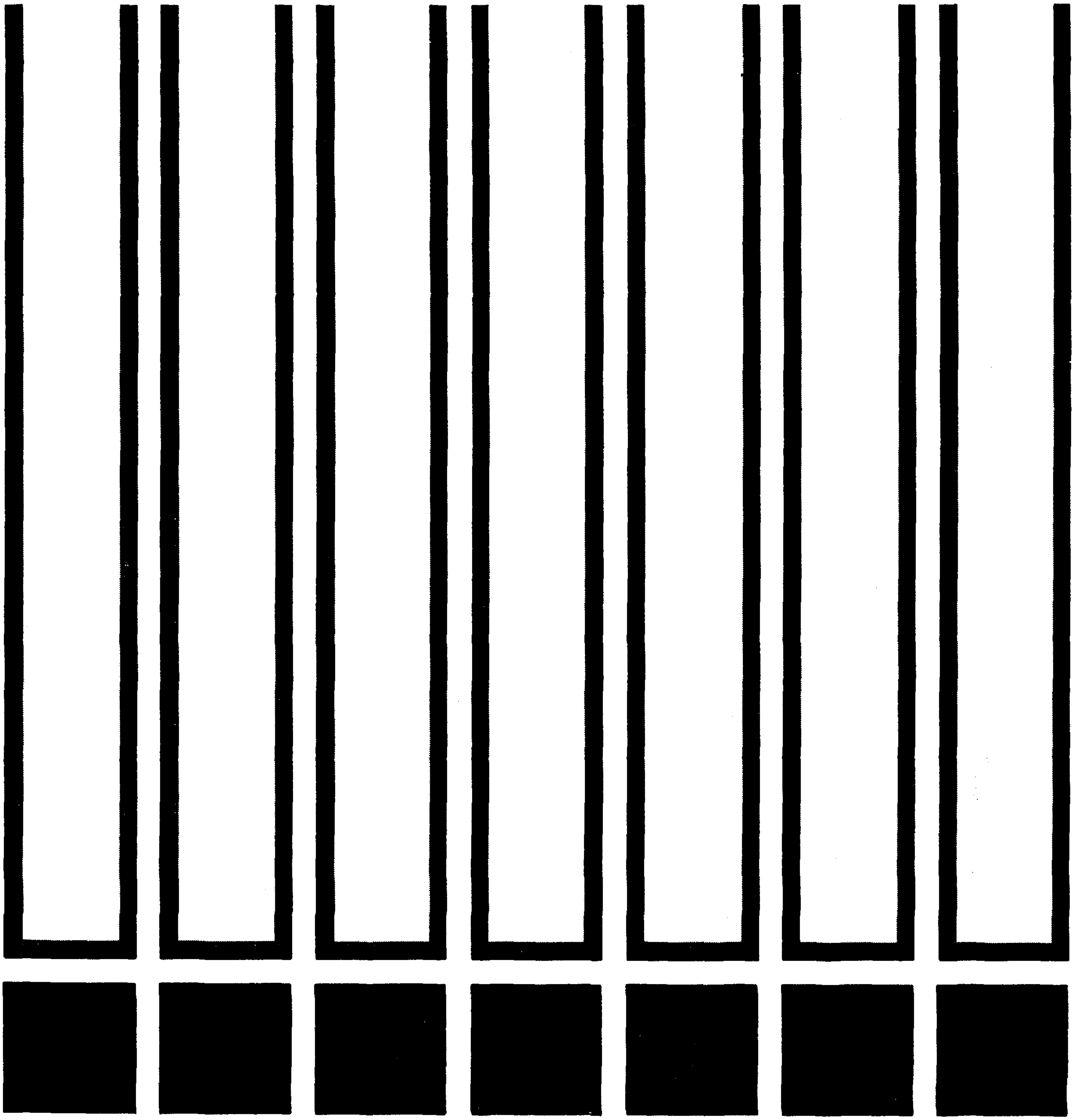
criteria for a recommended standard . . . .
occupational exposure to

inorganic fluorides



criteria for a recommended standard . . . .

OCCUPATIONAL EXPOSURE
TO
INORGANIC FLUORIDES

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Center for Disease Control
National Institute for Occupational Safety and Health
1975

HEW Publication No. (NIOSH) 76–103

PREFACE

The Occupational Safety and Health Act of 1970 emphasizes the need for standards to protect the health and safety of workers exposed to an ever-increasing number of potential hazards at their workplace.

The National Institute for Occupational Safety and Health has projected a formal system of research, with priorities determined on the basis of specified indices, to provide relevant data from which valid criteria for effective standards can be derived. Recommended standards for occupational exposure, which are the result of this work, are based on the deleterious effects of exposure. The Secretary of Labor will weigh these recommendations along with other considerations such as feasibility and means of implementation in developing regulatory standards.

It is intended to present successive reports as research and epidemiologic studies are completed and as sampling and analytical methods are developed. Criteria and standards will be reviewed periodically to ensure continuing protection of the worker.

I am pleased to acknowledge the contributions to this report on fluoride by members of my staff and the valuable constructive comments by the Review Consultants on Fluorides, by the ad hoc committees of the American Industrial Hygiene Association and the American Medical Association, by Robert B. O'Connor, M.D., NIOSH consultant in occupational medicine, and by Bruce J. Held on respiratory protection. The NIOSH recommendations for standards are not necessarily a consensus of all the consultants and professional societies that reviewed this criteria document on fluorides. Lists of the NIOSH Review Committee members and of the Review Consultants appear on the following pages.

John F. Finklea, MD

John F. Finklea, M.D.
Director, National Institute for
Occupational Safety and Health

The Office of Research and Standards Development, National Institute for Occupational Safety and Health, had primary responsibility for development of the criteria and recommended standard for fluoride. University of Washington, School of Public Health and Community Medicine developed the basic information for consideration by NIOSH staff and consultants under contract No HSM-99-73-36. Bryan D. Hardin had NIOSH program responsibility. Patricia G. Heitman served as criteria manager.

REVIEW COMMITTEE
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

Keith H. Jacobson, Ph.D.
Office of Research and Standards
Development

Stanley J. Reno
Regional Consultant DHEW
Region VIII

Herbert E. Stokinger, Ph.D.
Division of Laboratories and
Criteria Development

Ex Officio:

Herbert E. Christensen, D.Sc.
Deputy Director, Office of
Research and Standards Development

Department of Labor Liaison:

David Lee
Office of Standards

NIOSH REVIEW CONSULTANTS ON FLUORIDES

John E. Boysen, M.D.
Director, Marion County Health Department
Ocala, Florida 32670

Miles O. Colwell, M.D.
Vice President, Health and Environment
Aluminum Company of America
Pittsburgh, Pennsylvania 15219

Edward P. Radford, M.D.
Professor of Environmental Medicine
School of Hygiene and Public Health
The Johns Hopkins University
Baltimore, Maryland 21205

Frank A. Smith, Ph.D.
Associate Professor of Radiation Biology (Toxicology)
School of Medicine and Dentistry
University of Rochester
Rochester, New York 14642

Lawrence Stein, Ph.D.
Chemistry Division
Argonne National Laboratory
Argonne, Illinois 60439

A.J. Trommershausen
Director, Industrial Hygiene Services
Kaiser Industries Corporation
Oakland, California 94666

CRITERIA DOCUMENT: RECOMMENDATIONS FOR AN OCCUPATIONAL EXPOSURE STANDARD FOR INORGANIC FLUORIDES

Table of Contents

I. RECOMMENDATIONS FOR AN INORGANIC FLUORIDE STANDARD

III. BIOLOGIC EFFECTS OF EXPOSURE

IV. ENVIRONMENTAL DATA AND ENGINEERING CONTROLS

V. DEVELOPMENT OF STANDARD

I. RECOMMENDATIONS FOR AN INORGANIC FLUORIDE STANDARD

The National Institute for Occupational Safety and Health (NIOSH) recommends that worker exposure to inorganic fluorides be controlled by requiring compliance with the following sections. The standard is designed to protect the health and safety of workers for up to a 10-hour workday, 40-hour workweek over a working lifetime. Compliance with all sections of the standard should prevent adverse effects of exposure to fluorides in the workplace air. Fluoride is measurable by techniques that are valid, reproducible, and available. Sufficient technology exists to permit compliance with the recommended standard. The standard will be subject to review and revision as necessary.

Fluorides (abbreviated as "F" throughout this document) are defined as:

Those compounds of fluoride which are inorganic solids at normal workroom temperatures (20 C), and are compounds without radioactive elements, and are compounds having components which do not have exposure limits more restrictive than that proposed here for fluoride. In addition this definition includes any gaseous fluorides which are emitted simultaneously with particulate fluorides as defined above.

"Occupational exposure to fluorides" is defined as exposure above one-half the recommended workroom environmental limit.

Section 1 - Environmental (Workplace Air)

(a) Concentration

Occupational exposure shall be controlled so that no worker is exposed to a concentration of fluorides greater than 2.5 mg of F (combined ionic fluoride, atomic weight 19) per cubic meter of air determined as a time-weighted average (TWA) exposure for up to a 10-hour workday, 40-hour workweek.

(b) Sampling and analysis

Procedures for sampling, calibration of equipment, and analysis of fluoride samples shall be as provided in Appendices I and II, or by any method shown to be equivalent in precision, accuracy, and sensitivity to the methods specified.

Section 2 - Medical

(a) Medical Examinations

Comprehensive medical examinations including complete urinalysis shall be made available to all workers subject to exposure to fluorides prior to placement and biannually thereafter. These examinations should place emphasis on musculoskeletal, pulmonary, and gastrointestinal symptoms, and kidney dysfunction. Evidence of kidney dysfunction or moderate to severe osteosclerosis should be evaluated by a physician for consideration of disqualification for placement in fluoride exposure. Employees who experience breathing difficulty while using respirators shall be evaluated by a physician to determine the ability of the worker to wear respirators. In addition, a radiological examination of the male pelvis with proper gonadal shielding should be considered when preplacement examinations are conducted and every 6 years when the average of preshift urinary fluoride concentrations for the preceding 6 years exceeds 4.0 mg/liter. The physician evaluating the X-rays shall have knowledge of the radiological signs of fluorosis, and shall be made aware of the fluoride exposure of the worker.

(b) Biologic Monitoring

Urinary postshift F analysis shall be made available at an interval not exceeding every 3 months to at least one-fourth of all workers subject to occupational exposure to inorganic fluorides. Participating workers shall be rotated to provide all exposed workers the opportunity for urinalysis every year. Spot urine samples shall be collected at the conclusion of the workshift after 4 or more consecutive days of exposure. Urinary preshift F analysis shall be made available to all exposed workers at least annually. Preshift spot samples shall be collected at the start of the workshift at least 48 hours after a previous occupational exposure. Results shall be corrected for a specific gravity of 1.024. Procedures for sampling and analysis shall be as described in Appendix II.

If an individual's postshift urinary F level exceeds 7.0 mg/liter, preshift spot urine samples shall be collected within 2 weeks at the start of a workshift at least 48 hours after a previous occupational exposure and a repeat postshift spot sample shall be collected at the conclusion of the workshift. This shall be done at the end of the workweek in which the preshift sample is collected. If the F level of the second sample is above either the preshift limit of 4.0 mg/liter or the postshift limit of 7.0 mg/liter, steps shall be taken to evaluate dietary sources, personal hygiene, basic work practices, and environmental controls.

In case the group (job classification) median postshift urinary F levels exceed 7.0 mg/liter, the working environment shall be evaluated through an industrial hygiene survey and steps shall be taken to ensure compliance with the environmental limit. Urinary F analyses shall be performed monthly until the cause of elevated urinary F has been corrected as demonstrated by a return of the group median to a value not exceeding 7.0 mg/liter. The primary method of control will be engineering and work practices. Use of administrative controls for the individual or group can also be considered.

(c) Medical Records

Medical records including information on all biologic determinations shall be available to the medical representatives of the employer, of the Secretary of Labor, of the Secretary of Health, Education and Welfare, and of the employee at his request. These records shall be kept for the duration of employment plus 20 years.

Section 3 - Labeling (Posting)

(a) Containers of materials composed of fluoride in quantities comprising 5% or more of the total content shall bear the following label in addition to or in combination with labels required by other statutes, regulations, or ordinances:

WARNING
CONTAINS FLUORIDES
MAY BE FATAL IF SWALLOWED

Avoid breathing dusts, fumes, or mists of solutions.
Use with adequate ventilation.
Wash thoroughly after handling.
For skin or eye contact, flush with plenty of water.
If swallowed, get medical attention immediately.
Store away from acids.

(b) Fluxes containing fluorides shall bear the following label in addition to or in combination with labels required by other statutes, regulations or ordinances:

CAUTION
CONTAINS FLUORIDES
FUMES HARMFUL IF INHALED

Use with adequate ventilation.
Do not get on skin or in eyes.
Wash thoroughly after handling.

(c) Containers of welding rods with coatings comprised of fluorides shall bear the following label in addition to or in combination with labels required by other statutes, regulations, or ordinances:

CAUTION
COATING CONTAINS FLUORIDES
FUMES HARMFUL IF INHALED

Use with adequate ventilation.

(d) The following warning sign shall be affixed in a readily visible location at or near entrances to areas in which respirators are required for protection against occupational exposure to fluorides. The sign shall be printed both in English and in the predominant language of non-English-speaking workers, if any, unless they are otherwise trained and informed of the hazardous areas. All illiterate workers shall receive such training.

CAUTION
FLUORIDE EXPOSURE AREA
RESPIRATORY PROTECTION REQUIRED

Section 4 - Personal Protective Equipment and Work Clothing

(a) Protective clothing

(1) The type of protective clothing required will be dictated by the chemical nature and the physical form of the fluorides present and the duration and magnitude of worker exposure. The primary purpose of protective clothing is twofold: the worker must be protected from the effects of eye or skin contamination; and fluoride-contaminated clothing must not be allowed to present an exposure hazard to persons outside the workplace, eg, in the worker's home. Typical items of protective clothing include coveralls, shoe covers, caps, gloves, and goggles. Control practices may include provision of lockers for separation of protective clothing and street clothing, frequent changing of protective clothing, or provision of a vacuum or other mechanical means of removing gross contamination without producing significant airborne concentrations of fluorides. The type and manner of use of protective clothing shall be determined by professional occupational health personnel.

(2) Personnel working with acidic solutions of fluorides shall wear impervious gloves, shoe covers or boots, and aprons. Unless eye and face protection is afforded by a respirator hood or facepiece, chemical goggles and face shields shall be worn. Before removing protective

clothing, the worker shall rinse off fluoride solutions with clean water.

(3) All required eye protection shall conform to 29 CFR 1910.133.

(4) All protective clothing and equipment shall be maintained in a clean, sanitary, and workable condition.

(b) Respiratory Protection

(1) Engineering controls shall be used wherever feasible to maintain fluoride concentrations below the prescribed limit. Compliance with the permissible exposure limit may not be achieved by the use of respirators except:

(A) During the time period necessary to install or test the required engineering controls.

(B) For nonroutine operations such as a brief exposure to concentrations in excess of the permissible exposure limit as a result of maintenance or repair activities.

(C) During emergencies when air concentrations of fluoride may exceed the permissible limit.

(2) When a respirator is permitted by paragraph (b)(1) of this Section, it shall be selected and used pursuant to the following requirements:

(A) For the purpose of determining the type of respirator to be used, the employer shall measure the atmospheric concentration of fluoride in the workplace initially and thereafter whenever process, worksite, climate, or control changes occur which are likely to increase the fluoride concentrations; this requirement shall not apply when only atmosphere-supplying positive pressure respirators will be used. Odors, irritation, or other symptoms of exposure do not provide adequate warning of exposure to concentrations of fluorides above the recommended limit. The employer shall ensure that no worker is being exposed to fluoride in excess of the environmental limit because of improper respirator selection, fit, use, or maintenance.

TABLE I-1

RESPIRATOR SELECTION GUIDE

Maximum Use Concentration (Multiples of TWA Limit)	Respirator Type
Less than or equal to 5x (no acid gases present)	Single use, dust respirator.
Less than or equal to 10 x (no acid gases present)	Half-mask respirator with replaceable dust filter, or supplied-air, demand type respirator with half mask facepiece.
Less than or equal to 10x (acid gases present with particulates)	Half-mask respirator with replaceable combination acid gas/dust filter* cartridge or supplied-air demand type respirator with half-mask facepiece.
Less than or equal to 50x (no acid gases present)	Full facepiece with replaceable high efficiency filter or supplied-air demand type respirator with full facepiece.
Less than or equal to 50x (acid gases present with particulates)	Full facepiece with replaceable combination acid gas/high efficiency filter* or supplied-air demand type respirator with full facepiece.
Less than or equal to 100x (no acid gases present)	Powered air purifying respirator with replaceable high efficiency filter* and a full facepiece, hood, or helmet, or supplied-air pressure-demand or continuous flow type with full facepiece, hood or helmet.

TABLE I-1 (continued)

RESPIRATOR SELECTION GUIDE

Maximum Use Concentration (Multiples of TWA Limit)	Respirator Type
Greater than 100x (with or without acid gases present)	Self-contained breathing apparatus, pressure-demand mode with full facepiece or combination supplied-air pressure-demand type, full facepiece, with auxiliary self-contained air supply.
Evacuation or escape from known or unknown concentrations if acid gases present.***	Gas mask or mouthpiece respirator with acid gas canister.**

---

* Since some filter elements, because of materials of construction, are not suitable for exposure to hydrogen fluoride, manufacturer's recommendations should be followed.

** Above 50 mg/cu m of HF, severe eye irritation can occur, necessitating the use of eye protection for escape (eg full facepiece or chemical goggles).

***Any dust respirator can be used for escape from known or unknown concentrations of fluorides in particulate form only.

(B) A respiratory protection program meeting the requirements of 29 CFR 1910.134 as amended shall be established and enforced by the employer.

(C) The employer shall provide respirators in accordance with Table I-1 above and shall ensure that the employee uses the respirator provided.

(D) Respiratory protective devices described in Table I-1 shall be those approved under the provisions of 30 CFR 11, or approved under a Bureau of Mines schedule if the approval is recognized as being valid by the appropriate government agency or agencies.

(E) Respirators specified for use in higher concentrations of fluoride may be used in atmospheres of lower concentrations.

Section 5 - Informing Employees of Hazards from Fluorides

At the beginning of employment in an area in which there exists occupational exposure to fluorides, employees shall be informed of the hazards, relevant symptoms of acute overexposure, chronic fluorosis, appropriate emergency procedures when applicable, and proper conditions and precautions for safe use or exposure. Instruction shall include, as a minimum, applicable information in Appendix III. The information shall be posted in the work area and maintained on file, and be readily accessible to the worker at all places of employment where fluorides are involved in unit processes and operations.

A continuing educational program shall be instituted to ensure that all workers have current knowledge of job hazards, proper maintenance procedures, and clean-up methods, and that they know how to use respiratory equipment and protective clothing.

Information as required shall be recorded on the "Material Safety Data Sheet" shown in Appendix III or on a similar form approved by the Occupational Safety and Health Administration, US Department of Labor.

Section 6 - Work Practices

(a) Control of Airborne Fluorides

Fluorides shall be controlled at sources of dispersion by means of effective and properly maintained local exhaust ventilation or by fully enclosed process operations. Other methods may be used if they are shown to effectively control atmospheric levels of fluorides below the recommended environmental limit. Ventilation systems shall be subjected to regular preventive maintenance and cleaning to ensure maximum effectiveness, which shall be verified by periodic airflow measurements. An adequate source of make-up air shall be provided. Ventilation system discharges should be provided with air cleaners as required to meet ambient air quality standards.

(1) Ventilation for electroplating or other open surface tanks containing solutions of fluorides shall conform to the requirements of 29 CFR 1910.94.

(2) Ventilation for welding with electrodes having coatings containing fluorides shall conform to the requirements of 29 CFR 1910.252.

(b) Housekeeping

Workrooms, including remote points of accumulation, shall be kept clean and free of fluoride residues by any method which avoids redispersion in the workroom atmosphere in concentrations exceeding the environmental limit.

(c) Waste Disposal

Disposal of fluorides and discharges of fluorides into the environment should conform to all applicable local, state, and federal regulations.

(d) Storage

Fluorides should be stored in dry, well-ventilated areas, and should be protected from contact with acids.

(e) Handling

Fluorides shall be handled so as to minimize dispersion in the workroom atmosphere and into the community air. Skin contact shall be minimized.

(f) Unauthorized Entry

In areas where there is occupational exposure to fluorides a policy defining unauthorized entry shall be established. Considerations shall include the chemical and physical nature of the fluoride present, fluctuations in the airborne concentration, the duration of exposure, and any need for medical clearance.

Section 7 - Sanitation Practices

(a) Plant sanitation shall meet the requirements of 29 CFR 1910.141.

(b) Good personal hygiene shall be enforced. Hands, arms, and face shall be thoroughly washed prior to eating and at the end of the shift. Facilities shall be provided for this purpose in conformance with 29 CFR 1910.141(d).

(c) Emergency eye-wash fountains shall be conveniently placed in work areas where solutions of fluorides or acidic fluorides are handled or used.

(d) Food shall not be stored, prepared, dispensed (including vending machines), or eaten in areas where occupational exposure to fluorides may occur. Drinking (except from authorized fountains), smoking, and chewing (eg, tobacco or gum) shall be prohibited in fluoride exposure areas. The employer shall furnish an uncontaminated area for these purposes in conformance with 29 CFR 1910.141(g). Tobacco or foodstuffs should not be carried in fluoride-contaminated clothing.

(e) Protective clothing shall be changed at least weekly. Appropriate locker rooms shall be available for changing into required protective clothing in conformance with 29 CFR 1910.141(e). Where necessary, storage facilities sufficient to prevent gross contamination of street clothing shall be provided where bulk fluorides are handled under dusty conditions.

Section 8 - Monitoring and Recordkeeping Requirements

Work areas shall not be considered to have fluoride exposure if work area air levels, as determined on the basis of an industrial hygiene survey or by the judgment of a compliance officer, do not exceed half of the environmental limit. Records of these surveys, including the basis for concluding that air levels are below half the limit, shall be maintained until a new survey is conducted. Surveys shall be repeated when any process change indicates a need for reevaluation or when in the judgment of the compliance officer it is deemed necessary. Requirements set forth below apply to areas in which there is fluoride exposure.

Employers shall maintain records of work area exposures to fluorides based upon the following sampling and recording schedules:

(a) In all monitoring, samples representative of the exposure in the breathing zone of employees shall be collected. An adequate number of samples shall be collected to characterize every operation or process. The minimum number of representative workshift TWA determinations for an operation or process shall be based on the number of workers exposed as provided in Table I-2.

(b) The first sampling of the work environment shall be completed within 6 months of the promulgation of a standard incorporating these recommendations.

(c) Samples of the work environment shall be taken within 30 days after first operation of a new process or process changes.

(d) Samples shall be collected at least semiannually in accordance with Appendix I for the evaluation of the work environment with respect to the recommended environmental limit.

(e) Monitoring of an operation or process shall be repeated within one month when the fluoride concentration has been found to exceed the recommended environmental limit. When the results of the repeat survey

TABLE I-2

SAMPLING SCHEDULE

Number of Employees Exposed	Minimum Number of Employees Whose Individual Exposures Shall Be Determined
1-20	50% of the number of workers
21-100	10 plus 25% of the excess over 20 workers
over 100	30 plus 5% of the excess over 100 workers

reveal a concentration in excess of the limit, suitable controls shall be initiated and monitoring shall continue at monthly intervals until the adequacy of these controls has been demonstrated.

(f) Monitoring of an operation or process shall also be repeated when group median biological monitoring reveals urinary fluoride excretion above the postshift level of 7.0 mg/liter, adjusted to a specific gravity of 1.024. Monitoring of the work environment in response to high postshift urinalyses should be conducted on the same day as the repeat postshift urinalysis required by section 2.

(g) Records of all sampling, medical examinations, and biological monitoring shall be maintained for the duration of employment plus 20 years. Records shall be maintained so that exposure information is available for individual employees and shall indicate the type of personal protective devices, if any, in use at the time of sampling.

II. INTRODUCTION

This report presents the criteria and the recommended standard based thereon which were prepared to meet the need for preventing occupational diseases arising from exposure to inorganic fluorides. The criteria document fulfills the responsibility of the Secretary of Health, Education, and Welfare, under Section 20(a)(3) of the Occupational Safety and Health Act of 1970 to "...develop criteria dealing with toxic materials and harmful physical agents and substances which will describe exposure levels at which no employee will suffer impaired health or functional capacities or diminished life expectancy as a result of his work experience."

The National Institute for Occupational Safety and Health (NIOSH), after a review of data and consultation with others, formalized a system for the development of criteria upon which standards can be established to protect the health of workers from exposure to hazardous chemical and physical agents. It should be pointed out that criteria for a recommended standard should enable management and labor to develop better engineering controls resulting in more healthful work environments and mere compliance with the recommended standard should not be used as a final goal.

These criteria for a standard for fluorides are part of a continuing series of criteria developed by NIOSH. The proposed standard applies only to the processing, manufacture, and use of fluorides as applicable under the Occupational Safety and Health Act of 1970. The standard was not designed for the population-at-large, and any extrapolation beyond occupational exposures is not warranted. It is intended to (1) protect against injury from fluorides, (2) be measurable by techniques that are valid, reproducible, and available to industry and official agencies, and (3) be attainable with existing technology.

The extensive industrial use of and exposure to fluorides, the concern over adverse effects of fluorides on plants and farm animals, and perhaps most importantly, the interest in adding fluoride to the water supply to reduce dental caries have stimulated a great amount of scientific controversy as well as scientific study. It is not feasible for any review of fluorides and their effects on health to discuss all the available evidence. This criteria document has attempted to review critically that evidence bearing on a recommended occupational health standard for inorganic fluorides. For additional information on the effects of fluorides on health, other scientific references are available and recommended. Among these are the National Academy of Sciences-National Research Council review Fluorides, [1] the World Health Organization monograph Fluorides and Human Health, [2] the chapter by Hodge and Smith "Fluorides and Man" [3] found in the 1968 issue of the Annual Review of Pharmacology, the chapter by Hodge and Smith "Biological Properties of Inorganic Fluorides," found in the series Fluorine Chemistry, [4] and "Pharmacology of Fluorides" found in the 1966 and 1970 issues of the Handbook of Experimental Pharmacology. [5]

The criteria and recommended standard contained herein is intended to apply to occupational exposure to inorganic fluorides whose primary effect at low exposure levels is bone deposition. Bone deposition in excess of minimal change may in certain circumstances prove to be detrimental to human health. Other risks of fluoride exposure, such as those posed by strong oxidizing substances (eg, elemental fluorine) and gaseous fluorides alone (eg, hydrogen fluoride) will be the subject of additional or different recommendations in later criteria documents.

III. BIOLOGIC EFFECTS OF EXPOSURE

Extent of Exposure/Hazard

Fluorides are ubiquitous, being found in air, water, food, [6] coal, [7,8] and generally in the earth's crust. [6] The primary source of fluoride compounds used in industry is the mineral fluorspar (fluorite, calcium fluoride, CaF_2). [9,10] Secondary sources are cryolite (sodium aluminum fluoride, Na_3AlF_6) and phosphate rock. Phosphate rock as a source of F is expected to increase in importance as the supply of fluorspar becomes exhausted. Rock phosphate contains a lower concentration of F than fluorspar, but there is more of it available. US fluoride reserves of calcium fluoride as of 1970 were 5.4 million tons, while in rock phosphate they were 279 million tons. [9]

Greenland has been the world's only supplier of natural cryolite for over 100 years. [11] Mining ceased in 1969, but shipments of natural cryolite continue in small amounts from stockpiles. Most cryolite is now made synthetically from fluorspar or fluosilicic acid, a byproduct of fluoride-containing gases emitted during the processing of phosphates to make fertilizer. [12]

Production and consumption of phosphate rock rose at an increasing rate from 1950 to 1968, dipped sharply in 1969, then resumed its rise reaching 1967 levels again in 1971. [13] Florida and North Carolina accounted for about 83% of the total domestic production. The western states accounted for 11% and Tennessee 6%. Marketable production in 1971 was almost 39 million tons while apparent consumption [13] was almost 28 million tons. It was estimated [14] that 200,000 tons of "fluorine" (presumably fluoride) were evolved in processing 22 million tons of phosphate rock in 1968.

With minor fluctuations caused by economic conditions and work stoppages, the consumption of fluorspar in the United States has increased steadily. In 1939, mine shipments of fluorspar totaled 182,771 short tons, imports for consumption were 16,302 short tons, and consumption was 176,800 short tons. [15] Comparable figures in 1971 were: mine shipments--272,071 short tons, imports for consumption--1,072,405 short tons, and consumption--1,344,742 short tons. [12] In 1939, the chief consumer of fluorspar was the steel industry, using 73% of the total fluorspar consumed. [15] The steel industry used only 43% of the total fluorspar consumed in 1971. [12] The chemical industry used 14% of total fluorspar consumed in 1939, [15] while in 1971 the chemical industry used 37%. [12] The end use of fluorspar consumed is shown in Table XII-1. In addition to the direct use of fluorspar (938 tons) by the aluminum industry, the equivalent of about 200,200 tons in the form of hydrofluoric acid and the equivalent of 51,000 tons in the form of fluosilicic acid obtained as a byproduct from the fertilizer industry were utilized indirectly. [12] Both hydrofluoric acid and fluosilicic acid provided a source of fluoride in the manufacture of cryolite which is used by the aluminum industry in the electrolytic reduction process. [11] The largest users of fluorides are the steel industry, the aluminum industry, and the chemical industry.

Table XII-2 lists occupations with potential exposure to fluorides, indicating the wide and varied industrial use. NIOSH estimates that approximately 350,000 workers are potentially exposed to fluorides in the United States. A representative list of fluorides and their pertinent properties is presented in Table XII-3.

Because of air quality requirements, attention is being directed toward recycling fluoride emissions with subsequent reuse in industrial processes, and toward replacing fluorides with substances of lesser hazard, cost, and greater availability. [12,14,16] This may lead to some reduction of fluoride exposure in the future.

Historical Reports

In 1932, Moller and Gudjonsson [17] first reported roentgenologic bone changes in man due to occupational fluoride exposure. Their investigation of the incidence of silicosis among workers in dust-generating industries included a cryolite processing plant in Copenhagen. Because cryolite contained small amounts of quartz and the processing operation was very dusty, a radiologic survey was performed to determine the presence or absence of silicosis in exposed workers. Although half of the workers examined showed evidence of silicosis, the concern in this condition was greatly overshadowed by the unexpected increased density of ribs, claviculae, and cervical vertebrae in many X-rays of the thorax. Most of the workers had no complaints related to the increased bone density, although some complained that they had been troubled with "rheumatism." Follow-up roentgenologic examination of the entire bone system revealed increases in osseous radiopacity, calcification of ligaments, and hyperosteoses of varying degree and extent in 30 of the 78 cryolite workers examined. From earlier animal studies, the authors concluded that the osseous changes observed in the cryolite workers were caused by the fluoride contained in the cryolite. Consequently, Roholm

[18] studied in great detail the health status of cryolite workers and in 1937 published the results in his classic monograph. He confirmed the findings by Moller and Gudjonsson [17] and described the clinical and pathoanatomical picture of occupational fluoride intoxication.

Effects on Humans

(a) Sources of F

F in small amounts is present in the great majority of human foods and beverages as summarized in Table XII-4. Cholak [19] reviewed data up to 1959, and Hodge and Smith [20] provided a compilation from that date to 1970. The widest variation in concentration was found in meat and fish, while fruits and vegetables had the lowest range; grains were intermediate. Tea sometimes contained relatively high amounts of F. [19] Machle et al [21] found in 1939 that there was no correlation between the concentration of F in locally grown food and the amount of F in the local water supply. However, as pointed out by Marier and Rose [22] in 1966, food containing 0.2-0.3 ppm increased in F content to 0.6-1.0 ppm F after canning using fluoridated water. Waldbott [23] found elevated levels of fluoride in foods grown and produced near a phosphate fertilizer plant. Two reviews, those of McClure [24] and of Armstrong and Knowlton, [25] gave 0.2-0.3 mg as the approximate average daily F intake through food. Much of the work on F in foods is now over 20 years old. During this time more communities have begun fluoridating their water.

F in water has received much more emphasis than F in food, perhaps because it has been observed to vary more. Cholak [19] tabulated data on water samples from 49 states (excluding Alaska) and found concentrations ranging from 0 to 33.5 ppm F. Although the highest values were rarely encountered, 40 states had some values over 1.0 ppm and 32 states had some over 2.0 ppm F.

Cholak [19] reported the fluoride content of the particulate matter collected from the air in 15 American cities. The values were 0-0.1 ppb (approximately 0.08 μg F/cu m) for 9 cities, 0.2-0.3 ppb (approximately 0.16-0.23 μg F/cu m) for 3, and 0.4-0.5 ppb (approximately 0.3-0.39 μg F/cu m), for 3; Los Angeles had a concentration of 1.10 ppb (approximately 0.86 μg F/cu m). Sufficient air was sampled so that the aliquot chosen for analysis always contained over 5 μg F. In 1945, Agate et al [26] took 11 samples in a 5-month period from the atmosphere at various points up to 1 mile outside a factory in Scotland which produced aluminum by the electrolyte process. He found values ranging from 0.02 to 0.22 mg F/cu m. The highest concentration was found 200 yards from the factory.

(b) Absorption and Excretion of F

(1) Ingestion and Excretion

Machle et al [27] found by balance studies that a young man in Cincinnati in 1941 absorbed about 80% of the F he ingested in food and water. Largent [28] conducted balance studies on 5 groups of 2 subjects each, residing in areas with 5 different naturally occurring F concentrations in water. Duplicate samples of all food and water ingested (normal diets of their choosing) were analyzed over periods ranging from 45-160 days. As shown in Table III-1, Largent concluded that 8 subjects appeared to be storing F at low rates. However, he noted that part, if not all, of the fluoride assumed to have been stored may have been lost through perspiration which was not analyzed.

TABLE III-1

FLUORIDE BALANCE STUDIES ON 10 HUMAN SUBJECTS

F conc. in drinking water, ppm	F Intake mean mg/day		Excretion mean mg/day		Balance
	Food	Fluids	Urine	Feces	
2	1.17	2.42	2.86	0.36	+0.37
	0.94	2.55	2.88	0.19	+0.26
5.55	1.35	8.14	8.32	0.66	+0.51
	1.32	3.81	4.49	0.63	+0.01
6.1	1.02	6.74	8.09	0.45	-0.78
	1.98	6.34	7.76	0.59	-0.03
8	3.13	12.4	12.9	1.37	+1.2
	2.47	11.3	10.4	1.42	+2.0
20	1.48	20.8	12.3	1.39	+8.6
	1.16	15.6	11.4	1.88	+3.5

From Largent [28]

Spencer et al [29] reported balance studies on 10 male ambulatory patients who were in good physical and nutritional state and who did not have renal, gastrointestinal, or skeletal disease. Each patient was provided a regulated diet during a control period of 14-24 days and a dietary F supplement for a period of 22-42 days. A postdietary F evaluation period of 12-24 days followed. The F balances were all strongly positive and retention ranged from 1.57 to 2.19 mg/day in the control period (average 42.9% of intake), 4.58-6.61/day in the F supplement period (average 39.3% of intake), and 0.9-1.65 mg/day in the postsupplement evaluation period (average 31.9% of intake). The F balances did not consider loss of F in sweat, but environmental conditions were such as to minimize sweating.

McClure and Kinser [30] studied over 1,900 urine samples from US Army recruits and male high school students. A definite relationship was observed between urinary F levels and the F content of domestic water. At concentrations approximating 0.5 ppm of fluoride in the local domestic water, urine specimens showed a detectable increase in F. Urinary F levels continued to be proportional to water fluoride levels until a level of about 5.0 ppm of F in the local water supply was reached.

