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Radium in Humans

A Review of U.S. Studies

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Notation

ACBM	Advisory Committee for Biology and Medicine (of the AEC)
AEC	Atomic Energy Commission
ALD	Associate Laboratory Director (of Argonne)
BIM	Biological and Medical Research (Argonne division)
CHR	Center for Human Radiobiology (of Argonne)
DBER	Division of Biomedical and Environmental Research (of the AEC)
ER	Environmental Research (Argonne division)
ESH	(Assistant Secretary for) Environment, Safety, and Health (of the U.S. Department of Energy)
ICD	International Classification of Diseases
ICRP	International Commission on Radiological Protection
JCAE	Joint Committee on Atomic Energy
MIT	Massachusetts Institute of Technology
NBS	National Bureau of Standards (now the National Institute of Standards and Technology)
NCRP	National Committee on Radiation Protection and Measurement
NJRRP	New Jersey Radium Research Project
OER	Office of Energy Research (of the U.S. Department of Energy)
RER	Radiological and Environmental Research (Argonne division)
RPY	Radiological Physics (Argonne division)
SNOP	Systematized Nomenclature of Pathology

Preface

This document was originally conceived as a compilation of activities at Argonne National Laboratory that were directed toward the study of radium in humans. However, it soon became obvious that this was a very limited approach, because such a compilation would include no background on the widespread uses of radium in industry and in the medical profession, nor would it address the early history of the discovery of the hazards of radium. Such an approach would also ignore contributions to the study of radium effects made at other laboratories. This document now addresses these topics, quite briefly to be sure, in order to give an overall picture of what might be called the radium era, that period from the early part of this century, when radium was rapidly exploited as a tool and a medication, to the present time, when radium is not generally used and the study of its effects has been terminated.

This history was initiated with the approval of the U.S. Department of Energy in January 1988, but it was supported only sporadically until Dr. Robert G. Thomas became manager of the radium studies program at Argonne in 1991. With his support I was able to complete the first draft of the manuscript by the time the program was terminated at the end of September 1993. Discussions of a few papers presented at an international seminar in April 1994 were added subsequently.

Unfortunately, size and time limitations required that much be omitted from this document. The contributions of many individuals involved have not been addressed, and many productive avenues of research have not been mentioned. My intention has been to include sufficient references to guide the interested reader to additional studies on the radium cases. The text mentions the series of annual reports produced by the short-lived New Jersey Radium Research Project. Readers should be aware that both at the Massachusetts Institute of Technology and at Argonne National Laboratory semiannual or annual reports were produced every year. These reports contain a very large body of information on the respective radium studies.

The subjects covered in this text and those omitted reflect the interests and biases of the author. No doubt other writers would have included a different set of items. The author's hope is that the variety of subjects presented is sufficient to whet the interests of readers who are unfamiliar with the history and consequences of the uses and abuses of radium in this country.

As I look back on an exciting and satisfying career studying the effects of radium deposited internally in man, one overall conclusion appears to be worth emphasizing. Given the number of people who acquired radium internally, it is remarkable how few suffered significant damage. To be sure, those who eventually developed radium-induced malignancies suffered severely. Those who acquired very large internal quantities of radium, as did many of the early dial painters, also suffered from what we today suspect were acute radiation doses leading to early deaths. However, the great majority of exposed individuals went through life with no recognizable consequences of their exposures. They lived as long as, and apparently in as good health as, their unexposed neighbors. This fact seems to have been little appreciated and seldom mentioned, but it may be the most important finding of the entire study.

In the same vein, we should note that acquiring sufficient radium internally to put one at risk is not easy. Many individuals were referred to the Center for Human Radiobiology at Argonne for body content measurements after exposure to radium in the workplace or at a contaminated site, only to find no detectable radium in their bodies. Intravenous injection is by far the most effective method for obtaining a high radium burden. Oral ingestion of material with a high specific activity is also effective, in spite of the fact that 80% of what is ingested is not absorbed into the body. Except for a few radium chemists, who may have inhaled as well as ingested radium, only exposure in the dial painting industry, from medical treatments, or from self-administered radium has led to serious consequences. This fact has also not been widely disseminated.

The appendix to this review lists all of the measured radium cases, a total of 2,403 individuals whose records were in the files at the end of 1990. For each case the route of exposure, the dates of exposure, the years of birth and death, the measured body content, the calculated intake and dose, and the cause of death have been listed. This appendix was created from the Argonne radium databases by Thomas J. Kotek, to whom I am indebted for valued assistance during the writing of this manuscript.

These 2,403 cases, however, are not all of the measured cases. Some individuals referred to Argonne for measurement after suspected radium exposure were not considered radium cases when no radium was found by gamma-ray measurements. In contrast, individuals who worked in the radium dial industry or as radium chemists but were found to contain no measurable radium were listed as radium cases on the basis of their exposure history.

Only those who were alleged to have been exposed but whose actual exposure could not be verified have been omitted. This practice can perhaps be justified on the basis that all human beings contain radium, obtained from their exposure to food or water on our radioactive planet, yet we are not all considered radium cases because of these environmental exposures.

This report could not have been prepared without the help of many members of Argonne's support staff. The largest contributions were made by artist Michele Szawars, word processors Barbara Salbego and Judith Robson, and editor Karen Haugen. To all contributors, I am grateful.

Foreword

The opportunity to study a discrete human population exposed to a potentially toxic agent is rare, and it is rarer still to be able to collect sufficient data on the exposed subjects to reach definitive conclusions about the potency of the toxicant. Such an opportunity emerged when skeletal lesions, including tumors, became associated with workers (luminizers) who painted luminous dials on timepieces, with radioactive radium as the energy source for luminosity. This correlation was observed in the 1920s, when the dial painting industry was centered in the northeastern United States.

The initial medical finding that radium caused the lesions was followed shortly by interest among researchers in the physical sciences in dosimetric measurements of the radium body burdens of the exposed subjects. This interest was stimulated by a turning point in medical history. In the past, the dose of any material was the amount taken into the body through the mouth, a muscle, or a vein. Now, in contrast, it was possible to measure the radium dose in the organ of primary deposition (the skeleton), and this dose could be correlated with the clinically observed skeletal damage.

The concept that radiation should have its own exposure standards grew from early radiobiological effects on humans working with the new radiation energy sources. The first official exposure standard for a radionuclide in the body was established for ^{226}Ra at a level of 0.1 μCi, by a task group assembled by the U.S. National Bureau of Standards in 1941. On the basis of the average radiation dose to the skeleton from deposited radium, a "practical threshold" dose of 10 Gy was established many decades ago by the pioneer of the work contained in this book, Dr. Robley Evans, then at the Massachusetts Institute of Technology. It was Evans who, through appearances before the Atomic Energy Commission and congressional committees, spearheaded the establishment of the Center for Human Radiobiology at Argonne National Laboratory in 1968.

This book is rightly timed. The Internal Emitter Program at Argonne, which was the focal point for data collection and the principal source of information from this type of research over the last 25 years, ended at the direction of the U.S. Department of Energy on September 30, 1993. Much remains to be studied within the medial and dosimetric files on the radium-exposed subjects. The intention is that these files will be proprietarily accessible to scientists who have the means and interest to pursue the unknowns they contain. Many of the luminizers are still alive, in their later decades of life, and are somewhat accessible. At their present age, any

additional tumors of the bone or other organs could be attributed to normal incidence, so the time for radiation-effected clinical findings has passed. Subtle changes may be observed, however, through future molecular studies. Although such findings would not alter the practical threshold for tumorigenicity, they would be interesting indicators of radiobiological effects long after exposure. The radiographs from partial or whole-body X-rays used to observe subtle changes in bones of the exposed subjects are also of interest to the medical community, as has been recently attested by well-known radiologists.

The scientists associated with this program since its inception are numerous, and some are still actively analyzing the data today, in their retirement years. Naming such persons entails the risk of missing someone important. The primary contributors are mentioned throughout the book, through the many references cited. This treatise is a monument to these scientists, to accompany their many accomplishments over the years. The author, Dr. Rowland, deserves credit for his contributions to the radium studies; he served as the first director of the Center for Human Radiobiology.

As the most recent principal investigator for the Internal Emitter Program, it is my pleasure to introduce this book to its many interested readers throughout the world.

Robert G. Thomas
Program Manager
Environmental Research Division
Argonne National Laboratory

1 Introduction

This document was originally conceived as a description of the radium studies that took place at Argonne National Laboratory. It soon became evident, however, that to document the widespread use of radium, a brief review of the application of radium in medicine and in the U.S. dial painting industry is required. Further, because the Argonne studies were not the only such efforts, brief overviews of the other radium programs are included. Even so, much material has been omitted. The extensive references included will allow the interested reader to find additional information.

The effects of internally deposited radium in humans have been studied in this country for more than 75 years. Some 2,400 subjects have had their body contents of radium measured, and a majority of them have been followed for most of their adult lives, to understand and quantify the effects of radium. Many more individuals acquired radium internally but were never measured. Some of this group have been located and followed until death; in these cases the cause of death is known without a body content measurement.

As a consequence of the efforts made to locate, measure, and follow exposed individuals, a great deal of information about the effects of radium is available. Nevertheless, great gaps remain in our knowledge of radium toxicity. For example, when an adult woman, over a period of several months or years, ingests 5,000 μCi of ^{226}Ra or receives 1,000 μCi of ^{226}Ra by intravenous injection,* she has a relatively high probability (30-50%) of developing a bone sarcoma or head carcinoma induced by internal radium, 5-50+ years later. If an adult male receives the same quantity of radium, he too is at risk, but the probability that he will develop a malignancy appears to be much lower. Is this difference real?

The probability of the induction of one type of radium-induced malignancy, the bone sarcoma, in an adult female appears to be very low when the quantity of ^{226}Ra absorbed into the blood is less than 100 μCi. The probability then rises rapidly with increasing radium intake. This rise is not linear, but it is better characterized by the square or cube of the radium intake. Are the data that lead to this conclusion valid?

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* This document is a review of work covering much of this century. Most of the published information reports activities in curies. The author has chosen to retain this convention instead of converting activities to becquerels, the International System unit. (The becquerel [Bq] is equal to 27 pCi.) A different approach has been adopted for dose. The traditional unit, the rad, is equivalent to 0.01 Gy (gray). Therefore, all dose values are stated in cGy and are numerically equal to previously published dose values in rads.

No symptoms from internal radium have been recognized at levels lower than those associated with radium-induced malignancy. Radium levels 1,000 times the natural ^{226}Ra levels found in all individuals apparently do little or no recognizable damage. These statements may suggest that a threshold exists for radium-induced malignancies; at least, they recognize that the available data demonstrate a steep dose response, with the risk dropping very rapidly for lower radium doses.

These and other unexpected conclusions have come from Argonne's study of radium in humans. The Argonne study is the largest ever undertaken of the effects on humans of an internally deposited radioelement, in which the insult has been quantitated by actual measurements of the retained radioisotope. The study has now been terminated, even though more than 1,000 subjects with measured radium burdens are still alive. The original plan was to follow all measured cases until death, so that a large body of radiation experience would be obtained and preserved for future study. However, the data accumulated to date will apparently constitute the total available human radium experience. This document is written as a brief summary of current knowledge accumulated in this incomplete study.

2 Historical Background

The Medical Uses of Radium

After the isolation of radium in 1898 by the Curies, this element presented intriguing challenges for chemists and physicists. These challenges were in part due to radium's rarity and to the difficulty of separating minute fractions of it from its surroundings, but even more to the complex array of daughter products that accompanied the parent element. Physicians, too, were intrigued with radium and with the energy lost during the process of radioactive decay. Interest centered on the use of the gamma rays given off during decay, primarily by the daughter products of radium. These gamma rays appeared similar to the recently discovered X-rays. Also of interest were the effects of energetic heavy particles within the human body. This new form of energy deposition suggested that radium decay might have some therapeutic potential. Such speculation led to the first laboratory trials of radium in the United States.

The limited availability of radium and its high monetary value encouraged two brothers, Joseph M. and James J. Flannery, to form a company called the Colorado Chemical Mining Company to mine carnotite ore and extract radium from it (Lounsbury 1938). The Flannerys were spared the problem of obtaining mining properties, because the American Vanadium Company, of which they were founders, owned numerous claims in Colorado and Utah (Bruyn 1955). In 1910 the Flannerys organized another company, the Standard Chemical Company, to handle their new operations and set up a plant in Canonsburg, Pennsylvania, where radium was extracted from the ore. The first commercial radium was produced by the end of 1912, and by 1914 the Flannerys provided 4 g of radium to England for use in the war efforts (Silverman 1950). By 1920, 30 g of radium had been produced, and by 1922 the Canonsburg plant was producing radium at a rate of about 18 g per year, satisfying the U.S. demand.

