# FINAL REPORT

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

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**PUBLIC HEALTH SERVICE**
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**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

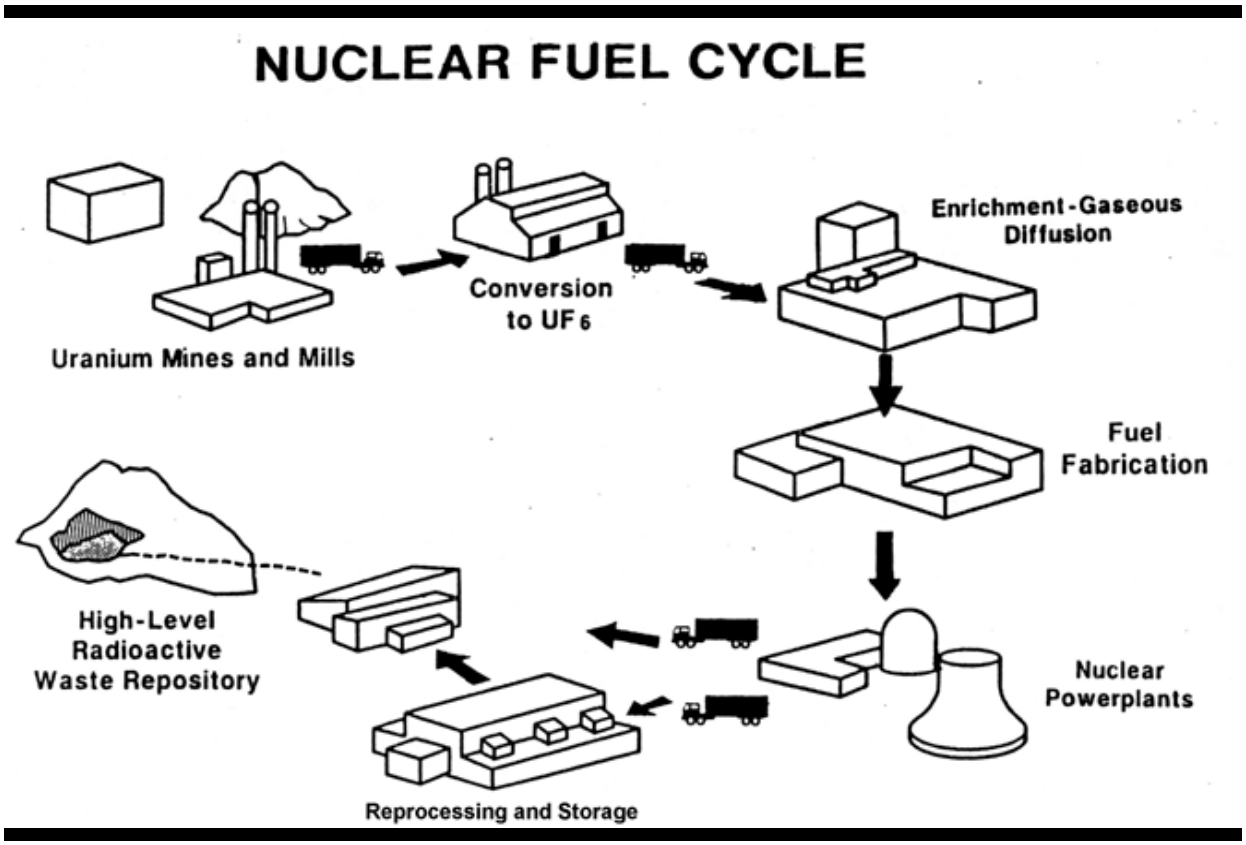


**Figure 1**: Nuclear Fuel Cycle *Source: Adapted from Health Physics Society: Science Teachers Workshop Material*

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].

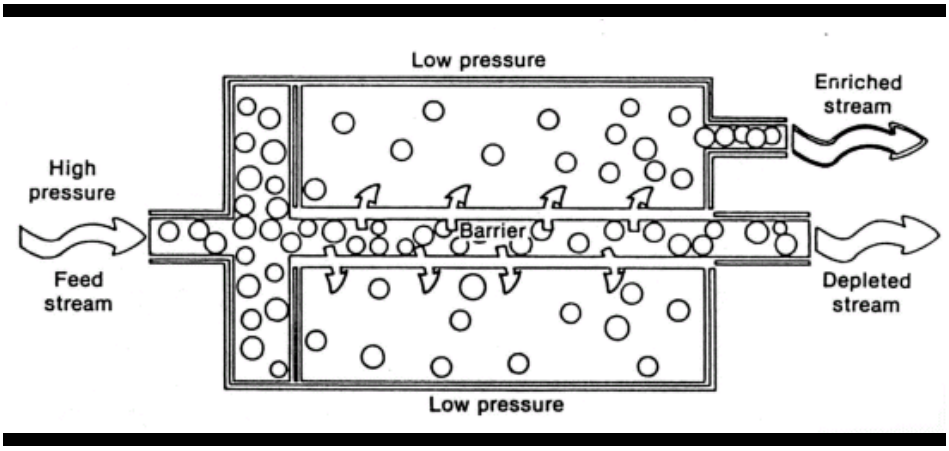


**Figure 3**: Gaseous Diffusion Stage
*Source: DOE EGG-2530; Health Physics Manual of Good Practices for Uranium Facilities, 1988.*

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).