Oral absorption and excretion of F have also been studied under conditions of changing periods of intake. Machle and Largent [31] carried out elaborate balance studies on Largent himself using as F supplements sodium fluoride, calcium fluoride in solution and in powdered form, bone meal, and cryolite. Duplicate samples of food, urine, and feces were analyzed, but sweat was not included. Results are shown in Table III-2. Although not part of the balance studies, a number of determinations of F excreted in perspiration revealed that no significant loss of F in sweat occurred during the experiment.

The combined data showed a straight-line correlation between absorption and urinary excretion as well as a linear relation between absorption and retention of F. [31] From this experiment, the authors concluded that (1) gastrointestinal F absorption was dependent on the solubility of the salt; (2) at normal levels of fluoride intake, the combined urinary and fecal output equaled the intake; (3) at supplementary levels of oral intake, storage occurred; (4) the quantity of F retained in storage correlated directly with gastrointestinal absorption; and (5) there existed a relationship between urinary excretion and retention of F.

TABLE III-2

FLUORIDE BALANCE STUDIES OVER AN 89-WEEK PERIOD

Mean Values for Each Period of Study MgF/Day

Time in Weeks	Supplementary F Form	Mg F Added	F in Ingesta Food	F in Ingesta Fluid	F in Excreta Urine	F in Excreta Feces
20	Control Period	0	0.15	0.30	0.37	0.04
14	NaF	6	0.13	0.35	2.42	0.19
11	Control	0	0.15	0.26	0.39	0.04
4	CaF2 soln	6	0.10	0.15	2.17	0.27
4	Control	0	0.11	0.21	0.52	0.06
7	Control	0	0.09	0.18	0.55	0.05
3	CaF2 solid	6	0.16	0.28	1.78	2.44
5	Bone meal	6	0.09	0.22	1.08	3.99
12	Control	0	0.19	0.40	0.42	0.04
3	Cryolite	6	0.17	0.44	2.66	1.52
12	Control	0	0.15	0.24	0.41	0.04

From Machle and Largent [31]

Zipkin et al [32] studied residents of Montgomery County, Maryland, and Grand Rapids, Michigan, before and after fluoridation of the water supply. F concentration in Grand Rapids was 0.1-0.2 ppm before fluoridation; the prefluoridation level for Montgomery County was not given. Some 360 male adults and children participated in the Montgomery County study, and, although it is not entirely clear, probably about the same number of male children in the Grand Rapids study. In Montgomery County, 11 urine collections were made on each subject over a period of 3.5 years. As early as 1 week after fluoridation, the ratio of urinary fluoride to water fluoride was 1.1 to 1 for adults. Six weeks after water supplies were fluoridated to 1.0 ppm, Montgomery County adults were excreting 1.0 ppm, but children at ages 5-14 did not reach this level until about 3 years following fluoridation. In Grand Rapids, the prefluoridation urine values averaged 0.2 ppm F. After fluoridation to 1.0-1.1 ppm F, approximately 5 years elapsed before the concentration of fluoride in the urine of the school children reached the level present in the drinking water.

Starting from the opposite point, with persons previously exposed to higher F concentrations who then experienced lower ones, Largent [33] reported contrasting findings. He and 2 other subjects had previously stored, as documented by balance studies, 1700, 500, and 1900 mg F, respectively; a fourth subject, age 20, had a life-long exposure to water containing 2.4-4.4 ppm F. No X-ray changes were seen in any subject. The subjects discontinued supplementary F. Over the next 19 days, during which duplicate samples of all food and drink (intake averaged 0.4-0.8 mg F/day) and all urine and feces were analyzed for F, an excess of F was excreted which seemed to follow a straight-line relationship (depletion of stored F vs time). Data plotted for periods which greatly exceeded 19 days could be fitted to an exponential curve of the form: $Y = A + Be^{-kt}$. In that equation, Y was the amount of stored fluoride remaining at any given time, t; B was the maximum amount that would be mobilized and excreted eventually if the low level of fluoride intake continued without change; k was a constant which, along with B, determined the slope of the curve at any particular time; A was the difference obtained by subtracting the value of Be^{-kt} from Y when t = 0, and this constant represented the amount of fluoride that in all likelihood would never be recovered from the tissues. The difficulties recognized in applying the equation included: (1) the accuracy of the value assigned to A was recognized to be suspect since A included all the errors associated with the unmeasured loss of F through sweat as well as unmeasured storage of F prior to the initiation of any balance study; and (2) the value assigned to k had to be adjusted from 0.01 during the first 19 days to 0.04 beyond the 40th week of the balance studies in order to achieve a good data fit.

Largent [34] assumed that when persons previously exposed to levels of 3-4 ppm F in the drinking water were switched to water of lower F content, urinary excretion of F would continue, with a resultant negative F balance until a new steady state was achieved. Applying this concept to the experience of residents of Bartlett, Texas, for example, he predicted a new equilibrium in 200-225 weeks after the change in drinking water from 8 ppm to 1.1-1.5 ppm, at which point urine excretion would be 1.1-1.5 ppm. According to the study of Likins et al [35] of Bartlett residents in 1956, the initial drop in urine concentration was very rapid. The urinary F concentration of 51 white male subjects over age 20 involved in the study dropped from an average of 7.7 mg/liter to 5.1 mg/liter in one week, although they still averaged 2.5 mg/liter 27 months later. The period of observation was not sufficient to prove or disprove Largent's projection of equilibration at 225 weeks, but the observations by Likins et al over the 113 weeks of the study were consonant with Largent's hypothesis.

The promptness of urinary excretion of F following oral absorption was also studied by Largent. [28] He ingested 4 capsules, each containing 3.95 mg rock phosphate, over a period of 6 hours. Urinary excretion rose to maximum levels of 10.6 mg F/liter within 2 hours of ingesting the first capsule and dropped to less than 5.0 mg F/liter within 8 hours after ingestion of the last capsule.

As demonstrated by Zipkin and Leone, [36] after a single dose of NaF providing 5 mg F, about 20-25% was found in the urine in 3 hours. In the 8 normal adults participating in this experiment, urine values (after F supplement) returned to presupplement levels in less than 24 hours. The authors stated that the remaining F was excreted in the feces and perspiration; however, no test results were reported.

The appropriateness of spot urine samples was considered by Largent and Ferneau. [37] A combined total of 17 24-hour specimens was obtained from 3 unexposed workers and 18 24-hour specimens from an exposed worker with high urinary F excretion (see Table III-3). During the course of collecting each 24-hour sample, one spot sample of 25-100 ml was collected separately; all other volumes voided during the period were combined. It was concluded that spot urine samples could be used to discover areas within industry that need special attention.

Rye [38] conducted several studies of the correlation between spot urine samples and 24-hour collections. Two individuals were studied 5 times each, including 2 control periods without exposure and one 8-hour period of exposure to 2.4 ppm gaseous F, during the 24-hour urine

TABLE III-3
COMPARISON OF SPOT AND 24-HOUR URINE SAMPLES

	24-hour collections mg F/liter			Spot Samples mg F/liter		
	N	Average	SD	N	Average	SD
3 Nonexposed workers	17	0.437	0.184	17	0.381	0.14
1 Exposed worker	18	17.44	3.35	18	18.00	6.68

From Largent and Ferneau [37]

collection. Since the only details were given in the author's narrative, it is quoted, in part, as follows:

"The general pattern of fluoride excretion in all of these trials was essentially the same. The rate of excretion increased in the first 2 hours from 0.5 mg per liter to as high as 4 mg per liter, and climbed somewhat erratically to a peak of 7-8 mg per liter in the ensuing ten hours. Following this, it receded erratically, with secondary peaks of 4-5 mg per liter, and usually returned to the range of 0.5-1 mg per liter by evening of the same day or early morning of the following day. In the succeeding 24-72 hours, without any additional exposure, the rate of excretion rose at approximately the same time of day as during exposure to a peak of 4-5 mg per liter....the total urinary excretion of fluoride amounted to 4.5 mg on the day of exposure, 3.8 mg for the first postexposure day, 3.6 for the second postexposure day, and 1.2 mg for the third postexposure day." In contrast, Zipkin et al [32] collected 3 spot samples at 3-hour intervals during a 24-hour period from each of 9 subjects who were drinking water containing 1.0 ppm F. There was no evidence of a circadian rhythm. The total group of spot samples ranged from 0.3 to 2.0 mg F/liter, while the variation among 24-hour samples was 0.5-1.2 mg F/liter. The greatest individual range was 0.6-2.0 mg F/liter in spot samples; the corresponding 24-hour value was 1.2 mg F/liter. The average concentration in the 24-hour samples was 0.9 mg F/liter.

The effect of correcting to a standard specific gravity has been explored. Buchwald [39] presented experimental data on urinary F excretion to demonstrate the effectiveness of the specific gravity correction in correlating the results of urine analyses. Urinary F in spot specimens of

5 normal persons living in the Slough area of England and using water from the same source (F content not reported) was found to range from 0.50 to 1.40 mg F/liter. The specific gravity (SG) of the specimens ranged from 1.010 to 1.028. Correction to 1.016 gave F concentrations of 0.80-0.81 mg F/liter. An SG of 1.016 was used since it was considered to be a more realistic standard for persons in the British Isles than the value most widely used for specific gravity correction in the United States, ie 1.024. Elkins et al [40] calculated the mean specific gravities of 35,506 urine samples covering a time span of about 25 years and concluded that although the true mean value was 1.022, correction to 1.024 should continue to be the practice as this value is the one most often cited in the US literature. The authors considered the actual value used to be less important than the concept of correcting to some standard value when results are to be compared with data reported by other investigators. The adjustment can be accomplished by the formula utilized by Elkins and Pagnotto [41] (see Appendix II).

Several investigators have reported on the extrarenal excretion of absorbed F. Crosby and Shepherd [42] compared F excretion in urine and sweat under reasonably controlled laboratory conditions. Urine and sweat were collected from 2 subjects, a 43-year-old male and a 22-year-old female under 4 test conditions, each test repeated 3 times: (1) no supplementary F intake and no induced sweating; (2) no supplementary F intake and induced sweating; (3) 3 mg sodium fluoride (in solution) supplementary F intake and no induced sweating; and (4) 3 mg sodium fluoride supplementary intake and induced sweating. Under conditions of sweating in which the subjects were enclosed in plastic bags for periods of 1.5-2 hours at an air temperature of about 104 F, significant quantities of F were recovered in sweat as shown in Table III-4.

McClure et al [43] conducted a study on 5 young men aged 19-27. The men spent 8 hours each day for 5 days in an experimental chamber designed to control environmental temperature and humidity. Two environmental conditions were maintained, one "comfortable" and the other "hot-moist." During "comfortable" conditions the temperature was maintained at 84-85 F and the relative humidity was 49-52%. During "hot-moist" periods the temperature was 100-101 F and relative humidity was 66-70%. The men were provided with daily F supplements of 1.5 mg or 3.0 mg. Urinary and fecal excretions were collected during all actual test periods. A record of dietary F intake was kept throughout the study.

The dermal excretion of F was determined by thoroughly washing the

TABLE III-4

RESULTS OF EXPERIMENTAL FLUORIDE EXCRETION STUDIES--TWO HUMAN VOLUNTEERS

Subject	Mg F intake	F in sweat, ppm	F in urine, ppm
Male	0	0.30	0.64
	0	0.47	0.49
	0	0.72	0.40
	3	0.93	2.16
	3	1.14	2.24
	3	0.98	1.63
Female	0	0.91	1.47
	0	0.51	0.85
	0	0.84	0.90
	3	1.71	1.83
	3	1.24	1.43
	3	1.45	1.20

From Crosby and Shepherd [42]

body prior to entrance into the experimental test chamber, collecting all sweat running from the body during the experimental exposure, and by a final thorough body washing at the termination of the exposure period. Tests indicated the methods used accounted for well over 90% of the sweat excreted.

The authors found that sweat contained 0.3-1.8 ppm F during F digestion periods. Two weeks after terminating the test regimen, sweat contained 0.2 to 0.3 ppm F. The data indicated that the concentration of F in sweat was related to the quantity of F ingested. Elimination of absorbed F was found to be primarily distributed between urine and perspiration in accordance with environmental conditions. The kidney excreted an average of 77% of the absorbed F, while the skin excreted 23% through insensible perspiration during "comfortable" periods. During "hot-moist" periods the kidney eliminated on the average 49% of the absorbed F, while the sweat during the 8-hour period accounted for an average of 44%.

Largent [34] found that one subject excreted 0.015-0.019 mg F/hour in sweat with little change when F intake was increased to 6 mg NaF/day. A second subject, beginning at the basal level, excreted up to 0.065 mg/hour in sweat when the intake was raised to 3 mg daily. Machle et al [27] found that "under conditions of high fluorine intake" (not otherwise specified) the value for F in sweat, 0.514 mg/liter (SD 0.066), was approximately 1/6 the value for urine, 3.256 mg/liter (SD 0.093). The preceding studies demonstrate that estimates of stored F may be high unless F loss through sweating is taken into account.

Saliva concentrations of F were measured by Domzalska and Lassocinska [44] simultaneously with urine values. Three age groups (18-24 years, 26 to 45 years, and 46-78 years) totaling 112 residents had mean saliva F concentrations of 1.73, 0.89, and 0.82 ppm, respectively. Concomitant average urine values for each age group were 1.12, 1.15, and 0.96 mg F/liter, respectively. A study by Carlson et al [45] with the radioisotope F-18 showed that less than 1% of the activity was recovered in parotid samples when 1 mg F in solution labeled with 5 microcuries of radioactive fluoride (F-18) was ingested by each of 2 test subjects. Excretion concentration ratios (urine to plasma) of radioactive F ranged from 7.3 to 118.3 in one subject and from 12.0 to 46.2 in the second; F clearances were always much less than creatinine clearances with ratios ranging from 0.39 to 0.70 in one subject and from 0.21 to 0.53 in the second.

(2) Inhalation and Excretion

In 1951, Collings et al [46] conducted a study on 2 human subjects who had no previous exposure to F other than dietary intake in order to determine their responses to inhaled F. Their mean rates of urinary F excretion before exposure were 0.9 and 1.2 mg F/day, respectively. The 2 subjects were exposed during one 8-hour period in the laboratory to an atmosphere containing an average of 5.0 mg F/cu m (a range of measurements of 3.9-8.7 mg F/cu m). The F dust was produced from rock phosphate containing 3.5% F. About 95% of the dust particles were 5m in diameter or smaller. Atmospheric samples were taken serially throughout the entire 8 hours of exposure. No symptoms were noted during the exposure. Urine specimens were collected at 2-hour intervals during exposure and for 2 days after exposure. Thereafter, specimens were collected at progressively longer intervals for three more days. The urine samples consisted of the entire output of urine during the interval represented. In persons not sweating excessively, this output is approximately 1500 ml/day. [47] Analysis of the 2-hour serial urine specimens showed a rapid rise in urinary output during the exposure with peaks reached 2-4 hours after cessation of exposure. Within 12-16 hours after exposure, urine levels had fallen almost to normal levels, although a slight elevation above preexposure levels continued into the following day. The daily urinary output data of the 2 subjects were as shown in Table III-5.

The authors [46] stated that the elevation over base levels still present in the second 24-hour collections would mean a small carryover in the case of an employee returning to work the following day. This study documented rapid absorption and excretion of F when respirable rock phosphate dust was inhaled. In a follow-up study, Collings et al [48] compared urinalysis results of 2 subjects exposed 6 hours each to atmospheric fluorides averaging 4.8 mg F/cu m in an industrial environment (further details not given). One subject was considered to have had no

Table III-5

URINARY FLUORIDE LEVELS OF TWO SUBJECTS
WITH NO PREVIOUS FLUORIDE EXPOSURE

Subject	Baseline urinary F mg F/day	Urinary output, mg F/24 hours			
		day 1 exposure	day 2	day 3	day 4
A	0.9	10.0	1.96	1.28	0.73
B	1.2	9.95	2.99	1.10	0.98

From Collings et al [46]

previous F storage; the other subject had worked approximately 8 years in a plant involving exposure to fluorides. An excretion curve plotted for the 2 subjects was remarkably similar for the first day following exposure; thereafter for an additional and concluding period of slightly less than 3 days, the previously exposed worker maintained a consistently higher base level than the subject with no previous F exposure. The authors suggested that the persistent high base line might have been due to a cumulative effect from repeated exposures. Urinary excretion values ranged from about 0.1 to 1.3 mg F/2 hours and were determined on the entire urine output during the study. An additional 7 industrial workers exposed to airborne F concentrations of 2.5 to over 10 mg F/cu m were found to excrete urinary F concentrations ranging from 5.3 to 23.0 mg F/liter (postshift). The preshift values 48 hours later were 2.0-4.2 mg F/liter. Among 8 other workers with exposures ranging from 0 to over 10 mg F/cu m, preshift urinary F concentrations ranged from 0.8 to 4.0 mg F/liter. Their postshift excretion was 7.0-17.0 mg F/liter. [48]

Largent and Ferneau [37] presented some observations on the urinary excretion of F in workmen engaged in various types of work in 3 magnesium foundries: L, B, and P. One air sample collected in foundry L contained 6 mg F/cu m. In 2 air samples taken from foundry B, the concentrations were 3.1 and 8.4 mg F/cu m. The mean of 123 spot urine samples in foundry L was 2.23 mg F/liter with the means of 4 subsamples within foundry L ranging from 1.56 to 3.45 mg F/liter. In foundry B the mean of 12 spot samples was 3.13 mg F/liter. Six spot urine samples in foundry P averaged 6.39 mg F/liter. The authors noted that the highest urinary F concentrations in foundry L were found in the core spraying area despite the existence of extensive ventilation and the use of respirators by exposed workers.

In 1960, Rye [38] reported on clinical observations of employees in the production of phosphate rock and triple superphosphate (TSP). In the production of TSP, 3 employees were exposed to airborne F during the handling of the cured product. The atmospheric F concentration was 2-4 ppm, being composed of approximately 60% dust and 40% gas (HF). The TSP dust was extremely fine, the major portion of the airborne particles being below 1 m in size. For 67 days, 24-hour urinary F samples were collected from an employee who worked 8 hours a day, 5 days a week with an occasional 2-3 hours of overtime. A background concentration of 0.8-1.3 mg F liter had been established on this employee prior to undertaking the study. Within 2-3 hours from the start of exposure to TSP dust the rate of urinary F excretion increased and reached a maximum in the 8 hours following cessation of exposure. The rate of urinary F excretion remained above the control value during this entire 67-day period and returned to near preexposure levels only after a vacation of an unspecified period of time. The F excretion was lowest, 3-4 mg/liter, at the beginning of the workweek and climbed to a level of 8-9 mg F/liter over the 5-day exposure period. After a 2-day absence from exposure, the rate was again 3-4 mg F/liter. A significant observation was the occurrence of periodic episodes of diuresis followed by a drop of F excretion approaching the preexposure level. This occurred in 5- to 7-day cycles throughout the study but never on weekends. Neither the amount of urine excreted during diuresis, nor the duration of these episodes was given. Rye [38] determined that in comparison to urinary F excretion in employees exposed to HF, a greater length of time was required for the F excretion to reach its maximum following exposure to

TSP dust. Also the return to normal F excretion took longer for TSP dust exposure (5-6 days) than for gaseous HF exposures (3 days). This difference was explained by the author on the basis of lung clearance. The delay in lung absorption of the dust deposited in the lower respiratory tract, in effect, continued the apparent effects of exposure beyond the duration of the atmospheric exposure.

Two studies on the urinary F excretion of arc welders exposed to F-containing fumes from low-hydrogen electrodes were reported by Smith [49] in 1968. On the average, 30% of each 8-hour working shift was spent welding. The range of values of 154 air samples was 0.1-10.0 mg F/cu m, with a median of 1.3 mg F/cu m. The time-weighted average for an 8-hour day was calculated as 0.4 mg F/cu m. Urinary F excretion levels as determined in spot samples obtained from 24 welders with a mean age of 41.5 years are shown in Table III-6. These samples were obtained at the end of a normal workshift.

TABLE III-6

URINARY FLUORIDE EXCRETION VALUES COMPARED TO FLUORIDE EXPOSURE VALUES AMONG 24 WELDERS

			Postshift spot sample mg F/liter		TWA exposure mg F/cu m
	N	Mean	Range	SD	
Welders	24	2.4	0.5-4.8	0.23	0.4
Confined*	5	2.9	1.5-4.8	0.55	1.0
Enclosed**	10	2.7	1.2-4.8	1.70	0.3
In the open***	9	1.8	0.5-2.8	0.22	0.3
Control subjects	16	1.0	0.4-1.5	1.08	--

*Confined = welding performed inside compartments
**Enclosed = welding performed inside a compartment having at least one side open to the atmosphere
***In the open = welding performed in open air or large workshop
From Smith [49]

The difference between the urinary F concentrations for welders in confined and enclosed conditions was not statistically significant. This was explained by the difficulty in defining enclosed areas which ranged from near-confined to near-open. This became apparent with the relatively high standard deviation (1.70 mg F/liter) of urinary F concentrations in the enclosed welders.

Krechniak [50] reported on F hazards in welders in 1969. Air samples were obtained from the welders' breathing zones in an experimental welding center utilizing automatic welding machines and in a shipyard production hall (see Table III-7).

Significant differences in urinary F concentrations were noted in groups of workers classified by years of exposure. The highest F concentrations in welders operating automatic welding machines were explained by the different degrees of exposure. When the authors used a maximum allowable urinary concentration of 2.8 mg F/liter, the basis for

TABLE III-7

COMPARISON OF URINARY FLUORIDE VALUES
OF AUTOMATIC MACHINE WELDERS VS MANUAL WELDERS

	F concentration in air, mg/cu m		No. of workers	Urinary F before shift, mg/liter	
	gas	particulate		average	range
Experimental center	2.29	0.36	29	3.2	(0.2-8.9)
Production hall	1.4	0.32	122	1.3	(0.1-3.9)
Control subjects			10	0.3	(0.15-0.55)

From Krechniak [50]

which was not documented, 46% of the automatic welding machine operators exceeded this value whereas only 6% of the manual welders did so.

Mangold and Beckett [51] reported environmental F levels and urinary F excretions in workers involved in silver brazing utilizing a flux containing 27% potassium fluoride. Their comparative study of exposure values and urinary F values in 30 different jobs in a pipe shop and 30 different jobs aboard ships revealed an average of 0.51 mg F/cu m (range 0.28-0.80) aboard ship vs an average of 0.12 mg F/cu m (range 0.02-0.16) in the pipe shop. Urinary F values were lower aboard ship, averaging 0.6 mg F/liter (range of 0.3-3.6) than in the pipe shops where the average was 1.5 mg F/liter (range 1.0-2.0). The authors were primarily concerned with nitrogen dioxide exposures and offered no explanation for the inverse relationship between airborne and urinary fluorides.

(c) F in Tissues Other than Bone

(1) Blood

Blood samples for F analysis were collected by Smith et al [52] from an unspecified number of volunteer residents of Rochester, New York (water contained 0.06 ppm F) and Newburgh, New York (water contained 1.0-1.2 ppm F). In Rochester, there was no detectable F in 30 of 34 fasting blood samples; of the samples containing measurable quantities of fluoride, one contained 0.4 g F/100 ml blood, one had 1-2 g F/100 ml and 2 were in the range of 3-6 g F/100 ml. A comparison of nonfasting blood samples between the 2 cities revealed a mean blood F concentration of 1.38 g/100 ml in Rochester and a mean of 3.93 g/100 ml in Newburgh where the F concentration in water was approximately 20 times that of Rochester.

Table XII-5 summarizes F values in blood from studies appearing in the literature during the 22-year period 1950-72. Taves [53] attributed the differences in values prior to 1970 to differences in methods of determination, which were yet to be resolved. Such difficulties in the measurement of F in blood prior to 1970 have precluded the widespread use of blood F values in studies of F metabolism. Hall et al [54] described a simplified method of measuring F in biological fluids using the ion-selective electrode. Recovery by this method of F added to plasma was 101.5%. Tests of 26 serum samples obtained from humans undergoing routine medical examinations prior to entering a hospital for various surgical procedures revealed a mean F concentration of 37 g/liter with a range of 16-55 g/liter.

(2) Other Tissues

Roholm [18] compared autopsy tissues of 2 cryolite workers with those of a nonexposed subject. Wide differences in F content were noted between lung and stomach values as shown in Table III-8. It appears from these data that the lung retains inhaled F.

Placental tissue was studied by Gardner et al [55] in Rochester, New York, where water contained 0.06 ppm F, and Newburgh, where water supplies contained 1.0-1.2 ppm F. The Rochester samples had a mean concentration of 0.74 ppm F while the Newburgh samples averaged 2.09 ppm F. These values were above corresponding blood values which averaged 0.014 and 0.040 ppm, respectively.

Feltman and Kosel [56] reported their observations on pregnant human subjects receiving 1.2 mg F as NaF daily in tablet form compared to those

TABLE III-8

SOFT TISSUE FLUORIDE LEVELS FOUND AT AUTOPSY
(mg/100 g dry tissue)

Subjects	Stomach	Liver	Spleen	Kidney	Heart	Lung
Cryolite worker currently exposed	1.2	0.44	0.67	2.4	0.70	79.2
Cryolite worker not exposed for 17 months	1.6	0.32	0.66	2.3	0.53	10.8
Unexposed subject, accidental death, age 50	1.4	0.50	1.80	1.1	0.81	0.73

From Roholm [18]

who did not. The water in the study area was reported free of F, but the type of analysis and actual values were not given. Placental tissue in 141 specimens contained 26.4-784 µg F/100 g (average 149.2) compared to the control range, for 251 subjects, of 15.9-764 µg (average 105.8). The authors suggested that the controls may have derived additional F from pharmaceuticals, but this might also apply to the test subjects. The differences in cord blood in 162 specimens showed the same overlap: 4.4-225.6 µg F/100 ml in 162 specimens from the NaF group (average 32.7) and 0.34-292 µg F/100 ml in 240 controls (average 12.9). Although placental tissue may have increased F, Armstrong et al [57] reported maternal artery values averaging 0.11 ppm (SE ±0.011) and maternal venous values of 0.10 (SE 0.008) based upon 14 patients at Caesarian section.

(d) Effects of F

(1) Gastrointestinal Effects

The most striking demonstration of acute gastrointestinal effects was the accidental ingestion of sodium fluoride by some 263 persons at a mental hospital, 47 of whom died. As reported by Lidbeck et al, [58] some complained of numbness of the mouth. Extremely severe nausea, vomiting, and diarrhea occurred abruptly and at times simultaneously. Vomitus and stools were bloody in many instances. Abdominal burning and cramp-like pain accompanied these symptoms. Samples of the scrambled eggs, which were the vehicle, revealed 3.2-13% NaF (it had been mistaken for powdered milk) and F was easily demonstrated by etch test from ash of organs at autopsy of poisoned inmates. A more precise estimate of the amount ingested could not be made. Death can occur in man in 2-4 hours after a single dose of 2.5-5.9 g of F is ingested. [1]

(2) Bone Effects

There have been a number of studies on the F content of human bone after various exposures (Table XII-6). With nonindustrial exposure at 1 ppm or less in drinking water, the reported range of F concentrations in bone extended as high as 160 mg/100 g bone, [59] but most studies have yielded considerably less. [18,28,60-64] The importance of water as a source of F was indicated by studies of Zipkin et al. [60] They studied a number of individuals exposed to varying concentrations of F in their drinking water, as summarized in Table XII-6. According to their data [60] and to that of others, [18,28,61-65] a positive correlation could be demonstrated between the amount of F to which the individual was exposed in drinking water and the amount that was found in bone at autopsy.

McClure et al [62] autopsied 2 women, 1 exposed for the last 24 years of her life to water containing 0.2 ppm F, and the other for her last 34 years to drinking water containing 8 ppm F. Comparative bone F data are shown in Table III-9.

Although the values for subject B are some 10 times the amount of F found in subject A, X-ray and clinical studies prior to death showed no abnormalities. Tissues were not examined microscopically.

Call et al [61] conducted autopsies on a group of 88 Utah residents over 15 years of age at death who had resided in industrial areas. They found the mean fluoride concentration of the bones of these subjects to be 56.8 mg/100 g. No disorder associated with fluorides was evident in any person. The bone F content varied somewhat by anatomic site, showing the

TABLE III-9

EFFECT OF INGESTED FLUORIDE ON BONE RETENTION

	Woman A	Woman B
Duration of exposure, years	24	34
F content of drinking water, ppm	0.2	8.0
Bone F, mg/100g dry fat-free bone		
femur	62	551
tibia	67	-
fibula	80	512
calvarium	92	653
lumbar vertebra	77	550
rib	89	630
dorsal vertebra	86	-
iliac crest	78	-
thoracic vertebra	-	550
misc. vertebra	-	530
pelvis	-	577
acromioclavicular joint	-	540

From McClure et al [62]

highest mean concentration in lumbar vertebrae at 61.5 mg/100 g dry fat-free bone (SD 3.39, SE 0.38), and the lowest in iliac crests (51.0 mg/100 g, SD 3.14, SE 0.4). These subjects were on average 61.8 years of age. Call et al in the same study conducted autopsies on a second group of 37 subjects who were less than 15 years of age at death or who had resided in Utah less than 10 years. The average bone fluoride concentration of the subjects was 44.6 mg F/100 g of bone ash. The average age of these subjects was 43.5 years. The analysis of water supplies in 1 of the 3 counties involved revealed less than 0.5 ppm of fluoride. The difference between the average bone fluoride concentrations of the two groups was significant ($p < 0.01$). It was concluded that this difference was due in part to differing age and perhaps also to the differing degrees of exposure. It was also concluded that for both groups fluoride content of bone increased with age. This last statement was not further elaborated upon.

The fluoride in the ribs of 23 humans of different ages with no known abnormal exposure to fluoride was studied by Glock et al. [63] At 2 London hospitals, ribs were taken during autopsies of persons resident in London where certain sources of water contained up to 0.5 ppm F. The results (presented in Table XII-6) suggested that the F in bone increased by up to 0.002% for each year of age.

Rare instances possibly representing neurologic complications of fluoride osteosclerosis have been observed in western countries. Goldman et al [66] reported a case of neuropathy attributed to an F-induced bone density increase in a 55-year-old Indian man of the American Southwest. There was bilateral flexion contracture of the knees and elbows, complete rigidity of the neck and spine, and decreased sensation to light touch and pin prick over the dorsum of the right foot. The patient drank water containing 4-8 ppm F and also often drank tea, which frequently contains large amounts of F (see Table XII-4). The amount of intake was not further quantified. X-rays revealed generalized increased bone density of the spine, ribs, and pelvis, suggestive of skeletal fluorosis. Extensive accompanying osteophytosis was present. An extracted tooth showed 61.4 mg/100 g ("bulk" canal), and 483.9 and 529.9 mg F/100 g in the crown and supragingival calculi. The latter values suggest a concomitant increased bone F.

Sauerbrunn et al [59] reported a case in which initial hyperreflexia, hypalgesia of the feet, and fasciculations in the arms progressed to spastic quadraparesis attributed to spinal cord compression by bone disease. At autopsy, the sternum, calvarium, and vertebrae were extremely dense. The vertebral column was massive but the spinal cord was symmetrical in its upper cervical portion and disclosed no gross abnormality. A toxicologic study revealed an elevated bone fluoride content of 610 mg/100 g of dried bone. The patient had spent most of his life in parts of Texas, drinking water from wells where, the authors commented, analyses had shown F concentration of 2.4-3.5 ppm. According to his sister he also drank a considerable amount of tea. He apparently did not have any industrial exposure. The authors believed the principal disorder to be due to amyotrophic lateral sclerosis. The probable cause of the fluoride effect was prolonged polydipsia.

While most investigations have focused on possible adverse effects of fluoride in bone, a significant effort has been made to establish the presence of beneficial effects. Ast and Chase [67] compared the incidence of decayed, missing, and filled (DMF) teeth in 6- to 12-year-old children in 2 New York towns before and after fluoridation of the water supply in one. The DMF ratio per 100 erupted permanent teeth was 20.2 in Kingston in examinations conducted in 1945-1946, and 20.6 in Newburgh in examinations conducted in 1944-1945. Beginning in May 1945, the F content of the water supply of Newburgh was increased from about 0.1 ppm F to 1.2 ppm; the water supply of Kingston remained essentially F-free. After an average of 6.4 years of F experience in Newburgh, the DMF rate was 11.9 as opposed to 22.5 for Kingston. In 1956, Schlesinger et al [68] evaluated populations of the same 2 towns for purposes of comparing the state of health exclusive of DMF rates. Some 800 children in Newburgh and 700 in Kingston were enrolled during the first 3 years of the study. By the end of the 10-year followup, about 40% were lost from the study. At final examination, the children ranged in age from 7-18 years. There was no statistical difference between children of the 2 towns in respect to height and weight, onset of menstruation, bone density on X-ray, skeletal maturation by X-ray, hemoglobin levels, urinalysis (including Addis counts), ophthalmologic and otologic examinations (including visual acuity and hearing), or trends in stillbirth and infant mortality rates. No F values, other than the concentration in drinking waters, were given. Bernstein et al [69] studied lumbar spine films taken of outpatients over age 45 willing to participate in the study. There were 300 persons exposed to a high F content in the water (4-5.8 ppm) and 715 from an area where it was only 0.15-0.3 ppm. Frequency of collapsed vertebrae was greater in women where F was low ($p < 0.05$ for ages 55-64, and $p < 0.01$ for ages 65 and over). A lower incidence of increased bone density was seen in both men and women living in the low F area than in the high F area ($p < 0.01$ for women age 55 and over, and $p < 0.05$ for men 55-64). Actual numbers in each group were not given.

Leone et al [70] presented X-ray findings of a 10-year study of 190 persons, approximately one-half residing in an area with 8 ppm F in drinking water, and the other half in an area where the water supply contained 0.4 ppm F. In the high F group, 13 of 89 persons were considered to have increased bone density. Ten years later only one new person exhibited an increase in bone density. In the control group, 3 of 101 persons initially had increased bone density with no change occurring 10 years later. A complete autopsy, including chemical analysis of bone, was performed on one participant from the high F area who died of a cerebral vascular accident at age 79 shortly after the conclusion of the X-ray study. Chemical analyses revealed approximately 6 times the F content (0.6 mg/100 g bone) of the same bone from individuals from nonfluoride regions. Her X-rays had revealed a moderate degree of increased bone density with some coarsened trabeculation.

Stevenson and Watson [71] reviewed medical records covering an 11-year period from 1943-1953 encompassing 170,000 X-ray examinations of the spine and pelvis. All records were of patients residing primarily in Texas and Oklahoma where water supplies contained up to 8 ppm of fluoride. A diagnosis of fluoride osteosclerosis could be made in only 23 patients. These patients lived in communities whose water supplies contained 4-8 ppm of fluoride. All patients were given clinical examinations including serology, sedimentation rate, red and white blood cell counts, differential white cell count, hemoglobin, blood urea, and routine urine studies. In addition, most had electrocardiographic investigations. In each case, the authors failed to find any relationship between X-ray findings and the clinical diagnosis of the patient's condition. It was concluded that fluoride osteosclerosis did not develop in patients drinking water with an F content of less than 4 ppm and in the 23 cases of osteosclerosis clinical examination failed to establish any relationship between the roentgenologic findings and the clinical diagnosis of the patient's condition.