The Standard Chemical Company, in an attempt to expand the market for radium, examined a number of potential uses. Of particular relevance were those related to the manufacture of dial paint and to the medical uses of radium. The company formed a wholly owned subsidiary, the Radium Chemical Company, Inc., to handle the sales of radium. This organization produced a dial paint containing radium and experimented with the addition of rare earths to the zinc sulfide in the dial paint for better light emission under alpha-particle bombardment. The Standard Chemical Company also maintained a well-equipped and well-staffed biological laboratory to investigate medical uses of radium. Out of the latter came the establishment

in 1913 of the journal *Radium* (Figure 1), in which physicians could record the results of the treatment of many diseases through internal or external application of the element. These experiments appeared to be serious investigations of the medical uses of the new material.

The Standard Chemical Company put much effort into its research on the biological effects of internally administered radium. Its research laboratory supported the publication *Radium* until 1921. The director of the laboratory, Dr. Frederick Proescher, treated many patients with radium, in order to identify the conditions responding most readily to radium treatments and the doses required. In an early series of four papers, Proescher (1913, 1914a-c) stated that he had injected 34 individuals with radium and described in detail the results for 16 subjects with arthritis. These 16 individuals received multiple radium injections totaling 70-350 μCi. Proescher stated that doses up to 1,000 μCi were tolerated well; he estimated that the lethal dose to a human was 60,000 μCi.

None of the individuals who received radium in Proescher's studies have been identified. However, another publication from the staff of the Standard Chemical Company, by Seil et al. (1915), did identify Mr. Seil himself as one of the early recipients of radium. The authors of this paper were investigating the rate and routes of radium loss from the body; they published data on two adult subjects. One was a 23-year-old male who received two intravenous injections of 100 μCi of ^{226}Ra two months apart, and the other was Seil, who received two 50-μCi doses of radium by mouth seven days apart. Seil, who was then 33 years old, died at the age of 69; the cause of death was listed as diverticulosis of the colon. Subsequently, after an exhumation, his body content of ^{226}Ra was determined to be 0.15 μCi, and his initial systemic intake was calculated as having been 73 μCi.

The *initial systemic intake* is a measure of the total quantity of radium that entered the systemic circulation. It may be considered equal to the quantity intravenously injected or to about one-fifth of the amount taken orally (Maletskos et al. 1966). The initial systemic intake is calculated from a subsequent measurement of the body content and the time interval between acquisition and the measurement. In a case like Seil's, the recorded intake was assumed to be the only such intake, an assumption that probably was not valid.

Although the producers of radium investigated its medical possibilities, its greatest use and endorsement as a medication came from others (Figure 2). One of these early proponents was a physician, Dr. C. Everett Field, listed for

RADIUM

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INFLUENCE OF INTRAVENOUS INJECTION OF SOLUBLE RADIUM SALTS IN HIGH BLOOD PRESSURE.

By FREDERICK PROESCHER, M. D.

FIGURE 1 The April 1914 issue of the journal *Radium* carried this title page.

a short time (1915-1916) as manager of the New York office of the Radium Chemical Company. He subsequently published both in medical journals and in privately printed pamphlets on the virtues of radium therapy and had a lucrative practice until the late 1920s, when the hazards of internal radium became known. In one publication, Field (1926) stated that he had administered 6,000 intravenous radium treatments in the preceding 12 years.

Field apparently believed in the internal use of radium, because after his death he was found to have a significant radium burden. He died in 1951 at the age of 81. The cause of death was listed as a cerebral hemorrhage. His body content was measured as 0.34 μCi of ^{226}Ra, and his initial systemic intake was calculated to have been 92 μCi.

A midwestern physician, Dr. Findley John, was also known to have used radium in his practice. In a 1951 interview with an Argonne staff member (Dr. W.B. Looney), John, then 82 years of age, stated that he had treated

THE COUNCIL ON PHARMACY AND CHEMISTRY OF THE AMERICAN MEDICAL ASSOCIATION HAS ACCEPTED FOR INCLUSION WITH NEW AND NON-OFFICIAL REMEDIES THE FOLLOWING PRODUCTS OF THE STANDARD CHEMICAL COMPANY OF PITTSBURGH, PENNSYLVANIA:

Radium Bromide
Radium Chloride
Radium Sulfate
Radium Carbonate

"Standard" Radium Solution for Drinking
"Standard" Radium Solution for Bathing
"Standard" Radium Compress
"Standard" Radium Earth

For Literature Address

RADIUM CHEMICAL COMPANY

Forbes and Meyran Avenues, Pittsburgh, Pa.

You are cordially invited to visit our exhibit at the annual meeting of the Illinois State Medical Society, Springfield, Illinois, May 18th, 19th and 20th, 1915, Masonic Temple. Booth Number 18.

FIGURE 2 This advertisement for radium preparations, which appeared in the May 1915 issue of the journal *Radium*, included a statement of acceptance by the American Medical Association.

hundreds of patients, both orally and intravenously, with 10 μg of ^{226}Ra every five days, with total doses of 100-300 μg. (Note that, for ^{226}Ra, 1 μg is equivalent to 1 μCi.) John also stated that he had taken about 1,000 μg himself and had given his wife about 200 μg. (John's intake was subsequently estimated to have been 711 μCi; his wife's was estimated at 508 μCi.) John never identified any of the subjects he treated with radium, but some radium patients identified by their symptoms subsequently stated that John had treated them. A total of 29 individuals who had received radium from John have been located; 21 of them were measured for radium, and most had high intake levels. Five ultimately developed bone sarcomas and another a mastoid air cell carcinoma, two malignancies known to be induced by internal radium.

Perhaps the best known form of radium available to the public in the 1920s was radium water (bottled drinking water spiked with radium and sold over the counter or by mail). The brand that has received the greatest notoriety was "Radithor." Radithor was sold by the case; 30 bottles, a month's supply, were sold at one time. Each bottle was claimed to contain 2 μCi of radium in distilled water. Radithor, a product of William J.A. Bailey, was sold by his Bailey Radium Laboratories of East Orange, New Jersey. The full history of Bailey's activities was documented by the Bureau of Investigation of the American Medical Association (1932).

Radithor met its end after the death of one Eben MacBurney Byers. *Time* magazine for April 11, 1932, under the heading "Medicine," published a full-page account of the death of this prominent sportsman and Pittsburgh businessman. *Time* stated that Byers died of radium poisoning after several years of consumption of Radithor. Gettler and Norris (1933), in an account of findings derived from an autopsy on Byers, stated that he had consumed about 1,400 bottles of Radithor and that his body contained 74 μCi of radium. The cause of death was stated to be necrosis of the jaw, abscess of the brain, secondary anemia, and terminal pneumonia. (After a 1965 exhumation, the actual body content was found to be 6.1 μCi, and the initial systemic intake was calculated as 349 μCi of ^{226}Ra and 600 μCi of ^{228}Ra. With the generally accepted value of 20% for absorption of ingested radium, the total radium ingested was about 5,000 μCi, suggesting that Byers may have consumed somewhat more than the 1,400 bottles estimated by Gettler and Norris.)

More than 80 subjects in the files of Argonne's Center for Human Radiobiology are listed as having consumed Radithor, but this number must represent only a very small fraction of individuals who purchased this product. Apparently some 400,000 bottles of Radithor were sold in 1925-1930 at $1 per bottle (Macklis 1990). William J.A. Bailey himself is

among those listed; he died at the age of 64 of a carcinoma of the bladder. After an exhumation in 1970, his body content was found to be 1.1 μCi, and his systemic intake was calculated as 159 μCi of ^{226}Ra and 140 μCi of ^{228}Ra.

In addition to the use of radium by individual physicians and others, this radioactive material was also used in certain medical institutions. An account of treatments for hypertension and pain at a midwestern clinic published by Allen et al. (1927) described eight patients treated with radium water in 1922 and another series treated with intravenous injections starting in 1925. A total of 116 subjects were found to have been treated with radium during this short period. Unfortunately, these patients were not identified until the early 1970s, and by that time most of the few survivors were difficult to locate.

One study of the effects of intravenously administered radium has provided researchers with invaluable evidence regarding the retention of radium within the human body. A series of four papers, Schlundt et al. (1929), Barker and Schlundt (1930), Schlundt and Failla (1931), and Schlundt et al. (1933), discussed the measurement of radium in the body and the elimination of radium from the body. The last paper described the intravenous injection of radium into 32 patients, primarily classified as having dementia praecox (now called schizophrenia), at the Elgin (Illinois) State Hospital.

Schlundt et al. (1929) reported body content measurements for seven radium-containing individuals, with no mention of how the subjects obtained their radium. Barker and Schlundt (1930) reported body content measurements for ten cases, with the route of administration (oral or intravenous) and the dates of multiple administrations. These ten individuals, who received 80-1,455 μCi of ^{226}Ra, were identified. Three were subsequently located by Argonne staff, and body burden determinations were made on two of them. One of these individuals was both a dental surgeon and physician. He received 330 μCi of ^{226}Ra, all intravenously. His body content was reported to have been 9-14 μCi in 1929. When he was measured at Argonne in 1951, his body burden was 10.7 μCi, and his initial systemic intake was calculated to have been 1,180 μCi. He probably received radium at times other than those listed by Barker and Schlundt (1930). He and his family are also known to have received radium from John. His son, measured in 1951, contained 5.6 μCi (systemic intake 720 μCi), and his wife, measured in 1952, contained 1.0 μCi (systemic intake 193 μCi). The dentist died of aplastic anemia in 1953 at the age of 79; his wife died of a bone cancer in 1952, at age 74. The son died of a heart problem at age 55.

Except for the papers in the Schlundt series, no records have ever been obtained of physicians who, in private practice, administered radium to their patients. Thus, the 1933 publication (Schlundt et al. 1933) that documented the quantity of radium given to each Elgin patient was of unique importance. Many of the patients were still at Elgin in the 1950s, and their individual files documented the dates and quantities of their radium injections. The radium body contents of 19 of these patients were measured a number of times in the 1950s by Argonne staff members.

W.P. Norris and his associates reviewed two sets of body content measurements, made six months apart and reported by Schlundt et al. (1933) on the Elgin State Hospital patients, and two sets made by Argonne personnel 233 and 258 months after the original measurements. Norris and associates also acquired from various sources sealed radium ampoules that had been obtained in the 1930s from H.M. Armstrong of the Radium Extension Service of Chicago, the source of the radium used at Elgin and by many Chicago area physicians (and produced by the U.S. Radium Corporation). These ampoules were found in 1953 to contain 10 μCi of radium, within a few percent, and no more than 0.05% mesothorium (^{228}Ra).

Norris et al. (1955) found that the retention of radium in the Elgin cases could be best fitted by a power function of the form $R_t = 0.54\ t^{-0.52}$, where R_t is the fractional retention at time t, and t is expressed in days ($t \geq 1$ day). The Elgin cases were thus of considerable value in determining the pattern of radium loss from the body over a long period.

These groups of radium cases, particularly the larger group receiving radium at the midwestern clinic, might have constituted useful cohorts for epidemiologic studies, but this promise was unfulfilled. Both groups suffered from the common fault that although quantities of radium injected and the dates were usually included in the medical records, other evidence suggested that some individuals received further, unrecorded radium treatments. Thus, in these cases the recorded treatments must be taken as the lower limits of the actual radium injection levels. Even without this problem, the small size of these groups limits their value for epidemiologic studies. A detailed comparison of these two groups is provided in Chapter 3.

Untold numbers of individuals received radium internally from private physicians, from a medical institution, or by medicating themselves with radium waters (Figure 3). Evans (1966) estimated that several thousands of individuals had received radium from physicians and calculated that 400,000-500,000 bottles of Radithor were sold to the public. The small fraction of these cases that are known might provide evidence of the effects of

FIGURE 3 This advertisement for radium preparations to be used internally and externally appeared in the June 1916 issue of the journal *Radium*.

high levels of radium, but these few cases are not suitable subjects for epidemiologic studies of the effects of internally deposited radium. Thus, the radium dial painters have been the only cohort of exposed persons found suitable for such epidemiologic studies.

