National Institute for Occupational Safety and Health
Announcement of Findings

Portsmouth GDP Edition

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Glossary of Terms

Cohort: A group of persons identified by common characteristics who are studied over a period of time.

External Radiation: Exposure which was measured by personal dosimeters worn on the outside of workers clothing.

Internal Radiation: Exposure to radioactive materials that have been inhaled or ingested by workers which was determined from the number of alpha counts in worker urine samples.

Healthy Worker Effect: Fewer deaths are observed for workers when compared to the U.S. population; usually due to the employment of healthy workers and the exclusion of the severely ill and chronically disabled from employment.

Hematopoietic: The system in the human body that forms blood. Diseases of this system include leukemias and multiple myeloma among others.

Dose Response: A relationship in which a change in the amount, intensity, or duration of an exposure is associated with either an increase or a decrease in risk of a specified health outcome.

Mortality Patterns of Uranium Enrichment Workers at the Portsmouth Gaseous Diffusion Plant

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Study Population: A total of 8,877 workers (both sexes and all races) employed for at least one day at Portsmouth Gaseous Diffusion Plant (PORTS) between September 1, 1954 and December 31, 1991.

How This Study Was Done: This epidemiologic study examined the causes of deaths among all PORTS workers employed by the facility between September 1, 1954 and December 31, 1991. Deaths among the workers were compared with rates for the general U.S. population. Possible relationships were evaluated for deaths from several types of cancer and exposures to ionizing radiation and certain chemicals (fluoride, uranium metal, and nickel). Based upon previous health studies of nuclear facility workers, including an earlier NIOSH investigation at PORTS, deaths from cancers of the stomach, lung, and the lymphatic and the hematopoietic systems including leukemia, were evaluated in more detail. The study report and findings were reviewed by experts outside NIOSH.

Study Findings: Approximately 88% of the cohort was still alive through the end of 1991. Overall cohort mortality was significantly less than expected, when compared to the United States population, as was mortality from all cancers. A total of 1,088 deaths from all causes occurred in this cohort through 1991. A total of 1,518 deaths could be expected based upon rates in the general U.S. population. The lower mortality among these workers is consistent with the healthy worker effect which is found in most occupational epidemiologic studies.

No statistically significant excesses in mortality from any specific cause were identified. Analyses of possible relationships between causes of death and the identified exposures failed to reveal any dose-response trends. For leukemia, no effect of cumulative exposure to either external or internal radiation was identified. Additionally, no dose-response relationships were observed for cancers of the stomach, lung, Hodgkin's disease, lymphoreticulosarcoma, and all cancers combined.

Further NIOSH Information:

- For a summary of NIOSH research involving Department of Energy workers, visit online at: www.cdc.gov/niosh/2001-133.html

DOE Compensation Information:

- Questions regarding the DOE compensation program may be directed (toll free) to the DOE Office of Worker Advocacy at : (877) 447-9756, www.eh.doe.gov/advocacy
- Questions may also be directed (toll free) to the Department of Labor's DOE Compensation Initiative Office at: (866) 888-3322, www.dol.gov

This study was conducted by researchers at the National Institute for Occupational Safety and Health.

Worker deaths from cancers of the lympho-hematopoietic tissue, including leukemia, equaled U.S. rates. Stomach cancer deaths were greater than expected (12.7 deaths expected, 15 deaths found) but this difference was not statistically significant. Deaths from these cancers had been found to be slightly elevated in a previous NIOSH study of PORTS.

Study Limitations:

- The young average age of this cohort with 88% still living in 1991 made it difficult to fully assess patterns of death associated with work exposures.
- Estimated radiation and chemical exposures developed for this study are subject to error since monitoring results for chemicals and various forms of radiation were incomplete.
- Potentially important factors that may have an effect on the observed outcome, such as lifestyle factors (e.g., smoking), radiation due to medical procedures, and other workplace exposures could not be evaluated.

Study Advantages:

- A comprehensive exposure assessment was conducted, making use of nearly all health physics and industrial hygiene data from 1954 through 1991.
- The patterns of exposures used in this study were evaluated by experienced workers at the site. Analysis with these estimates increased the ability to detect associations between exposures and death.
- This study design was a good tool for evaluating the causes of death in workers with chronic low-level radiation exposures.

Important Announcements

NIOSH researchers will discuss study findings in a live video presentation from the Portsmouth Gaseous Diffusion Plant to the Paducah and Oak Ridge facilities on October 4, 2001, at 2:00 p.m., EDT. Broadcast of this presentation will be held at the Building X-100 Videoconference Room at the PORTS site. A second presentation at PORTS will be held from 6:30-8:30 p.m. at the Vern Riffe Vocational School. The presentation will be videotaped and made available at each of the sites. For more information please contact the Department of Energy (DOE) representative, Mr. Walter Perry at (865) 576-0885.

Questions concerning this study should be directed to NIOSH at (513) 841-4400. For a copy of the abstract or final technical report for this study, call 1-800-356-4674.

NIOSH/HERB Contact Points for Further Information...

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FINAL REPORT

MORTALITY PATTERNS AMONG URANIUM ENRICHMENT WORKERS at the PORTSMOUTH GASEOUS DIFFUSION PLANT PIKETON, OHIO

PREPARED BY THE STAFF OF
THE HEALTH-RELATED ENERGY RESEARCH BRANCH
DIVISION OF SURVEILLANCE, HAZARD EVALUATIONS, AND FIELD STUDIES
NATIONAL INSTITUTE FOR OCCUPATIONAL SAFETY AND HEALTH

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
CENTERS FOR DISEASE CONTROL AND PREVENTION

July, 2001

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EXECUTIVE SUMMARY

In 1992, the National Institute for Occupational Safety and Health (NIOSH) received a Congressional request to conduct an update study of mortality at the Portsmouth Gaseous Diffusion Plant in Piketon, Ohio. The plant enriches uranium by increasing the proportion of the uranium-235 isotope (^{235}U) using a gaseous diffusion process.

This report presents the methods and findings of the study. Specifically, this report addresses: 1) the use of available employment records to assemble a cohort of gaseous diffusion workers; 2) the use of existing health physics and industrial hygiene data to reconstruct past occupational exposures to selected physical and chemical agents; 3) a survey of electromagnetic field exposures to the contemporary workforce; 4) the overall mortality experience of the workers compared to that of the general population; 5) the cause-specific mortality experience of the workers by degree of occupational exposures using: a) individually measured alpha count activity in urine (resulting from exposure to soluble uranium), b) personal dosimetry for exposure to external (gamma and X ray) ionizing radiation, and c) estimated historical occupational exposures to chemical compounds of uranium, nickel, and fluorine.

Overall mortality at the facility was significantly less than expected [SMR = 0.72; 95% confidence interval (CI) = 0.67 - 0.76] as was mortality from “All Cancers” [SMR = 0.82; CI = 0.73 - 0.92]. Statistically non-significant excesses of mortality were found for some specific

causes of death including cancers of the stomach and of the lympho-hematopoietic tissue, both of which had been noted in an earlier NIOSH study at this facility.

Epidemiologic evaluation of the data to detect trends in risk of death after radiation/chemical exposures was performed in nested case control and sub-cohort analyses. Analyses of possible relationships between cause of death and external radiation produced the same results as those resulting from the analysis performed upon the full cohort. A statistically non-significant elevated SMR for cancer of the stomach was present similar to that observed in the overall analysis. Non-significant excesses of deaths were observed due to diseases of the arteries, veins and pulmonary circulation; and due to diseases of the musculoskeletal system and connective tissue. No dose-response relationships were observed for cancers of the stomach, lung, Hodgkin's disease, lymphoreticulosarcoma, and all cancers combined.

The analyses of possible relationships between cause of death and internal radiation exposure produced essentially the same results observed in the total cohort and for external radiation. Analyses of possible relationships between cause of death and exposures to fluorine and fluoride compounds, uranium metal, and nickel for the respective sub-cohorts working in departments where airborne concentrations for these agents were measured failed to reveal any dose-response relationships.

Temporal effects, effect modification and interaction, confounding by concomitant occupational exposures and risk factors such as age, race and gender were also examined. No patterns were identified for any of these exposures or factors. Statistically non-significant excesses of deaths due to cancers of the stomach were again noted in the sub-cohorts restricted to departments with measured airborne concentrations of fluorine and fluoride compounds and metallic uranium.

Evidence for and against associations of mortality and occupational factors were evaluated and are presented and discussed. No statistically significant excesses in mortality were identified even though extensive efforts were taken to utilize contaminant exposure information, latency periods, and appropriate comparison groups.

Reasons for the lower than expected mortality experienced by this cohort and the limitations of the study are discussed. Recommendations are made for future studies at the site. The limitations of exposure monitoring data collected for compliance purposes in epidemiologic studies are discussed. Recommendations are made to expand exposure monitoring so that future epidemiologic study results can be more specific. These recommendations include more inclusive monitoring programs based on statistical sampling principles and more complete documentation of reasons for monitoring certain areas or groups of workers.

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Glossary of Abbreviations, Symbols, and Acronyms

AEC	Atomic Energy Commission (predecessor of the Department of Energy)
BEIR	Committee reports on the Biological Effects of Ionizing Radiation. Board on Radiation Effects Research, Commission on Life Sciences, National Research Council. National Academy of Sciences
ClF_3	Chlorine Trifluoride
CIP/CUP	Cascade Improvement Program/Cascade Uprating Program
Co	Cobalt
Cs	Cesium
DHHS	Department of Health and Human Services
DOE/EM	Department of Energy/Environmental Management
DOE	Department of Energy
dpm	Disintegrations per minute
dpm-years	Disintegrations per minute - years
EGG	EG&G, Inc. A contractor to the Department of Energy
EM	Exposure Matrix
ERDA	Energy Research Development Administration
F_2	Fluorine
FFC	Fluorine and fluoride compounds
GAT	Goodyear Atomic Corporation

GCEP	Gaseous Centrifuge Enrichment Plant in Piketon, Ohio
GLM	General Linear Model. SAS Institute
HASL	The Health and Safety Laboratory of the AEC New York Operations Office (now the DOE Environmental Measurements Laboratory)
HF	Hydrogen fluoride
ICD	International Classification of Diseases
IH	Industrial Hygiene
LOD	Limit of detection
LOD/2	Limit of detection divided by two
LTAS	Life Table Analysis System. The NIOSH computer program written to calculate the expected numbers of cause specific deaths.
mg	milligrams
ml	milliliter
MMES	Martin Marietta Energy Systems
NDI	National Death Index, National Center for Health Statistics
NIOSH	National Institute for Occupational Safety and Health
OCAW	Oil Chemical and Atomic Workers. Predecessor of Paper, Allied-Industrial, Chemical and Energy Workers International Union [PACE]
ORAU	Oak Ridge Associated Universities
PDF	Personal dose file
PGDP	Paducah Gaseous Diffusion Plant in Paducah, Kentucky
PORTS	Portsmouth Gaseous Diffusion Plant in Piketon, Ohio

ppm	Parts per million
PYAR	Person-years at risk [of dying]
R/hr	Roentgens per hour
Ra	Radium
REIRS	Radiological Exposure Information Retrieval System
SEG	Similar Exposure Group
SMR	Standardized Mortality Ratio
SSA	Social Security Administration
UF_4	Uranium tetrafluoride (Green Salt)
UF_6	Uranium hexafluoride
UO_2F_2	Uranyl fluoride
USEC	United States Enrichment Corporation
USUR	United States Uranium Registry
^{x}U	An isotope of uranium. *x* specifies the atomic mass.

INTRODUCTION

In 1979, the Oil, Chemical, and Atomic Workers Union (OCAW)[1], requested that the National Institute for Occupational Safety and Health (NIOSH) evaluate the potential health effects from occupational exposures at the Portsmouth Gaseous Diffusion Plant (PORTS) in Piketon, Ohio [Metzenbaum 1979]. Specifically, OCAW was concerned that exposure to uranium hexafluoride (UF_6) may have caused cancers among production and maintenance workers.

In 1982, following a number of site visits and workplace environmental surveys, NIOSH initiated a cohort study to determine whether mortality was associated with occupational exposures at PORTS [NIOSH 1987a,b]. A report on the study findings, issued in January 1987, revealed statistically non-significant elevated standardized mortality ratios (SMRs) for stomach cancer (SMR = 1.69) and for malignancies of the lymphatic and hematopoietic system (SMR = 1.46) in a cohort that overall displayed a less than expected total cancer mortality experience. Workers at PORTS and at a similar gaseous diffusion plant in Paducah, Kentucky continued to voice concerns, especially about leukemia risks. In 1992, NIOSH was asked by Senator John Glenn (Ohio) to update the cohort mortality study [Glenn 1992]. That same year, the Health-Related Energy Research Branch at NIOSH, working under a Memorandum of Understanding established between Department of Health and Human Services (DHHS) and the Department of Energy

[1]OCAW is now The Paper, Allied-Industrial, Chemical and Energy Workers International Union.

(DOE), commenced a study to update the mortality experience of this workforce through December 1991.

This report presents the methods and findings of the NIOSH investigation. Specifically the report addresses:

1. The use of available employment records to assemble a cohort of PORTS workers.
2. The use of existing health physics and industrial hygiene data to reconstruct past occupational exposures to selected physical and chemical agents.
3. The overall mortality experience of the PORTS workers compared to that of the general population.
4. The cause-specific mortality experience of the PORTS workers by type of exposure using:
   a. estimated personal alpha count activity in urine (resulting from exposure to soluble uranium),
   b. personal dosimetry for exposure to external (gamma and X ray) radiation, and
   c. estimated historical occupational exposures to airborne uranium, nickel, fluorine compounds, and to electromagnetic fields.
5. The existence of exposure-response relationships between specific physical and chemical exposures and certain causes of death (stomach cancer, malignancies of the lymphatic and hematopoietic system) of special *a priori* interest.

The Uranium Industry

The nuclear fuel cycle in the U.S. has been previously described by the United States Uranium Registry [USUR 1981]. The industry consists of uranium mines, mills, conversion plants, and

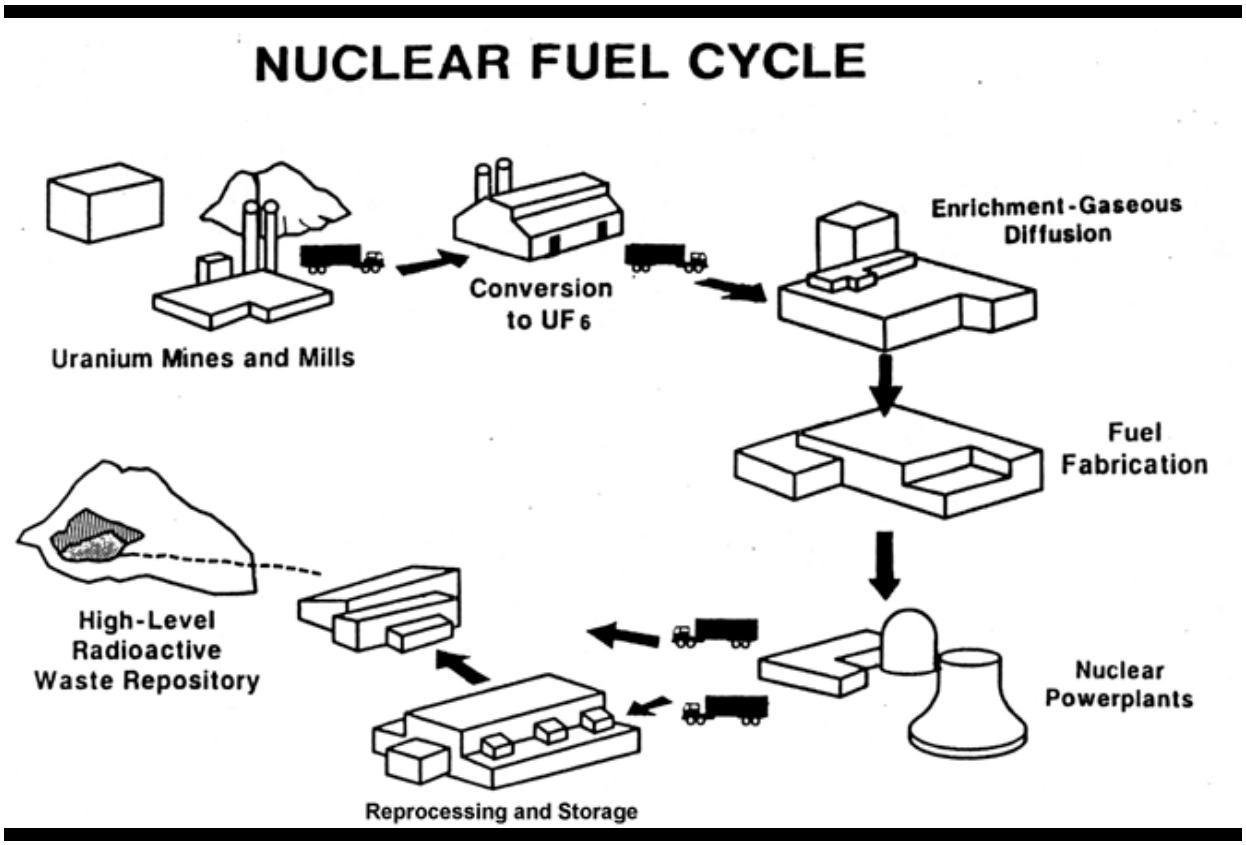


Figure 1: Nuclear Fuel Cycle *Source: Adapted from Health Physics Society: Science Teachers Workshop Material*

enrichment plants (Figure 1).

The role of PORTS in the uranium industry has been to produce uranium enriched in the uranium-235 isotope (^{235}U) . Enriched uranium is fissionable and is used by the commercial

nuclear power industry and the U.S. Department of Defense in its military nuclear programs, e.g., nuclear weapons and fuel for naval nuclear vessels.

PROCESS AND FACILITY

Uranium is a naturally occurring radioactive element consisting of three isotopes: ^{238}U (99.276%), ^{235}U (0.719%) and a trace amount of ^{234}U (0.0057%) [Schleien 1992]. The objective of the enrichment process is to take naturally-occurring uranium and convert it to a product with an increased proportion of fissionable ^{235}U. In 1943, construction began on the first gaseous diffusion plant (code named K-25) in Oak Ridge, Tennessee. Production of enriched uranium started in 1945 [DOE 1978] and continued until 1985. Two other plants, the Paducah Gaseous Diffusion Plant (PGDP) in Paducah, Kentucky, and the Portsmouth Gaseous Diffusion Plant (PORTS) in Piketon, Ohio, were constructed in the early 1950's. Production of enriched uranium began in 1953 and 1954, respectively, and continues to date [AEC 1968; ERDA 1977].

PORTS is located in Pike County, Ohio, on a 4000 acre federally-owned reservation. It was a DOE government-owned, contractor-operated (Martin Marietta Energy Systems [MMES]) facility during the time period covered by this investigation. The plant consists of 109 permanent buildings, containing over 10 million square feet of floor area (Figure 2) [MMES 1992].

Figure 2: Portsmouth Gaseous Diffusion Plant
Source: Linking Legacies; DOE/EM-0319; January 1997

From inception until November 16, 1986, PORTS was operated by the Goodyear Atomic Corporation (GAT), a subsidiary of Goodyear Tire and Rubber Company, under contract with the U.S. government. On November 16, 1986, the management of PORTS was transferred to Martin Marietta Energy Systems, Inc. (MMES), which remained the primary contractor through the study end date (December 31, 1991).

PORTS can process naturally-occurring uranium supplied by uranium mills or partially enriched uranium (up to three percent ^{235}U) supplied by the PGDP [Bostock 1988]. The uranium is further enriched at PORTS to an assay of 3-5% ^{235}U for use in commercial nuclear power plants and may be enriched to greater than 98% ^{235}U for use in nuclear weapons and as fuel for nuclear reactors powering naval vessels. This is the only facility in the U.S. that has the capability of enriching uranium to these very high proportions of ^{235}U [MMES 1985a].

Uranium enrichment is an iterative physical process [AEC 1968]. Uranium is combined with fluorine and heated to make uranium hexafluoride gas (UF_6). PORTS receives its feed material already in the form of UF_6. Uranium hexafluoride is a solid at room temperature which necessitates the operation of the process at temperatures and pressures that maintain the uranium hexafluoride in the gaseous state. The gaseous diffusion process begins when the UF_6 is forced through barrier tubes contained inside the converter, a component of the individual separation units referred to as a stage. A stage consists of a motor, compressor, and the converter which

contains the barrier tubes (Figure 3) [DOE 1978]. The stages are grouped into functional units called cells for the provision of auxiliary services and maintenance purposes [ERDA 1977].

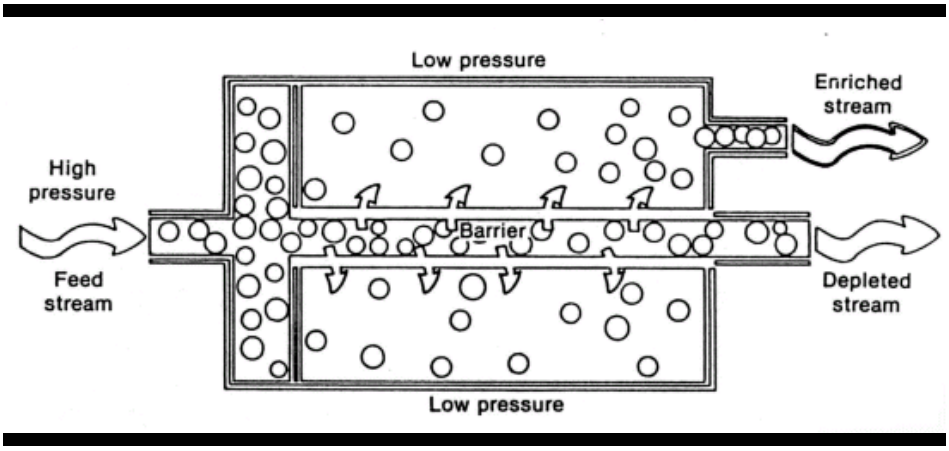


Figure 3: Gaseous Diffusion Stage
Source: DOE EGG-2530; Health Physics Manual of Good Practices for Uranium Facilities, 1988.

Various contaminants and light gases have entered the process from a variety of sources over time. (Purge stages that separate and purge the light gas contaminants that leak into the system are also a part of the cascade process.) Acidic gases (e.g., fluorine, chlorine trifluoride, etc.) enter the cascade through process equipment maintenance activities. Chlorofluorocarbons from the cascade cooling system equipment also leak into the process stream. The presence of contaminants (including transuranics) in the process feed materials and also originating as a part of the decay series of uranium contribute to the chemical and radiological agents present within the cascade process equipment.

Compressors, driven by electric motors, compress the process gas and maintain its flow through the cascade. When the gas contacts the barrier, the $^{235}UF_6$ molecules pass through more readily

than the $^{238}UF_6$ due to their lower mass and higher velocity. The theoretical maximum separation that can be achieved through the barrier is equal to the square root of the ratio of the weight of the gas molecules. Since the square root of that ratio for UF_6 molecules is 1.0043, thousands of separation stages are connected in series, or cascaded, to reach the desired enrichment [MMES 1985a] (Figure 4).

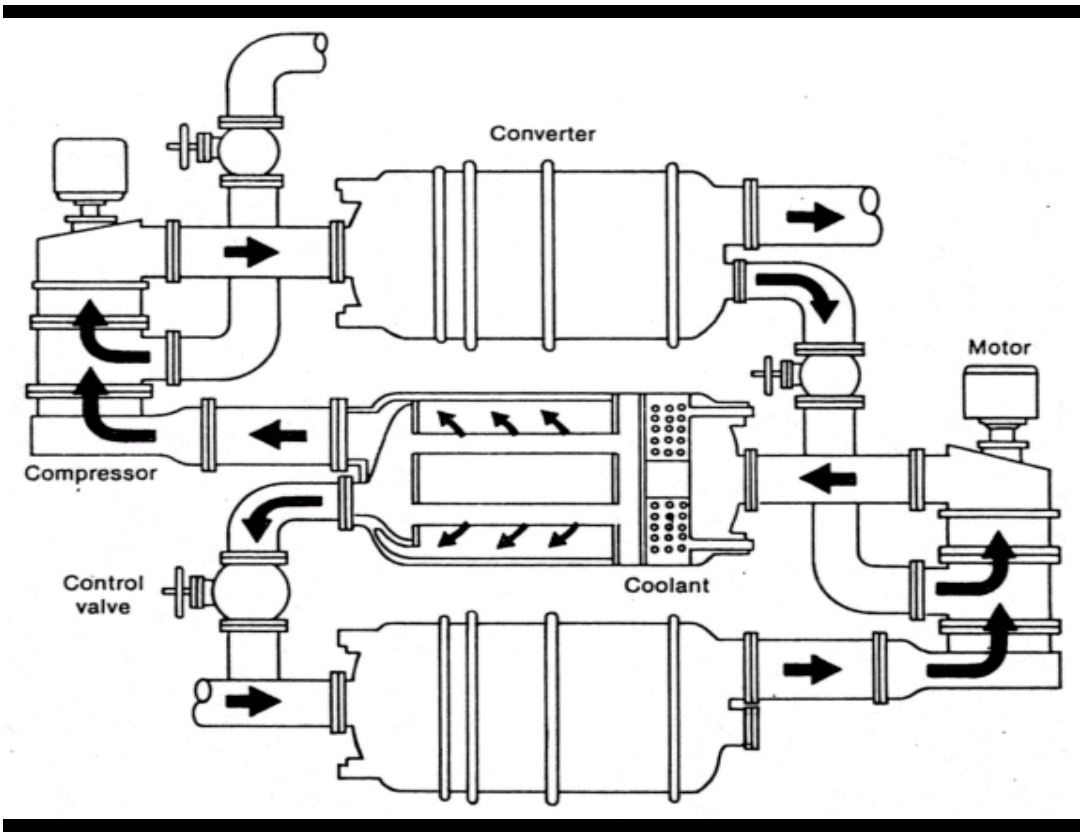


Figure 4: Stage Arrangement

Source: DOE EGG-2530; Health Physic Manual of Good Practices for Uranium Facilities, 1988.

The gaseous diffusion enrichment process is housed in three large process buildings: X-333, X-330, and X-326. These transite sided buildings contain about 8 million square feet of floor

space. In the past, 4,080 stages of compressors and converters were housed in these buildings [AEC 1968]. Because of a reduced demand for highly enriched uranium, the number had been reduced to 2,820 as of the study end date. The process equipment is operated as a closed system. Internal process equipment pressures are maintained below atmospheric pressure for large portions of the system. Heat from the process gas is removed by stage coolers utilizing Freon™ 114 which subsequently transfers the heat to a recirculating water system. The chemical properties of UF_6 including its extreme reactivity with water, corrosivity to most metals, and its incompatibility with organic compounds influences process design and the materials that may be used. The materials used in the construction of the process equipment are primarily nickel-plated steel, Monel™, and aluminum. Hydrogen fluoride has been used for conditioning and treatment of process equipment [ERDA 1977].

Releases of UF_6 into the atmosphere result in the production of uranyl fluoride (UO_2F_2) and HF upon reaction with water vapor. Nitrogen or dry-air buffer systems are used to isolate locations where leakage into or out of the process stream is most likely. The pressure in the buffer systems is higher than that of the process gas. Each process unit also has its own lubrication system that continuously supplies oil to the motor and compressor bearings. Alumina is used in the cold recovery traps. Transformers located in the buildings are Askarel™ filled [ERDA 1977].

PORTS underwent a major facility upgrade program, referred to as the “Cascade Improvement Program/Cascade Uprating Program” (CIP/CUP), between 1974 and 1981. The CIP program

involved the installation of improved barrier in the process equipment and modifications to the compressors, piping, and cooling systems. The CUP program, which ran partially concurrent with CIP involved the uprating of electrical equipment and increasing the efficiency of the process cooling systems [ERDA 1977]. This refurbishment required an increase in the number of craft/trades workers on-site. Staffing levels on all three work shifts increased markedly. A Gaseous Centrifuge Enrichment Plant (GCEP) was also constructed in the early 1980's [DOE 1977a]. This facility, however, ceased operations after initial testing and was never used for production.

Major cascade support functions (and the buildings in which they are carried out) include maintenance (Building X-720), decontamination of process components (X-705), chemical cleaning of components (X-705), assembly of converters and maintenance (X-700), UF_6 feed vaporization (X-342A and X-343), and the product shipment building (X-344).

Building X-720 is the maintenance and stores building and provides most of the equipment maintenance support for the diffusion cascades. Major maintenance areas include the compressor, motor, carpentry, sheet metal, and paint shops plus central stores, tool crib, instrumentation calibration and maintenance, and offices for these activities. Equipment is rebuilt and parts are fabricated here. Materials used in X-720 include various types of solvents, paints, lubricants and coolants [DOE 1992].

The X-705 building is where large components such as compressors and motors from the cascade are disassembled and decontaminated. X-705 also houses the laundry and equipment for the reclamation of uranium from decontamination solutions [DOE 1992].

The X-700 building is where equipment is degreased before reinstallation in the cascade. Converter maintenance activities that require controlled humidity conditions occupy a large portion of this structure. The welding shop is also located in X-700 and is where converters are assembled, modified, and maintained. Welding and metal cutting may also be performed in situ within the process buildings on off-line, purged equipment [DOE 1992]. The lining of the process equipment is a source of nickel fume from welding and cutting tasks.

Building X-342A is a backup facility for X-343 (built in 1981), housing backup equipment for feed vaporization and sampling. X-342A also is the fluorine generation facility for the site. Building X-343 houses seven steam-heated autoclaves which serve two purposes: the vaporization of UF_6, which is then fed into the process buildings through heated pipe ways; and the liquefaction of UF_6 for sampling. Building X-344 is the sampling facility and product shipment building. It contains four autoclaves for sampling UF_6 product and for transferring product from large DOE cylinders into smaller customer cylinders for shipment off-site. Sampling of small cylinders of highly enriched uranium may also be performed in X-344 [MMES1992b].

The potential for exposures to uranium and fluorine containing compounds may occur anywhere that UF_6 is present in the equipment, if UF_6 is released to the atmosphere. This is due to the reaction of UF_6 with moisture in the air which results in the exothermic production of uranyl fluoride (UO_2F_2) and hydrogen fluoride (HF) [Fisher et al.1990]. The potential for exposure to fluorine compounds in the absence of uranium was limited to those activities where fluorine gas or HF was in use for conditioning equipment, cascade maintenance, and in X-342A where fluorine gas is generated from HF.

About 70 support buildings of various sizes are located on the site. The X-600 series buildings are used for utility-related functions. Included are a steam plant, well fields, pump houses, water treatment plant, sewage treatment plant, and numerous cooling towers. Other service functions include an analytical chemistry laboratory building (X-710), medical dispensary (X-101), administration (X-100, X-103, X-104, and X-1000), and cafeteria building (X-102).

The X-500 series facilities at PORTS are related to the electrical power operations. These facilities include switch yards, switch houses, valve houses, and test and repair facilities. The two large switch yards, X-530 and X-533, receive power at 345 kilovolts (kV). The power is transformed to 13.8 kV, and then delivered from the switch yards to the switch houses for distribution to the process and support areas.

Gaseous diffusion is an extremely energy-intensive process, consuming thousands of megawatts of electrical power every day of the year. The peak design power capacity is about 2,100 megawatts [USEC 1994]. The site has extensive utility systems to manage electrical power distribution.

WORKER EXPOSURES

Concern about the biological effects of uranium on humans had been expressed from the start of the Manhattan Project in the early 1940s. In 1958, a symposium was held on the occupational health experience in the industry [HASL 1958]. A second meeting was held in 1975, at which the expressed health concerns led to the formation of the United States Uranium Registry (USUR) [ERDA 1975]. USUR has since published two summary reports on radiological health aspects of the commercial sectors of the uranium industry [USUR 1982, USUR 1983].