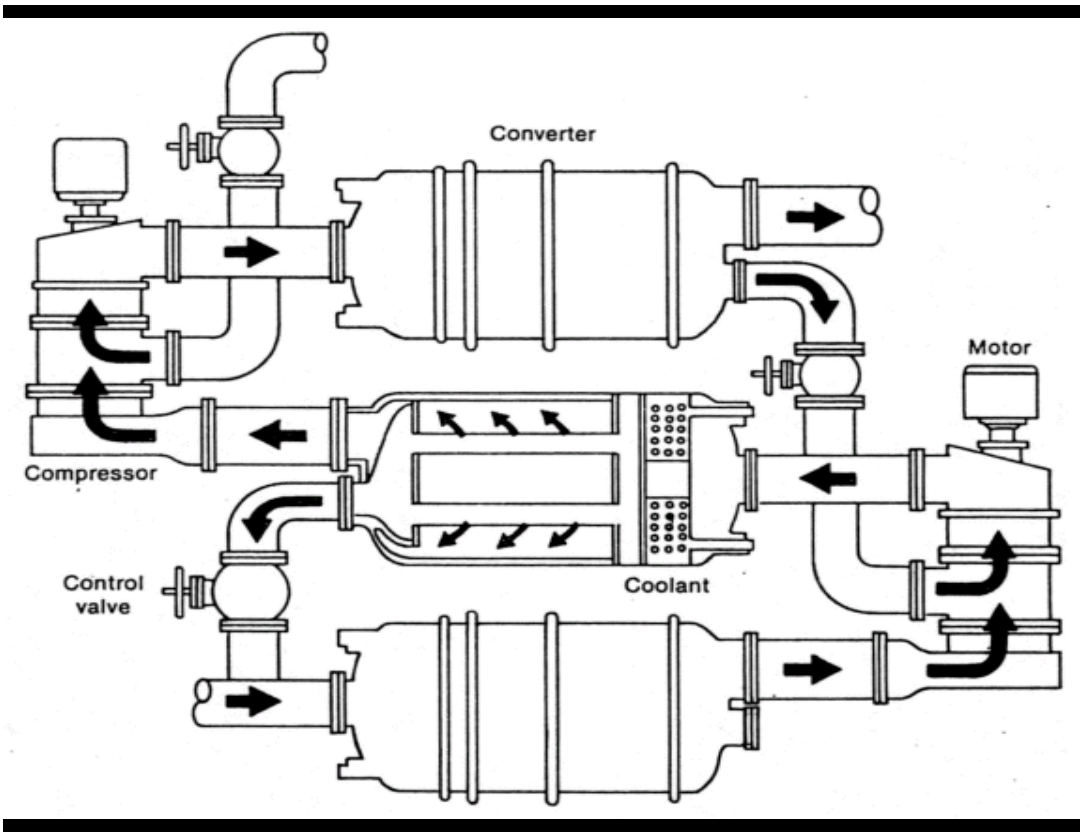


**Figure 4**: Stage Arrangement

*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 |
|---|---|---|---|---|
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MEDICAL EXAMINATIONS

| DATE | EXAMINING PHYSICIAN | CLASSI-FICATION | DISABILITIES | RESTRICTIONS |
|---|---|---|---|---|
| | | | | |
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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:

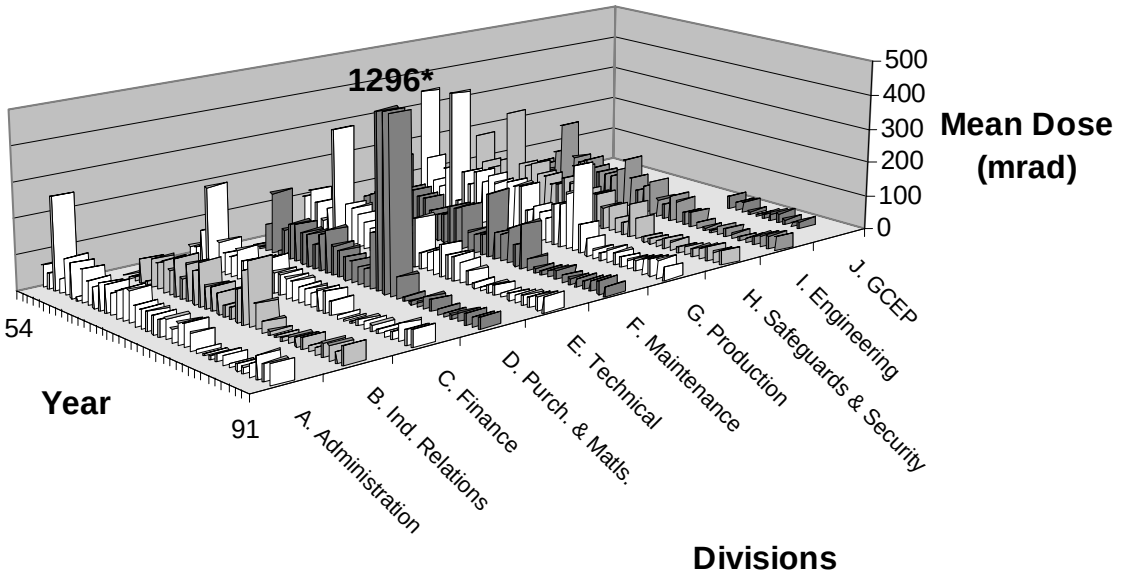
! Ten "Divisions" were identified from historical organizational charts and given an alphabetical designation as follows : A = General Administration; B = Industrial Relations; C = Finance; D = Purchasing and Materials; E = Technical; F = Maintenance; G = Production; H = Safeguards and Security; I =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

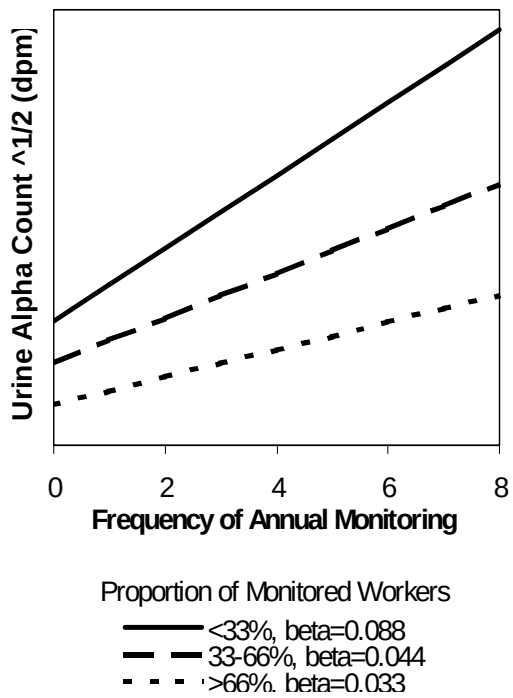



**Figure 7:** Imputation of Urine Alpha Counts for Workers with Frequency of Monitoring Equal to Zero

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).

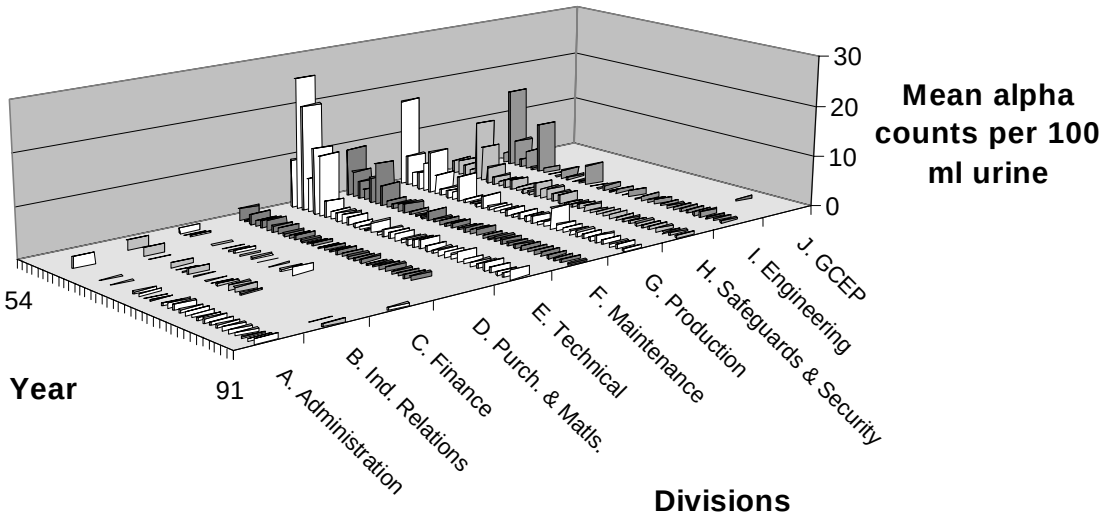



**Figure 8:** Internal Radiation Exposure Matrix. Mean Alpha Counts per 100 ml in Urine.

## 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 ($\beta$= -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 $\beta$= -0.0032, p=0.072; cumexp*age $\beta$=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 $\beta$= -0.0004, p=0.225 for risk at less than 60 years of age and $\beta$=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 $\beta$=-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 $\beta$ 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.