The Dial Painting Industry

The early industrial uses of radium capitalized on the element's ability to cause some specific materials to fluoresce. This fluorescence was well known, because it provided one of the early methods of measuring the strength of an alpha-emitting source. Observation through a microscope of the number of flashes of light given off in a measured time interval by a suitable screen allowed the number of alpha particles given off by a small source to be determined. Not surprisingly, attempts were made to find materials that would glow continually under constant, heavy bombardment.

The first use of such a radioluminous material in the United States was in 1903 by Dr. George F. Kunz, who is reported to have painted the hands of his wrist watch so that he could read it in the dark.* Kunz filed for a patent in September 1903 and was subsequently issued a patent for a radioluminous compound to be used on watches. A very similar patent was filed by Mr. Hugo Lieber in May 1904 for a radioluminous compound. The Ansonia Clock Company of New York was the first firm to produce and sell radioluminous products. The radium for their products was undoubtedly acquired from Europe.

The individual who was primarily responsible for expanding the use of radioluminous products was Sabin A. von Sochocky, who had earned both a medical degree and a Ph.D. in Europe before immigrating to this country in 1906. In 1913, apparently to earn money to finance his medical research, he developed a radium paint and sold some 2,000 radium dial watches. Because a large and growing market existed in this field, von Sochocky continued his research on radioluminous compounds. In 1915 he, with Dr. George S. Willis, founded the Radium Luminous Materials Corporation, which provided

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* This information, and much of what follows, was found in the notes of Swen Kjaer, which are now in the files of the Internal Emitter Program at Argonne National Laboratory. Kjaer, an employee of the U.S. Department of Labor, Bureau of Labor Statistics, traveled extensively, visiting institutions and individuals who reportedly were using radium in their work. The notes record Kjaer's findings on field trips made in 1925 and 1928. These notes were turned over by Kjaer to the Radium Research Program in New Jersey in 1959, then subsequently to Professor Robley Evans at the Massachusetts Institute of Technology, and in turn to Argonne when the other programs were terminated.

radium-luminous paint to other companies and was itself involved in the actual painting of dials. This firm prospered, primarily because the demand for luminous compounds was stimulated by the war. The original plant was in Newark, New Jersey, but the company moved to larger quarters in Orange, New Jersey, in 1917. The firm was reorganized as the U.S. Radium Corporation in 1921. von Sochocky left the firm in 1922 and formed a new company, the General Radium Corporation, which supplied radium for medical uses but was not involved in the manufacture of luminous compounds.

A radium extraction plant and a radium dial painting studio were set up on the site in Orange, New Jersey. Changing the name of the firm to the U.S. Radium Corporation reflected an interest in selling radium to the medical profession as well as to the dial painting industry. The rapid growth of the dial painting industry at this time is documented in an article published in *Scientific American* in 1920 (Mount 1920), which stated that more than 1,000,000 watches and clocks had already been produced by this rapidly growing industry.

The U.S. Radium Corporation started using mesothorium (^{228}Ra) in its dial paint in May 1919, a move that allowed it to substantially reduce the price of its product. The company was said to have eventually controlled the entire output of mesothorium in the United States. Its raw material was obtained from the Welsbach Company in Glouchester, New Jersey, and the Lindsay Light Company of Chicago; both of these companies extracted thorium from monazite sand. The residue was sold to the U.S. Radium Corporation. The final refining of this product, which was described as 50% mesothorium and 50% barium bromide, was undertaken in the laboratory in Orange, New Jersey.

Soon after its formation, the U.S. Radium Corporation began to experience problems with the health of its dial painters. These problems are well summarized in a letter written by its president, dated June 18, 1928, to the Commissioner of Public Health of the City of New York. The following statements are taken from that letter; they outline the sequence of events that took place and give a clear impression of the corporate point of view.

> The luminous material business of this corporation was started in 1914 or '15 as a private enterprise. . . .
>
> About the year 1917, the present corporation, then known as The Radium Luminous Material Corporation, took over the business of the parent company and greatly expanded its operations. Mines were acquired, a plant for the extraction of

radium was opened in Orange, and a very much enlarged plant for the application of luminous material was built. . . .

The [dial painting] work was easy, the operators well paid, and as conditions turned out, we unfortunately gave work to a great many people who were physically unfit to procure employment in other lines of industry. Cripples and persons similarly incapacitated were engaged. What was then considered an act of kindness on our part has since been turned against us, as all previous employees, regardless of what they may have been suffering from or are suffering from at the present time, in the minds of the general public can be attributed to "Radium Poisoning." Our operations were carried on at a very large scale until about 1920, when our large customers began to install, with our assistance, application plants in their own factories. This policy naturally reduced our force considerably, until in 1923 or 1924, we had only a mere handful of operators engaged in luminous material application.

In the early part of 1924, it was called to our attention, through a highly reputable dental firm, that some of our employees were suffering from what was believed to be phosphorus poisoning. . . .

There was a thought at the time that if a condition existed it might be due to the brushes, and therefore all brushes were thoroughly sterilized before being used by the operators. Absolute instructions were given not to point the brushes with the lips. We also engaged Dr. Cecil K. Drinker of the Industrial Hygiene Section of the Harvard School of Medicine to make a survey of our plant and to make recommendations. Dr. Drinker made several trips to the plant, made numerous tests, and finally reported on June 3, 1924, that since he could find no direct cause for the apparent trouble which existed, radium looked suspicious. . . .

In the early part of 1925, two suits were instituted against the corporation, claiming injury from their employment. About this time the Consumer's League of New Jersey took an active interest in the matter, and enlisted the support of Dr. Frederick L. Hoffman, Statistician for the Prudential Insurance Company. Although not a physician and having no knowledge regarding radium, Dr. Hoffman read a paper before the American Medical Association Convention in Atlantic City, May 1925, and named

the disease in New Jersey "Radium Necrosis." Dr. Hoffman claims credit for having discovered this so-called disease, and we believe the published literature bears out his contention. . . .

Confronted with law suits, and with a paper purporting to have discovered a brand new disease among our former employees, also propaganda from the Consumer's League of New Jersey, we took further advice from competent medical authorities on what should be done. It was suggested that the Industrial Hygiene section of the College of Physicians and Surgeons, Columbia University, might undertake scientific investigations to ascertain if there really was danger existing in the industry. Dr. Frederick B. Flinn became interested in March 1925, and began a scientific study of the question. . . .

Based on the scientific data acquired, which was undoubtedly the most thorough study of the subject, Dr. Flinn reached the conclusion that there was no industrial hazard in the industry. However, soon after this article appeared there was brought to his attention a case of a former applicator who contained a certain amount of radioactivity. While there were other implications involved, after a period of 6 or 8 months observation and treatment, and a final autopsy, Dr. Flinn reached the conclusion that undoubtedly radioactivity contributed to the condition of the girl. . . .

Perhaps it would not be amiss to discuss briefly the recent suits against this corporation, which have received so much unwarranted and untrue publicity. . . .

Through a, no doubt, cleverly designed campaign of publicity, the public was appealed to and the appeal met a responsive chord. . . .

The spectacle of five women filled with radium, doomed to a speedy and terrible death, according to experts, presented a gruesome picture indeed. . . .

The fact that we settled these suits in no way is indicative of the merits of the complainants [*sic*] contentions, or that we admit responsibility or liability. From a legal aspect there is very little question but that we had a perfect defense, both from the standpoint of the Statute of Limitations and from the fact that there was no negligence on our part. . . .

Interestingly, this letter did not mention the findings and the many publications of Dr. H.S. Martland, who firmly and without hesitation stated that the problems in the industry were due to radium and mesothorium. Martland's contributions are detailed below under "Early Findings on Health Effects of Radium."

The U.S. Radium Corporation found it necessary to close its dial painting plant in Orange, New Jersey, by 1926, because the unfavorable publicity generated by the newspaper accounts of the tragedies that befell the early dial painters made finding employees difficult. The company moved its offices and facilities to New York City and continued its business, but never again at the level of activity that prevailed at Orange.

Dial painting also took place in the Midwest, primarily in Illinois. The Radium Dial Company was organized as a division of the Standard Chemical Company in 1917, to give the company an entry into the dial painting industry. The Radium Dial Company was operating a dial painting studio in Chicago as early as 1918, located in the Marshall Field Annex building.

The Radium Dial Company moved from Chicago to Peru, Illinois, in 1920, to be closer to its major customer, the Westclox Clock Company (Figure 4). Radium Dial did not remain long in Peru, but found a more appropriate site in the former township high school building in Ottawa, Illinois. This studio was in operation by 1922 and continued operating at that site until it closed in the middle 1930s. Radium Dial also opened a studio in Streator, Illinois, in 1925, but it closed this studio after only about nine months of operation.

In an interview with an officer of the firm in 1925, Kjaer learned that Radium Dial had employed about 1,000 young women since operations in Illinois started in 1917. Each painter handled about a millicurie of radium each month. The quantity issued at any time varied from 0.18 μCi to 0.98 μCi of ^{226}Ra, depending on the size of the dials to be painted. The company stated that it was practically impossible to keep the workers from tipping their brushes in their mouths. In 1925, Radium Dial was turning out about 4,300 dials each day (Figure 5).

The size of the dial industry in the 1920s can be estimated by the number of firms purchasing luminous compounds from the U.S. Radium Corporation and the Radium Chemical Company. The former claimed that about 120 firms bought luminous paint. Radium Chemical placed the total number at about 100 and stated that its major customers were Radium Dial in

FIGURE 4 Dial painters in a studio in Peru, Illinois, pause in their work. Completed dials are visible beside each painter, and painting materials are ready on the desks.

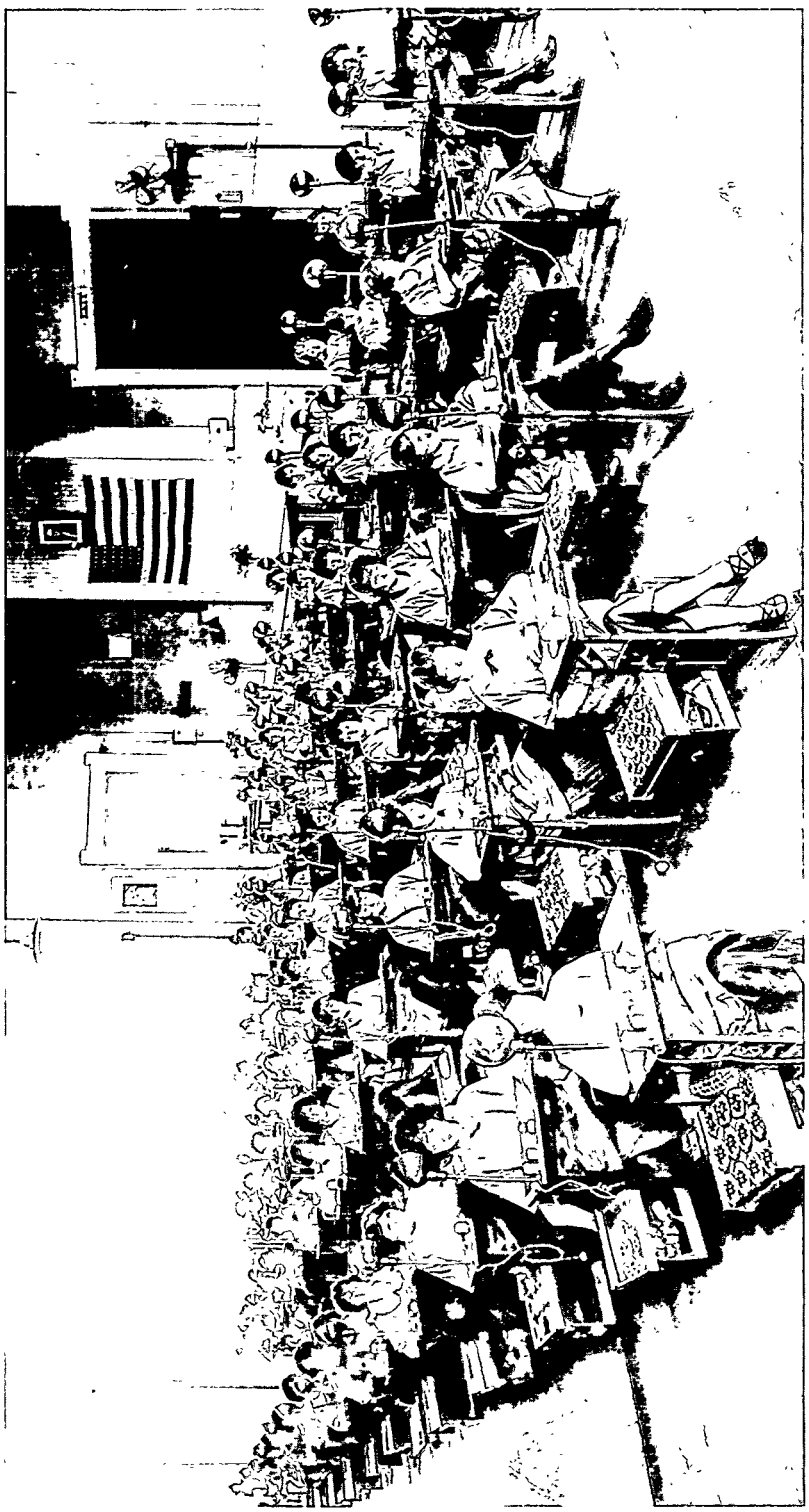

FIGURE 5 A large number of dial painters worked in this studio in Ottawa, Illinois, in 1925. The completed dials next to the painters and the radium-containing paint on their desks suggest that a significant flux of gamma rays must have been present at all times. Thus, each dial painter must have received an external dose from the paint and the completed dials in addition to the internal dose from ingested radium.