The predominant radiation exposure for workers in the enrichment and conversion facilities is to highly soluble compounds of uranium originating as UF_6. Uranium is an alpha-emitter which concentrates in bone, and is also toxic to the kidneys. There is some external ionizing radiation exposure as well. Other potentially hazardous exposures, described in the section that follows, during the uranium enrichment process include fluorines (especially as hydrogen fluoride in air), nickel, and electromagnetic fields (EMF).

Hazardous Exposures

Uranium

Many opportunities for exposure to uranium exist during enrichment. While UF_6 is the material used during enrichment, other chemical forms of uranium exist in the process. Each form presents different risks of chemical and radiological hazards to the workers.

Uranium can enter the body by four pathways - inhalation, ingestion, injection and skin absorption [Voelz et al. 1992]. Injection and absorption of uranium can occur when an open wound or abraded skin is contaminated, but such occurrences are rare at PORTS. Most uranium exposure is from the inhalation of airborne uranium, and some exposure is from ingestion.

All of the uranium isotopes at PORTS emit alpha particles during radioactive decay, and should be regarded as having tumorigenic potential. Historically, the risk to humans from exposures to uranium has been inferred from risks calculated for long-lived, bone-volume-seeking radium-226 (^{226}Ra) [Mays et al. 1985]. For natural uranium compounds found in the environment, bone has been considered to be the target organ, and bone cancer (sarcomas) has been the health outcome of greatest concern. However, numerous chemical forms of uranium with varying solubility have been processed for the nuclear weapons and power industries. The primary portal of entry for this agent in the nuclear industry is inhalation. Thus, it is reasonable to investigate whether uranium exposure at PORTS is associated with tumors of the lung.

The Committee on the Biological Effects of Ionizing Radiation (BEIR IV) evaluated the epidemiologic literature through 1986 and found little conclusive evidence for any carcinogenic effect of uranium [BEIR 1988]. In the animal radiobiology literature, high-specific-activity artificial uranium isotopes (^{232}U and ^{233}U), have produced bone sarcomas in mice. Highly-enriched uranium was reported to produce bone sarcomas in rats. It is therefore reasonable to assume that natural or enriched uranium could cause bone cancer in humans. However, the statistical power to see such an effect in a worker population would be very limited because of the rarity of the disease.

Leukemia might also be an expected outcome because uranium in bone irradiates the bone marrow [BEIR 1988]. (There may also be other causal paths between ingested uranium and leukemia.) However, studies of occupational exposures to radium, in which ingestion is a primary route of absorption, have not indicated a leukemogenic effect [Mays et al. 1985; Spiers et al. 1983].

In animal studies insoluble uranium compounds have been shown to induce metaplastic changes in lung epithelium and malignant lung tumors [BEIR 1988]. Lungs and tracheobronchial lymph nodes were the only organs affected. Rats exposed to highly-soluble, high specific activity uranium (^{232}U and ^{235}U) developed lung tumors, primarily adenocarcinomas. Generally speaking, the more insoluble the form of uranium, the longer the material is retained in the lung [Fisher et

al. 1990] and the higher the radiological dose is to the lung from the inhalation exposure to more highly enriched, less soluble, uranium compounds [ATSDR 1999].

Soluble uranium compounds absorbed by the blood are excreted primarily by the kidneys [Fisher et al. 1990]. Uranium has been identified as a nephrotoxic metal but is considered to be a less potent nephrotoxin than cadmium, lead, and mercury. Researchers have suggested that the renal damage from exposure to high linear energy transfer alpha-emitting heavy metals such as uranium may be the result of a complementary effect of both the chemical toxicity and the radiotoxicity of the metal [ATSDR 1999]. Despite the extensive experimental literature on kidney pathology induced by uranium exposure, kidney tumors caused by uranium have not been identified in laboratory animals and kidney tumors have not been considered a likely outcome in humans [ATSDR 1999].

Researchers at NIOSH have studied the mortality experience of uranium miners and millers [Waxweiler et al. 1983a; Archer et al. 1973; Hornung et al. 1987; Roscoe et al. 1989]. Uranium miners have long been known to be at increased risk of lung cancer mortality, but that risk is believed to be due to inhaled radon decay products such as lead-210, polonium-214, polonium-218 and bismuth-214. Therefore, the miner experience is not informative as to the risks during subsequent stages of the uranium processing industry.

Uranium millers are exposed primarily to insoluble uranium. These worker populations are small and their exposures have not been well characterized. In contrast, larger populations of uranium-exposed workers have been employed at several DOE facilities. Numerous cohort mortality studies have been published on these workers who have been exposed to varying forms of soluble, partially soluble, and insoluble uranium [Cragle et al. 1997; Cragle et al. 1984; Cragle et al. 1988; Frome et al. 1990; Polednak et al. 1981; Checkoway et al. 1988; Dupree et al. 1987; NIOSH 1987a].

The major published study relating specifically to uranium enrichment workers is by Frome et al. [1990]. They conducted an analysis of mortality in World War II era workers at K-25 (the first gaseous diffusion plant), X-10, and the Tennessee Eastman Corporation (TEC) plant (now Y-12 plant). TEC used an electromagnetic separation process of partially soluble UCl_4. Lung cancer mortality was elevated in the K-25 and TEC cohorts relative to the general population. The increased risk of lung cancer continued well past employment time, which is consistent with the long induction period of occupationally induced lung cancer.

This increase in mortality over time emphasizes the need to study cohorts at least twenty or more years after initial exposures. Findings of increased risk of death from lung cancer might have been expected given the radiobiology literature. In fact, the lung cancer effect in the TEC cohort had been previously reported [Polednak and Frome 1981]. A subsequent case-control analysis of

this cohort found a statistically significant dose-response relationship between lung cancer and uranium exposure for workers hired after age 45 [Cookfair et al. 1983].

External Ionizing Radiation

The sources of external ionizing radiation exposure found throughout PORTS are: (1) low energy gamma (γ) radiation from ^{235}U and thorium (^{231}Th and ^{234}Th); (2) beta particles from protactinium (^{234m}Pa), a decay product in the ^{238}U decay chain; and (3) bremsstrahlung radiation (photons) produced by interaction of beta particles from uranium decay products and technetium (^{99}Tc) with surrounding dense materials. (Technetium-99 was introduced into the PORTS cascade from recycled nuclear reactor fuel and it concentrated in the top end of the enrichment process because of its smaller mass relative to the uranium isotopes.)

Very low levels of gamma and bremsstrahlung radiation (0.2 to 1.0 microRoentgen per hour [µR/hr] above background) are found throughout the process buildings [GAT 1986a]. The highest levels of external ionizing radiation (up to 5 milliroentgens per hour [mR/hr]) have been found in UF_6 feed cylinder handling areas. Other potential sources of external ionizing radiation exposure, but with limited exposure potential to the workforce, include high energy photons from various calibration sources such as cesium (^{137}Cs), radium (^{226}Ra), and Cobalt (^{60}Co), machine-generated X rays at tube potentials ranging from 70 to 200 kilovolts, and neutrons [Cardarelli 1997; GAT 1986a]. The Radiation Exposure Information Reporting System (REIRS) report

indicates that exposures to external ionizing radiation in gaseous diffusion plants are significantly lower than in most other DOE sites [DOE 1977b].

Fluoride, Fluorine and Hydrogen Fluoride

Many workers at PORTS have been exposed to fluorides, fluorine, and hydrogen fluoride aerosols (hydrofluoric acid) primarily in the enrichment process buildings and equipment. UF_6, which is solid at room temperature, is heated to a gaseous state to be introduced into the cascade process. UF_6, if released from the cascade system, combines with atmospheric water to produce uranyl fluoride (UO_2F_2) and hydrogen fluoride (HF). Also present in the cascade areas is chlorine trifluoride (ClF_3) which is combined with fluorine (F_2) and used as a conditioning gas in cell treatments [MMES 1985b].

Fluoride exposures occur through inhalation or ingestion of materials containing the F^- ion. Absorption of excessive amounts of fluoride increases the radiographic density of bones. Workers exposed to airborne fluoride concentration of 5 milligrams per cubic meter (mg/m^3) of air or greater, complain of eye and respiratory tract irritation and nausea [Proctor et al. 1988]. Excess fluoride storage in bone and teeth occurs from the ingestion of as little as 3 mg/day. The development of crippling fluorosis may occur after 10-20 years of absorbing 20-80 mg of fluoride daily.

Fluorine (F_2) is a yellow gas that produces severe irritation of the eyes, mucous membranes, skin, and lungs. Upon contact with water, fluorine reacts to produce ozone and hydrofluoric acid. HF gas, like fluorine gas, is a severe respiratory irritant. Inhalation of HF may produce choking and coughing. Following an asymptomatic period of several hours to 1 or 2 days, fever, cough, dyspnea, cyanosis, and pulmonary edema may develop. Exposure to 30 parts per million (ppm) [24 mg/m^3] for several minutes has been reported to result in irritation of the eyes, nose, and respiratory tract. Exposure to 2.6 to 4.8 ppm [2 to 4 mg/m^3] for periods of up to 50 days has caused slight irritation of the nose, eyes, and skin, but no signs or symptoms of pulmonary irritation [Hathaway et al. 1991].

It has been reported that large numbers of workers were exposed to fluorine containing materials during the initial startup phases of the gaseous diffusion plants in the 1950s [Lyon 1962]. During the upgrade processes from the mid-1970s to the early 1980s, there were more workers on-site than usual and more working on process equipment. The upgrade processes required the opening, purging, and performance of work internal to the system on a scale, frequency, and degree of invasiveness not encountered during routine operations and maintenance. This increased the number of workers that could potentially be exposed to fluorine containing materials associated either with releases of UF_6 or from process equipment conditioning and maintenance using fluorine gas. The greatest potential for exposure to fluorine containing materials is during maintenance activities on or in cascade process equipment. Reaction products from the fluorine compounds may also be generated in association with opening the system.

The E area of Building X-705 has also been identified as having exposures to fluorine and fluorides. This area operated between about 1967 and 1978 and involved converting uranium oxide to UF_6. Building X-705 also housed a fluorination process for converting UF_4 to UF_6 which operated for about three years in the late 1950s to early 1960s.

Industrial hygiene data for fluoride, fluorines, and hydrogen fluoride are limited and the specific fluorine containing agent of concern is often ambiguous. The majority of quantitative data is from more recent years. The greatest number of industrial hygiene samples for fluorine containing compounds were collected between 1986 and 1991, with a large increase in sample collection in one cascade building (X-333). Increases in the amount of quantitative sampling for airborne chemical contaminants in the late 1980's and into the 1990's coincided with an increase in site industrial hygiene capabilities. This also reflects an increase in sampling for airborne contaminants as maintenance activities increased following the passage of time since the Cascade Improvement Program/Cascade Uprating Program CIP/CUP (1974-1981).

Nickel

Various epidemiologic studies have reported excess lung and nasal cancer in nickel workers [Mastromatteo 1986; EPA 1986; Wong 1983]. These studies were primarily of nickel refinery workers, but also included nickel alloy workers [Enterline et al. 1982]. Workers at PORTS have had and continue to have exposure to nickel and nickel alloys from welding and metal cutting during maintenance and fabrication of process equipment.

Electromagnetic Fields

Because each stage of the cascade uses an electric motor to drive a compressor, electrical power use is immense (about 2,000 megawatts daily). This large electrical consumption generates 60 Hz electromagnetic fields (EMF) in numerous locations throughout production areas and switchyard facilities. All workers, even those in office jobs, have exposure because ordinary electrical current present in typical building wiring generates EMFs. Brain cancer and leukemia mortality have been associated with occupational exposures to magnetic fields [Floderus et al. 1993; Savitz et al. 1995]; however, such associations have not been found in other studies of magnetic fields [Sahl et al. 1993].

Other Agents

As in any large industrial setting, there are a multitude of other agents to which workers at PORTS can have frequent exposure; depending upon their job, tasks, and work location. Among these are oils, solvents, chlorofluorocarbons, polychlorinated biphenyls, asbestos, welding fumes, acids, paints, temperature extremes, and noise. Other chemicals such as arsenic were introduced into the cascade by contaminated feed materials. In this study, however, exposure to these agents were not specifically addressed for two reasons: (1) a lack of exposure data, and (2) only a small number of workers may have been infrequently exposed for short durations (minutes to hours).

Records of Exposures

Health Physics Records

A formal program of personnel monitoring has been in place at PORTS since operations began. For external radiation, personal dosimetry in the form of film badges (1954-1979) and thermoluminescent dosimeters (TLDs) (1980-present) have been used. For internal radiation, urine analyses and whole body counts have been conducted. The urine analysis program evaluated alpha activity and uranium metal content. Whole body counting was initiated in 1965 utilizing the *in-vivo* facility at the DOE Y-12 plant in Oak Ridge, Tennessee, to measure the deposition of insoluble uranium compounds in the body.

External Radiation

Several administrative and technological changes have occurred in the external monitoring program which are displayed in Table 1. Based on the computerized dosimetry and personnel files provided by the site, 9,215 workers were ever employed. About 81% (N=7,456) of the workforce was monitored at some time during their work tenure. The 19% that were not monitored may have been exposed, since monitoring programs excluded potentially exposed workers during two periods of time. For those workers who were monitored there were 493,214 individual dosimeter readings, consisting of 277,279 film badge records and 215,935 TLD records. Exposures to external penetrating radiation, based on the data received from the site, ranged from nondetected to a lifetime cumulative dose equivalent of about 10 rem.

For the purposes of this study, dose equivalent data (radiation units of mrem) have been converted to absorbed dose data (radiation units of mrad) assuming a quality factor of 1. The quality factor is a multiplying factor used with absorbed dose to express its effectiveness in causing detrimental biological effects [Borders 1991]. The distribution of individual doses was highly skewed. The median cumulative absorbed dose was 60 mrad and seventy-five percent of the workers had less than 240 mrad. The mean cumulative absorbed dose was 268 mrad (Table 2). Relative to natural background rates of exposure to ionizing radiation (about 360 mrem per year), this facility can be considered to have very low exposures [BEIR 1990]. A more comprehensive description of the documents used to assess external exposures to ionizing radiation is presented in the METHODS section of this report.

Internal Radiation

The amount of absorbed alpha radiation from inhaling soluble uranium in UF_6 was monitored by analyzing the activity in disintegrations per minute (dpm) in samples of urine collected from workers. Relative to background excretion rates of uranium in urine, the amount of alpha activity in an unexposed worker's urine sample could not be detected with the historical measurement process used by PORTS. Therefore, any measurable alpha activity can be considered to be occupationally derived. These bio-assays were standardized to dpm per 100 milliliters (ml) of urine. The PORTS supplied a computer tape that contained records for about 240,000 individual samples. Urine samples were collected from workers for a variety of reasons, but the two most common were: (1) "Routine" samples collected to periodically monitor

individuals working in an area where there was potential for radiation exposure, and (2) "Specials" or "Recall" samples collected when an individual's routine sample result was above the recall limit (>5 dpm). When the latter happened, the worker was required to submit repeated samples over time and the change in alpha activity in the urine was followed. Not everyone was required to submit a sample if they worked in an area where potential exposure to alpha radiation occurred. Rather, the practice was to attempt an evaluation of urine samples from a large proportion of the individuals who worked in areas of higher potential exposure, and a smaller proportion with lesser frequency from those employees who worked in areas with lower potential exposure. About 47% of the total workforce had at least one urine analysis record.

There were about 13,000 records of *in-vivo* whole body counts to measure body burdens of internally deposited radioactive materials. Whole body counting began in the later part of the 1960s and continued until 1989. The procedure was performed infrequently and was never considered a routine activity. The mobile whole body counter was shared with other DOE facilities where there was greater need. Employees at PORTS have also been sent to Oak Ridge for *in-vivo* analysis.

Industrial Hygiene Records

Industrial hygiene (IH) surveys have been conducted by site personnel since 1954. Some documents have been found that describe the rationale for selecting buildings and jobs to be surveyed, but no explanation accompanied individual survey results.

Beginning in 1981, IH results were kept in a computer database. The database identifies approximately 120 different agents that have been monitored during the period from 1981 to 1991. The number of sample results on a specific chemical or physical agent ranges from one (e.g. ethanolamine, ethyl butyl ketone, electric field, methyl amyl ketone) to 924 (noise) over the eleven year time period. The most frequently evaluated agents from 1981 through 1991 were trichloroethylene (1981: 73 samples), noise (1982-1987: 45 - 296 per year), and asbestos (1988-1991: 97 - 204 per year).

In addition to the IH computer records, original field survey notes and written reports exist in paper copy. About 14,000 reports (27,000 pages) containing IH data were identified spanning the time period from October 1954 through December 31, 1991. The surveys only identified the building in which they were conducted. With few exceptions, there was no indication of the department name, specific job operation, or individuals monitored. In those rare cases when such data did exist, invariably they were from surveys conducted in the late 1980s or the 1990s. Table 3 presents the quantities of industrial hygiene data available by location (building or area) for the

study period and for the three chemical contaminants of primary interest. Table 4 identifies some of the characteristics of the data as found in the site records.

A majority of samples taken by plant industrial hygienists were area samples, collected by grab or short term (one hour or less) methods. Sampling methods included direct reading colorimetric tubes, filters, and impinger solutions. Equipment used for sample collection changed through time as technology advanced and included Greenberg-Smith impingers, midget impingers, electrostatic precipitators and vacuum pumps.

There are no records of surveys or any other indication that PORTS worker exposure to EMF was measured at any time.

Records of Workers

Personnel Records

Three record systems were available upon which to build the cohort of PORTS workers:

1. The original employment forms ("Form A-59," Figure 5a and 5b);
2. A "Computer Master File" that had been assembled by NIOSH for the previously reported analysis (1987);

3. MMES computer file of all MMES workers, including those who transferred from Goodyear Atomic Corporation (GAT) at the time of the contract transfer and workers subsequently hired by MMES.

The Form A-59 has remained basically unchanged since the start of operations at PORTS. The form, stored in the personnel file of each employee, contained personal identifying information such as name, Social Security Administration (SSA) number, and badge number. It also contained demographic information such as date of birth, address, gender, marital status, and other information about previous military service, education, and previous employment. The A-59s also contain the full work history from hire date to termination date, the date of each job action, the department number involved in the action, and the position (job name) of the work performed. The A-59 forms were the source documents for all other electronic personnel data files received from the site.

A-59 (11-58) REV.

BIRTH DATA

BIRTH DATE

CITY OF BIRTH

STATE COUNTRY

READS

SPEAKS

SIGNATURE

PHOTO

RT. THUMB PRINT

PRESENT ADDRESS: ST. NO.

CITY STATE

COUNTRY PHONE

IN CASE OF EMERGENCY NOTIFY PERSON BELOW

NAME RELATIONSHIP PHONE

ADDRESS CITY STATE

NEW ADDRESS CITY STATE PHONE

NEW ADDRESS

MILITARY SERVICE: BRANCH RANK FROM TO

DRAFT STATUS DRAFT BOARD NO.

CLEARANCE

DATE DATE

PERSONAL		HOME		PHYSICAL		DEPENDENTS
SINGLE	WIDOWER	OWN HOME	BOARD	HEIGHT: FT.		TOTAL NO. OF DEPENDENTS
MARRIED	DIVORCED	RENT HOME	LIVE WITH PARENTS	SEX	COLOR EYES	NO. OF DEPENDENT CHILDREN
WIDOW	SEPARATED	RENT ROOM	LIVE WITH HUSBAND WIFE	WEIGHT	COLOR HAIR	AGE OF DEPENDENTS

SCHOOL	NAME AND ADDRESS OF SCHOOL ATTENDED	YEAR FINISHED	COURSE	DEGREE	YR.
GRAMMER		1 2 3 4 5 6 7 8			
HIGH SCHOOL		1 2 3 4 5			
TECHNICAL OR COLLEGE		1 2 3 4 5			
GRADUATE		1 2 3			
BUSINESS OR CORRES. COURSE		1 2 3			

LEAVES OF ABSENCE

DATE POSTED	EFFECTIVE DATE	REINSTATE-MENT DATE	REASON CODE	DATE POSTED	EFFECTIVE DATE	REINSTATE-MENT DATE	REASON CODE	DATE POSTED	EFFECTIVE DATE	REINSTATE-MENT DATE	REASON CODE

Figure 5a: Form A-59 Personnel Information

EMPLOYMENT HISTORY

DATE	DEPT.	POSITION	EXIT DATE	CODE	DATE POSTED
		RATING P F G VG E			
		REHIRE E NE			
		CLEARANCE P OC OF DATE			
		EXIT CODE V			
		COMMENTS			

PREVIOUS EMPLOYMENT

FROM	TO	NAME AND ADDRESS OF EMPLOYER	POSITION	REASON FOR LEAVING

MEDICAL EXAMINATIONS

DATE	EXAMINING PHYSICIAN	CLASSI-FICATION	DISABILITIES	RESTRICTIONS

SPECIAL NOTATIONS

Figure 5b: Form A-59 Personnel Information

The NIOSH "Computer Master File" was assembled in 1982 as part of the earlier NIOSH study [NIOSH 1987a], and was also based on the A-59 forms. In addition to being in machine-readable format, the advantage of using this file was that it had been the working file for the previous study. NIOSH investigators, during the previous study, examined the badge numbers (on the records included in the computer tape provided by the company) which had been issued sequentially. At that time, if a badge number was missing in the sequence, the investigators went back to the company for the original paper record corresponding to the missing badge number and added the worker to the master file. This process was also used as a check on the completeness of the cohort during the assembly of the computer master file for the initial NIOSH study. Therefore, this file already contained some vital status follow-up and corrected demographic information. This file, however, did not contain information on employees who began work at PORTS after 1982, nor did it contain the updated work histories of employees who were active in 1982 and continued to work past that time. The MMES computer file included all employees ever hired by Martin Marietta after they took control of the contract in 1985, and all of the GAT employees.

METHODS

Cohort Definition

The cohort was defined as all PORTS employees who worked for at least one day at the facility between September 1, 1954, and December 31, 1991. The cohort was assembled by combining the previous NIOSH master file of GAT workers and the computerized MMES file. The data contained in these two files were compared. For each difference the original A-59 was referenced for resolution. The A-59 data were treated as the primary source and took precedence over all other sources.

The new NIOSH computerized file contained all breaks in service that occurred for individual workers over the course of their respective work histories. Reasons for these breaks in service included military leave, schooling, and personal reasons, such as extended illness of self or family members. These breaks in service usually did not appear on the computerized MMES file, but they generally appeared on the A-59s used to resolve discrepancies.

The new NIOSH computerized file consisted of 9,215 workers. Among these workers, there were 53,304 distinct changes within their work histories. Three hundred thirty-eight workers' histories were eliminated because none of them had occurred between September 1, 1954, and December 31, 1991. Therefore, 8,877 workers were included in the cohort.

Vital Status Ascertainment

A list of all individuals in the cohort not known to be deceased by the end of the previous study (1982) but not actively employed as of 1992 was compiled. This list was compared first to the computer death tapes of SSA and then submitted to the National Death Index (NDI) for vital status follow-up through December 31, 1991. Additionally, personnel from the Oak Ridge Associated Universities (ORAU) searched their extensive record system of DOE employees assembled during previous epidemiologic studies of DOE facility workers. ORAU also used Pension Benefit, Inc. which searched some additional sources, including U.S. Civil Service retirement, military retirement, and state motor vehicle registration information.

For individuals determined as deceased, death certificates were requested from the state in which the death occurred. From these certificates, an ORAU nosologist determined the underlying and contributing causes of death. The causes were coded according to the revision of the International Classification of Diseases (ICD) code in effect at the time of death. To ensure consistency between previous (1982) and current nosology, a sample of 100 death certificates was randomly chosen and submitted to the nosologist who had coded the original NIOSH study. There was 100% agreement in the coding.

Cohort Exposure Characterization

This study required the development and use of several exposure matrices (EM) to address potential radiological and chemical exposures. Two radiological EMs required the collapse of numerous department names and numbers that existed in the work histories through time into groups referred to as "similar exposure groups" (SEG). The chemical EMs evaluated airborne chemical contaminants and required that a connection be established between the location (building) in which the samples were collected and the work history components (department numbers). Because IH data for the site has always been recorded "by building" without identifying associated department(s) and work histories were maintained by department without indication of work location (building), a link had to be created to enable a connection between historic IH data and work histories. This resulted in assigning specific department numbers through time to certain buildings on the site and thus linking associated chemical contaminant information. Electromagnetic field (EMF) exposures were measured by NIOSH over several surveys between 1993 and 1995. These measurements were used to assign exposures to cohort members based on the departments in which they worked. The following sections describe the specific EMs in more detail.

External Radiation

A *Personal Dose File* (PDF) was constructed for each worker who had potential exposure to external radiation (gamma and/or X ray). There were 493,214 individual dosimeter

measurements (277,279 film badge records and 215,935 TLD records) taken on 7,456 monitored workers (81% of the workforce) from the plants inception to 1991. If a worker was monitored throughout his or her work history, then that worker's PDF was entirely derived from their dosimetry results. This occurred for 1,292 workers. However, if a worker was not monitored for a period but had potential exposure to external ionizing radiation, then an EM was used to assign an estimated dose to the worker's PDF. The dose values were based on annual averages then divided into daily rates. The number of days employed in each year contributed to the worker's PDF. This procedure was carried out for every year of the worker's tenure.

The EM was a cross tabulation of calendar time by the department [NIOSH, 1993]. Where calendar time and department intersect on the matrix, an estimated exposure value was assigned. This value was usually some measure of central tendency that describes the aggregate of measured exposures in the specified location during the specified time period. To estimate the historical occupational exposure to an unmonitored worker, the worker's job history was compared to the EM. When the worker was in an area of potential exposure as indicated in the EM, but did not have a record of personal dosimetry for that time period, the exposure estimate from the EM was assigned to the worker as his or her exposure.

Changes in the organizational structure from the beginning of operations in 1954 had to be documented. Existing organizational charts, personnel accounting reports, and interviews with management and labor representatives were conducted to generate a record of historical

departmental changes. There were a total of 833 uniquely labeled department titles that existed at the plant from 1954 through 1991. The SEGs were defined and used to construct the EM as follows:

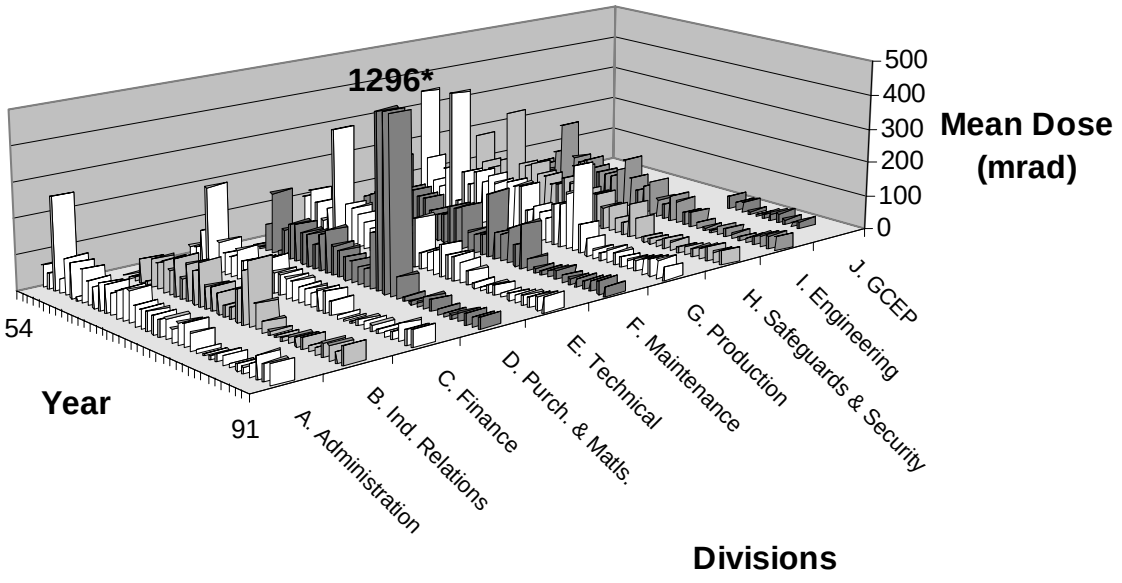
! Ten "Divisions" were identified from historical organizational charts and given an alphabetical designation as follows : A = General Administration; B = Industrial Relations; C = Finance; D = Purchasing and Materials; E = Technical; F = Maintenance; G = Production; H = Safeguards and Security; I = Engineering; and J = Gaseous Centrifuge Enrichment Project.

! Within each Division, workers were grouped by the department title in which they worked and then each grouping was assigned a unique SEG code. The first character of the SEG was an alpha code that designated the Division and the last two characters were numeric codes that designated the department. For example, SEGs for the top level management in the General Administration Division was A00 and Field Level Management was designated A01. This naming convention continued for Miscellaneous Support Staff positions (A02), followed by A03, A04, ... where SEGs were identified by the department titles. This method reduced the categories from 833 unique departments to 136 SEGs between 1954 - 1991.

! Every job in an employee's work history was assigned to an appropriate SEG.

! All dosimetry data associated with each SEG were used to estimate the average absorbed dose for the EM. Since the exposures to external ionizing radiation were low (typically less than 50 mrad per 3 month monitoring period), a large proportion of the dosimeter readings were below the limit-of-detection (LOD) (30 mrad for film badges and 10 mrad for TLDs). Historically, administrative practices at the site required that these measurements be recorded as zeros. The potential number of zeros that could appear each year varied as the monitoring frequencies were reduced over time from weekly, bi-weekly, and monthly to quarterly. NIOSH researchers changed these zero values to reflect the LOD / 2 but normalized the change to reflect an annual dose estimate based on a quarterly monitoring frequency [Hornung et al., 1990]. Therefore a maximum dose of 60 mrad [(30 mrad / 2) X 4 qtrs yr^{-1}] based on film badges or 20 mrad [(10 mrad / 2) X 4 qtrs yr^{-1}] based on TLD measurements was added to a the workers' annual dose file if they were monitored and had zero recorded as an exposure. Unmonitored workers would be assigned the SEG average exposure based on the results from monitored workers.

The external EM was used to assign exposures to workers in potentially exposed job categories who were not always monitored. This resulted in a matrix of estimated exposures by Division over the study period (Figure 6). An explanation for the extreme values for the purchasing and



* For three years, the mean dose in the Purchasing and Materials Division exceeded the range of the histogram.

Figure 6: Mean Annual Dose-Equivalent from External Ionizing Radiation by Division.

materials divisions is presented in the DISCUSSION section of this report. Appendix B is a brief summary of the approach used for estimating external radiation exposures.

This procedure increased the number of workers having cumulative external radiation dose estimates from 7,456 to 8,678 (Table 2). Since the total number of subjects in the analyses was 8,877, the remaining 199 subjects were assigned a cumulative dose equal to zero because they were never monitored or worked in a department in which no other workers were monitored. Individual dose estimates were derived from (1) an individual's personal dose file, (2) an assigned SEG mean dose, or (3) a combination of both personal and SEG data. Application of the EM increased the site's collective absorbed dose from 1,998 to 9,294 rad.

Internal Radiation

The results of about 240,000 urine analyses were in the plant records. The study protocol called for using these records of personal (individual) internal alpha data as a measure of dose in this epidemiologic study [NIOSH 1994]. After close examination of the data, it was determined that these data could not be used for this purpose because:

! The data consisted of periodic alpha activity counts and uranium mass in urine resulting from exposure to soluble uranium for individual workers. Plant records indicate that the primary purpose of the urine monitoring program was not to characterize typical exposures, but rather to determine body burdens among workers with the highest potential exposures [GAT 1963, 1971b]. When there was a high level of either alpha activity or uranium mass detected, repeat measures were made until the urine levels fell below plant allowable limits. Over 90% of the uranium mass results were recorded as zero.

! Soluble uranium has a very short biological half life (about 6 hours) [ICRP 1988]. Thus, the periodic routine sampling at various frequencies (monthly, quarterly, semi-annually) would often miss uptakes of soluble uranium. Even when detected, the sampling data could not be used to estimate internal dose unless the time of intake was known. Additionally, not everyone who was potentially exposed was monitored.