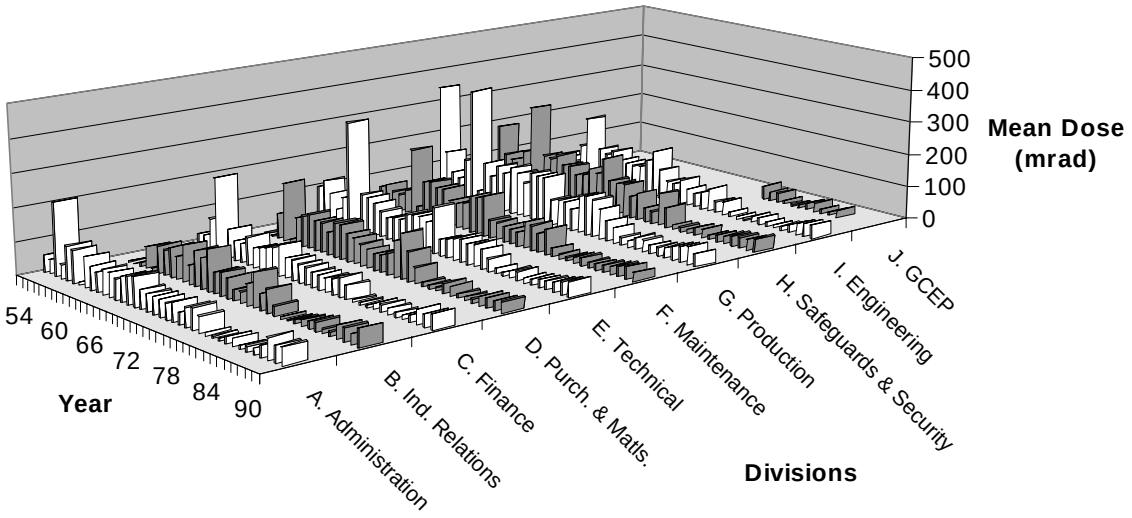


**Figure 9:** Adjusted External Radiation Exposure Metric. Minimum of 90 Days Monitoring for Workers to be Considered in the Creation of the EM.

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.