Ottawa, Illinois; Elgin Watch in Elgin, Illinois; Waterbury Clock in Waterbury, Connecticut; New Haven Clock in New Haven, Connecticut; Waltham Watch and Clock in Waltham, Massachusetts; and Ansonia Clock in New York City. The U.S. Radium Corporation plant in Orange, New Jersey, and the Radium Dial Company plant in Ottawa, Illinois, were by far the largest application plants. By the end of 1921 an estimated 28 Ci of radium had been used in luminous compounds for watch and clock dials, gun sights, and similar applications.

One aspect of the use of luminous paint containing radium that has seldom been mentioned was its availability to the public. The U.S. Radium Corporation sold a product called Undark, a dial paint for general use. One of the company's advertisements stated the following:

> You Can Apply UNDARK Yourself
>
> For those who desire to experiment with Undark or who wish to illuminate articles in the home, we manufacture the UNDARK Radium Illuminating Set. It contains a small quantity of UNDARK radium luminous material of medium luminosity, a grade adaptable for most application requirements, one vial of practice material (for experimental purposes before UNDARK is used), one vial of adhesive, one vial of thinner, a mixing cup, glass mixing rod and a camel's hair brush.

The price of Undark was $3 per set.

In 1929, over the name of the Commissioner of Labor Statistics of the U.S. Department of Labor, a document titled *Radium Poisoning* was published (Stewart 1929). This document stated that investigators had visited 31 plants ". . . covering all establishments of any importance engaged in dial painting and other establishments engaged in the commercial manufacture of radioactive preparations." A total of 23 radiation-related fatalities were found and another 19 living individuals who were suffering from poisoning due to radioactive materials. Of the 42 cases, 33 were dial painters; of these, 15 were dead and 18 still living. All were stated to have tipped their brushes with their lips. Of the dial painters, 25 were from the U.S. Radium Corporation plant in Orange, New Jersey, as were five deceased individuals who were not dial painters.

Stewart's document (1929) strongly suggested that radium dials were not necessary for the public and that the importing and manufacture of radium dials should be prohibited, but it also outlined guidelines for protecting workers. Stewart did discuss "modern" methods of applying

luminous paint to dials, by which hand painting could be eliminated. He also noted that these new methods required each worker to handle twice the quantity of radium, with an evident tradeoff between reduced ingestion and increased risk from external radiation.

The investigation of the 31 plants revealed only some 250 workers who were directly exposed to radioactive material at the time of the visits. The investigators estimated that during the 16 years of luminous dial painting in the United States, fewer than 2,000 individuals had been involved in the work (Stewart 1929).

A description of the working conditions in the dial painting industry, after the tipping of brushes by mouth had been stopped, is available from a survey made of a number of plants (Bloomfield and Knowles 1933). The dial painters were reported to have used 50-500 μg of radium each day, and their measured external radiation dose was found to be 0.7-46 cGy/yr in the various plants. The average radon level in the plants was 51×10^{-8} Ci per 10 m^3 of air (approximately 10 m^3 of air is inhaled in a working day) or 51 pCi of radon per liter; the dust in the air contained 26×10^{-10} Ci of radium per 10 m^3 or 0.26 pCi of radium per liter. These values are useful for comparison with other potential sources of radon and radium contamination.

The dial workers were not the largest of the radium-exposed populations, but individuals who worked in the dial painting industry have been the easiest to find and study for several reasons. The publicity that arose when the hazards of the industry were realized brought to the attention of the public and the workers the potential hazard of dial painting. Further, all industrial concerns maintain employment records, and these records, when available, have been an important source of names of employees. Also of value was the fact that the employees knew many of their coworkers and were able to identify them when employment lists were not available. Company photographs also proved to be useful in tracing employees, because individuals identified in a given picture often knew the names of others present when the photograph was taken (Figure 6).

For reasons like these, the largest known group of radium-exposed individuals is the dial workers; for epidemiologic purposes the female workers are the most valuable. Most of the workers were female, and many of them were of similar age when they were first hired. Table 1 summarizes the numbers of the workers in the industry who had their radium body contents measured, grouped by the time of first employment. Table 1 also gives the average age at employment and the average initial systemic intake of ^{226}Ra of those hired in each time period.

FIGURE 6 This photograph of a studio in Ottawa, Illinois, shows variations in the placement of the radium-containing paint on the desk of each dial painter. A woman who kept the paint close to her body would have received a larger external dose to the breasts than one who kept her work at a greater distance.

TABLE 1 Employment Statistics for Measured Dial Workers

	Females			Males		
Employment Period	Number Employed	Average Age (years)	Initial Systemic ^{226}Ra Intake (μCi)	Number Employed	Average Age (years)	Initial Systemic ^{226}Ra Intake (μCi)
Before 1915	7	18.0	47.35	2	21.0	3.71
1915-1919	215	19.6	78.68	29	23.6	1.96
1920-1924	331	19.0	73.07	12	30.9	2.26
1925-1929	267	18.5	9.83	4	20.3	1.36
1930-1934	43	20.9	1.20	6	28.2	3.97
1935-1939	54	20.5	1.33	6	26.0	0.18
1940-1944	446	22.4	0.99	14	28.9	1.99
1945-1949	167	22.2	0.76	10	26.2	0.36
1950-1954	106	26.7	0.62	21	27.2	0.36
1955-1959	41	25.8	0.11	23	26.8	0.26
After 1959	70	28.8	0.08	34	28.3	0.13
Total	1,747			161		

The terminology used in this document regarding employees in the dial painting industry should be carefully noted. All of those who are known to have worked in the industry are called "dial workers." Only those who actually applied paint containing radium to the dials or hands of watches and clocks, or those who used the paint on other products, are classified as "dial painters." This difference has been misunderstood in the past, because many have assumed that only dial painters were exposed to radium. Many individuals described as "dial handlers" or "dial remote" or by other codes have been found to contain radium, but at much lower levels than the actual dial painters. For example, the average systemic intake for dial painters was more than 10 times the average intake for those identified as dial handlers or dial remote workers and 100 times the average for those identified as dial blue-collar or dial remote white-collar workers.

Much of the history of the dial painting era can be visualized from Table 1. When the hazards of dial painting became known after 1925, the number of new employees dropped markedly. At the same time the practice of tipping the brush with the lips was stopped. This simple change in work habits greatly reduced the quantity of radium that entered the bodies of the workers. World War II increased the demand for aircraft dials and other items that could be seen in the dark and significantly increased the number of new employees needed in the industry. However, the knowledge previously acquired about the hazards of radium was sufficient to protect the workers from excess intakes, so the employees hired during the war years showed no increase in the amount of radium acquired. Indeed, no radium-induced malignancy has yet been observed in anyone who entered the dial painting industry after 1925.

The number of men in the industry was small, and very few men actually painted dials. Men in the industry were usually involved in other occupations in the dial painting plants, and few had large radium intakes. Thus, it is not surprising that no men in the dial painting industry were ever diagnosed with one of the radium-induced malignancies.

Some employees in the dial painting industry were never identified, and many of those identified never had their radium body contents measured. Many died before the large search for former workers was underway. Table 2 summarizes the numbers of identified but unmeasured employees in the dial industry by their periods of first employment. The total number of identified employees of the dial painting industry was 4,133; of these 88% were female. Only 34% of the identified males were measured, while 48% of the identified females were brought to a laboratory for measurements and examinations.

TABLE 2 Employment Statistics for Identified but Unmeasured Dial Workers

Employment Period	Number of Females	Number of Males	Total Number
Before 1915	11	3	14
1915-1919	297	111	408
1920-1924	221	33	254
1925-1929	177	11	188
1930-1934	30	2	32
1935-1939	31	4	35
1940-1944	761	43	804
1945-1949	181	46	227
1950-1954	108	16	124
1955-1959	27	6	33
After 1959	26	17	43
Date unknown	40	23	63
Total	1,910	315	2,225

Workers who started painting after 1950 are poorly represented in the measured population, because of the time period in which these employees were identified. Most of the postwar employees of the U.S. Radium Corporation in Bloomsburg, Pennsylvania, were identified in the late 1970s, and thus only a few of them were measured before budget restrictions stopped the recruitment of new cases. The files list 1,533 unmeasured employees from this plant.

Early Findings on Health Effects of Radium

The early studies of the effects of internally deposited radium reflect the pioneering efforts of many individuals, but the role of Dr. Harrison S. Martland is preeminent. Martland was the chief medical examiner of Essex County, New Jersey, putting him in an excellent position to investigate the problems at the dial painting plant in Orange, New Jersey. The published record, however, was started by a dentist, Dr. Theodor Blum (1924), who described an unusual mandibular osteomyelitis in a dial painter, a condition he called "radium jaw." Next came the report by Dr. Frederick L. Hoffman (1925), a statistician for the Prudential Insurance Company, whose attention had been drawn to the dial painters by the New Jersey Consumer's League.

Hoffman reported on 5 dead and 12 living individuals who had developed resistant infections of the jaw with buccal lesions and marked anemia.

Before these publications appeared, the U.S. Radium Corporation had engaged Dr. W.B. Castle and Drs. Cecil and Katherine Drinker of Harvard to look into the conditions at the plant, with special reference to zinc poisoning. Their publication was delayed at the request of the U.S. Radium Corporation, because of litigation. When it was finally released, the publication (Castle et al. 1925) suggested that the deleterious health effects noted among the workers were probably due to the radium in the paint.

At this time Martland's name, along with those of his coworkers, first appeared on publications, but Martland had been aware of problems at the Orange, New Jersey, plant for some time. The group's first major publication (Martland et al. 1925) was an impressive document reporting the detection of gamma rays from living dial painters and the exhalation of radon from their lungs. Three cases were detailed in this first publication. One subject had died of pernicious anemia with leukopenia and terminal infection, one was suffering from chronic leukopenia of the pernicious type and had bone necrosis, and the third was still healthy. All tested positive for internal radioactivity. The authors described the autoradiographic detection of radioactivity in bone samples by means of dental X-ray films. Martland et al. (1925), assuming from autopsy specimens that radium was fixed primarily in the reticuloendothelial system (an assumption later found to be not entirely correct), stated the following: "Introduced into the body they [radioactive elements] form fixed deposits in the main organs of the reticuloendothelial system, chiefly the spleen, bones, marrow and liver. Here they continuously emit radiations until they decay according to their individual life history."

In October of 1925, three months before the Martland et al. (1925) paper was published, Martland (1925) read the paper before the New York Pathological Society. The published discussion contains some relevant comments.

Dr. F.B. Flinn asked the following question (Martland 1925): "In checking our work with that of Dr. Martland, we do not find so much in the marrow. We found it chiefly in the calcareous portion, and not in the marrow. Why is it that these girls taking a small amount of radium through the alimentary tract have this condition, and that no cases of necrosis occur by intravenous injection?"

Martland's reply suggested that he was thinking about the jaw necrosis (Martland 1925): "We think that the bone necrosis in these cases has nothing to do with the actual deposition of radioactive element in the bone itself, but it

leaves on the gum, on the roof of the mouth and teeth minute particles that stimulate the normal bacteria of the mouth to increased activity."