Given the shortcomings in the available urine analysis data for epidemiologic purposes, the challenge was to develop an exposure metric that would be better than a generic surrogate for internal exposure, such as duration of employment. Duration of employment was eliminated as a possible surrogate because a worker's internal dose depended as much on the level of UF_6 enrichment as it did on the frequency and duration of exposure. Even though the integrated dose from an exposure incident could not be calculated, we decided that the urine alpha counts were the best measurements most likely to be correlated with internal dose. In an epidemiologic study, a primary way of judging causality is the presence of a dose-response relationship. Unlike a dosimetry program, it was not imperative that the actual dose be known.

Therefore, the urine analysis alpha count monitoring data were used to construct an exposure file which is a surrogate for internal dose. The exposure file consisted of the mean urine alpha count, in dpm per 100 ml, from all "routine" samples taken for an individual in a specific job category in a particular year (dpm-year). This mean alpha count became the "score." For the years where a worker had no personal samples taken, an exposure matrix was constructed to assign the score based on the exposure experience of co-workers.

Although the basic construction of the EM for assessing internal exposures was similar to that for assessing external exposures, it was more complex. The specific procedure was as follows:

! The same ten Divisions at PORTS that had been identified for the external EM were used again. These were General Administration, Industrial Relations, Finance, Purchasing and Materials, Technical, Maintenance, Production, Safeguards and Security, Engineering, and GCEP (the name given to the centrifuge project). As described previously, each Division was given an alpha code of A through J, respectively (Appendix B). A two digit numeric code was then assigned to each identified department title. As an illustration, Code 04 was assigned to jobs that involved instrumentation in the Cascade. Combining the alpha Division Code F (Maintenance Division) with the numeric Department Code 04, formed SEG F04. Department 731 Cascade Instrument Maintenance which existed from 1955-1985, and Department 712 Cascade Instrument Maintenance, which existed from 1982 to 1991, were represented by the same SEG (F04) for Cascade Instrument Maintenance.

! Site health physics records indicated there was an operating philosophy by plant management that called for more monitoring of urine for workers with greatest potential for exposures. In practice, this intent was manifested two ways. First, workers with the greatest potential for exposure were monitored more frequently. Second, SEGs where workers were monitored more frequently also had a greater proportion of workers monitored.

To assess whether this operating philosophy was practiced, SEGs were stratified into tertiles based on the proportion of monitored employees (< 33% of employees monitored, 33% to 66% monitored, and > 66% monitored). This stratification was arbitrary, chosen as a matter of practicality. Because the monitoring results were count data, the number of alpha disintegrations per minute was assumed to follow a Poisson distribution. Therefore, in preparation for use in a regression analysis and to produce homogeneity of the variances, the alpha count data were transformed by their square root.

! Within each tertile, and for every SEG, the square root of the mean urine alpha count was plotted against the frequency of providing a routine sample. The resulting coefficient for the slope was calculated using PROC GLM, SAS Institute [SAS 1990]. The coefficients were 0.033, 0.044 and 0.088 for the highest, middle, and lowest tertiles respectively (Figure 7). All three of the coefficients had positive trends that were statistically significant. Moreover, the stratification showed that the departments where the majority of workers were monitored had the strongest association

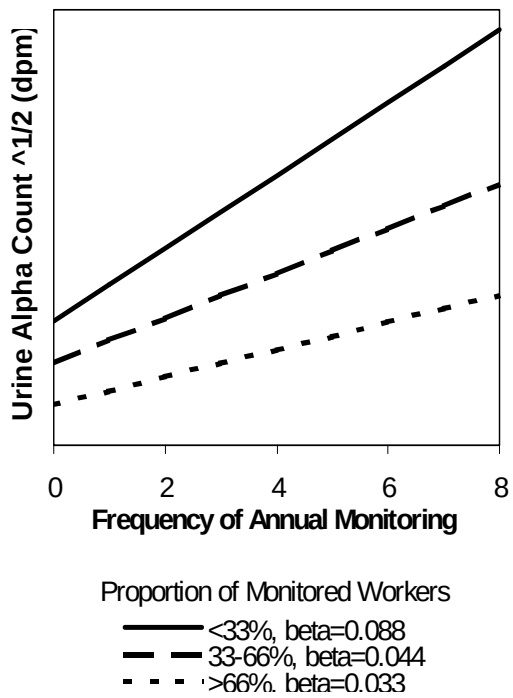


Figure 7: Imputation of Urine Alpha Counts for Workers with Frequency of Monitoring Equal to Zero

between alpha counts and frequency of individual monitoring. This is consistent with the monitoring program which called for more frequent monitoring of workers with greater potential for exposure.

! Because the internal exposure potential continually changed over time, the regression coefficient derived from the appropriate tertile was applied to the existing measurements within each annual SEG to determine the value of the intercept (a monitoring frequency of zero). The specific steps were (1) the mean monitoring frequency of an SEG was multiplied by the regression coefficient of the appropriate tertile; (2) this result was subtracted from the mean of the square roots of all the individual alpha count results; and, (3) the resulting value was squared. This transformed value represented an annual SEG-specific imputed urine alpha count for those workers who had not been measured during any annual period.

! Finally, the annual cumulative alpha count value (imputed or measured) was divided into 365 equal parts (1/365 of the calculated dpm-year). The worker's specific job history was referenced and for every day of employment in a particular SEG, one day's urine alpha count concentration was added to that worker's personal exposure file.

The final EM consisted of assigned alpha counts for individuals who worked in potentially-exposed job categories but were not personally monitored (Figure 8).

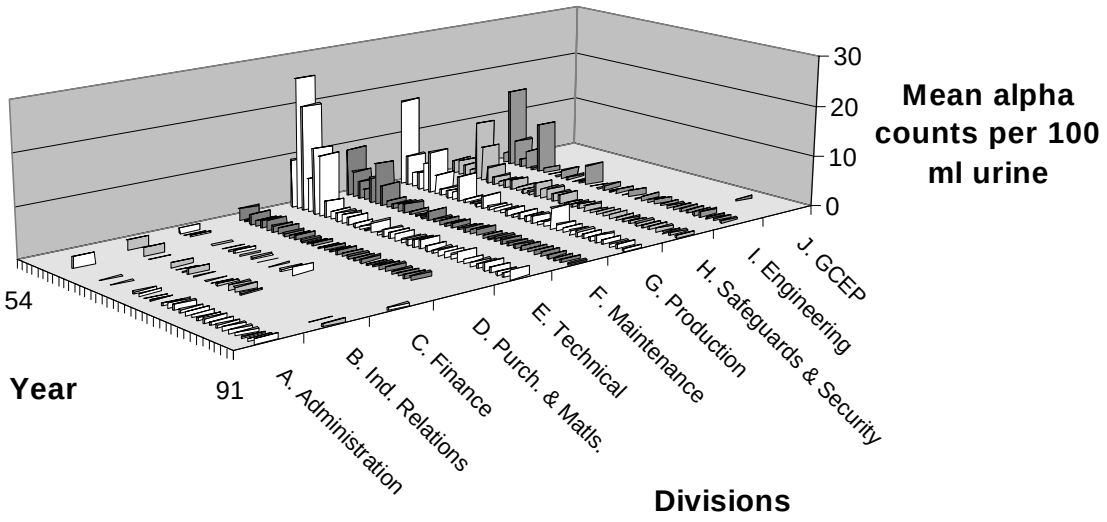


Figure 8: Internal Radiation Exposure Matrix. Mean Alpha Counts per 100 ml in Urine.

Chemical Exposures

Because of the widespread presence of toxic chemicals at PORTS, an attempt was made to identify those chemicals to which worker exposure could be characterized from existing records, and to establish sub-cohorts of exposed workers to study. The specific chemical exposures identified were fluorine/fluoride compounds, uranium, and nickel. All 14,200 existing IH reports were reviewed to determine if any of these three groups of compounds had been monitored. A total of 3,123 survey records was identified, containing 7,185 sample results for these chemicals.

All results were converted to common measures. For instance, measurements for airborne fluorine-containing compounds reported as part per million (ppm), milligram per cubic meter (mg/m^3), or microgram per cubic meter ($\mu g/m^3$), were all standardized to concentrations of the fluoride ion in $\mu g/m^3$. Likewise, measurements for airborne uranium and nickel were converted to concentrations expressed as $\mu g/m^3$ of the metal. Table 3 presents summary information regarding the availability of industrial hygiene sampling data for airborne uranium, fluorine compounds, and nickel.

Attempts were made to create EMs for each of the three substances. Unlike the external and internal radiation exposure data, which included the individually monitored person's identity and the department name in which he/she worked, the IH surveys were mostly area samples. Only the building number (where the survey was performed) and the date of the survey were recorded on the original record. In an effort to use these data, all departments in the surveyed buildings were assumed to have had the same average exposure - a condition that was not very probable. This approach, however, did have the advantages of using actual data for the development of an EM. A single department could be present in more than one building, and buildings often contained more than one department.

Categorizing by one year intervals would have rendered an EM that was sparsely filled and where the mean exposure estimates were highly variable. Therefore, larger groupings of time intervals were used to provide more stable exposure estimates. The time periods were 1954 through 1959,

1960 through 1969, 1970 through 1979, 1980 through 1989, and 1990 through 1991. Dividing the time into these intervals accounted for the major changes at PORTS: the start up period in the 1950s, the production period of the 1960s, the upgrade program of the 1970s, and the post upgrade production of the 1980s up through 1991.

All building-specific IH measurements for the contaminant of interest taken within each of these time periods were averaged. Measurements that were recorded as zero, non-detectable, or less than detectable, were assigned the agent specific limit of detection (using the smallest, non-zero value reported in the data set) divided by two (LOD/2) [Hornung et al. 1990]. When no measurements existed for a building during a given interval, a concentration was assigned by taking the average of the measurements from the preceding and subsequent time intervals. If there were no measurements to use from a preceding interval (e.g. the first time period covered by the EM: 1954-1959), then the average of the earliest time interval for which measurements did exist was extended back in time and applied. Similarly, if there were no measurements from a subsequent interval, then the average of the last interval's measurements was extended forward in time. In this way EMs that covered the entire time period of plant operation were assembled.

Surveys had been conducted only in buildings where exposures were most likely to occur. Therefore, just because a building was not surveyed does not mean that exposures were zero. Out of necessity, however, individuals working within one of the surveyed buildings were

considered to be part of these chemical-exposure sub-cohorts, and only their exposures experienced within these buildings were estimated.

There were only 712 recorded samples that had been taken for nickel. Of these, 169 samples occurred in one particular building (X-700 Building; Converter Maintenance). Nickel became airborne as a result of welding on nickel-containing metals. Building X-700 was the location where welding activities occurred on a continuing basis. Most other welding was performed on the equipment location in the process buildings. The nickel sampling data were sufficient to develop an EM for the X-700 building. The remaining nickel samples were scattered over the rest of the PORTS site and over time.

After EMs were developed for fluorine compounds and uranium, the ratio of the mean and the median measurements frequently exceeded an order of magnitude, indicating that the data were not normally distributed. The vast majority of measurements were consistently very low, with occasional excursions that were orders of magnitude higher.

The alternative metric selected was the proportion of time that an airborne area concentration exceeded the American Conference of Governmental Industrial Hygienists (ACGIH) Threshold Limit Values (TLVs®) for uranium and fluorine that existed in the mid-1950s [ACGIH 1992a, 1992b]. An illustration of how uranium exposure was estimated for workers in building X-326 is given in Table 5.

For example, if a worker was located in Building X-326 which housed the cascade cells where highly enriched uranium was processed during 1954 through 1959, then he or she would be assigned a uranium exposure score of 7.5 for each day worked in this building. The score of 7.5 was assigned because 7.5 percent of the measurements taken for uranium in this time period exceeded 50 µg U/m^3. If the worker changed jobs and worked in the Feed Vaporization Building (X-342) for the period 1960-1969, where one percent of the samples exceeded 50 µg U/m^3, the worker would receive a score of one for each day worked during this period. Each day's score was accumulated to make a lifetime cumulative score. In this way, ratio data that estimated relative exposure to uranium were expressed in a form that could be used for examining an exposure-response relationship. Fluoride exposures were handled similarly, except that the score was based on the proportion of samples exceeding 2500 µg/m^3.

The total number of deaths occurring among workers who had fluorine compounds and/or uranium exposure scores was stratified by quartiles of accumulated exposure. These were examined to see if these quartiles corresponded with logical exposure ranges. The quartiles roughly approximated a geometric increase in accumulated exposure. Therefore, four strata of geometrically-increasing lifetime cumulative exposure scores were calculated. For fluorine compounds. the strata were <30; 30 to 60; 60 to 120; and greater than 120 exposure score-years. For uranium they were <50; 50 to 100; 100 to 200; and greater than 200 exposure score-years.

There were only 35 deaths among workers with quantifiable nickel exposures. Four strata of geometrically increasing exposure concentrations were made. These were <25,000; 25,000 to 50,000; 50,000 to 100,000; and greater than 100,000 $\mu g/m^3$-years

Electromagnetic Fields

Historical data on electromagnetic field (EMF) exposure measurements were not available at the plant. Because consumption of electrical energy at the plant has remained high throughout its operational history, NIOSH conducted a survey of current exposures. Job titles were initially grouped into six categories thought to have similar exposures to magnetic fields: 1) cascade operators; 2) other operators; 3) electrical maintenance workers and welders; 4) other maintenance workers; 5) technicians (including fire and security); and 6) managers and office workers. From each of these groupings, workers were randomly selected for exposure measurements from supervisors' lists of employees. Workers in all jobs, including managers, office staff and security were represented in these measurements.

NIOSH staff completed the personal magnetic field measurements on 252 workers representing most of the jobs at the facility between 1993 and 1995. Measurements were made using a minimum of half-shift samples. Emdex® instruments were used to measure magnetic field intensities at frequencies from 40 to 1000 hertz (Hz) including the 60 Hz power frequency. Daily time-weighted average exposures were computed for each worker monitored. Measured daily

EMF exposures ranged from 0.20 to 82.6 milligauss (mG), with a mean of 3.2 mG and a median of 1.1 mG (Table 6).

In order to investigate a potential EMF exposure-response relationship, EMF exposure estimates were needed for all members of the cohort. Although electricity consumption has remained high at PORTS, this factor alone was not considered adequate to make time-specific EMF exposure estimates over the operating history of PORTS. Other reasons for not making time-specific estimates include: 1) the inability to collect key information required to place workers near sources of EMF; 2) the small number of current workers with EMF measurements in excess of those typically found in household or similar environments; and, 3) changes in the work environment which affect EMF exposure were very complex and difficult to assess over time. For example, the use of electronic equipment in offices and control panels has increased over time but the influence of these changes on EMF exposure remains unknown. Therefore, adjustments to EMF exposure estimates based on changes in plant electrical configurations (motor windings, lighting, and office equipment) were not attempted.

As a result of the surveys to evaluate worker exposures to EMF at PORTS (and the Paducah gaseous diffusion plant), job title alone was found to be insufficient to predict EMF exposures. Information on the task performed and task location were needed. Department identifiers were available for all subjects but, as was noted for the development of the department and building links used for the chemical exposure assessment, the department identifiers were not sufficient to

place workers in a defined work location near electrical equipment within the assigned building. The investigator noted that some of the common assumptions about workplace chemical exposures did not apply to magnetic fields present at the site. One example was the determination that large groups of professionals and office workers had higher magnetic field exposures than did production and maintenance workers. The assembly of exposure groups from the EMF survey data required defining them by location in addition to job title. This resulted in 26 exposure groups with arithmetic means ranging from 0.43 to 24.9 mG (with most groups defined by location in addition to job title.) Efforts were made to assign cumulative EMF exposures to this workforce but because most of the exposures overlapped with typical EMF levels in homes and other non-occupational settings a decision was made not to complete the dose-response analysis for EMF. Additional details on this exposure assessment are described in a recent publication [Wenzl, 1999].

Life-Table Analysis

The overall mortality experience of the entire PORTS cohort, without regard to specific chemical or ionizing radiation exposures, was examined using the NIOSH Life-Table Analysis System (LTAS) [Waxweiler, 1983b; Steenland et al, 1990]. This computer program was used to generate expected numbers of cause-specific deaths (for each of the 92 causes of death listed in Appendix A), based upon the person-years at-risk (PYAR) of dying within 5-year age and 5-year calendar time periods. The United States death rates, specific for race, gender, and 5-year age and calendar time periods, were used as the referent for calculating the expected numbers of deaths. The age, race, and calendar time-specific PYARs were applied to the rates of cause-specific deaths, yielding expected numbers of deaths. Expected and observed deaths were accumulated for each age and calendar time period from September 1, 1954 through December 31, 1991. Persons not found to be deceased during vital status follow-up were considered alive for this analysis. Observed and expected deaths also were stratified by 5-year latency (time since initial employment) and 5-year duration of employment categories.

The same LTAS method of generating expected numbers of cause-specific deaths was used for the following sub-cohorts:

1. Workers with measured or otherwise estimated exposures to external radiation.
2. Workers with measured or otherwise estimated internal deposition of alpha-emitting radionuclides.

3. Workers in buildings where there was potential exposure to fluorine compounds.

4. Workers in buildings where there was potential exposure to uranium.

5. Workers in buildings where there was potential exposure to nickel.

In lieu of stratifying by duration of employment, as was done for the overall cohort, categories of increasing measures of cumulative exposure were created. Categories of personal dosimetry of external radiation, and of urinary alpha count activity for internal radiation, were used for radiation exposure. For fluorine compounds and uranium exposures, stratification was made on categories of increasing proportions of exposures exceeding an exposure limit (agent specific TLV® for the 1950s). For nickel exposure, categories of cumulative exposures were used. For EMF exposure, assigned daily exposure in mG was accumulated as mG-days.

To derive Standardized Mortality Ratios (SMRs), the observed numbers of deaths that occurred were divided by the numbers of deaths expected. Two-sided 95% confidence limits were calculated around each SMR, assuming a Poisson distribution.

Case-Control Analyses

For certain cancer deaths, nested case-control analyses were conducted using conditional logistic regression. Stomach and hematopoietic cancers were chosen based on the original NIOSH study report of excess findings [NIOSH 1987a]. Lung cancer and leukemia were chosen based on questions raised in the radiobiology literature. Bone cancer has also been implicated in the

literature as a consequence of radiation exposure, but the PORTS cohort had an insufficient numbers of cases to support a case-control study.

For lung cancer, 5 controls were chosen for each case. For the other causes investigated, 10 controls were chosen for each case. Controls were matched to cases on age at the death of the case, gender, and race. Three models were used to examine the data: 1) the log-linear (or exponential, which is the usual model of logistic regression), 2) the power function (log of cumulative exposure), and 3) the linear relative risk model ($1+\beta$ dose) using the EPICURE statistical software package [EPICURE, 1993].

Odds of exposure to external ionizing radiation, internal ionizing radiation, and the chemicals nickel, fluorine, and uranium were calculated. Also examined were the temporal effects of time since last exposure, age at first exposure, age at last exposure, duration of exposure, and exposure rate. Effect modification (interaction) was tested using these factors as well as attained age, race, and gender.

RESULTS

Standardized Mortality Ratios

Total Cohort

A total of 9,215 workers was identified from the plant records (Table 7). About 77% were white males, 17% were white females, four percent were non-white males, and two percent were non-white females. Of these workers, about 49% percent were employed for less than five years, and only about 13% were employed for more than 20 years (Table 8). Slightly less than half of the workers were hired prior to 1965, and about 10% since 1985 (Table 9). Three hundred and thirty-eight (3.7%) of these workers were eliminated from the cohort because they did not work at PORTS between September 1, 1954 and December 31, 1991 (Table 10). An examination of their work histories indicated that most were GAT employees who worked prior to 1954 during the time that the plant site was being designed and constructed. The remainder either were hired after the study end date or deciphering their work histories was not possible.

The 8,877 workers who met the cohort definition criteria of at least 1 day of employment between September 1, 1954 and December 31, 1991, consisted of 6,849 (77%) white males, 1,462 (17%) white females, 372 (4%) non-white males, and 194 (2%) non-white females. These 8,877 workers collectively contributed over 200,000 person-years to the study cohort (Table 11).

A total of 1,088 (12%) of the 8,877 final cohort members were identified as deceased by the study end date of December 31, 1991. There were 1,518 expected deaths (SMR = 0.72; CI = 0.67-0.76) for all genders and races combined (Table 12). This overall SMR indicated that significantly fewer deaths had occurred than had been expected in comparison with the experience of the U.S. general population. An examination of the all-cause SMRs, over seven strata of duration-of-exposure and six strata of latency (time since first employment), exhibited no readily discernable patterns of mortality (Table 13).

There were statistically non-significant excesses of death due to cancers of the stomach (15 obs; SMR = 1.18; CI = 0.65-1.94), female genital organs (6 obs; SMR = 1.27; CI = 0.47-2.77), bone (2 obs; SMR = 1.68; CI = 0.20-6.05) lympho-reticulosarcoma (7 obs; SMR = 1.37; CI = 0.55-2.82), and Hodgkin's disease (5 obs; SMR = 1.38; CI = 0.45-3.23) (Table 12).

Deaths from all cancers combined were significantly less than expected; 313 were observed versus 382 expected (SMR = 0.82; CI = 0.73-0.92). This included deficits in malignant neoplasms (MN) of the buccal cavity and pharynx (5 obs; SMR = 0.49; CI = 0.16-1.14), MN of digestive organs and peritoneum (76 obs; SMR = 0.84; CI = 0.66-1.05), MN of respiratory system (112 obs; SMR = 0.79; CI = 0.15-0.95), MN of breast (6 obs; SMR = 0.68; CI = 0.25-1.48), MN of male genital organs (16 obs; SMR = 0.77; CI = 0.44-1.25), and MN of urinary organs (12 obs; SMR = 0.70; CI = 0.36-1.22).

There were also fewer deaths due to non-cancer causes such as diseases of the heart (406 obs; SMR = 0.75; CI = 0.68-0.83), circulatory system (75 obs; SMR = 0.72; CI = 0.57-0.91), diabetes mellitus (16 obs; SMR = 0.67; CI = 0.38-1.09), respiratory system (45 obs; SMR = 0.50; CI = 0.36-0.66), digestive system (41 obs; SMR = 0.52; CI = 0.37-0.70), accidents (71 obs; SMR = 0.61; CI = 0.48-0.77), and violence (34 obs; SMR = 0.51; CI = 0.35-0.72). However, in an *a posteriori* observation uncharacteristic of the deficit seen for most other causes of death, there was a non-significant increase in the category of death entitled "diseases of the arteries, veins and pulmonary circulation." (41 obs; SMR = 1.20; CI = 0.86-1.62)

Observed and expected numbers of cause-specific deaths were computed separately for white males, non-white males, white females, and non-white females. Numbers of deaths among females and non-white males displayed the same basic patterns of cause-specific death rates as white males, but were generally too small for meaningful interpretation. The rates for white male deaths were essentially the same as those for all genders and races combined.

All of the five-year calendar time period and age strata for cancer of the stomach, lympho-reticulosarcoma, and Hodgkin's disease, were reviewed for clustering of deaths in time. The 15 deaths from stomach cancer appear to cluster between 1970 and 1985 (Table 14). But the SMRs displayed no particular pattern by age (Table 15). Likewise, the SMRs for stomach cancer displayed no particular pattern by latency or duration-of-exposure periods (Table 16).

Two of the deaths from lympho-reticulosarcoma occurred in individuals who were within the age group 25-29. Only 0.13 cases would have been expected in this age group. One occurred in the calendar time period 1955-1959 while the other occurred in the period 1965-1969. The work histories and death certificates were examined for the purpose of comparing work and demographic attributes. No commonalities were found, except that they both worked in engineering type jobs (one worked in a division that developed instrumentation, the other worked as a statistician in an engineering division).

There were five deaths from Hodgkin's disease. Two occurred in the time period 1955-1959, one in 1965-1969, one in 1970-1974, and one in 1975-1979. All five occurred in individuals 30 to 55 years of age, which was about twice (SMR = 2.22; CI = 0.72-5.18) the number of expected deaths for this age group.

External Radiation

For the analysis of possible relationships between cause of the death and external radiation exposure, the cohort was restricted to those who had worked in departments where workers had been monitored for exposure to external radiation by film badge and/or by TLD. This restriction resulted in a sub-cohort of 8,564 workers (Table 17). Overall, the 8,564 workers were either monitored for external radiation or were assigned a dose by application of an EM consisted of 6,708 (78%) white males, 1,306 (15%) white females, 362 (4%) non-white males and 188 (2%) non-white females. Collectively, they contributed 194,494 person-years at risk of dying

(Table 18). The LTAS stratified the PYARs by categories of increasing cumulative absorbed dose to external radiation determined by personal dosimetry or EM. The categories were < 30 mrad; 30 mrad to 100 mrad; 100 mrad to 500 mrad; 500 mrad to 1 rad; 1 to 5 rad; and > 5rad.

The resulting SMRs for all cause-specific deaths were essentially the same as those resulting from the analysis performed on the full cohort (Table 19). There were 1,043 (12%) workers who died during the study period, while 1,461 were expected to have died (SMR 0.71; CI = 0.67-0.75) for all genders and races combined. There were 298 cancer deaths while 366 were expected (SMR = 0.81; CI = 0.72-0.91). The all-cause SMRs examined over the six strata of increasing cumulative absorbed dose revealed no discernable relationship, nor did an examination by seven strata of increasing latency (time since first recorded or assigned absorbed dose) (Table 20).

A statistically non-significant elevated SMR for cancer of the stomach was present similar to that observed in the overall analysis (14 Obs; SMR = 1.14; CI = 0.62-1.92) (Table 21). There were no apparent patterns of mortality readily evident over the six strata of cumulative absorbed dose nor the seven strata of latency. Statistically non-significant excesses of death due to cancers of the bone (2 obs; SMR = 1.75; CI = 0.21-6.32), lymphoreticulosarcoma (7 obs; SMR = 1.43; CI = 0.57-2.95), and Hodgkin's disease (5 obs; SMR = 1.44; CI = 0.47-3.37) were all similar to the overall cohort findings.

As was seen in the overall cohort, there remained a non-significant excess of deaths from diseases of the arteries, veins and pulmonary circulation (40 obs; SMR = 1.21; CI = 0.87-1.65). There also was a statistically non-significant increase of deaths due to diseases of the musculoskeletal system and connective tissues (5 obs; SMR = 1.62; CI = 0.53-3.79).

Examination by increasing categories of cumulative external radiation and by latency (duration of time since first exposure) of cancers of the stomach, lung, Hodgkin's disease, lymphoreticulosarcoma, and all cancers combined failed to reveal any patterns suggestive of an dose-response relationship (Tables 21 through 25).

Internal Radiation

For the analysis of possible relationships between cause of death and internal radiation exposure, the cohort was restricted to those who had worked in departments where biological (urine) samples had been taken from workers to monitor their personal exposures to alpha activity from uranium. This restriction resulted in a sub-cohort of 6,827 workers (Table 26). There were 5,593 (82%) white males, 834 (12%) white females, 282 (4%) non-white males, and 118 (2%) non-white females. Of these, 885 (13%) were identified as deceased. The proportion of males in this sub-cohort was about six percent greater, and of women, about 24% less than in the total cohort of 8,877. Collectively, the sub-cohort contributed over 150,000 person-years at risk of dying (Table 27). Because the first enriched uranium was not produced until 1954, there were no person-years at-risk for dying in 1954 [ERDA 1977]. The LTAS stratified the PYARs by

increasing cumulative alpha activity determined from personal urine samples or EM. The categories were less than two dpm-years; two to four dpm-years; four to eight dpm-years; eight to 16 dpm-years; 16 to 32 dpm-years; and greater than 32 dpm-years.

In this sub-cohort there were 885 total observed deaths compared with 1,214 expected (SMR = 0.73; CI = 0.68-0.78) for all genders and races combined (Table 28). The observed and expected numbers of deaths yielded essentially the same SMR and confidence intervals found for all causes of deaths in the total cohort. An examination of the total mortality over the six geometrically increasing categories of cumulative urine alpha dpm-years, revealed consistently low SMRs (statistically significant at $p<0.01$) of 0.75, 0.66, 0.70, 0.71, 0.77, and 0.73 for the categories <2 dpm-years, 2-4, 4-8, 8-16, 16-32, and >32 dpm-years, respectively (Table 29). There were statistically non-significant excesses of deaths due to cancers of the stomach (11 obs; SMR = 1.07; CI = 0.53-1.92), larynx (5 obs; SMR = 1.25; CI = 0.40-2.91), bone (2 obs; SMR = 2.16; CI = 0.26-7.81), lymphoreticulosarcoma (6 obs; SMR = 1.49; CI = 0.54-3.23) and Hodgkin's disease (3 obs; SMR = 1.08; CI = 0.22-3.15). Deficits of deaths were observed for most of the other 92 individual causes examined, as was a deficit of all cancer combined (239 obs; SMR = 0.78; CI = 0.69-0.89).

As was seen in the overall cohort, there remained a non-significant elevated SMR for diseases of the arteries, veins and pulmonary circulation (35 obs; SMR = 1.27; CI = 0.88-1.77). There also

was a statistically non-significant elevated SMR for the diseases of the musculoskeletal system and connective tissues (5 obs: SMR = 2.02; CI = 0.65-4.72).

Examination by increasing categories of urine alpha activity and by latency (duration of time since first exposure) of cancers of the stomach, lung, Hodgkin's disease, and all cancers combined, revealed no clear patterns suggestive of an exposure-response relationship for these cancers (Tables 30 through 33). Similarly, when lymphoreticulosarcoma was examined this way, there were no obvious patterns (Table 34). In some individual exposure/latency categories, however, the observed numbers of lymphoreticulosarcoma deaths differed substantially from that expected. Three of the cases fell within the five to 10 year latency stratum (SMR = 5.36; CI = 1.08-17.56), and two of these occurred in the stratum of eight to 16 dpm-years category (SMR = 20.33; CI = 2.28-88.21). All five deaths from cancers of the larynx occurred in workers with urine cumulative alpha activities greater than eight dpm-years, while only 2.2 were expected (SMR = 2.30; CI = 0.74-5.69).

Fluorine and Fluoride Compounds (FFC)

For the analysis of possible relationships between cause of death and fluorine and fluoride exposures, the cohort was restricted to those who worked in departments where airborne concentrations of FFC had been measured. This restriction resulted in a sub-cohort of only 1,446 workers (Table 35). Collectively, they contributed 29,432 person-years at risk of dying (Table 36). The LTAS stratified the PYARs by increasing cumulative exposure-scores to FFC. The

categories of scores were <30 exposure-years; 30 to 60 exposure-years; 60 to 120 exposure-years; and >120 exposure-years.

A total of 139, or 9.6% of this sub-cohort, was deceased, compared with 201.8 expected (SMR = 0.69; CI = 0.58-0.81) when all races and both genders were considered together (Table 37). The previously noted increase in cancer of the stomach was still present in this sub-cohort (3 obs; SMR = 1.80; CI = 0.37-5.26). All three cases were between the ages of 45-60 years, and all three were in the lowest two exposure strata (Table 38). There was only one case of lympho-reticulosarcoma (SMR = 1.56; CI = 0.04-8.69) and no cases of Hodgkin's disease or bone cancer. There was only one-half the number of cancer deaths as expected (26 obs; SMR = 0.52; CI = 0.34-0.76).

Uranium

For the analysis of possible relationships between cause of death and uranium exposures, the cohort was restricted to those who worked in departments where airborne concentrations to uranium metal were measured. This restriction resulted in a sub-cohort of only 1,832 workers (Table 39). Collectively, they contributed a total of 42,176 person-years at-risk of dying (Table 40). A total of 190 (10.4%) of the sub-cohort was deceased compared with 278.2 expected deaths (SMR = 0.68; CI = 0.59-0.79) when all races and both genders were considered together (Table 41). The previously noted excess of cancer of the stomach was still present in this sub-cohort (3 obs; SMR = 1.31; CI = 0.27-3.84). There was only one case of lympho-

reticulosarcoma (SMR = 1.09; CI = 0.03-6.08) and no cases of Hodgkin's disease. There were 36 deaths from all cancers (SMR = 0.52; CI = 0.36-0.72).