In Sweden, Alffram et al [72] reported studies on bone mineral mass in women without known bone disease. Sixty-two healthy women, 45-72 years of age, were chosen in Malmo (water F 0.2-0.4 ppm), while 47 women were selected in Billesholm (water F 4-6.8 ppm). The women had been residents of either town for at least 20 years. The evaluation of bone mass was based on 3 measurements: the combined thickness of the 2 cortices measured with a caliper on anteroposterior (AP) radiograms of the second metacarpal, the combined thickness of the cortici on AP radiograms of the proximal end of the radius, and the attenuation of a photon-beam passing through the femur laterally in the epicondylar area. While no single comparison showed an outstanding correlation between bone mass, age, and F content of water, all parameters indicated the same tendency for bone density to be greater in Billesholm residents. There was no difference in the age-adjusted incidence of fracture of the femur in 3 cities in Sweden with water F concentrations under 0.1, 0.2-0.4, and 0.8-1.2 ppm.

The limit of the ability of the skeleton to store fluoride has not been established. In theory, sufficient fluoride to convert the hydroxyapatite of bone to fluoroapatite could be deposited, about 35,000 ppm fluoride, according to Hodge. [73] In the view of Largent [28] and

Zipkin et al, [32] the fact that this value has not been observed is due largely to the ability of the kidney to achieve a steady state at a level below such absolute saturation.

The dependence on urinary excretion to limit F retention in bone is emphasized by the experience with patients with renal disease. Evidence of this comes from a variety of sources. Linsman and McMurray [74] reported the case of a young soldier with marked anemia. Chronic renal disease was present and the patient died 3 months later. During life his bones showed marked increase in density on X-ray, with thickened trabeculae, most pronounced in the trunk. The patient had no musculoskeletal complaints. At autopsy, bone ash from a lumbar vertebral body contained 750 mg F/100 g and from the sternum 690 mg/100 g. The lower left bicuspid tooth contained 450 mg F/100 g ash. The patient, as the history was reconstructed, was probably exposed during all but 2 years of his life to drinking water containing 1.2-5.7 ppm F. One sister, who shared this exposure, had moderately severe mottling of the enamel but no osteosclerosis on X-ray.

Juncos and Donadio [75] reported 2 cases of fluorosis with renal failure. The first, an 18-year-old boy, had experienced an increase in thirst and urination since infancy. The only other history of renal disease was intermittent proteinuria. There was an atrophic right kidney with no visualization of the left kidney. The artesian well water which supplied his drinking water contained 2.6 ppm F. The teeth were mottled and very opaque. X-rays showed increased bone density. The second case was that of a 17-year-old girl with progressive azotemia associated with urinary tract infection. Since infancy she had drunk large amounts of water containing 1.7 ppm F. Teeth were opaque with diffuse brownish mottling. X-ray of the spine showed blurring and rather coarse trabeculation. It was postulated by the authors that renal disease in both instances had led to increased intake and retention.

Call et al [61] found higher bone fluoride levels in persons with marked kidney disease compared to controls, but not in persons with lesser degrees of renal involvement. Comparative results are shown in Table III-10.

Two patients with known renal disease were studied by Largent. [28] When the subjects were on normal diets, little or no storage of F was apparent. When, however, extradietary fluoride was ingested at the rate of 6 mg/day in the form of sodium fluoride, one subject who was observed for 6 days stored 76.0% of the F ingested and the other subject observed for 13 days stored 81.5% of the F ingested. The explanation of the association of renal disease with greater bone burdens of F is not final. Although it was not mentioned by Largent, [28] it may be that patients with chronic renal

TABLE III-10

BONE FLUORIDE LEVELS AMONG SUBJECTS WITH AND WITHOUT KIDNEY DISEASE

Subjects, age 60-73	N	mg F/100 g dry, fat-free bone	
		Mean	SD
No kidney disease	16	55.7	33
Some pyelonephritis	10	57.0	20.6
Unilateral renal disease	4	64.5	26.7
Marked chronic bilateral pyelonephritis	5	128.2	41.7

From Call et al [61]

disease drink more water, [75,76] as well as excrete less F.

A special relation between intake and bone deposition exists in patients on renal dialysis with fluoridated water. With little or no renal excretion and a dialysate concentration at 1 ppm (about 6 times that of serum), Taves et al [77] found the average net gain by their patient to be 10 mg/dialysis. The patient received dialysis treatments every 2-3 weeks for almost a year. Her home water supply contained 0.2 mg F/liter. At autopsy, the ash of a vertebral bone specimen contained 550 mg F/100 g. The microscopic diagnosis of the bone was, however, mild to moderate osteoporosis plus a severe degree of osteomalacia and dissecting osteitis compatible with moderately severe renal osteodystrophy.

(3) F and Thyroid

Because F is a halogen one could postulate it might be a goitrogen. Maumeme in 1854 [78] suggested that F in certain waters accounted for the rather rapid development of goiter in persons exposed to them. Day and Powell-Jackson [79] studied 13 Himalayan villages where the water iodine was 0.001 ppm or less, on the assumption that at other localities higher iodine levels might protect against the goitrogenic effect of F. The authors found the prevalence of goiter generally increased with increasing concentration of F in the water. The prevalence of goiter was also found to correlate very well with the hardness of water. Since water hardness and fluoride levels were themselves correlated, it was not possible for the authors to conclude from the data that the association between fluoride and goiter was independent of that between water hardness and goiter.

On the other hand, Latham and Grech [80] found no correlation of F in water with goiter in an area in Tanzania with sufficient F content to cause an overall rate of 95% dental fluorosis. Singh et al [81] also found no association between water F and goiter in their Punjab study. Although the Punjab was affected by endemic goiter along its northern border, the fluorotic belt along its southern border had no increase in goiter. Furthermore, 20 patients with fluorosis had protein-bound iodine (PBI) levels ranging between 4-7 g/100 ml compared to 5-8 g/100 ml in normal controls in a nonfluorotic area. Leone et al [82] studied 106 persons who had used water containing 3.48 ppm F for more than 10 years and 109 persons using essentially F-free water for the same period. The distribution of PBI values given by class intervals for both groups showed no significant difference between the 2 groups.

Galletti and Joyet [83] found no appreciable uptake of radioactive F-18 by the thyroid in either normal humans or patients with hyperactive glands.

On the basis of these studies, [79-83] it can be concluded that fluorides do not interfere with the uptake of iodine by the thyroid, do not accumulate in the thyroid, and do not cause thyroid goiter.

(4) F as an Allergen

In 1938 Evang [84] reported an investigation of a Norwegian aluminum plant. Fluorides were utilized in the potroom operation. The operation involved filling carbon-lined iron pots with 500-600 kg of cryolite, partly in powder form and partly in the form of fused red-hot cryolite from a pot which was in operation. After electric current was passed through the pot and the cryolite fused, the pot was fed by hand with alumina and small amounts of cryolite and aluminum fluoride.

During the depression, the plant reduced its full operation consisting of 270-280 workers and then, beginning in 1936, gradually enlarged again. At the time of the investigation about 190 men were employed. A large number of workmen had been entirely away from this work for 4-6 years and had resumed work for 6 months or less, while the rest had worked on reduced time during the same 4-6 years.

In order to get the most complete information possible about the number of cases of asthma in the plant and in the district in general, all available sources of disease information were solicited. After eliminating duplicate names, 54 individuals were found who suffered from asthma, of these 32 were in the population outside of the plant: 11 were women, 11 were children under 14 years, and 10 were men. [84] Under the category "asthma" were included only the patients who had been described as having had severe attacks or shortness of breath of an asthmatic character, who reacted to the medicines ordinarily employed for asthma (adrenalin, potassium iodide, ephedrin, asthma powder, etc), and whose attacks had been followed by free intervals without symptoms from the lungs. Cases of chronic or often-recurring bronchitis or similar ailments were not included. The frequency of asthma in the plant (excluding 4 discharged workers) was 22/190 or 95/1000, while in the population outside the plant it was 32/3800 or 8/1000. [84] Subtracting all asthma patients with a known family tendency, the rates were 74/1000 and 5/1000, respectively. Approximately 110 of the 190 plant employees (about 3/5) worked in the potroom; only one case of asthma occurred in the workmen who had not worked in the potroom. Some of the potroom workers thought that they could refer the first attack to an accidental cause, most often to a "cussed pot" which, when being worked, gave off a particularly large amount of gas, the composition of which was unspecified. A total of 18 of the 22 workmen with asthma stated that they had no attacks at all when they were outside of the potroom, while 4, after their illness first occurred, also had an attack in other places, although most frequently at the plant.

Evang [84] commented on the lack of complete information regarding all cases of asthma in the district, the dependence on individual histories and records taken for other purposes, and the lack of a clear definition of "asthma." He felt, however, the prevalence of asthma was increased in potroom workers. The question that could not be answered within this study was the contribution to the onset of asthma made by other agents, enumerated by the author [84] as follows: CO, tar smoke, carbon dust, aluminum oxide, and water vapor in addition to HF, cryolite, and aluminum fluoride. No exposure data for any of these were available.

In 1960 Midttun [85] reported his work as a factory medical officer. On the basis of his experience and his contacts with engineers in Germany and colleagues in Yugoslavia, he instituted precautions in the aluminum factory where he worked in order to prevent the development of asthma in aluminum workers. Those who were to do pot work were carefully selected, ie, they were not accepted for work in the potroom if there was any family history of allergy or if they had had bronchitis previously. (Some of the potroom workers had been employed prior to Midttun's appointment, however.) Some 400 persons had some potroom assignment with exposures to HF and other gaseous F compounds, aluminum fluoride, cryolite, alumina, tar vapors, and sulfur dioxide. The surrounding climate was considered a good one for asthmatics; many asthma patients who moved to the area felt that the climate had a beneficial effect on their illness. Statistics of absence from work were not given, but reportedly indicated that in 1955, the year the new policies went into effect, there was less absence on account of respiratory tract disease among potroom workers than in other groups. These workers were considered a medically selected group. Nearly 80% were under 40 years of age, and approximately 60% were newcomers to the aluminum industry. The average eosinophil count was 90/cu mm with only 4% over 300. The same distribution was found in a control group of 50 office employees.

The majority of workers starting in the potroom for the first time developed nausea, headaches, and irritation of conjunctiva and respiratory passages as a result of exposure to dust, fumes, and smoke. [85] These symptoms were worst during the first few days of exposure and then generally disappeared. Four workers developed acute asthma on their first encounter with the potroom, and all 4 stopped work and left within a couple of weeks; they were therefore not examined more closely or recorded. From 1955 to 1957, 33 asthma cases were recorded out of about 400 potroom workers. Thirteen (39%) of these had a history of bronchitis and 4% a family history of asthma. Average length of exposure for the 33 cases was 4 years, ranging from 2 months to 30 years. Three of the affected workers left the factory, ie, one was granted a disability pension; the second was assigned to office work and had no additional asthma attacks, even though he still suffered from bronchitis; the third remained symptom-free. Twenty-one men were removed from the potroom; 3 were still affected by asthma and remained partly unable to work, the others were described as managing well, but had relatively long absences from work due to colds.

Nine men continued working in the potroom and were well when conditions were good. Eosinophil counts averaged 184/cu mm for the 33 men, with a range of 56-843. Intracutaneous tests with household dust extract and a representative dust extract from the potroom gave no definite positive results.

In 1958, 19 new asthma cases were reported [85] (2 acute) with 17 occurring in the last 4 months of the year when the total F air concentrations were twice as high as usual. One had a history of bronchitis and another had a family history of asthma. The average exposure time of this group was 3 years, ranging from 2 months to 10 years. Blood counts in 7 cases before asthma developed showed an average eosinophil count of 53/cu mm (40-100) and average leucocyte count of 5600 (4200-7400). During the attack, the average eosinophil count in all 19 cases was 376/cu mm (87-1000) and after the attack 166/cu mm (37-281). Leucocyte counts averaged 9000 (5700-17,100) during and 7450 after attacks. Sedimentation rates were normal.

In 1959, 2 new serious cases of status asthmaticus occurred [85] without prodromal symptoms in workers with 2 and 10 years of exposure. It was observed that workers who developed asthma while working on pots did well if they worked as far away from the pots as possible, while asthmatics who worked outside the potrooms had attacks when they were reexposed, for example, when skimming the tapping ladle. In 1958, a potroom malfunction resulted in the escape of a great deal of gas into the workroom air. The total amount of F released during this period was double the normal level of 1-2 mg F/cu m. The incidence of asthma among potroom workers increased at this time and a high incidence of serious dermatitis was reported.

Reduction of atmospheric F concentrations to normal levels resulted in a reduction of these health problems.

In discussing the findings, the author [84] concluded that the family history, previous bronchitis, as well as the clinical pictures, with eosinophilia and leucocytosis, suggested an allergic reaction to an F compound. The long exposure time before the occurrence of asthma, 3 years in most cases, suggested a long duration of sensitization. In summary, he reported an average of 10.8 cases/year of bronchial asthma over a 5-year period among an average of 400 workers.

The question of allergy to F in the nonoccupational environment has been raised by Shea et al [86] in several case reports. They reported the following: giant urticaria, dermatitis, stomatitis, diarrhea, glandular swelling, and allergic nasal disease--all believed to follow F exposure in dentifrices, vitamin drops, or other vehicles. In 2 of 7 individuals whose cases were reported in some detail, challenge on one occasion each with the suspected vehicle reproduced the symptoms; challenge was not attempted in the others. At the request of the United States Public Health Service, the executive committee of the American Academy of Allergy [87] reviewed case reports of allergy, including those of Shea et al [86] for the purpose of evaluating the possible relation of F to allergy. On the basis of the lack of correlation between the effects attributed to F and the recognized patterns of allergy as well as a lack of sufficient clinical and laboratory findings, these authorities made the categorical statement that there was no evidence of allergy or intolerance to fluorides as used in the fluoridation of community water supplies. [87]

(5) F and Enzymes

There have been a wide variety of biochemical systems studied to determine effects of F on enzymes. Based on an analysis of a number of published reports, Largent [34] listed the level of F in ppm which inhibited in vitro the enzyme specified: liver esterase 0.1; acid phosphatase, 2; enolase, 2; 5-nucleotidase 19; glutamine synthetase 19; citrullinase, 19; dehydrogenase, 25; 5-adenylic acid deaminase 95; prostatic phosphorylase 190; nucleotide pyrophosphatase 950. Frajola [88] had 2 subjects ingest 36 mg sodium fluoride (16 mg of F ion) daily for varying periods of time. Of the 5 enzymes tested (lactic dehydrogenase (LDH), acid phosphatase, alkaline phosphatase, and serum glutamic-oxalacetic transaminase (SGOT), and serum glutamic-pyruvic transaminase (SGPT)), only LDH showed some decrease in the first subject. The second subject did not exhibit this effect, nor did the first subject when the trial was repeated. Actual figures were not given.

The sensitivity of various enzymes to fluoride in vitro in the pH range 5-8 was reported by Wiseman. [89] In the presence of divalent metal ions human salivary acid phosphatase was 55% inhibited at a F concentration of 3.8 ppm; erythrocyte inorganic pyrophosphatase was inhibited 52% at 0.38 ppm; human plasma cholinesterase was 61% inhibited at 0.95 ppm F; sheep brain glutamine synthetase was 50% inhibited at 0.95 ppm, and liver methionine "activating" enzyme was 28% inhibited at a F concentration of 15.2 ppm. The significance, if any, of the above results was not discussed.

Ferguson [90] found that when 15 subjects were given 5 mg of sodium fluoride and compared double blind to 15 controls, alkaline phosphatase activity showed a reduction of approximately 20% during the first 6 weeks by starch gel electrophoresis. Protein patterns were unchanged. When he studied 89 persons in 3 towns before and after water fluoridation, there were comparable depressions in serum alkaline phosphatase in the first 3-4 weeks. By 8-12 weeks, however, the values had risen to normal. The control values for serum alkaline phosphatase activity, from which the percentages of normal activity were calculated, were derived from measurements in 33 subjects in town A during the month prior to fluoridation. The lower limit of normal for the methods used to measure the enzyme activity (Boehringer test kit and starch gel electrophoresis of serum) was said to be "around 57% of the control values found in these studies." There was graphic presentation of these control values, with means ranging between 20-35 milliunits/ml serum. The author failed to mention that if 57% of the control values were at the lower limit of normal, then the mean values of all groups tested remained within normal limits.

Epidemiologic Studies

Moller and Gudjonsson [17] in 1932 first reported the results of their examination of 78 workers engaged in the crushing and refining of cryolite. Roentgenologic examinations of the skeletal system revealed that in 30 of the 78 workers there was increased bone density varying in degree and extent. The changes were most marked in the spinal column and pelvis, extending throughout the skeletal system only in the most severe cases. Besides increases in osseous radiopacity, calcification of ligaments and hyperosteoses were observed. Increased bone density was found in 14 of the

60 workers employed less than 10 years and in 16 of the 18 employed more than 10 years. However, marked changes were found in 1 male worker after 5 years and in 2 women after 6 years of employment. The 2 workers with the most pronounced bone changes, who had worked in the factory for 25 and 11 years, respectively, were entirely devoid of mobility in the lumbar and thoracic spine. The older of the 2 had noticed the stiffness for 10 years. Otherwise he was in good condition except for a slight tendency to dyspnea with exertion. Despite the musculoskeletal symptoms he did not complain of pain either in his back or in his joints. The other man had first noticed stiffness in the back 6 years previously and complained of "rheumatic" pain across the hip even when lying down. Head movement was markedly restricted. Both men were able to move other joints freely. Of the 78 workers examined, 42 complained of nausea, loss of appetite, and vomiting. In addition, most of the workers suffered from shortness of breath during work. Of the 30 workers with increased bone density, 11 were anemic with an average red cell count of 3.7 million and hemoglobin of 77%. Of these 11 workers, 7 had severe bone changes. Serum calcium was determined in 10 cases and found to be normal. Urinalysis showed no abnormalities. Examination of the teeth indicated nothing of a specifically pathological character.

For comparison, several workers from other chemical factories were evaluated. [17] Nine men who had worked for up to 40 years in an enamel factory were examined; neither bone changes nor the acute dyspeptic symptoms experienced by the cryolite workers were observed. The enamel was made by mixing, crushing, and melting cryolite, feldspar, and quartz. During the melting process "great quantities" of hydrogen fluoride were released. Five men exposed for several years to hydrogen fluoride in the etching of glass exhibited none of the symptoms found in the workers engaged in the crushing and refining of cryolite. Six men were examined from a sulfuric acid factory where sodium fluosilicate was formed as a by-product. Five of the 6 who were exposed for 4-9 years to F vapors but not to fluoride dust had never experienced any discomfort nor had they any signs of bone changes. The 6th worker, engaged for 1.5 years in drying sodium fluosilicate, complained of acute dyspeptic symptoms when working in heavy dust but had no bone changes. The authors attributed the abnormal findings in cryolite workers to the absorption of fluoride from swallowed cryolite dust.

Roholm [18] followed up on the findings of Moller and Gudjonsson. [17] He conducted an extensive survey of the health status of cryolite workers, and published the results in 1937. The processing of the cryolite ore in the factory under investigation included a series of dust-producing operations such as stamping, grinding, sieving, and packing. Gravimetric dust tests conducted in the grinding and manual-sorting rooms ranged from about 30 to 40 mg of dust/cu m. In some places where fine grained cryolite was handled or worked within enclosed spaces around machinery, the dust content increased up to 994 mg/cu m. Working periods under these conditions were short and workers wore masks. The dust content had been much higher prior to 1923-24 when enclosure of the machinery was begun. The cryolite content of the dust averaged 97%, varying from 94.17 to 99.04%. Cryolite contains 54.3% fluoride. From these data, the average exposure was estimated as 15-20 mg F/cu m. Quartz content of the dust was 1-5%. In the grinding and manual-sorting rooms approximately 80% of the dust was less than 10 m in diameter and about 50% was less than 5 m.

In 1933 the factory employed 69 workers. [18] All these workers were examined except one who declined. The remaining 68 workers ranged in age from 20 to 69 years and were comprised of 47 males and 21 females. Their period of employment averaged 10 years; 21 workers (30%) had been employed for 10 years or more, and of this number 5 workers (7.4%) for 25 years or more. Eighty percent of the workers complained of loss of appetite, nausea, and vomiting. Moderate dyspnea was experienced by 42.7%. Feelings of stiffness and rheumatic pains were reported by 35.3% of the workers, while 33.8% complained of either diarrhea or constipation. Tiredness, sleepiness, indisposition, headaches, and giddiness were experienced by 22.1%, and 11.8% had intermittent skin rash. In the first of 3 described phases of osseous fluorosis, the density of the bone was slightly increased in the spinal column and pelvis, but this increase was doubtful or absent in other bones. In some cases there was incipient osteophyte formation on the lumbar vertebrae. In the second phase the bony structure presented a diffuse structureless density with uneven and somewhat blurred contours. The changes were most pronounced in the pelvis and vertebrae. In the long bones the medullary cavity was usually moderately narrowed. Ligament calcifications were observed in the spinal column. In the third phase, the bone appeared more or less diffuse, and had increased density in which no details could be distinguished. There was considerable ligament calcification, particularly in the thoracic and lumbar spine. The width of the medullary cavity of the femur and tibia was in some cases decreased to half the normal and partial occlusion of the cavity of the metacarpals and phalanges occurred. Roholm summarized the bone changes as follows: "The pathological process may be characterized as a diffuse osteosclerosis, in which the pathological formation of bone starts both in periosteum and in endosteum. Compacta densifies and thickens. The spongiosa trabeculae thicken and fuse together. The medullary cavity decreases in diameter. There is considerable new formation of bone from periosteum, and ligaments that normally do not calcify or only in advanced age undergo a considerable degree of calcification. All signs of bone destruction are absent from the picture."

Table III-11 indicates the relationship between the varying degrees of dust the 68 workers were exposed to and the classifications of bone changes that were observed. The classification of "light," "moderate," and "heavy" dust exposure by Roholm was apparently made by visual inspection of the workplace. Dust samples were taken only from the grinding and hand-sorting rooms and from space around enclosed machine separators. Less dust exposure was noticed in the raw cryolite store, when cryolite was transported in the open, and near the wet processes.

Of the 8 workers exposed to "slight" dust concentrations, 7 (87.5%) had little or no bone changes; of the 49 workers exposed to "moderate" dust concentrations, 37 (75.5%) had mild to moderate bone changes and of the 11 workers exposed to "heavy" dust concentrations, 9 (82%) had moderate to marked osteosclerosis. On physical examination the severe cases showed moderate to marked restriction of mobility of the spine. On the whole, the restriction of mobility increased directly with the osseous changes. In the 26 mild osteosclerosis cases, 12 showed slight restriction (46%) and 2 moderate restriction (7.6%). In the 24 moderate osteosclerosis cases, 7 had slight restriction (29%) and 5 moderate restriction (21%). In the 7

TABLE III-11

BONE CHANGES IN 68 CRYOLITE WORKERS EXPOSED TO VARIOUS DUST LEVELS

Characteristic Bone Change	Number of Workers	% of Total	Average Age	Average Length of Employment	Dust Exposure		
					Slight	Medium	Heavy
No change	11	16.2	36	8 (2.8-24)	2	9	-
Phase 1	26	38.2	36.8	9.3 (2.4-33.8)	5	19	2
Phase 2	24	35.3	40.5	9.7 (4.8-28.9)	1	18	5
Phase 3	7	10.3	58.2	21.1 (11.2-31.2)	-	3	4

From Roholm [18]

marked osteosclerosis cases, 3 had moderate restriction (43%) and 4 great restriction (57%). There was no irritation of the mucous membranes of the nose and throat.

The F content of teeth of 5 workers, who had been employed for about 10 years, averaged 2.5 mg/g ash (varying from 1.1 to 5.3 mg/g) while teeth of 2 persons not exposed to F averaged 0.26 mg/g (0.19 to 0.30 mg/g). [18]

Eleven workers (14%) exhibited physical signs resembling emphysema. [18] Chest X-rays showed incipient or moderate signs of pulmonary fibrosis in 34 of the 68 workers. In workers with 9 years or more of employment the majority (13 of 18) had fibrosis. Dust levels were high, ranging from 30 to 40 mg/cu m. The quartz content of the dust varied from 1 to 5% of the total. Examination of the blood (hemoglobin, RBC, WBC, differential, bleeding time, coagulation time, blood platelet count, erythrocyte resistance, and sedimentation rate) showed no significant deviation from the normal. No abnormalities were found in the examination of the nervous system. The size of the thyroid gland was not changed. Urine contained no albumin or sugar; microscopy was normal. Serum calcium determinations on 18 workers were within normal range. Two selected workers, both with osteosclerosis, excreted 2.54 and 2.09 mg F in a 24-hour urine sample while 2 controls excreted 0.22 and 0.12 mg F/24 hours.

One-hundred-twenty-seven persons who had formerly worked at the factory for at least 6 months were questioned about loss of appetite, nausea, vomiting, irregular bowel movements, cough, headache, and tiredness while on the job. [18] They complained of symptoms more frequently than did currently employed workers, but otherwise there was great conformity between the 2 groups. X-ray examinations of 32 of these former employees who had worked at the factory for 4-38 years (average 13.5 years) revealed typical bone changes in 9 (28.1%), 5 cases of first phase, 3 of second, and 1 of third phase. Of 22 persons who had been away from the factory for at least 3 years, only 3 (13.6%) had any level of osteosclerosis.

Observation of pulmonary fibrosis in 23.3% of 30 former workers examined in comparison with 50% of the employed workers indicated to Roholm [18] that the pulmonary fibrosis did not progress after cessation of work and perhaps even had a tendency to diminish in contrast to silicosis. Therefore, he tentatively concluded that cryolite and not quartz was the active agent causing the fibrosis in cryolite workers.

X-ray examination [18] of 9 workers from a cryolite mine, whose employment at the mine varied from 16 months to nearly 8 years, showed first phase bone changes in only one worker who had worked 7 1/4 years at the mine. Pulmonary fibrosis was observed in 3 of the 9 workers. Seven workers complained of transitory gastric symptoms.

Morbidity statistics [18] of the cryolite workers, as expressed in number of sick days due to various illnesses, did not exceed the average in industry as a whole but indicated a high frequency of "rheumatic" complaints.

Post-mortem examinations of 2 cryolite workers were described in detail. [18] The first was a 68-year-old male who had been employed at the cryolite factory for about 24 years until some weeks before his death from an intestinal obstruction. The second, a 52-year-old male who had worked in the cryolite plant about 9 years, died of syphilitic heart disease about 17 months after leaving the factory. In general no changes were found in the gastrointestinal tract and liver. A slight degree of chronic nephritis was found in 1 case, while the kidneys of the second were normal. No lung changes were revealed which could be characterized as silicotic. The various parts of the bones were thickened, all surfaces were uneven and there was more or less wide-spread calcification of the ligaments. Microscopic examination of the bones showed increased formation of osseous tissue, both from periosteum and from endosteum. However, in the second case there were signs of both active breakdown of the sclerotic osseous tissue and formation of a mainly normal osseous tissue. This normal reconstruction of bone was not seen in the first case. Stomach, liver, spleen, kidneys, and heart were analyzed for F and compared with values for an unexposed 50-year-old man who had died after an accident. In the cryolite workers, F ranged from 0.32 to 2.4 mg/100 g dry substance and in the unexposed man from 0.50 to 1.8 mg/100 g. The results showed that there was probably no marked excess deposition of fluoride in these organs of cryolite workers. However in the lung tissue from both cryolite workers there was a high F content, 79.2 and 10.8 mg/100g compared with 0.73 mg/100 g in the control. The F content of the bone ash of the 2 cryolite workers ranged from 3.1 to 13.1 mg/g ash compared to 0.48-2.1 mg/g of 11 controls obtained from the Copenhagen University's Institute of Pathological Anatomy.

Of 5 children [18] borne by women who worked at the cryolite factory before or during pregnancy or who started to work there soon after giving birth, 3 had mottled enamel. The degree of the changes was approximately dependent upon the length of the nursing period. Since these children had nursed for a relatively long period (up to 2 years), Roholm [18] attributed the condition to excretion of F in the milk.

In 1938 Hjort [91] reported the study of 62 potmen ranging in age from 32 to 68 (average 42) who had worked in the furnace room of an aluminum plant for 10-18 years. X-ray examinations of 56 of these workers showed no bone changes resembling fluorosis. In 37.5% of the workers, indications of spondylosis deformans, mostly of a mild degree, were found. Erythrocyte count and hemoglobin values were normal. Eosinophilia (4.5-8.5%) was found in 18 workmen. However, eosinophilia was also observed in 9 of 20 controls. Stomach secretions of 55 workers were normal. The 2 1/2 year diary representing the practice of a physician providing medical care for the workers showed 47 out of 89 potroom workers visited the doctor on 92 occasions. A study of the illnesses reported by the workers and compared with a control group of 67 workers from an electrode plant with 70 visits gave no indications of the occurrence of acute or chronic symptoms or signs related to F exposure. There were 3 cases of asthma in the furnace group and 2 cases of asthma were also reported in the control group. However, since these 2 men had occasionally worked in the potroom, they should not have been included in the control group.

In 1941 Brun et al [92] published the results of their study of urinary F analysis in cryolite workers. The normal excretion of F in 24-hour urine samples was determined on 30 randomly selected hospital patients with no known F exposure (control group), ranging in age from 2 months to 76 years. Osteosclerosis was reported in some (number not given) of 24 cryolite workers, 8 craftsmen, and 4 of 6 former workers. The urinary F concentration in the control group was 0.30-1.60 mg/liter, averaging 0.92 mg/liter, whereas that of 24 cryolite workers employed 1-34 years ranged between 2.41-43.41 mg/liter, with an average of 16.05 mg/liter. In one cryolite worker, hospitalized for a prolonged period of time, the urinary F concentration fell from 7.74 mg/liter on the first day in the hospital to 1.52 mg/liter on the 25th day of hospital stay. Eight craftsmen, reportedly exposed to less dust, excreted an average of 4.81 mg F/liter (ranging from 1.78 to 11.67 mg F/liter). Six former workers who had worked at the plant for 16-38 years, but who had been away from exposure for 2-11 years, excreted from 0.92 to 7.42 mg F/liter. The 4 workers with osteosclerosis in this group showed higher F excretion, 2.8-7.42 mg/liter. All urine samples were single measurements. The samples of the cryolite workers and craftsmen were taken during working hours.

Tourangeau [93] in 1944 reported on potroom working conditions in aluminum plants. The F concentration (as HF) in the potroom at one plant was 4 ppm (3.3 mg F/cu m) and in that of the other plant where ventilation was better, it was not more than 3 ppm (2.5 mg F/cu m). Air contaminant concentrations were determined only at the time of the study and therefore may not represent the actual long-term exposure of the workers. Medical records showed the average number of respiratory illnesses over a 4-year period did not vary greatly among 698 potworkers as compared with unexposed employees (number not given) with an incidence of 59.4 and 53.2/1,000 employees, respectively. The incidence of diseases of the digestive tract, rheumatism, cardiovascular disease, renal disorder, disease of the skin, eye and nose disorders, anemia, pyorrhea, and infections was 100.7/1,000 in potworkers while it was 131.1/1,000 in the control group. Physical examinations of 104 potworkers, aged 20-55 years, with exposure periods ranging from 1 to 20 years, disclosed the state of health of these workers to be excellent. Chest X-rays of 105 potworkers exhibited increased hilar markings and accentuation of the reticulum of the stroma in 16 (15.2%) workers, and deformation of the cardiac shadow in 17 (16.2%) workers. These abnormalities seemed to be more frequent in men who had worked for 3 years and more in the potroom. Four workers (3.8%) showed scarring of tubercular lesions and 3 (2.8%) exhibited pleural sequelae. X-ray examinations of the skeletal system in 10 workers who had been working in the potroom from 7-30 years seemed to show generalized osteosclerosis in 2 workers, and areas of localized densities in 3. None of these workers suffered from serious incapacity. The quality of X-rays of the skeletal system, which was stated to be poor, made the validity of the radiological evaluation suspect.

In 1947 Bowler et al [94] reported an investigation of F exposure in a magnesium foundry in southern England. Airborne dust at various points in the factory contained from 0.143 to 0.714 mg F/cu m in all areas except the mixing mill where it was 6.370 mg/cu m. Of about 190 foundry employees, only 124 volunteered to participate and not all of these were examined due to staggered holidays, progressive reduction in personnel, and reluctance of some workers to participate. The age of the participants ranged from 15 to 59 years with about 50% in the 30-39 age range. The results of hematological examinations (hemoglobin and red, white, and differential counts) showed no abnormalities. The vital capacities of 103 workers showed no significant difference between the groups except that workers in the core shop and foundry with less than 6 and less than 2 years' exposure, respectively, had rather low mean vital capacities. It was stated that low vital capacities in these workers might have been due to their generally poor physical condition inasmuch as they were not sufficiently fit to qualify for military service. It is not possible from the data presented to compare ventilatory function in exposure groups or with a control group. Chest X-rays of 78 workers revealed evidence of reticulation in 28 (36%) of the workers examined. An increasing frequency of reticulation was noted with age--those in the 15-29 age group had 25% showing reticulation, 29% in the 30-39 age group, 42% in the 40-49 age group, and 88% in the 50-59 age group. Radiologic examination of the jaw, spine, pelvis, radius, and ulna (numbers of workers examined not given) revealed only one case with increased density of the bones, ossification of the ligamentous attachments, and increased density of the tips of the spinal processes. He was a 37-year-old man who had worked as a furnaceman for 5 1/2 years. He exhibited no symptoms or abnormal physical signs. His hemoglobin was 112%. Although his urinary F concentration was not measured, his blood F level was reported as 4.6 ppm.

Nine subjects who volunteered to give 24-hour weekend urine specimens were found to excrete from 0.35 to 3.80 mg F/24 hours with a mean of 1.10 mg. [94] Urinary F concentrations of 54 spot samples, obtained at unspecified times, with an average specific gravity of 1.027 (range of 1.005-1.037) showed an average of 2.53 mg F/liter ranging from 0.5 to 7.5 mg F/liter. Urinalyses from a control group of 40 residents from England and Scotland (20 from each) revealed average values of 1.58 and 0.51 mg F/liter, respectively. Fluoride content of the water supply in the district in which the workers lived was 0.18 ppm. Fluoride concentrations in the water supply of the control groups ranged from 0.07 to 0.18 ppm. There was no significant difference in urinary F concentrations in the 3 exposure groups labeled core shop, foundry, and furnacemen, with mean F concentrations of 2.48, 2.65, and 2.23 mg/liter, respectively. In the core shop and furnacemen groups there was no significant difference in urine levels between those who had been exposed for more than 5 years and those with shorter exposure. The foundry workers showed a progressive increase in urinary F concentrations with lengthening exposure. Those with under 2 years' exposure had an average of 1.60 mg/liter, those with 2-5 years' exposure had an average of 2.78 mg/liter and those with 6 or more years' exposure had an average of 3.92 mg/liter.

In 1948, the Industrial Hygiene Division of the US Public Health Service [95] conducted a medical and environmental study to determine the potential health hazards associated with the use of sodium fluoride at open hearth furnaces in 4 steel plants. Medical examinations of 350 workers were distributed as follows: 187 employed in 2 plants using sodium fluoride, 63 in a plant formerly using sodium fluoride, and 100 in a plant which had never used sodium fluoride. Air samples were taken in the breathing zone by means of an electrostatic precipitator followed in series by a standard impinger containing 1% sodium hydroxide. Results of 59 samples from operations using NaF were distributed as follows: ladle crane operators, 1.0-51.2 mg NaF/cu m (about 0.45-23 mg F/cu m), pouring platform workers 0.2-21.9 mg NaF/cu m (about 0.09-9.9 mg F/cu m), tapping workers, "negligible" to 40.1 mg NaF/cu m, and general area workers, "negligible" to 14.0 mg NaF/cu m. Time-weighted averages for these workers ranged from "negligible" to 2.2 mg NaF/cu m (about 1.0 mg F/cu m). Medical procedures included a medical history, present subjective complaints, examination of the eyes, nose, and throat, X-rays of the chest, left wrist and forearm, routine blood analysis and urinalysis including the F concentation. The most common, and, according to the authors, the most significant symptoms found were cough, "hoarseness," sore throat, and congestion of the nose. Although an inverse relationship of these symptoms to the level of sodium fluoride exposure was found, the authors concluded it was difficult to separate the independent effects of sodium fluoride, tar smoke, and sulfur dioxide. Analysis of arm-bone X-ray films for increased density, narrowing of the marrow cavity, and increased thickening or roughening along the areas of ligament attachment failed to reveal any findings which the authors considered to be abnormal. Examination of 38 urine specimens for F revealed all to be within unspecified "normal" limits.