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**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]<br>56.3[B]<br>77.1[C]<br>73.4[D] | 295<br>3.2<br>4.4<br>77.4 | 1,104<br>12.0<br>16.4<br>70.3 | 140<br>1.5<br>2.1<br>69.3 | 6,725<br>73.0 |
| Unknown[E] | 733<br>7.9<br>60.4<br>10.4 | 43<br>0.5<br>3.5<br>11.3 | 380<br>4.1<br>31.3<br>24.2 | 59<br>0.6<br>4.8<br>29.2 | 1,215<br>13.2 |
| Dead | 1,142<br>12.4<br>89.6<br>16.2 | 43<br>0.5<br>3.4<br>11.3 | 87<br>0.9<br>6.8<br>5.6 | 3<br>0.0<br>0.2<br>1.5 | 1,275<br>13.8 |
| Total | 7,061<br>76.6 | 381<br>4.1 | 1,571<br>17.0 | 202<br>2.2 | 9,215<br>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]<br>0.24[C]<br>-[D] | - | - | - | - | - | - | 0<br>0.24<br>- |
| **7 days-5 years** | 1<br>0.20<br>4.89 | 65<br>98.0<br>0.66[F] | - | - | - | - | - | 66<br>98.25<br>0.67[F] |
| **5 years - 10 years** | 1<br>0.25<br>4.01 | 37<br>50.09<br>0.74 | 29<br>77.10<br>0.38[F] | - | - | - | - | 67<br>127.44<br>0.53[F] |
| **10 years-15 years** | 0<br>0.18<br>- | 25<br>57.25<br>0.44[F] | 38<br>40.63<br>0.94 | 39<br>64.79<br>0.60[F] | - | - | - | 102<br>162.85<br>0.63[F] |
| **15 years-20 years** | 0<br>0.10<br>- | 42<br>63.40<br>0.66[F] | 16<br>38.17<br>0.42[F] | 16<br>18.73<br>0.85[F] | 19<br>53.89<br>0.35[F] | - | - | 93<br>174.29<br>0.53[F] |
| **20 years-25 years** | 0<br>0.13<br>- | 69<br>74.96<br>0.92 | 38<br>44.16<br>0.86 | 12<br>15.17<br>0.79 | 10<br>16.17<br>0.62 | 31<br>50.60<br>0.61[F] | - | 160<br>201.20<br>0.80[F] |
| **> 25 years** | 0<br>0.47<br>- | 192<br>264.81<br>0.73[F] | 138<br>155.80<br>0.89 | 44<br>42.00<br>1.05 | 31<br>45.47<br>0.68[E] | 64<br>56.83<br>1.13 | 131<br>188.52<br>0.69[F] | 600<br>753.90<br>0.80[F] |
| **Total** | 2<br>1.56<br>1.28 | 430<br>608.57<br>0.71[F] | 259<br>355.86<br>0.73[F] | 111<br>140.70<br>0.79[E] | 60<br>115.53<br>0.52[F] | 95<br>107.43<br>0.88 | 131<br>188.52<br>0.69[F] | 1088<br>1518.17<br>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]<br>0.57[C]<br>0 | - | - | - | - | - | 0<br>0.58<br>0 |
| **5 years-10 years** | - | 0<br>0.33<br>- | 1<br>0.61<br>1.65 | - | - | - | - | 1<br>0.94<br>1.06 |
| **10 years-15 years** | - | 0<br>0.42<br>- | 0<br>0.33<br>- | 0<br>0.52<br>- | - | - | - | 0<br>1.27<br>0 |
| **15 years-20 years** | - | 1<br>0.50<br>1.99 | 0<br>0.32<br>- | 0<br>0.17<br>- | 1<br>0.46<br>2.19 | - | - | 2<br>1.44<br>1.39 |
| **20 years-25 years** | - | 3<br>0.64<br>4.69 | 0<br>0.39<br>- | 0<br>0.14<br>- | 0<br>0.15<br>- | 1<br>0.45<br>2.21 | - | 4<br>1.77<br>2.26 |
| **> 25 years** | - | 3<br>2.33<br>1.29 | 0<br>1.40<br>- | 2<br>0.36<br>5.52 | 0<br>0.37<br>- | 0<br>0.49<br>- | 3<br>1.76<br>1.70 | 8<br>6.73<br>1.19 |
| **Total** | - | 7<br>4.80<br>1.46 | 1<br>3.05<br>0.33 | 2<br>1.19<br>1.68 | 1<br>0.97<br>1.03 | 1<br>0.94<br>1.06 | 3<br>1.76<br>1.70 | 15<br>12.73<br>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]<br>15.69[C]<br>0.70[D] | 22<br>21.73<br>1.01 | 21<br>47.13<br>0.45[E] | 10<br>13.08<br>0.76 | 3<br>3.40<br>0.88 | 0<br>0.16<br>- | 67<br>101.18<br>0.66[E] |
| **5 years - 10 years** | 9<br>7.28<br>1.24 | 11<br>13.52<br>0.81 | 23<br>44.11<br>0.52[E] | 14<br>39.05<br>0.36[E] | 11<br>25.66<br>0.43[E] | 0<br>0.80<br>- | 68<br>130.43<br>0.52[E] |
| **10 years - 15 years** | 3<br>8.92<br>0.34[F] | 4<br>12.20<br>0.33[F] | 21<br>41.22<br>0.51[E] | 24<br>42.20<br>0.57[E] | 49<br>59.91<br>0.82 | 1<br>1.90<br>0.53 | 102<br>166.35<br>0.61[E] |
| **15 years - 20 years** | 4<br>10.12<br>0.40 | 7<br>12.49<br>0.56 | 23<br>34.69<br>0.66[F] | 15<br>32.12<br>0.47[E] | 44<br>83.53<br>0.53[E] | 1<br>3.22<br>0.31 | 94<br>176.17<br>0.53[E] |
| **20 years - 25 years** | 12<br>11.42<br>1.05 | 17<br>14.74<br>1.15 | 33<br>39.54<br>0.83 | 23<br>29.28<br>0.79 | 70<br>99.35<br>0.70[G] | 0<br>6.62<br>-[E] | 155<br>200.94<br>0.77[E] |
| **25 years - 30 years** | 14<br>13.59<br>1.03 | 14<br>17.86<br>0.78 | 30<br>48.13<br>0.62[E] | 29<br>34.32<br>0.84 | 106<br>118.00<br>0.90 | 11<br>11.76<br>0.94 | 204<br>243.66<br>0.84[F] |
| **>30 years** | 11<br>22.25<br>0.49[F] | 22<br>29.32<br>0.75 | 66<br>85.78<br>0.77[F] | 53<br>60.25<br>0.88 | 186<br>219.23<br>0.85[F] | 15<br>25.00<br>0.60[F] | 353<br>441.83<br>0.80[E] |
| **Total** | 64<br>89.26<br>0.72[E] | 97<br>121.86<br>0.80[F] | 217<br>340.59<br>0.64[E] | 168<br>250.30<br>0.67[E] | 469<br>609.08<br>0.77[E] | 28<br>49.46<br>0.57[E] | 1043<br>1460.56<br>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]<br>0.10[C]<br>-[D] | 0<br>0.12<br>- | 0<br>0.29<br>- | 0<br>0.09<br>- | 0<br>0.02<br>- | 0<br>0.00<br>- | 0<br>0.61<br>- |
| **5 years - 10 years** | 0<br>0.05<br>- | 0<br>0.09<br>- | 1<br>0.32<br>3.15 | 0<br>0.31<br>- | 0<br>0.20<br>- | 0<br>0.01<br>- | 1<br>0.97<br>1.03 |
| **10 years - 15 years** | 0<br>0.07<br>- | 0<br>0.09<br>- | 0<br>0.31<br>- | 0<br>0.34<br>- | 0<br>0.48<br>- | 0<br>0.01<br>- | 0<br>1.31<br>- |
| **15 years - 20 years** | 0<br>0.08<br>- | 0<br>0.10<br>- | 1<br>0.28<br>3.61 | 0<br>0.27<br>- | 2<br>0.71<br>2.83 | 0<br>0.03<br>- | 3<br>1.47<br>2.05 |
| **20 years - 25 years** | 0<br>0.10<br>- | 0<br>0.13<br>- | 2<br>0.34<br>5.89 | 0<br>0.26<br>- | 1<br>0.89<br>1.12 | 0<br>0.06<br>- | 3<br>1.77<br>1.69 |
| **25 years - 30 years** | 0<br>0.12<br>- | 2<br>0.16<br>12.57[E] | 1<br>0.43<br>2.30 | 0<br>0.31<br>- | 3<br>1.09<br>2.76 | 0<br>0.11<br>- | 6<br>2.22<br>2.70 |
| **>30 years** | 0<br>0.19<br>- | 0<br>0.25<br>- | 0<br>0.76<br>- | 0<br>0.52<br>- | 1<br>1.94<br>0.52 | 0<br>0.23<br>- | 1<br>3.90<br>0.26 |
| **Total** | 0<br>0.72<br>- | 2<br>0.94<br>2.14 | 5<br>2.73<br>1.83 | 0<br>2.09<br>- | 7<br>5.31<br>1.32 | 0<br>0.45<br>- | 14<br>12.24<br>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]<br>0.54[C]<br>1.86[D] | 1<br>0.82<br>1.22 | 1<br>1.69<br>0.59 | 0<br>0.41<br>- | 1<br>0.08<br>12.74 | 0<br>0.00<br>- | 4<br>3.53<br>1.13 |