Nickel

For the analysis of possible relationships between cause of death and nickel exposures, the cohort was restricted those who worked in departments where airborne concentrations to nickel were measured. This restriction resulted in a sub-cohort of only 465 workers (Table 42). Collectively, they contributed a total of 8,077 person-years at-risk (Table 43). This is an extremely small sub-cohort upon which to base an analysis; only the most extreme occupationally-induced epidemic of mortality would be seen in a cohort of this size. The LTAS stratified the PYARs by increasing cumulative exposure to nickel. The categories were <25,000 $\mu g/m^3$-days; 25,000 to 50,000 $\mu g/m^3$-days; 50,000 to 100,000 $\mu g/m^3$-days; and 100,000 $\mu g/m^3$-days.

There were 35 deaths (7.5% of the sub-cohort) with 45.8 deaths expected (SMR = 0.76; CI = 0.53-1.06) when all races and both genders were considered together (Table 44). There was no indication of an exposure-response relationship for any cause of death, nor were there any readily apparent patterns by age, latency, or calendar time.

Case Control

Lung Cancer

The 107 deaths from lung cancer were examined in a case-control analysis. For each lung cancer death, five controls (non-cases) were selected from the risk set. Controls were selected from the risk set and were required to survive at least to the age at which the case died. Controls were also matched on race and gender.

Analyses were performed using continuous measures of cumulative external exposure, assuming log-linear, power function (log of exposure), and linear relative risk models. These analyses showed non-significant negative regression coefficients for each millirad of exposure to external radiation of -0.00008, -0.102, and -0.000054, respectively. The cumulative exposure was categorized by 0 to 100 mrad, 100 to 500 mrad, 0.5 to 1.0 rad, and greater than 1.0 rad. The resulting coefficients were all negative (β= -0.114, -0.341, -0.989, and -0.254, respectively). Potential interaction between cumulative exposure and age was examined in a log-linear model (cumexp β= -0.0032, p=0.072; cumexp*age β=0.00005, p=0.074). Because of the resulting small p-values, cumulative exposure was dichotomized and examined at the age that the coefficient suggested the relative risk became positive. This resulted in a β= -0.0004, p=0.225 for risk at less than 60 years of age and β=0.0005, p=0.200 for greater than 60 years of age. Thus, no statistically significant associations were seen. No confounding was seen when examining exposures to uranium, nickel, fluorides, or EMF. Similarly, time since last exposure and age at

first exposure were also not found to have any significant effect upon the relative risk of lung cancer.

The continuous measure of urine alpha counts was used as an index of internal radiation exposure. Because this counting represents a scale of relative exposure rather than an actual dose estimate, only cumulative exposure was examined. Both linear and log-linear coefficients were positive (0.0027 and 0.0037 respectively), but in both instances the p-value exceeded 0.5 (Table 45).

To discount the effect of exposures received directly prior to death (which would not likely be causally related to a death from lung cancer), exposures were lagged 5 years prior to the age of death of the case (Table 46). Cumulative external absorbed dose lagged five years evaluated in a log-linear model resulted in a per millirad negative β=-0.00006; $p>0.5$. This means there was a statistically non-significant inverse relationship between lung cancer and cumulative external radiation exposure. Categorizing the cumulative absorbed dose by 0 to 100 mrad, 100 to 500 mrad, 0.5 to 1.0 rad, and greater than 1.0 rad resulted in all negative β coefficients of -0.014, -0.045, -0.779, and -0.067, respectively. None were statistically significant. An interaction term between cumulative absorbed dose and age provided essentially the same results as the non-lagged analysis. No statistically significant relationships were revealed by dichotomizing age attained at <60 years and of >60 years.

There were also no clear patterns of dose-response relationships under a 10, 15, or 20 year lag assumption (Tables 47 and 48), although at 10 years the log-linear regression model that examined the interaction of absorbed-dose and age resulted in negative coefficients for exposure, and positive coefficients for the interaction term with p-values of 0.049 and 0.044, respectively. This suggests that there may be a positive exposure-response beginning at older ages. However, when cumulative exposure was divided at <60 years and >60 years of age, neither were statistically significant. The direction of this effect is the same with a 15 and 20 year lag assumption, but the p-values are larger.

Hematopoietic Cancer

There were 36 deaths from hematopoietic cancers examined. Cases were matched to non-cases from the risk set (at a ratio of 1:10) on race, gender, and attained age. The analyses were the same as those performed for lung cancer except that only lag periods for zero and five years were evaluated. Each of the log-linear, linear, and power function models showed no significant trends with cumulative external absorbed dose (Tables 49 and 50). There was no confounding apparent by other chemical exposures, time since last exposure, or age first exposed, nor was there evidence of effect modification by attained age.

Internal radiation was not observed to have an effect on hematopoietic cancer risk. No trends in relative risk with cumulative exposure were found for any of the three forms of the risk function. There was a suggestion of declining relative risk with time since last exposure (TSLE)

(z = -1.931, p=0.053), but this holds little meaning since the effect of internal exposure was either flat or declining before inclusion of the TSLE term in the model=Eq~ÄäÉë=RN=åÇ=ROFK

Leukemia

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Stomach Cancer

There were 15 deaths due to stomach cancer in the cohort. They were matched to non-cases from the risk set (at a ratio of 1:10) on attained age, race, and gender. The analyses performed were the same as those performed on previously examined cancer deaths. No suggestion of positive findings emerged from these analyses (Table 55).

DISCUSSION

The most evident health outcome in this cohort is the better than expected all-cause mortality experience. As with other epidemiologic studies of occupational cohorts, it had been anticipated that mortality rates for this cohort would be lower than the expected due to the "healthy worker effect" [Fox et al, 1976]. What was not anticipated was the extent of this effect. The healthy worker effect is usually a manifestation of some measure of good health status required to initially seek and obtain employment (selection bias) and possibly the advantages to survival associated with being employed (survival bias). These effects combine to create a propensity of good health among worker cohorts relative to the general population. Factors that may lead to increased survival might include sufficient income, good housing, access to health care, and the perception and esteem that come from job purpose and status. In the present case, the effect is unusually conspicuous among these PORTS workers. For instance, deaths from homicide is a mere 33% of that expected. This suggests that there may be some additional factors influencing the mortality rates. Perhaps the requirement of obtaining and keeping a high-level security clearance led to a "super healthy worker" selection and survival bias.

Consideration was given to whether there may be artifacts contributing to the appearance of greater survival. For example, if the vital status ascertainment was incomplete then number of deaths would be undercounted while at the same time the person-years of deceased individuals

would not stop accumulating. If under-ascertainment was a factor, this would result in the appearance of a better than expected mortality experience for the cohort where none exists.

When large, across-the-board, deficits in mortality are seen, it is appropriate to suspect under-ascertainment of deaths as a contributing factor. Therefore, the completeness of the follow-up activities should be suspect and validation of the follow-up activity should be confirmed. Public health agencies no longer have access to Social Security Administration assistance in determining vital status of cohort members. This was a time-proven method of establishing the vital status of a cohort. The National Death Index (NDI), formed in 1978, provided us with an alternate method of follow-up, and has the added benefit of providing information about the state that maintains the death certificates [CDC 1990]. But only deaths occurring subsequent to 1978 are included in the NDI. Relying solely on the NDI for determining vital status of a cohort has been evaluated by others and is generally accepted as performing in a relatively complete manner. However some selectivity is involved in all follow-up methods [Boyle et al, 1990; Omohundro et al, 1993].

The PORTS cohort had been previously studied and vital status had been determined through the SSA, Internal Revenue Service, U.S. Post Office, the Ohio Bureau of Motor Vehicles, and PORTS records. This had been completed through 1982 for most of the cohort and for all of the cohort through 1979. This is past the time that NDI has been demonstrated to be virtually complete. The demographic information available for the PORTS cohort was exceptionally good

with regard to the information required for NDI follow-up, so there is no reason to believe that there is a quality of data problem that may have led to less than adequate follow-up. But that does not rule out the possibility of error, such as a mis-formatted computer data tape, or a missing data. Therefore, to rule out such error in follow-up, the entire file was recreated and submitted to NDI. The search results were the same. Further attempts to establish the whereabouts of each and every member of the cohort, by asking former neighbors, running credit checks, scanning city directories, and other techniques is feasible. However such additional effort is prohibitive because it is very labor intensive, expensive, and likely to be of little value.

The less than expected mortality is very prominent in this cohort but it is not unique. Other cohorts of DOE workers have shown the same general finding, including the earlier study of this cohort by NIOSH [NIOSH 1987a; Wing et al, 1991; Wiggs et al, 1994; Gilbert et al, 1993]. This recurring theme emphasizes the relatively young age of all DOE cohorts studied thus far. Initial selection bias was unduly influential because sufficient time has not yet elapsed for the healthy worker effect to diminish.

Inhalation is the primary route of entry of radioactive material in this facility. Because there would be some resident time in the lung before this soluble compound is absorbed systemically, it is reasonable to postulate that lung cancer is a primary radiogenic tumor of concern in this population. A large combined Oak Ridge cohort study, with Poisson regression modeling to estimate risk at specific sites, found an elevated risk of lung cancer at the K-25 site (SMR=1.10),

where the gaseous diffusion process was similar to that at PORTS [Frome et al, 1997]. In this cohort, however, the SMR for lung cancer in the overall population was significantly less than expected (106 obs; SMR = 0.78; CI = 0.64-0.95). No trends emerged from analyses of five-year age or five-year latency strata. Among the PORTS workers monitored for exposure to soluble uranium by urine analysis, no apparent mortality trends were observed with urine alpha activity count or age. Moreover, there was no evidence in the internally-adjusted case-control analyses that risk increased as exposure increased.

With regard to leukemia risk, there was no effect apparent for cumulative exposure to either external or internal radiation. There was, however, an effect modification with attained age. The effect for workers under 40 years of age was positive, but not statistically significant. The lack of statistical significance may be due to a lack of power to detect such trends. The power was limited because there was a total of only 13 leukemia deaths, and only four of those were less than 40 years of age. Similar results were found when examining internal exposure and were probably due to external and internal exposures being highly correlated (r=0.69).

One of the primary objectives of this study was to develop methods to utilize the voluminous radiation and IH data available on this population in order to increase the sensitivity of the study. In an earlier study, NIOSH evaluated the urine monitoring data but reasoned that the mass-uranium data had not been collected in a way that allowed back calculation to actual exposure

[NIOSH 1987a]. In that study, the mass-uranium[2] data was used to classify the cohort by the frequency of monitoring for urine uranium content. Departments were ranked on how often they exceeded 50 µg/l uranium, and then this distribution was dichotomized. Although this was a reasonable way to use the data, it only allowed for a simple dichotomous comparison of what was believed to be a surrogate for radiation exposure. A more powerful analysis requires a graduated measure of exposure across the cohort. In the present study analyses, the urine alpha counts were used. In addition, existing historical IH data for fluoride compounds, uranium, and nickel exposures were also used to investigate job-related mortality.

Although these EMs were established according to well-reasoned criteria, these procedures sometimes lead to anomalies in the estimates. For instance, the rules for establishing the external radiation EM resulted in some dose estimates being unduly influenced by monitored workers with very short work durations. This may have resulted in assignment of doses to unmonitored workers that were in excess of what they actually experienced. An example of this was seen in Figure 6 in the Purchasing and Materials Division where the unlikely dose values of 499, 578 and 1,296 mrad/yr were assigned to unmonitored workers for the years of 1976, 1977, and 1978 respectively. If the rules used in developing the EM had required an arbitrary minimum of 90 days of monitoring before including the dosimetry results into the EM, then these extreme estimates would have been lower (compare Figure 6 to Figure 9). The doses for workers in the

[2]Brown and Bloom used uranium metal in urine, not urine alpha activity, as a surrogate for exposure to internal alpha radiation.

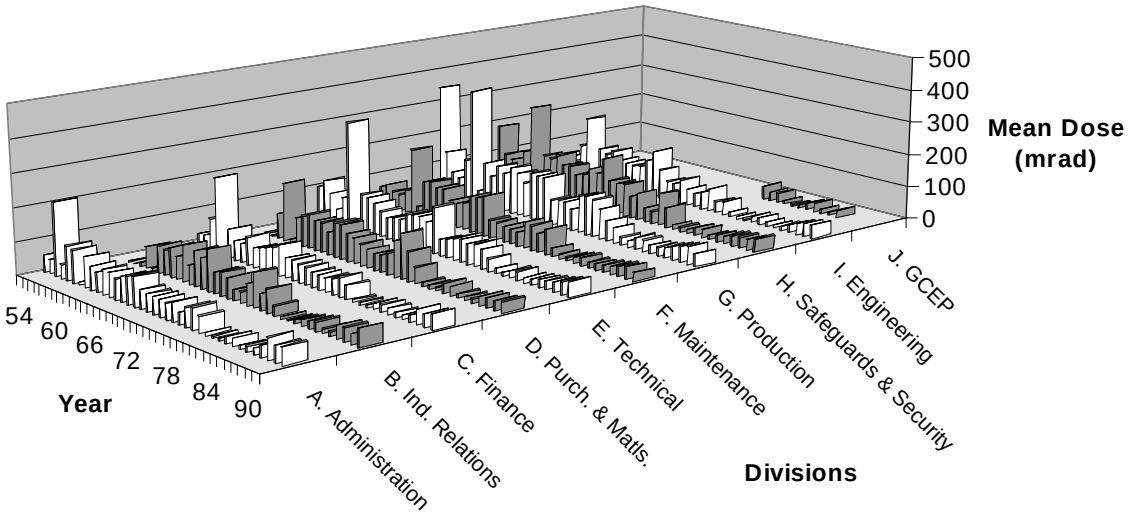

Figure 9: Adjusted External Radiation Exposure Metric. Minimum of 90 Days Monitoring for Workers to be Considered in the Creation of the EM.

Purchasing & Materials Division for the similar years (1976-1978) were reduced to 108, 136, and 89 mrad/yr respectively. Nonetheless, the original EM was used to conduct the epidemiologic analyses since it was based on established criteria.

No dose-response relationships were found from exposures to any of these agents with any particular cause of death. This could be viewed as evidence against a causal relationship between exposure to internal radiation and chemicals with mortality from a number of cause-specific deaths. However, the accuracy of the exposure metrics developed for these analyses could be responsible. To address this concern, a group of plant management and labor representatives

who possessed many decades of experience were presented with EM-derived estimates of exposure (by division and year). While the group debated about how the exposure estimates were made, this group of experienced people believe the historical pattern of exposure was correctly portrayed (Figure 8). They agreed that the departments and times where internal and external radiation exposures were known to have occurred were properly proportioned relative to their expectations. They could even identify in the graph small perturbations in exposure from temporary changes in plant activities known to them but not to the NIOSH investigators (work stoppages and process interruptions).

Independent evaluation of the exposure estimates developed for the chemical contaminant EM (uranium, fluoride compounds, and nickel) using other data or even a subset of site data was not possible. The site had never extracted and assembled the historic industrial hygiene sampling data. The majority of the samples collected were not individual personal exposure samples. Sampling data was associated with site building; not department numbers, job titles, identified tasks, or sample collection purposes, and rarely with any individual. The site had no method of associating buildings (identified in industrial hygiene survey forms) with departments. Employee work histories were associated with department numbers, not buildings. Historic exposure data from site records for uranium (measured as a mass airborne concentration), fluoride compounds, and nickel along with the department-building link (to enable assigning work histories with chemical exposures or site locations) have subsequently been obtained by investigators conducting medical surveillance for PORTS former workers.

Bone sarcoma has been a primary malignancy of concern because uranium is an alpha-emitting, bone-seeking radionuclide like radium [ICRP 1979; Mays et al, 1985]. Because of the size and age of this cohort, it was known at the outset that the power to statistically detect a difference in the occurrence of bone cancer would be extremely low (Table 56) [Rinsky et al, 1992]. Indeed, the SMR for bone cancer among those workers who were in departments where urine samples were monitored was 2.16, based on two cases (0.92 expected). The confidence intervals were extremely wide (CI = 0.26-7.80), reflecting the lack of power commensurate with small numbers. The power to detect a two-fold increase of bone cancer at a 95% confidence level was a mere 12% [Rinsky et al, 1992]. With the small number of expected bone cancer deaths, a six-fold effect would be necessary to statistically detect an increase (80% power). Further investigation into these two cases revealed the following information:

Case one was a 55 year-old white male who died in 1964. His death certificate reads:

> **"IMMEDIATE CAUSE (a)** *Metastatic carcinoma to spinal column*
> **DUE TO (b)** resulting in respiratory paralysis and death."

The nosologist coded this as a 196.2 which, in the Seventh Revision of the International Classifications of Diseases (ICD), is the code for malignant neoplasm of the vertebral column (WHO 1957). According to his personnel file, he had worked as a maintenance mechanic, from November 2, 1953 to July, 1955, in departments 730, 731 and 724. As best as can be determined, from the earliest

plant documentation available, these Departments were associated with process and cascade maintenance. He may, therefore, have worked in the first gaseous diffusion cell placed on stream in 1954 [ERDA 1977]. It is unlikely, however, that he would have had much exposure to radiation during this time period because by 1956 there was only a total of 380 cells. It could be argued, on the other hand, that if he worked in the early history of the facility that high exposures related to startup activities may have been present, but were not documented. This individual had previous employment at several chemical plants in the Kanawha Valley of West Virginia, and served in the U.S. Navy from 1943-1945.

Case two was a 68 year old white male who died in 1987. His death certificate reads:

"IMMEDIATE CAUSE (a) *Osteogenic sarcoma of spine."*

There were no other contributing causes of death listed. The nosologist coded this as a 170.2 which, in the ninth revision of the ICD, is the code for malignant neoplasm of the vertebral column (WHO 1978). According to his personnel file, he had worked as a Draftsman and Designer from 1956 through 1983 in Departments 761, 737 and 741. None of these departments was associated with radiation exposure. Department 761 was housed in the 100 Building, which was administration, and 737 and 741 were in the 720 Building where offices and maintenance shops were housed. According to his personnel file, this individual

had worked previously for various companies as a draftsman, pipefitter, plumber and electrician.

Given that these two cases did not work in areas where radiation exposures were likely, and that the cause of death as written for one of the cases is not clear as to the origin of the cancer,[3] few helpful data are provided to address the effect of radiation on bone cancer.

Uranium, as a metal, is known to be nephrotoxic. There were 5 deaths from chronic nephritis (SMR = 0.60; CI = 0.19-1.39) from the entire cohort. For the sub-cohort of 1,832 workers where an attempt was made to quantify the amount of uranium metal exposure received, there were two deaths from chronic nephritis (SMR = 1.34; CI = 0.16-4.83). Therefore, uranium metal related nephotoxicity in this cohort is not statistically evident.

[3]The death certificate indicates that the malignancy was a metastatic tumor but it does not specify whether it was a primary bone tumor or from some other site. The designation "carcinoma" rather than sarcoma also makes it suspect as a primary bone tumor.

LIMITATIONS AND STRENGTHS

As in all observational epidemiologic studies, where subjects are neither chosen for their optimal contribution nor are strict experimental conditions controlled by the researchers, this study has a number of potential limitations. They include:

- an early assessment of the cohort (only 12% of the cohort was deceased by December 31, 1991);
- the presence of a strong healthy worker effect bias;
- exposure data that was collected for compliance purposes and therefore was not optimally suited for epidemiologic application;
- a reliance on estimates of past exposures derived from complex models when no personal exposure data were available;
- the essentially complete lack of information about concomitant exposures resulting from non-inventoried chemicals, neutron and medical exposures, and transuranics; and
- an inability to study any cause of illness that does not result in death (diseases with low case fatality rate). This would exclude, for example, the study of some chronic respiratory conditions that are of great importance in assessing the health of an industrial population.

The strengths of this study are substantial and include:

! an exhaustive assessment of all existing health physics and industrial hygiene data;

! use of all personal radiation dosimetry and biological sampling that existed at the facility, resulting in a substantial improvement over reliance on indices often used a surrogates for exposure (such a duration of employment) which are subject to mis-classification and confounding;

! a validation of the mortality search results from the National Death Index, and a validation of the consistency of nosology coding over time;

! determination of the reasonableness of dose estimates made by independently estimating internal and external radiation exposure trends and then comparing them to the judgements of a group of long term plant personnel;

! the mortality study is a good tool for evaluating the major health consequences of chronic low-level radiation exposure, namely maligancies of the blood, lung, stomach, and bone, because these diseases have high case fatality rates; and

! the ability to quickly and economically update this study and keep track of the mortality experience as the cohort ages and as new epidemiologic methodology is developed.

CONCLUSIONS

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In the course of this study, several Health Hazard Evaluations were conducted that addressed specific issues of occupational exposures [Cardarelli 1997, Ahrenholz 1996, Singal et al. 1994]. These hazard evaluations resulted in modifications to specific record keeping practices, engineering controls, and process operations.

This report can serve as a beginning of a periodic assessment of the mortality experience at this facility. The time consuming and labor intensive process of assembling EMs and links between buildings and departments has been completed through 1991. Some additional work would be required to characterize organizational changes after 1991. By 2003, it will be possible for the National Death Index to update vital status through 2001. Thus, a ten year update will be possible in only two years.

In summary, this was an exhaustive effort to understand the exposures and their potential effects on this population of nuclear workers. At this point in time, there do not seem to be deviations in mortality of sufficient magnitude to detect epidemiologically. It remains to be determined whether this is because there has been no adverse mortality resulting from these exposures or if it is a matter of the relatively young age of this cohort.

RECOMMENDATIONS

A number of health physics, industrial hygiene, and epidemiologic recommendations are offered below with the goal of providing a more healthful workplace and facilitating future epidemiologic evaluation of the workforce. The majority of exposure assessment at PORTS, as at other industrial facilities, is geared towards compliance. In contrast, these recommendations highlight the need to approach the issue of occupational exposures from a public health/disease prevention perspective.

Health Physics

1. A bioassay program should be developed to characterize internal radiation exposures among the entire workforce. This is in addition to the current bioassay program which was designed for compliance. The program should be developed in cooperation with statisticians, health physicists, epidemiologists, or other health related professionals to ensure a comprehensive program that is not biased. Additionally, this program should include analyses for transuranic exposures.

2. Routine monitoring for workers most likely exposed to neutron radiation should be conducted. Although, the chronic neutron exposure may be below any level legally requiring a monitoring program, the potential for acute and/or episodic exposures is

evident. Other neutron specific recommendations can be found in the NIOSH Health Hazard Evaluation Report #HETA 96-0198-2651 [Cardarelli, 1997].

3. Administrative decisions and changes in both the external and internal Health Physics dosimetry program (e.g. doses below the limit of detection, abnormal chip ratios, and investigative reports) should be better documented and routinely reported to the workforce. This would serve to educate, inform, and solicit questions about how the changes or decisions affect a worker's dose record.

4. Historically, control dosimeters were positioned in areas of elevated background radiation which resulted in artificially low recordable doses. Efforts should be made to maintain control dosimeters in an area of normal (natural) background levels.

Industrial Hygiene

1. Workers should be identified in IH surveys when samples are taken. The worker's specific tasks during monitoring (i.e. task-based exposure monitoring), as well as their location and time spent at the task, should be recorded. When the worker being monitored is a "roving worker," such as a trades person, maintenance person, guard, supervisor, or custodian, it should be recorded as such.

2. Monitoring results below the limit of detection should also report that limit of detection. Recording results as zero and "less-than" an exposure or dose limit should be avoided.

3. Measurements should be taken on a sample of the entire workforce (workers at different potential exposure levels) to provide a better picture of the entire workforce's exposure. Included in the documentation should be an indication of what other jobs, departments, or work areas the measurements represent. The development of models that estimate exposures could also be carefully considered for various applications. This latter approach could reduce, but would not eliminate, the need for actual measurements.

4. Exposure estimates for groups of workers should be confirmed by repeat sampling on an established schedule. Any changes in work practices or processes should be evaluated to determine the impact on exposures. Even when monitoring is not necessary, a record of that decision should be made. Whenever a sample is collected, the purpose of that monitoring should appear on the record. When a sample result or a pattern of exposure is discovered that indicates a need for control, or change in work practices, its relevance to other workers or work areas should be evaluated and documented.

5. All sampling records should be systematically assembled and retained. Indexes that cross-classify sample results should be established. Examples include indexes by substance, task, building, department, calendar time, shift, time of day, personal

protective equipment, work practices, and engineering controls that were or were not in use.

6. Sampling and analytical methods should be identified in the record. In addition, all equipment calibration information should be retained.

7. When area samples are collected, a link between the location surveyed and the department numbers, job titles, and tasks should be recorded.

8. A system of collecting and archiving all health and safety program manuals, policies, and required personal protective equipment directives should be developed that can readily be used to reconstruct practices in effect at the time samples were collected.

9. A comprehensive chemical inventory for every area should be continuously maintained to assist in retrospective exposure assessments for workers, areas, and tasks.

Epidemiology

1. This epidemiologic study could serve as the beginning of a mortality surveillance program for this population. Provided adequate financial support can be identified, periodic update of the cohort mortality is also recommended. This would allow for examination of the data after adequate latency periods have been achieved, and provide

increased statistical power with each update. It would also allow the employment of new epidemiologic methodologies as they become available.

2. Recently, several papers indicate a differential in risk posed by radiation exposure experienced at older ages [Ritz et al, 1999; Richardson et al, 1998, Richardson et al, 1999]. Examinations of radiation-exposed cohorts by Ritz et al. and Richardson et al. have shown that an excess in risk of cancer deaths is apparent only when "age at exposure" is aptly weighted. Biostatisticians and epidemiologists at NIOSH are developing related methodologies that might have application for the PORTS cohort. As these methodologies become available, the study should be revisited to exam the issue of age-at-exposure.

3. Public health researchers should consider the results of this study within the context of other existing cohorts. In the present case, the feasibility of conducting an epidemiologic study at the Paducah Gaseous Diffusion Plant (PGDP) should be revisited to determine the benefits and limitations of such a study. Additionally, there would be the benefit of increased power by combining these two cohorts. Similarly, future case-control studies of lung, hematopoietic, leukemia, and stomach cancers among uranium enrichment workers should consider including workers from all three (PORTS, PGDP, and K-25) gaseous diffusion plants.

4. Information on non-mortality endpoints (cancer incidence and other diseases or disorders) associated with exposures encountered at this facility should be included in the medical records generated from medical surveillance activities involving this work force.

BIBLIOGRAPHY

ACGIH [1992a]. American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. Fluorides. Cincinnati, Ohio.

ACGIH [1992b]. American Conference of Governmental Industrial Hygienists. Documentation of the Threshold Limit Values and Biological Exposure Indices. Uranium (Natural), Soluble and Insoluble Compounds. Cincinnati, Ohio.

AEC [1968]. U.S. Atomic Energy Commission. AEC Gaseous Diffusion Plant Operations. Washington, D.C.: ORO-658

Ahrenholz SH [1996]. Lockheed Martin Utility Services, Inc., Piketon, Ohio. Center for Disease Control and Prevention, Public Health Service, National Institute for Occupational Safety and Health. HETA 94-0077-2568. Cincinnati, Ohio 1996.

Archer VE, Wagoner JK, Lundin FE [1973]. Cancer mortality among uranium mill workers. Journal of Occupational Medicine.: 15;11-14.

ATSDR [1999]. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Uranium (Update). DHHS, Public Health Service.

Beck WL, Cookfair DL, Sowder CL [1983]. Radiation dosimetry for epidemiologic lung cancer study. In: epidemiology applied to health physics. Proceedings of the sixteenth midyear topical meeting of the Health Physics Society. January 9-13, 1983; Albuquerque, New Mexico. U.S. Dept. of Energy Report.: CONF-830101, pp.436-443.

BEIR [1988]. Health risks of radon and other internally deposited alpha emitters. National Academy Press. National Research Council. Committee on the Biological Effects of Ionizing Radiation. Washington, DC.

Borders RJ [1991]. The Dictionary of Health Physics & Nuclear Sciences Terms. RSA Publications, Hebron, CT. p. 254.

Bostock [1988]. Introduction to Paducah Gaseous Diffusion Plant (PGDP) Operations. Paducah, Kentucky. KY-772.

Boyle CA, Decoufle P [1990]. National sources of vital status information: Extent of coverage and possible selectivity in reporting. American Journal of Epidemiology. 131:No.1.

Cardarelli J [1997]. Portsmouth Gaseous Diffusion Plant, Piketon, Ohio. Center for Disease Control and Prevention, Public Health Service, National Institute for Occupational Safety and Health. HETA 96-0198-2651. Cincinnati, Ohio 1997.

CDC [1990]. Centers for Disease Control. National Center for Health Statistics User's Manual. National Death Index. Department of Health Human Services. Publication No. (PHS) 90-1148.

Checkoway H, Pearce N, Crawford-Brown DJ [1988]. Radiation doses and cause-specific mortality among workers at a nuclear materials fabrication plant. American Journal of Epidemiology.:127,255-266.

Cookfair DL, Beck WL, Shy C [1983]. Lung cancer among workers in a uranium processing plant. Epidemiology Applied to Health Physics. U.S. Dept. of Energy Report. Albuquerque, New Mexico.: CONF-830101,pp. 398-406.

Cragle DL, Hollis DR, Shy CM [1984]. A retrospective cohort mortality study among workers occupationally exposed to metallic nickel powder at the Oak Ridge Gaseous Diffusion Plant. In: Nickel in the Human Environment. Sunderman FW ed. International Agency for Research on Cancer, Lyon.: IARC No. 53, pp. 57-63.

Cragle DL, McLain RW, Qualters JR [1988]. Mortality among workers at a nuclear fuels production facility. American Journal Industrial Medicine.:14,379-401.

Cragel DL, Watkins JP, Ingle JN, Robertson-Demurs K, Tankersley WG, West CM [1996]. Mortality among a cohort of white male workers at a uranium processing plant: Fernald Feed Materials Production Center, 1951-1989. Oral Presentation to Workers and Community. Harrison, Ohio, September 5, 1996.

DOE [1977a]. U.S. Department of Energy. United States Gas Centrifuge Program for Uranium Enrichment.: UCC-ND 1977 (Rev. 1).

DOE [1977b]. U.S. Department of Energy. Radiation Exposure Information Reporting System (REIRS). Office of Environmental Compliance and Overview, Operational and Environmental Safety Division.: Reports 10 through 15.

DOE [1978]. U.S. Department of Energy. United States Gaseous Diffusion Program for Uranium Enrichment. Report No. UCC-ND1978.

Dupree EA, Cragle DL, McLain RW [1987]. Mortality among workers at a uranium processing facility. The Linde Air Products Company ceramics plant, (1943-1949). Scand J Work Environment Health.:13,100-107.

Enterline PE, Marsh GM [1982]. Mortality among workers in a nickel refinery and alloy manufacturing plant in West Virginia. Journal of the National Cancer Institute. 68:925-933.

EPICURE. [1993]. Hirosoft International Corp. Seattle, Washington.

EPA [1986]. U.S. Environmental Protection Agency. Health Assessment Document for Nickel and Nickel Compounds. Office of Health and Environmental Assessment. Final Report. Washington, D.C.

ERDA [1975]. U.S. Energy Research and Development Administration. Conference on Occupational Health Experience with Uranium. Washington, D.C.: ERDA - 93.

ERDA [1977]. U.S. Energy Research and Development Administration. Final Environmental Impact Statement: Portsmouth Gaseous Diffusion Plant Site, Piketon, Ohio. Washington, D.C.: ERDA-1555 (Vol. 1).

Fisher DR, Swint MJ, Kathren RL [1990]. Technical Report: Evaluation of health effects in Sequoyah fuels corporation workers from accidental exposure to uranium hexafluoride. Pacific Northwest Laboratory. Richland, WA.: NUREG/CR-5566; PNL-7328

Floderus B, Persson T, Stenlund C, Wennberg A, Ost A, Knave B [1993]. Occupational exposure to electromagnetic fields in relation to leukemia and brain cancer. Cancer Causes and Control. 4:465-476.