Flinn, Professor of Industrial Hygiene at Columbia University, had been engaged by the U.S. Radium Corporation to investigate the problems at its plant in Orange, New Jersey. In his comments after Martland's presentation, Flinn described a measurement he had made to find the quantity of paint ingested by the dial painters. He went to a factory where the painters did not point the brushes with their lips but instead drew the brush through a cloth to bring it to a point. He had painters save all the cloths they used in one week. From these cloths, Flinn determined that 500 mg of paint (of the 10 g used daily) could have been ingested per day if the painters had pointed their brushes with their lips. (No translation into the quantity of radium involved was provided. At Martland's estimate of 1 mg of radium to 30-40 g of zinc sulfide, 500 mg of paint would have contained 13-17 μCi of radium. Kjaer gave an average value of 25 μg of radium to 5 g of paint, or 2.5 μCi of radium per 500 mg of paint.)

Flinn (1926) subsequently published a paper on the Orange, New Jersey, plant, concluding that no industrial hazard existed in the dial painting industry. He did note that five painters from Orange had died of some cause that could not be determined.

A year later, Flinn (1927) described a dial painter from the Waterbury Clock Company in Connecticut who had tripped and broken her femur without falling. This woman suffered from bone necrosis, and measurements showed that her body contained about 100 μCi of radium. She died in 1927, leading Flinn to believe that "radium is partially if not the primary cause of the pathological condition. . . ." Finally, Flinn (1928) concluded that "radioactive material is at the bottom of the trouble even if the mechanism by which it is caused is not altogether clear and not previously suspected."

The last statement did not sit well with Martland, who had continued to publish accounts of death and disease among the dial painters (Martland 1926; Reitter and Martland 1926). In his next publication, Martland (1929) clearly indicated that radium had previously been identified as the cause of the dial painters' problems.

The papers of this period by Martland and his coworkers are truly remarkable. The researchers thoroughly investigated the new phenomenon of radioactivity. They concerned themselves deeply with the symptoms in the individual cases and showed a good deal of compassion toward the victims of the industry. Martland and his colleagues bemoaned the fact that the laws of

New Jersey did not provide compensation for previously unknown forms of occupational diseases.

Although Martland believed that the anemias in the dial painters were due to ingested radium and mesothorium, these anemias had been reported primarily from the U.S. Radium Corporation plant in Orange, New Jersey. Kjaer, in an unpublished memo dated July 21, 1923, suggested that an intense flux of gamma rays in that plant might have been involved. Kjaer wrote:

> One circumstance, which may have been overlooked in the investigation of the attacks of radium necrosis among the employees in the dial-painting factory of the U.S. Radium Corporation in Orange, N.J., is the housing of this factory together with the laboratory. This is something that does not exist in any other dial-painting establishment in the United States, as far as known, and may account for the aplastic pernicious anemia found attending the five deaths.
>
> The proximity of the laboratory might prove to have been an important factor and thorough investigation of this point should be made. The quantity of radium-active element kept in the plant, the possibility of distribution of the radiation through the dial-painting division and precautions taken, not alone at the present time but during the previous years, are subjects that should be thoroughly gone into. . . .
>
> As final refining of mesothorium was performed in the establishment where these attacks took place, there must necessarily have been a large quantity of this element in the plant. The probabilities are that final extraction of radium from American ore was also made in the plant, though there is no data on this subject and the only fact is that the U.S. Radium Corporation controlled the entire output of mesothorium in the United States, furnished by the Welsbach Company of Gloucester, N.J., and Dr. H.N. McCoy from the Lindsay Light Company of Chicago, Illinois.

No notice appears to have been taken of this concept that an intense gamma-ray flux might have been present in the Orange, New Jersey, plant. No remaining data permit estimation of the external dose rate throughout the dial painting area of the plant in Orange.

The next chapter in the history of these dial workers was soon to be written. Martland and Humphries (1929) called attention to the occurrence of

two cases of osteogenic sarcoma among 15 women whose deaths were attributed to radium-mesothorium poisoning. The authors pointed out that this incidence was too high to be coincidental and went on to postulate "that the preexisting deposits of radioactive substances in the bones . . . played an important etiologic role in the subsequent development of the sarcoma." They called attention as well to the existence of four publications implicating external radiation treatments as the cause of sarcomas in humans.

Two years later, Martland (1931) reported that 5 deaths with osteogenic sarcomas had occurred among 18 deaths in workers at the Orange plant. In addition, he described another death with a bone sarcoma in a dial painter who had worked in New York and Connecticut. Martland also reported details on two living individuals with sarcomas and a third he believed would develop a sarcoma. He thus was able to state, "We have . . . in the study of the radium dial painters, proof that radiation can produce malignancy. . . ."

It was possible, from autopsy and exhumation material, to determine with current techniques the radium levels of four of the five sarcoma cases Martland (1931) described. The systemic intake levels ranged from about 243 μCi to over 400 μCi of ^{226}Ra and from 72 μCi to over 2,200 μCi of ^{228}Ra.

Martland (1931) also reviewed the symptoms of radium poisoning. The early cases "were characterized by the presence of jaw necroses and the development of anemias." The jaw necroses were thought to be the consequence of "bacterial infection, usually by way of the teeth, . . ." which resulted in "intractable necrosis of the jaw. . . ." The anemias he believed to be leukopenic, of the regenerative type. Most of these cases occurred within five to ten years after the worker entered the industry.

The later cases showed "crippling bone lesions, developing years after they have left their work as dial painters, and often after several years of good health." These individuals often experienced spontaneous fractures of their bones, and some of them developed bone sarcomas.

Table I in Martland (1931) lists 18 deceased workers from the Orange, New Jersey, plant. Four of these were male chemists, with anemia listed as the cause of death. One individual, who died in 1922, was not seen by Martland. This victim, Case 00-033 in the Argonne files and Case 2 in Martland's (1931) Table I, was exhumed in 1969. Very little radium was found; the body content was only 0.006 μCi of ^{226}Ra, and the calculated systemic intake was only 0.2 μCi of ^{226}Ra and 0.2 μCi of ^{228}Ra. Had this victim been examined at death with the techniques then available, no radium would have been found. This observation suggests that the anemia in this case (and

perhaps in others) might have been due to exposure to external gamma rays in the laboratory, and it lends support to the previously quoted memo by Kjaer.

In a study of 30 radium cases, Aub et al. (1952) summarized their clinical findings as "bone changes of a destructive and reactive nature, spontaneous fractures, loss of teeth, necrosis and osteomyelitis of the maxillae, mandible, or temporal bone, and tumors of bone or surrounding tissues." The authors reported that three of their patients had epidermoid carcinomas of the nasopharynx, a new finding for patients with internally deposited radium. The living patients in this study had been exposed to radium at least 25 years earlier, while the deceased individuals had survived at least 15 years and some more than 25 years after exposure.

Aub et al. (1952) mentioned the long life span of their patients relative to those in the Martland series. Noting that in many patients the first symptom was osteomyelitis of the jaw or loss of many teeth, they speculated that dental hygiene might have been an important factor in determining whether and when jaw disease occurred. Thus, they suggested, in determining the latent period between exposure and symptoms from radium, jaw disease should not be considered.

The Role of the Radium Cases in Radiation Protection

The tensions preceding World War II made preparations for war necessary. Radium-painted dials would clearly be very useful for aircraft and naval vessels, for gun sights, and for other applications. However, the early history of the dial workers indicated that adequate measures would be required to protect employees producing these objects. Standards for radium exposure would have to be adopted, as would procedures to be followed in the workplace.

Standards require measurements, and measurements require instrumentation and appropriate techniques. Thus, the emphasis shifted from the physicians to the physicists. The physicist who was on the scene when he was needed was Robley D. Evans.

In 1933, the year after he obtained his Ph.D. degree at the California Institute of Technology, Evans published his first paper on radium poisoning (Evans 1933), a review of what was then known on the subject. About this time, now at the University of California at Berkeley, Evans made his first measurements of exhaled radon and radium excretion on a former dial painter then living in California.

Evans moved to the Massachusetts Institute of Technology (MIT) in 1934. There, with Dr. Joseph Aub from Boston, Evans continued to work with dial painters, developing the technique that permitted the body content of radium to be determined by summing the exhaled radon fraction and the fraction of radon that decayed within the body. The latter fraction was measured by detecting, with a Geiger counter, the gamma rays emitted from within the body (Evans 1937; Evans and Aub 1937; Aub et al. 1952).

The one-meter-arc method Evans (1937) introduced to measure the radon that decayed within the body required the determination of three unknowns: the gamma-ray attenuation coefficient of the body, the depth of burial of the source within the body, and the response or calibration of the detector. Four measurements were required: the detector response from the body, bent into a one-meter arc, facing first toward and then away from the instrument; then the response with a source placed behind the body; and finally the response from the source alone in the same position. From such an absolute measurement, alternative placements of the patient and the detector yielding higher count rates in more comfortable positions could be calibrated.

With these techniques, dependable body content measurements were made on 27 subjects by 1940. At this time an advisory committee to the National Bureau of Standards (NBS) was charged with the preparation of an NBS (1941) handbook, *Safe Handling of Radioactive Luminous Compound.* Earlier suggestions that levels of 10 μg or 1 μg might be appropriate safety guidelines for the radium content of humans (Rajewsky 1936) had been derived from experience with fewer cases and before accurate methods of measuring the total body burden were available.

The advisory committee, consisting of Martland, Flinn, Failla, Evans, and five others, reviewed the 27 measured cases. The committee found that 7 subjects had carried radium within their bodies for many years and had residual body burdens of 0.5 μCi or less with no symptoms. The other 20 individuals, all with symptoms, had residual body burdens of 1.2-23 μCi of radium. The committee decided on a *tolerance level* of 0.1 μCi of residual body content (NBS 1941; NCRP 1941). This level, later called the *maximum permissible body burden*, was based on effects observed in individuals who had carried radium for 20 or more years, but it was applied on the basis that the body content should never exceed this level. Because the original body contents of the observed individuals were undoubtedly much greater than their residual body contents, a safety factor of one or two orders of magnitude was built into this level. The history of these actions was reviewed by Evans (1980, 1981) and Stannard (1988).

During the war years, Evans continued his work on the dial painters, with emphasis on their safety. Two papers appeared on safety and housekeeping in the dial painting plants (Evans 1943; Morris et al. 1943). In addition, Evans was involved in a study of the effects of radium administered to laboratory animals. Using the rat in an attempt to imitate human radium poisoning, Evans et al. (1944) studied the effects of internally deposited radium. They found that, per unit body weight, 150 times as much radium was required to produce particular chronic symptoms in the rat as in man (or 250 times, expressed as the ratio of radium to calcium in the skeleton). The authors observed that "the tolerance values in animals may be interesting where there is an absolute lack of observations on man, but even a few measurements on man must be regarded as overwhelmingly more important in determining the tolerance dosage for man than the most elaborate experiments on animals." Readers familiar with Evans will recognize this finding as a basis for his often-quoted statement that "the proper subject for the study of man is man" (Evans 1981).

With the conclusion of the war, the interest in the radium cases increased even as the wartime needs for radium dials diminished. The development of atomic energy implied that employees in the nuclear industries might risk incorporation of alpha-emitting radioelements into their bodies. Thus, the potential risks from the low levels of radium in the survivors of the early radium industry were of considerable interest.

3 The Postwar Studies of Radium Cases

The Program at the Massachusetts Institute of Technology

Even before the Atomic Energy Commission (AEC) was created, Evans (1981) was asked by the Office of Naval Research to continue his radium studies. After the formation of the AEC in 1946, the laboratory at MIT received continual support from this source for its radium work. Within a few years, a paper was published that reviewed in detail 30 patients who had carried internally deposited radium for many years (Aub et al. 1952).

Among Evans's contributions to this manuscript was a large section on the physical and chemical properties of radium and mesothorium, describing the decay of mesothorium (^{228}Ra) and its daughter products and showing their contributions to the total activity in a patient as a function of the age of the material. At the time the manuscript was written, the half-life of ^{228}Ra was thought to be 6.7 years instead of the presently accepted value of 5.7 years. With this change and with the generally accepted value of 37% for radon retention after long residence times in the body instead of Evans's value of 55%, this work stands today as the best single analysis of the internal decay of radium daughter products in the human body.