| **5 years - 10 years** | 1<br>0.34<br>2.92 | 3<br>0.70<br>4.31 | 3<br>2.55<br>1.18 | 0<br>2.01<br>- | 0<br>1.01<br>- | 0<br>0.04<br>- | 7<br>6.65<br>1.05 |
| **10 years - 15 years** | 0<br>0.57<br>- | 1<br>0.72<br>1.38 | 3<br>2.77<br>1.08 | 3<br>3.00<br>1 | 4<br>3.51<br>1.14 | 0<br>0.13<br>- | 11<br>10.70<br>1.03 |
| **15 years - 20 years** | 0<br>0.76<br>- | 0<br>0.87<br>- | 2<br>2.46<br>0.81 | 0<br>2.52<br>- | 3<br>6.34<br>0.47 | 0<br>0.26<br>- | 5<br>13.20<br>0.38[E] |
| **20 years - 25 years** | 3<br>1.07<br>2.82 | 2<br>1.32<br>1.51 | 2<br>3.60<br>0.56 | 2<br>2.65<br>0.75 | 6<br>9.48<br>0.63 | 0<br>0.65<br>- | 15<br>18.77<br>0.80 |
| **25 years - 30 years** | 1<br>1.47<br>0.68 | 1<br>1.91<br>0.52 | 8<br>5.27<br>1.52 | 1<br>3.65<br>0.27 | 7<br>12.91<br>0.54 | 3<br>1.34<br>2.24 | 21<br>26.56<br>0.79 |
| **>30 years** | 2<br>2.57<br>0.78 | 4<br>3.35<br>1.19 | 4<br>10.25<br>0.39[E] | 4<br>6.85<br>0.58 | 21<br>25.47<br>0.82 | 5<br>3.07<br>1.64 | 40<br>51.56<br>0.78 |
| **Total** | 8<br>7.31<br>1.09 | 12<br>9.70<br>1.24 | 23<br>28.58<br>0.80 | 10<br>21.08<br>0.47[E] | 42<br>58.78<br>0.71[E] | 8<br>5.52<br>1.45 | 103<br>130.97<br>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]<br>0.00[D]<br>-[E] | 1<br>0.13<br>7.56 | 1<br>0.32<br>3.12 | 0<br>0.10<br>- | 0<br>0.03<br>- | 0<br>0.00<br>- | 2<br>0.68<br>2.93 |
| **5 years - 10 years** | 0<br>0.04<br>- | 0<br>0.07<br>- | 1<br>0.21<br>4.68 | 0<br>0.20<br>- | 0<br>0.17<br>- | 0<br>0.00<br>- | 1<br>0.70<br>1.42 |
| **10 years - 15 years** | 0<br>0.04<br>- | 0<br>0.05<br>- | 0<br>0.16<br>- | 0<br>0.14<br>- | 1<br>0.26<br>3.83 | 0<br>0.01<br>- | 1<br>0.65<br>1.53 |
| **15 years - 20 years** | 0<br>0.03<br>- | 0<br>0.04<br>- | 1<br>0.11<br>9.45 | 0<br>0.08<br>- | 0<br>0.23<br>- | 0<br>0.01<br>- | 1<br>0.49<br>2.05 |
| **20 years - 25 years** | 0<br>0.02<br>- | 0<br>0.03<br>- | 0<br>0.07<br>- | 0<br>0.05<br>- | 0<br>0.16<br>- | 0<br>0.01<br>- | 0<br>0.34<br>- |
| **25 years - 30 years** | 0<br>0.02<br>- | 0<br>0.02<br>- | 0<br>0.06<br>- | 0<br>0.04<br>- | 0<br>0.13<br>- | 0<br>0.01<br>- | 0<br>0.27<br>- |
| **>30 years** | 0<br>0.02<br>- | 0<br>0.02<br>- | 0<br>0.06<br>- | 0<br>0.04<br>- | 0<br>0.16<br>- | 0<br>0.02<br>- | 0<br>0.32<br>- |
| **Total** | 0<br>0.26<br>- | 1<br>0.36<br>2.79 | 3<br>0.99<br>3.02 | 0<br>0.65<br>- | 1<br>1.14<br>0.87 | 0<br>0.06<br>- | 5<br>3.46<br>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]<br>0.07[C]<br>14.04[D] | 0<br>0.09<br>- | 1<br>0.22<br>4.46 | 0<br>0.07<br>- | 0<br>0.02<br>- | 0<br>0.00<br>- | 2<br>0.47<br>4.22 |
| **5 years - 10 years** | 0<br>0.04<br>- | 0<br>0.05<br>- | 1<br>0.19<br>5.35 | 0<br>0.20<br>- | 1<br>0.16<br>6.43 | 0<br>0.00<br>- | 2<br>0.63<br>3.17 |
| **10 years - 15 years** | 0<br>0.04<br>- | 0<br>0.06<br>- | 0<br>0.17<br>- | 0<br>0.17<br>- | 2<br>0.32<br>6.16 | 0<br>0.01<br>- | 2<br>0.77<br>2.61 |
| **15 years - 20 years** | 0<br>0.05<br>- | 0<br>0.06<br>- | 0<br>0.16<br>- | 1<br>0.13<br>7.72 | 0<br>0.40<br>- | 0<br>0.01<br>- | 1<br>0.81<br>1.24 |
| **20 years - 25 years** | 0<br>0.04<br>- | 0<br>0.05<br>- | 0<br>0.15<br>- | 0<br>0.11<br>- | 0<br>0.38<br>- | 0<br>0.03<br>- | 0<br>0.76<br>- |
| **25 years - 30 years** | 0<br>0.04<br>- | 0<br>0.05<br>- | 0<br>0.15<br>- | 0<br>0.10<br>- | 0<br>0.37<br>- | 0<br>0.04<br>- | 0<br>0.75<br>- |
| **>30 years** | 0<br>0.03<br>- | 0<br>0.04<br>- | 0<br>0.14<br>- | 0<br>0.09<br>- | 0<br>0.35<br>- | 0<br>0.04<br>- | 0<br>0.70<br>- |
| **Total** | 1<br>0.31<br>3.17 | 0<br>0.41<br>- | 2<br>1.18<br>1.70 | 1<br>0.87<br>1.15 | 3<br>2.00<br>1.50 | 0<br>0.13<br>- | 7<br>4.89<br>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]<br>2.29[C]<br>1.31[D] | 7<br>3.29<br>2.13 | 6<br>7.14<br>0.84 | 4<br>1.90<br>2.12 | 1<br>0.45<br>2.23 | 0<br>0.02<br>- | 21<br>15.08<br>1.39 |
| **5 years - 10 years** | 4<br>1.26<br>3.18 | 3<br>2.46<br>1.22 | 7<br>8.72<br>0.80 | 3<br>7.23<br>0.42 | 3<br>4.05<br>0.74 | 0<br>0.15<br>- | 20<br>23.87<br>0.84 |
| **10 years - 15 years** | 0<br>1.81<br>- | 2<br>2.38<br>0.84 | 8<br>8.82<br>0.91 | 6<br>9.29<br>0.65 | 14<br>11.28<br>1.24 | 0<br>0.40<br>- | 30<br>33.99<br>0.88 |
| **15 years - 20 years** | 0<br>2.25<br>- | 2<br>2.64<br>0.76 | 9<br>7.42<br>1.21 | 3<br>7.32<br>0.41 | 12<br>17.75<br>0.68 | 0<br>0.70<br>- | 26<br>38.07<br>0.68 |
| **20 years - 25 years** | 4<br>2.98<br>1.34 | 5<br>3.70<br>1.35 | 9<br>10.00<br>0.90 | 9<br>7.34<br>1.23 | 17<br>24.87<br>0.68 | 0<br>1.66<br>- | 44<br>50.54<br>0.87 |
| **25 years - 30 years** | 3<br>3.99<br>0.75 | 5<br>5.10<br>0.98 | 13<br>14.00<br>0.93 | 7<br>9.69<br>0.72 | 21<br>33.23<br>0.63[E] | 4<br>3.37<br>1.19 | 53<br>69.38<br>0.76[E] |
| **>30 years** | 5<br>7.00<br>0.71 | 6<br>8.99<br>0.67 | 18<br>27.10<br>0.66 | 18<br>18.25<br>0.99 | 52<br>66.41<br>0.78 | 5<br>7.80<br>0.64 | 104<br>135.55<br>0.77[F] |
| **Total** | 19<br>21.59<br>0.88 | 30<br>28.55<br>1.05 | 70<br>83.21<br>0.84 | 50<br>61.00<br>0.82 | 120<br>158.04<br>0.76[F] | 9<br>14.11<br>0.64 | 298<br>366.50<br>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]<br>0.26[D]<br>-[E] | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0.26<br>- |
| **7 days-5 years** | 36<br>49.70<br>0.72 | 6<br>14.59<br>0.41[F] | 6<br>11.89<br>0.50 | 3<br>6.53<br>0.46 | 2<br>4.78<br>0.42 | 0<br>0.51<br>- | 53<br>88.00<br>0.60[G] |
| **5 years-10 years** | 27<br>39.62<br>0.68[F] | 10<br>17.86<br>0.56 | 10<br>20.47<br>0.49[F] | 9<br>16.87<br>0.53 | 5<br>14.24<br>0.35[F] | 2<br>4.66<br>.43 | 63<br>113.72<br>0.54[G] |
| **10 years-15 years** | 18<br>43.35<br>0.42[G] | 11<br>19.87<br>0.50[F] | 10<br>24.87<br>0.40[G] | 17<br>26.98<br>0.63 | 21<br>24.22<br>0.87 | 11<br>9.01<br>1.22 | 88<br>148.28<br>0.59[G] |