Fox AS, Collier PR [1976]. Low mortality rates in industrial cohort studies due to selection for work and survival in industry. British Journal of Preventive & Social Medicine. 30:225-230.

Frome EL, Cragel DL, Watkins JP, Wing S, Shy CM, Tankersley WG, West CM [1997]. A mortality study of employees of the nuclear industry in Oak Ridge, Tennessee. Radiation Research. 148:64-80.

Frome EL, Cragle DL, McLain RW [1990]. Poisson regression analysis of the mortality among a cohort of world war ii nuclear industry workers. Radiation Research.: 123,138-152.

GAT [1959]. Goodyear Atomic Corporation: Radiation Protection Program Summary. Letter from Ben Kalmon to D.W. Doner, dated November 12, 1959; GAT-212-59-136.

GAT [1961]. Goodyear Atomic Corporation: Revised Radiation Protection Program. Letter from Ben Kalmon to D.W. Doner, dated April 20, 1961; GAT-212-61-52.

GAT [1963]. Goodyear Atomic Corporation: The GAT Urinary Bioassay Program. May, 1963.

GAT [1971a]. Goodyear Atomic Corporation: Reporting and Investigating Accidents and Radiation Exposure. Letter from C.D. Tabor to R.V. Anderson, dated March 1, 1971. GAT-212-71-41.

GAT [1971b]. Goodyear Atomic Corporation: The GAT Urinary Bioassay Program. Update of May, 1963; March, 1971.

GAT [1986a]. Goodyear Atomic Corporation: Thermoluminescent Dosimeter Response and Calibration. A.C. Bassett. (GAT-S-56). Piketon, OH.

GAT [1986b] Goodyear Atomic Corporation: IH-HP Bulletin #3-86. December 1986.

Gilbert ES, Omohundro E, Buchanan JA [1993]. Mortality of workers at the hanford site:1945-1986. Health Physics Society. 64(6):577-590.

Glenn J. [1992]. Memorandum of February 7, 1992, from J. Glenn, Chairman of the U.S. Senate Committee on Governmental Affairs, to The Honorable Louis W. Sullivan, Secretary, U.S. Department of Health and Human Services.

HASL [1958]. Health and Safety Laboratory. U.S. Atomic Energy Commission Symposium on Occupational Health Experience and Practices in Uranium Industry. New York City, New York.: HASL-58.

Hathaway GJ, Proctor NH, Hughes JP, Fischman ML [1991]. Chemical Hazards of the Workplace. New York. Van Nostrand Reinhold.: Third edition.

Hornung RW, Reed LD [1990]. Estimation of average concentration in the presence of nondetectable values. Appl. Occup. Environ. Hyg. 5:46-51.

Hornung RW, Meinhardt TJ [1987]. Quantitative risk assessment of lung cancer in U.S. uranium miners. Health Physics.: 52:417-430.

ICRP [1979]. Annals of the ICRP. Limits for Intakes of Radionuclides by Workers. Publication 30 Part I, Volume 2.

ICRP [1988]. Annals of the ICRP. Individual Monitoring for Intakes of Radionuclides by Workers: Design and Interpretation. Publication 54 Radionuclides, Monitoring Intakes.: ISBN Volume 19 No.1-3.

Lyon JS [1962]. Observations of personnel working with fluorine at a gaseous diffusion plant. Occupational Medicine. 4;199-201.

Mastromatteo E [1986]. Nickel. American Industrial Hygiene Association Journal. 10;589-601.

Mays CW, Rowland RE, Stehney AF [1985]. Cancer risk from the lifetime intake of Ra and U isotopes. Health Physics. 1985: Vol. 48,635-647.

Metzenbaum HM [1979] Letter dated November 2, 1979, from H.M. Metzenbaum, United States Senate, Committee on Labor and Human Resources, to Anthony Robbins, National Institute for Occupational Safety and Health.

MMES [1985a]. Final Safety Analysis Report. Introduction, Summary Safety Analysis, Site, and Facility and Process Description - Part 1. Portsmouth, OH: GAT/GDP-1073 (Vol. 1).

MMES [1985b]. Portsmouth Gaseous Diffusion Plant Final Safety Analysis Report. U.S. Department of Energy. Accident Analysis, Quality Assurance, Conduct of Operations and Appendices. 1985: Vol. 3. GAT/GOP-1073.

MMES [1992a]. Martin Marietta Energy Systems, Inc. PORTS Telephone Directory. Piketon, Ohio.

MMES [1992b]. Martin Marietta Energy Systems, Inc. PORTS Technical Site Information. Report number POEF-2059.

Mulhausen, JR Damiano, J [1997]. Comrehensive Exposure Assessment. In: The Occupational Environment - Its Evaluation and Control, pp. 307-309. S.R. DiNardi, Ed. American Industrial Hygiene Association, Fairfax, VA.

NIOSH [1987a]. Brown DP, Bloom T. Mortality among uranium enrichment workers. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health; Cincinnati, Ohio. 1987: NTIS PB 87-188991.

NIOSH [1987b]. Bloom TF. Health physics survey report of Portsmouth Gaseous Diffusion Plant, Piketon, Ohio. U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health. Cincinnati, Ohio. 1987 ed. (Publication IWS-121-10).

NIOSH [1993]. National Institute for Occupational Safety and Health. Epidemiologic use of nondetectable values in radiation exposure measurements. NIOSH Research Issues Workshop. National Institute for Occupational Safety and Health. Cincinnati, Ohio. 1993.

NIOSH [1994]. National Institute for Occupational Safety and Health. Health-Related Energy Research Branch. Protocol: Mortality Patterns Among Uranium Enrichment Workers at the Portsmouth Gaseous Diffusion Plant. July, 1994.

Omohundro EL, Gilbert ES [1993]. An evaluation of the adequacy of vital status follow-up in the Hanford worker mortality study. Pacific Northwest Laboratory: PNL-8981; UC-605.

ORAU [1982]. Oak Ridge Associated Universities. "Dosimetry records and radiation hazards questionnaire." [Unpublished information]. Oak Ridge Associated Universities; P.O. Box 117; Oak Ridge, TN. 37830.

Polednak AP, Frome EL [1981]. Mortality among men employed between 1943 and 1947 at a uranium-processing plant. Journal of Occupational Medicine.:23,168-178.

Proctor NH, Hughes JP, Fischman ML [1988]. Chemical Hazards of the Workplace. New York. Van Nostrand Reinhold.: Second edition.

Richardson DB, Wing S [1998]. Methods for investigating age differences in the effects of prolonged exposures. Am. J. Ind. Med. 2;33(2):123-130.

Richardson DB, Wing S [1999]. Greater sensitivity to ionizing radiation at older age: follow-up of workers at Oak Ridge National Laboratory through 1990. Int. J. Epidemiology 6;28(3):428-436.

Rinsky RA, McBrearty A. [1992]. Power Program for the IBM PC. University of Cincinnati. Unpublished.

Ritz B, Morgenstern H, Moncau J [1999]. Age at exposure modifies the effects of low-level ionizing radiation on cancer mortality in an occupational cohort. Epidemiology 3;10(2):135-140.

Roscoe RJ, Steenland K, Halperin WE [1989]. Lung cancer mortality among non smoking uranium miners exposed to radon daughters. JAMA.: 262,629-633.

Sahl, J., Kelsh, M, Greenland, S [1993]. Cohort and nested case-control studies of hematopoietic cancers and brain cancer among electric utility workers. Epidemiology 4:104-114.

SAS [1990]. SAS Institute Inc. The GLM Procedure. Note: Fourth Edition. Version 6. 1990.

Savitz DA. Loomis DP [1995]. Magnetic field exposure in relation to leukemia and brain cancer mortality among electric utility workers. Am J of Epidemiology. 141(2):123-34.

Seixas, NS Robins, TG Moulton, LH [1988]. The use of geometric and arithmetic mean exposures in occupational epidemiology. Amer. J. Ind. Med. 14:465-477.

Shleien B, [1992]. The Health Physics and Radiological Health Handbook. Second Edition. Silver Spring, MD: Scinta, Inc. Table 8.12 Typical Uranium Isotopic Abundances. p 286.

Singal M, Ahrenholz SH [1994]. Closeout letter of November 18, 1994, re HETA 94-0369 from M. Singal and S.H. Ahrenholz, Division of Surveillance, Hazard Evaluations, and Field Studies, to J. Moore, Martin Marietta Utility Services, Inc., Piketon, Ohio.

Spiers FW, Lucas HF, Rundo J, Anast GA [1983]. Leukemia incidence in U.S. dial workers. Health Physics: Vol 44 (Suppl 1) 65-72.

Steenland K, Beaumont J, Spaeth S [1990]. New developments in the life table analysis system of the National Institute for Occupational Safety and Health. Journal Occupational Medicine. 32:1091-1098.

USEC [1994]. United States Enrichment Corporation. Uranium Enrichment Processes. Portsmouth, Ohio. Martin Marietta: Rev. 4, 10/94 ed.

USUR [1981]. Hanford Environmental Health Foundation and Pacific Northwest Laboratory. Occupational exposures to uranium processes, hazards and regulations: a field study of the commercial fuel cycle. Richland, Washington. 1981: Report No. PNL-3341 and USUR-01.

USUR [1982]. Hanford Environmental Health Foundation and Pacific Northwest Laboratory. Radiological health aspects of commercial uranium conversion, enrichment, and fuel fabrication. Richland, Washington.: PNL-4438 and USUR - 03.

USUR [1983]. Hanford Environmental Health Foundation and Pacific Northwest Laboratory. Radiological health aspects of uranium milling. Richland, Washington.: PNL-4606 and USUR -04.

Voelz GL, Sullivan JB, Krieger GR. eds. [1992]. Uranium in hazardous materials toxicology, clinical principles of environmental health. Williams & Wilkins, Baltimore.: pp. 1155-1159.

Waxweiler RJ, Archer VE, Roscoe RJ [1983a]. Mortality patterns among a retrospective cohort of uranium mill workers. In: epidemiology applied to health physics: Proceedings of the Sixteenth Midyear Topical Meeting of the Health Physics Society ed. Albuquerque, New Mexico. U.S. Department of Energy.: Jan 9. CONF -830101, pp. 428-435.

Waxweiler RJ [1983b]. A modified life-table analysis system for cohort studies. Journal of Occupational Medicine. 25:115-124.

Wenzl TB [1999]. Assessment of magnetic field exposures for a mortality study at a uranium enrichment plant. Am. J. Ind. Hyg. 60:818-824.

WHO [1957]. International Classification of Diseases, 7th Revision. Volume 1. Geneva, Switzerland: World Health Organization.

WHO [1978]. International Classification of Diseases, 9th Revision. Volume 1. Geneva, Switzerland: World Health Organization.

Wiggs LD, Johnson ER, Cox-DeVore CA [1994]. Mortality through 1990 among white male workers at the Los Alamos National Laboratory: considering exposures to plutonium and external ionizing radiation. Health Physics Society. 67(6):577-588.

Wing S, Shy CM, Wood JL [1991]. Mortality among workers at Oak Ridge National Laboratory: evidence of radiation effects in follow-up through 1984. Journal of the American Medical Association. 265:1397-1402.

Wong O [1983]. Critical evaluation of epidemiologic studies of nickel-exposed workers. Final Report. Environmental Health Associates, Inc. Berkeley, California.

Table 1
External Radiation Monitoring Practices
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	1954-59	**1960-64**	**1965-69**	**1970-79**	**1980-86**	**1987-91**
Frequency	Weekly, Monthly, Quarterly	Monthly, Quarterly	Quarterly		Monthly, Quarterly, Yearly	Quarterly
% Monitored	Fraction Monitored	All radiation workers monitored		Fraction Monitored	All Monitored	
Technology	Film Dosimeters				Thermoluminescent Dosimeters	

A monitored worker is a radiation worker who wore a dosimeter.

Table 2
Distribution of Health Physics Dosimetry Data
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Data Source	# of Workers	Cumulative Absorbed Dose Statistics (mrad)						Collective Dose (mrad)
		5%	25%	Mean	Median 50%	75%	95%	
Raw Site Data	7,456	0	0	268	60	240	119	1,998,208
After exposure matrix was applied	8,678†	20	141	1,071	506	1,327	4,017	9,294,138

† Workers assigned doses based on the exposure matrix and adjusted values of zero by LOD/2.

Table 3
Initial and Final Historic Industrial Hygiene Data Available for Industrial Hygiene Job Exposure Matrix (JEM)
Airborne Uranium Metal, Fluorine/Fluoride Compounds and Nickel
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Year Built	Building No.	Master Building or Area Name	Airborne Uranium Metal results for final JEM	Fluorine & Fluoride Compounds results for final JEM	Nickel sample results for final JEM	Total Samples Available for Final JEM	Total Raw Samples Coded for 38 year study period[A]
1955	X-330	Process Building (middle - cascade enrichment process)	356	587	39	982	1221
1955	X-333	Process Building (bottom - cascade enrichment process)	174	765	14	953	1184
1954	X-342[B]	Feed Vaporization & Fluorine Generation (process feed and uranium handling facility)	351	533	19	903	923
1955	X-705	Decontamination Building	413	179	124	716	778
1956	X-326	Process Building (top - cascade enrichment process)	383	297	35	715	748
1954	X-720	Maintenance & Stores Building	279	39	287	605	652
1955	X-700	Converter Shop & Cleaning Building	141	217	169	527	604

Table 3 Continued.

Year Built	Building No.	Master Building or Area Name	Airborne Uranium Metal results for final JEM	Fluorine & Fluoride Compounds results for final JEM	Nickel sample results for final JEM	Total Samples Available for Final JEM	Total Raw Samples Coded for 38 year study period[A]
1954	X-770	Mechanical Testing	179	130		309	312
1954	X-342A[B]	Feed Vaporization and Fluorine Generation Building (AKA[C] Building X-342)	135	115		250	254
1955	X-710	Technical Services Building (laboratory, library, offices)	127	106		233	235
1958	X-344[B]	Uranium Hexafluoride Sampling Facility	22	44	3	69	73
1954	X-600	Steam Plant (See Note)		31	19	50	51
1954	X-342B	Fluorine Storage Building		27		27	34
1954	X-760	Chemical Engineering Building	7	2	9	18	21
1954	X-103	Auxiliary Office Building	6	10		16	16
		No building designation		4		4	11

Table 3 Continued.

Year Built	Building No.	Master Building or Area Name	Airborne Uranium Metal results for final JEM	Fluorine & Fluoride Compounds results for final JEM	Nickel sample results for final JEM	Total Samples Available for Final JEM	Total Raw Samples Coded for 38 year study period[A]
1956	X-744G	Bulk Storage Building - Non Uranium Enrichment Area	6	8	3	17	17
1956	X-300	Plant Control Facility (Process Monitoring Building)		2	5	7	8
NA[D]	NA	Sampling Skid Shack	6	1		7	7
1985	X-3346	Feed and Withdrawal Facility (Gaseous Centrifugal Enrichment Process)			6	6	6
NA	X-749	South Contaminate Materials Storage Yard	5			5	5
1980	X-345	Special Nuclear Materials Storage Building		3		3	4
NA	X-533	Electrical Switchyard		4		4	4
NA	Unknown	NA	2			2	3

Table 3 Continued.

Year Built	Building No.	Master Building or Area Name	Airborne Uranium Metal results for final JEM	Fluorine & Fluoride Compounds results for final JEM	Nickel sample results for final JEM	Total Samples Available for Final JEM	Total Raw Samples Coded for 38 year study period[A]
1954	X-101	Health Services Center (Dispensary)		3		3	3
1958	X-344A[B]	Uranium Hexafluoride Sampling Facility (AKA Building X-344)		2		2	2
NA	X-344G	No structure or area identified with this number		1		1	2
1954	X-533C	Test and Repair Building (located in X-533 Electrical Switch yard)		2		2	2
NA	NA	Perimeter Road		1		1	1
1958	X-344B	Maintenance Storage Building (general storage)		1		1	1
NA	X-560	No structure or area identified with this number				0	1
NA	X-745C	West Waste Process Gas Yard (outdoor storage UF_6 cylinders)	1			1	1

Table 3 Continued.

Year Built	Building No.	Master Building or Area Name	Airborne Uranium Metal results for final JEM	Fluorine & Fluoride Compounds results for final JEM	Nickel sample results for final JEM	Total Samples Available for Final JEM	Total Raw Samples Coded for 38 year study period[A]
1954	X-746	Materials Receiving - Inspection				0	1
		Totals over the study period:[E]	2593 (36.1%)	3114 (43.3%)	732 (10.2%)	6439 (89.6%)	7185 (100%)

[A] Total number of samples extracted and coded for the three contaminants of interest out of industrial hygiene records spanning the 38 year period covered by the study.
[B] Data for buildings X-342 and X-342A; X-344 and X-344A were subsequently combined as X-342 and X-344.
[C] AKA: Also known as. Some building numbers had multiple numerical designations, other variations with appended letters actually constituted different structures.
[D] NA: Not applicable. This information was not available, not applicable, or could not be discerned from other sources.
[E] The column totals represent the number of data points (and percent of total industrial hygiene data) available for each contaminant out of the total collection of industrial hygiene samples extracted and coded for the three contaminants of interest that could potentially be used in developing a Job Exposure Matrix. Fifty or more samples with numerical results were required to be available for a contaminant within a designated building throughout the time period of the study in order for assignment of non-zero contaminant values to be made in the final chemical contaminant JEM. A walk-through and determination of historic building utilization was obtained by project investigators for the buildings associated with uranium enrichment processes.
Note: Buildings identified in shaded cells were not included in the JEM developed from historic data. Additionally, X-710 and X-720 were also excluded because the majority of workers associated with these buildings worked across the site (not in these buildings.)

Table 4
Summary Tally of Initially Extracted Industrial Hygiene Data
Available 1954-1991 for All Buildings and Three Chemical Contaminants
(Airborne Uranium Metal, Fluorine/Fluoride Compounds, and Nickel)
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Numerical Status	Number of Samples	Percent of Total
Discrete (non-zero) numerical value	3983	55.4
Results reported as zero	764	10.6
Reported as greater than some value	391	5.4
Reported as less than some value	1762	24.5
Result missing from survey	137	1.9
Result reported as non-detectable	110	1.5
Miscellaneous unuseable*	38	0
Total:	7185	100

* Miscellaneous unuseable survey results included: surveys identified a contaminant but no sample for the contaminant of interest was collected; surveys that were exclusively health physics evaluations but were initially included because of the survey subject; duplicate copies of surveys; etc. This group constituted 0.005% of the information assembled. Industrial hygiene and health physics surveys could be reported on the same forms and a few of these were not eliminated by the survey file selection process. A separate evaluation of how many surveys were missed by the initial file selection process indicated that about 2% of the excluded files may have contained useable industrial hygiene data for uranium, fluorine containing compounds, and nickel. This could have produced approximately 222 additional sample results of varying quality (if the remaining 11,134 files had been coded.)

Table 5
Building X-326
Distribution of Airborne Uranium Metal
Existing Industrial Hygiene Data
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Time Period	Mean ($\mu g/m^3$)	Median ($\mu g/m^3$)	Max ($\mu g/m^3$)	Percent of samples $\geq 50 \mu g/m^3$ [A]	Number of samples
1954-1959	163.7	1.5	14305	7.5 %	212
1960-1969[B]	0.2	0.00005	5.0	0 %	82
1970-1979	24.0	0.1	974	2.2 %	45
1980-1989	32.8	1.0	1000	2.9 %	34
1990-1991	0.69	0.35	3.0	0 %	10

[A] Threshold Limit Value® for uranium metal circa 1950.
[B] The decade of the 1960's presented 55% of the sampling results for uranium as zero (45/82). The number of samples collected for any one year ranged from 0 to 12, with an average per year of 8.2 samples for uranium. Building X-326 was the last process building on site to be constructed (1956). The low airborne uranium concentrations associated with sampling results during this decade may indicate an optimal operation period following start-up and prior to potentially increasing needs for maintenance and repair associated with prolonged periods of operation or the upgrade activities of the next decade. No specific information was found regarding process activity or other factors to raise questions about the validity of these data compared to those obtained during other time periods. X-326 also produced the most highly enriched uranium, presenting the greatest radiological hazard per gram of material released.

Table 6
Distribution of Magnetic Field Exposures
Portsmouth Gaseous Diffusion Plant (PORTS)
NIOSH Surveys Conducted in 1993 and 1995

Group	Number	Units	25th percentile	median	75th percentile	mean
Measured workers	252	milligauss (mG)	0.7	1.1	2.2	3.2
Cumulative exposure for cohort	8,877	mG-years	2.9	8.9	22.6	22.0

Table 7
Distribution of Vital Status by Race and Gender
9,215 Uranium Enrichment Workers
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Male		Female		
Vital Status	**White**	**Non-White**	**White**	**Non-White**	**Total**
Alive	5,186[A] 56.3[B] 77.1[C] 73.4[D]	295 3.2 4.4 77.4	1,104 12.0 16.4 70.3	140 1.5 2.1 69.3	6,725 73.0
Unknown[E]	733 7.9 60.4 10.4	43 0.5 3.5 11.3	380 4.1 31.3 24.2	59 0.6 4.8 29.2	1,215 13.2
Dead	1,142 12.4 89.6 16.2	43 0.5 3.4 11.3	87 0.9 6.8 5.6	3 0.0 0.2 1.5	1,275 13.8
Total	7,061 76.6	381 4.1	1,571 17.0	202 2.2	9,215 100

[A] Frequency
[B] Percent of total cohort
[C] Row percent
[D] Column percent
[E] All workers were assumed alive unless confirmed deceased (death certificate or matched to NDI)

Table 8
Distribution of Years of Employment
9,215 Uranium Enrichment Workers
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Frequency	Cumulative Frequency	Percent	Cumulative Percent
<1 year	1334	1,334	14.5	14.5
1 - < 5 years	3,173	4,507	34.4	48.9
5 - < 10 years	1,531	6,038	16.6	65.5
10 - < 15 years	943	6,981	10.2	75.8
15 - < 20 years	1,022	8,003	11.1	86.8
20 - < 25 years	332	8,335	3.6	90.5
25 - < 30 years	298	8,633	3.2	93.7
30 + years	582	9,215	6.3	100.0

Table 9
Distribution of Year First Employed
9,215 Uranium Enrichment Workers
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Frequency	Cumulative Frequency	Percent	Cumulative Percent
<1955	2,994	2,994	32.5	32.5
1955 -1964	1,315	4,309	14.3	46.8
1965 - 1974	1,191	5,500	12.9	59.7
1975 - 1984	2,867	8,367	31.1	90.8
1985 - 1991	848	9,215	9.2	100.0

Table 10
Distribution of 8,877 Uranium Enrichment Workers
By Race, Gender, and Vital Status
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Total Number of Workers	Rejected	Analyzed
White Females	1,571	109	1,462
Non-White Females	202	8	194
White Males	7,061	212	6,849
Non-White Males	381	9	372
Total	9,215	338	8,877

	Total Number of Workers	Rejected	Analyzed
Alive	6,725	127	6,721[A]
Dead	1,275	64	1,088 [A]
Unknown	1,215	147	1,068
Total	9,215	338	8,877

[A] 123 deaths occurred after the study end date of 12/31/91 and therefore they were counted as alive.

Table 11
Distribution of Person-Years at Risk
Latency by Duration of Employment
8,877 Uranium Enrichment Workers
Portsmouth Gaseous Diffusion Plant (PORTS)
All Races, Both Genders
September 1954 through December 1991

Latency[A]	Duration of Employment							Total Person-Years
	1 day-7 days	7 days - 5 years	5 - 10 years	10 - 15 years	15 - 20 years	20 -25 years	> 25 years	
1 day - 7 days	112	-	-	-	-	-	-	112
7 days - 5 years	58	40,139	-	-	-	-	-	40,196
5 years - 10 years	49	17,779	21,681	-	-	-	-	39,509
10 years - 15 years	31	14,274	7,679	12,748	-	-	-	34,732
15 years - 20 years	11	11,003	4,953	2,252	7,143	-	-	25,362
20 years - 25 years	10	9,283	4,196	1,217	1,075	4,811	-	20,592
> 25 years	22	17,933	8,668	2,023	1,448	2,118	10,179	42,391
Total	293	110,410	47,176	18,240	9,667	6,929	10,179	202,894

[A] time since initial employment.

Table 12
Summary of Observed and Expected Deaths
8,877 Uranium Enrichment Workers
Portsmouth Gaseous Diffusion Plant (PORTS)
All Races, Both Genders
September 1954 through December 1991

NIOSH Death Category	Cause	Observed Deaths	Expected Deaths	SMR	95% Confidence Limits
1-2	Tuberculosis	0	3.79	[A]	-
1	Respiratory tuberculosis	0	3.34	-	-
2	Other tuberculosis	0	0.45	-	-
3-6	Malignant Neoplasm (MN) of buccal cavity and pharynx	5	10.29	0.49	0.16 - 1.14
3	MN of lip	0	0.15	-	-
4	MN of tongue	0	2.35	-	-
5	MN of other parts of buccal cavity	2	2.73	0.73	0.09 - 2.65
6	MN of pharynx	3	5.06	0.59	0.12 - 1.73
7-14	MN of digestive organs and peritoneum	76	90.44	0.84	0.66 - 1.05
7	MN of esophagus	4	9.40	0.43	0.12 - 1.09
8	MN of stomach	15	12.73	1.18	0.66 - 1.94
9	MN of intestine except rectum	30	32.61	0.92	0.62 - 1.31
10	MN of recturm	5	7.47	0.67	0.22 - 1.56
11	MN of biliary passages, liver and gall bladder	4	5.90	0.68	0.18 - 1.73
12	MN of liver not specified	0	2.30	-	-
13	MN of pancreas	17	18.49	0.92	0.54 - 1.47
14	MN of peritoneum and other and unspecified of digestive organs	1	1.54	0.65	0.02 - 3.60
15-17	MN of respiratory	112	142.04	0.79[A]	0.65 - 0.95
15	MN of larynx	5	4.88	1.02	0.33 - 2.39
16	MN of trachea, bronchus and lung	107	135.62	0.78[B]	0.64 - 0.94
17	MN of other parts of respiratory system	1	1.54	0.65	0.02 - 3.61
18	MN of Breast	6	8.83	0.68	0.25 - 1.48
19-22	MN of female genital organs	6	4.71	1.27	0.47 - 2.77
19	MN of cervix uteri	2	1.42	1.41	0.17 - 5.08
20	MN of other and unspecified parts of uterus	1	0.81	1.23	0.03 - 6.84
21	MN of ovary, fallopian tube and broad ligament	3	2.36	1.27	0.27 - 3.71

[A] Two-Sided Poisson $p<0.05$
[B] Two-Sided Poisson $p<0.01$

Table 12 Continued

NIOSH Death Category	Cause	Observed Deaths	Expected Deaths	SMR	95% Confidence Limits
22	MN of other female genital organs	0	0.11	-	-
23-24	MN of male genital organs	16	20.74	0.77	0.44 - 1.25
23	MN of prostate	16	18.92	0.85	0.48 - 1.37
24	MN of other male genital organs	0	1.82	-	-
25-26	MN of urinary organs	12	17.11	0.70	0.36 - 1.22
25	MN of kidney	7	9.58	0.73	0.29 - 1.51
26	MN of bladder and other urinary organs	5	7.53	0.66	0.21 - 1.55
27-33	MN of other and unspecified sites	44	51.46	0.86	0.62 - 1.15
27	MN of skin	8	8.90	0.9	0.39 - 1.77
28	MN of eye	0	0.25	-	-
29	MN of brain and other nervous system	9	12.69	0.71	0.32 - 1.35
30	MN of thyroid gland	0	0.69	-	-
31	MN of bone	2	1.19	1.68	0.20 - 6.05
32	MN of connective tissue and soft tissue	2	2.19	0.91	0.11 - 3.30
33	MN of other and unspecified sites (minor)	23	25.54	0.90	0.57 - 1.35
34-37	Neoplasms of lymphatic and hematopoietic tissue	36	36.39	0.99	0.69 - 1.37
34	Lymphosarcoma and reticulosarcoma	7	5.11	1.37	0.55 - 2.82
35	Hodgkin's disease	5	3.61	1.38	0.45 - 3.23
36	leukemia and aleukemia	13	14.03	0.93	0.49 - 1.58
37	Other neoplasms of lymphatic hematopoietic tissue	11	13.63	0.81	0.40 - 1.44
38-40	Benign and unspecified neoplasms	0	4.93	[A]	-
38	Benign neoplasms of the eye, brain and other parts of nervous system	0	0.70	-	-
39	Neoplasms of eye, brain, other parts of nervous system unspecified nature	0	2.31	-	-
40	Other benign and unspecified nature neoplasms	0	1.92	-	-
41	Diabetes mellitus	16	23.82	0.67	0.38 - 1.09
42-45	Diseases of the blood and blood forming organs	4	4.29	0.93	0.25 - 2.38
42	Pernicious anemia	0	0.06	-	-
43	Anemias of other and unspecified type	1	1.51	0.66	0.02 - 3.68
44	Coagulation defects, purpura, other hemorrhagic conditions	1	1.15	0.87	0.02 - 4.82
45	All other diseases of blood forming organs	2	1.57	1.27	0.15 - 4.59
46-47	Mental, and personality disorders	8	13.06	0.61	0.26 - 1.21

Table 12 Continued

NIOSH Death Category	Cause	Observed Deaths	Expected Deaths	SMR	95% Confidence Limits
46	Alcoholism	3	7.79	0.39	0.08 - 1.13
47	Other mental disorders	5	5.27	0.95	0.31 - 2.22
48-49	Nervous system and sensor organs	13	18.58	0.70	0.37 - 1.20
48	Multiple sclerosis	2	2.00	1.00	0.12 - 3.60
49	Other nervous systems and sensor organs	11	16.58	0.66	0.33 - 1.19
50-55	Diseases of the heart	406	538.40	0.75[B]	0.68 - 0.83
50	Rheumatic heart disease, including fever	3	10.58	0.28[A]	0.06 - 0.83
51	Ischemic heart disease	346	430.87	0.80[B]	0.72 - 0.89
52	Chronic disease of endocardium	3	4.34	0.69	0.14 - 2.02
53	Other myocardial degeneration	1	2.86	0.35	0.01 - 1.95
54	Hypertension with heart disease	4	11.77	0.34[A]	0.09 - 0.87
55	Other diseases of the heart	49	77.98	0.63[B]	0.46 - 0.83
56-58	Other diseases of circulatory system	75	103.64	0.72[B]	0.57 - 0.91
56	Hypertension without heart disease	3	3.87	0.77	0.16 - 2.27
57	Cerebrovascular disease	31	65.46	0.47[B]	0.32 - 0.67
58	Diseases of the arteries, veins and pulmonary circulation	41	34.30	1.20	0.86 - 1.62
59-65	Diseases of the respiratory system	45	90.56	0.50[B]	0.36 - 0.66
59	Acute respiratory infections except influenza and pneumonia	0	0.52	-	-
60	Influenza	0	0.95	-	-
61	Pneumonia (except newborn)	9	27.19	0.33[B]	0.15 - 0.63
62	Chronic and unspecified bronchitis	3	2.92	1.03	0.21 - 3.01
63	Emphysema	5	14.27	0.35[B]	0.11 - 0.82
64	Asthma	1	2.54	0.39	0.01 - 2.18
65	Pneumoconioses and other respiratory diseases	27	42.17	0.64[A]	0.42 - 0.93
66-69	Diseases of the digestive system	41	78.79	0.52[B]	0.37 - 0.70
66	Diseases of the stomach and duodenum	4	7.21	0.55	0.15 - 1.42
67	Hernia and intestinal obstruction	0	2.50	-	-
68	Cirrhosis of the liver	24	46.62	0.51[B]	0.33 - 0.77
69	Other diseases of digestive system	13	22.46	0.58[A]	0.31 - 0.99