For purposes of evaluating a dose-response relationship (exposure to airborne F and urine or skeletal changes), it is unfortunate that the authors did not provide either individual or group comparisons. Further, actual urine F values found were not listed, nor was the criterion for the range of "normal" specified.

Agate et al [26] in 1949 reported on clinical, radiological, hematological, and biochemical findings of 324 Scottish workers from 2 aluminum factories and 75 persons residing in the communities surrounding the Fort William plant. The factories under consideration produced aluminum by the electrolytic reduction process. The F concentrations in the furnace rooms at the Fort William plant reportedly ranged from 0.14 to 3.43 mg/cu m. This concentration range was based on a total of 17 air samples, 5 of which were taken over a 12-day period in one year, and the remaining were collected the next year over a 3-day period. In the furnace rooms in the Kinlochleven foundry, F ranged from 0.34 to 0.91 mg/cu m. Atmospheric concentrations of F in various parts of the factory outside the furnace rooms ranged from 0.015 to 0.141 mg/cu m. This concentration range was based on a total of 7 samples, all of which were taken over a 2-day period in the same year. The concentrations of F in the air outside the factory at various distances (200 yards to 1 mile) on different days, varying with wind direction and speed at time of sampling, ranged from 0.020 to 0.220 mg/cu m. A total of 11 samples were taken to determine this range. Eight samples were taken in one year, and the remainder taken in the second year. Particulate F as a percentage of total F ranged from 19 to 85% in these areas.

At the time of the clinical examination, 668 men and 114 women were employed at the Fort William plant with 358 men and 42 women working in the furnace rooms. [26] Two groups within the factory population were selected. Group I was comprised of 220 men and 22 women employed in the furnace rooms as well as 12 men from the Kinlochleven plant who were included because of their length of service (24-42 years). Group II consisted of 44 men and 25 women from the Fort William plant working outside the furnace rooms. For various reasons (absenteeism, sick leave, and refusal to be examined) the population samples were not strictly representative. Group III consisted of 26 men and 51 women volunteers from residents of the surrounding district. Physical examinations included particular attention to the movements of the thoracic cage, joints, spinal column, X-rays of the skeletal system, blood, and urine examinations. A past and present history of aches and pains in the back was reported by 5.3% in Group I, by 4.4% in Group II, and by 6.5% in Group III. Statistical evaluation of these data after age and sex adjustment showed no evidence that the 3 groups differed. There was no evidence of any difference in the incidence of fractures among the 3 groups. Abnormal radiological findings were seen in 48 (25.4%) of the 189 workers in Group I, in 5 (8.3%) of 60 employees drawn from Group II, and in 3 (4%) of 75 subjects from Group III. The criteria of abnormal radiological findings were all those radiological abnormalities, however slight, which could not be explained as due to any disease. The reason given for this classification was the fact that the earliest radiological signs of fluorosis had not yet been defined. The authors, therefore, stressed that abnormal X-ray appearances must not be considered synonymous with skeletal fluorosis. In most cases the abnormal X-ray appearances showed normal bone density and pattern and only in a small proportion was there an appearance of increased density. No numbers were given. Within broad age groups (under 40 years and over 40 years), there was an increasing incidence of

X-ray abnormalities with increasing length of exposure in Group I workers, with the exception of the 10-14 year exposure period in the younger age group, for which only 3 observations were available. The increase of X-ray abnormalities with increasing length of exposure was statistically significant only in the age group 40 and over ($p < 0.01$). The data presented also suggested that age may have had a direct influence upon the incidence of abnormal X-ray findings, but statistically the difference was not significant ($p < 0.30$). Various forms of digestive disturbances were reported in 14.7% of Group I, 10.1% of Group II, and 5.2% of Group III. Standardization for age and sex showed an indication of a relatively higher incidence of digestive disturbance rate in Group I, compared with the control group. No complaints of dyspnea on exertion were elicited. The incidence of cough persisting over a number of weeks and accompanied by the production of sputum was higher in Group I (12.83%) using the same standardization process compared with the less exposed Group II (6.9%) and controls (3.9%). Hemoglobin and blood counts were within normal limits in all 3 groups.

The mean urinary F for male employees working full-time in the furnace room was 9.03 mg/24 hours. [26] For the Group I workers whose exposure was considered less severe, urinary F values for males was 5.19 mg/24 hours and 3.64 mg/24 hours for females. The range for the total Group I (furnace room) workers was 0.5-23.3 mg/24 hours. Male workers in other parts of the factory (Group II) excreted 1.83 mg/24 hours, as contrasted with females who excreted 1.58 mg/24 hours. Male and female local residents excreted 0.84 mg/24 hours. A comparison between the mean urinary excretion of the fully exposed furnace room workers who had abnormal X-ray findings and those who had not, showed no statistically significant difference in urinary F excretion (9.36 mg and 9.08 mg/24 hrs, respectively). There was no statistically significant difference between abnormal X-ray findings and age or length of exposure among the less severely exposed furnace room workers. Comparing the incidence of X-ray abnormalities of the fully exposed and less severely exposed male workers there was an indication of a lower incidence in the latter group.

Two case histories with abnormal X-ray findings were presented. [26] One was a 32-year-old man who had been exposed to F for 15 years. He had a rigid spine from the lower cervical region to the sacrum and his X-ray showed slight lipping of the vertebrae and calcification of the anterior and lateral intervertebral ligaments. All other bones appeared to be normal, but there was a suggestion of abnormal ossification of the interosseous membrane between the radius and ulna. Since these abnormal findings were associated with osteoporosis of the vertebrae, a diagnosis of ankylosing spondylitis rather than skeletal fluorosis was made. Urinary F was 2.1 mg/24 hours. The other case involved a 63-year-old man who had been exposed to F for 43 years; his vertebrae showed lipping, beak-like exostosis, and osseous bridges, and his pelvis showed an amorphous or granular appearance with loss of the trabecular pattern. He reported no clinical symptoms. The urinary F was 14.9 mg/24 hours.

In summary, even though the examined population was not randomly sampled because of voluntary participation, one can reasonably assume that the data obtained by Agate et al [26] gave a broad, though not necessarily statistically precise, picture of the risks of F. A larger proportion of the workers exposed to the heaviest F concentrations complained of chronic, productive cough. Bone X-ray abnormalities were found in 48 out of 189 furnace room workers, but only a small number showed appearances consistent with those of skeletal fluorosis. The survey would have been greatly enhanced if more detailed data on the X-ray findings had been presented. Among the older furnace room workers examined, the incidence of X-ray abnormalities was found to increase with increasing length of exposure.

In 1951, Largent et al [96] compared urinary fluoride excretion with roentgenographic bone changes in 16 workers engaged in the manufacture of inorganic fluorides. Three of these employees worked solely with hydrogen fluoride and are not considered here. Off-work urine samples obtained over several consecutive days were collected on 7 occasions over a 3-year period. Average F concentrations in urine over the 3-year period ranged from 0.7 to 16.38 mg/liter among the 16 workers. Length of service ranged from 6 to 26 years and age of the workers from 30 to 72 years. Urine samples taken from 2 office workers with no known exposure had F concentrations ranging between 0.48 and 0.88 mg/liter during the 3-year period. Two of the 13 workmen exposed to inorganic fluorides showed marked increase in bone density. Three others showed slight increase in bone density. None had any symptoms of fluorosis. In one case the roentgenographic changes were described as marked increase in the density of the bones of the pelvis, the head and neck of the femurs, the lumbar and dorsal spine, ribs, the clavicles, scapulae, and the bones of the forearms. The average urinary F concentration in this 57-year-old workman with 25 years of service was 16.38 mg/liter. In the second case there was moderate increase in the density of the lower thoracic and lumbar spine and the pelvic bones. The average urinary F concentration in this 42-year-old worker who had 6 years of service was 11.65 mg/liter. The remaining 11 workmen whose mean urinary F concentration ranged from 0.70 to 6.51 mg/liter did not show any increased bone density.

In summary, [96] the 2 workers who showed a marked increase in bone density had an average urinary F concentration of 11.65 mg/liter or more, and those that showed slight increases in X-ray density excreted 9.30-12.29 mg F/liter. No bone changes were noticed among 11 workers with mean urinary F concentrations of 6.51 mg/liter or less. The authors concluded that, while the data were obtained from too few individuals during too short a period of time to warrant any sweeping generalization, workers excreting urinary fluoride concentrations of less than 10 mg/liter should not be expected to show demonstrable bone density changes.

In 1960 Rye [38] reported on clinical observations of 28 employees in the production of phosphate rock. The phosphate rock production consisted of open pit mining, and drying, screening, and grinding the ore. The phosphate rock contained 3.5-4.2% F and less than 1% silica. Ten percent of the dust particles were below 10 μm in diameter. The average of urine spot samples taken before, after, and during the workshift was 0.5-1.5 mg F/liter while the total 24-hour F excretion ranged from 1.0 to 1.3 mg. No spot sample contained more than 4 mg F/liter. Annual chest X-ray films recorded since 1948 as well as films of the lower spine, pelvis, and upper femur revealed no significant changes in 28 men employed for periods of 2-35 years.

Derryberry et al [97] in 1963 reported on the health status of 74 workers in a phosphate fertilizer manufacturing plant in relation to fluoride exposure. Fluorides in the form of dust and gases in varying combinations and concentrations were produced throughout the process in the manufacture of phosphate fertilizer. Data were collected from clinical examinations, the working environment, and urinary excretion of F throughout the 25 years of operation. From 1952 to 1962, urine specimens had been collected (a specimen at the end of the shift on 5 consecutive days) at yearly intervals. In 1958, a special study was initiated to evaluate the effects of fluoride on the workers' health. An average daily exposure for each job was established by determining the time-weighted concentrations of F. From these data a weighted atmospheric exposure was calculated for the period of employment of each worker. In the clinical study presented, a group of 74 workers, representative of all exposure areas, was chosen from 300 workers employed in the phosphate fertilizer manufacturing process on the basis of having been exposed to relatively high F concentrations as reflected by consistently high urinary F excretion throughout their employment. A control group, selected to match the exposed group as nearly as possible in age, race, and socioeconomic class, consisted of 67 workers with no previous exposure to F. The average ages of the groups were 45.3 and 45.9 years, respectively, with an average F exposure duration for the first group of 14.1 years and a range of 4.5-25.9 years.

Past medical histories [97] showed a greater incidence of metabolic and endocrine disease in the controls than in the exposed (7.5% vs 1.4%, with $p < 0.10$). The incidence of respiratory disease (pneumonia, pleurisy, or influenza) was significantly higher ($p < 0.05$) in the exposed group than in the control group (25.7% vs 11.9%). Physical findings showed 26.9% of controls to be overweight vs 14.9% of the exposed ($p < 0.10$). Among other differences, there was significantly more anemia (4.5%) in the control group than in the exposed group (0.0%) based on a hematocrit of less than 40% ($p < 0.05$), and more albuminuria in the exposed group (12.2% vs 4.5%, with $p < 0.10$). Minimal or questionable increase in bone density (recognizable with prior knowledge that the individual had a potential exposure to F, but not such as would be recognized in routine practice) was seen in 23% of the exposed group and in 1.5% of the controls ($p < 0.01$). Chest X-ray revealed a higher incidence, which was questionably significant, of pulmonary changes (emphysema, nonspecific fibrosis, costophrenic adhesions, and healed tuberculous lesions) in the exposed group (14.9% vs 6.0%, with $p < 0.10$).

The average urinary postshift F excretion [97] of exposed workers was 4.67 mg F/liter ranging from 2.14 to 14.7 mg F/liter. Those with increased or questionable density excreted an average of 5.18 mg F/liter with a range of 2.8-8.9 mg F/liter and those without increased or questionable bone density excreted an average of 4.53 mg F/liter with a range of 2.1-14.7 mg F/liter (see Table III-12). To evaluate F retention under variable conditions of exposure, the range and the percentage of urinary specimens containing or exceeding a reference level of 4 mg F/liter was used to classify individual workers as follows: low exposure, when less than 30% of the specimens exceeded this level; moderate exposure, when 30-50% contained 4 mg F/liter or more and high exposure, when over 50% contained 4 mg F/liter or more. In the low exposure group, 2 workers showed increased or questionably increased bone density and 17 workers had no increased bone density. In the moderate exposure group, 5 exhibited increased or questionable bone density changes while 16 had normal bone appearance. In the high exposure group, 10 had increased or questionably increased bone density compared with 24 without changes.

The authors [97] computed a "weighted atmospheric exposure" for each individual based on time-weighted average F exposures at each job and the period of time each worker was employed at that job. The range of individual average weighted exposure to F was 0.50-8.32 mg/cu m, with 1.78-7.73 mg/cu m being associated with increased or questionably increased bone density. The difference in averages between the increased bone density group (average exposure 3.38 mg F/cu m) and the remainder of the exposed group (average exposure 2.65 mg F/cu m) is significant by both t test ($t = -2.75$, $p = 0.0045$) and rank test ($z = -2.2$, $p = 0.014$). Table III-13 shows the incidence of increased bone density in increments of 1 mg F/cu m of time-weighted exposure during employment. It demonstrates that exposure to higher atmospheric concentrations resulted in a relatively greater incidence of increased bone density.

The wide range in individual urinary F excretion and exposure of

TABLE III-12

RELATIONSHIP OF AVERAGE URINARY FLUORIDE EXCRETIONS TO INCIDENCE OF INCREASED BONE DENSITY IN 74 INORGANIC FLUORIDE WORKERS

Average urinary F excretion mg/liter	2-2.9	3-3.9	4-4.9	5-5.9	6-6.9	7-7.9	8-8.9	9-9.9	10+
Number of workers	10	23	20	9	3	1	5	2	1
Number with increased bone density	1	4	5	3	1	0	3	0	0

From Derryberry et al [97]

TABLE III-13

RELATIONSHIP OF WEIGHTED EXPOSURE DURING EMPLOYMENT TO INCIDENCE OF INCREASED BONE DENSITY IN 74 INORGANIC-FLUORIDE WORKERS

Weighted exposure mg F/cu m	0- 1.50	1.51- 2.50	2.51- 3.50	3.51- 4.50	4.51- 5.50	5.51- 6.50	6.51- 7.50	7.51- 8.50
Number of workers	13	26	17	11	1	2	0	4
Number with increased bone density	0	5	6	4	0	1	0	1
Percent	0	19	35	36	0	50	0	25

From Derryberry et al [97]

workers with and without increased bone density suggested to the authors considerable variation in individual reactions to F as manifested by bone density changes. Comparison of data also suggested that increased density resulted from frequent exposure at high environmental concentrations of F, but was not related to the age of the worker or to the number of years of exposure. [97]

Parsons et al [98] in 1964 reported on a survey of respiratory symptoms, lung function tests, and chest X-rays of 280 men employed in 2 fluorspar mines at St. Lawrence, Newfoundland. In a previous study the incidence of lung cancer had been shown to be 20 times greater in this group of miners than for the rest of the province. [95] The high incidence of carcinoma among the miners was attributed [98] to the high level of radioactivity (radon daughters) in the air and water in the mines. In view of the likely presence of radon in this area and the absence of other reports of cancer among F workers, it appears at this time that the authors were correct in attributing the observed cancer to radon. Of the 258 men radiographically studied only 5 (1.93%) showed pneumoconiosis. Environmental studies [99] of airborne dust revealed a 4-38% quartz content with a mean of 19%. Calcium fluoride in airborne dust ranged from 26% to 96% with a mean of 62%. Air concentrations of F in the mine reportedly ranged from 0.00 to 1.92 mg/cu m. Chronic bronchitis was found in 22.1% of the miners and mill workers compared with 16.2% in surface workers and 4% in nonminers. Pulmonary function tests showed lower values in maximum breathing capacity and maximal mid-expiratory flow rate in the exposed group though these differences were not significant at the 5% level. Peak flow rate values were similar in all groups with the exception of surface workers in the 50- to 59-year range who had significantly lower values. Smoking habits of the exposed group were not significantly different when compared with the nonexposed group, though there were fewer nonsmokers and more heavy smokers in miners and crusher-house workers. Lower values of lung function tests appeared to be associated more closely with the presence of chronic bronchitis than with exposure to dusty conditions.

In 1970, Vischer et al [100] reported a study on a group of 17 male workers, 52-66 years old. They were selected for possible fluorosis, based on typical radiological findings, history of F exposure at work in the potrooms of an aluminum factory for 11-46 years (average 29 years), and on elevated F values in bone as determined in dried and defatted samples of the iliac crest obtained by open or needle biopsy. No quantitative estimates were available in regard to the levels of F exposure. All of the subjects showed normal levels of serum alkaline phosphatase, calcium, and phosphate. The bone biopsies revealed an average F content of 332.0 mg F/100 g bone and ranged from 135.0 to 472.0. Microscopic examination of 11 bone biopsies showed 4 cases with evidences of fluorosis, including bone remodeling with increased density and changes in mineralization. The other biopsies showed only disturbances of mineralization. One biopsy appeared practically normal. On X-ray examination all cases showed exostosis or ossification of ligaments of the spine, but in only 9 of the 17 subjects could a clear-cut increase in density or change of structure of the bone be determined. Decreased motility of the spine was found in all patients and 3 had evidence of radicular syndromes. All but one subject complained of pain and stiffness in the extremities, shoulders, neck, and lower back. No other bone or joint diseases were found, except for a rather high incidence of arthritic lesions. The authors suggested that the relationship between fluorosis and the high incidence of musculoskeletal symptoms and arthritic lesions be evaluated in an epidemiologic study to clarify whether the cases presented had significantly more clinical symptoms than a comparable control group.

Studies by the US Public Health Service [101] in 1967 evaluated the effects of chemical irritants, including particulate and gaseous fluorides (HF), in a chemical plant. Twenty-eight room air samples for HF were taken with concentrations ranging from 0.077 to 10.0 ppm (.063 - 8.2 mg/cu m). Four samples, near control panels, exceeded 1 ppm (.82 mg/cu m). These samples had values of 1.3, 2.6, 4.4, and 10.0 ppm (1.04, 2.13, 3.60, 8.2 mg/cu m). Two general area samples were also found to exceed 1 ppm. Those samples had values of 1.8 and 1.9 ppm (1.48 and 1.56 mg/cu m). Thirty-four particulate samples were all under 0.5 mg/cu m, with a range of 0.1-0.49 mg/cu m. Before-shift and after-shift urinary F excretions were analyzed in 25 workers exposed to HF or particulate fluorides, and in 10 nonexposed office employees. Both of these groups were exposed at F concentrations of 0.6-1.1 ppm through the plant water supply. Before-shift specimens were collected after the workmen had been away from the plant on their days off, and after-shift samples were collected after each consecutive workday for 5 days, and pooled for each man. The before-shift urinary F concentration ranged from 0.33 to 4.48 mg/liter, compared to 0.95-26.6 mg/liter for the after-shift samples. Corresponding levels for the control group were 0.50-1.88 mg F/liter for the preshift, and 0.50-2.38 mg F/liter for after-shift specimens. Of 11 workers assigned to HF operations, 4 exceeded 5 mg F/liter (6.85, 8.80, 17.5, and 26.6). The 2 workers with the highest urinary F levels had accidental exposures ("gas out" and reboiler leak) during the week of urine collection. These 2 workers, along with 3 others who had long-term exposure to fluorides were given skeletal X-rays. No evidence of skeletal fluorosis was found in any of the X-ray readings.

Pulmonary function tests were performed on 305 chemical workers including workers exposed to gaseous and particulate F, and a control group of 88 workers in a box plant. [101] The chemical plant workers had a mean age of 44 years and were on the average 14 years older than the box plant workers. The observed values for forced vital capacity (FVC), 1 second forced expiratory volume (FEV1) and FEV1/FVC for the total group were within about 3% of the predicted value with no significant difference between chemical workers and the control group.

The residual volume (RV) expressed as percentage of total lung volume (TLVol) was 30.8% in the chemical workers as compared to 26.8% for the box plant workers, with both values within normal limits (35% upper limit of normal). This difference was explained by the higher average age of the chemical workers since RV/TLVol usually increases with advancing years. The FEV1/FVC of less than 70% as an indicator for pulmonary function impairment was also evaluated. In the chemical plant workers, 17% had values of less than 70% and for the box plant worker the value was 7%. Irrespective of exposure, those with a FEV1/FVC of less than 70% had depressed FEV1, a larger residual volume, a larger TLVol, and the RV/TLVol was increased to a borderline normal value of about 34%. The smoking histories of 52 chemical workers with a FEV1/FVC of less than 70% (group A) and those 253 chemical workers with a FEV1/FVC of 70% and above (group B) were quite different. Only 6% of group A had never smoked while 23% in group B had not. A higher proportion of group A were heavier smokers; 75% in group A and 56% in group B smoked a pack or more of cigarettes a day. In group A, 21% gave a history of mild dyspnea while in group B only 7% were dyspneic. Cough was reported in 37% of group A but only in 1% of group B. From the analysis of the pulmonary function data the authors concluded that the ventilatory function in most of the chemical and box plant workers was within acceptable normal limits for their age. Some decrease in function which the authors considered, without stated reasons, not to be work-related was found in a small percentage of the workers. Pulmonary function tests were performed on workers exposed to HF, fluorides, sulfuric acid, sulfur dioxide, chlorine, and ammonia but test results were not separately recorded for each substance. Viewing the results as a whole, the authors concluded that there was no relationship between the chemical irritant the workers were exposed to and the FEV1/FVC values.

H R Henderson provided additional data on environmental and urinary F levels on the same plant population (written communication, September 1974). Data obtained between March 1968 and April 1973 using an automatic HF analyzer showed the following results: 2.1% of the total 23,280 samples were 5 ppm, and over; 1.3% were 4-5 ppm; 1.5% were 3-4 ppm; 3.2% were 2-3 ppm; 11.4% were 1-2 ppm; and 80.3% were 0-1 ppm. Periodic urinary fluoride levels taken for 6-10 years on 13 HF workers revealed the average preshift levels for the workmen ranged from 2.0 to 5.7 mg/liter, while average after-shift samples ranged from 4.2 to 24.7 mg/liter. One of the 4 workers with postshift urinary F concentrations exceeding 5 mg/liter who had negative X-rays for osteosclerosis showed minimal osteosclerosis when reexamined by Henderson 2 years later. His average preshift urinary F level was 5.3 mg/liter, ranging from 2.6 to 16.3 mg F/liter. The average of his after-shift urinary F level was 11.5 mg/liter, ranging from 2.0 to 30.0 mg/liter. No definitive information was provided regarding his atmospheric F exposure levels.

In 1972, Kaltreider et al [102] reported the results of health surveys in 2 aluminum plants. In 1945-46, potroom workers at a New York aluminum plant were examined and air samples were analyzed. The electrolytic cells in this operation were not provided with local exhaust ventilation; the general ventilation in the potroom was about one change/minute. The time-weighted 8-hour air concentrations of F (ranges not given) for the different job classifications were as follows: pot tenders 2.4-3.0 mg/cu m (36% gaseous F primarily in the form of HF); tapper-carbon changers 3.0-4.0 mg/cu m (50% gaseous F primarily in the form of HF); and cranemen 4.0-6.0 mg/cu m (50% gaseous F primarily in the form of HF). The average urinary F excretion in spot samples taken during the working day was 8.7 mg/liter for pot tenders, 9.8 mg/liter for tapper-carbon changers, and 9.6 mg/liter for cranemen. There were 107 potroom workers with an average age of 51.9 years (ranging from 27 to 65 years) and an average length of employment of 19.1 years (ranging from 2 to 40 years), and all were examined. As controls, 108 employees at the same plant with an average age of 50.7 years (ranging from 22 to 70 years) and with no history of F exposure were used. The control group showed an average urinary F excretion of 0.7 mg/liter. With the exception of a higher incidence (no data given) of aching joints, particularly those of the upper extremities, in the exposed group, the medical histories were not different in the 2 groups. Hypertension (diastolic over 90 mm Hg) was more frequent among the potroom workers (25 vs 13 in the control group). Limited motion of the dorsolumbar spine was found in 22 (20.6%) of the potroom workers compared to none in the control group. Restrictive motility of the elbows was found in 11 of the exposed group vs 1 in the control group. The higher incidence of restricted mobility was considered traumatic in origin since the frequent use of sledge hammers and bars resulted in repetitive jarring of the arms. Otherwise the physical examination and laboratory procedures including urinalysis, blood counts, and forced vital capacity revealed no significant difference in the 2 groups.

Roentgenographic examination demonstrated increased bone density in 76 out of 79 potroom workers with more than 5 years of employment. [102] The increased bone density in 46 (58.3%) was classified as slight, characterized only by accentuation of trabeculation and slight blurring of the bone structure. Four (5.1%) had "moderate, diffuse structureless" bone appearance and 26 (33%) were classified as having marked fluorosis showing homogeneous marble-white opacity of the bones. In 10 cases calcification of pelvic ligaments was seen. Marked increase in density of the ribs was found in 17 cases. The degree of bone density did not appear to be related to any single job classification. When the degree of bone density was compared with years of service, no good correlation existed; however, moderate or marked bone density was observed only after 15 years of employment. The workers with marked osteosclerosis had markedly restricted movements of the spinal column. In summary, 30 of 79 potroom workers exposed to airborne F concentrations averaging 2.4-6.0 mg F/cu m and excreting an on-the-job average of 8.7-9.8 mg F/liter of urine developed moderate or marked increased bone density after 15 years of employment.

A second health survey in 1960 [102] involved 231 potroom workers of a second New York aluminum plant. The average age of those examined was 46 years, ranging from 21 to 64 years, with length of potroom service ranging from 0 to 45 years (86% had more than 10 years of service). The pots in the potroom were hooded. To serve as controls, 152 men employed in the fabrication department, with no known fluoride exposure, were examined in 1963 and 1965. Their average age was 48.8 years ranging from 31 to 64 years. Due to great variation of the workers' exposure, it was impossible to arrive at a weighted average exposure for each job classification and urinary F excretions were therefore used as an exposure index. Spot urine samples collected during the day in 1960, 1961, and 1962 showed an average of 4.63 mg F/liter. Beginning in 1966, urine samples were collected from all potroom employees at the end of the last shift of the week, and the samples were corrected to a specific gravity of 1.024. There was a marked increase in average urinary F in these samples (8.09 mg/liter) when compared with samples taken during 1960-1962 (4.63 mg F/liter). This difference could be explained by the time the samples were taken (end of the last shift of the week vs during the day). An increase of average urinary fluorides to 8.09 mg/liter in February 1966 and to 9.77 mg/liter in February 1967 indicated a breakdown in the control program. Improvements in environmental controls and personal hygiene practices led to substantially decreased urinary F levels over the following years down to 3.09 mg/liter in November 1970. Urinary F levels taken after 5 working days in 1970 revealed 113 or 39.8% with urinary F levels of 0-1.9 mg/liter; 104 (36.7%) had levels of 2.0-3.9; 43 (15.1%) had levels of 4.0-5.9; and 24 (8.5%) had levels of 6.0 and higher.

In order to estimate "fluoride body burden," urine samples were collected at the beginning of the shift commencing in 1966, after 48 and 72 hours off work. [102] Preshift urinary F averaged 1.4 mg/liter in 147 employees after 48 hours off work and 1.5 mg/liter in 148 employees after 72 hours off work. This was compared with an average postshift urinary F concentration of 3.0 mg/liter in 284 employees taken after 3-5 working days. In the group absent 48 hours, 87.1% had urinary F levels of 0-1.9 mg/liter and 10.9% had levels of 2.0-3.9 mg/liter. In the group absent 72 hours, 85.1% had levels of 0-1.9 mg/liter and 13.5% had levels of 2.0-3.9 mg/liter.

Medical history and physical examination findings revealed no statistical difference between the exposed and the control groups. [102] Specifically, there was no significant difference between the groups in the incidence of upper respiratory infection, chronic obstructive pulmonary disease, metabolic or endocrine disorders, symptomatic degenerative arthritis, dermatologic lesions, or cardiovascular diseases. Electrocardiograms (ECG) taken on 120 potroom workers in 1967 and compared with 152 ECGs of the control group showed no significant differences. Biochemical profiles done on 120 potroom workers in 1967 showed one worker with hyperglycemia, 4 workers had slightly elevated blood urea nitrogen, 7 workers had elevated uric acid, 5 workers had elevated cholesterol levels, and 5 workers had increased total bilirubin. The authors believed that the abnormal values for total bilirubin were fallacious since all occurred on the same day and none of the employees showed clinical signs of jaundice. The values for calcium and phosphorus were all normal. A biochemical profile was not run on the control group. Chest X-rays revealed no diffuse linear or nodular fibrosis. Roentgenographic examination of the spine showed no increased bone density in the 231 potroom workers. In the control group 2 cases of "suspicious" evidence of increased bone density were found but were not considered typical of fluoride deposits.

In 1972, the National Institute for Occupational Safety and Health conducted a study of an aluminum reduction facility for the primary purpose of collecting and analyzing airborne contaminants. [103] In addition, a limited amount of medical information was provided by company management officials. Of about 200 potroom workers receiving annual pulmonary function testing, 10 individuals with known or suspected respiratory problems were selected for more detailed tests consisting of chest X-rays, spirometry and steady-state CO diffusion studies. Smoking histories and the actual values were not reported but 7 of the 10 workers were stated to have marked-to-severe obstructive airway changes, as evidenced by a reduction of FEV1 and maximum breathing capacities. The examining physician expressed concern about the young age distribution of the workers with reduced pulmonary function. One of the affected workers was 39, and 3 were in their early 40's. In addition, the results of urinary F analyses covering a 1-year period and involving about 155 workers (including potroom and cryolite workers) were made available by the company. The average of the preshift samples was 2.35 with a range of 2.0-2.8 mg F/liter. The average of the postshift samples was 4.8, ranging from 3.2 to 6.5 mg F/liter. Results of breathing zone and general air samples for gaseous (primarily HF) and particulate F over a 5-day period were all less than 0.34 mg F/cu m.

Animal Toxicity

A number of experiments have been conducted in an effort to determine the relationship between fluoride intake and retention in dairy heifers. [104-106] It was found [104] that the amount of F in blood, urine, and bones was correlated with the amount of F in the diet (see Table III-14). Cattle appeared to behave as other animal species in eliminating some of the F retained in the skeleton when shifted to a low F diet. [106]

Krechniak [50] reported the effects on animals exposed to welding fumes in a 7-cu m chamber. The welding electrodes used contained about 17% calcium fluoride and the average F content in the welding fumes was 8.9%. The air exchange rate in the experimental chamber was 1.6 cu m/min. For the experiment, 150 rats and 45 rabbits were exposed for various periods up to 95 and 110 days, respectively, 3 hours daily excluding holidays, at concentrations of F averaging 3.4 mg/cu m of particulates plus 1.45 mg/cu m

TABLE III-14

EFFECTS OF FLUORIDE ON DAIRY CATTLE

	Age	Normal Conditions	Chronic fluorosis	
			Borderline	Severe
F in moisture-free diet, ppm	2	Up to 15	30-40	60-109
	4	Up to 15	30-40	60-109
	6	Up to 15	30-40	60-109
F in bone, ppm	2	401-0714	1,605-2,130	3,027-4,206
	4	706-1,138	2,379-3,138	4,504-6,620
	6	653-1,221	2,794-3,788	5,622-8,676
F in urine, ppm	2	2.27-3.78	8.04-10.54	14.71-19.86
	4	3.54-5.3	10.32-13.31	18.49-25.63
	6	3.51-6.03	11.29-14.78	20.96-30.09
F in blood, ppm	2	Up to 0.30	0.15-0.40	.50 and above
	4	Up to 0.30	0.15-0.40	.50 and above
	6	Up to 0.30	0.15-0.40	.50 and above

From Shupe et al [104]

of volatile fluoride compounds (assumed by Krechniak to be HF). Some animals were killed at different time intervals and F in the organs and tissues was determined. In the lungs of both animal species the F levels increased significantly after 3 days of exposure ($p < 0.05$) compared to controls. At the end of the exposure the F content of the lungs in rats was 5 times control levels, while in the rabbits the F content increased less than 3 times. Twenty weeks after termination of the experiment a distinct reduction, but not total elimination of F from the lungs, had taken place.

The F level of the blood increased rapidly to twice the normal level and remained constant during the exposure. [50] The F content of muscles, liver, kidney, and brain increased significantly after a month of exposure, remained at the elevated level, and decreased to near control levels during the recovery period which lasted a minimum of 30 days. The highest F retention occurred in the bones and teeth and was directly proportional to the length of exposure. Little F was eliminated from the bones and teeth during the recovery period. Urinary F excretion in the rabbits gradually increased from 2.0 mg/liter after 3 days of exposure to 6.2 mg/liter after 28 days of exposure. The controls excreted an average of 1.3 mg F/liter. After 40 days of exposure, urinary F levels dropped slightly to 4.2 mg/liter and remained at this level for the remainder of the experiment. During the recovery period there was a slight increase to 5.4 mg/liter. Fecal F also showed a significant increase during and after exposure possibly caused by ingestion of welding dust by the animals.

Correlation of Exposure and Effects

All epidemiologic studies and case reports with the exception of the study by Derryberry et al [97] have a common weakness, in that effects were reported which undoubtedly were cumulative effects of past exposure while atmospheric levels were measured at the time of the study, thus failing to give long-term environmental data. Nevertheless, limited correlations between exposure levels and effects can be drawn.

Roholm [18] reported digestive disturbances (nausea, vomiting, loss of appetite, diarrhea, or constipation) in over 80% of a population exposed to cryolite at an estimated 15-20 mg F/cu m. Moller and Gudjonsson [17] in a study of cryolite workers found digestive disturbance in 54% of the exposed population. No environmental data were given but based on the high percentage of osteosclerotic bone changes (39%) in the exposed population one can assume high exposure levels. The study of Agate et al [26] indicated a relatively higher incidence of symptoms of the digestive tract in a group of aluminum furnace room workers exposed to atmospheric F levels of 0.14-3.43 mg F/cu m when compared with a control group (14.7% vs 5.2%). Midttun [85] reported nausea in the majority of new employees in the potroom of an aluminum factory but the symptoms passed in a few days. Atmospheric data indicated an exposure at 1-2 mg F/cu m. Other clinical studies [93,94,97,102] gave no indication of increased incidence of digestive disturbance for F exposures ranging from 0.143 to 8.32 mg/cu m.

Roholm [18] reported increased bone density in 83.8% of a population of cryolite workers who were exposed to levels of 15-20 mg F/cu m at the time of the study. Minimal changes were found in 38.2%, moderate changes in 34.3%, and marked changes in 10.3%. Kaltreider et al [102] reported increased bone density in 96% of a group of potroom workers in an aluminum factory with a time-weighted average 8-hour exposure of 2.4-6.0 mg F/cu m. Minimal osteosclerotic changes were seen in 58.3%, moderate changes in 5.1%, and marked changes in 33%. Derryberry et al [97] reported minimal or questionable skeletal fluorosis in 23% of a group of 74 fertilizer manufacturing workers with a time-weighted atmospheric F exposure of 1.78-7.73 mg F/cu m (average 3.38). Of those found to have minimal or questionable increases in bone density, 71% were exposed to time-weighted F levels greater than 2.5 mg F/cu m (see Table III-13). Tourangeau [93] possibly found (X-ray interpretations were questionable) 2 cases of generalized and 3 cases of localized osteosclerosis in a selected group of 10 aluminum potroom workers out of a total group of 104. Exposure of these workers was estimated as less than 4 ppm (3.3 mg F/cu m). Agate et al [26] apparently found only a few cases "showing appearances consistent with skeletal fluorosis" in a group of 189 aluminum furnace room workers with an environmental exposure of 0.14-3.43 mg F/cu m. Bowler et al [94] reported only 1 case suggestive of osseous fluorosis in a group of 124 magnesium foundry workers with an atmospheric exposure level of 0.143-6.37 mg F/cu m.