| **15 years-20 years** | 35<br>37.93<br>0.92 | 8<br>17.92<br>0.42[G] | 14<br>23.32<br>0.60 | 16<br>29.03<br>0.55[F] | 15<br>34.41<br>0.36[G] | 7<br>11.64<br>0.60 | 95<br>156.24<br>0.61[G] |
| **20 years-25 years** | 35<br>35.85<br>0.98 | 15<br>16.43<br>0.91 | 20<br>27.39<br>0.73 | 17<br>32.48<br>0.52[G] | 37<br>43.03<br>0.86 | 20<br>20.25<br>0.99 | 144<br>175.42<br>0.82[G] |
| **> 25 years** | 75<br>93.83<br>0.80 | 36<br>43.78<br>0.82 | 63<br>67.867<br>0.93 | 92<br>105.40<br>0.87 | 121<br>138.22<br>0.87 | 54<br>82.00<br>0.66[G] | 441<br>531.57<br>0.84[G] |
| **Total** | 226<br>300.53<br>0.75[G] | 86<br>130.46<br>0.66[G] | 123<br>175.80<br>0.70[G] | 154<br>217.27<br>0.71[G] | 201<br>259.39<br>0.77[G] | 95<br>130.17<br>0.73[G] | 885<br>1213.62<br>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]<br>0.00[D]<br>-[E] | 0<br>0.00<br>- | 0<br>0.00<br>- | 0<br>0.00<br>- | 0<br>0.00<br>- | 0<br>0.00<br>- | 0<br>0.00<br>- |
| **7 days-5 years** | 0<br>0.30<br>0 | 0<br>0.10<br>- | 1<br>0.09<br>11.16 | 0<br>0.05<br>- | 0<br>0.03<br>- | 0<br>0.00<br>- | 1<br>0.57<br>1.75 |
| **5 years-10 years** | 0<br>0.29<br>- | 0<br>0.13<br>- | 0<br>0.16<br>- | 0<br>0.13<br>- | 0<br>0.11<br>- | 0<br>0.04<br>- | 0<br>0.87<br>- |
| **10 years-15 years** | 0<br>0.34<br>- | 0<br>0.16<br>- | 0<br>0.20<br>- | 1<br>0.21<br>4.71 | 0<br>0.19<br>- | 0<br>0.07<br>- | 1<br>1.18<br>0.85 |
| **15 years-20 years** | 0<br>0.31<br>- | 1<br>0.15<br>6.55 | 0<br>0.20<br>- | 0<br>0.25<br>- | 0<br>0.29<br>- | 0<br>0.11<br>- | 1<br>1.32<br>0.76 |
| **20 years-25 years** | 0<br>0.31<br>- | 0<br>0.15<br>- | 0<br>0.24<br>- | 0<br>0.30<br>- | 0<br>0.39<br>- | 1<br>0.18<br>5.57 | 1<br>1.56<br>0.64 |
| **> 25 years** | 2<br>0.81<br>2.46 | 1<br>0.38<br>2.58 | 1<br>0.60<br>1.66 | 0<br>0.99<br>- | 2<br>1.26<br>1.59 | 1<br>0.77<br>1.59 | 7<br>4.77<br>1.47 |
| **Total** | 2<br>2.36<br>0.85 | 2<br>1.08<br>1.85 | 2<br>1.50<br>1.33 | 1<br>1.87<br>0.53 | 2<br>2.28<br>0.88 | 2<br>0.17<br>1.71 | 11<br>10.27<br>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]<br>0.01[D]<br>-[E] | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0.01<br>- |
| **7 days - 5 years** | 2<br>2.90<br>0.95 | 0<br>0.54<br>- | 0<br>0.39<br>- | 0<br>0.16<br>- | 0<br>0.11<br>- | 0<br>0.02<br>- | 2<br>3.33<br>0.60 |
| **5 years - 10 years** | 5<br>2.38<br>2.10 | 1<br>1.14<br>0.88 | 0<br>1.13<br>- | 0<br>0.76<br>- | 0<br>0.58<br>- | 1<br>0.20<br>5.11 | 7<br>6.17<br>1.13 |
| **10 years - 15 years** | 2<br>3.10<br>0.64 | 0<br>1.49<br>- | 1<br>1.76<br>0.57 | 2<br>1.76<br>1.14 | 2<br>1.42<br>1.42 | 1<br>0.52<br>1.92 | 8<br>10.04<br>0.80 |
| **15 years - 20 years** | 4<br>3.05<br>1.31 | 0<br>1.47<br>- | 0<br>1.91<br>- | 0<br>2.33<br>- | 2<br>2.69<br>0.74 | 0<br>1.03<br>- | 6<br>12.49<br>0.48 |
| **20 years - 25 years** | 3<br>3.37<br>0.89 | 2<br>1.58<br>1.27 | 1<br>2.63<br>0.38 | 3<br>3.26<br>0.92 | 4<br>4.31<br>0.93 | 3<br>1.97<br>1.52 | 16<br>17.12<br>0.93 |
| **> 25 years** | 10<br>10.35<br>0.97 | 3<br>4.87<br>0.62 | 6<br>7.69<br>0.78 | 11<br>11.95<br>0.92 | 12<br>16.43<br>0.73 | 5<br>10.09<br>0.50 | 47<br>61.39<br>0.77 |
| **Total** | 26<br>24.36<br>1.07 | 6<br>11.10<br>0.54 | 8<br>15.52<br>0.52 | 16<br>20.22<br>0.79 | 20<br>25.53<br>0.78 | 10<br>13.83<br>0.72 | 86<br>110.56<br>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]<br>0[D]<br>-[E] | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- |
| **7 days-5 years** | 0<br>0.28<br>- | 1<br>0.09<br>10.60 | 0<br>0.09<br>- | 0<br>0.06<br>- | 0<br>0.05<br>- | 0<br>0<br>- | 1<br>0.58<br>1.74 |
| **5 years-10 years** | 0<br>0.17<br>- | 0<br>0.07<br>- | 0<br>0.09<br>- | 1<br>0.10<br>9.96 | 0<br>0.10<br>- | 0<br>0.03<br>- | 1<br>0.57<br>1.74 |
| **10 years-15 years** | 0<br>0.14<br>- | 0<br>0.06<br>- | 0<br>0.08<br>- | 0<br>0.10<br>- | 0<br>0.11<br>- | 0<br>0.05<br>- | 0<br>0.53<br>- |
| **15 years-20 years** | 1<br>0.09<br>11.56 | 0<br>0.04<br>- | 0<br>0.05<br>- | 0<br>0.07<br>- | 0<br>0.09<br>- | 0<br>0.04<br>- | 1<br>0.39<br>2.59 |
| **20 years-25 years** | 0<br>0.05<br>- | 0<br>0.02<br>- | 0<br>0.04<br>- | 0<br>0.05<br>- | 0<br>0.07<br>- | 0<br>0.04<br>- | 0<br>0.27<br>- |
| **> 25 years** | 0<br>0.08<br>- | 0<br>0.04<br>- | 0<br>0.06<br>- | 0<br>0.08<br>- | 0<br>0.12<br>- | 0<br>0.07<br>- | 0<br>0.45<br>- |
| **Total** | 1<br>0.81<br>1.23 | 1<br>0.33<br>3.05 | 0<br>0.40<br>- | 1<br>0.47<br>2.15 | 0<br>0.54<br>- | 0<br>0.24<br>- | 3<br>2.79<br>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]<br>0.04[D]<br>-[E] | 0<br>0.00<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0.04<br>- |
| **7 days-5 years** | 9<br>8.08<br>1.11 | 1<br>2.21<br>0.45 | 2<br>1.73<br>1.16 | 1<br>0.66<br>1.16 | 0<br>0.62<br>- | 0<br>0.08<br>- | 13<br>13.57<br>0.96 |
| **5 years-10 years** | 8<br>8.22<br>1.00 | 4<br>3.60<br>1.11 | 2<br>3.78<br>0.53 | 4<br>2.81<br>1.43 | 0<br>2.23<br>- | 1<br>0.74<br>1.35 | 19<br>21.17<br>0.90 |
| **10 years-15 years** | 6<br>9.67<br>0.62 | 1<br>4.43<br>0.23 | 3<br>5.30<br>0.57 | 5<br>5.32<br>0.94 | 4<br>4.37<br>0.92 | 3<br>1.61<br>1.86 | 22<br>30.70<br>0.72 |
| **15 years-20 years** | 11<br>8.95<br>1.23 | 2<br>4.13<br>0.48 | 4<br>5.35<br>0.75 | 2<br>6.33<br>0.32 | 4<br>7.27<br>0.55 | 0<br>2.77<br>- | 23<br>34.80<br>0.66[F] |
| **20 years-25 years** | 10<br>9.43<br>1.06 | 3<br>4.26<br>0.70 | 5<br>7.04<br>0.71 | 4<br>8.37<br>0.48 | 10<br>10.99<br>0.91 | 6<br>5.00<br>1.20 | 38<br>45.10<br>0.84 |
| **> 25 years** | 21<br>28.11<br>0.75 | 10<br>12.95<br>0.77 | 17<br>20.24<br>0.84 | 31<br>31.08<br>1.00 | 34<br>41.87<br>0.81 | 11<br>25.17<br>0.44[G] | 124<br>159.43<br>0.78[G] |