Table 12 Continued

NIOSH Death Category	Cause	Observed Deaths	Expected Deaths	SMR	95% Confidence Limits
70-78	Diseases of the genito-urinary system	8	16.40	0.49[A]	0.21 - 0.96
70	Acute glomerulonephritis nephrotic syndrome & acute renal failure	1	1.64	0.61	0.02 - 3.38
71	Chronic & unspec. Nephritis, renal failure & other renal sclerosis	5	8.38	0.60	0.19 - 1.39
72	Infection of kidney	1	1.42	0.70	0.02 - 3.90
73	Calculi of urinary system	0	0.35	-	-
74	Hyperplasia of prostate	0	0.33	-	-
75	Other diseases of male genital organs	0	0.26	-	-
76	Diseases of the breast	0	0.00	-	-
77	Diseases of the female genital organs	0	0.10	-	-
78	Other genito-urinary system diseases	1	3.91	0.26	0.01 - 1.42
79-80	Diseases of the skin and subcutaneous tissue	3	1.17	2.55	0.53 - 7.47
79	Infections of the skin and subcutaneous tissue	1	0.39	2.59	0.07 - 14.39
80	Other diseases of the skin and subcutaneous tissue	2	0.79	2.54	0.31 - 9.16
81-83	Diseases of the musculoskeletal system and connective tissue	6	3.25	1.85	0.67 - 4.02
81	Arthritis and spondylitis	2	1.15	1.74	0.21 - 6.27
82	Osteomyelitis and periostitis	0	0.19	-	-
83	Other diseases of MS system	4	1.90	2.10	0.57 - 5.37
84	Symptoms and ill-defined conditions	8	18.72	0.43[B]	0.18 - 0.84
85-89	Accidents	71	115.94	0.61[B]	0.48 - 0.77
85	Transportation accidents	48	67.50	0.71[A]	0.52 - 0.94
86	Accidental poisoning	3	6.50	0.46	0.10 - 1.35
87	Accidental falls	5	9.50	0.53	0.17 - 1.23
88	Other accidents	14	30.33	0.46[B]	0.25 - 0.77
89	Medical complications and misadventure	1	2.12	0.47	0.01 - 2.62
90-91	Violence	34	66.37	0.51[B]	0.35 - 0.72
90	Suicide	27	45.04	0.60[B]	0.39 - 0.87
91	Homicide	7	21.33	0.33[B]	0.13 - 0.68
92-92	Other causes	37	34.46	1.07	0.76 - 1.48
3-37	All Cancers	313	382.00	0.82[B]	0.73 - 0.92
1-92	All Deaths	1088	1518.17	0.72[B]	0.67 - 0.76

Table 13
Observed and Expected Deaths From All Causes of Death
Latency by Duration of Employment
8,877 Uranium Enrichment Workers
Portsmouth Gaseous Diffusion Plant (PORTS)
All Races, Both Genders
September 1954 through December 1991

	Duration of Employment							
Latency[A]	**1 day - 7 days**	**7 days - 5 years**	**5 years - 10 years**	**10 years - 15 years**	**15 years - 20 years**	**20 years - 25 years**	**> 25 years**	**Total**
1 day - 7days	0[B] 0.24[C] -[D]	-	-	-	-	-	-	0 0.24 -
7 days-5 years	1 0.20 4.89	65 98.0 0.66[F]	-	-	-	-	-	66 98.25 0.67[F]
5 years - 10 years	1 0.25 4.01	37 50.09 0.74	29 77.10 0.38[F]	-	-	-	-	67 127.44 0.53[F]
10 years-15 years	0 0.18 -	25 57.25 0.44[F]	38 40.63 0.94	39 64.79 0.60[F]	-	-	-	102 162.85 0.63[F]
15 years-20 years	0 0.10 -	42 63.40 0.66[F]	16 38.17 0.42[F]	16 18.73 0.85[F]	19 53.89 0.35[F]	-	-	93 174.29 0.53[F]
20 years-25 years	0 0.13 -	69 74.96 0.92	38 44.16 0.86	12 15.17 0.79	10 16.17 0.62	31 50.60 0.61[F]	-	160 201.20 0.80[F]
> 25 years	0 0.47 -	192 264.81 0.73[F]	138 155.80 0.89	44 42.00 1.05	31 45.47 0.68[E]	64 56.83 1.13	131 188.52 0.69[F]	600 753.90 0.80[F]
Total	2 1.56 1.28	430 608.57 0.71[F]	259 355.86 0.73[F]	111 140.70 0.79[E]	60 115.53 0.52[F]	95 107.43 0.88	131 188.52 0.69[F]	1088 1518.17 0.72[F]

[A] time since initial employment
[B] observed deaths
[C] expected deaths
[D] standardized mortality ratio
[E] two-sided Poisson $p<0.05$
[F] two-sided Poisson $p<0.01$

Table 14
Observed and Expected Deaths Due to Stomach Cancer
By Calendar Time Periods
8,877 Uranium Enrichment Workers
Portsmouth Gaseous Diffusion Plant (PORTS)
All Races, Both Genders
September 1954 through December 1991

Sept. 1954 through 1954			1955 through 1959			1960 through 1964			1965 through 1969			1970 through 1974		
Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR
0	0.17	-	0.00	0.39	-	1	0.63	1.58	1	0.90	1.11	4	1.34	2.99

1975 through 1979			1980 through 1984			1985 through 1989			1990 through 1991		
Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR	Observed	Expected	SMR
2	1.91	1.05	5	2.62	1.91	1	3.37	0.30	1	1.55	0.64

Table 15
Observed and Expected Deaths Due to Stomach Cancers
Five Year Age Intervals
8,877 Uranium Enrichment Workers
Portsmouth Gaseous Diffusion Plant (PORTS)
All Races, Both Genders
September 1954 through December 1991

Ages	Observed	Expected	SMR
15-19	0	0.00	-
20-24	0	0.01	-
25-29	0	0.05	-
30-34	0	0.16	-
35-39	0	0.36	-
40-44	0	0.65	-
45-49	2	1.09	1.83
50-54	1	1.70	0.59
55-59	3	2.30	1.31
60-64	5	2.46	2.03
65-69	3	1.94	1.54
70-74	0	1.21	-
75-79	0	0.56	-
80-84	1	0.19	5.15
85+	0	0.04	-
Total	15	12.73	1.18

Table 16
Observed and Expected Deaths Due to Stomach Cancer
Latency by Duration of Employment
8,877 Uranium Enrichment Workers
Portsmouth Gaseous Diffusion Plant (PORTS)
All Races, Both Genders
September 1954 through December 1991

	Duration of Employment							
Latency[A]	**1 day-7 days**	**7 days-5 years**	**5 years-10 years**	**10 years-15 years**	**15 years-20 years**	**20 years-25 years**	**> 25 years**	**Total**
1 day-7 days	-	-	-	-	-	-	-	0
7 days-5 years	-	0[B] 0.57[C] 0	-	-	-	-	-	0 0.58 0
5 years-10 years	-	0 0.33 -	1 0.61 1.65	-	-	-	-	1 0.94 1.06
10 years-15 years	-	0 0.42 -	0 0.33 -	0 0.52 -	-	-	-	0 1.27 0
15 years-20 years	-	1 0.50 1.99	0 0.32 -	0 0.17 -	1 0.46 2.19	-	-	2 1.44 1.39
20 years-25 years	-	3 0.64 4.69	0 0.39 -	0 0.14 -	0 0.15 -	1 0.45 2.21	-	4 1.77 2.26
> 25 years	-	3 2.33 1.29	0 1.40 -	2 0.36 5.52	0 0.37 -	0 0.49 -	3 1.76 1.70	8 6.73 1.19
Total	-	7 4.80 1.46	1 3.05 0.33	2 1.19 1.68	1 0.97 1.03	1 0.94 1.06	3 1.76 1.70	15 12.73 1.18

[A] time since initial employment
[B] observed deaths
[C] expected deaths
[D] standardized mortality ratio

Table 17
Distribution of 8,564 Uranium Enrichment Workers
with Potential for External Radiation Exposure
By Race, Gender, and Vital Status
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Total Number of Workers	Rejected	Analyzed
White Females	1,571	265	1,306
Non-White Females	202	14	188
White Males	7,061	353	6,708
Non-White Males	381	19	362
Total	**9,215**	**651**	**8,564**
		Total Alive	7,521
		Total Deceased	1,043

Table 18
Distribution of Person-Years at Risk
Latency by Cumulative Absorbed Dose
8,564 Uranium Enrichment Workers
Portsmouth Gaseous Diffusion Plant (PORTS)
All Races, Both Genders
September 1954 through December 1991

	Cumulative Absorbed Dose (rad)						
Latency	**<0.03**	**0.03 -0.10**	**0.10 -0.50**	**0.50-1.0**	**1.0-5.0**	**>5.0**	**Total**
Under 5 years	6,347	8,913	18,257	4,981	1,470	109	40,077
5 years - 10 years	2,291	4,587	13,276	10,658	7,614	295	38,719
10 years - 15 years	1,843	2,987	9,087	8,134	11,361	430	33,842
15 years - 20 years	1,491	2,121	5,736	4,223	10,353	421	24,344
20 years - 25 years	1,244	1,748	4,674	2,770	8,476	596	19,508
25 years - 30 years	1,124	1,426	3,997	2,399	7,350	804	17,101
>30 years	1,247	1,527	4,701	2,886	9,401	1,141	20,904
Total	15,586	23,309	59,729	36,051	56,024	3,794	194,494

Table 19
Summary of Observed and Expected Deaths
All Races, Both Genders for 8,564 Uranium Enrichment Workers
with Potential for External Radiation Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

NIOSH Death Category*	Cause	Observed Deaths	Expected Deaths	SMR	95% Confidence Limits
1-2	Tuberculosis	0	3.58	-	-
3-6	MN of buccal cavity and pharynx	4	9.93	0.40	0.11 - 1.03
7-14	MN of digestive organs and peritoneum	72	86.90	0.83	0.65 - 1.04
8	MN of stomach	14	12.24	1.14	0.62 - 1.92
15-17	MN of respiratory	109	137.18	0.79[A]	0.65 - 0.96
16	MN of trachea, bronchus and lung	103	130.97	0.79[A]	0.64 - .095
18	MN of Breast	6	7.49	0.80	0.29 - 1.74
19-22	MN of female genital organs	4	3.95	1.01	0.28 - 2.59
23-24	MN of male genital organs	15	20.07	0.75	0.42 - 1.23
25-26	MN of urinary organs	12	16.50	0.73	0.37 - 1.27
27-33	MN of other and unspecified sites	42	49.51	0.85	0.61 - 1.15
31	MN of bone	2	1.14	1.75	0.21 - 6.32
32	MN of connective tissue and soft tissue	2	2.11	0.95	0.11 - 3.43
34-37	Neoplasms of lymphatic and hematopoietic tissue	34	34.97	0.97	0.67 - 1.36
34	Lymphosarcoma and reticulosarcoma	7	4.89	1.43	0.57 - 2.95
35	Hodgkin's disease	5	3.46	1.44	0.47 - 3.37
36	leukemia and aleukemia	11	13.48	0.82	0.41 - 1.46
37	Other neoplasms of lymphatic hematopoietic tissue	11	13.14	0.84	0.42 - 1.50
38-40	Benign and unspecified neoplasms	0	4.71	-[A]	-
41	Diabetes mellitus	15	22.83	0.66	0.37 - 1.08
42-45	Diseases of the blood and blood forming organs	4	4.11	0.97	0.27 - 2.49
46-47	Mental, and personality disorders	7	12.66	0.55	0.22 - 1.14
48-49	Nervous system and sensor organs	13	17.81	0.73	0.39 - 1.25
50-55	Diseases of the heart	390	518.35	0.75[B]	0.68 - 0.83
56-58	Other diseases of circulatory system	74	99.17	0.75[A]	0.59 - 0.94
58	Diseases of the arteries, veins, and pulmonary circulation	40	32.95	1.21	0.87-1.65
59-65	Diseases of the respiratory system	43	86.92	0.49[B]	0.36 - 0.67
63	Emphysema	5	13.67	0.37[A]	0.12 - 0.85
66-69	Diseases of the digestive system	39	75.86	0.51[B]	0.37 - 0.70
70-78	Diseases of the genito-urinary system	8	15.64	0.51	0.22 - 1.01
79-80	Diseases of the skin and subcutaneous tissue	2	1.11	1.80	0.22 - 6.49
81-83	Diseases of the musculoskeletal system and connective tissue	5	3.08	1.62	0.52 - 3.79
84-84	Symptoms and ill-defined conditions	8	18.08	0.44[A]	0.19 - 0.87
85-89	Accidents	68	112.28	0.61[B]	0.47 - 0.77
90-91	Violence	33	64.50	0.51[B]	0.35 - 0.72
90	Suicide	27	43.65	0.62[B]	0.41 - 0.90
91	Homicide	6	20.85	0.29[B]	0.11 - 0.63
92-92	Other causes	36	33.36	1.08	0.75 - 1.49
3-37	All Cancers	298	366.50	0.81[B]	0.72 - 0.91
1-92	All Deaths	1043	1460.56	0.71[B]	0.67 - 0.76

* ICD Codes for NIOSH Death Categories are listed in Appendix A

[A]Two-sided Poisson $p < 0.05$
[B] Two-sided Poisson $p < 0.01$

Table 20
Observed and Expected Deaths from All Causes of Death
Latency by Cumulative Absorbed Dose
All Races, Both Genders for 8,564 Uranium Enrichment Workers
with Potential for External Radiation Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Cumulative Absorbed Dose (rad)						
Latency[A]	**<0.03**	**0.03-0.10**	**0.10-0.50**	**0.50-1.0**	**1.0-5.0**	**>5.0**	**Total**
Under 5 years	11[B] 15.69[C] 0.70[D]	22 21.73 1.01	21 47.13 0.45[E]	10 13.08 0.76	3 3.40 0.88	0 0.16 -	67 101.18 0.66[E]
5 years - 10 years	9 7.28 1.24	11 13.52 0.81	23 44.11 0.52[E]	14 39.05 0.36[E]	11 25.66 0.43[E]	0 0.80 -	68 130.43 0.52[E]
10 years - 15 years	3 8.92 0.34[F]	4 12.20 0.33[F]	21 41.22 0.51[E]	24 42.20 0.57[E]	49 59.91 0.82	1 1.90 0.53	102 166.35 0.61[E]
15 years - 20 years	4 10.12 0.40	7 12.49 0.56	23 34.69 0.66[F]	15 32.12 0.47[E]	44 83.53 0.53[E]	1 3.22 0.31	94 176.17 0.53[E]
20 years - 25 years	12 11.42 1.05	17 14.74 1.15	33 39.54 0.83	23 29.28 0.79	70 99.35 0.70[G]	0 6.62 -[E]	155 200.94 0.77[E]
25 years - 30 years	14 13.59 1.03	14 17.86 0.78	30 48.13 0.62[E]	29 34.32 0.84	106 118.00 0.90	11 11.76 0.94	204 243.66 0.84[F]
>30 years	11 22.25 0.49[F]	22 29.32 0.75	66 85.78 0.77[F]	53 60.25 0.88	186 219.23 0.85[F]	15 25.00 0.60[F]	353 441.83 0.80[E]
Total	64 89.26 0.72[E]	97 121.86 0.80[F]	217 340.59 0.64[E]	168 250.30 0.67[E]	469 609.08 0.77[E]	28 49.46 0.57[E]	1043 1460.56 0.71[E]

[A]Time since initial dose
[B] Observed deaths
[C] Expected deaths
[D] Standardized mortality ratio
[E] Two-sided Poisson $p < 0.01$
[F] Two-sided Poisson $p < 0.05$

Table 21
Observed and Expected Deaths Due to Stomach Cancer
Latency by Cumulative Absorbed Dose
All Races, Both Genders for 8,564 Uranium Enrichment Workers
with Potential for External Radiation Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Cumulative Absorbed Dose (rad)						
Latency[A]	**<0.03**	**0.03-0.10**	**0.10-0.50**	**0.50-1.0**	**1.0-5.0**	**>5.0**	**Total**
Under 5 years	0[B] 0.10[C] -[D]	0 0.12 -	0 0.29 -	0 0.09 -	0 0.02 -	0 0.00 -	0 0.61 -
5 years - 10 years	0 0.05 -	0 0.09 -	1 0.32 3.15	0 0.31 -	0 0.20 -	0 0.01 -	1 0.97 1.03
10 years - 15 years	0 0.07 -	0 0.09 -	0 0.31 -	0 0.34 -	0 0.48 -	0 0.01 -	0 1.31 -
15 years - 20 years	0 0.08 -	0 0.10 -	1 0.28 3.61	0 0.27 -	2 0.71 2.83	0 0.03 -	3 1.47 2.05
20 years - 25 years	0 0.10 -	0 0.13 -	2 0.34 5.89	0 0.26 -	1 0.89 1.12	0 0.06 -	3 1.77 1.69
25 years - 30 years	0 0.12 -	2 0.16 12.57[E]	1 0.43 2.30	0 0.31 -	3 1.09 2.76	0 0.11 -	6 2.22 2.70
>30 years	0 0.19 -	0 0.25 -	0 0.76 -	0 0.52 -	1 1.94 0.52	0 0.23 -	1 3.90 0.26
Total	0 0.72 -	2 0.94 2.14	5 2.73 1.83	0 2.09 -	7 5.31 1.32	0 0.45 -	14 12.24 1.14

[A]Time since initial exposure
[B] Observed deaths
[C] Expected deaths
[D] Standardized mortality ratio
[E]Two-sided Poisson $p < 0.05$

Table 22
Observed and Expected Deaths Due to Lung Cancer
Latency by Cumulative Absorbed Dose
All Races, Both Genders for 8,564 Uranium Enrichment Workers
with Potential for External Radiation Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Cumulative Absorbed Dose (rad)						
Latency[A]	**<0.03**	**0.03-0.10**	**0.10-0.50**	**0.50-1.0**	**1.0-5.0**	**>5.0**	**Total**
Under 5 years	1[B] 0.54[C] 1.86[D]	1 0.82 1.22	1 1.69 0.59	0 0.41 -	1 0.08 12.74	0 0.00 -	4 3.53 1.13
5 years - 10 years	1 0.34 2.92	3 0.70 4.31	3 2.55 1.18	0 2.01 -	0 1.01 -	0 0.04 -	7 6.65 1.05
10 years - 15 years	0 0.57 -	1 0.72 1.38	3 2.77 1.08	3 3.00 1	4 3.51 1.14	0 0.13 -	11 10.70 1.03
15 years - 20 years	0 0.76 -	0 0.87 -	2 2.46 0.81	0 2.52 -	3 6.34 0.47	0 0.26 -	5 13.20 0.38[E]
20 years - 25 years	3 1.07 2.82	2 1.32 1.51	2 3.60 0.56	2 2.65 0.75	6 9.48 0.63	0 0.65 -	15 18.77 0.80
25 years - 30 years	1 1.47 0.68	1 1.91 0.52	8 5.27 1.52	1 3.65 0.27	7 12.91 0.54	3 1.34 2.24	21 26.56 0.79
>30 years	2 2.57 0.78	4 3.35 1.19	4 10.25 0.39[E]	4 6.85 0.58	21 25.47 0.82	5 3.07 1.64	40 51.56 0.78
Total	8 7.31 1.09	12 9.70 1.24	23 28.58 0.80	10 21.08 0.47[E]	42 58.78 0.71[E]	8 5.52 1.45	103 130.97 0.79[E]

[A] time since initial exposure
[B] observed deaths
[C] expected deaths
[D] standardized mortality ratio
[E] two-sided Poisson $p < 0.05$

Table 23
Observed and Expected Deaths Due to Hodgkin's Disease
Latency by Cumulative Absorbed Dose
All Races, Both Genders for 8,564 Uranium Enrichment Workers
with Potential for External Radiation Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Cumulative Absorbed Dose (rad)						
Latency[A]	**<0.03**	**0.03-0.10**	**0.10-0.50**	**0.50-1.0**	**1.0-5.0**	**>5.0**	**Total**
Under 5 years	0[C] 0.00[D] -[E]	1 0.13 7.56	1 0.32 3.12	0 0.10 -	0 0.03 -	0 0.00 -	2 0.68 2.93
5 years - 10 years	0 0.04 -	0 0.07 -	1 0.21 4.68	0 0.20 -	0 0.17 -	0 0.00 -	1 0.70 1.42
10 years - 15 years	0 0.04 -	0 0.05 -	0 0.16 -	0 0.14 -	1 0.26 3.83	0 0.01 -	1 0.65 1.53
15 years - 20 years	0 0.03 -	0 0.04 -	1 0.11 9.45	0 0.08 -	0 0.23 -	0 0.01 -	1 0.49 2.05
20 years - 25 years	0 0.02 -	0 0.03 -	0 0.07 -	0 0.05 -	0 0.16 -	0 0.01 -	0 0.34 -
25 years - 30 years	0 0.02 -	0 0.02 -	0 0.06 -	0 0.04 -	0 0.13 -	0 0.01 -	0 0.27 -
>30 years	0 0.02 -	0 0.02 -	0 0.06 -	0 0.04 -	0 0.16 -	0 0.02 -	0 0.32 -
Total	0 0.26 -	1 0.36 2.79	3 0.99 3.02	0 0.65 -	1 1.14 0.87	0 0.06 -	5 3.46 1.44

[A] time since initial exposure
[B] observed deaths
[C] expected deaths
[E] standardized mortality ratio

Table 24
Observed and Expected Deaths Due to Lymphoreticulosarcoma
Latency by Cumulative Absorbed Dose
All Races, Both Genders for 8,564 Uranium Enrichment Workers
with Potential for External Radiation Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Cumulative Absorbed Dose (rad)						
Latency[A]	**<0.03**	**0.03-0.10**	**0.10-0.50**	**0.50-1.0**	**1.0-5.0**	**>5.0**	**Total**
Under 5 years	1[B] 0.07[C] 14.04[D]	0 0.09 -	1 0.22 4.46	0 0.07 -	0 0.02 -	0 0.00 -	2 0.47 4.22
5 years - 10 years	0 0.04 -	0 0.05 -	1 0.19 5.35	0 0.20 -	1 0.16 6.43	0 0.00 -	2 0.63 3.17
10 years - 15 years	0 0.04 -	0 0.06 -	0 0.17 -	0 0.17 -	2 0.32 6.16	0 0.01 -	2 0.77 2.61
15 years - 20 years	0 0.05 -	0 0.06 -	0 0.16 -	1 0.13 7.72	0 0.40 -	0 0.01 -	1 0.81 1.24
20 years - 25 years	0 0.04 -	0 0.05 -	0 0.15 -	0 0.11 -	0 0.38 -	0 0.03 -	0 0.76 -
25 years - 30 years	0 0.04 -	0 0.05 -	0 0.15 -	0 0.10 -	0 0.37 -	0 0.04 -	0 0.75 -
>30 years	0 0.03 -	0 0.04 -	0 0.14 -	0 0.09 -	0 0.35 -	0 0.04 -	0 0.70 -
Total	1 0.31 3.17	0 0.41 -	2 1.18 1.70	1 0.87 1.15	3 2.00 1.50	0 0.13 -	7 4.89 1.43

[A] time since initial exposure
[B] observed deaths
[C] expected deaths
[D] standardized mortality ratio

Table 25
Observed and Expected Deaths Due to All Cancers Combined
Latency by Cumulative Absorbed Dose
All Races, Both Genders for 8,564 Uranium Enrichment Workers
with Potential for External Radiation Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Cumulative Absorbed Dose (rad)						
Latency[A]	**<0.03**	**0.03-0.10**	**0.10-0.50**	**0.50-1.0**	**1.0-5.0**	**>5.0**	**Total**
Under 5 years	3[B] 2.29[C] 1.31[D]	7 3.29 2.13	6 7.14 0.84	4 1.90 2.12	1 0.45 2.23	0 0.02 -	21 15.08 1.39
5 years - 10 years	4 1.26 3.18	3 2.46 1.22	7 8.72 0.80	3 7.23 0.42	3 4.05 0.74	0 0.15 -	20 23.87 0.84
10 years - 15 years	0 1.81 -	2 2.38 0.84	8 8.82 0.91	6 9.29 0.65	14 11.28 1.24	0 0.40 -	30 33.99 0.88
15 years - 20 years	0 2.25 -	2 2.64 0.76	9 7.42 1.21	3 7.32 0.41	12 17.75 0.68	0 0.70 -	26 38.07 0.68
20 years - 25 years	4 2.98 1.34	5 3.70 1.35	9 10.00 0.90	9 7.34 1.23	17 24.87 0.68	0 1.66 -	44 50.54 0.87
25 years - 30 years	3 3.99 0.75	5 5.10 0.98	13 14.00 0.93	7 9.69 0.72	21 33.23 0.63[E]	4 3.37 1.19	53 69.38 0.76[E]
>30 years	5 7.00 0.71	6 8.99 0.67	18 27.10 0.66	18 18.25 0.99	52 66.41 0.78	5 7.80 0.64	104 135.55 0.77[F]
Total	19 21.59 0.88	30 28.55 1.05	70 83.21 0.84	50 61.00 0.82	120 158.04 0.76[F]	9 14.11 0.64	298 366.50 0.81[F]

[A] time since initial exposure
[B] observed deaths
[C] expected deaths
[D] standardized mortality ratio
[E] two-sided Poisson p < 0/05
[F] two-sided Poisson p < 0.01

Table 26
Distribution of 6,827 Uranium Enrichment Workers
with Potential for Internal Radiation Exposure
By Race, Gender, and Vital Status
Portsmouth Gaseous Diffusion Plant (PORTS)
January 1955 through December 1991

	Total Number of Workers	Rejected	Analyzed
White Females	1,571	737	834
Non-White Females	202	84	118
White Males	7,061	1,468	5,593
Non-White Males	381	99	282
Total	**9,215**	**2,388**	**6,827**
		Total Alive	5,942
		Total Deceased	885

Table 27
Distribution of Person-Years at Risk
Latency by Exposure (Cumulative Urine Alpha Activity)
All Races, Both Genders for 6,827 Uranium Enrichment Workers
with Potential for Internal Radiation Exposure
January 1955 through December 1991

	Exposure[A] (dpm-years)						
Latency	**<2**	**2-4**	**4-8**	**8-16**	**16-32**	**>32**	**Total**
1 day - 7 days	112	0	0	0	0	0	112
7 days - 5 years	18,147	5,055	4,049	2,497	1,929	196	31,871
5 years - 10 years	10,640	4,604	5,365	4,446	3,864	1,301	30,220
10 years - 15 years	8,236	3,882	4,502	4,981	4,270	1,650	27,122
15 years - 20 years	5,061	2,187	2,638	3,521	4,053	1,764	19,225
20 y ears - 25 years	3,536	1,495	2,103	2,544	3,648	1,917	15,243
> 25 years	5,540	2,470	3,555	4,921	7,084	4,680	28,250
Total	51,272	19,293	22,211	22,910	24,849	11,507	152,042

[A] disintegrations per minute-years (dpm-years)

Table 28
Summary of Observed and Expected Deaths
All Races, Both Genders for 6,827 Uranium Enrichment Workers
with Potential for Internal Radiation Exposure
January 1955 through December 1991

NIOSH Death Category	Cause	Observed Deaths	Expected Deaths	SMR	95% Confidence Limits
1-2	Tuberculosis	0	2.90	-	-
3-6	MN of buccal cavity and pharynx	4	8.38	0.48	0.13 - 1.22
7-14	MN of digestive organs and peritoneum	58	72.74	0.80	0.61 - 1.03
8	MN of stomach	11	10.27	1.07	0.53 - 1.92
15-17	MN of respiratory	92	115.80	0.79[A]	0.64 - 0.97
15	MN of larynx	5	4.01	1.25	0.40 - 2.91
16	MN of trachea, bronchus and lung	86	110.55	0.78[A]	0.62 - 0.96
18	MN of Breast	3	4.62	0.65	013 - 1.90
19-22	MN of female genital organs	0	2.36	-	-
23-24	MN of male genital organs	13	17.05	0.76	0.41 - 1.30
25-26	MN of urinary organs	10	13.89	0.72	0.34 - 1.32
27-33	MN of other and unspecified sites	35	41.07	0.85	0.59 - 1.19
31	MN of bone	2	0.93	2.16	0.26 - 7.81
32	MN of connective tissue and soft tissue	1	1.72	0.58	0.01 - 3.23
34-37	Neoplasms of lymphatic and hematopoietic tissue	24	28.89	0.83	053 - 1.24
34	Lymphosarcoma and reticulosarcoma	6	4.04	1.49	0.54 - 3.23
35	Hodgkin's disease	3	2.79	1.08	0.22 - 3.15
36	leukemia and aleukemia	6	11.11	0.54	0.20 - 1.18
37	Other neoplasms of lymphatic hematopoietic tissue	9	10.96	0.82	0.37 - 1.56
38-40	Benign and unspecified neoplasms	0	3.85	-	-
41	Diabetes mellitus	14	18.91	0.74	0.40 - 1.24
42-45	Diseases of the blood and blood forming organs	4	3.37	1.19	0.32 - 3.03
46-47	Mental, and personality disorders	7	10.48	0.67	0.27 - 1.38
48-49	Nervous system and sensor organs	13	14.62	0.89	0.47 - 1.52
50-55	Diseases of the heart	339	436.24	0.78[B]	0.70 - 0.86
56-58	Other diseases of circulatory system	63	82.55	0.76[A]	0.59 - 0.98
58	Diseases of the arteries, veins, and pulmonary circulation	35	27.56	1.27	0.88 - 1.77
59-65	Diseases of the respiratory system	39	72.65	0.54[B]	0.38 - 0.73
63	Emphysema	4	11.52	0.35[A]	0.09 - 0.89
66-69	Diseases of the digestive system	31	63.17	0.49[B]	0.33 - 0.70
70-78	Diseases of the genito-urinary system	8	12.86	0.62	0.27 - 1.23
79-80	Diseases of the skin and subcutaneous tissue	0	0.91	-	-
81-83	Diseases of the musculoskeletal system and connective tissue	5	2.47	2.02	0.65 - 4.72
84	Symptoms and ill-defined conditions	7	14.95	0.47[A]	0.19 - 0.97
85-89	Accidents	60	89.98	0.67[B]	0.51 - 0.86
90-91	Violence	28	51.91	0.54[B]	0.36 - 0.78
90	Suicide	24	35.43	0.68	0.43 - 1.01
91	Homicide	4	16.48	0.24[B]	0.07 - 0.62
92	Other causes	28	26.95	1.04	0.69 - 1.50
3-37	All Cancers	239	304.81	0.78[B]	0.69 - 0.89
1-92	All Deaths	885	1213.62	0.73[B]	0.68 - 0.78

[A]Two-sided Poisson $p < 0.05$ [B] Two-sided Poisson $p < 0.01$

Table 29
Observed and Expected Deaths from All Causes of Death
Latency by Exposure (Cumulative Urine Alpha Activity)
All Races, Both Genders of 6,827 Uranium Enrichment Workers
with Potential for Internal Radiation Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
January 1955 through December 1991

	Exposure[B] (dpm-years)						
Latency[A]	**<2**	**2-4**	**4-8**	**8-16**	**16-32**	**> 32**	**Total**
1 day-7 days	0[C] 0.26[D] -[E]	0 0 -	0 0 -	0 0 -	0 0 -	0 0 -	0 0.26 -
7 days-5 years	36 49.70 0.72	6 14.59 0.41[F]	6 11.89 0.50	3 6.53 0.46	2 4.78 0.42	0 0.51 -	53 88.00 0.60[G]
5 years-10 years	27 39.62 0.68[F]	10 17.86 0.56	10 20.47 0.49[F]	9 16.87 0.53	5 14.24 0.35[F]	2 4.66 .43	63 113.72 0.54[G]
10 years-15 years	18 43.35 0.42[G]	11 19.87 0.50[F]	10 24.87 0.40[G]	17 26.98 0.63	21 24.22 0.87	11 9.01 1.22	88 148.28 0.59[G]
15 years-20 years	35 37.93 0.92	8 17.92 0.42[G]	14 23.32 0.60	16 29.03 0.55[F]	15 34.41 0.36[G]	7 11.64 0.60	95 156.24 0.61[G]
20 years-25 years	35 35.85 0.98	15 16.43 0.91	20 27.39 0.73	17 32.48 0.52[G]	37 43.03 0.86	20 20.25 0.99	144 175.42 0.82[G]
> 25 years	75 93.83 0.80	36 43.78 0.82	63 67.867 0.93	92 105.40 0.87	121 138.22 0.87	54 82.00 0.66[G]	441 531.57 0.84[G]
Total	226 300.53 0.75[G]	86 130.46 0.66[G]	123 175.80 0.70[G]	154 217.27 0.71[G]	201 259.39 0.77[G]	95 130.17 0.73[G]	885 1213.62 0.73[G]