Correlation of increased bone density and urinary F levels were reported in several studies. Bowler et al [94] found one case of osteosclerosis in 54 magnesium foundry workers with an average urinary F level of 2.53 mg/liter and a range of 0.9-7.5 mg/liter. Agate et al [26] reported 48 cases of increased bone density in 189 aluminum foundry workers. Twenty-four hour urinary F excretion of the total exposed group ranged from 0.5 to 23.3 mg F/24 hrs. The mean urinary fluoride excretion of fully exposed furnace room workers who had abnormal X-ray appearances was 9.36 mg F/24 hrs. Workers who had normal X-rays excreted 9.08 mg F/24 hrs. The difference was judged to be statistically insignificant. Largent et al [96] found 2 cases of marked osteosclerosis out of 13 workers in an inorganic fluoride manufacturing plant. These 2 cases had average postshift urinary F excretions of 11.65 and 16.38 mg F/liter, ranging from 6.08 to 23.90 and 9.28 to 24.00 mg F/liter, respectively. No changes in bone density were found in workers with a postshift average of 6.51 mg F/liter or less. Derryberry et al [97] reported 17 cases of minimal or questionable osteosclerosis in a group of 74 workers in a fertilizer production plant. The average urinary F excretion (average of 38 postshift samples per worker) in the affected group was 5.18 mg F/liter ranging from 2.2 to 8.9, compared to 4.53 mg F/liter with a range of 2.1-14.7 in 57 workers demonstrating no increase in bone density. Table III-12 shows the incidence of increased bone density compared to urinary fluoride levels at 1 mg F/liter increments. It demonstrates that as the average urinary fluoride level increased the percentage of suspected cases of osteofluorosis also increased.

Kaltreider et al [102] found osseous fluorosis in 76 out of 79 aluminum potroom workers with many exhibiting marked restriction of motility of the spine. Urinary spot samples collected during working days at the time of the survey showed an average F excretion of 8.7 mg F/liter for 32 pot tenders, 9.8 mg F/liter for 25 tapper-carbon changers, and 9.6 mg F/liter for 8 cranemen. In a later study [102] at a different aluminum plant, no cases of increased bone density in a group of 231 potroom workers were found. The average urinary F level from spot samples taken during working days for the first 3 years of the survey was 4.63 mg F/liter for the total group. During the last 3 years of the study the average urinary F level based on spot samples obtained at the end of the shift on last day of the workweek and corrected for a specific gravity of 1.024, was 4.64 mg F/liter. During the intervening years the average increased to 8.9 mg F/liter which, the authors noted, clearly indicated some breakdown in the control program. Subsequent changes "both in the control program and hygienic practices" (details not provided) resulted in a lowering of values as previously noted.

The relationship of preshift urine samples to postshift samples was studied by comparing samples collected after 5 working days with samples collected after 48 hours and 72 hours off work (preshift). [102] Thirteen workers in a chemical plant excreted an average of 2.0-5.7 mg F/liter (preshift) and an average postshift range of 4.2-24.7 mg F/liter. X-ray films of 1 worker revealed minimal osteofluorosis. (HR Henderson, written communication, September 1974) Average pre- and postshift urinary F concentrations for this employee were 5.3 and 11.5 mg/liter with a range of 2.6-16.3 mg F/liter and 2.0-30.0 mg F/liter, respectively. Data on the worker's fluoride exposure were not provided.

Roholm [18] reported signs of pulmonary fibrosis in workers exposed to 15-20 mg F/cu m with total dust concentrations ranging from 30 to 40 mg/cu m. Quartz content of the dust ranged from 1 to 5%. In a group of magnesium foundry workers with an F exposure of 0.143-6.37 mg/cu m, Bowler et al [94] found evidence of reticulation of the lung stroma in 28 of 78 workers examined. Radiological examinations of the chests of 105 pot workers by Tourangeau [93] demonstrated increased hilar markings and reticulation of the stroma in 16 workers (15.2%). These changes were seen more frequently in workers with more than 3 years' exposure. This group of workers was exposed to an average of 3.3 mg F/cu m. Derryberry et al [97] reported a significantly higher incidence of borderline pulmonary changes in a group of workers exposed to 0.5-8.32 mg F/cu m when compared with a control group (14.9% vs 6.0%). No diffuse linear or nodular fibrosis was found by Kaltreider et al [102] in a group of potmen with an exposure ranging from 2.4 to 6.0 mg F/cu m.

Increased incidence of bronchial asthma has been reported in Norwegian aluminum foundry workers by Midttun [85] and Evang. [84] Midttun found 54 cases of asthma in a group of 400 potroom workers over a 5-year period. Air samples taken under normal working conditions showed F concentrations between 1-2 mg F/cu m. Epidemiologic studies in aluminum plants outside Norway and in other inorganic fluoride operations failed to show any significant evidence of increased incidence of asthma. [38,93,94,102] Evang, [84] who first reported the occurrence of bronchial asthma in aluminum foundry workers, suggested the cause to be the distillation products of tar, pitch, and coal. On the basis of the data available, it seems inappropriate to attribute asthma to fluoride exposure. However, this point should be further examined in future studies.

The widespread use of fluorides within industry and within the community has resulted in extensive study of the properties of fluoride-containing compounds and their effects on man and animals. This study has in turn given rise to a great amount of scientific controversy. It is not possible for any single scientific review to be totally comprehensive in dealing with these aspects. This criteria document has attempted to review critically that evidence bearing on a recommended occupational health standard for inorganic fluorides. Further information regarding uses and effects of fluorides can be found in the National Academy of Sciences-National Research Council _Fluorides_, [1] the World Health Organization monograph _Fluorides and Human Health_, [2] the chapter by Hodge and Smith "Fluorides and Man" [3] in the 1968 issue of the Annual Review of Pharmacology, the chapter by Hodge and Smith "Biological Properties of Inorganic Fluorides," found in the series _Fluorine Chemistry_, [4] and "Pharmacology of Fluorides" in the 1966 and 1970 issues of the _Handbook of Experimental Pharmacology_. [5]

IV. ENVIRONMENTAL DATA AND ENGINEERING CONTROLS

Sampling Methods

Numerous methods of sampling and analysis for fluorides have been developed and reported. The National Academy of Sciences [1] summarized much of the current data available on processing of biological samples. Pack and Hill [107] and others have reviewed sampling and analytical methods. [108-115] Tentative methods of analysis based on techniques available prior to 1970 have been published. [116]

In atmospheric sampling of fluorides, the usual sampling problems arise and are occasionally complicated by the presence of both gaseous and particulate forms. Sampling methods that separate gaseous from particulate fluoride rely on the reactivity of the gaseous forms with aluminum, alkali, or other materials. Habel [117] used a quartz filter device containing a membrane filter followed by a series of impingers to achieve a separation of the two fluoride components. Pack et al [118] used an assembly of 3 concentric aluminum tubes attached to an impinger to accomplish a sharp separation of gaseous and particulate compounds. A less distinct separation occurred when sodium bicarbonate-coated glass tubes were used in conjunction with an impinger. Mandl et al [119] found that when sodium bicarbonate-coated glass tubes were used, efficiency of gaseous uptake was affected by tube diameter. Mandl et al [119] also used a tape sampler which used 2 treated tapes, one to remove particulates and one to remove gaseous fluoride compounds.

Membrane filters were reported [118] to collect aerosols at average efficiencies greater than 98% of the amount absorbed in 2 impingers in series while retaining less than 7% of gaseous fluoride. Mandl et al [119] reported that the pretreatment of filters with citric acid solution reduced the retention of gaseous fluoride. Other investigators reported that impregnation of the filters with potassium carbonate, [120] calcium oxide, [121] and sodium formate [122] allowed collection of the gaseous fluorides. Separation of gaseous and particulate fluorides was accomplished by Jahr [123] by the use of an untreated membrane prefilter, of 0.8-μm pore size, in conjunction with a sodium formate-treated 5-μm secondary filter. After sampling, the filter assembly was warmed to 75 C to allow diffusion of adsorbed HF from the first to the second filter. He determined the diffusion efficiency to be better than 99% after 20 hours of heating at 75 C. GH Farrah (written communication, September 1974) has submitted a method to the American Society for Testing and Materials (ASTM) Sub-Committee D 22.04 (tentative method, unpublished) using an untreated membrane filter backed by a carbonate/glycerol impregnated cellulose pad to effect particulate-gaseous separation. Collection efficiency, precision, and accuracy of the analytical method have not been submitted.

NIOSH compared two different sampling methods to collect airborne fluorides in potrooms and a cryolite recovery operation associated with operations in an aluminum reduction plant. [124] The first method consisted of a membrane filter for particulates, followed by a midget impinger filled with 0.1 N sodium hydroxide to absorb the gaseous fluorides. The second used a 0.8-μm pore size filter which was impregnated with sodium formate so that gaseous as well as particulate fluorides would be captured. Nine of 12 simultaneous, paired general area samples collected a higher total F (combined particulate and gaseous F) by the sodium formate-impregnated membrane method than by the membrane filter-impinger method. The analytical method (ion-selective electrode) was the same for both sampling methods. Unfortunately, filter blank values were not included in the data.

Treated filters were used for rapid determination by Harrold and Hurlburt. [125] Luxon [126] used wool filters. The use of limed papers was investigated by Adams, [127,128] Robinson, [129] and Wilson et al. [130]

Farrah, [114] in discussing standard impingers, stated that although the standard impinger is easily cleaned, fairly rugged and compact and gives high collection efficiencies for gaseous and particulate fluorides, it has several operational drawbacks. Attention must be given to maintaining the liquid level within the impinger, and the liquid must be protected from freezing in cold weather. Glass fiber filter paper [131] proved to be a simple rugged system for efficient collection of both gaseous and particulate fluorides but suffered from high blank values.

For personnel monitoring of total fluorides, the sampling method of choice is the sodium formate-treated membrane filter [122-124] as outlined in Appendix I, because it permits the simultaneous collection of gaseous and particulate fluorides on a single filter, has a high collection efficiency, and the collection technique is relatively simple.

Analytical Methods

Water soluble forms of fluorides can often be analyzed directly but unless determined otherwise, it should be assumed that the samples contain refractory forms of fluoride.

Various techniques for the isolation of fluoride from the sample have been developed. Tentative isolation methods including distillation, diffusion and ion exchange have been published. [116] Fluorides collected in bubblers and impingers can be concentrated by anion exchange resin in the hydroxyl form [132,133] or in the acetate form. Talvitie and Brewer [134] used cation exchange resin as a bacteriostatic agent to preserve urine and then passed the urine through an anion exchange resin. Bowley [135] used ion exchange coupled with colorimetry and titration. One of the oldest techniques for the separation of fluoride has been the steam distillation method devised by Willard and Winter. [136] Many modifications of the distillation method have been published. [137,138] Singer and Armstrong [139] introduced a diffusion method in which the sample is sealed in a multichamber cell with acid and a collecting base. Modifications with a variety of cells have applied the technique to urine. [140-144] The isolation method offering the greatest advantage is the diffusion method since large eluent volumes, constant operator attention, extensive glassware, and resin beds are not necessary for good analytical results.

Many manual and semiautomated methods for analysis of fluoride are available. The choice of a method depends on the type of sampling, separation, sensitivity, and accuracy required. Titrimetric methods were among the first to be used effectively for air samples. Willard and Winter

[136] titrated with a standard solution that complexed fluoride. Armstrong modified this approach. [145] A frequently used complexing agent is thorium with alizarin red as the endpoint dye. Many modifications have been made with various dyes and complexing agents. [146,147] Talvitie and Brewer [134] used a cation exchange resin to form hydrofluoric acid which was titrated with base. A detailed review of most of the methods known through 1950 is contained in reviews by McKenna. [108,109,110]

Trace amounts of fluoride ion may be estimated directly in body fluids or water by measuring their inhibitory effect on the enzymatic hydrolysis of ethyl butyrate. [148] Colorimetric methods outnumber all other methods of fluoride analysis, although they were not used successfully until 1943. [149] Thorium with an alizarin sulfonate lake was decolorized by the fluoride. [150,151] Another colorimetric method involved the use of zirconium-alizarin sulfonate. [152] A bleaching system by Megregian [153] used zirconium-eriochrome cyanine-R complex. According to Rausa and Trivello, [154] Belcher in 1959 introduced alizarin complexone which forms a red chelate with cerium(III) and forms a blue, stable complex in the presence of fluoride ion. Another colored complex of lanthanum(III)-alizarin complexone is also used. [137,155-157] The lanthanum methods are less sensitive but have a greater range and fewer interferences. Other dyes which have been suggested are thorium-chrome azurol-S, [158] and aluminum-dyes. [159]

The Megregian zirconium-eriochrome cyanine-R method was applied by many laboratories [116,160-163] because it is almost as sensitive and develops much faster than the zirconium-alizarin system. [152,164] The zirconium-xylenol orange method forms a positive color which has greater sensitivity. [165,166] In 1958, Bellack and Schouboe [167] introduced trisodium 4,5-dihydroxy 3-(p-sulfophenylazo) 2,7-naphthalenedisulfonate (SPADNS) as a dye for use with zirconium. It proved more stable and reacted more rapidly with a broader absorption spectrum which made it easy to use with simple filter photometers. This system can be substituted in many procedures based on the zirconium-alizarin system. [8,22] Other metal-dye complex systems have been used although not widely applied. [168,169] Noteworthy among them is the fluorescence-quenching technique using magnesium 8-hydroxyquinoline. [170]

The most commonly used and adaptable colorimetric methods are zirconium-alizarin, zirconium-SPADNS, the zirconium-eriochrome cyanine-R, and the lanthanum-alizarin or cerium-alizarin complexone systems. With minor differences in sensitivity and interferences, they are almost all interchangeable. Chemical interferences, eg, chloride can cause erroneous results in these types of methods.

Polarographic and other electrochemical methods have been developed for fluoride, but have received little attention in the published literature. [171]

A recently developed gas chromatographic detection method for fluoride [68,172-176] may be useful for analyzing gaseous fluorides in the presence of particulate fluorides. X-ray diffraction was applied to fluoride analysis by Lennox and Leroux. [177] Neutron activation analysis was applied by Spoonemore [178] and atomic absorption, [179] nondispersive infrared analysis, [180] photonuclear activation, [178] pyrohydrolysis, [181] and particulate identification have been used for analyzing fluoride. Mass spectrometric methods [182] have not been widely used.

A number of fully and semiautomatic sampling instruments have been devised for the measurement of fluoride levels in air and urine. [95,137,138,142,163,170,183-191]

The development of the fluoride ion-selective electrode provided a rapid, direct method of analysis. [192] The electrode employs a crystal that develops a potential dependent only on fluoride ion activity which is measured in reference to a calomel electrode by an expanded-scale pH meter. Both potentiometric and direct measurements were developed. [193-196] Methods have been published for analysis of fluoride in air and stack samples, [122] blood, plasma, urine, bones, teeth, and other materials. [95,183,191,197-202] The fluoride ion-selective electrode has made measurement of urinary fluoride concentrations easy, rapid, and routine. [197,198,203] It offers the most rapid, simple, and specific method for measuring fluorides in urine when used between pH values of 4.5-8. Sun [198] compared results of ion-selective electrode urinalysis obtained by direct measurement, microdiffusion-colorimetric, and microdiffusion-electrode methods. No significant difference was found in 45 samples analyzed by ion-selective electrode, either directly or after microdiffusion. A comparison has been made between SPADNS and electrode methods for stack emission samples. [204] In some cases interfering substances such as aluminum made prior preparation or separation of the sample necessary before the final analysis with the electrode, but interference effects were generally found far less often with electrode analysis than with chemical analyses when a TISAB solution containing CDTA is used. [192] The great working range of concentration makes the method very useful (0.03 μg/ml to 20 mg/ml) with sufficient accuracy (±1%) for most determinations. [122,172,183,193-196,199,200,205,206] Further studies are indicated for ionic fluorides in biological fluids. [207] The American Society for Testing and Materials [208] outlined 2 methods for water analysis (SPADNS photometric and ion-selective probe) which are commonly applied with minor modifications to collected air samples of soluble fluorides and to urine samples. Incomplete solubility or complexing of the fluoride are conditions which require prior processing of the sample.

The method of choice for fluoride analysis in air or urine samples is determination by ion-selective electrode as outlined in Appendix II. The relative adaptability, ease of analysis, reproducibility, and linearity of response over an analytical range of nearly 100,000 provide a sound basis for the choice of method. If test conditions indicate an isolation step is necessary because of the possible presence of complexing ions, eg, aluminum, iron, and silicon, diffusion should first be used for separation and concentration.

Environmental Levels and Engineering Controls

A number of studies have been conducted to determine the workroom levels of fluorides in various industrial operations. Engineering controls were often mentioned, but few details of control installations were reported, and in most cases effectiveness of control methods was not determined by actual measurement. Generally, these studies tended to indicate that fluoride levels in industry were not grossly excessive, suggesting that effective control is readily obtainable by standard methods. The following studies, categorized by type of industrial operation, have been selected as examples.

(a) Foundry Operations

In 1942, Williams [209] reported concentrations of various airborne materials generated during the casting of magnesium metal. Fluorides were used as oxidation inhibitors and fluxes. Fluorides used as inhibitors were not identified except as soluble fluoride salts. Fluxes were generally composed of ammonium acid fluoride, ammonium fluoborate, or ammonium fluosilicate. Cores were sprayed with aqueous 20% solutions of fluorides, also not identified. Furnaces were reportedly ventilated. Major fluoride exposures occurred during addition of fluoride inhibitors to molten magnesium and during the shakeout of finished castings. Samples indicated that HF was present in the workroom air in insignificant amounts compared to particulate fluorides. Results of sample analyses are shown in Table IV-1.

Williams [209] stated that concentrations exceeding 10 mg F/cu m caused nasal irritation but that filter respirators appeared to provide adequate protection. He also stated that provision of exhaust ventilation for furnace and shakeout operations would keep fluorides within "reasonable

TABLE IV-1

CONCENTRATIONS OF FLUORIDE GENERATED
DURING THE CASTING OF MAGNESIUM METAL

Operation	Range (mg F/cu m)	Average (mg F/cu m)
Shakeout	0.94-18.0	8.77
Molding	0.44-10.6	1.88
Melting	0.74-1.76	1.26
Core spraying	0.58-0.94	0.7

From Williams [209]

limits" throughout the foundry. He recommended that core spraying operations be conducted in booths, apparently meaning ventilated booths.

In 1943, Largent and Ferneau [37] studied the urinary excretion of fluorides by workers in 3 magnesium foundries. Only 3 air samples for total fluorides were taken, an insufficient number to provide conclusive indications of the severity of exposure. The foundry from which the largest number of samples were collected was recently constructed, was clean, and was provided with extensive ventilation for core making, core spraying, melting, and shakeout operations. The cooling sheds were also reported as being well ventilated. Highest exposures, as indicated by urinalysis, were found in the core spraying and pouring areas, although extensive ventilation was present in both locations. Core spraying took place in ventilated booths and, in addition, the workers were provided with respirators. Largent and Ferneau [37] conjectured that laxity in wearing the respirators was responsible for the elevated urinary fluoride levels.

Methods for the collection of fluoride fumes in air were studied in 1945 by Williams and Silverman. [210] To confirm their laboratory results, they sampled the air of a magnesium foundry. Parallel samples were taken using electrostatic precipitators and Whatman filter papers. Fluoride concentrations were found ranging from 0.079 to 1.13 mg F/cu m. Unfortunately, no details of sampling locations or engineering controls were given.

Stimulated by the work of Largent and Ferneau, [37] Bowler et al [94] in 1947 made a similar study of an English foundry engaged in the casting of magnesium alloys. They did not attempt to determine gaseous fluorides. Fluoride concentrations ranged from 0.143 to 6.37 mg F/cu m. No mention was made of controls except the statement that the "baleout" type of furnace was partially enclosed. Whether or not this enclosure was effective could not be determined by the sampling and analytical results, since sample locations were identified only as "near furnaces."

(b) Electric Arc Welding, Brazing, and Soldering

Low hydrogen coated welding electrodes use fluorides, generally calcium fluoride, as a coating component. Fluxes used in silver brazing and aluminum soldering contain fluorides. [211,212] A number of studies have been made of the fluoride hazard of these operations. In 1941, Tebbens and Drinker [213] studied the fumes and gaseous products of welding with coated electrodes and the engineering factors required for control. Laboratory tests indicated that from 1 to 5 mg F/cu m were produced by welding with a single rod in a volume of 1,000 cu ft without exhaust ventilation. Gaseous fluorides were not found in significant amounts. Based on further tests to determine ventilation requirements, they recommended 250 cubic feet per minute per welder for control by general dilution ventilation when using fluoride-coated rods in spaces of 2000 cu ft or less, and 150-200 cu ft/minute for control by local exhaust ventilation with the intake positioned a few inches from the arc.

Studying welding fumes in steel fabrication, Drinker and Nelson [214] in 1944 observed that hydrogen fluoride was not present in fumes from fluoride-coated rods. They recommended local exhaust ventilation for control with a minimum air flow of 200 cu ft per minute per welder.

In 1953, Ferry [215] measured the fluoride concentrations to which a welder might be exposed under varying conditions. He first determined the concentrations of fluoride in various locations with respect to the welder's helmet, finding the lowest concentrations at the rear of and inside the helmet, and the highest concentrations outside the helmet at the front and sides. Concentrations inside the helmet ranged from 0.7 to 2.8 mg F/cu m. The range of concentrations outside the helmet was 0.35-30.0 mg F/cu m. He further confirmed that gaseous fluorides were not present in determinable amounts. Field samples were taken to determine welders' exposures without local exhaust ventilation and to determine general air concentrations in work areas. While the 6 areas investigated varied in type of work and size, general ventilation was reportedly good or fair in all. Breathing zone samples were collected under the welders' helmets, while general air samples were collected in the room air away from the direct path of welding fumes. Results of analyses were reported only in ranges vs number of samples in each area. Breathing zone concentrations ranged from undetectable to 10.0 mg F/cu m. Of 63 samples, 40 were below 1.5 mg/cu m, while 33 were below 1.0 mg F/cu m. General air samples ranged from undetectable to 0.64 mg F/cu m. Work habits of welders were considered to be the most significant cause of variation in results. Ferry [215] concluded that the number of variables was too great to permit accurate exposure evaluation by air sampling and recommended monitoring by urinalysis.

In 1966, Smith [216] studied the total welding fume and fluoride exposure of welders using low hydrogen electrodes under a variety of conditions in the fabrication of pressure vessels and submarines. The environments studied were classified as "confined" (totally enclosed volume of less than 2000 cu ft), "enclosed" (compartments with at least one side open and a volume generally greater than 2000 cu ft), and "open" (open air or large workshop). Total fume samples collected on cellulose filter papers in these 3 degrees of confinement indicated wide variations in concentrations, ranging from 0.4 to 112.0 mg/cu m.

The effects of using local exhaust ventilation were demonstrated [216] by the results shown in Table IV-2. While the median concentrations indicate some control of total fumes by use of ventilation, the ranges indicate that, in some instances, exposures were higher with ventilation than without. Smith stated [216] that locating exhaust ducting too far from the fume source reduced effectiveness. Presumably this was the reason for the discrepant results listed in the table. Three hundred and two welding fume samples taken in 6 different industrial situations contained median fluoride concentrations ranging from 0.55 to 2.93 mg /cu m. Seventy-eight percent of the samples contained less than 2.5 mg F/cu m and 7% contained more than 5 mg F/cu m. Samples taken in "confined" areas generally showed higher concentrations of fluorides than did samples taken in areas classified as "enclosed" or "open." Fluoride concentrations greater than 2.5 mg/cu m were found in all confined and enclosed welding locations. Thirty-seven percent of the samples taken in confined environments showed concentrations of fluoride exceeding this value. Smith [216] calculated that the weighted exposures for an 8-hour day in the areas studied ranged from 0.17 to 0.88 mg F/cu m. He stressed the importance of adequate ventilation for control.

In 1969, Krechniak [50] studied the fluoride hazard of automatic submerged arc welding in a shipyard production hall where 30-50 welding stands were in operation. Air samples were obtained from the welder's breathing area. Particulate sampling for fluorides was carried out using

TABLE IV-2

FLUORIDE FUME EXPOSURE OF WELDERS
USING LOW HYDROGEN ELECTRODES

Environmental Condition	Number of Samples	Total Fume Concentration (mg total fume/cu m)		
		Maximum	Minimum	Median
Confined,				
Without exhaust	66	112.0	1.0	21.0
With exhaust	40	41.9	6.3	17.6
Enclosed,				
Without exhaust	76	57.6	0.7	10.2
With exhaust	25	77.8	1.4	5.6

From Smith [216]

filter paper. In order to determine volatile F compounds, air was "blown" into polyethylene bottles containing dilute sodium hydroxide. Analysis showed an average concentration of gaseous fluoride compounds of 1.4 mg HF/cu m and 0.32 mg particulate F/cu m at the welder's stand. Pantucek [217] did not find gaseous fluorides in fumes from basic coated electrodes; however, the sampling and analytical methods were not specified.

In investigating exposures to cadmium and oxides of nitrogen, Mangold and Beckett [51] determined fluoride exposures due to silver brazing with fluoride flux. Air samples taken in a large open shop showed fluoride concentrations ranging from 0.02 to 0.16 mg/cu m with a mean of 0.12 mg/cu m. Samples taken aboard ship under more confined conditions showed that fluorides ranged from 0.28 to 0.80 mg/cu m with a mean concentration of 0.51 mg/cu m. The investigators did not feel that fluorides presented a hazard in this type of operation. No local exhaust ventilation was used.

In 1964, McCann [218] investigated the health hazard from flux used in joining aluminum electric cables. The flux was believed to contain ammonium fluoborate, aminoethylethanolamine, and zinc oxide. The majority of the fluoride in the fume was particulate rather than gaseous matter. The cable joiners worked in relatively confined spaces such as tent-covered trenches, houses, substations, and manholes. Fumes were generated when the cable was brushed with flux and when hot solder was poured over each core end after fluxing. Sampling tests were performed in a simulated work situation. Ventilation was purposely made poor, and large amounts of flux were used. Breathing zone concentrations were found to range from 1.2 to 5.3 mg F/cu m. McCann [218] calculated peak concentrations of 5.0-34.6 mg F/cu m. While these levels were recognized as being high, it was stated that under the worst conditions jointers were exposed to such levels for only a few minutes a day. Recommended preventive measures included education of the jointers, minimal use of flux, good personal hygiene, use of barrier creams and gloves, and avoiding breathing the fumes. Filter respirators were recommended for confined spaces. Apparently, it was considered impractical to utilize local exhaust ventilation under the work circumstances. Time-weighted exposure data were not reported.

(c) Aluminum Production

In 1946, Yaffe [219] reported an investigation of concentrations of fluorides in aluminum plants. Samples were collected to determine airborne concentrations of gaseous and particulate fluorides in locations which would be representative of exposures of potmen, tappers, and carbon crews. Exposures of crane operators were not reported. Total fluoride in the air ranged from 0.18 to 5.84 mg F/cu m, of which about 50% was gaseous (primarily HF). Average time-weighted exposures were calculated by Yaffe

[219] who found no significant differences in the exposures of the various occupational categories. Time-weighted average (TWA) exposures ranged from 0.8 to 1.0 mg F/cu m. Engineering controls were not reported.

In 1949, Agate et al [26] reported investigations of fluoride exposures in an aluminum plant. Gaseous and particulate fluorides were determined separately. In addition, particle size determinations were performed on particulate fluorides. Three furnace rooms were included in the investigation. Furnace room "A" was described as having ventilation provided only by convection currents and natural ventilation through doors and windows. Furnaces in furnace room "B" were enclosed and provided with an exhaust system, while furnace room "C" was described only as clean, cool, and visually clear of fumes. Total fluoride ranged from 0.37 to 3.43 mg/cu m in furnace room "A" with gaseous fluorides (primarily HF) ranging from 44 to 74% of the total. Furnace room "B" samples contained total fluorides ranging from 0.41 to 2.72 mg/cu m, with 39-65% gaseous. Furnace room "C" airborne fluorides ranged from 0.14 to 0.60 mg/cu m with gaseous fluorides comprising 15-62% of the total. Background air samples taken throughout the factory showed concentrations of total fluorides ranging from 0.015 to 0.141 mg /cu m. Particle size distributions were essentially the same for all furnace rooms, 100% of the particles being below 60 μm in size, with only 6-12% by weight being below 10 μm.

Breysse [220] studied exposures of aluminum potliner removers in 1959. Samples taken inside pots while removing liners ranged from 0.6 to 2.35 mg F/cu m. Airborne concentrations varied directly with the method of pretreatment, specifically with the amount of water used. A method using "wagon drills" to break up potliners was to be instituted which would preclude the necessity for entry of personnel into the pots to break up the liners. It was felt that this would reduce exposures considerably. Dust control measures were recommended, but they were not described.

A succinct discussion of the 3 major aluminum production methods and the problems encountered in use of ventilation for control of cell effluent was presented by Hickey [221] in 1968, who stated that the prebake, horizontal stud, and vertical stud Soderberg processes could all be equipped with ventilated enclosures on the cells. However, effectiveness of this ventilation depended on strict maintenance procedures, careful initial design, and maintaining enclosure integrity (doors and shields closed) as much as possible. Hickey [221] stated that the control of cell effluent from the prebake process involved the fewest problems. Few hydrocarbons were emitted and particulate matter was easily collected in multiple cyclones or fabric filters. Particle size of particulate fluorides was reported to range from 0.05 to 0.8 µm. He added that good door and shield maintenance and rigid operational practices could hold effluent from the building itself (and presumably this involves air within the building to which workers may be exposed) to 5-10% of total cell effluent.

In 1970, Hiszek et al [222] studied health hazards of fluorides in aluminum plants. Working conditions were briefly mentioned in minimal detail. Every electrolytic cell was equipped with "fume-absorbing" or burning devices which disposed of most of the effluent gases. Based on 88 measurements with air cleaning devices in full operation, the average fluoride content of the plant air was 1.1 mg/cu m.

In a health survey of aluminum workers, Kaltreider et al [102] in 1972 reported workroom air levels of 2.4-6.0 mg F/cu m in a prebake type aluminum plant in which the cells were not hooded. Thirty-six to 50% of the fluorides sampled were gaseous (primarily HF).

(d) Phosphate Fertilizer Production

Although the production of phosphate fertilizer is one of the major sources of airborne fluorides, few studies have been made detailing worker exposure. In 1961, Rye [38] reported clinical observations of phosphate workers. He reported that the dried phosphate rock contained an average of 3.5-4.2% fluoride. The study covered 3 operations: the production of phosphate rock, phosphoric acid, and triple superphosphate (TSP). The production of phosphate rock involved drying, screening, and grinding operations productive of dust, 10% of which was below 10 µm in size. Controls for this process were not reported, nor were airborne fluoride concentrations. However, analysis of urine for fluoride and the physical examination of workers did not reveal evidence of excessive fluoride exposure. Treatment of the phosphate rock with sulfuric acid to produce phosphoric acid generated gaseous fluorides. Engineering controls were present but were not described. Airborne concentrations of fluoride, calculated as HF, were below 3 ppm. The production of TSP produced both gaseous and particulate airborne fluorides in the proportion of 60% particulate to 40% gaseous. Exhaust ventilation was installed on the mixing cone and setting belt, but further details were not given. Total fluoride concentration in the air of the storage building, where the major exposure occurred, was given as 2-4 ppm as HF (approximately 1.5-3.1 mg F/cu m). Rye stated that a major portion of the particulate matter was below 1 µm in size. Respirators were required to be worn, and administrative controls in the form of duty rotation were used to reduce exposure in the storage building.

In 1962, Derryberry et al [97] reported results of a study of worker health in a phosphate fertilizer plant. Operations involved handling and calcining of phosphate rock, production of elemental phosphorus, conversion of phosphorus to phosphoric acid, acidulation of pulverized phosphate rock with phosphoric acid, production of calcium metaphosphate, and storage, aging, bagging, and shipping of finished products. Gaseous and particulate fluorides were produced throughout these processes. Most of the dusts and gases were reportedly removed by scrubbers and local exhaust systems, but details were not given. Time-weighted exposures ranged from 0.50 to 8.32 mg F/cu m with an average of 2.81 mg F/cu m. Unfortunately, this range was not broken down to indicate exposures at each of the various industrial operations, nor was the distribution of total F in terms of gaseous and particulate components reported.

Kuznetsova [223] in 1969 reported air-sampling results in a phosphate plant. Near the conveyor, the mean particulate F content of the air was 1.3 mg F/cu m and 0.27 mg F/cu m at the processing equipment platforms. In the crushing department, equipment operators were exposed to 1.3 mg F/cu m. Similar exposures occurred near the drying drums. In the double superphosphate department, the highest concentration, 2.8 mg F/cu m, was found in the work area of the equipment operators. The powdering and drying process produced 3.1 mg F/cu m of air. Existence of control measures was not reported.

(e) Water Supply Fluoridation

In 1950, Zufelt [224] reported exposures in a water fluoridation facility. The charging hopper was equipped with exhaust ventilation. The feed machine had been made as dusttight as possible. Operators were fitted with rubber gloves and dust respirators for use when filling the hopper. Atmospheric fluoride concentrations ranged from 0.025 to 0.134 mg/cu m, calculated as HF (approximately 0.024-0.127 mg F/cu m) in the general atmosphere of the operating area before and after hopper filling operations. A sample taken in the breathing zone of an operator who was filling the hopper with sodium fluoride showed 8.89 mg /cu m as HF (about 8.46 mg F/cu m). This operation was completed in 5 minutes, and never occurred more than once a day. Details of the ventilation system were not reported.

(f) Mining and Cryolite Manufacture

Roholm [18] in his classic monograph discussed the processing of cryolite ore mined in Greenland. Operations included crushing and grinding, drying, conveying, and packing. Dust from rooms in which the various processes took place was exhaust ventilated through filters. The collected dust was used in manufacturing. Attempts had been made to enclose and ventilate machines and drying stoves, but details were not given. Masks were supplied to workers. Total dust content of the air ranged from 22 to 994 mg/cu m. Since this dust was about 97% cryolite these figures correspond to approximately 12-536 mg/cu m as fluoride.

Fluorspar mining was the subject of a study by de Villiers et al [225] reported in 1971. Sixty-seven samples at 27 locations underground showed a mean of 0.44 mg F/cu m, while 7 samples taken above ground showed a mean of 0.17 mg F/cu m. Control measures, if any, were not reported.

(g) Pottery Manufacture

During a health survey of the pottery industry, Luxon [226] determined the nature and amount of fluoride compounds emitted during the firing of pottery. Etching of window panes had indicated the presence of such compounds. He found that the compounds were particulate and were probably composed of ammonium fluosilicates. Breathing level workroom air samples showed concentrations up to 3.5 mg F/cu m. Exhaust ventilation fitted to the entrances of the kilns was found to afford effective control, but specifications of the exhaust system were not reported.

(h) Steel Manufacture

Although steel manufacturing uses the largest quantity of fluorspar as a metallurgical fluxing agent, [12,15] only a limited number of studies have been made of fluoride exposures resulting from this process. Markuson, [227] in 1947, reported on the use of sodium fluoride in the manufacture of rimmed steel. Fluoride fumes were generated when 4-ounce paper bags of sodium fluoride were added to the mold while molten steel was being poured. Samples were taken for total fluoride, including both gaseous and particulate fluorides. Samples of crane cab air showed 3.02 mg F/cu m. The highest concentration found on the pouring platform was 1.3 mg F/cu m. On a time-weighted average basis, exposures were 0.85 mg F/cu m and 0.365 mg F/cu m for an 8-hour day, respectively. Crane cabs were later equipped with filtered ventilation, but the author doubted their efficacy because of the extensive and frequent maintenance required. Local exhaust ventilation was recommended, but details were not given.