During the postwar period, the number of dial workers brought into the study increased markedly. The discovery of an employment list of the Waterbury Clock Company in Waterbury, Connecticut, and the addition of workers from the New England Watch Company (also in Waterbury, Connecticut) and the Standard Chemical Company in Pittsburgh kept the MIT laboratory busy. The laboratory also switched from the Geiger counter to the newly developed thallium-activated sodium iodide, NaI(Tl), crystal for the detection of gamma rays. This change increased sensitivity and allowed for the first time the detection of ^{228}Ra daughters, specifically the ^{208}Tl gamma ray of 2.62 MeV (if present in sufficient quantity). A shielded whole-body counter, called the "controlled background facility," was built. Ultimately the responsibility for radium cases from the New Jersey Radium Research Project (see below), along with all records, was transferred to the MIT program. When this program at MIT was terminated in 1969, files on nearly 800 measured radium cases were transferred to the Center for Human Radiobiology at Argonne.

A critical and significant study of the relative gastrointestinal absorption of radium and thorium was undertaken at Evans's laboratory at MIT. This study addressed the problem of thorium in dial paint; early dial paint was known to contain not only radium as ^{226}Ra and ^{228}Ra (mesothorium, MsTh),

but sometimes also thorium in the form of ^{228}Th (radiothorium, RdTh). Was thorium, like radium, absorbed through the gut after oral uptake? Indeed, the absorption of radium itself from dial paint was not well known. If thorium was well absorbed, it would deliver a relatively large dose in spite of its short half-life (1.9 years), because elimination of thorium from the body was thought to be very slight after entry into the bloodstream. However, the short half-life precluded measurement of unsupported ^{228}Th or its daughters 20 or more years after acquisition, so no estimate of the dose contributed in a given case by the unsupported component of this isotope could be made from a whole-body measurement.

To resolve this question, Dudley (1960) proposed an experiment with mock dial paint containing tracer doses of short-lived radium and thorium isotopes. He suggested using ^{224}Ra (half-life 3.6 days) and ^{234}Th (half-life 24 days) as the tracer isotopes. The actual experiment, which employed elderly volunteers (aged 63-83 years), was reported in great detail in the *MIT Annual Report* (Maletskos et al. 1966) and was later presented at the symposium on the Delayed Effects of Bone-Seeking Radionuclides, at Sun Valley, Idaho, in September 1967 (Maletskos et al. 1969). The experiment showed that the absorption of radium from dial paint was on the order of 20%, while that of thorium was only 0.02%, lower by a factor of 1,000. Thus, the thorium content of the dial paint could reasonably be ignored, because almost all the subsequent dose was due to the radium isotopes and their decay products. Further, the absorption of radium was now quantified, an achievement that had considerable importance for several other studies.

Evans and Dudley (1960), in testimony before the Joint Committee on Atomic Energy (JCAE), drew attention to the fact that the standard for ^{226}Ra, namely 0.1 μCi retained in the skeleton, was based on direct observations on humans. The standard involved no assumptions about values of relative biological effectiveness, nonuniform distributions, retention, or extrapolation from one species to another. It was, Evans and Dudley concluded, the basic standard for the chronic effects on humans of bone-seeking radionuclides.

Subsequently, Evans (1966) elaborated on this idea, suggesting that this basic standard provided the "pivot point" for the establishment of rational radiation protection guides for ^{90}Sr, ^{239}Pu, and other bone-seeking radionuclides. Because no human experience was available for these radioisotopes, the protection guides would have to be established from animal experiments. Evans continued:

> Here the central requirement is to measure the ratio of the toxicity of, for example, plutonium to the toxicity of radium in an experimental animal. If these toxicity ratios . . . are found to be

reasonably independent of species . . . then the toxicity ratios may be assumed tentatively to also apply to man.

$$\frac{\text{(human } ^{90}\text{Sr toxicity, continuous exposure) [unknown]}}{\text{(human Ra toxicity, brief exposure) [known]}}$$

$$= \frac{\text{(dog } ^{90}\text{Sr toxicity, continuous ingestion) [measured]}}{\text{(dog Ra toxicity, brief exposure) [measured]}}$$

In this way the radiation protection guide pivot point of 0.1 μg of radium, as measured in man, may then be used as the basis for rational protection guides for other radionuclides in man.

Evans continually came back to the ideas of the pivot point, the use of animal toxicity ratios to determine the radiation protection standards for other bone-seeking internal emitters from the radium standard, and the importance of the several thousands of humans who had acquired internal burdens of radium. These ideas are emphasized in an article in *Health Physics* (Evans 1974), in which Evans also estimated that at least 2,000 dial workers were employed during the high-ingestion period before mouth tipping was discontinued in 1926. Further, he suggested that between 500 and several thousand radium chemists were employed in some 23 refineries and radium laboratories. Finally, he said that the list of those potentially exposed to internal radium must include those who received radium for medical reasons, whether by ingestion or injection; their number Evans estimated at several thousand.

In 1967 Evans, who was looking forward to retirement, was concerned about the continuation of the ongoing studies of radium in man, the most active of which were under his direction at MIT. During the symposium on the Delayed Effects of Bone-Seeking Radionuclides at Sun Valley in 1967, Evans suggested the formation of a National Center for Human Radiobiology to carry on the studies of internal emitters in humans (see the discussion in Evans et al. 1969). Toward this end he subsequently drafted a report titled *Comments on a National Center of Human Radiobiology*, which he submitted to the Division of Biology and Medicine of the AEC in December 1967. Included in that report were the following eight relevant points, on which Evans elaborated:

1. Radiation protection guides that are valid for humans are essential for the nuclear era.

2. There is no substitute for man as the relevant experimental species.

3. A unique group of humans exist with relevant radium (^{226}Ra) and mesothorium (^{228}Ra) burdens already carried for 40-50 years.

4. There is no prospect of duplication of this experimental material.

5. Our moral obligation to future generations requires that all possible information be obtained by this generation of investigators, on these humans.

6. These studies are just now blossoming. Quantitative dose versus response relationships are emerging. The findings are relevant to other types of radiation carcinogenesis, such as lung cancer in uranium miners.

7. It is prudent to intensify these studies on at least a national scale, to complete all of the study and control groups, to observe the survivors throughout their full life spans, and to perform detailed laboratory studies on their tissues after death.

8. To carry out this mission, a more nearly immortal organization should be established which might be called the National Center of Human Radiobiology, with a multidisciplinary staff of competent young scientists and with long-range funding.

In January 1968, Evans's proposal was discussed at the meeting of the AEC Advisory Committee for Biology and Medicine (ACBM). The ACBM recommended that an *ad hoc* group be assembled to study the proposal. In February Evans met with the staff of the JCAE and with the General Manager of the AEC concerning the establishment of a center. After this meeting, the chairman of the ACBM appointed a subcommittee of the ACBM, with outside experts, to advise the AEC on the proposal. The members of this subcommittee were E.R. Moseley, Chairman; W.F. Bale; J.C. Bugher; L.K. Bustad; C.L. Comar; L.C. McGee; H.M. Parker; and S. Warren. In May 1968 the subcommittee submitted its report, approving the concept of continuing the human studies and specifying that the program be conducted at Argonne. It also specified that an advisory committee be formed for the new center and that Evans be a member of that committee.

The consequences of Evans's proposal were far reaching and had a positive effect on the study of those exposed to radium.

Argonne Radium Studies, 1945-1960

Argonne National Laboratory was formed on July 1, 1946, from the wartime Metallurgical Laboratory. The Met Lab, as of July 30, 1945, included a Health Division containing three sections: Clinical Medicine, Biological Research, and Medical Industrial Hazards. These sections were subsequently transformed into a Medical Division, a Biology Division, and a Health Physics Division in the new national laboratory. Several changes in division names, functions, and areas of responsibility took place in the late 1940s before these divisions in the early 1950s became a Health Services Division under R.J. Hasterlik, a Biological and Medical Division under A.M. Brues, and a Radiological Physics Division under J.E. Rose.

The first record of any Argonne study of radium in humans appeared in the first quarterly report of the Biology Division, dated August-October 1946 (Table 3). In this document, the Radiochemical Group under W.P. Norris reported on attempts to remove radium from the body of a man who had worked with radium for 35 years. This subject, reported to contain about 2 μCi of radium, was admitted to Billings Hospital on July 4, 1945, under the care of Brues and L. Jacobson. Radon breath measurements were made on two occasions, and the body content was estimated at 2.08 μCi and 1.80 μCi, with the assumption that 45% of the radon formed in the body was exhaled. Although the Radiation Physics Group was eager to measure the external gamma radiation emitted by the patient, clearance could not be obtained for him to enter Site B, a former brewery just south of the University of Chicago campus, where this group was located at that time.

The patient was given ammonium chloride and parathormone and placed on a low-calcium diet, according to the usual "deleading" technique. Urinary excretion of radium was increased by about a factor of five by this regime. A more complete analysis of the data appeared in the quarterly report for August-October 1950.

In the quarterly report for May-August 1947, a cryptic two-sentence paragraph from the Radiation Physics Group of the Biology Division indicated that some additional work on humans might have been underway. The report, authored by V. Clemens and S. Brar, stated the following: "A series of measurements has been made to determine the amount of radon present in the air that is exhaled from the lungs of human subjects. These

TABLE 3 Argonne Periodic Reports Including Information about Radium Studies[a]

Title	Number
Quarterly Report, August to October, 1946, Biology Division	CH-3711
Quarterly Report February 1947 to May 1947, Biology Division	CH-3830
Quarterly Report May 1947 to August 1947, Biology Division	ANL-4078
Division of Biological and Medical Research Quarterly Report, May, June, July, 1950	ANL-4488
Division of Biological and Medical Research Quarterly Report, August, September, October, 1950	ANL-4531
Division of Biological and Medical Research Quarterly Report, February, March, April, 1951	ANL-4625
Monthly Progress Report: Report of the Work of the Biological and Medical Research, Radiological Physics, and Health Services Divisions for the Quarterly Period Ending June 30, 1951	ANL-4658
Division of Biological and Medical Research Quarterly Report, August, September, October, 1951	ANL-4713
Division of Biological and Medical Research Quarterly Report, November, December, 1951, January, 1952	ANL-4745
Monthly Progress Report: Report of the Work of the Biological and Medical Research, Radiological Physics, and Health Services Divisions for the Quarterly Period Ending June 30, 1952	ANL-4849
Monthly Progress Report: Report of the Work of the Biological and Medical Research, Radiological Physics, and Health Services Divisions for the Quarterly Period Ending September 30, 1952	ANL-4902
Division of Biological and Medical Research Quarterly Report, November and December 1952, and January 1953	ANL-4948

TABLE 3 (Cont.)

Title	Number
Monthly Progress Report: Report of the Work of the Biological and Medical Research, Radiological Physics, and Health Services Divisions for the Quarterly Period Ending June 30, 1953	ANL-5086
Monthly Progress Report: Report of the Work of the Biological and Medical Research, Radiological Physics, and Health Services Divisions for the Quarterly Period Ending December 31, 1953	ANL-5200
Report on Biological, Medical, and Biophysics Programs, July 1955	ANL-5456
Radiological Physics Division Semiannual Report, January through June, 1957	ANL-5755
Radiological Physics Division Semiannual Report, July through December, 1957	ANL-5829
Radiological Physics Division Semiannual Report, January through June, 1958	ANL-5919
Radiological Physics Division Semiannual Report, July through December, 1958	ANL-5967
Radiological Physics Division Semiannual Report, January through June, 1959	ANL-6049
Radiological Physics Division Semiannual Report, July through December, 1959	ANL-6104
Radiological Physics Division Semiannual Report, January through June, 1960	ANL-6199
Radiological Physics Division Semiannual Report, July through December, 1960	ANL-6297
Radiological Physics Division Semiannual Report, January through June, 1961	ANL-6398
Radiological Physics Division Semiannual Report, July through December, 1961	ANL-6474
Radiological Physics Division Semiannual Report, January through June, 1962	ANL-6646

TABLE 3 (Cont.)