[A] time since initial exposure
[B] cumulative urine alpha activity, disintegrations per minute-year (dpm-years)
[C] observed deaths
[D] expected deaths
[E] standardized mortality ratio
[F] two-sided Poisson $p < 0.05$
[G] two-sided Poisson $p < 0.01$

Table 30
Observed and Expected Deaths Due to Stomach Cancer
Latency by Exposure (Cumulative Urine Alpha Activity)
All Races, Both Genders of 6,827 Uranium Enrichment Workers
with Potential for Internal Radiation Exposure
January 1955 through December 1991

	Exposure[B] (dpm-years)						
Latency[A]	**< 2**	**2-4**	**4-8**	**8-16**	**16-32**	**> 32**	**Total**
1 day-7 days	0[C] 0.00[D] -[E]	0 0.00 -	0 0.00 -	0 0.00 -	0 0.00 -	0 0.00 -	0 0.00 -
7 days-5 years	0 0.30 0	0 0.10 -	1 0.09 11.16	0 0.05 -	0 0.03 -	0 0.00 -	1 0.57 1.75
5 years-10 years	0 0.29 -	0 0.13 -	0 0.16 -	0 0.13 -	0 0.11 -	0 0.04 -	0 0.87 -
10 years-15 years	0 0.34 -	0 0.16 -	0 0.20 -	1 0.21 4.71	0 0.19 -	0 0.07 -	1 1.18 0.85
15 years-20 years	0 0.31 -	1 0.15 6.55	0 0.20 -	0 0.25 -	0 0.29 -	0 0.11 -	1 1.32 0.76
20 years-25 years	0 0.31 -	0 0.15 -	0 0.24 -	0 0.30 -	0 0.39 -	1 0.18 5.57	1 1.56 0.64
> 25 years	2 0.81 2.46	1 0.38 2.58	1 0.60 1.66	0 0.99 -	2 1.26 1.59	1 0.77 1.59	7 4.77 1.47
Total	2 2.36 0.85	2 1.08 1.85	2 1.50 1.33	1 1.87 0.53	2 2.28 0.88	2 0.17 1.71	11 10.27 1.07

[A] time since initial exposure
[B] cumulative urine alpha activity, disintegrations per minute-years (dpm-years)
[C] observed deaths
[D] expected deaths
[E] standardized mortality ratio

Table 31
Observed and Expected Deaths Due to Lung Cancer
Latency by Exposure (Cumulative Urine Alpha Activity)
All Races, Both Genders of 6,827 Uranium Enrichment Workers
with Potential for Internal Radiation Exposure
January 1955 through December 1991

	Exposure[B] (dpm-years)						
Latency[A]	**< 2**	**2-4**	**4-8**	**8-16**	**16-32**	**> 32**	**Total**
1 Day - 7 Days	0[C] 0.01[D] -[E]	0 0 -	0 0 -	0 0 -	0 0 -	0 0 -	0 0.01 -
7 days - 5 years	2 2.90 0.95	0 0.54 -	0 0.39 -	0 0.16 -	0 0.11 -	0 0.02 -	2 3.33 0.60
5 years - 10 years	5 2.38 2.10	1 1.14 0.88	0 1.13 -	0 0.76 -	0 0.58 -	1 0.20 5.11	7 6.17 1.13
10 years - 15 years	2 3.10 0.64	0 1.49 -	1 1.76 0.57	2 1.76 1.14	2 1.42 1.42	1 0.52 1.92	8 10.04 0.80
15 years - 20 years	4 3.05 1.31	0 1.47 -	0 1.91 -	0 2.33 -	2 2.69 0.74	0 1.03 -	6 12.49 0.48
20 years - 25 years	3 3.37 0.89	2 1.58 1.27	1 2.63 0.38	3 3.26 0.92	4 4.31 0.93	3 1.97 1.52	16 17.12 0.93
> 25 years	10 10.35 0.97	3 4.87 0.62	6 7.69 0.78	11 11.95 0.92	12 16.43 0.73	5 10.09 0.50	47 61.39 0.77
Total	26 24.36 1.07	6 11.10 0.54	8 15.52 0.52	16 20.22 0.79	20 25.53 0.78	10 13.83 0.72	86 110.56 0.78[F]

[A] time since initial exposure
[B] cumulative urine alpha activity, disintegrations per minute-years (dpm-years)
[C] observed deaths
[D] expected deaths
[E] standardized mortality ratio
[F] two-sided Poisson $p < 0.05$

Table 32
Observed and Expected Deaths Due to Hodgkin's Disease
Latency by Exposure (Cumulative Urine Alpha Activity)
All Races, Both Genders of 6,827 Uranium Enrichment Workers
with Potential for Internal Radiation Exposure
January 1955 through December 1991

	Exposure[B] (dpm-years)						
Latency[A]	**< 2**	**2-4**	**4-8**	**8-16**	**16-32**	**> 32**	**Total**
1 day-7 days	0[C] 0[D] -[E]	0 0 -	0 0 -	0 0 -	0 0 -	0 0 -	0 0 -
7 days-5 years	0 0.28 -	1 0.09 10.60	0 0.09 -	0 0.06 -	0 0.05 -	0 0 -	1 0.58 1.74
5 years-10 years	0 0.17 -	0 0.07 -	0 0.09 -	1 0.10 9.96	0 0.10 -	0 0.03 -	1 0.57 1.74
10 years-15 years	0 0.14 -	0 0.06 -	0 0.08 -	0 0.10 -	0 0.11 -	0 0.05 -	0 0.53 -
15 years-20 years	1 0.09 11.56	0 0.04 -	0 0.05 -	0 0.07 -	0 0.09 -	0 0.04 -	1 0.39 2.59
20 years-25 years	0 0.05 -	0 0.02 -	0 0.04 -	0 0.05 -	0 0.07 -	0 0.04 -	0 0.27 -
> 25 years	0 0.08 -	0 0.04 -	0 0.06 -	0 0.08 -	0 0.12 -	0 0.07 -	0 0.45 -
Total	1 0.81 1.23	1 0.33 3.05	0 0.40 -	1 0.47 2.15	0 0.54 -	0 0.24 -	3 2.79 1.08

[A] time since initial exposure
[B] cumulative urine alpha activity, disintegrations per minute-years (dpm-years)
[C] observed deaths
[D] expected deaths
[E] standardized mortality ratio

Table 33
Observed and Expected Deaths Due to All Cancers Combined
Latency by Exposure (Cumulative Urine Alpha Activity)
All Races, Both Genders of 6,827 Uranium Enrichment Workers
with Potential for Internal Radiation Exposure
January 1955 through December 1991

	Exposure[B] (dpm-years)						
Latency[A]	**< 2**	**2-4**	**4-8**	**8-16**	**16-32**	**> 32**	**Total**
1 day-7 days	0[C] 0.04[D] -[E]	0 0.00 -	0 0 -	0 0 -	0 0 -	0 0 -	0 0.04 -
7 days-5 years	9 8.08 1.11	1 2.21 0.45	2 1.73 1.16	1 0.66 1.16	0 0.62 -	0 0.08 -	13 13.57 0.96
5 years-10 years	8 8.22 1.00	4 3.60 1.11	2 3.78 0.53	4 2.81 1.43	0 2.23 -	1 0.74 1.35	19 21.17 0.90
10 years-15 years	6 9.67 0.62	1 4.43 0.23	3 5.30 0.57	5 5.32 0.94	4 4.37 0.92	3 1.61 1.86	22 30.70 0.72
15 years-20 years	11 8.95 1.23	2 4.13 0.48	4 5.35 0.75	2 6.33 0.32	4 7.27 0.55	0 2.77 -	23 34.80 0.66[F]
20 years-25 years	10 9.43 1.06	3 4.26 0.70	5 7.04 0.71	4 8.37 0.48	10 10.99 0.91	6 5.00 1.20	38 45.10 0.84
> 25 years	21 28.11 0.75	10 12.95 0.77	17 20.24 0.84	31 31.08 1.00	34 41.87 0.81	11 25.17 0.44[G]	124 159.43 0.78[G]
Total	65 72.31 0.90	21 31.58 0.66	33 43.43 0.76	47 54.77 0.86	52 67.34 0.77	21 35.38 0.59[F]	239 304.81 0.78[G]

[A] time since initial exposure
[B] cumulative urine alpha activity, disintegrations per minute-years (dpm-years)
[C] observed deaths
[D] expected deaths
[E] standardized mortality ratio
[F] two-sided Poisson $p < 0.05$
[G] two-sided Poisson $p < 0.01$

Table 34
Observed and Expected Deaths Due to Lymphoreticulosarcoma
Latency by Exposure (Cumulative Urine Alpha Activity)
All Races, Both Genders of 6,827 Uranium Enrichment Workers
with Potential for Internal Radiation Exposure
January 1955 through December 1991

	Exposure[B] (dpm-years)						
Latency[A]	**< 2**	**2-4**	**4-8**	**8-16**	**16-32**	**> 32**	**Total**
1 day-7 days	0[C] 0[D] -[E]	0 0 -	0 0 -	0 0 -	0 0 -	0 0 -	0 0 -
7 days-5 years	1 0.21 4.66	0 0.07 -	0 0.06 -	0 0.04 -	0 0.03 -	0 0 -	1 0.42 2.36
5 years-10 years	0 0.17 -	1 0.07 13.81	0 0.09 -	2 0.10 20.33[G]	0 0.09 -	0 0.03 -	3 0.56 5.36[F]
10 years-15 years	0 0.17 -	0 0.07 -	1 0.10 10.25	0 0.13 -	0 0.14 -	0 0.05 -	1 0.67 1.50
15 years-20 years	0 0.15 -	0 0.07 -	1 0.09 10.96	0 0.13 -	0 0.17 -	0 0.07 -	1 0.68 1.47
20 years-25 years	0 0.12 -	0 0.06 -	0 0.09 -	0 0.12 -	0 0.16 -	0 0.08 -	0 0.63 -
> 25 years	0 0.18 -	0 0.09 -	0 0.13 -	0 0.21 -	0 0.29 -	0 0.17 -	0 1.07 -
Total	1 1.02 0.98	1 0.43 2.33	2 0.57 3.49	2 0.73 2.76	0 0.89 -	0 0.41 -	6 4.04 1.49

[A] time since initial exposure
[B] cumulative urine alpha activity, disintegrations per minute-years (dpm-years)
[C] observed deaths
[D] expected deaths
[E] standardized mortality ratio
[F] two-sided Poisson $p < 0.05$
[G] two-sided Poisson $p < 0.01$

Table 35
Distribution of 1,446 Uranium Enrichment Workers
with Potential for Fluorine and Fluoride Compound Exposure
By Race, Gender, and Vital Status
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Total Number of Workers	Rejected	Analyzed
White Females	1,571	1,452	119
Non-White Females	202	161	41
White Males	7,061	5,868	1,193
Non-White Males	381	288	93
Total	**9,215**	**7,769**	**1,446**
		Total Alive	1,307
		Total Deceased	139

Table 36
Distribution of Person-Years at Risk
Latency by Exposure
All Races, Both Genders of 1,446 Uranium Enrichment Workers
with Potential for Fluorine and Fluoride Compound Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
January 1955 through December 1991

	Exposure[B] (score-years)				
Latency[A]	**< 30**	**30 to <60**	**60 to <120**	**=>120**	**Total**
1 day - 7 days	23	0	0	0	23
7 days - 5 years	2,931	1,434	1,207	1,188	6,762
5 years -10 years	1,719	1,203	1,358	2,101	6,383
10 years - 15 years	1,429	980	1,190	2,111	5,711
15 years - 20 years	946	726	783	1,085	3,541
20 years - 25 years	776	593	591	586	2,548
≥ 25 years	1,442	889	931	1,198	4,461
Total	9,270	5,828	6,061	8,271	29,432

[A] time since initial exposure
[B] exposure = a "score-year" based on the percent of IH samples taken within a particular area and calendar year where the exposure to fluorine or fluoride compounds exceeded 2500 micrograms per cubic meter.

Table 37
Summary of Observed and Expected Deaths
All Races, Both Genders of 1,446 Uranium Enrichment Workers
with Potential for Fluorine and Fluoride Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

NIOSH Death Category	Cause	Observed Deaths	Expected Deaths	SMR	95% Confidence Limits
1-2	Tuberculosis	0.00	0.44	-	-
3-6	MN of buccal cavity and pharynx	0	1.43	-	-
7-14	MN of digestive organs and peritoneum	8	11.84	0.68	0.29 - 1.33
8	MN of stomach	3	1.67	1.80	0.37 - 5.26
15-17	MN of respiratory	9	19.58	0.46[A]	0.21 - 0.87
16	MN of trachea, bronchus and lung	9	18.69	0.48[A]	0.22 - 0.91
18	MN of Breast	1	0.57	1.74	0.04 - 9.66
19-22	MN of female genital organs	0	0.28	-	-
23-24	MN of male genital organs	0	2.55	-	-
25-26	MN of urinary organs	2	2.24	0.89	0.11 - 3.23
27-33	MN of other and unspecified sites	2	7.09	0.28	0.03 - 1.02
31	MN of bone	0	0.16	-	-
32	MN of connective tissue and soft tissue	0	0.31	-	-
34-37	Neoplasms of lymphatic and hematopoietic tissue	4	4.86	0.82	0.22 - 2.10
34	Lymphosarcoma and reticulosarcoma	1	0.64	1.56	0.04 - 8.69
35	Hodgkin's disease	0	0.49	-	-
36	leukemia and aleukemia	2	1.85	1.08	0.13 - 3.89
37	Other neoplasms of lymphatic hematopoietic tissue	1	1.88	0.53	0.01 - 2.95
38-40	Benign and unspecified neoplasms	0	0.63	-	-
41	Diabetes mellitus	3	3.14	0.96	0.20 - 2.80
42-45	Diseases of the blood and blood forming organs	1	0.55	1.80	0.05 - 10.02
46-47	Mental, and personality disorders	3	1.98	1.52	0.31 - 4.43
48-49	Nervous system and sensor organs	1	2.44	0.41	0.01 - 2.28
50-55	Diseases of the heart	47	68.87	0.68[B]	0.50 - 0.91
56-58	Other diseases of circulatory system	7	12.56	0.56	0.22 - 1.15
59-65	Diseases of the respiratory system	7	11.04	0.63	0.25 - 1.31
63	Emphysema	0	1.63	-	-
66-69	Diseases of the digestive system	5	10.89	0.46	0.08 - 1.07
70-78	Diseases of the genito-urinary system	2	1.10	1.00	0.12 - 3.62
79-80	Diseases of the skin and subcutaneous tissue	0	0.14	-	-
81-83	Diseases of the musculoskeletal system and connective tissue	1	0.40	2.48	0.06 - 13.80
84-84	Symptoms and ill-defined conditions	2	2.69	0.74	0.09 - 2.68
85-89	Accidents	15	17.56	0.85	0.48 - 1.41
90-91	Violence	12	10.70	1.12	0.58 - 1.96
90	Suicide	9	6.84	1.32	0.60 - 2.50
91	Homicide	3	3.86	0.78	0.16 - 2.27
92	Other causes	7	5.35	1.31	0.52 - 2.69
3-37	All Cancers	26	50.43	0.52[B]	0.34 - 0.76
1-92	All Deaths	139	201.81	0.69[B]	0.58 - 0.81

[A] two-sided Poisson $p < 0.05$ [B] two-sided Poisson $p < 0.01$

Table 38
Observed and Expected Deaths Due to Stomach Cancer
Latency by Exposure
All Races, Both Genders of 1,446 Uranium Enrichment Workers
with Potential for Fluorine and Fluoride Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
January 1955 through December 1991

	Exposure[B] (score-years)				
Latency[A]	**< 30**	**30 to <60**	**60 to <120**	**>120**	**Total**
1 day- 7 days	0[C] 0.0[D] -[E]	0 0.0 -	0 0.0 -	0 0.0 -	0 0.0 -
7 days - 5 years	0 0.05 -	0 0.03 -	0 0.02 -	0 0.02 -	0 0.11 -
5 years - 10 years	0 0.04 -	0 0.04 -	0 0.05 -	0 0.05 -	0 0.17 -
10 years - 15 years	1 0.05 19.27	1 0.05 21.46	0 0.06 -	0 0.08 -	2 0.24 8.40[F]
15 years - 20 years	0 0.05 -	1 0.06 17.79	0 0.06 -	0 0.08 -	1 0.25 4.00
20 years - 25 years	0 0.06 -	0 0.06 -	0 0.06 -	0 0.06 -	0 0.24 -
> 25 years	0 0.18 -	0 0.13 -	0 0.15 -	0 0.20 -	0 0.66 -
Total	1 0.43 2.33	2 0.36 5.56	0 0.39 -	0 0.49 -	3 1.67 1.80

[A] time since initial exposure
[B] cumulative exposure score to fluorine and fluoride
[C] observed deaths
[D] expected deaths
[E] standardized mortality ratio

Table 39
Distribution of 1,832 Uranium Enrichment Workers
with Potential Uranium Metal Exposure
By Race, Gender, and Vital Status
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Total Number of Workers	Rejected	Analyzed
White Females	1,571	1,399	172
Non-White Females	202	155	47
White Males	7,061	5,550	1,511
Non-White Males	381	279	102
Total	**9,215**	**7,383**	**1,832**
		Total Alive	1,642
		Total Deceased	190

Table 40
Distribution of Person-Years at Risk
Latency by Exposure
All Races, Both Genders for 1,832 Uranium Enrichment Workers
with Potential for Uranium Metal Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
January 1955 through December 1991

	Exposure[B] (score-years)				
Latency[A]	**<50**	**50 to <100**	**100 to <200**	**=>200**	**Total**
1 day - 7 days	30	0	0	0	30
7 days - 5 years	4,848	1,973	1,360	664	8,846
5 years - 10 years	2,695	1,551	2,261	1,982	8,490
10 years - 15 years	2,198	1,277	1,954	2,217	7,648
15 years - 20 years	1,442	846	1,300	1,571	5,161
20 years - 25 years	1,200	714	989	1,160	4,064
≥25 years	2,286	1,350	1,694	2,603	7,935
Total	14,702	7,714	9,560	10,199	42,176

[A] time since initial exposure
[B] exposure = a "score-year" based on the percent of IH samples taken within a particular area and calendar year where the exposure to uranium exceeded 50 micrograms per cubic meter.

Table 41
Summary of Observed and Expected Deaths
All Races, Both Genders for 1,832 Uranium Enrichment Workers
with Potential for Uranium Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

NIOSH Death Category	Cause	Observed Deaths	Expected Deaths	SMR	95% Confidence Limits
1-2	Tuberculosis	0	0.64	-	-
3-6	MN of buccal cavity and pharynx	0	1.96	-	-
7-14	MN of digestive organs and peritoneum	10	16.26	0.62	0.29 - 1.13
8	MN of stomach	3	2.29	1.31	0.27 - 3.84
15-17	MN of respiratory	11	26.78	0.41[A]	0.20 - 0.74
16	MN of trachea, bronchus and lung	11	25.57	0.43[A]	0.21 - 0.77
18	MN of Breast	1	0.98	1.02	0.03 - 5.65
19-22	MN of female genital organs	0	0.49	-	-
23-24	MN of male genital organs	1	3.39	0.29	0.01 - 1.64
25-26	MN of urinary organs	2	3.06	0.65	0.08 - 2.36
27-33	MN of other and unspecified sites	5	9.84	0.51	0.16 - 1.19
31	MN of bone	0	0.22	-	-
32	MN of connective tissue and soft tissue	0	0.43	-	-
34-37	Neoplasms of lymphatic and hematopoietic tissue	6	6.80	0.88	0.32 - 1.92
34	Lymphosarcoma and reticulosarcoma	1	0.91	1.09	0.03 - 6.08
35	Hodgkin's disease	0	0.72	-	-
36	leukemia and aleukemia	3	2.59	1.16	0.24 - 3.39
37	Other neoplasms of lymphatic hematopoietic tissue	2	2.57	0.78	0.09 - 2.81
38-40	Benign and unspecified neoplasms	0	0.88	-	-
41	Diabetes mellitus	4	4.35	0.92	0.25 - 2.35
42-45	Diseases of the blood and blood forming organs	2	0.76	2.63	0.32 - 9.51
46-47	Mental, and personality disorders	3	2.68	1.12	0.23 - 3.28
48-49	Nervous system and sensor organs	2	3.39	0.59	0.07 - 2.13
50-55	Diseases of the heart	66	94.57	0.70[A]	0.54 - 0.89
56-58	Other diseases of circulatory system	12	17.16	0.70	0.36 - 1.22
59-65	Diseases of the Respiratory System	8	15.02	0.53	0.23 - 1.05
63	Emphysema	0	2.22	-	-
66-69	Diseases of the digestive system	8	15.12	0.53	0.23 - 1.04
70-78	Diseases of the genito-urinary system	2	2.79	0.72	0.09 - 2.59
73	Calculi of urinary system	0	0.05	-	-
74	Hyperplasia of prostate	0	0.04	-	-
79-80	Diseases of the skin and subcutaneous tissue	0	0.20	-	-
81-83	Diseases of the musculoskeletal system and connective tissue	1	0.57	1.76	0.04 - 9.75
84	Symptoms and ill-defined conditions	3	3.67	0.82	0.17 - 2.39
85-89	Accidents	21	25.18	0.83	0.52 - 1.28
90-91	Violence	14	14.61	0.96	0.52 - 1.61
90	Suicide	10	9.53	1.05	0.50 - 1.93
91	Homicide	4	5.08	0.79	0.21 - 2.01
92	Other causes	8	7.09	1.13	0.49 - 2.22
3-37	All Cancers	36	69.55	0.52[A]	0.36 - 0.72
1-92	All Deaths	190	278.22	0.68[A]	0.59 - 0.79

[A] two-sided Poisson $p < 0.01$

Table 42
Distribution of 465 Uranium Enrichment Workers
with Potential Nickel Exposure
By Race, Gender, and Vital Status
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

	Total Number of Workers	Rejected	Analyzed
White Females	1,571	1,544	27
Non-White Females	202	186	16
White Males	7,061	6,686	375
Non-White Males	381	334	47
Total	**9,215**	**8,750**	**465**
		Total Alive	430
		Total Deceased	35

Table 43
Distribution of Person-Years at Risk
Latency by Exposure
All Races, Both Genders for 465 Uranium Enrichment Workers
with Potential for Nickel Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
January 1955 through December 1991

	Exposure (mg Ni / m^3 exposure-days)				
Latency[A]	**<25**	**25 to <50**	**50 to <100**	**=>100**	**Total**
1 day - 7 days	8	0	0	0	8
7 days - 5 years	479	326	437	1,050	2,292
5 years - 10 years	380	257	334	1,297	2,268
10 years - 15 years	327	217	272	1,225	2,041
15 years - 20 years	126	117	58	342	643
20 years - 25 years	80	95	25	70	270
≥25 years	169	185	51	150	555
Total	1,569	1,197	1,177	4,134	8,077

[A] time since initial exposure

Table 44
Summary of Observed and Expected Deaths
All Races, Both Genders for 465 Uranium Enrichment Workers
with Potential for Nickel Exposure
Portsmouth Gaseous Diffusion Plant (PORTS)
January 1955 through December 1991

NIOSH Death Category	Cause	Observed Deaths	Expected Deaths	SMR	95% Confidence Limits
1-2	Tuberculosis	0.00	0.09	-	-
3-6	MN of buccal cavity and pharynx	0	0.30	-	-
7-14	MN of digestive organs and peritoneum	2	2.55	0.78	0.09 - 2.83
8	MN of stomach	0	0.35	-	-
15-17	MN of respiratory	3	4.09	0.73	0.15 - 2.15
16	MN of trachea, bronchus and lung	3	3.90	0.77	0.16 - 2.25
18	MN of Breast	0	0.28	-	-
19-22	MN of female genital organs	0	0.15	-	-
23-24	MN of male genital organs	0	0.54	-	-
25-26	MN of urinary organs	0	0.46	-	
27-33	MN of other and unspecified sites	2	1.59	1.26	0.15 - 4.54
31	MN of bone	0	0.04	-	-
32	MN of connective tissue and soft tissue	0	0.08	-	-
34-37	Neoplasms of lymphatic and hematopoietic tissue	1	1.09	0.91	0.02 - 5.07
34	Lymphosarcoma and reticulosarcoma	0	0.13	-	-
35	Hodgkin's disease	0	0.11	-	-
36	Leukemia and aleukemia	1	0.42	2.36	0.06 - 13.14
37	Other neoplasms of lymphatic hematopoietic tissue	0	0.44	-	-
38-40	Benign and unspecified neoplasms	0	0.14	-	-
41	Diabetes mellitus	0	0.73	-	-
42-45	Diseases of the blood and blood forming organs	0	0.14	-	-
46-47	Mental, and personality disorders	2	0.52	3.85	0.47 - 13.88
48-49	Nervous system and sensor organs	0	0.58	-	-
50-55	Diseases of the heart	11	14.15	0.78	0.39 - 1.39
56-58	Other diseases of circulatory system	2	2.73	0.73	0.09 - 2.64
56	Hypertension without heart disease	1	0.11	8.96	0.23 - 49.78
57	Cerebrovascular disease	0	1.70	-	-
58	Diseases of the arteries, veins and pulmonary circulation	1	0.92	1.08	0.03 - 6.01
59-65	Diseases of the respiratory system	1	2.40	0.42	0.01 - 2.31
63	Emphysema	0	0.32	-	-
66-69	Diseases of the digestive system	1	2.36	0.42	0.01 - 2.36
70-78	Diseases of the genito-urinary system	0	0.44	-	-
79-80	Diseases of the skin and subcutaneous tissue	0	0.03	-	-
81-83	Diseases of the musculoskeletal system and connective tissue	1	0.10	10.22	0.26 - 56.76
84	Symptoms and ill-defined conditions	0	0.68	-	-
85-89	Accidents	3	4.76	0.63	0.13 - 1.84
90-91	Violence	4	3.23	1.24	0.34 - 3.17
90	Suicide	2	1.88	1.06	0.13 - 3.83
91	Homicide	2	1.34	1.49	0.18 - 5.38
92	Other causes	2	1.71	1.17	0.14 - 4.21
3-37	All Cancers	8	11.07	0.72	0.31 - 1.42
1-92	All Deaths	35	45.85	0.76	0.53 - 1.06

Table 45
Radiation Exposure-Response Analyses
107 Lung Cancer Deaths
No Lag Period
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Absorbed Dose (rad)

Model	β Coefficient	Relative Risk	Std. Error	Likelihood Ratio Chi-square	p value
Cumulative exposure (log-linear)	-0.00008	0.92 at 1 rad	0.00017	0.220	>0.5
Log (cumulative exposure + 1)	-0.102	0.49 at 1 rad	0.054	3.62	0.057
Cumulative exposure (linear)	-0.000054	0.95 at 1 rad	0.00015	0.161	>0.5
Categorical cumulative exposure (log-linear)				**Wald Test (z)**	
0-100 mrad	-0.114	0.89	0.623	-0.183	>0.5
100 - 500 mrad	-0.341	0.71	0.631	-0.540	>0.5
0.5 - 1 rad	-0.989	0.37	0.672	-1.471	0.141
>1 rad	-0.254	0.78	0.670	-0.379	>0.5
Cumulative exposure (linear)	-0.0032		0.0018	-1.79	0.072
Cumulative exposure * age	0.00005		0.00003	1.787	0.074
Cumulative exposure (age <60 yrs)	-0.0004	0.67 at 1 rad	0.00033	-1.214	0.225
Cumulative exposure (age >60 yrs)	0.0005	1.65 at 1 rad	0.00039	1.282	0.20

Alpha activity in urine (dpm-year)

Cumulative exposure (linear)	0.0027	†	0.0085	0.318	>0.5
Cumulative exposure (log-linear	0.0037		0.0071	.0513	>0.5

† Because internal exposure was measured as alpha activity in urine output rather than actual or estimated exposure, it is not useful to calculate risk per unit.