In 1948, the US Public Health Service [95] conducted a survey of potential health hazards associated with use of sodium fluoride at 4 steel plants. Differential sampling indicated the absence of gaseous fluorides, while X-ray analysis showed that fluoride fumes were composed of the parent material, sodium fluoride. Ventilation existed only as natural ventilation modified by convection currents from the heat sources in the plants, all of which had monitor roofs. Crane operator exposures ranged from 1.0 to 51.2 mg NaF/cu m (about 0.45-23.1 mg F/cu m) while pouring platform exposures ranged from 0.2 to 21.9 mg NaF/cu m (about 0.09-9.9 mg F/cu m) with a median of 1.6 mg NaF/cu m (about 0.72 mg F/cu m). Exposures were calculated from the median values of the daily samples and from an estimate of the average hours per day spent in each exposure area. Time-weighted exposures ranged from 0.4 mg NaF/cu m (about 0.18 mg F/cu m) for ladlemen, pourers, and platform men, to 2.2 mg NaF/cu m (about 1.0 mg F/cu m) for ladle cranemen. Despite the lack of engineering controls, extensive physical examinations failed to disclose evidence of physiological damage by the sodium fluoride. Therefore, no controls were recommended except positive pressure ventilation for crane cabs, with filtering of intake air.

V. DEVELOPMENT OF STANDARD

Basis for Previous Standards

In 1945, Cook [228] compiled a list of standards and recommendations for maximum allowable concentrations of industrial atmospheric contaminants. The following 2 different maximum allowable concentrations were cited for fluoride dusts: 1 mg F/cu m, a nonofficial concentration in use as a guide by the Massachusetts Department of Labor and Industries Division of Occupational Hygiene, September 1945, and 2 mg F/cu m, an official concentration listed in Rules and Regulations for the Prevention and Control of Occupational Diseases by the Oregon State Board of Health, August 1945. No supporting documentation was given for these time-weighted averages for a 40-hour workweek.

The American Conference of Governmental Industrial Hygienists [229] in 1947 adopted a Maximum Allowable Concentration (MAC) for fluorides of 2.5 mg F/cu m. It was not stated if this MAC was intended as a ceiling value or as a time-weighted average. The April 1948 meeting of this same organization [230] adopted 2.5 mg F/cu m as a threshold limit value (TLV) which has remained unchanged since. This TLV for fluorides is a time-weighted average.

Data presented in the 1966 Documentation of the Threshold Limit Values for Substances in the Workroom Air [231] did not conclusively support this TLV. The report by Ronzani [232] related to the irritant effects produced when animals were exposed to 3 ppm hydrogen fluoride and was not concerned with human exposure to fluorides. The reports by Roholm, [18,233] Largent, [33] and notes taken by HE Stokinger (written communication, 1974) on Irwin's presentation at a 1954 symposium, were also considered when the ACGIH Documentation was published. Largent [33] concluded that fluorides might be stored in human tissues over periods of years when as little as 3 mg of NaF was ingested daily, but he had only indirect evidence based on 4 subjects. Data by Irwin as cited in the Documentation [231] supported the ACGIH TLV of 2.5 mg F/cu m. Irwin reported no significant bone changes in workers when fluoride air levels were below 4-5 ppm (3-4 mg F/cu m). Gaseous and particulate fluoride concentrations were approximately equal. Slight bone changes were recorded after 20 years among cryolite workers exposed at air levels of 5 ppm. According to Irwin (HE Stokinger, written communication, 1974) 29 of 265 workers excreting an average of 9.6 mg F/day in the urine showed a marked increase in density of the pelvic bone while only 2 of 402 employees excreting an average of 6.3 mg F/day in the urine showed marked increase in bone density. These notes did not further document the relationship between air concentrations and bone changes.

The 1971 Documentation [234] concluded that "the limit of 2.5 mg F/cu m is sufficiently low to prevent irritative effects and to protect against disabling bone changes." The Documentation reviewed studies concerning workroom air concentration and ingestion and excretion by Roholm, [18] Largent, [34] Williams, [209] Markuson, [227] Elkins, [235] Collings et al, [48] Irwin (unpublished work referred to above), Derryberry et al, [97] Agate et al, [26] Machle and Largent, [31] Largent, [33] Hodge, [236] Princi, [237] and Waldbott. [23]

In 1963, the Second International Symposium on Permissible Limits for Air of Workplaces, as reported by Truhaut, [238] adopted a MAC of 2.5 mg

F/cu m which was considered as a time-weighted average. No documentation was given to support the MAC.

The American Industrial Hygiene Association's Hygienic Guide on Fluoride-Bearing Dusts and Fumes (Inorganic), [211] published in 1965, recommended a MAC (8 hours) of 2.5 mg F/cu m. Data from the ACGIH Documentation of Threshold Limit Values were used as the basis for the value.

The United States of America Standards Institute [239] (now referred to as the American National Standards Institute) listed an acceptable 8-hour time-weighted average concentration for fluoride in dusts as 2.5 mg F/cu m (Z37.28-1966). This standard was supported by observations on animals exposed to HF and fluoride by Stokinger [240] and a review of Heyroth [241]; but it was pointed out that there was insufficient evidence and that further study was necessary.

The Department of Environmental Resources in Pennsylvania [242] adopted the ACGIH TLV of 2.5 mg F/cu m and it also set a short-term limit for fluoride of 10 mg F/cu m for 30 minutes. In Pennsylvania's Short Term Limits for Exposure to Airborne Contaminants: A Documentation, [243] reports by the American Industrial Hygiene Association (Hygienic Guide Series--1956) and Heyroth [241] were considered.

According to a Czech source, [244] the Soviet Union, Poland, Hungary, Czechoslovakia, and East Germany recommended an 8-hour maximum allowable concentration for fluoride of 1.0 mg F/cu m, but West Germany recommended 2.5 mg F/cu m.

In the Soviet Union, Smelyanskiy and Ulanova [245] listed a mandatory maximum permissible concentration of 0.001 mg F/liter (1.0 mg F/cu m) in the workroom air which was established by the Main State Health Inspector of the USSR, January 10, 1959, No. 279-59. Maximum permissible concentrations were considered concentrations which could only be exceeded if workers were in an industrial area for an unspecified brief period.

The Czechoslovak Committee of MAC [244] suggested a mean MAC value of 1 mg F/cu m for all soluble fluorides. Total fluoride intake (inhalation plus food and water) were considered. The committee concluded that 8 hours of work with a mean concentration of 1 mg F/cu m would result in a daily urinary excretion of 5.5 mg F, taking into account 1 mg F/day of food and water intake. This level of urinary excretion was stated to be a "critical value," ie, F would be accumulated in the skeleton but without the appearance of osteosclerosis. A peak MAC of 2 mg F/cu m was also suggested. This peak could occur for a short time as long as the mean was not exceeded. These mean and peak values were based on studies by Roholm [18] and Largent. [33,34]

The present federal standard for fluoride is an 8-hour time-weighted average of 2.5 mg/cu m (29 CFR 1910.1000), published in the *Federal Register* 39:23541, June 27, 1974, as amended. The standard is based on the United States of America Standard Z37.28-1966. [239]

Basis for Recommended Environmental Standard and Biological Monitoring

(a) Occupational Environmental Standard

The occupational exposure limit is intended to prevent deleterious effects of skeletal fluorosis (osteosclerosis or increased bone density due to excessive absorption and retention of fluorides). Severe osteofluorosis was found by Moller and Gudjonsson [17] and Roholm [18] among cryolite workers who exhibited calcification of ligaments and pronounced bone density increases in the pelvis and spinal column. The most severe cases exhibited moderate-to-marked restriction of spinal motility. [18] Kaltreider et al [102] found skeletal fluorosis in 76 of 79 aluminum potroom workers who had worked at their jobs for more than 5 years. Twenty-six or 33% of the workers classified as having marked or advanced fluorosis showed restricted movements of the spine.

Three studies [18,93,94] reported pulmonary fibrotic changes on radiological examination. No attempt was made to correlate clinical symptoms with pulmonary function tests. Atmospheric F concentrations within the industries studied ranged from 15 to 20 mg/cu m, 2-3 mg/cu m, and 0.143-6.37 mg/cu m, respectively. The results of one study [18] indicated that pulmonary fibrosis caused by exposure to cryolite dust tended to diminish in workers who had been away from the factory for at least 3 years. Since the cryolite dust contained quartz (1-5%) as well as fluorides, the study did not conclude that the fluoride component of the cryolite was responsible for the fibrotic changes. X-ray findings reported in other epidemiological studies did not indicate the presence of pulmonary fibrosis. [38,95,96,102]

The increased incidence of bronchial asthma reported in 2 Norwegian aluminum factories by Evang [84] and Hjort [91] is not substantiated by other studies. Neither study was able to conclude that fluoride was the causal agent.

A study relevant to the development of an environmental standard is the one by Derryberry et al. [97] They provided comprehensive environmental and urinary fluoride excretion data on each worker included in the survey and demonstrated correlation with clinical and radiological findings. Environmental fluoride levels were evaluated from approximately 750 atmospheric samples over a period of approximately 25 years. An average daily fluoride exposure for each job was established and from these data a weighted atmospheric exposure was calculated for the period of employment of each worker. The range of individual average weighted exposure to F was 0.50-8.32 mg/cu m, with 1.78-7.73 mg/cu m being associated with a questionable or marginal increase in bone density. The difference in averages between the increased bone density group (average exposure 3.38 mg F/cu m) and the remainder of the exposed group (average exposure 2.62 mg F/cu m) is significant by both T test ($t = -2.75$, $p = 0.0045$) and rank test ($z = -2.2$, $p = 0.014$).

While the work of Derryberry et al [97] is of value in developing an environmental standard, inherent weaknesses found within the study limit the extent to which the findings of the authors can be directly applied to the determination of an environmental limit. The test population utilized in the study was very small. This placed limitations on the statistical significance of the findings. In the study, 17 out of 74 workers exposed to various concentrations of F were diagnosed as showing bone density changes of minimal or questionable degree. The authors stated that these diagnoses were made by a radiologist who had prior knowledge that the tested individuals had potential fluoride exposures and who did not feel that the radiographs showed sufficient increase in bone density to be recognized as such in routine radiological practice.

The Derryberry et al [97] study is of value, however, in that it is a comprehensive study and it does indicate that a threshold for minimal increases in bone density exists. Considering all aspects of the study, this threshold is best represented by the average exposure of 3.38 mg F/cu m.

It is virtually impossible to correlate medical findings with exposure levels from other available studies. [26,93,102] These studies include the following shortcomings: exposure levels were taken only at the time of the study and may not represent long-term exposure; insufficient individual or group data were given to correlate exposure with medical findings; and workers were often selected for the study. Tourangeau [93] observed 2 cases of generalized and 3 cases of localized osteosclerosis (degree not given) in a selected group of 10 out of 104 aluminum potroom workers. Unfortunately, the fluoride exposure of this selected group was not provided. Kaltreider et al [102] found 76 cases of osteosclerosis in a group of 79 aluminum foundry workers with a weighted average 8-hour atmospheric exposure of 2.4-6.0 mg F/cu m. Of the affected 76 cases, 46 had slight osteosclerosis, 4 moderate osteosclerosis, and 26 marked osteosclerosis with restrictive motion of the spine. Unfortunately, exposure levels were measured only at the time of the study. Agate and co-workers [26] found abnormal X-ray appearances not considered to be necessarily synonymous with skeletal fluorosis in 48 of 189 furnace room workers selected for study. These abnormal findings ranged from skeletal fluorosis to minor bone irregularities. A breakdown in the subject selection method employed in the study forced the authors to resort to conducting clinical examinations on volunteers from the study population. Exposure levels were given as between 0.14 and 3.43 mg F/cu m within the furnace areas.

Support of the present environmental limit by the 1971 Documentation [231] and the United States of America Standards Institute (now referred to as ANSI) [236] as well as the evidence provided by Derryberry et al [97] indicate that a time weighted average exposure limit of 2.5 mg F/cu m will prevent the occurrence of deleterious health effects resulting from F deposition.

It is recognized that many workers handle small amounts of fluoride or work in situations where, regardless of the amount used, there is only negligible contact with the substance. Under these conditions, it should not be necessary to comply with many of the provisions of this recommended standard, which has been prepared primarily to protect worker health under more hazardous circumstances. Concern for worker health requires that protective measures be instituted below the enforceable limit to ensure that exposures stay below that limit. For these reasons, "exposure to fluoride" has been defined as exposure above half the environmental limit, thereby delineating those work situations which do not require the expenditure of health resources, of environmental and medical monitoring, and associated recordkeeping. Half the environmental limit has been chosen on the basis of professional judgment rather than on quantitative data that delineate nonhazardous areas in which a hazard may exist. However, because of nonrespiratory hazards such as those resulting from skin irritation or eye contact, it is recommended that appropriate work practices and protective measures be required regardless of the air concentration.

(b) Biological Monitoring

Since the recovery of fluorides from physiological fluids can be related to environmental exposures and/or osteofluorosis, a valuable means of monitoring employee health is provided. Many authors [28,31,32,246,247] have demonstrated that under exposure conditions leading to F retention in bone, urinary F excretion reflects the amount of fluorides absorbed. Largent and Machle [31] demonstrated in their balance studies that a straight-line correlation existed not only between absorption and urinary excretion but also between absorption and retention of fluorides. Separate studies [38,46,48,49] demonstrated rapid absorption and urinary excretion of fluorides when respirable inorganic fluoride dust or fumes were inhaled.

(1) Postshift Urinary F Biological Level

Collings et al [46,48] and Largent [28] demonstrated a rapid rise in urinary fluoride output within 2 hours of exposure which remained at high levels for 2-4 hours after cessation of exposure. Thus, end-of-shift urine samples, as recommended by NIOSH, will reflect exposure conditions occurring during the working day. This is especially desirable since it supplies data relevant to employees' working practices and engineering control measures.

Although numerous studies have demonstrated [61,62,70-72,96,97] that slightly increased bone density is not synonymous with disability or a reduced functional capability, the urinary excretion limits should be established below that point at which skeletal fluorosis begins to occur rather than at that point where the first signs of disability or functional impairment are approached.

The study by Derryberry et al [97] provides long-term individual worker postshift urinary excretion data which can be compared to reported cases of suspected increased bone density. In this study, average postshift urinary fluoride levels for the examined group of workers were determined from 2,850 urine samples, averaging 38 specimens per man. Reference to Table III-12 demonstrates that as the average urinary fluoride level excretion increased the percentage of suspected osteosclerosis increased gradually until the excretion range of 8-8.9 mg F/liter was reached, at which point 3 of 5 members of the group in that range revealed an increase in bone density.

A definite though asymptomatic increase in bone density was found by Kaltreider et al [102] in 76 of 79 aluminum potroom workers. The results of spot urine samples collected during the shift at unspecified times were given only as the averages for the 3 occupational groups of pot tender, tapper-carbon changer, and craneman and revealed urinary F concentrations of 8.7, 9.8, and 9.6 mg/liter, respectively (no correction for specific gravity). In a second study [102] in which the fluoride exposure was reported to be "quite modest" in comparison with exposures of the earlier study, there were no cases of increased bone density among 231 potroom workers excreting an average postshift urinary F concentration of 3.0 mg/liter corrected to 1.024 specific gravity.

The above results [97,102] indicate that an average of postshift urinary F concentrations less than 8.0 mg/liter will lead to few cases of definite osteofluorosis. It is concluded that a postshift biological level of 7.0 mg/liter calculated as an average will provide an acceptable margin of safety.

(2) Preshift Urinary F Biological Level

Upon cessation of F exposure, the initial rapid rise of urinary F concentration is followed by a return to stable and relatively low levels of F excretion within 24 hours. [28,36,46] Urinary F concentrations approached preshift values within 1-6 days. [27,28,36,38,46,48] These studies reveal that the time required for the preshift sample to stablilize is quantitatively related to the urinary F concentration in the postshift sample, and that urinary F analyses conducted preshift after a period of 1 day or more without exposure will provide a stable baseline value indicative of a worker's residual F retention (body burden).

The relationship of the concentration of fluoride in the preshift urine sample to the onset of osteofluorosis has not been precisely demonstrated, but a limited number of industrial exposure studies [101,102, H R Henderson, written communication, September 1974] as well as one nonindustrial exposure study enable some inferences to be drawn regarding the relationship of osteofluorosis to the preshift urine sample.

Preshift urinary F excretions were analyzed in 25 chemical workers exposed to HF or particulate fluorides in concentrations ranging from 0.077 to 10.0 ppm as HF and 0.1-0.49 mg/cu m as particulate F. [101] Preshift specimens, which were collected after the workmen had been away from the plant on their days off, ranged from 0.33 to 4.48 mg F/liter. Corresponding levels for a control group of 10 office workers were 0.5-1.88 mg F/liter.

Additional data on environmental and urinary F levels of the same plant population were made available by the company (H R Henderson, written communication, September, 1974). Over a 10-year period periodic urinary F levels of 13 HF workers revealed the average preshift levels for the workmen ranged from 2.0 to 5.7 mg/liter. One of 4 workers with high preshift urinary F concentrations who had negative X-rays for osteosclerosis when examined 2 years earlier demonstrated minimal osteosclerosis upon a follow-up examination. His average preshift urinary F level was 5.3 mg/liter, ranging from 2.6 to 16.3 mg F/liter.

No osteofluorosis was found in a group of 147 potroom workers excreting a preshift average of 1.4 mg F/liter of urine ranging from 0 to 11.9 mg F/liter. [102] Urine samples were collected after 48 hours off work and the values were corrected to a specific gravity of 1.024.

Stevenson and Watson [71] reviewed medical records of patients residing primarily in Texas and Oklahoma where water supplies contained up to 8 ppm of fluoride. A diagnosis of fluoride osteosclerosis was made in 23 patients living in communities whose water supplies contained 4-8 ppm of F. It was concluded that fluoride osteosclerosis did not develop in patients drinking water with an F content of less than 4 ppm.

The preceding data [102, 101, H R Henderson, written communication, September 1974] suggest that preshift urinary values up to 5.3 mg F/liter were not associated with osteofluorosis. While the findings of the nonindustrial exposure study [71] cannot be strictly applied to the determination of a preshift level, the results of the study indicate that a preshift level below 5.3 is needed. It is concluded that a preshift level of 4 mg/liter will provide adequate worker protection. The validity of the value, as a preshift level, should be tested and adjusted in the future as more information is gained.

(c) Urine Specific Gravity

Urinary fluoride values corrected to a standard specific gravity provide better correlation with F intake than uncorrected values, as demonstrated by Buchwald. [39] Elkins et al [40] concluded that the true mean value for the specific gravity of 35,506 samples analyzed in their lab was 1.022. They concluded that since the value most widely used for specific gravity correction in the United States is 1.024, it should continue as a standard reference to enable data comparisons among different investigators.

(d) Renal Disease

The importance of urinary excretion in minimizing fluoride retention in bone is illustrated by experience with patients with renal disease. Evidence of interference with normal clearance of fluorides and increased bone deposition caused by kidney dysfunction rather than induced by fluoride has been presented by several authors. [28,61,74,75] Emphasis should, therefore, be put on eliciting a history of renal disease and providing a means of disease detection during physical examinations.

(e) Radiological Examination

The onset of increased bone density is most apparent in the lumbar spine and pelvis. [97] Since changes in the osseous system may be the only evidence of increased absorption and retention of fluorides, a periodic X-ray of the pelvis may be valuable in cases where urinary F levels have been found to be high. It should be noted that the first changes produced by fluoride absorption and retention are difficult to recognize without prior knowledge that the individual had a fluoride exposure. Radiological examination of the pelvis can result in irradiation of the gonads and embryos. [248] This may lead to deviation from normal mutation rates and may produce developmental abnormalities in the human embryo. Due to the difficulty of ensuring adequate protection for female gonads and for embryos, it is recommended that radiological examination of the female pelvis not be conducted. Since male gonads can be protected adequately during pelvic X-ray, preplacement male pelvic exams should be considered to obtain baseline radiologic information.

VI. WORK PRACTICES

Prevention of occupational diseases from fluorides involves the prevention of inhalation or ingestion of fluorides. Inhalation of fluorides is best prevented by controlling fluorides at their sources of emission by means of enclosure of processes or by local exhaust ventilation. Ingestion of fluorides is prevented by means of good housekeeping and personal hygiene procedures. Properly designed and maintained ventilation systems will prevent dispersal of fluorides in the workroom atmosphere, not only preventing their inhalation, but preventing their accumulation on surfaces, thus minimizing maintenance problems and the chance for redispersal during cleanup with attendant inhalation and ingestion hazards. Good ventilation practices, such as those outlined in a current edition [249] of Industrial Ventilation--A Manual of Recommended Practice, published by the American Conference of Governmental Industrial Hygienists, should be followed. Where exhaust ventilation is used, an adequate source of make-up air should always be provided, with conditioning for temperature and humidity as required.

Mechanization and enclosure of processes offer additional methods of engineering control which may be effectively used under some circumstances.

Many of the industrial processes emitting fluorides produce simultaneous exposure to gaseous as well as particulate fluorides. If respiratory protection is required, it must be of the type to provide protection against both. In 1971, the AIHA and ACGIH Joint Committee on Respirators [250] published a list of sorbents for contaminants listed in the ACGIH 1970 TLV table. For fluoride exposure, a combination of a toxic dust or fume filter and a soda-lime activated carbon cartridge was recommended. Manufacturers' equivalents of this combination should be provided when cartridge or canister respiratory protection is used.

Soluble and acidic fluorides can cause skin, eye, and respiratory irritation. [212,251] Adequate protective clothing and eye and face protection must be utilized when exposure to these compounds occurs. Eyewash fountains and showers should be available for immediate care. Eyes affected by fluorides should be immediately flushed with copious amounts of water, forcibly holding open the lids, if necessary.

Fluorides which form acidic solutions when mixed with water should be stored away from water. [212] Many fluorides produce hydrogen fluoride in contact with acids. Hydrogen fluoride is extremely corrosive and hazardous. Unwarranted contact of fluorides with acids should be avoided.

Fluorides are not fire or explosion hazards. However, acidic solutions of fluorides may generate hydrogen in contact with metals. Water may be used on fires involving fluorides.

Labeling of fluoride fluxes and use of ventilation for welding with fluoride coated rods are required by 29 CFR 1910.252. Close observance of the specified distances between ventilation intakes and source of welding or brazing fumes is important for effective control.

Workers should exercise care in handling of bags and barrels of fluorides to prevent container rupture and spillage. [252] Spills are preferably cleaned up with an industrial vacuum cleaner, but wet methods may be of value in instances where such procedures will not increase exposure or pollute sewers and drains above environmental limits.

Good personal hygiene involves washing thoroughly before eating or drinking, and after handling fluorides. [211,212,252] Contaminated impervious protective clothing should be washed off before removal. Food or beverages should not be consumed in work areas. [253]

A number of states have promulgated standards for fluoride content of community air and vegetation. (See Chapter VII.) It is therefore imperative that any discharge of fluorides to the environment comply with all applicable regulations. This may require air cleaning devices on the discharge ducts of ventilation systems used for control of fluorides in workroom atmospheres, and close control of effluents discharged to streams, onto the ground, or to sewers and drains.

VII. COMPATIBILITY WITH EMISSION STANDARDS

There is no federal community air standard for fluorides. A number of states have promulgated standards for control of emission of fluorides. These standards were not established on the basis of protection of human health but on the basis of damage to livestock and vegetation. The levels established are well below those found to adversely affect human health. For example, Wyoming has adopted a fluoride regulation and standard which states [254] that fluorides measured as HF shall not exceed 0.80 μg/cu m or 1.0 ppb (part per billion) as a 24-hour average. Pennsylvania's standard [255] sets the limit for water soluble fluorides as 5 μg HF/cu m averaged over 24 hours. Montana's standard is 1 ppb as HF, and New York's is 3 ppb averaged over a 24-hour period. [256] Washington [257] has established 2 standards, one for forage and one for community air. Fluoride concentration of forage by dry weight is not to exceed 40 ppm F averaged over 12 consecutive months, 60 ppm averaged over 2 months, and 80 ppm more than once in any 2 consecutive months. Gaseous fluorides in community air are not to exceed an average of 3.7 μg HF/cu m for 12 hours, 2.9 μg/cu m for 24 hours, 1.7 μg/cu m over 7 days, 0.84 μg/cu m over 30 days, and 0.5 μg/cu m over the period March 1 through October 31 of any year.

Standards for fluorides in effluent from aluminum smelting operations have been proposed by the Environmental Protection Agency in the <u>Federal Register</u> 38(230):33170-83, dated 30 November 1973. The proposed 40 CFR 421 specifies various concentrations of fluoride in effluent ranging from 0.05 kg/1,000 kg of product a day to 2.0 kg/1,000 kg of product a day, depending on the process and the technology used. These standards apparently are not based on any human health effects, but on the best practicable or best available technology. For this reason they are not directly comparable with the recommended environmental limit of Chapter I.

VIII. REFERENCES

1. Committee on Biologic Effects of Atmospheric Pollutants: Fluorides. Washington, DC, National Academy of Sciences, National Research Council, Division of Medical Sciences, 1971

2. Fluorides and Human Health. World Health Organization Monograph Series No. 59. Geneva, World Health Organization, 1970, pp 255-62

3. Hodge HC, Smith FA: Fluorides and man. Ann Rev Pharmacol 8:395-408, 1968

4. Hodge HC, Smith FA: Biological Properties of Inorganic Fluoride, in Simons JH (ed): Fluorine Chemistry. New York, Academic Press, 1965, vol IV

5. Handbook of Experimental Pharmacology Heffter-Heubner-new series, in Smith F (ed): Pharmacology of fluorides. New York, Springer-Verlag, vol XX, part I (1966), part II (1970)

6. Cholak J: Current information on the quantities of fluoride found in air, food, and water. Arch Ind Health 21:312-15, 1960

7. Crossley HE: Fluorine in coal--III. The manner of occurrence of fluorine in coals. J Soc Chem Ind 63:289-92, 1944

8. Abernethy RF, Gibson FH: Method for Determination of Fluorine in Coal, report RI 7054. US Dept of the Interior, Bureau of Mines, 1967

9. MacMillan RT: Fluorine, in Mineral Facts and Problems, bulletin 650. US Dept of the Interior, Bureau of Mines, 1970

10. Grogan RM: Fluorspar and cryolite, in Industrial Minerals and Rocks (Nonmetallics Other than Fuels), ed 3 rev. New York, American Institute of Mining, Metallurgical and Petroleum Engineers, 1960, pp 363-82

11. Readling CL: Fluorspar and cryolite, in Minerals Yearbook 1969--Vol I-II. Metals, Minerals and Fuels. US Dept of the Interior, Bureau of Mines, 1971, pp 505-13

12. Wood HB: Fluorspar and cryolite, in Minerals Yearbook, vol 1. Metals, Minerals, and Fuels. US Dept of the Interior, Bureau of Mines, 1973, pp 517-30

13. Lewis RW, Stowasser WF: Phosphate rock, in Minerals Yearbook 1971, vol 1: Metals, Minerals, and Fuels. US Dept of the Interior, Bureau of Mines, 1973, pp 971-83

14. Barber JC, Farr TD: Fluoride recovery from phosphorus production. Chem Eng Prog 66:56-62, 1970

15. Davis HW, Trought ME: Fluorspar and cryolite, in Keiser HD (ed): Minerals Yearbook--Review of the 1940. US Dept of the Interior, Bureau of Mines, 1941, pp 1295-1312

16. Tarbutton G, Farr TD, Jones TM, Lewis HT Jr: Recovery of by-product fluorine. Ind Eng Chem 50:1525-28, 1958

17. Moller PF, Gudjonsson SV: Massive fluorosis of bones and ligaments. Acta Radiol 13:269-94, 1932

18. Roholm K: Fluorine intoxication--A clinical-hygienic study with a review of the literature and some experimental investigations. London, HK Lewis & Co Ltd, 1937

19. Cholak J: Fluorides: A critical review--I. The occurrence of fluoride in air, food, and water. J Occup Med 1:501-11, 1959

20. Hodge HC, Smith FA: Fluorides, in Lee DH, Minard D (eds): Metallic Contaminants and Human Health. New York, Academic Press, 1972, pp 163-87 (Fogarty International Center Proceedings No. 9)

21. Machle W, Scott EW, Treon J: Normal urinary fluorine excretion and the fluorine content of food and water. Am J Hyg 29:139-45, 1939

22. Marier JR, Rose D: The fluoride content of some foods and beverages--A brief survey using a modified Zr-SPADNS Method. J Food Sci 31:941-46, 1966

23. Waldbott GL: The physiologic and hygienic aspects of the absorption of inorganic fluorides. Arch Environ Health 2:155-67, 1961

24. McClure FJ: Fluorine in foods. Public Health Rep 64:1061-74, 1949

25. Armstrong WD, Knowlton M: Fluorine derived from food. J Dent Res 21:326, 1942

26. Agate JN, Bell GH, Boddie GF, Bowler RG, Buckell M, Cheeseman EA, Douglas THJ, Druett HA, Garrad J, Hunter D, Perry KMA, Richardson JD, Weir JB: Industrial Fluorosis--A Study of the Hazard to Man and Animals near Fort William, Scotland. Medical Research Council Memorandum no. 22. London, His Majesty's Stationery Office, 1949

27. Machle W, Scott EW, Largent EJ: The absorption and excretion of fluorides--Part I. The normal fluoride balance. J Ind Hyg Toxicol 24:199-204, 1942

28. Largent EJ: Excretion of fluoride, in Muhler JC, Hine MK (eds): Fluorine and Dental Health--The Pharmacology and Toxicology of Fluorine. Bloomington, Indiana University Press, 1959, pp 128-56

29. Spencer H, Lewin I, Wistrowski E, Samachson J: Fluoride metabolism in man. Am J Med 49:807-13, 1970

30. McClure FJ, Kinser CA: Fluoride domestic waters and systemic effects--II. Fluorine content of urine in relation to fluorine in drinking water. Public Health Rep 59:1575-91, 1944

31. Machle W, Largent EJ: The absorption and excretion of fluoride--II. The metabolism at high levels of intake. J Ind Hyg Toxicol 25:112-23, 1943

32. Zipkin I, Likins RC, McClure FJ, Steere AC: Urinary fluoride levels associated with use of fluoridated waters. Public Health Rep 71:767-72, 1956

33. Largent EJ: Rates of elimination of fluoride stored in the tissues of man. Arch Ind Hyg Occup Med 6:37-42, 1952

34. Largent EJ: Fluorosis--The Health Aspects of Fluorine Compounds. Columbus, Ohio State Univ Press, 1961, pp 11, 12, 21, 33, 43, 44, 48, 122, 123, 125, 126

35. Likins RC, McClure FJ, Steere AC: Urinary excretion of fluoride following defluoridation of a water supply. Public Health Rep 71:217-20, 1956

36. Zipkin I, Leone NC: Rate of urinary fluoride output in normal adults. Am J Public Health 47:848-51, 1957

37. Largent EJ, Ferneau IF: Exposure to fluorides in magnesium founding. J Ind Hyg Toxicol 26:113-16, 1944

38. Rye WA: Fluorides and phosphates--Clinical observations of employees in phosphate operation, in Proceedings of the 13th International Congress on Occupational Health, July 25-29, 1960, pp 361-64, 1961

39. Buchwald H: The expression of urine analysis results--Observations on the use of a specific gravity correction. Ann Occup Hyg 7:125-36, 1964

40. Elkins HB, Pagnotto LD, Smith HL: Concentration adjustments in urinalysis. Am Ind Hyg Assoc J 35:559-65, 1974

41. Elkins HB, Pagnotto LD: The specific gravity adjustment in urinalysis. Arch Environ Health 18:996-1001, 1969

42. Crosby ND, Shepherd PA: Studies on patterns of fluid intake--Water balance and fluoride retention. Med J Aust 44:341-46, 1957

43. McClure FJ, Mitchell HH, Hamilton TS, Kinser CA: Balances of fluorine ingested from various sources in food and water by five young men. J Ind Hyg Toxicol 27:159-70, 1945

44. Domzalska E, Lassocinska A: Content of fluorides in human urine, saliva and blood depending on the age of the subject. Polish Med J 9:228-34, 1970

45. Carlson CH, Armstrong WD, Singer L: Distribution and excretion of radiofluoride in the human. Proc Soc Exp Biol Med 104:235-39, 1960

46. Collings GH, Fleming RBL, May R: Absorption and excretion of inhaled fluorides. Arch Ind Hyg Occup Med 6:368-73, 1952.