Title	Number
Radiological Physics Division Summary Report, July 1962 through June 1963	ANL-6769
Radiological Physics Division Annual Report, July 1963 through June 1964	ANL-6938
Radiological Physics Division Annual Report, July 1964 through June 1965	ANL-7060
Radiological Physics Division Annual Report, July 1965 through June 1966	ANL-7220
Radiological Physics Division Annual Report, July 1966 through June 1967	ANL-7360
Radiological Physics Division Annual Report, July 1967 through June 1968	ANL-7489
Radiological Physics Division Annual Report, July 1968-June 1969	ANL-7615
Radiological Physics Division Annual Report, Center for Human Radiobiology, July 1969-June 1970	ANL-7760-II
Radiological Physics Division Annual Report, Center for Human Radiobiology, July 1970-June 1971	ANL-7860-II
Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, July 1971-June 1972	ANL-7960-II
Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, July 1972-June 1973	ANL-8060-II
Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, July 1973-June 1974	ANL-75-3-II
Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, July 1974-June 1975	ANL-75-60-II

TABLE 3 (Cont.)

Title	Number
Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, July 1975-June 1976	ANL-76-88-II
Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, July 1976-June 1977	ANL-77-65-II
Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, July 1977-June 1978	ANL-78-65-II
Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, July 1978-June 1979	ANL-79-65-II
Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, July 1979-June 1980	ANL-80-115-II
Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, July 1980-June 1981	ANL-81-85-II
Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, July 1981-June 1982	ANL-82-65-II
Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, July 1982-June 1983	ANL-83-100-II
Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, July 1983-June 1984	ANL-84-103-II

[a] These reports are available to the U.S. Department of Energy and its contractors through the Office of Scientific and Technical Information, P.O. Box 62, Oak Ridge, Tennessee 37831; prices are available from (615) 576-2413. The reports are available to the public from the U.S. Department of Commerce, Technology Administration, National Technical Information Service, 5825 Port Royal Road, Springfield, Virginia 22161, telephone (703) 487-4650.

measurements have been conducted in collaboration with Dr. J.J. Nickson of the Health Division."

These brief references to the study of radium in humans indicate an interest in the subject at the newly formed Argonne National Laboratory. What was lacking were radium patients to study, and what was needed was someone to find such patients. By 1950 many of the staff who were to play key roles in the radium program were already assembled at Argonne. They were Brues and Norris of the Biological and Medical Research (BIM) Division; R.J. Hasterlik and A.J. Finkel of the Health Division; and J.E. Rose, L.D. Marinelli, C.E. Miller, A.F. Stehney, R.E. Rowland, and H.F. Lucas of the Radiological Physics (RPY) Division.

The three divisions mentioned above were involved in the radium studies during the 1950s. Indeed, their research interests overlapped to the extent that for a time these efforts were combined. In July of 1950 all research efforts were assigned to BIM under Brues and three associate division directors, H. Lisco, L.D. Marinelli, and E.L. Powers. Two service divisions remained, RPY under Rose and Health Services under Hasterlik. This merging of the research staff, however, was hardly noticeable; staff members remained in their original divisions and continued their research, but apparently they were financed as if they were all in the one division.

No record has been located that indicates when this merger was dissolved, but it certainly did not last very long. A clear indication of this was a reorganization of the RPY Division on July 1, 1953. On that date all of the health physics operations, including monitoring and analytical chemistry, were transferred into the Industrial Hygiene and Safety Division, leaving only research activities within the RPY Division.

Even before its move from the University of Chicago location at Site B, the RPY Division had started to measure natural radioactivity in samples from the new Argonne site near Lemont and its environs, to determine baseline levels of environmental radioactivity for evaluation of possible future contamination. In the course of this work, Stehney discovered that water from deep wells in the area contained radium concentrations hundreds of times greater than the concentration in Lake Michigan (Stehney 1955). These were the only instances of high radium levels in water known at that time. Stehney and the members of his group expanded the scope of the measurements, and Lucas eventually mapped a large region of the upper Midwest that had high concentrations of radium in well water (Lucas 1985).

The RPY Division staff also undertook the difficult task of measuring the body radium levels and the daily rates of intake of persons drinking water

with these elevated radium levels. After considerably improving the sensitivity of the radon-in-breath method, they obtained estimates of the amount of radium in a number of subjects who had been drinking water from a known supply for a long period of time (Stehney and Lucas 1956). Included were 30 inmates of a nearby prison who had been incarcerated for periods ranging from a few months to 25 years. Marshall et al. (in ICRP 1973) later deduced an estimate of 0.21 for the fractional absorption of radium from the gut to blood on the basis of the data from these prisoners, very close to the value of 0.20 obtained by Maletskos et al. (1969).

A key event in the radium program was the arrival at Argonne of W.B. Looney, who came in July of 1950 as a postdoctoral AEC fellow in medical science. Looney was assigned, under the direction of Hasterlik, to a project that originated from a suggestion by Marinelli, to study the group of 32 mental patients given radium chloride intravenously at the Elgin State Hospital in 1931.

In his two years at Argonne, Looney was apparently the only staff member who devoted all of his time to the study of the radium cases. He spent several weeks at the Elgin State Hospital, locating the subjects given radium in the 1930s by examining hundreds of patient files. He also contacted physicians who had treated patients with radium and obtained from them the names of some of their patients. Further, he was responsible for the clinical studies on the patients he was able to bring to Argonne.

The clinical studies for each patient at that time included a patient history, a physical examination, complete skeletal X-rays, complete dental X-rays with a dental consultation, a complete blood count, differential white blood cell count, sedimentation rate, a urinalysis, serum calcium, serum phosphorus, serum alkaline phosphatase, and a Kahn test.

An Argonne report dated July 1951 and titled *A Progress Report of Clinical Studies on Twenty-Four Patients* had as its authorship "the Radium Toxicity Group, report compiled and written by William B. Looney, Division of Biological and Medical Research" (Looney 1951). Thus, one year after his arrival, Looney was responsible for the first Argonne document on human radium studies. Many others were to follow.

In September of 1951, Looney, Norris, and Stehney met with Marinelli to discuss the publication of the data that had been gathered in the prior year. They agreed to publish in one journal a series of papers covering all facets of the work. They envisaged the following six papers: (1) an introduction, to be authored by the heads of the divisions; (2) measurements of radon in exhaled breath, by Stehney and colleagues; (3) measurements of the nonemanating

fraction of radium in the body, by Marinelli and his group; (4) radium metabolism, by Norris and colleagues; (5) clinical findings, with Looney as the senior author; and (6) a discussion, authored by the heads of the divisions.

Ultimately, although four of these papers were published, the first and the last never materialized. However, it was 1955 before the series appeared in print. In the meantime Looney had left Argonne and was most eager to see the results of his two years of effort published. He had submitted for publication a paper on autoradiography of radium bone (Looney and Woodruff 1953), but that and his Argonne progress report (or "green back," as they were called) were all he had to show for his efforts.

After leaving Argonne, Looney published what he termed a thorough analysis of the late effects of early medical and industrial uses of radioactive materials. This analysis took the form of three long articles in the *Journal of Bone and Joint Surgery* (Looney 1955, 1956a, 1956b). He also wrote, with his Argonne coworkers, the fifth paper in the list above (Looney et al. 1955). Two additional papers were based entirely or in part on his Argonne studies (Looney 1954, 1956c). Finally, he published an article in *Science* (Looney 1958), suggesting that the maximum permissible level of radium be lowered. This profusion of publications, each with Looney, the former postdoctoral appointee, as the senior author, did not please the senior members of the Argonne radium program.

In retrospect, Looney served as a catalyst that got the Argonne studies of radium underway, and his role has been overlooked and underestimated. For several years after he left, while the examination of the Elgin State Hospital patients continued, the rate at which new radium cases were acquired and studied was very low. As noted above, 24 cases had been studied by July 1951; that number increased to 39 by January 1952, but after Looney left the rate slowed, so that only 43 cases were in the files by January 1953. In his paper published in 1955, coauthored by his Argonne colleagues, 45 cases are described: 6 dial painters, 19 Elgin cases, and 20 subjects given radium therapeutically. What was needed now was a new catalyst, a new finder of radium cases, to revitalize the program.

However, this period was not wasted. To allow detection of the lowest possible radium levels, techniques were developed and tested, equipment was built, and methods were pushed to the limit. Further, several new projects were underway that were to have direct bearing on the understanding of the metabolism of radium.

One of these new projects was the development of a method to concentrate radon from large volumes of expired breath on cooled charcoal

(Stehney and Lucas 1956). This method substantially improved the accuracy of the estimation of retained body burden of radium. Previous measurements of the radon collected in a 1-L flask required an assumed respiratory volume. The new method was first tested by Lucas in 1950. The basal metabolic rate mouthpiece and the helmet originally used were considered unsuitable for the collection of data from large numbers of elderly individuals, and a new system was devised that used well-designed, easily sterilized face masks and pressure-assisted breathing (Lucas 1991).

The radium content of an individual was measured in 1950 by the method developed by Evans, in which a Geiger counter was placed directly behind the seated subject. Marinelli proposed that the Geiger counter be replaced by the recently developed NaI(Tl) crystal. Further, he suggested that the spectrum of gamma-ray energies detected by the crystal be examined to determine the region where the best signal-to-background ratio could be obtained.

Both of these concepts had the potential to increase markedly the sensitivity of the measurements, but neither could be applied without considerable effort. Sodium iodide crystals were not readily available, and those acquired (primarily from the Harshaw Chemical Company, Cleveland, Ohio) were small and often flawed. Further, although these crystals produced light flashes proportional to the gamma energy lost within the crystal, the electronic circuitry required to analyze and count the light pulses was unavailable.

Single-channel pulse height analyzers were designed and built by using the facilities of Argonne's Instrumentation Division, located at that time at Site A in the Argonne Woods Forest Preserve. The RPY Division and the BIM Division were in 1950 still located at Site B. C.E. Miller headed a team consisting of H. May and Rowland, who were moved to Site A to build and test three single-channel analyzers that saw years of use for radium and many other types of measurements.

Subsequently, both Miller and May worked to seal the NaI(Tl) crystals in aluminum cans suitable for analysis. They developed close relations with the Harshaw Chemical Company, which brought the RPY Division larger and better crystals. The growth of crystal size with time allowed two crystals used in 1952, one 1.25 in. in diameter by 0.5 in. thick and the other 1.25 in. in diameter by 2 in. thick, to be replaced in 1956 by a crystal 8 in. in diameter and 4 in. thick.

In 1952 the RPY Division purchased a set of four multichannel analyzers from the Canadian Marconi Company (location unknown). These

analyzers could be used individually as a 6-channel analyzer or ganged to make a single 24-channel analyzer. These were large pieces of equipment; 6 channels with their power supplies were housed in a single 6-ft-tall relay rack. Ganged together to provide 24 channels, they were 8 ft long.

By 1952 both RPY (in 1951) and BIM (in 1952) had moved to their new, permanent quarters at Site D, the current Argonne location (Figure 7). One of the first projects at the new site was to construct a low-background facility in which the gamma rays from contaminated individuals could be measured. A room in Building 202 was dedicated to the purpose, and its walls were covered with 1/8 in. of sheet lead to reduce the background radiation.

Whole-body gamma-ray measurements had previously been made in a lead-lined room at Site B. The new facility was found to have a much lower background, perhaps because the new building was uncontaminated. However, the Site B facility was the only one available when a number of employees from a firm in Cincinnati, Ohio, were brought to Argonne to have their radium levels determined. These employees had been exposed to a release of radium sulfate on July 24, 1951, when a platinum capsule containing 50 mg of radium ruptured (Saenger et al. 1952). They were

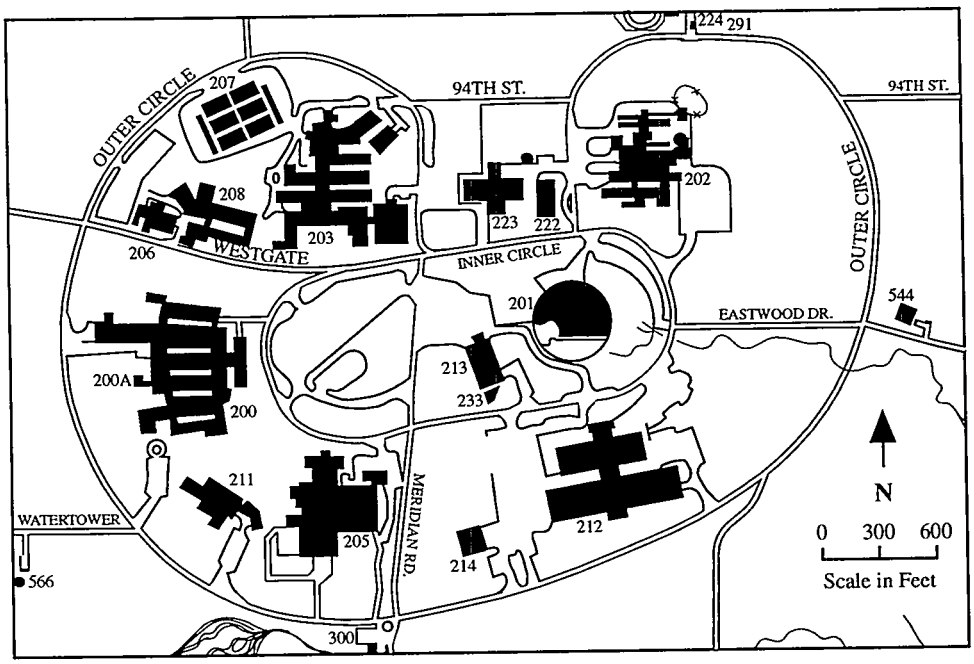

FIGURE 7 Buildings 202 and 203, in the 200 area at Argonne National Laboratory, housed Argonne's radium studies after 1952.

found to have inhaled measurable levels of this compound in particulate form.