| **Total** | 65<br>72.31<br>0.90 | 21<br>31.58<br>0.66 | 33<br>43.43<br>0.76 | 47<br>54.77<br>0.86 | 52<br>67.34<br>0.77 | 21<br>35.38<br>0.59[F] | 239<br>304.81<br>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]<br>0[D]<br>-[E] | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- | 0<br>0<br>- |
| **7 days-5 years** | 1<br>0.21<br>4.66 | 0<br>0.07<br>- | 0<br>0.06<br>- | 0<br>0.04<br>- | 0<br>0.03<br>- | 0<br>0<br>- | 1<br>0.42<br>2.36 |
| **5 years-10 years** | 0<br>0.17<br>- | 1<br>0.07<br>13.81 | 0<br>0.09<br>- | 2<br>0.10<br>20.33[G] | 0<br>0.09<br>- | 0<br>0.03<br>- | 3<br>0.56<br>5.36[F] |
| **10 years-15 years** | 0<br>0.17<br>- | 0<br>0.07<br>- | 1<br>0.10<br>10.25 | 0<br>0.13<br>- | 0<br>0.14<br>- | 0<br>0.05<br>- | 1<br>0.67<br>1.50 |
| **15 years-20 years** | 0<br>0.15<br>- | 0<br>0.07<br>- | 1<br>0.09<br>10.96 | 0<br>0.13<br>- | 0<br>0.17<br>- | 0<br>0.07<br>- | 1<br>0.68<br>1.47 |
| **20 years-25 years** | 0<br>0.12<br>- | 0<br>0.06<br>- | 0<br>0.09<br>- | 0<br>0.12<br>- | 0<br>0.16<br>- | 0<br>0.08<br>- | 0<br>0.63<br>- |
| **> 25 years** | 0<br>0.18<br>- | 0<br>0.09<br>- | 0<br>0.13<br>- | 0<br>0.21<br>- | 0<br>0.29<br>- | 0<br>0.17<br>- | 0<br>1.07<br>- |
| **Total** | 1<br>1.02<br>0.98 | 1<br>0.43<br>2.33 | 2<br>0.57<br>3.49 | 2<br>0.73<br>2.76 | 0<br>0.89<br>- | 0<br>0.41<br>- | 6<br>4.04<br>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]<br>0.0[D]<br>-[E] | 0<br>0.0<br>- | 0<br>0.0<br>- | 0<br>0.0<br>- | 0<br>0.0<br>- |
| **7 days - 5 years** | 0<br>0.05<br>- | 0<br>0.03<br>- | 0<br>0.02<br>- | 0<br>0.02<br>- | 0<br>0.11<br>- |
| **5 years - 10 years** | 0<br>0.04<br>- | 0<br>0.04<br>- | 0<br>0.05<br>- | 0<br>0.05<br>- | 0<br>0.17<br>- |
| **10 years - 15 years** | 1<br>0.05<br>19.27 | 1<br>0.05<br>21.46 | 0<br>0.06<br>- | 0<br>0.08<br>- | 2<br>0.24<br>8.40[F] |
| **15 years - 20 years** | 0<br>0.05<br>- | 1<br>0.06<br>17.79 | 0<br>0.06<br>- | 0<br>0.08<br>- | 1<br>0.25<br>4.00 |
| **20 years - 25 years** | 0<br>0.06<br>- | 0<br>0.06<br>- | 0<br>0.06<br>- | 0<br>0.06<br>- | 0<br>0.24<br>- |
| **> 25 years** | 0<br>0.18<br>- | 0<br>0.13<br>- | 0<br>0.15<br>- | 0<br>0.20<br>- | 0<br>0.66<br>- |
| **Total** | 1<br>0.43<br>2.33 | 2<br>0.36<br>5.56 | 0<br>0.39<br>- | 0<br>0.49<br>- | 3<br>1.67<br>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<br>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<br>189.3-189.9 |
| **27-33** | MN of other and unspecified sites | | | |
| **27** | MN of skin | 190,191 | 172.0-172.4<br>172.6-172.9<br>173.0-173.4<br>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<br>198,199 | 194-196<br>197.0-197.7<br>197.9,198<br>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<br>239.6-239.7 |
| **40** | Other benign and unspecified nature neoplasms | 210-222<br>224-236<br>238-239 | 208,210-223<br>226-237<br>239 | 210-223<br>226-237.4<br>238.0239.5<br>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<br>281.1-281.4<br>282-285 | 280.000<br>281.1-281.8<br>282-285 |
| **44** | Coagulation defects,purpura,other hemorrhagic conditions | 296 | 286,287 | 286,287 |
| **45** | All other diseases of blood forming organs | 294-295<br>297-299 | 209<br>288,289 | 288,289 |
| **46-47** | Mental and personality disorders | | | |
| **46** | Alcoholism | 322 | 303 | 303 |
| **47** | Other mental disorders | 300-321<br>323-326 | 290-302<br>304-315 | 290-302<br>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<br>350-398 | 370-333<br>341-389 | 320-337<br>341-389 |
| **50-55** | Disease of the heart | | | |
| **50** | Rheumatic heart disease,including fever | 400-402<br>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<br>402,404 | 402-404 |
| **55** | Other diseases of the heart | 430-434 | 420-423<br>425-427<br>429 | 420-423<br>425-428<br>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<br>400.2,400.3<br>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<br>444.3-458 | 415-417<br>440-459 |
| **59-65** | Diseases of the respiratory system | | | |
| **59** | Acute respiratory infections except influenza and pneumonia | 470-475<br>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<br>527.2 | 500-519 | 470-478<br>494-519 |
| **66-69** | Diseases of the digestive system | | | |
| **66** | Diseases of the stomach and duodenum | 540-541<br>543 | 531-537 | 531-537 |
| **67** | Hernia and intestinal obstruction | 560-561<br>570 | 550-553<br>560 | 550-553<br>560 |
| **68** | Cirrhosis of the liver | 581 | 571 | 571 |
| **69** | Other diseases of digestive system | 530-539,542<br>544-545<br>550-553<br>571-578<br>580,582-587 | 444.2<br>520-530<br>540-543<br>561-570<br>572-577 | 520-530<br>540-543<br>555-558<br>562-570<br>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<br>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<br>605-609 | 591-593<br>595-599 | 588,589,591<br>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<br>720,721 |
| **82** | Osteomyelitis and periostitis | 730 | 720 | 730 |
| **83** | Other diseases of MS system | 731-749<br>726-727 | 716-718<br>721-738 | 710,717-719<br>722-729<br>731-739 |
| **84** | Symptoms and ill-defined conditions | 780-793<br>795 | 780-793<br>795,796 | 780-796,798<br>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<br>940-941 | 800-848<br>929.0-929.1 |
| **86** | Accidental poisoning | 870-895 | 850-877<br>942 | 850-869<br>929.2 |
| **87** | Accidental falls | 900-904 | 880-887<br>943 | 880-888<br>929.3 |
| **88** | Other accidents | 910-936<br>960-962 | 890-929<br>944-946 | 890-928<br>929.4-929.9 |
| **89** | Medical complications and misadventure | 940-959 | 930-936<br>947-949 | 870-879<br>930-949 |
| **90-91** | Violence | | | |
| **90** | Suicide | 963<br>970-979 | 950-959 | 950-059 |
| **91** | Homicide | 964<br>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.