47. Clinical Norms--A Handbook of Biomedical Data. Butler, NJ, Francis Roberts Agency Inc, 1969, p 75

48. Collings GH Jr, Fleming RBL, May R, Bianconi WO: Absorption and excretion of inhaled fluorides--Further observations. Arch Ind Hyg Occup Med 6:368-73, 1952

49. Smith LK: Urinary fluoride excretion in electric arc welders exposed to low hydrogen electrode fumes. Ann Occup Hyg 11:203-07, 1968

50. Krechniak J: Fluoride hazards among welders. Fluoride Q Rep 2:13-24, 1969

51. Mangold CA, Beckett RR: Combined occupational exposure of silver brazers to cadmium oxide, nitrogen dioxide and fluorides at a Naval shipyard. Am Ind Hyg Assoc J 32:115-18, 1971

52. Smith FA, Gardner DE, Hodge HC: Investigations on the metabolism of fluoride--II. Fluoride content of blood and urine as a function of the fluorine in drinking water. J Dent Res 29:596-600, 1950

53. Taves DR: Normal human serum fluoride concentrations. Nature 211:192-93, 1966

54. Hall LL, Smith FA, de Lopez OH, Gardner DE: Direct potentiometric determination of total ionic fluoride in biological fluids. Clin Chem 18:1455-58, 1972

55. Gardner DE, Smith FA, Hodge HC, Overton DE, Feltman R: The fluoride content of placental tissue as related to the fluoride content of drinking water. Science 115:208-09, 1952

56. Feltman R, Kosel G: Prenatal and postnatal ingestion of fluorides--Fourteen years of investigation--Final report. J Dent Med 16:190-99, 1961

57. Armstrong WD, Singer L, Makowski EL: Placental transfer of fluoride and calcium. Am J Obstet Gynecol 107:432-34, 1970

58. Lidbeck WL, Hill IB, Beeman JA: Acute sodium fluoride poisoning. JAMA 121:826-27, 1943

59. Sauerbrunn BJL, Ryan CM, Shaw JF: Chronic fluoride intoxication with fluorotic radiculomyelopathy. Ann Intern Med 63:1074-78, 1965

60. Zipkin I, McClure FJ, Leone NC, Lee WA: Fluoride deposition in human bones after prolonged ingestion of fluoride in drinking water. Public Health Rep 73:732-39, 1958

61. Call RA, Greenwood DA, LeCheminant WH, Shupe JL, Nielsen HM, Olson LE, Lamborn RE, Mangelson FL, Davis RV: Histological and chemical studies in man on effects of fluoride. Public Health Rep 80:529-38, 1965

62. McClure FJ, McCann HG, Leone NC: Excessive fluoride in water and bone chemistry--Comparison of two cases. Public Health Rep 73:741-46, 1958

63. Glock GE, Lowater F, Murray MM: The retention and elimination of fluorine in bones. Biochem J 35:1235-39, 1941

64. Boissevain CH, Drea WF: Spectroscopic determination of fluorine in bones, teeth, and other organs, in relation to fluorine in drinking water. J Dent Res 13:495-500, 1933

65. Kilborn LG, Outerbridge TS, Lei HP: Fluorosis with report of an advanced case. Can Med Assoc J 62:135-41, 1950

66. Goldman SM, Sievers ML, Templin DW: Radiculomyopathy in a southwestern Indian due to skeletal fluorosis. Ariz Med 28:675-77, 1971

67. Ast DB, Chase HC: The Newburgh-Kingston caries fluorine study--IV. Dental findings after six years of water fluoridation. Oral Surg 6:114-23, 1953

68. Schlesinger ER, Overton DE, Chase HC, Cantwell KT: Newburgh-Kingston caries-fluorine study--XIII. Pediatric findings after ten years. J Am Dent Assoc 52:296-306, 1956

69. Bernstein DS, Sadowsky N, Hegsted DM, Guri CD, Stare FJ: Prevalence of osteoporosis in high- and low-fluoride areas in North Dakota. JAMA 198:499-504, 1966

70. Leone NC, Stevenson CA, Hilbish TF, Sosman MC: A roentgenologic study of a human population exposed to high-fluoride domestic water--A ten-year study. Am J Roentgenol Radium Ther Nucl Med 74:874-85, 1955

71. Stevenson CA, Watson AR: Fluoride osteosclerosis. Am J Roentgenol Radium Ther Nucl Med 78:13-18, 1957

72. Alffram PA, Hernborg J, Nilsson BER: The influence of a high fluoride content in the drinking water on the bone mineral mass in man. Acta Orthop Scand 40:137-42, 1969

73. Hodge HC: Fluoride, in Comar CL, Bronner F (eds): Mineral Metabolism--The Elements. New York, Academic Press, 1964, vol 2, part A, pp 573-602

74. Linsman JF, McMurray CA: Fluoride osteosclerosis from drinking water. Radiology 40:474-84, 1943

75. Juncos LI, Donadio JV Jr: Renal failure and fluorosis. JAMA 222:783-85, 1972

76. Faccini JM: Fluoride and bone. Calcif Tissue Res 3:1-16, 1969

77. Taves DR, Terry R, Smith FA, Gardner DE: Use of fluoridated water in long-term hemodialysis. Arch Intern Med 115:167-72, 1965

78. Maumeme ME: Experience pour determiner l'action des fluorures sur l'economie animale. C R Acad Sci 39:538-40, 1854

79. Day TK, Powell-Jackson PR: Fluoride, water hardness, and endemic goitre. Lancet 1:1135-38, 1972

80. Latham MC, Grech P: The effects of excessive fluoride intake. Am J Public Health 57:651-60, 1967

81. Singh A, Jolly SS, Bansal BC, Mathur CC: Endemic fluorosis--Epidemiological, clinical and biochemical study of chronic fluorine intoxication in Punjab (India). Medicine 42:229-46, 1963

82. Leone NC, Leatherwood EC, Petrie IM, Lieberman L: Effect of fluoride on thyroid gland--Clinical study. J Am Dent Assoc 69:179-80, 1964

83. Galletti PM, Joyet G: Effect of fluorine on thyroidal iodine metabolism in hyperthyroidism. J Clin Endocrinol Metab 18:1102-10, 1958

84. Evang K: [Investigation among Norwegian aluminum workmen as to the occurrence of bronchial asthma, acute cryolite poisoning and "fluorosis."] Nord Hyg Tidskr 19:117-48, 1938 (Nor)

85. Midttun O: Bronchial asthma in the aluminum industry. Acta Allergol 15:208-21, 1960

86. Shea JJ, Gillespie SM, Waldbott G: Allergy to fluoride. Ann Allergy 25:388-91, 1967

87. Austen KF, Dworetzky M, Farr RS, Logan GB, Malkiel S, Middleton E Jr, Miller MM, Patterson R, Reed CE, Siegel SC, Van Arsdel PP Jr: A

statement on the question of allergy to fluoride as used in the fluoridation of community water supplies. J Allergy 47:347-48, 1971

88. Frajola WJ: Fluoride and enzyme inhibition, in Muhler JC, Hine MK (eds): Fluorine and Dental Health. Bloomington, Indiana University Press, 1959, pp 60-69

89. Wiseman A: Effect of inorganic fluoride on enzymes, in Handbuch der experimentellen Pharmakologic. Handbook of Experimental Pharmacology 20, Pt 2:pp 48-97, 1970

90. Ferguson DB: Effects of low doses of fluoride on serum proteins and a serum enzyme in man. Nature (New Biol) 231:159-60, 1971

91. Hjort E: [Investigation of possible fluorine poisoning among workers in an aluminum plant.] Nord Med Tidskr 15:47-54, 1938 (Nor)

92. Brun GC, Buchwald H, Roholm K: Die Fluorausscheidung im Harn bei chronischer Fluorvergiftung von Kryolitharbeitern. Acta Med Scand 106:261-73, 1941

93. Tourangeau FJ: [The health of workers in the aluminum extraction industry.] Laval Med 9:548-61, 1944 (Fr)

94. Bowler RG, Buckell M, Garrad J, Hill AB, Hunter D, Perry KMA, Schilling RSF: The risk of fluorosis in magnesium foundries. Br J Ind Med 4:216-22, 1947

95. Health of Workers Exposed to Sodium Fluoride at Open Hearth Furnace, bull No. 229. Federal Security Agency, Public Health Service, 1948

96. Largent EJ, Bovard PG, Heyroth FF: Roentgenographic changes and urinary fluoride excretion among workmen engaged in the manufacture of inorganic fluorides. Am J Roentgenol Radium Ther Nucl Med 65:42-48, 1951

97. Derryberry OM, Bartholomew MD, Fleming RBL: Fluoride exposure and worker health--The health status of workers in a fertilizer manufacturing plant in relation to fluoride exposure. Arch Environ Health 6:503-14, 1963

98. Parsons WD, deVilliers AJ, Bartlett LS, Becklake MR: Lung cancer in a fluorspar mining community--II. Prevalence of respiratory symptoms and disability. Br J Ind Med 21:110-16, 1964

99. deVilliers AJ, Windish JP: Lung cancer in a fluorspar mining community--I. Radiation dust, and mortality experience. Br J Ind Med 21:94-109, 1964

100. Vischer TL, Bernheim C, Guerdjikoff C, Wettstein P, Lagier R: Industrial fluorosis, in Vischer TL (ed): Fluoride in Medicine. Bern, Hans Huber Publishers, 1970, pp 96-105

101. Leidel NA, Key MM, Henschel AF, Sutton GW, Haij ME, Willens N, Svalina J, Knudson JC, Jones HH, Kupel RE, Kinser RE, Mauer PL: Environmental and Medical Survey Blockson Works--Olin Mathieson Corporation, Joliet, Ill, SR-20. US Dept of Health, Education, and Welfare, Public Health Service, Bureau of Disease Prevention and Environmental Control, National Center for Urban and Industrial Health, Occupational Health Program, 1967

102. Kaltreider NL, Elder MJ, Cralley LV, Colwell MO: Health survey of aluminum workers with special reference to fluoride exposure. J Occup Med 14:531-41, 1972

103. Johnson WM, Shuler PJ, Curtis RA, Wallingford KM, Mangin HJ, Parnes W, Donaldson HM: Industrial Hygiene Survey, Ormet Corporation Aluminum Facilities, Hannibal, Ohio. Cincinnati, US Dept Health, Education, and Welfare, Public Health Service, National Institute for Occupational Safety and Health, 1973

104. Shupe J, Miner ML, Greenwood DA, Harris LE, Stoddard GE: The effect of fluorine on dairy cattle--II. Clinical and pathological effects. Am J Vet Res 24:964-79, 1963

105. Shupe JL, Olson AE, Sharma RP: Fluoride toxicity in domestic and wild animals. Clin Toxicol 5:195-213, 1972

106. Phillips PH, Suttie JW, Zebrowski EJ: Effects of dietary sodium fluoride on dairy cows--VII. Recovery from fluoride ingestion. J Dairy Sci 46:513-16, 1963

107. Pack MR, Hill CA: Further evaluation of glass filters for sampling hydrogen fluoride. J Air Pollut Control Assoc 15:166-67, 1965

108. McKenna FE: Methods of fluorine and fluoride analysis--III. Nucleonics 9:51-58, 1951

109. McKenna FE: Methods of fluorine and fluoride analysis--I. Nucleonics 8:24-33, 1951

110. McKenna FE: Methods of fluorine and fluoride analysis--II. Nucleonics. 9:40-49, 1951

111. Elving PJ, Horton CA, Willard HH: Analytical chemistry of fluorine, in Simons JH (ed): Fluorine Chemistry. New York, Academic Press, 1954, vol 2, pp 58-67

112. Cholak J: Fluorides: A critical review--II. Analysis of fluorides. J Occup Med 1:648-54, 1959

113. West PW: Analytical methods for the study of air pollution. Pure Appl Chem 21:437-471, 1970

114. Farrah GH: Manual procedures for the estimation of atmospheric fluorides. J Air Pollut Control Assoc 17:738-41, 1967

115. Bourbon P: Analytical problems posed by pollution by fluorine compounds. J Air Pollut Control Assoc 17:661-63, 1967

116. Tentative method of analysis for fluoride content of the atmosphere and plant tissues (manual methods). Health Lab Sci 6:64-83, 1969

117. Habel K: The separation of gaseous and solid fluorine compounds during air quality measurements. Staub-Reinhalt Luft 28:26-31, 1968

118. Pack MR, Hill AC, Thomas MD, Transtrum LG: Determination of gaseous and particulate inorganic fluorides in the atmosphere, in Symposium on Air Pollution Control, ASTM Special Technical Publication No. 281. Baltimore, American Society for Testing Materials, 1960, pp 27-44

119. Mandl RH, Weinstein LH, Weiskopf GJ, Major JL: The separation and collection of gaseous and particulate fluorides, in Englund HM, Beery WT (eds): Proceedings of the Second International Clean Air Congress. New York, Academic Press, 1971, pp 450-58

120. Mukai K, Ishida H: The alkaline filter paper method for surveying fluorides in the atmosphere. Paper A70-10 presented at American Institute of Mining, Metallurgy and Petroleum Engineers, Denver, Colo, Feb 16-18, 1970

121. Levaggi DA, Oyung W, Feldstein M: A comparative study of the determination of fluoride on lime paper by a colorimetric method and the specific ion electrode method. Presented at the 10th State Health Department Methods Conference, San Francisco, Cal, Feb 19-21, 1969

122. Elfers LA, Decker CE: Determination of fluoride in air and stack gas samples by use of an ion specific electrode. Anal Chem 40:1658-61, 1968

123. Jahr J: [A new dual filter method for separate determination of hydrogen fluoride and dustlike fluorides in the air.] Staub Reinhalt Luft 31:17-22, June, 1972 (Ger)

124. Bierbaum PJ, Shuler PJ, Curtis RA, Mangin HJ: Industrial Hygiene Survey, Reynolds Metals Listerhill Reduction Plant, Sheffield, Ala. Cincinnati, US Dept Health, Education, and Welfare, Public Health Service, National Institute for Occupational Safety and Health, Division of Field Studies and Clinical Investigations, Environmental Investigations Branch, 1973

125. Harrold GC, Hurlburt RV: Device and technique for rapid determination of effluent fluorides. Anal Chem 21:1504-06, 1949

126. Luxon SG: A method for the estimation of particulate atmospheric contaminants in trace amounts. Ann Occup Hyg 3:30-32, 1961

127. Adams DF: Further applications of the Limed Filter Paper Technique in fluorine air pollution studies. J Air Pollut Control Assoc 7:88-91, 1957

128. Adams DF: A quantitative study of the limed filter paper technique for fluorine air pollution studies. Int J Air Water Poll 4:247-55, 1961

129. Robinson E: Determining fluoride air concentrations by exposing limed filter paper. Am Ind Hyg Assoc Q 18:145-48, 1957

130. Wilson WL, Campbell MW, Eddy LD, Poppe WH: Calibration of limed filter paper for measuring short-term hydrogen fluoride dosages. Am Ind Hyg Assoc J 28:254-59, 1967

131. Pack MR, Hill AC, Benedict HM: Sampling atmospheric fluorides with glass fiber filters. J Air Pollut Control Assoc 13:374-77, 1963

132. Nielsen JP, Dangerfield BS: Use of ion exchange resins for determination of atmospheric fluorides. Arch Ind Health 11:61-65, 1955

133. Nielsen HM: The determination of fluoride in soft tissue. Arch Ind Health 21:316-17, 1960

134. Talvitie NA, Brewer LW: Separation of fluoride by exchange--Application to urine analysis. Am Ind Hyg Assoc J 21:287-95, 1960

135. Bowley MJ: The micro determination of sulphate and fluoride in lime-silica-sulphate-fluoride phase systems. Analyst 94:787-93, 1969

136. Willard HH, Winter OB: Volumetric method for determination of fluorine. Ind Eng Chem (Anal Ed) 5:7-10, 1933

137. Derner HA: Semiautomated determination of fluoride in urine. Am Ind Hyg Assoc J 28:357-62, 1967

138. Mavrodineanu R, Coe RR: Improved apparatus and procedures for sampling and analyzing air for fluorides. Contributions from Boyce Thompson Institute 18:173-80, 1955

139. Singer L, Armstrong WD: Determination of fluoride procedure based upon diffusion of hydrogen fluoride. Anal Biochem 10:495-500, 1965

140. Rowley RJ, Farrah GH: Diffusion method for determination of urinary fluoride. Am Ind Hyg Assoc J 23:314-18, 1962

141. Taves DR: Determination of submicromolar concentrations of fluoride in biological samples. Talanta 15:1015-23, 1968

142. Cumpston AG, Dinman BD: A modified diffusion method for the determination of urinary fluoride. Am Ind Hyg Assoc J 26:461-64, 1965

143. Thomas MD, Amtower RE: The microdetermination of fluoride in vegetation. J Air Pollut Control Assoc 19:439-41, 1969

144. Farrah GH: Diffusion method for determination of urinary fluoride: Recent developments. Am Ind Hyg Assoc J 25:55-58, 1964

145. Armstrong WD: Modification of the Willard-Winter method for fluorine determination. J Am Chem Soc 55:1741-42, 1933

146. McClure FJ: Microdetermination of fluorine by thorium nitrate titration. Ind Eng Chem (Anal Ed) 11:171-73, 1939

147. Smith FA, Gardner DE: A comparison of the thorium nitrate back-titration and the salt-acid thorium titration for the determination of fluoride. Arch Biochem 29:311-14, 1950

148. Linde HW: Estimation of small amounts of fluoride in body fluids. Anal Chem 31:2092-94, 1959

149. Talvitie NA: Colorimetric determination of fluoride in natural waters with thorium and alizarin. Ind Eng Chem (Anal Ed) 15:620-21, 1943

150. Smith FA, Gardner DE: The determination of fluoride in urine. Am Ind Hyg Assoc Q 16:215-20, 1955

151. Rao DN, Venkateswarlu P: Colorimetric estimation of fluorine in natural waters--Thorium-alizarin method improved. Indian J Med Res 39:223-27, 1951

152. Megregian S, Maier FJ: Modified zirconium-alizarin reagent for determination of fluoride in water. J Am Water Works Assoc 44:239-48, 1952

153. Megregian S: Rapid spectrophotometric determination of fluoride with Zirconium-Eriochrome Cyanine R Lake. Anal Chem 26:1161-66, 1954

154. Rausa G, Trivello R: Un metodo spettrofotometrico per la determinazione del fluoro nelle urine. Igiene Moderne 63:89-103, 1970

155. Wade MA, Yamamura SS: Microdetermination of fluoride using an improved distillation procedure. Anal Chem 37:1276-78, 1965

156. West PW, Lyles GR, Miller JL: Spectrophotometric determination of atmospheric fluorides. Environ Sci Tech 4:487-91, 1970

157. Weinstein LH, Mandl RH, McCune DC, Jacobson JS, Hitchcock AE: Semiautomated analysis of fluoride in biological materials. J Air Pollut Control Assoc 15:222-25, 1965

158. Waldo AL, Zipf RE: The determination of fluoride in drinking water and biological materials. J Lab Clin Med 40:601-09, 1952

159. Schenk GH, Dilloway KP: Determination of fluoride by fluorescence quenching. Anal Let 2:379-85, 1969

160. Marshall BS, Wood R: A simple field test for the determination of hydrogen fluoride in air. Analyst 93:821-26, 1968

161. Mulder GJ: De microbepaling van fluoride in urine. Pharm Weekbl 94:329-45, 1959

162. Sarma PL: Spectrophotometric determination of fluoride by Zirconium-Eriochrome Cyanine R. Anal Chem 36:1684-85, 1964

163. Adams DF, Koppe RK, Matzek NE: Colorimetric method for continuous recording analysis of atmospheric fluoride--Test chamber and interference studies with the Mini-Adak analyzer. Anal Chem 33:117-19, 1961

164. Crossley HE: Fluorine in coal--II. The determination of fluorine in coal. J Soc Chem Ind 63:284-88, 1944

165. Macejunas AG: Spectrophotometric determination of fluoride using zirconium-xylenol orange. J Am Water Works Assoc 61:311-13, 1969

166. Cabello-Tomas ML, West TS: Kinetochromic spectrophotometry--I. Determination of fluoride by catalysis of the zirconium-xylenol orange reaction. Talanta 16:781-88, 1969

167. Bellack E, Schouboe PJ: Rapid photometric determination of fluoride in water--Use of sodium 2-(p-Sulfophenylazo)-1,8-dihydroxynaphthalene-3,6-disulfonate-Zirconium Lake. Anal Chem 30:2032-34, 1958

168. Panin KP: [Separate determination of inorganic fluorides, gaseous and in droplet aerosols, in air.] Gig Sanit 36:403-06, 1971 (Rus)

169. Demidov AV, Mokhov LA: Rapid methods for the determination of inorganic substances in the air, in USSR Literature on Air Pollution and Related Occupational Diseases--A Survey. US Dept Health, Education, and Welfare, Public Health Service, 1964, vol 10, pp 60-61

170. Ivie JO, Zielenski LF, Thomas MD, Thompson CR: Atmospheric fluorometric fluoride analyzer. J Air Pollut Control Assoc 15:195-97, 1965

171. Dubois L, Monkman JL, Teichman T: The determination of urinary fluorides. Am Ind Hyg Assoc J 23:157-63, 1972

172. Kojima T, Ichise M, Seo Y: Selective gas-chromatographic detection using an ion-selective electrode. Talanta 19:539-547, 1972

173. Fresen JA, Cox FH, Witter MJ: The determination of fluoride in biological materials by means of gas chromatography. Pharm Weekbl 103:909-14, 1968

174. Cropper E, Puttnam NA: Gas chromatographic determination of fluoride in dental creams. J Soc Cos Chem 21:533-40, 1970

175. Gutsche B, Herrmann R, Rudiger K: Ein fluorspezifischer Detektor fur die Gas-Chromatographie. Z Anal Chem 258:273-77, 1972

176. Gutsche B, Herrmann R: Simplified fluorine-specific detector for gas-chromatography. Z Anal Chem 259:126-27, 1972

177. Lennox D, Leroux J: Applications of x-ray diffraction analysis in the environmental field. Arch Ind Hyg Med 8:359-70, 1953

178. Spoonemore JW: A method of determining trace amounts of fluorine in lithium-fluoride samples by means of neutron activation analysis. MS Thesis. Seattle, University of Washington, 1963

179. Bond AM, O'Donnell TA: Determination of fluoride by atomic absorption spectrometry. Anal Chem 40:560-63, 1968

180. Bresler PI: [Photometric determination of fluorine and hydrogen fluoride in gas mixtures.] Zh Anal Khim 27:145-50, 1972 (Rus)

181. Kakabadse GJ, Manohin B, Bather JM, Weller EC, Woodbridge P: Decomposition and the determination of fluorine in biological materials. Nature 229:626-27, 1971

182. Van Leuven HCE: Organic multi-element analysis with a small mass spectrometer as detector--A preliminary note. Anal Chim Acta 49:346-66, 1970

183. Ke PJ, Regier LW, Power HE: Determination of fluoride in biological samples by a nonfusion distillation and ion selective membrane electrode method. Anal Chem 41:1081-84, 1969

184. Bien SM: A new micro-method for the determination of fluorides. J Dent Res 22:123-27, 1943

185. Adams DF: An automatic hydrogen fluoride recorder proposed for industrial hygiene and stack monitoring. Anal Chem 32:1312-16, 1960

186. Howard OH, Weber CW: An improved continuous internal electrolysis analyzer for gaseous fluorides in industrial environments. Am Ind Hyg Assoc J 23:48-57, 1962

187. Jacobson JS, McCune DC, Weinstein LH, Mandl RH, Hitchcock AE: Studies on the measurement of fluoride in air and plant tissues by the Willard-Winter and semiautomated methods. J Air Pollut Control Assoc 16:367-71, 1966

188. Howard OH, Weber CW: A portable continuous analyzer for gaseous fluorides in industrial environments. Arch Ind Health 19:355-64, 1959

189. Nichols PNR: The simple modification of a commercial sulphur dioxide meter for the determination of the atmospheric pollutants. Chem Ind 39:1654-55, 1964

190. Thompson CR, Zielenski LF, Ivie JO: A simplified fluorometric fluoride analyzer. Atmos Environ 1:253-59, 1967

191. Byczkowski S, Krechniak J, Gietazyn T: An attempt to evaluate exposure to fluorides based upon fluoride levels in hair. Fluoride 4:98-100, 1971

192. Harwood JE: The use of an ion-selective electrode for routine fluoride analyses on water samples. Water Research 3:273-80, 1969

193. Liberti A, Mascini M: Determination of fluorides in polluted air by use of an ion specific electrode. Fluoride 4:49-56, 1971

194. Liberti A, Mascini M: Continuous measurements of atmospheric pollutants by means of specific ion membrane electrodes, in Englund HM, Beery WT (eds): Proceedings of the Second International Clean Air Congress. New York, Academic Press, 1971, pp 519-22

195. Liberti A, Mascini M: Anion determination with ion selective electrodes using Grans plots--Application to fluoride. Anal. Chem 41:676-79, 1969

196. Harzdorf C: Verwendung einer fluoridspezifischen Elektrode bei automatisch-potentiometrischen titrationen. Z Anal Chem 245:67-70, 1969

197. Neefus JD, Cholak J, Saltzman BE: The determination of fluoride in urine using a fluoride-specific ion electrode. Am Ind Hyg Assoc J 31:96-99, 1970

198. Sun MW: Fluoride ion activity electrode for determination of urinary fluoride. Am Ind Hyg Assoc J 30:133-36, 1969

199. Materova EA, Grinberg GP, Evstifeeva MM: [A study of the fluoride function of precipitation and anion exchange membrane electrodes.] Zh Anal Khimii 24:821-24, 1969 (Rus)

200. Jacobson JS, Heller LI: Selective ion electrode analysis of fluoride in vegetation, in Englund HM, Beery WT (eds): Proceedings of the

Second International Clean Air Congress. New York, Academic Press, 1971, pp 459-62

201. Singer L, Armstrong WD, Vogel JJ: Determination of fluoride content of urine by electrode potential measurements. J Lab Clin Med 74:354-58, 1969

202. Anfalt T, Jagner D: Effect of acetate buffer on the potentiometric titration of fluoride with lanthanum using a lanthanum fluoride membrane electrode. Anal Chim Acta 47:483-94, 1969

203. Cernik AA, Cooke JA, Hall RJ: Specific ion electrode in the determination of urinary fluoride. Nature 227:1260-61, 1970

204. MacLeod KE, Crist HC: Comparison of the SPADNS-Zirconium Lake and specific ion electrode methods of fluoride determination in stack emission samples. Anal Chem 45:1272-73, 1973

205. Domzalska E: Wystepowanie chorob przyzebia u osob w roznym stopniu narazonych na dzialanie zwiazkow fluoru. Czas Stomatol 25:1005-11, 1972

206. Weber SJ: Specific ion electrodes in pollution control. Am Lab 15-18, 20-23, July 1970

207. Venkateswarlu P, Singer L, Armstrong WD: Determination of ionic (plus ionizable) fluoride in biological fluids--Procedure based on absorption of fluoride ion on calcium phosphate. Anal Biochem 42:350-59, 1971

208. Standard Methods of Test for Fluoride Ion in Water, in ASTM Standards. Industrial Water--Atmospheric Analyses Pt 23, 1972. ASTM Designation--D1179-72, pp 191-96

209. Williams CR: Atmospheric contamination from the casting of magnesium. J Ind Hyg Toxicol 24:277-80, 1942

210. Williams CR, Silverman L: Collection of fluoride fumes in air. J Ind Hyg Toxicol 27:115-27, 1945

211. Fluoride-Bearing Dusts and Fumes (Inorganic) F-Hygienic Guide Series. Michigan, Am Ind Hyg Assoc, 1965

212. Inorganic fluorides, National Safety Council Data Sheet 442. Chicago, National Safety Council, 1957

213. Tebbens BD, Drinker P: Ventilation in arc welding with coated electrodes. J Ind Hyg Toxicol 23:322-42, 1941

214. Drinker P, Nelson KW: Welding fumes in steel fabrication. Ind Med 13:673-75, 1944

215. Ferry JJ: Fluorine exposure during low-hydrogen welding. Am Ind Hyg Assoc Q 14:173-76, 1953

216. Smith LK: Fume exposures from welding with low hydrogen electrodes. Ann Occup Hyg 10:113-21, 1967

217. Pantucek M: Influence of filler materials on air contamination in manual electric arc welding. Am Ind Hyg Assoc J 32:687-92, 1971

218. McCann JK: Health hazard from flux used in joining aluminum electricity cables. Ann Occup Hyg 7:261-68, 1964

219. Yaffe CD: Atmospheric concentrations of fluorides in aluminum reduction plants. J Ind Hyg Toxicol 28:29-31, 1946

220. Breysse PA: Industrial hygiene study--Potliner removal operation Kaiser Aluminum and Chemical Corporation. Mead Works, Spokane, Washington. Approved by Ross W. Kusian. Environmental Research Laboratory, Dept of Public Health and Preventive Medicine, School of Medicine, University of Washington, July 13,1959, 4 pp

221. Hickey HR: A practical brief on controlling aluminum effluent reduction. Air Eng 10:20-22, 1968

222. Hiszek N, Horvath F, Mandi A, Villanyi G: [Health hazards caused by fluorine in aluminum plants.] Banyasz es Kohasz Lapok, Kohaszat 103:514-17, 1970 (Hung)

223. Kuznetsova LS: [The effects of the various operations in the manufacture of superphosphate on the sex organs of female workers.] Gig Tr Prof Zabol 13:21-25, 1969 (Rus)

224. Zufelt JC: Some practical aspects of the addition of sodium fluoride to a municipal water supply. Water Sewage Works 95:57-60, 1948

225. deVilliers AJ, Windish JP, Brent FdN, Hollywood B, Walsh C, Fisher JW, Parsons WD: Mortality experience of the community and of the fluorspar mining employees at St. Lawrence, Newfoundland. Occup Health Rev 22:1-15, 1971

226. Luxon SG: Atmospheric fluoride contamination in the pottery industry. Ann Occup Hyg 6:127-30, 1963

227. Markuson KE: The use of sodium fluoride in the manufacture of steel. Ind Med 16:434-36, 1947

228. Cook WA: Maximum allowable concentrations of industrial atmospheric contaminants. Ind Med 14:936-46, 1945

229. 1947 M.A.C. values. Ind Hyg Newsletter 7:15-16, 1947

230. Threshold Limit Values Adopted at April, 1948 Meeting of American Conference of Governmental Industrial Hygienists in Boston, Massachusetts. Cincinnati, ACGIH, 1948

231. American Conference of Governmental Industrial Hygienists: Documentation of Threshold Limit Values. Cincinnati, ACGIH, 1966, p 51

232. Ronzani E: Uber den Einfluss der Einatmungen von reizenden Gasen der Industrien auf die Schutzkrafte des Organismus gegenuber den infektiven Krankheiten--Experimental-Untersuchungen. Arch Hyg 70:217-69, 1909

233. Roholm K: Fluorvergiftung, eine "neue" Krankheit. Klin Wochenschr 15:1425-31, 1936

234. American Conference of Governmental Industrial Hygienists: Documentation of the Threshold Limit Values for Substances in Workroom Air, ed 3. Cincinnati, ACGIH, 1971, pp 116-17

235. Elkins HB: The Chemistry of Industrial Toxicology, ed 2. New York, John Wiley & Sons Inc, 1959, pp 7, 11, 20, 71-73, 220-22, 225, 235-36, 238-40, 254, 257

236. Hodge HC: Notes on the effects of fluoride deposition on body tissues. Arch Ind Health 21:350-52, 1960

237. Princi F: Fluorides: A critical review--III. The effects on man of the absorption of fluoride. J Occup Med 2:92-99, 1960

238. Truhaut R: Maximum allowable concentrations, 2nd International Symposium, Paris, 1963. Arch Environ Health 8:487-91, 1964

239. United States of America Standards Institute: Acceptable Concentrations of Hydrogen Fluoride and Inorganic Fluoride Dusts, Z37.28-1966. New York, United States of America Standards Institute, 1966

240. Stokinger HE: Toxicity following inhalation of fluorine and hydrogen fluoride, in Voegtlin C, Hodge HC (eds): Pharmacology and Toxicology of Uranium Compounds, ed 1. New York, McGraw-Hill Book Company Inc, 1949, pp 1021-1057 (National Nuclear Energy series--Manhattan Project Technical section Division VI--vol 1)

241. Heyroth FF: Halogens, in Patty FA (ed): Industrial Hygiene and Toxicology ed 2 rev; Toxicology (DW Fassett, DD Irish, eds). New York, Interscience Publishers Inc, 1963, vol 2, pp 832-44

242. Pennsylvania Dept of Environmental Resources: Title 25: Rules and regulations; Pt 1: Dept of Environmental Resources; Subpart D: Environmental Health and Safety; Article IV: Occupational Health and Safety; Chapter 201: Places of Employment; Subchapter A: Threshold Limits. 204-201.121. Adopted Sept 28, 1971, effective Nov 1, 1971

243. Short Term Limits for Exposure to Airborne Contaminants--A Documentation. Harrisburg, Pennsylvania Dept of Health, Div of Occupational Health, no date

244. Czechoslovak Committee of MAC: Documentation of MAC in Czechoslovakia. Prague, June 1969, pp 76-80

245. Smelyanskiy ZB, Ulanova IP: [New standards for permissible levels of toxic gases, fumes and dust in the air of work areas.] Gig Tr Prof Zabol 3:7-15, 1959 (Rus)

246. Largent EJ: Metabolism of Inorganic Fluorides, in Shaw JH (ed): Fluoridation as a Public Health Measure. Washington, DC, American Association for the Advancement of Science, 1954, pp 49-78

247. Largent EJ: Effects of fluorides on man and animals. Proceedings of the First Nation Air Pollution Symposium, 1949, pp 129-34

248. Boice JD, Burnett BM: Considerations of possible pregnancy in the use of diagnostic X-rays, in Health Physics in the Healing Arts, FDA Pub 73-8029, 1973

249. American Conference of Governmental Industrial Hygienists, Committee on Industrial Ventilation: Industrial Ventilation--A Manual of Recommended Practice, ed 13. Ann Arbor, Mich, Edwards Bros Inc, 1972

250. Table of Sorbents for Contaminants Listed in ACGIH 1970 TLV. Am Ind Hyg Assoc J 32:404-19, 1971

251. Electroplating, Data Sheet D-Gen 46, National Safety Council. National Safety News 64:32-33, 101-10, July 1951

252. Seferian S: Fluoride-handling safety. J Am Water Works Assoc 64:604-08, 1972

253. Fluorine and compounds, in International Labour Office: Encyclopedia of Occupational Health and Safety. New York, McGraw-Hill Book Company Inc, 1972, vol 1 A-K

254. Davis WL: Ambient air fluorides in Salt Lake County. Rocky Mt Med J 69:53-56, 1972

255. Pennsylvania Dept of Environmental Health: Title 25: Rules and Regulations; Part I: Dept of Environmental Resources; Subpart C: Protection of Natural Resources; Article III: Air Resources; Chapter 131: Ambient Air Quality Standards. Adopted September 2, 1971, revised January 27, 1972

256. Weaver NK: Atmospheric contaminants and standards--Status report. J Occup Med 11:445-61, 1969

257. Washington Administrative Code 18-48 (4/1/71)

258. Gafafer WM (ed): Occupational Diseases--A Guide to Their Recognition, publication no. 1097. US Dept of Health, Education, and Welfare, Public Health Service, 1964, pp 148-50

259. Greenwood DA: Fluoride intoxication. Physiol Rev 20:582-616, 1930

260. Weast RC (ed): Handbook of Chemistry and Physics--A Ready-Reference Book of Chemical and Physical Data. Cleveland, The Chemical Rubber Co, 1973

261. Oelschlager W: Fluoride in food. Fluoride Q Rep 3:6-11, 1970

262. Singer L, Armstrong WD: Regulation of human plasma fluoride concentration. J Appl Physiol 15:508-10, 1960

263. Armstrong WD, Singer L, Ensinck J, Rich C: Plasma fluoride concentrations of patients treated with sodium fluoride. J Clin Invest 43:555-56, 1964

IX. APPENDIX I

AIR SAMPLING PRACTICES FOR FLUORIDES

General Requirements

Air contaminant concentrations shall be determined within the worker's breathing zone and shall meet the following criteria in order to evaluate conformance with the standard:

(a) Samples collected shall be representative of the individual worker's exposure.

(b) Sample data sheets shall include:

(1) The date and time of sample collection

(2) Sampling duration

(3) Volumetric flowrate of sampling

(4) Ambient temperature and pressure

(5) A description of the sampling location

(6) Other pertinent information (eg, worker's name, shift, work process, etc)

(c) Sampling will be in accordance with the provisions of the procedures outlined herein.

Breathing Zone Sampling

(a) In order to assure that a sample is representative of a worker's exposure, collection shall be as near the breathing zone of the worker as practical. Sampling should not hamper the typical movement patterns associated with his work.

(b) A portable, battery-operated personal sampling pump capable of being calibrated to ±5% at the required flow, and a 2- or 3-piece filter cassette containing a filter (prepared as in Appendix II) shall be used to collect the sample.

(c) The sampling rate shall be accurately maintained at a value between 1.5-2.5 liters per minute and samples shall be taken for a time period appropriate to the estimated concentration of fluoride in the air; eg, at the environmental limit (2.5 mg F/cu m), a flowrate of 2.0 liters/min, and sample time of 60 min, fluoride would be present in solution at 7.5 ppm after sample treatment (20 ml water and 20 ml Total Ionic Strength Adjustment Buffer [TISAB]).

(d) A minimum of 4 samples shall be taken for each operation and averaged on a time-weighted basis.

(e) For determining background correction a field blank and a laboratory blank (clean filters) shall be analyzed simultaneously with the sample.

Calibration of Sampling Trains

Since the accuracy of an analysis can be no greater than the accuracy of the volume of air which is measured, the accurate calibration of a sampling pump is essential to the correct interpretation of the pump's indication. The frequency of calibration is dependent on the use, care, and handling to which the pump is subjected. In addition, pumps should be recalibrated if they have been subjected to misuse or if they have just been repaired or received from a manufacturer. If pumps receive hard usage, more frequent calibrations may be necessary. Regardless of use, maintenance and calibration should be performed on a regular schedule and records of these kept.

Ordinarily, pumps should be calibrated in the laboratory both before they are used in the field and after they have been used to collect a large number of field samples. The accuracy of calibration is dependent on the type of instrument used as a reference. The choice of calibration instrument will depend largely upon where the calibration is to be performed. For laboratory testing, a 1- or 2-liter buret or wet-test meter is recommended, although other standard calibrating instruments such as spirometer, Marriott's bottle, or dry gas meter can be used. The actual set-up will be similar for these instruments.