Table 46
External Radiation Exposure-Response Analyses
107 Lung Cancer Deaths
Five Year Lag Period
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Absorbed Dose (rad)

Model	β Coefficient	Relative Risk	Std. Error	Likelihood Ratio Chi-square	p value
Cumulative exposure (log-linear)	-0.00006	0.94 at 1 rad	0.00018	0.098	>0.5
Log (cumulative exposure + 1)	-0.071	0.61 at 1 rad	0.050	2.042	0.153
Cumulative exposure (linear)	-0.00004	0.96 at 1 rad	0.00016	0.070	>0.5
Categorical cumulative exposure (log-linear)				**Wald Test (z)**	
0-100 mrad	-0.014	0.99	0.453	-0.031	>0.5
100 - 500 mrad	-0.045	0.96	0.449	-0.101	>0.5
0.5 - 1 rad	-0.779	0.46	0.516	-1.509	0.131
>1 rad	-0.067	0.94	0.519	-0.128	>0.5
Cumulative exposure (linear)	-0.0035		0.0018	-1.79	0.074
Cumulative exposure * age	0.00005		0.00003	1.787	0.073
Cumulative exposure (age <60 yrs)	-0.00045	0.64 at 1 rad	0.00037	-1.223	0.221
Cumulative exposure (age >60 yrs)	0.00059	1.80 at 1 rad	0.00043	1.380	0.167

Table 47
External Radiation Exposure-Response Analyses
107 Lung Cancer Deaths
Ten Year Lag Period
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Absorbed Dose (rad)

Model	β Coefficient	Relative Risk	Std. Error	Likelihood Ratio Chi-square	p value
Cumulative exposure (log-linear)	-0.00005	1.05 at 1 rad	0.0002	0.052	>0.5
Log (cumulative exposure + 1)	-0.063	0.65 at 1 rad	0.048	1.756	0.185
Cumulative exposure (linear)	-0.00003	1.03 at 1 rad	0.0002	0.031	>0.5
Categorical cumulative exposure (log-linear)				**Wald Test (z)**	
0-100 mrad	0.074	1.08	0.365	0.204	>0.5
100 - 500 mrad	-0.216	0.81	0.379	-0.570	>0.5
0.5 - 1 rad	-0.662	0.52	0.432	-1.533	0.125
>1 rad	-0.013	0.99	0.475	-0.027	>0.5
Cumulative exposure (linear)	-0.0046		0.0023	3.873	0.049
Cumulative exposure*age	0.000072		0.000036	4.072	0.044
Cum exposure (age <60 yrs)	-0.00063	0.53 at 1 rad	0.00048	1.303	0.193
Cum exposure (age >60 yrs)	0.0009	2.46 at 1 rad	0.00053	1.685	0.092

Table 48
External Radiation Exposure-Response Analyses
107 Lung Cancer Deaths
Fifteen and Twenty Year Lag Periods
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Lag period = 15
Absorbed Dose (rad)

Model	β Coefficient	Relative Risk	Std. Error	Wald Test (z)	p value
Cumulative exposure (log-linear)	-0.0001	0.90 at 1 rad	0.0003	-0.362	>0.5
Cum exposure (log-linear)	-0.0043		0.0028	-1.522	0.128
Cumulative exposure * age	0.00007		0.00004	1.506	0.132

Lag period = 20
Absorbed Dose (rad)

Cumulative exposure (log-linear)	-0.00026	0.77 at 1 rad	0.00037	0.491	0.478
Cumulative exposure (log-linear)	-0.0067		0.0037	-1.795	0.073
Cumulative exposure * age	0.0001		0.00006	1.751	0.080

Table 49
External Radiation Exposure-Response Analyses
36 Hematopoietic Cancer Deaths
No Lag Period
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Absorbed Dose (rad)

Model	β Coefficient	Relative Risk	Std. Error	Likelihood Ratio Chi-square	p value
Cumulative exposure (log-linear)	0.00015	1.16 at 1 rad	0.00043	0.110	>0.5
Log (cumulative exposure + 1)	-0.045	0.75 at 1 rad	0.096	0.614	0.416
Cumulative exposure (linear)	0.00018	1.18 at 1 rad	0.00056	0.123	>0.5
				Wald Test (z)	
Log (cumulative exposure)	0.115		0.264	0.435	>0.5
Log (cumulative exposure) * age	-0.0029		0.0046	-0.639	>0.5

Table 50
External Radiation Exposure-Response Analyses
36 Hematopoietic Cancer Deaths
Five Year Lag Period
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Absorbed Dose (rad)

Model	β Coefficient	Relative Risk	Std. Error	Likelihood Ratio Chi-square	p value
Cumulative exposure (log-linear)	-0.00004	0.96 at 1 rad	0.0005	0.008	>0.5
Log (cumulative exposure + 1)	-0.110	0.47 at 1 rad	0.081	1.817	0.178
Cumulative exposure (linear)	-0.00004	0.96 at 1 rad	0.0005	0.008	>0.5
				Wald Test (z)	
Log (cumulative exposure)	-0.147		0.167	-0.883	0.377
Log (cumulative exposure) * age	0.0013		0.0033	0.404	>0.5

Table 51
Internal Radiation Exposure-Response Analyses
36 Hematopoietic Cancer Deaths
No Lag Period
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Alpha activity in urine (dpm-year)

Model	β Coefficient	Std. Error	Likelihood Ratio Chi-square	p value
Cumulative exposure (log-linear)	-0.008	0.019	0.175	>0.5
Log (cumulative exposure + 0.1 disintegration counts)	-0.047	0.045	1.063	0.297
Cumulative exposure (linear)	-0.008	0.012	0.218	>0.5
			Wald test (z)	
Cumulative exposure (log-linear)	0.069	0.070	0.960	0327
Cumulative exposure * age	-0.0015	0.0014	1.145	0.285
Cumulative exposure (<40 yrs)	0.029	0.039	0.742	0.458
Cumulative exposure (40-60 yrs)	-0.041	0.047	-0.875	0.381
Cumulative exposure (>60 yrs)	-0.113	0.066	-1.718	0.086
Time since last exposed	-0.042	0.022	-1.931	0.053

Table 52
Internal Radiation Exposure-Response Analyses
36 Hematopoietic Cancer Deaths
Five Year Lag Period
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Alpha activity in urine (dpm-year)

Model	β Coefficient	Std. Error	Likelihood Ratio Chi-square	p value
Cumulative exposure (log-linear)	-0.07	0.023	0.575	0.477
Log (cumulative exposure + 0.1 disintegration counts)	-0.044	0.046	0.903	0.339
Cumulative exposure (linear)	-0.012	0.0008	0.710	0.365
			Wald test (z)	
Cumulative exposure	0.116	0.092	1.264	0.206
Cumulative exposure * age	-0.0025	0.0018	-1.373	0.170
Cumulative exposure (<40 yrs)	0.136	0.094	1.443	0.149
Cumulative exposure (40 - 60 yrs)	-0.159	0.100	-1.587	0.113
Cumulative exposure (>60 yrs)	0.190	0.106	-1.785	0.074

Table 53
External Radiation Exposure-Response Analyses
13 Leukemia Cancer Deaths
No Lag Period and Five Year Lag Period
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Lag Period = 0 years
Absorbed Dose (rad)

Model	β Coefficient	Relative Risk	Std. Error	Likelihood Ratio Chi-square	p value
Cumulative exposure (log-linear)	-0.0013	0.27 at 1 rad	0.0011	1.893	0.225
Log (cumulative exposure +1)	-0.324	0.11 at 1 rad	0.137	5.588	0.018
Cumulative exposure (linear)	-0.0004	0.60 at 1 rad	0.0003	1.590	0.235
				Wald test (z)	
Cumulative exposure (log-linear)	0.013		0.0056	2.269	0.023
Cum exposure * age	-0.00029		0.00013	2.246	0.025
Cumulative exposure (<=40 yrs)	0.0032	23.9 at 1 rad	0.0023	1.40	0.162
Cumulative exposure (>40 yrs)	-0.0059	0.07 at 1 rad	0.0028	2.107	0.037

Lag Period = 5 years
Absorbed Dose (rad)

Cumulative exposure (log-linear)	0.012		0.007	1.693	0.090
Cum exp * age	-0.00028		0.00015	1.870	0.062
Cum exp (<40 yrs)	0.0005	1.65	0.0048	0.104	>0.5
Cum exp (>40 yrs)	-0.0037	0.03	0.0052	0.712	0.473

Table 54
Internal Radiation Exposure-Response Analyses
13 Leukemia Cancer Deaths
No Lag Period
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Alpha activity in urine (dpm-year)

Model	β Coefficient	Std. Error	Likelihood Ratio Chi-square	p value
Cumulative exposure (log-linear)	-0.026	0.036	0.603	0.467
Log (cumulative exposure + 0.1 disintegration)	-0.145	0.069	4.415	0.036
			Wald test (z)	
Cumulative exposure (linear)	-0.012	0.020	0.361	>0.5
Cumulative exposure (log-linear)	0.457	0.199	2.298	0.022
Cumulative exposure * age	-0.011	0.0048	-2.196	0.028
Cumulative exposure (<=40)	0.124	0.073	1.704	0.088
Cumulative exposure (>40)	-0.230	0.098	-2.337	0.019

Table 55
External Radiation Exposure-Response Analyses
15 Stomach Cancer Deaths
No Lag Period
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

Absorbed Dose (rad)

Model	β Coefficient	Relative Risk	Std. Error	Likelihood Ratio Chi-square	p value
Cumulative exposure (log-linear)	-0.00003	0.97 at 1 rad	0.00056	0.003	>0.5
Log (cumulative exposure + 1)	-0.042	0.75 at 1 rad	0.137	0.095	>0.5
				Wald Test (z)	
Cumulative exposure (log-linear)	0.0041		0.005	0.795	0.427
Cumulative exposure * age	-0.00007		0.00009	-0.797	0.425

Table 56
Power to Detect Excess Bone Cancer
8,877 Uranium Enrichment Workers
α = 0.05 2-Sided Expected Deaths = 0.925

Relative Risk	Power
1.25	4%
1.75	9%
2	12%
2.5	20%
3	29%
4	48%
5	66%
6	80%
7	87%

Appendix A
NIOSH Death Categories
Life-Table Analysis System (LTAS)
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

NIOSH Death Category	Cause	7th Revision ICD 1954 - 1967	8th Revision ICD 1968 - 1978	9th Revision ICD 1979 - 1991
1-2	Tuberculosis			
1	Respiratory tuberculosis	001-008	010-012	010-012
2	Other tuberculosis	010-019	013-019	013-018
3-6	MN of buccal cavity and pharynx			
3	MN of lip	140	140	140
4	MN of tongue	141	141	141
5	MN of other parts of buccal cavity	142-144	142-145	142-145
6	MN of pharynx	145-148	146-149	146-149
7-14	MN of digestive organs and peritoneum			
7	MN of esophagus	150	150	150
8	MN of stomach	151	151	151
9	MN of intestine except rectum	152,153	152,153	152,153
10	MN of rectum	154	154	154
11	MN of biliary passages, liver and gall bladder	155	155,156	155.0,155.1, 156
12	MN of liver not specified	156A	197.8	155.2
13	MN of pancreas	157	157	157
14	MN of peritoneum and other and unspecified of digestive organs	158,159	158,159	158,159

Appendix A Continued

NIOSH Death Category	Cause	7th Revision ICD 1954 - 1967	8th Revision ICD 1968 - 1978	9th Revision ICD 1979 - 1991
15-17	MN of respiratory			
15	MN of larynx	161	161	161
16	MN of trachea, bronchus and lung	162,163	162,163	162,163
17	MN of other parts of respiratory system	160,164	160,163	160, 163-165
18	MN of breast	170	174	174-175
19-22	MN of female genital organs			
19	MN of cervix uteri	171	180	180
20	MN of other and unspecified parts of uterus	172-174	181,182	179,181,182
21	MN of ovary, fallopian tube and broad ligament	175	183	183
22	MN of other female genital organs	176	184	184
23-24	MN of male genital organs			
23	MN of prostate	177	185	185
24	MN of other male genital organs	178,179	172.5,173.5 186,187	186,187
25-26	MN of urinary organs			
25	MN of kidney	180	189.0-189.2	189.0-180.2
26	MN of bladder and other urinary organs	181	1881.089.9	188 189.3-189.9
27-33	MN of other and unspecified sites			
27	MN of skin	190,191	172.0-172.4 172.6-172.9 173.0-173.4 173.6-173.9	172,173
28	MN of eye	192	190	190

Appendix A Continued

NIOSH Death Category	Cause	7th Revision ICD 1954 - 1967	8th Revision ICD 1968 - 1978	9th Revision ICD 1979 - 1991
29	MN of brain and other parts of nervous system	193	191,192	191,192
30	MN of thyroid gland	194	193	193
31	MN of bone	196	170	170
32	MN of connective tissue and soft tissue	197	171	171
33	MN of other and unspecified sites (minor)	156B,165,195 198,199	194-196 197.0-197.7 197.9,198 199	194-199
34-37	Neoplasms of lymphatic and hematopoietic tissue			
34	Lymphosarcoma and reticulosarcoma	200	200	200
35	Hodgkin's disease	201	201	201
36	Leukemia and aleukemia	204	204-207	204-208
37	Other neoplasms of lymphatic hematopoietic tissue	202,203,205	202,203	202,203
38-40	Benign and unspecified neoplasms			
38	Benign neoplasms of the eye, brain and other parts of nervous system	233	224,225	224,225
39	Neoplasms of eye brain, other parts of nervous system unspecified nature	237	238,743.4	237.5-237.9 239.6-239.7
40	Other benign and unspecified nature neoplasms	210-222 224-236 238-239	208,210-223 226-237 239	210-223 226-237.4 238.0239.5 239.8-239.9
41	Diabetes mellitus	260	260	260

Appendix A Continued

NIOSH Death Category	Cause	7th Revision ICD 1954 - 1967	8th Revision ICD 1968 - 1978	9th Revision ICD 1979 - 1991
42-45	Diseases of the blood and blood forming organs			
42	Pernicious anemia	290	281.0,281.9	281.0,281.9
43	Anemias of other and unspecified type	291-293	280.000 281.1-281.4 282-285	280.000 281.1-281.8 282-285
44	Coagulation defects,purpura,other hemorrhagic conditions	296	286,287	286,287
45	All other diseases of blood forming organs	294-295 297-299	209 288,289	288,289
46-47	Mental and personality disorders			
46	Alcoholism	322	303	303
47	Other mental disorders	300-321 323-326	290-302 304-315	290-302 304-319
48-49	Nervous system and sensor organs			
48	Multiple sclerosis	345	340	340
49	Other diseases nervous system and sensor organs	340-344 350-398	370-333 341-389	320-337 341-389
50-55	Disease of the heart			
50	Rheumatic heart disease,including fever	400-402 410-416	390-398	390-398
51	Ischemic heart disease	420	410-414	410-414
52	Chronic disease of endocardium	421	424	424
53	Other myocardial degeneration	422	428	429.0,429.1
54	Hypertension with heart disease	440-443	400.1, 00.9 402,404	402-404
55	Other diseases of the heart	430-434	420-423 425-427 429	420-423 425-428 429.2-429.9

Appendix A Continued

NIOSH Death Category	Cause	7th Revision ICD 1954 - 1967	8th Revision ICD 1968 - 1978	9th Revision ICD 1979 - 1991
56-58	Other disease of circulatory system			
56	Hypertension without heart disease	444-447	400.0 400.2,400.3 401,403	401,403,405
57	Cerebrovascular disease	330-334	430-438	430-438
58	Diseases of the arteries, veins and pulmonary circulation	450-468	440-444.1 444.3-458	415-417 440-459
59-65	Diseases of the respiratory system			
59	Acute respiratory infections except influenza and pneumonia	470-475 500	460-466	460-466
60	Influenza	480-483	470-474	487
61	Pneumonia (except newborn)	490-493	480-486	480-486
62	Chronic and unspecified bronchitis	501,502	490-491	490-491
63	Emphysema	527.1	492	492
64	Asthma	241	493	493
65	Pneumoconioses and other respiratory diseases	510-527.0 527.2	500-519	470-478 494-519
66-69	Diseases of the digestive system			
66	Diseases of the stomach and duodenum	540-541 543	531-537	531-537
67	Hernia and intestinal obstruction	560-561 570	550-553 560	550-553 560
68	Cirrhosis of the liver	581	571	571
69	Other diseases of digestive system	530-539,542 544-545 550-553 571-578 580,582-587	444.2 520-530 540-543 561-570 572-577	520-530 540-543 555-558 562-570 572-579

Appendix A Continued

NIOSH Death Category	Cause	7th Revision ICD 1954 - 1967	8th Revision ICD 1968 - 1978	9th Revision ICD 1979 - 1991
70-78	Diseases of the genito-urinary system			
70	Acute glomerulonephritis nephrotic syndrome & acute renal failure	590-591	580-581	580,581,584
71	Chronic & unspec.nephritis,renal failure & other renal sclerosis	592-594	582-584	582,583 585-587
72	Infection of kidney	600	590	590
73	Calculi of urinary system	602-604	592-594	592-594
74	Hyperplasia of prostate	610	600	600
75	Other diseases of male genital organs	611-617	601-607	601-608
76	Diseases of the breast	620-621	610-611	610-611
77	Diseases of the female genital organs	622-637	612-629	614-629
78	Other genito-urinary system diseases	601-603 605-609	591-593 595-599	588,589,591 593,595-599
79-80	Diseases of the skin and subcutaneous tissue			
79	Infections of the skin and subcutaneous tissue	690-698	680-686	680-686
80	Other diseases of the skin and subcutaneous tissue	700-716	690-708	690-709
81	Arthritis and spondylitis	720-725	710-715	711-716 720,721
82	Osteomyelitis and periostitis	730	720	730
83	Other diseases of MS system	731-749 726-727	716-718 721-738	710,717-719 722-729 731-739
84	Symptoms and ill-defined conditions	780-793 795	780-793 795,796	780-796,798 799

Appendix A Continued

NIOSH Death Category	Cause	7th Revision ICD 1954 - 1967	8th Revision ICD 1968 - 1978	9th Revision ICD 1979 - 1991
85-89	Accidents			
85	Transportation accidents	800-866	800-845 940-941	800-848 929.0-929.1
86	Accidental poisoning	870-895	850-877 942	850-869 929.2
87	Accidental falls	900-904	880-887 943	880-888 929.3
88	Other accidents	910-936 960-962	890-929 944-946	890-928 929.4-929.9
89	Medical complications and misadventure	940-959	930-936 947-949	870-879 930-949
90-91	Violence			
90	Suicide	963 970-979	950-959	950-059
91	Homicide	964 980-985	960-978	960-978
92	Other causes			

Appendix B
Adjustments Made to the External Dosimetry Data
Portsmouth Gaseous Diffusion Plant (PORTS)
September 1954 through December 1991

The purpose of this Appendix is to provide more information on how the site collective cumulative external dose increased from 1,998 rad to 9,289 rad. A detailed explanation of this process is forthcoming in a separate publication. However, a brief summary is provided in this final report for those interested in this exposure assessment process.

Objectives

1. Provide external radiation dose estimates for every worker in the cohort for dose-response analyses, while accounting for the possibility of a zero cumulative dose estimate.
2. Account for changes over time in administrative practices for recording external dosimetry results and dosimeter exchange frequencies from 1954 through 1991.
3. Account for technical changes over time in the type of dosimeters used in the radiation monitoring program from 1954 through 1991.
4. Develop imputation strategies for assigning external dose estimates to workers that were potentially exposed but not monitored.

Challenge

The challenge in this exposure assessment was to appropriately interpret historical dosimetry information collected for compliance purposes and convert it into dosimetry information applicable to epidemiologic research. This process of conversion is often the subject of controversy because the conversion methods are not always consistent with the practices typically encountered in the traditional compliance-driven health physics arena. This appendix briefly illustrates this challenge, and summarizes the process used in this

study to estimate worker exposures to external ionizing radiation based on their individual work history information.

Dose Conversion Process for Epidemiology Research

1. **Obtain Raw Data**: A complete copy of the computerized dosimetry (radiation exposure information) and personnel (work history) information were obtained from the site covering the years of operation from 1954 through 1991.

2. **Account for Organizational Changes**: The organizational dynamics at the site were described by departmental changes over time. Three sources used to create a complete history of these changes were (1) organization charts, (2) personnel accounting reports, and (3) a 1987 site-generated document of historical departmental changes developed for the initial NIOSH study. All three sources were used to create a database called the Department History Document/Database (DHD). The original DHD developed by the site was generated from these same sources but was not sufficiently complete for this study. We identified additional organizational charts and personnel accounting reports to complete the DHD through 1991. Since the DHD was created from official records and not subject to computerized manipulation, it was considered to be the "gold standard" for department histories as compared to department histories in personnel data files.

3. **Account for Health Physics Administrative Changes:** Changes in the Health Physics monitoring program influenced the quantity and quality of exposure data available for this study. For example, the dosimetry monitoring frequency, selection criteria for monitoring, and the dosimeter technology all experienced changes between 1954 and 1991. In the 1950s, not all workers were monitored but those who were monitored were selected on a statistical basis according to their exposure potential and time spent

in radiation areas [GAT 1959]. In the 1960s, a new film badge incorporating the security pass was issued, which meant that all workers were monitored. The frequency with which these badges were read depended upon the likelilhood of exposure, but all workers had their badge read at least once each year [GAT 1961]. In the 1970s, only those workers potentially exposed were monitored on a quarterly basis [GAT 1971a]. In the 1980s, new dosimeter technology was introduced, the thermoluminescent dosimeter (TLD), and the monitoring frequency was primarily based on the exposure potential and gender [GAT 1986b]. Women had their dosimeters read every month to prevent unnecessary fetal exposures. Men had their dosimeters read quarterly. Workers in a non-exposed category had their dosimeters read annually. Exceptions to this read-out practice were based on job classification, work area, and time spent in radiation areas. After 1987, all dosimeters were read quarterly, including women.

4. **Merge Computerized Databases:** Linking the variables ID, YEAR, or DEPARTMENT among the Health Physics Database, Departmental History Document Database, and Personnel Database completed three critical steps of this conversion process. First, if all these variables properly matched among the three databases then the exposure data were assigned to a NIOSH exposure group and used to construct an exposure matrix. Second, personnel and health physics information were combined to provide a detailed work history and dose history for every worker. And finally, unmonitored periods were identified for every worker. If any of these variables did not match then several procedures were implemented to correct the errors. Those provided procedures are too complex to describe here but will be in the forthcoming publication.

5. **Dose Adjustments for Monitored Workers:** The first step in increasing the site collective cumulative dose was to account for doses measured below the limit of detection (LOD) and administratively recorded as zeros. This is one example where compliance versus epidemiology requirements differ. The practice of recording doses

below the LOD as zeros is legally acceptable. However, for epidemiologic purposes, the LOD was considered to be high enough above background that maintaining a zero dose would result in an underestimate of a worker's cumulative dose. Data sets containing some values below the LOD are called "left-censored," i.e., nonzero values which cannot be measured but are known to be below some threshold. Conversely, adjusting the zero dose to the LOD would overestimate a worker's cumulative dose. Therefore, the estimate of a worker's cumulative dose incorporated a dose increase of half the LOD (LOD/2) for any recorded zero in their dosimetry file (Hornung and Reed, 1990). This simple adjustment was further confounded by multiple dosimeter exchange frequencies over time. To address these different exchange frequencies we normalized the "additional" dose to be consistent with a quarterly dosimeter exchange rate on an annual basis. A specific example is provided in the main text of this report. Workers who were monitored for only a portion of their work histories were assigned their individual dose results for monitored periods. During unmonitored periods, these workers were assigned a mean dose based on their assigned exposure groups as described below. Additionally, if a worker spent time in more than one exposure group during a given year, the estimated dose was apportioned according to the time spent in each exposure group.

6. **Dose Imputations for Unmonitored Workers:** The second step that increased the site collective cumulative dose was to impute doses for unmonitored workers who were potentially exposed. Unmonitored workers were assigned the mean dose for their assigned exposure group. Additionally, dose imputations covered periods of time when little or no monitoring was conducted, specifically during the 1950s and 1970s. During the 1950s and 1970s, several exposure groups (primarily in the Administration, Industrial Relations, Finance, and Purchasing and Materials Divisions) were missing exposure data due to the monitoring practices employed during those times. For example, the monitoring practice may have been based on legal and economic drivers

since these workers did not routinely experience exposure potentials equal to or greater than ten percent of the annual occupational exposure limit (500 mrem yr^{-1}), which was the plant's threshold requiring health physics monitoring. This is another example where compliance vs. epidemiologic needs differ and warrant dose adjustments when doing epidemiologic research. We believed these unmonitored workers of the 1950s and 1970s were potentially exposed since their job tasks did not change and exposure data existed for them in the 1960s and 1980s when most workers were monitored. Backward extrapolation was used to estimate a doses during the 1950s. During the 1970s, doses were linearly interpolated from exposure group trends before and after this decade.

7. **Zero Cumulative Dose Criteria:** A zero dose was assigned to a portion of the worker's work history under two conditions: 1) if a worker was neither monitored nor assigned to an exposure group, and 2) if a worker was not monitored but categorized into an exposure group that had no other monitored workers. These criteria allow for a worker to receive a zero cumulative dose based on their work history and exposure potential.

Tables B1 - B3 list three employee's work history while employed at the site, their dose of record as measured by the site, and the NIOSH assigned external dose used in the dose-response analyses. They were selected based on their length of employment (2.4 years, 6.6 years, and 18.9 years) to illustrate various methods of dose imputation strategies over the time.

Figure B1 illustrates the contributions from the NIOSH adjustments to the site collective dose. The collective dose provided by the site (1,998 rad) accounted for 21.5 percent of the collective dose as estimated by NIOSH. An additional 3,550 rad (38.2% of the NIOSH total) was added to the site collective dose to account for workers that had potential exposures but were not monitored, followed by 1,256 rad (27.5%) from the LOD adjustment, 792 rad (8.5%) from the

1954 - 1959 backward extrapolation adjustment, and 395 rad (4.2%) from the 1970 - 1979 interpolation adjustment (4.2 %).

Table B1: A worker with a length of employment of 2.4 years.

WORK HISTORY					DOSE RESULTS		
NIOSH Exposure Group	**Department Code**	**Department Title**	**Year**	**Days employed**	**Site Dose (mrem)**	**NIOSH Dose (mrem)**	**Comment***
F01	730	Cascade Maintenance	1954	36	Not Monitored	20.2	Adjusted Mean Dose
			1954	12	Not Monitored	0.0	LEAVE
			1954	99	Not Monitored	55.5	Mean Dose
F04	731	Cascade Instrument Maintenance	1954	25	Not Monitored	6.4	Mean Dose
			1955	215	0	1.4	Zero Adjusted Dose
			1955	12	0	0.0	LEAVE
			1955	138	0	0.9	Zero Adjusted Dose
			1956	107	60	75.0	Zero Adjusted Dose
F14	732	Field Welding	1956	121	Not Monitored	32.0	Mean Dose
			1956	11	Not Monitored	0.0	LEAVE
			1956	103	Not Monitored	27.3	Mean Dose
				Cumulative Dose (mrem)	**60**	**220**	**NIOSH dose rounded to two significant figures**

* See Comment definitions at the bottom of Table A3.

Table B2: A worker with a length of employment of 6.5 years.

WORK HISTORY					DOSE RESULTS		
NIOSH Exposure Group	**Department Code**	**Department Title**	**Year**	**Days employed**	**Site Dose (mrem)**	**NIOSH Dose (mrem)**	**Comment***
I03	761	Engineering Services	1975	149	Not Monitored	25.5	Mean Dose Slope
			1976	275	Not Monitored	177.4	Mean Dose
I05	611	Electrical and Instrument Engineering	1976	91	Not Monitored	11.8	Mean Dose Slope
			1977	274	Not Monitored	37.0	Mean Dose
J06	903	Oak Ridge Expansion Team	1977	91	Not Monitored	33.8	Mean Dose
			1978	365	Not Monitored	23.3	Mean Dose Slope
			1979	365	Not Monitored	23.3	Mean Dose Slope
H02	903	Safeguards/ SAR/EIS	1980	366	Not Monitored	31.8	Mean Dose
			1981	365	0	5.0	Zero Adjusted
J06	903	Operating Contractor Project Office	1982	15	0	5.0	Zero Adjusted
				Cumulative Dose (mrem)	**0**	**370**	**NIOSH dose rounded to two significant figures**

* See Comment definitions at the bottom of Table A3.

Table B3: A worker with a length of employment of 17.5 years.

WORK HISTORY					DOSE RESULTS		
NIOSH Exposure Group	**Department Code**	**Department Title**	**Year**	**Days employed**	**Site Dose (mrem)**	**NIOSH Dose (mrem)**	**Comment***
F16	741	Buildings and Grounds	1954	109	Not Monitored	0.0	No Dose
			1955	365	Not Monitored	0.0	No Dose
			1956	261	Not Monitored	0.0	No Dose
F12	726	Carpenter Shop	1956	105	Not Monitored	23.8	Adjusted Mean Dose
			1957	365	Not Monitored	55.6	Adjusted Mean Dose
			1958	365	60	66.9	Zero Adjusted
			1959	365	160	165.0	Zero Adjusted
			1960	366	0	5.0	Zero Adjusted
			1961	365	0	75.0	Zero Adjusted
			1962	365	0	60.0	Zero Adjusted
			1963	259	40	84.3	Zero Adjusted
			1963	60	Not Monitored	0.0	LEAVE
			1963	2	0	0.7	Zero Adjusted

Table B3 Continued

WORK HISTORY					DOSE RESULTS		
NIOSH Exposure Group	**Department Code**	**Department Title**	**Year**	**Days employed**	**Site Dose (mrem)**	**NIOSH Dose (mrem)**	**Comment***
			1963	44	Not Monitored	0.0	LEAVE
			1964	16	Not Monitored	0.0	LEAVE
			1964	2	0	1.4	Zero Adjusted
			1964	57	Not Monitored	0.0	LEAVE
			1964	291	155	198.6	Zero Adjusted
			1965	365	20	80.0	Zero Adjusted
F12	726	Carpenter Shop	1966	297	36	126.0	Zero Adjusted
			1966	68	Not Monitored	0.0	LEAVE
			1967	113	Not Monitored	0.0	LEAVE
			1967	252	0	120.0	Zero Adjusted
			1968	366	765	810.0	Zero Adjusted
			1969	365	270	285.0	Zero Adjusted
			1970	365	Not Monitored	95.8	Mean Dose Slope

Table B3 Continued.

WORK HISTORY					DOSE RESULTS		
NIOSH Exposure Group	**Department Code**	**Department Title**	**Year**	**Days employed**	**Site Dose (mrem)**	**NIOSH Dose (mrem)**	**Comment***
			1971	365	Not Monitored	90.1	Mean Dose Slope
			1972	91	Not Monitored	20.4	Mean Dose
				Cumulative Dose (mrem)	**1506**	**2400**	**NIOSH dose rounded to two significant figures**

Table Comment Definitions:

* **Adjusted Mean Dose** = Dose estimate was determined by backward extrapolation from the NIOSH Exposure Group dose trend during the 1960s and all zero recorded doses were assigned a value of LOD/2 and normalized to a quarterly monitoring frequency. (See pages 34 - 38)
* **Mean Dose** = Dose estimate was derived from the mean dose associated with all the monitored workers categorized in the NIOSH Exposure Group
* **Mean Dose Slope** = Dose estimate was calculated for the 1970 - 1979 period using linear interpolation based on the mean doses of the NIOSH exposure group during the 1960's and 1980's decades.
* **No Dose** = A zero dose was assigned because the worker was neither monitored for radiation exposure and worked in a NIOSH exposure group that had no other monitored workers.
* **Zero Adjusted Dose** = The worker was monitored and the recorded zero doses were assigned a value of LOD/2 and normalized to a quarterly frequency. Some workers were not monitored routinely throughout their work history, which may have resulted in lower or higher assigned dose estimates.

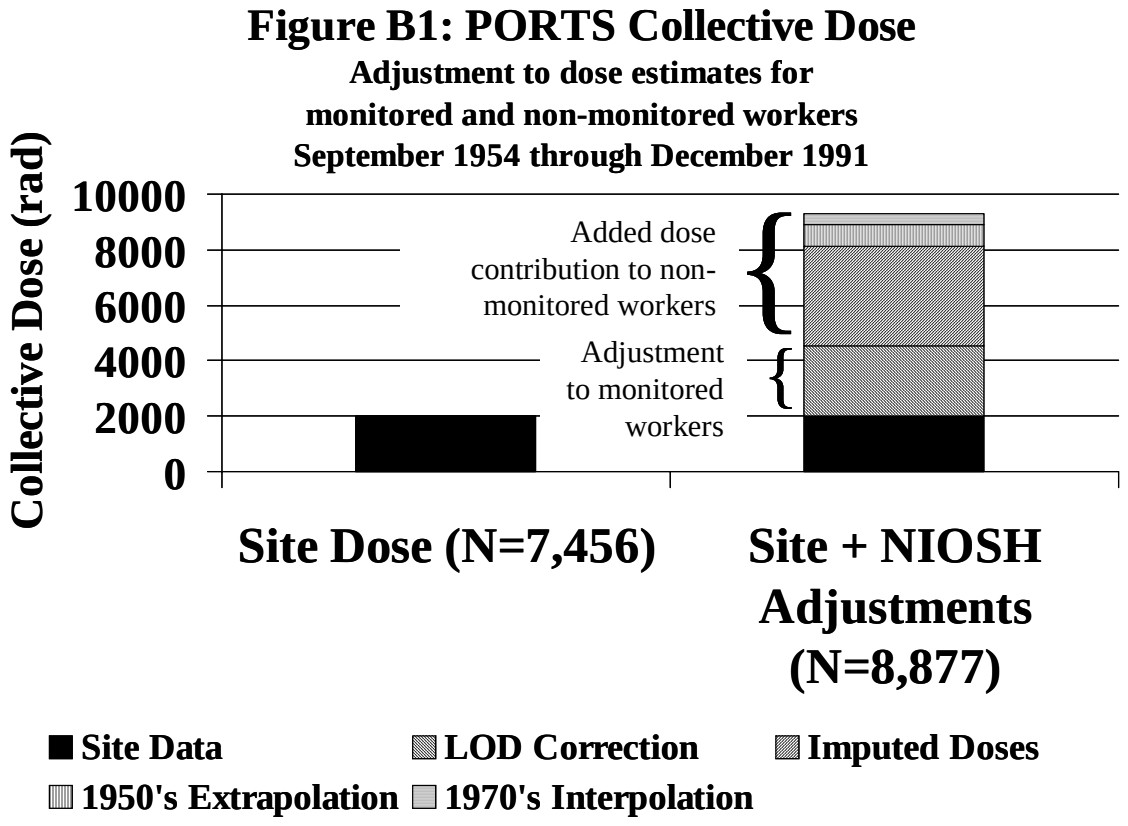
Figure B1: PORTS Collective Dose
Adjustment to dose estimates for
monitored and non-monitored workers
September 1954 through December 1991
Collective Dose (rad)
10000
8000
6000
4000
2000
0
Added dose
contribution to non-
monitored workers
Adjustment
to monitored
workers
Site Dose (N=7,456)
Site + NIOSH
Adjustments
(N=8,877)
Site Data
LOD Correction
Imputed Doses
1950's Extrapolation
1970's Interpolation