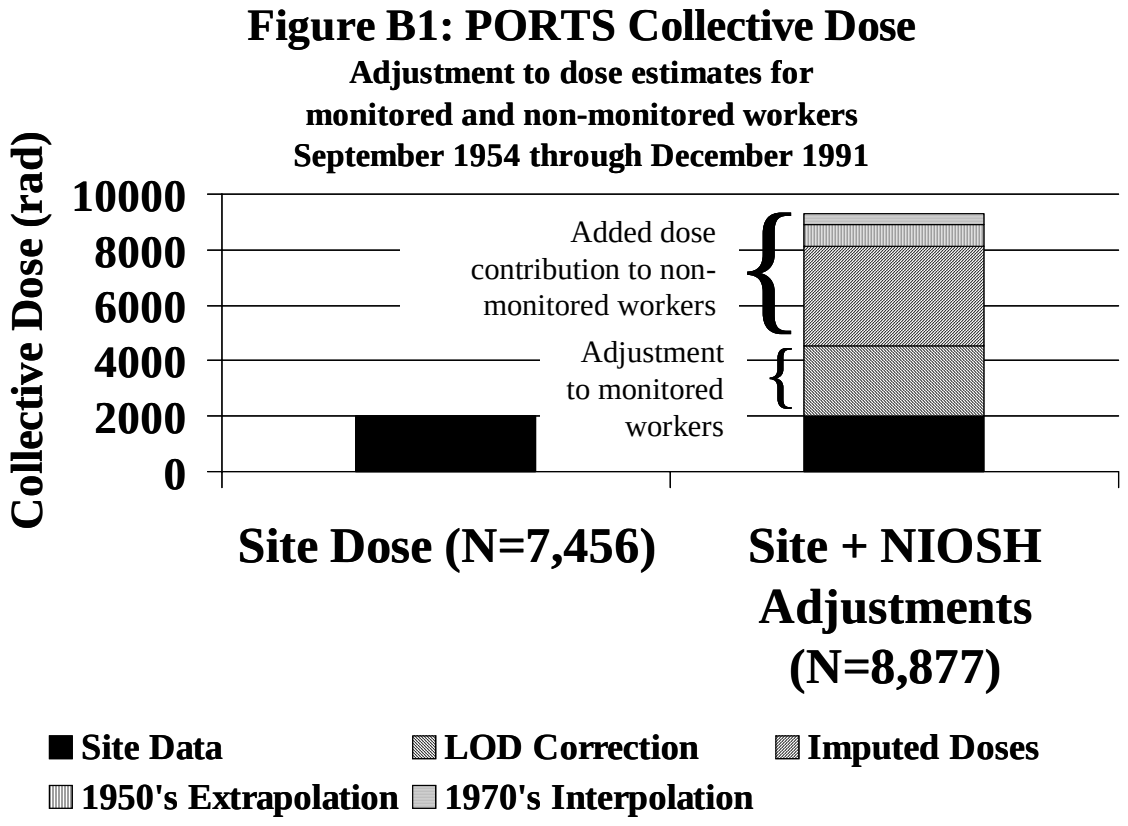
Figure B1: PORTS Collective Dose
Adjustment to dose estimates for
monitored and non-monitored workers
September 1954 through December 1991
Collective Dose (rad)
10000
8000
6000
4000
2000
0
Added dose
contribution to non-
monitored workers
Adjustment
to monitored
workers
Site Dose (N=7,456)
Site + NIOSH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