Instructions for calibration with the soapbubble meter follow. If another calibration device is used, equivalent procedures should be followed. The calibration setup for personal sampling pumps with a filter is shown in Figure XII-1.

(a) Check the voltage of the pump battery with a voltmeter both with the pump off and while it is operating to assure adequate voltage for calibration.

(b) Place a treated membrane filter in the holder.

(c) Assemble the sampling train as shown in Figure XII-1.

(d) Turn the pump on and moisten the inside of the soapbubble meter by immersing the buret in the soap solution. Draw bubbles up the inside until they are able to travel the entire buret length without bursting.

(e) Adjust the pump rotameter to provide a flowrate of 1.5-2.5 liters per minute. (Often a flowrate of 1.8 liters a minute is satisfactory for prolonged sampling periods.)

(f) Check the water manometer to insure that the pressure drop across the sampling train does not exceed 13 inches of water (approximately 1 inch of mercury).

(g) Start a soapbubble up the buret and, with a stopwatch, measure the time it takes for the bubble to transit a minimum of 1.0 liter.

(h) Repeat the procedure in (g) above at least 3 times, average the results, and calculate the flowrate by dividing the volume between the preselected marks by the time required for the soapbubble to traverse the distance.

(i) Data for the calibration include the volume measured, elapsed time, pressure drop, air temperature, atmospheric pressure, serial number of the pump, date, and name of the person performing the calibration.

(j) Corrections to the flowrate may be necessary if the pressure or temperature when samples are collected differs significantly from that when calibration was performed. Flow rates may be calculated by using the following formula:

$$q\ (\text{actual}) = q\ (\text{indicated}) \sqrt{\frac{P\ (\text{calibrated})}{P\ (\text{actual})} \times \frac{T\ (\text{actual})}{T\ (\text{calibrated})}}$$

where q = volumetric flowrate

P = pressure

T = temperature (in degrees Kelvin or Rankine)

(k) Use graph paper to record the air flow corrected to 25 C and 760 torr as the ordinate and the rotameter readings as the abscissa.

X. APPENDIX II

ANALYTICAL METHODS FOR FLUORIDE

Principle of the Method

(a) A known volume of air is drawn through a treated filter to collect fluoride particulate and gases.

(b) The filters are returned to the laboratory where the sample is isolated and/or analyzed. Hydrated aluminum fluoride should be treated as a special case, requiring an alkaline fusion for conversion to a soluble form.

(c) If water-insoluble material is present in the sample an isolation procedure is required.

(d) After isolation and initial sample preparation with TISAB, the fluoride concentration in the sample is measured by ion-selective electrode.

Range and Sensitivity

(a) The limit of detection of the method is 0.02 ppm of fluoride in the liquid sample.

(b) The upper limit of the method may be varied, according to the sample requirements, by appropriate selection of the size aliquot taken for analysis.

Interferences

In the isolation process, interfering materials that volatilize from an acid medium must be eliminated. Relatively large amounts of chloride may be fixed in the diffusion vessel by the cautious addition of 0.2 g silver perchlorate. Samples high in carbonates require caution upon acidification to control effervescence. The ion-selective electrode is relatively free from interferences. Certain cations (notably those of hydrogen, aluminum, silicon, and ferric iron) will bind fluoride and reduce the fluoride activity in acid solution. By pH adjustment and addition of a complexing agent such as CDTA (1,2-diamino cyclohexane N,N,N',N'-tetraacetic acid) these interferences are minimized.

Precision

The precision of electrode methods is 1.5-3.5%; an electrode method tested by 111 laboratories was found to have a precision of 0.030 at a concentration of 0.85 mg/liter (ppm) of fluoride. [208]

Advantages of the Method

(a) No evaporation losses.

(b) Rapid, simple analyses are usually possible.

(c) Potential interferences are easily minimized.

(d) Minimal clean-up time (no elaborate glassware); electrode easily rinsed clean with distilled water.

Apparatus

(a) General

(1) Filters, cellulose acetate, membrane, 0.8-μm. Two- or 3-piece plastic filter holders (field monitors) are convenient for taking the sample and transporting the filters.

(2) Glassware should be borosilicate; plastic ware should be used for preparation and storage of fluoride standards and samples. Apparatus is cleaned in a hot detergent solution, followed by a rinse in

warm, dilute nitric or hydrochloric acid solution and a rinse with distilled water. All sampling devices, volumetric glassware, and reagent solutions must be stored under suitable conditions to protect from airborne dust and fumes, and should be reserved for exclusive use in low-fluoride analyses.

(3) An ion-selective meter having a direct reading concentration scale or a pH meter with an expanded mV scale and a precision of ±1% should be used. A fluoride ion-selective electrode and single-junction or glass sleeve type saturated potassium chloride reference electrode are used in conjunction with the meter.

(4) A magnetic stirrer having fluorocarbon-coated stirring bars equipped with a heat shield between the stirring motor and sample should be used.

(b) Isolation by Diffusion

(1) Microdiffusion dish--disposable plastic Petri dish. The Conway Microdiffusion dish with Obrink modification, made of methyl methacrylate resin or similar plastic capable of withstanding 60 C may be used. [116]

(2) A thermostatically controlled oven capable of maintaining ±1 C in the 50-60 C range.

(3) A Mohr pipet with a capacity of 0.1 ml, having 0.01 ml subdivisions.

Reagents

All reagents are ACS reagent grade except where noted.

(a) Filter

Alcoholic Sodium Formate--dissolve 10 g sodium formate in 50 ml distilled water; dilute to 100 ml with ethyl alcohol.

(b) Diffusion

(1) Perchloric acid, 70-72%

(2) Silver perchlorate, anhydrous, C.P.

(3) Alcoholic sodium hydroxide, 1.0 N solution--dissolve 4 grams sodium hydroxide in 5 ml of water and dilute to 100 ml ethyl or methyl alcohol.

(c) Analysis

TISAB is commercially available or can be prepared as follows: Place approximately 500 ml of distilled water in a 1-liter beaker. Add 57 ml of glacial acetic acid, 58 g of sodium chloride, and 4 g of disodium CDTA. (CDTA is available commercially as cyclohexylene dinitrilotetraacetic acid, or 1,2-diaminocyclohexane N,N,N',N'-tetraacetic acid.) Stir to dissolve. Place beaker in a water bath for cooling, insert a calibrated pH electrode into the solution, and slowly add approximately 5 N sodium hydroxide until the pH is between 5.0 and 5.5. Cool to room temperature. Pour into a 1-liter flask and add distilled water to the mark.

Standards

Serially dilute commercial standards or prepare standards as follows:

(a) Standard solution #1: Dissolve 2.2105 g of pure sodium fluoride in distilled water and dilute to 1 liter in a volumetric flask, mix, and transfer to a plastic storage bottle (1 ml = 1.00 mg F).

(b) Standard solution #2: Dilute 10 ml of standard #1 to 1 liter with distilled water in a volumetric flask, mix, and transfer to a plastic storage bottle (1 ml = 0.01 mg F).

Filter Preparation

Cellulose acetate filter paper (0.8 m pore, 37-mm diam.) is dipped in a solution of 50% ethanol-10% sodium formate and allowed to air dry.

Filter Treatment

If in the judgment of the analyst water-insoluble fluorides are present in the air sample, remove the filter from the holder and place it in the bottom section of the microdiffusion vessel; follow diffusion procedure. If it is known that only water-soluble fluorides are present, remove the filter from the holder and place in a 50-ml plastic beaker. Add 10 ml TISAB solution and stir the sample for 30 minutes. Follow analysis procedure.

Diffusion Procedure

With a 0.1-ml Mohr pipet, place 0.05 ml of the alcoholic sodium hydroxide solution on the center of the inside top of the plastic Petri dish. Use the tip of the pipet to spread the droplet into a circular spot at least 3-4 cm in diameter. Dry for about 1 hour under slightly reduced pressure in a desiccator containing activated alumina. To the bottom portion of the Petri dish containing the filter, cautiously add 1 ml distilled water, 0.2 g silver perchlorate, and 2.0 ml perchloric acid. Immediately close the dish with a prepared lid. Place the unit in an oven maintained at 50-60 C and allow to remain 16-20 hours. Carefully remove the unit from the oven and take off the lid. Follow analysis procedure.

Analysis Procedure

(a) Air Samples

If the diffusion step was included, wash the Petri dish lid with successive portions of distilled water (10 ml total) and collect the washings in a 50-ml plastic beaker. To the beaker containing the diffusion washings, add 10 ml distilled water and 20 ml TISAB solution. If the diffusion step was not included, add 20 ml distilled water and 10 ml TISAB solution. The final solution should be a 1:1 dilution of 20 ml distilled water and 20 ml TISAB. Using an expanded scale pH meter or a ion-selective meter, place the electrodes in appropriate standard solutions and record the mV readings as the solution is stirred. Rinse and blot electrode surfaces between solutions. After the meter has been calibrated using manufacturer's recommendations and a standard curve has been established, place the electrodes in the sample solution and record the mV reading. The meter will require several minutes to come to equilibrium. It should be read at equivalent elapsed times for each sample. Determine F concentration by referring to the standard curve.

Note: Since the rate of stirring speed affects the electrode response time, a constant and consistent speed should be used for air and urine samples and standards.

(b) Urine Samples

Urine samples should be collected in plastic or wax-impregnated cardboard specimen bottles. Care must be taken when collecting and handling urine specimens to avoid F contamination. Upon receipt of the sample, either analyze immediately or refrigerate to retard bacterial action on urea which increases the sample pH through release of ammonia. Determine the specific gravity before analysis. Dilute the sample 1:1 with TISAB and proceed directly with electrode analysis. Determine F concentration in mg fluoride per liter of urine by referring to a previously prepared standard curve. Adjust the measured concentration to a specific gravity of 1.024 as follows [40,41]:

$$\text{Adjusted mg F/liter at specific gravity } 1.024 = \frac{0.024\ (A)}{B-1.000}$$

where A = measured F concentration mg/liter
B = measured specific gravity

If a urinometer is used which has been calibrated against water at 4 C, a temperature correction must be made. [41]

Note: Urine samples with specific gravities of less than 1.010 are not indicative of the normal range and should be discarded. Failure to do so will result in an erroneously high F concentration when an adjustment to a specific gravity of 1.024 is accomplished.

Calculations

(a) Calculate the fluoride concentration in the atmosphere at 25 C and 760 Torr, in terms of mg of fluoride/cu m as follows:

$$\text{Total fluoride (mg/cu m)} = \frac{(2550)(F)(273 + t)}{pv}$$

where F = mg of fluoride in total sample
p = sampling pressure in torr (mm of mercury)
t = sampling temperature in degrees Celsius
v = sample volume in liters
2550 = STP correction factor

(b) Conversion factors for fluoride in solution:

To convert from moles per liter (molarity) fluoride to:

parts per million (ppm)	multiply by 19,056
milligrams per liter	" " "

(c) Standard Curve Preparation

Using semilogarithmic graph paper, plot millivolts on the linear axis and fluoride concentration on the logarithmic axis.

XI. APPENDIX III

MATERIAL SAFETY DATA SHEET

The following items of information which are applicable to a specific product or material shall be provided in the appropriate block of the Material Safety Data Sheet (MSDS).

The product designation is inserted in the block in the upper left corner of the first page to facilitate filing and retrieval. Print in upper case letters as large as possible. It should be printed to read upright with the sheet turned sideways. The product designation is that name or code designation which appears on the label, or by which the product is sold or known by employees. The relative numerical hazard ratings and key statements are those determined by the rules in Chapter V, Part B, of the NIOSH publication, *An Identification System for Occupationally Hazard Materials*. The company identification may be printed in the upper right corner if desired.

(a) Section I. Product Identification

The manufacturer's name, address, and regular and emergency telephone numbers (including area code) are inserted in the appropriate blocks of Section I. The company listed should be a source of detailed backup information on the hazards of the material(s) covered by the MSDS. The listing of suppliers or wholesale distributors is discouraged. The trade name should be the product designation or common name associated with the material. The synonyms are those commonly used for the product, especially formal chemical nomenclature. Every known chemical designation or competitor's trade name need not be listed.

(b) Section II. Hazardous Ingredients

The "materials" listed in Section II shall be those substances which are part of the hazardous product covered by the MSDS and individually meet any of the criteria defining a hazardous material. Thus, one component of a multicomponent product might be listed because of its toxicity, another component because of its flammability, while a third component could be included both for its toxicity and its reactivity. Note that a MSDS for a single component product must have the name of the material repeated in this section to avoid giving the impression that there are no hazardous ingredients.

Chemical substances should be listed according to their complete name derived from a recognized system of nomenclature. Where possible, avoid using common names and general class names such as "aromatic amine," "safety solvent," or "aliphatic hydrocarbon" when the specific name is known.

The "%" may be the approximate percentage by weight or volume (indicate basis) which each hazardous ingredient of the mixture bears to the whole mixture. This may be indicated as a range or maximum amount, ie, "10-40% vol" or "10% max wt" to avoid disclosure of trade secrets.

Toxic hazard data shall be stated in terms of concentration, mode of exposure or test, and animal used, ie, "100 ppm LC50-rat," "25 mg/kg LD50-skin-rabbit," "75 ppm LC man," or "permissible exposure from 29 CFR 1910.1000," or if not available, from other sources of publications such as the American Conference of Governmental Industrial Hygienists of the American National Standards Institute, Inc. Flammable or reactive data could be flash point, shock sensitivity, or other brief data indicating nature of the hazard.

(c) Section III. Physical Data

The data in Section III should be for the total mixture and should include the boiling point and melting point in degrees Fahrenheit (Celsius in parentheses); vapor pressure, in conventional millimeters of mercury (mmHg); vapor density of gas or vapor (air = 1); solubility in water, in parts/hundred parts of water by weight; specific gravity (water = 1); percent volatiles (indicated if by weight or volume) at 70 degrees Fahrenheit (21.1 degrees Celsius); evaporation rate for liquids or sublimable solids, relative to butyl acetate; and appearance and odor. These data are useful for the control of toxic substances. Boiling point, vapor density, percent volatiles, vapor pressure, and evaporation are useful for designing proper ventilation equipment. This information is also useful for design and deployment of adequate fire and spill containment equipment. The appearance and odor may facilitate identification of substances stored in improperly marked containers, or when spilled.

(d) Section IV. Fire and Explosion Data

Section IV should contain complete fire and explosion data for the product, including flash point and autoignition temperature in degrees Fahrenheit (Celsius in parentheses); flammable limits, in percent by volume in air; suitable extinguishing media or materials; special firefighting procedures; and unusual fire and explosion hazard information. If the product presents no fire hazard, insert "NO FIRE HAZARD" on the line labeled "Extinguishing Media."

(e) Section V. Health Hazard Information

The "Health Hazard Data" should be a combined estimate of the hazard of the total product. This can be expressed as a TWA concentration, as a permissible exposure, or by some other indication of an acceptable standard. Other data are acceptable, such as lowest LD50 if multiple components are involved.

Under "Routes of Exposure," comments in each category should reflect the potential hazard from absorption by the route in question. Comments should indicate the severity of the effect and the basis for the statement if possible. The basis might be animal studies, analogy with similar products, or human experiences. Comments such as "yes" or "possible" are not helpful. Typical comments might be:

> Skin Contact--single short contact, no adverse effects likely; prolonged or repeated contact, irritation and possibly some blistering.
>
> Eye Contact--some pain and transient irritation; possible corneal scarring.

"Emergency and First Aid Procedures" should be written in lay language and should primarily represent first aid treatment that could be provided by paramedical personnel or individuals trained in first aid.

Information in the "Notes to Physician" section should include any special medical information which would be of assistance to an attending physician including required or recommended preplacement and periodic medical examinations, diagnostic procedures, and medical management of overexposed workers.

(f) Section VI. Reactivity Data

The comments in Section VI relate to safe storage and handling of hazardous, unstable substances. It is particularly important to highlight instability or incompatibility to common substances or circumstances such as water, direct sunlight, steel or copper piping, acids, alkalies, etc. "Hazardous Decomposition Products" shall include those products released under fire conditions. It must also include dangerous products produced by aging, such as peroxides in the case of some ethers. Where applicable, shelf life should also be indicated.

(g) Section VII. Spill or Leak Procedures

Detailed procedures for cleanup and disposal should be listed with emphasis on precautions to be taken to protect workers assigned to cleanup detail. Specific neutralizing chemicals or procedures should be described in detail. Disposal methods should be explicit including proper labeling of containers holding residues and ultimate disposal methods such as "sanitary landfill," or "incineration." Warnings such as "comply with local, state, and federal anti-pollution ordinances" are proper but not sufficient. Specific procedures shall be identified.

(h) Section VIII. Special Protection Information

Section VIII requires specific information. Statements such as "Yes," "No," or "If Necessary" are not informative. Ventilation requirements should be specific as to type and preferred methods. Respirators shall be specified as to type and NIOSH or US Bureau of Mines approval class, ie, "Supplied air," "Organic vapor canister," "Suitable for dusts not more toxic than lead," etc. Protective equipment must be specified as to type and materials of construction.

(i) Section IX. Special Precautions

"Precautionary Statements" shall consist of the label statements selected for use on the container or placard. Additional information on any aspect of safety or health not covered in other sections should be inserted in Section IX. The lower block can contain references to published guides or in-house procedures for handling and storage. Department of Transportation markings and classifications and other freight, handling, or storage requirements and environmental controls can be noted.

(j) Signature and Filing

Finally, the name and address of the responsible person who completed the MSDS and the date of completion are entered. This will facilitate correction of errors and identify a source of additional information.

The MSDS shall be filed in a location readily accessible to workers potentially exposed to the hazardous material. The MSDS can be used as a training aid and basis for discussion during safety meetings and training of new employees. It should assist management by directing attention to the need for specific control engineering, work practices, and protective measures to ensure safe handling and use of the material. It will aid the safety and health staff in planning a safe and healthful work environment and in suggesting appropriate emergency procedures and sources of help in the event of harmful exposure of employees.

MATERIAL SAFETY DATA SHEET

I PRODUCT IDENTIFICATION

MANUFACTURER'S NAME	REGULAR TELEPHONE NO. EMERGENCY TELEPHONE NO.
ADDRESS	
TRADE NAME	
SYNONYMS	

II HAZARDOUS INGREDIENTS

MATERIAL OR COMPONENT	%	HAZARD DATA

III PHYSICAL DATA

BOILING POINT, 760 MM HG		MELTING POINT
SPECIFIC GRAVITY (H_2O=1)		VAPOR PRESSURE
VAPOR DENSITY (AIR=1)		SOLUBILITY IN H_2O, % BY WT.
% VOLATILES BY VOL.		EVAPORATION RATE (BUTYL ACETATE=1)
APPEARANCE AND ODOR		

IV FIRE AND EXPLOSION DATA

FLASH POINT (TEST METHOD)		AUTOIGNITION TEMPERATURE	

FLAMMABLE LIMITS IN AIR, % BY VOL.	LOWER		UPPER	

EXTINGUISHING MEDIA	
SPECIAL FIRE FIGHTING PROCEDURES	
UNUSUAL FIRE AND EXPLOSION HAZARD	

V HEALTH HAZARD INFORMATION

HEALTH HAZARD DATA

ROUTES OF EXPOSURE

INHALATION

SKIN CONTACT

SKIN ABSORPTION

EYE CONTACT

INGESTION

EFFECTS OF OVEREXPOSURE

ACUTE OVEREXPOSURE

CHRONIC OVEREXPOSURE

EMERGENCY AND FIRST AID PROCEDURES

EYES:

SKIN:

INHALATION:

INGESTION:

NOTES TO PHYSICIAN

VI REACTIVITY DATA

CONDITIONS CONTRIBUTING TO INSTABILITY

INCOMPATIBILITY

HAZARDOUS DECOMPOSITION PRODUCTS

CONDITIONS CONTRIBUTING TO HAZARDOUS POLYMERIZATION

VII SPILL OR LEAK PROCEDURES

STEPS TO BE TAKEN IF MATERIAL IS RELEASED OR SPILLED

NEUTRALIZING CHEMICALS

WASTE DISPOSAL METHOD

VIII SPECIAL PROTECTION INFORMATION

VENTILATION REQUIREMENTS

SPECIFIC PERSONAL PROTECTIVE EQUIPMENT

RESPIRATORY (SPECIFY IN DETAIL)

EYE

GLOVES

OTHER CLOTHING AND EQUIPMENT

IX SPECIAL PRECAUTIONS

PRECAUTIONARY STATEMENTS

OTHER HANDLING AND STORAGE REQUIREMENTS

PREPARED BY:

ADDRESS:

DATE:

XII. TABLES AND FIGURE

TABLE XII-1

CONSUMPTION OF FLUORSPAR IN THE UNITED STATES IN 1971

End use or product	Short Tons of Fluorspar Consumed	Percentage of Total Fluorspar Consumed
Hydrofluoric acid	701,844	52.0
Glass	12,875	1.0
Enamel	2,258	0.2
Welding rod coatings	1,043	0.1
Primary aluminum	938	0.1
Primary magnesium	22,260	1.7
Other nonferrous metals	10,715	0.8
Iron and steel castings	31,565	2.4
Open hearth furnaces	82,133	6.1
Basic oxygen furnaces	377,266	28.0
Electric furnaces	81,511	6.1
Other uses or products	20,334	1.5
TOTAL	1344,742	100.0

From Wood [12]

TABLE XII-2

OCCUPATIONS WITH POTENTIAL EXPOSURE TO FLUORIDES

Abrasive wheel makers
Adhesive makers
Aluminum anodizers
Aluminum makers
Aluminum refiners
Aluminum solderers
Aluminum welders
Apatite workers
Bactericide workers
Beryllium refiners
Brewers
Building workers
Carbon electrode workers
Cement workers
Ceramic workers
Chemical manufacturers
Chemical polisher workers
Chlorofluorocarbon makers
Concreters
Construction workers
Copper refiners
Cryolite makers
Dentifrice makers
Diazosalt makers
Disinfectors
Electric arc welders
Electric arc workers
Electroplaters
Embalming fluid workers
Fertilizer makers
Fluoride workers
Fluorocarbon makers
Fluorspar workers
Flux makers
Foundry workers
Fruit workers
Fungicide workers
Glass etchers
Gold refiners
Grinding wheel makers
Hide and skin preservers
Hydrofluoric acid makers
Insect exterminators
Insecticide makers
Latex foam rubber workers
Laundry workers
Lead electroplaters
Lead smelters
Lenscoaters
Light metal casters
Magnesium foundry workers
Marble hardeners
Masonry preservers
Metal cleaners
Metal coating workers
Metal refiners
Mica makers
Miners (fluorspar, phosphate rock)
Mothproofers
Opal glass makers
Open hearth steel workers
Optical equipment makers
Ore flotation workers
Paint workers
Petroleum refinery workers
Phosphorescent tube makers
Phosphoric acid makers
Phosphorus makers
Photoengravers
Porcelain enamel workers
Radiation dosimeter makers
Rock phosphate acidulators
Rodent exterminators
Rubber coagulators
Sandy soil treaters
Silver refiners
Silver solder flux workers
Sinterers
Soil improvers
Stainless steel welding rod users
Submerged arc welders
Superphosphate makers
Synthetic stone makers
Tantalum refiners
Textile mordanters
Vegetable growers
Vitreous enamel workers
Water treaters
Welding rod makers
Wood preservative workers
Wood preservers and stainers
Zinc miners

Adapted from Gafafer and Greenwood [258,259]

TABLE XII-3

PROPERTIES OF REPRESENTATIVE FLUORIDES

Name and Formula	Formula Weight	Density g/cc	Melting Point Degrees C	Boiling Point Degrees C	Solubility, g/100 cc H2O	
					cold	hot
Aluminum fluoride, AlF3	83.98	2.882	sublimes		0.559	soluble
Aluminum fluosilicate (topaz) 2AlFO.SiO2	184.04	3.58			insoluble	insoluble
Ammonium fluoborate, NH4BF4	104.84	1.871	sublimes		25	97
Ammonium fluoride, NH4F	37.04	1.009	sublimes		100	decomposes
Ammonium fluosilicate (alpha cryptohalite), (NH4)2SiF6	178.14	2.011	decomposes		18.6	55.5
Ammonium hydrogen fluoride, NH4HF2	57.04	1.50	125.6		v.s.*	v.s.*
Calcium fluoride (fluorspar), CaF2	78.08	3.180	1360	c 2500	0.0016	0.0017
Calcium fluosilicate, CaSiF6	182.16	2.66			sl.s.**	
Cesium fluoride, CsF	151.90	4.115	682	1251	367	
Fluorapatite (phosphate rock), Ca10(PO4)6F2					sl.s.**	
Hydrogen fluoride, HF	20.01	0.991(Sp.G.)	-83.1	19.54	sol. in all proportions	v.s.*
Lithium fluoride, LiF	25.94	2.635	842	1676	0.27	
Magnesium fluoride, MgF2	62.31		1266	2239	0.0076	insoluble
Magnesium fluosilicate, MgSiF6	166.39				65	
Potassium fluoborate, KBF4	125.91	2.498	decomposes 350	decomposes	0.44	6.27
Potassium fluoride, KF	58.10	2.48	846	1505	92.3	v.s.*

TABLE XII-3 (continued)

PROPERTIES OF REPRESENTATIVE FLUORIDES

Name and Formula	Formula Weight	Density g/cc	Melting Point Degrees C	Boiling Point Degrees C	Solubility, g/100 cc H2O	
					cold	hot
Potassium fluosilicate, K2SiF6	220.25	2.665	decomposes		6.9	
Potassium fluotantalate, K2TaF7	392.14	4.56			sl.s.** decomposes	
Potassium fluotitanate, K2TiF6.H2O	258.11		780	decomposes	0.556	1.27
Potassium fluozirconate, K2ZrF6	283.41	3.48			0.781	25
Potassium hydrogen fluoride, KHF2	78.11	2.37	decomposes ca 225	decomposes	41	v.s.*
Rubidium fluoride, RbF	104.47	3.557	775	1410	130.6	
Sodium aluminum fluoride (cryolite), 3NaF.AlF3	209.94	2.90	1000		sl.s.**	
Sodium fluoborate, NaBF4	109.79	2.47	slowly decomposes 384	decomposes	108	210
Sodium fluoride, NaF	41.99	2.558	988	1695	4.22	
Sodium fluosilicate, Na2SiF6	188.06	2.679	decomposes		0.652	2.46
Sodium hydrogen fluoride, NaHF2	61.99	2.08			soluble	soluble
Strontium fluoride, SrF2	125.62	4.24	1450+	2489	0.011	0.012
Zinc fluoride, ZnF2	103.37	4.95	872	1500	1.62	soluble

* v.s. = very soluble   ** sl.s. = slightly soluble

From Weast [260]

TABLE XII-4

FLUORIDE CONTENT OF FRESH FOOD AND BEVERAGE, MILLIGRAMS PER KILOGRAM*

Reference and year		Fruit	Vegetable	Meat excl Bone	Breads & Grain	Fish	Black Tea
[18]	1937		0.01-5.9				
[21]	1939	0.21-0.70	0.08-1.34	0.20-1.67	0.82-1.33	1.58-1.63	
[24]	1949	0.00-1.32	0.08-6.4	<0.02-3.3	<0.1-0.7	<0.2-26.9	7.8-398.8
[19]	1959	0.02-1.32	0.1-3.0	0.01-7.7	<0.1-20.0	0.1-24.0	3.2-4.00
[20]	1970	0.03-0.84	0.02-0.9	0.14-2.0	0.18-2.8	0.05-19.0	0.1-2.0
[261]	1970	0.07-0.72	0.06-1.05 (polished peas, 14.06)	0.19-0.51	0.04-0.24 (polished rice, 10.67)	1.37-5.21	10.08-10.78

---

* The variability of the given values may be explained by the variability in F content among different plant and animal species and/or the differences in analytical techniques.

TABLE XII-5

F LEVELS IN BLOOD--MICROGRAMS PER 100 MILLILITERS

Author	Year	Values	Number of Tests	Method	Subjects' Exposure
Smith et al [52]	1950	0.33	34	Smith & Gardner	0.06 in water
		1.38	20		
		3.93	12		1.0-1.2
Singer & Armstrong [262]	1960	14	16	Singer & Armstrong	0.15 in water
		15	18		1.1 "
		19	36		1.1 "
		16	26		2.5 "
		26	22		5.4 "
Armstrong et al [263]	1964	79	5	Singer & Armstrong	50-100 mg F/day therapeutically
		85			
		65			
		177			
		45			
		48			
		23			
		34			
		30			
		25			
		27			
		32			
		28			
Taves [53]	1966	1.3	16	modified diffusion process of Singer & Armstrong	about 1 mg F/liter in water
Armstrong et al [57]	1970	11	12-16	Singer & Armstrong	
Hall et al [54]	1972	3.7	26	ion-selective electrode	about 1 mg F/liter in water

TABLE XII-6

FLUORIDE IN BONE

Subjects	Age	F in Water ppm	Years in Area	Bone Site	F in bone (mg/100 g) dry, fat-free	F in bone (mg/100 g) ash
New York City resident		0.0		rib		10
Washington, D.C., resident		0.0		humerus		100
Washington, D.C. resident		0.0		tibia		20
Washington, D.C. resident [64]		0.0		toe		160
Autopsy; most deaths sudden; 33 subjects [60]	55.5 (SE 2.8)	0.1-0.4	10-87	vertebra	54 (SE*5)	
				rib	50 (SE 4)	
				iliac crest	41 (SE 3)	
Longterm resident [62]	74	0.2	24	lumbar vertebra	77	177
				calvarium	92	135
				iliac crest	78	135
In counties with industrial ¯; 90 subjects [61]	15+	<0.5	10+	vertebrae	61.5	
				iliac crest	51.0	
				rib	53.7	
Autopsy; most deaths sudden; 5 subjects [60]	75.6 (SE 1.5)	1.0	10+	vertebra	160 (SE 2)	
				rib	139 (SE 2)	
				iliac crest	138 (SE 1.6)	
11 subjects [18]	33-80			rib	93 average 48-210	
Colorado resident		2.0		rib	300	
" "				rib	300	
" "				rib	350	
" [64]				rib	120	

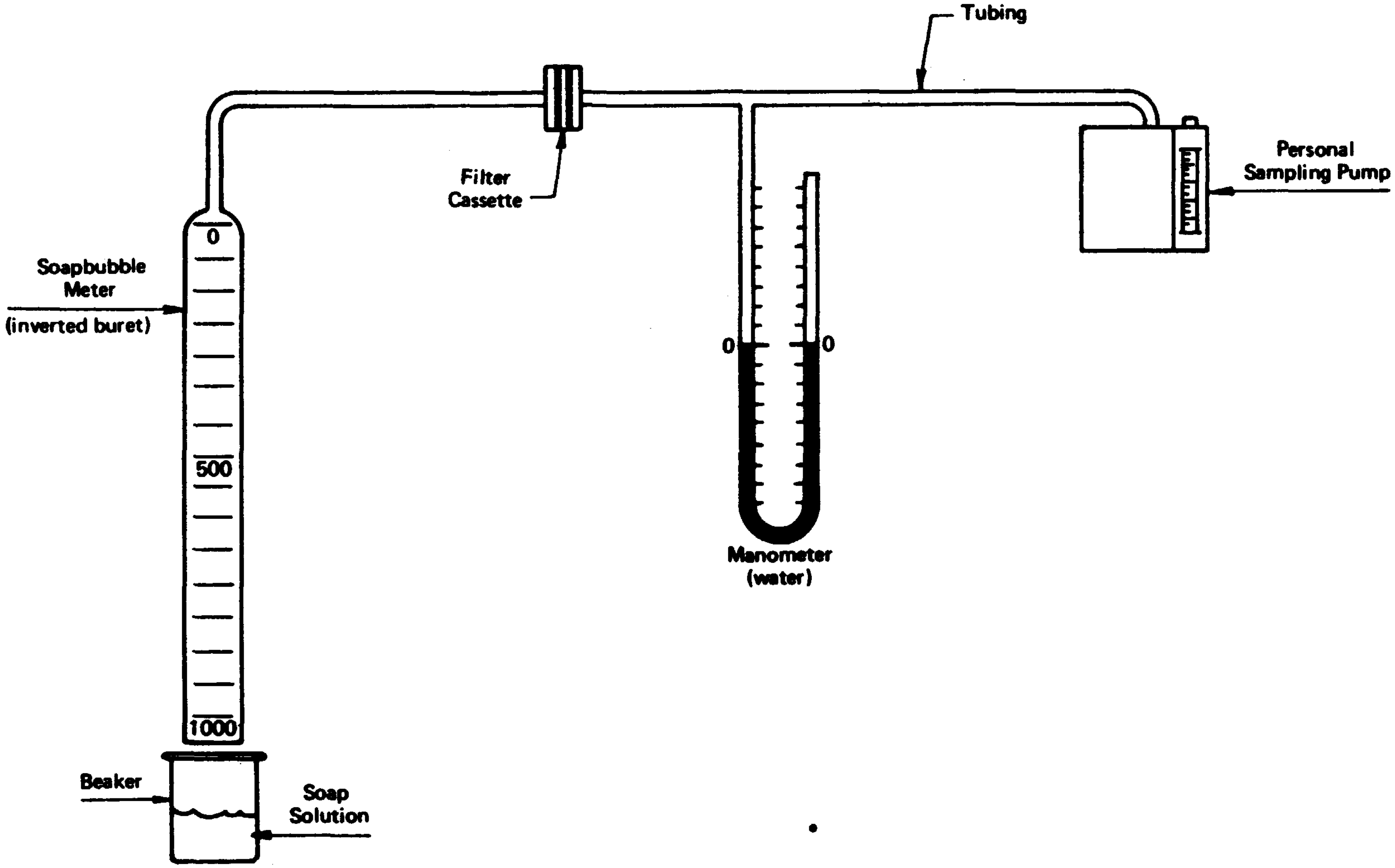
TABLE XII-6 (continued)

FLUORIDE IN BONE

Subjects	Age	F in Water (ppm)	Years in Area	Bone Site	F in bone (mg/100 g)	
					dry, fat-free	ash
27 subjects [60]	65.9 (SE 3.0)	2.6	10-76	vertebra	286 (SE 2.3)	
				rib	257 (SE 2)	
				iliac crest	257 (SE 2.3)	
4 subjects [60]	56 (SE 11.0)	4.0	10-48	vertebra	411 (SE 6.8)	
				rib	398 (SE 4.0)	
				iliac crest	13 (SE 5.3)	
Long-term resident [62]	78	8	34	lumbar vertebra	550	913
				calvarium	653	914
China resident [65]	37	2.4& 13.1	life	lumbar vertebra	1175	1865
				skull	1502	2209
				rib	1355	2083
				ilium	1124	1756
				calcaneum	1322	2066
London residents [63]		under 0.5		rib	24	
	1			"	33	
	2			"	69	115
	15			"	88	174
	22			"	180	397
	25			"	61	
	27			"	280	591
	32			"	89	194
	37			"	310	687
	37			"	150	280
	54			"	230	
	59			"	90	186
	60			"	230	473

*Standard Error

FIGURE XII - 1. CALIBRATION SETUP FOR PERSONAL SAMPLING PUMP WITH FILTER CASSETTE



☆ U.S. GOVERNMENT PRINTING OFFICE: 1976 - 657-696/5522 Region No.5-II

DEPARTMENT OF
HEALTH, EDUCATION, AND WELFARE
PUBLIC HEALTH SERVICE
CENTER FOR DISEASE CONTROL
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH
RM. 532, U. S. POST OFFICE, CINCINNATI, OHIO 45202

OFFICIAL BUSINESS
PENALTY FOR PRIVATE USE, $300

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF H.E.W.
HEW 399

U.S.MAIL

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE
Public Health Service
Center for Disease Control
National Institute for Occupational
Safety and Health

HEW Publication No. (NIOSH) 76-103