These accident cases were of particular interest to Marinelli, who devoted a great deal of time to the measurements and the analysis of the results (Marinelli et al. 1953). He was surprised to find that these subjects exhaled no more than 25% of the radon formed within their lungs, in contrast to the 60-70% exhalation found when radium was deposited within the skeleton. The low exhalation was thought to be due to the low solubility of radium sulfate within the lungs. Some of these individuals were brought back to Argonne several times over the next few years to monitor the disappearance of radium from their lungs.

Two subsequent accidents tested the capabilities of the newly formed multidisciplinary research team in RPY. The first of these, on June 2, 1952, involved an exposure to neutrons and gamma rays at the ZPR-1 reactor at Argonne. Although this accident did not involve radium, it did require evaluation of the dose received by reactor workers. A number of approaches were employed in RPY and in other divisions to evaluate the dose from highly overexposed film badges.

June 2, 1952, was also the date on which a large quantity of fallout from an atomic bomb fell on Argonne. At about 3 a.m. on June 3, when Stehney was determining the decay rate of neutron-induced activities in samples irradiated during the accident, the fallout on the roof of the building caused the counters he was using to show higher and higher backgrounds. Some time was required to determine the cause of the abnormal behavior.

Less than two weeks later (June 13, 1952), a radium source used to calibrate health physics instruments released radium during use in the basement of Building 203. The technicians using the source were exposed, and the contamination subsequently spread throughout the building via the air circulation system. The exposed employees were monitored for several years, until neither their originally low body content nor their radium excretion was measurable.

Subsequently, under the direction of Rose and Miller, the first "iron room" or whole-body counter was assembled in 1954 (Miller et al. 1956). This counter, constructed in the A-Wing basement of Building 203, was built from pre-World War II steel, to assure that the metal did not contain radionuclides from fallout produced by postwar bomb testing. In subsequent years this room was provided with 1/8 in. of lead on the inside walls to further reduce the gamma-ray background. Later, Lucas developed a system to supply radon-free air for this room. However, the room had such a large rate

of exchange with the outside air, because seals were lacking around the door, that the installed system could not maintain a low radon level in the room. This system was also intended to reduce the level of radioactive argon, ^{41}A, produced on site, but the levels of both radon and ^{41}A remained variable.

Later (1957) a second iron room was assembled in the A-Wing basement of Building 203. Ultimately three such rooms were on the site, for a third constructed in Building 202 was operating by 1958. These shielded rooms, NaI(Tl) crystals, and multichannel analyzers allowed measurements of radium body burden at far lower levels than ever before (Figure 8). This capacity improved the accuracy of the results and allowed meaningful measurements to be made on radium subjects with levels too low for earlier evaluation methods.

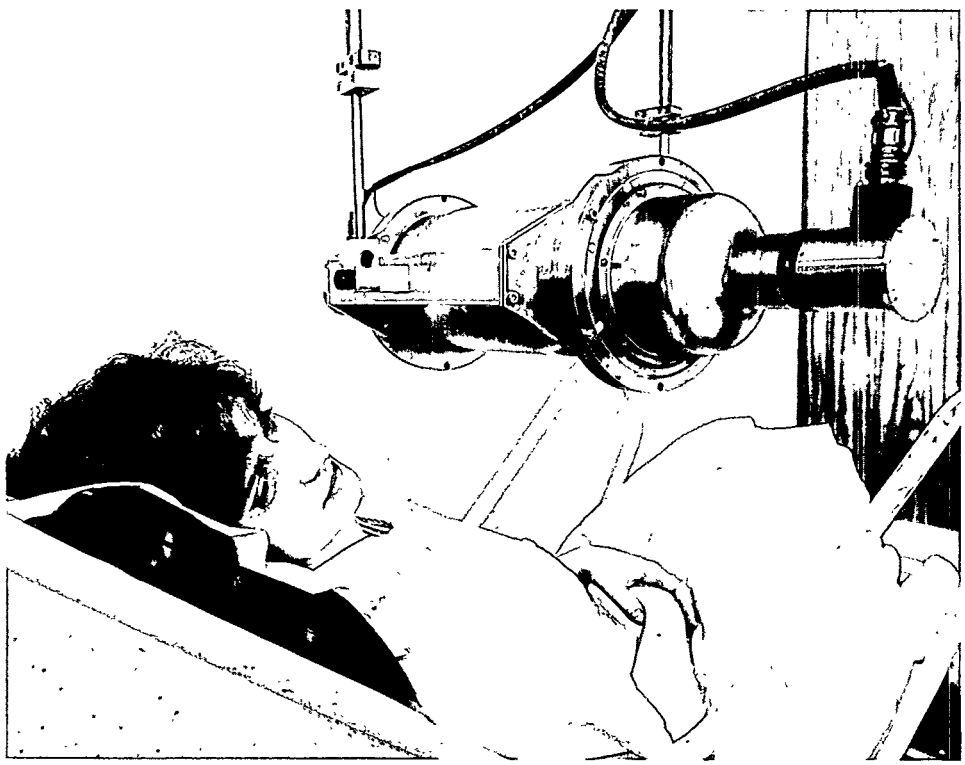

FIGURE 8 Inside the whole-body counter in Argonne's Center for Human Radiobiology, a patient is ready for a measurement of gamma rays emitted from her body. The patient wears clean clothing supplied by the Center and reclines on a metal chair beneath the cylindrical NaI(Tl) crystal that serves as the detector.

While these efforts were underway, an extremely important analysis was undertaken by Norris and his associates. They reviewed the two sets of measurements, made six months apart and reported by Schlundt et al. (1933), on the Elgin State Hospital patients, and two sets made by Argonne personnel 233 and 258 months after the original measurement. They also acquired from various sources sealed radium ampoules that had been obtained in the 1930s from H.M. Armstrong of the Radium Extension Service of Chicago, the source of the radium used at Elgin and by many Chicago area physicians; the radium had been produced by the U.S. Radium Corporation. These ampoules were found in 1953 to contain 10 μCi of radium, within a few percent, and no more than 0.05% of mesothorium.

Norris et al. (1955) found that the retention of radium in the cases under review could best be described by a power function of the form $R_t = 0.54\ t^{-0.52}$, where R_t is the fractional retention at time t (in days) for times greater than one day. By differentiation, the fraction of the injected dose excreted per day is found to be $-dR_t/dt = 0.28\ t^{-1.52}$; dividing this expression by the retention expression yields the coefficient of elimination (the fraction of the total body radium excreted per day), which is equal to $0.52\ t^{-1}$.

Thus, from the Elgin cases, Norris and his colleagues derived a retention function thought to be applicable to any radium case in which the radium was acquired over a period that was short compared to the retention time. The retention function was unique, for it had been customary to think of retention as being described by an exponential function rather than a power function as found here. To be sure, the retention of radium in humans might be described better by a series of exponential terms, but the determination of the coefficients of the various terms would require a large number of very precise measurements of very similar cases. The power function is probably the best fit to such a series of terms, is easy to use, and has been found to describe the observed retention quite adequately.

Thus, the Elgin patients, with known injection levels, played a major role in the early understanding of the metabolism of radium in the human body. Unfortunately, the data are slightly suspect and thus subject to some uncertainty. Schlundt et al. (1933) stated that their patients received no radium in the six months between their first and second measurements. However, whether they received additional radium after the Schlundt measurements is uncertain.

This uncertainty arises because of the previously mentioned Dr. Findley John. In the 1933 paper, Schlundt credited John and a Dr. A.J. Carlson with providing the initiative for the radium injections. This statement fits with the

subsequent finding, in the paper by Barker and Schlundt (1930) reporting the measurement of the radium contents of ten subjects made available by physicians who used radium in their private practices, that six of the subjects had received their radium from John. In addition, a publication (John 1927) describing the responses of a number of his patients to radium administration reported a case in which a patient apparently recovered from a neurological disorder after several 10-μg injections of radium. Thus, it is not surprising that in Looney's interview with John in 1951, John stated that he had been the initiator of the study at Elgin.

The concern about additional radium treatments arose from an interview that took place in 1973. Stehney met with a nurse who had worked for John for a period in 1932, after the reported radium injections at Elgin in 1931. The nurse told Stehney that John often went to the Elgin State Hospital and took glass ampoules of "radioactivity" with him. This report was verified in an interview Stehney had with J.T. Nerancy in 1973. Nerancy, who was one of the coauthors of the Schlundt et al. (1933) paper, stated that John had brought the radium to Elgin for the study. Nerancy thought that there might have been another 50 patients at Elgin to whom physicians had given radium. This last statement was partially verified by the discovery of the records of nine patients in which the administration of radium was recorded, in addition to the 32 patients mentioned in the 1933 paper. Many of the group of nine received their radium in 1933, after the 1931 injections reported by Schlundt. The question of whether any of the original 32 patients received additional radium injections after their reported doses in 1931 has not yet been answered.

The major technical accomplishments of the Argonne radium program up to 1956 were summarized by Marinelli in a semiannual report as follows:

- The natural levels of radium in persons living in the Chicago area, evaluated by Stehney and Lucas, were found to lie in the range 0.4×10^{-10} to 3.7×10^{-10} g, with the highest level in individuals who had consumed drinking water with naturally elevated radium levels. This study led to a mapping of the areas in the Midwest with high radium levels in drinking water.

- Efforts to detect radium in the human body at the lowest possible levels, primarily by reducing the gamma-ray background and eliminating radioactive materials from the neighborhood of the detection equipment, fell short of the self-assigned goal of 5×10^{-11} g of radium by about a factor of ten. These efforts included the construction of the first iron room in the basement of Building 203, with its 8-in. steel

walls, floor, and ceiling. This work to increase the sensitivity of the gamma-ray measurements was primarily under the direction of C.E. Miller.

- Subtraction, from the gamma spectrum of a radium-contaminated individual, of the contribution from ^{40}K as detected by the NaI(Tl) crystal allowed measurement of 5×10^{-9} Ci of RaB,C with an accuracy of about 5%. The amount to be subtracted for each individual was determined after ingestion of a small quantity of the short-lived isotope ^{42}K to evaluate the total body potassium.

- The work revealed that the NaI(Tl) spectrum of unexposed individuals was changing during 1955 and 1956. This change was due to the appearance of an additional photopeak in the spectra, which grew in magnitude during this period and was subsequently found to represent ^{137}Cs from the testing of nuclear weapons.

A study initiated during this period primarily at the urging of Marinelli concerned the metabolism of radium in bone. Hoecker and Roofe (1951) had published alpha-track autoradiographs made from bones from former dial painters. The number of tracks and hence the concentrations of radium varied widely from one microscopic area of bone to another. At about the same time, A. Engstrom in Sweden and R. Amprino in Italy had made microradiographs of thin slices of bone and had shown that the density changes in bone (recorded on the high-resolution X-ray images) clearly detailed the structure of bone on a microscopic scale (Engstrom 1946; Amprino and Engstrom 1952). Marinelli suggested that the two techniques, applied to a single section of bone, might make it possible to understand the metabolism of radium in bone at the histologic level. He asked Rowland to investigate these techniques, which ultimately brought to the study a much clearer understanding of the uptake and retention of radium in bone.

A tentative start in this area was underway by 1952, but real growth took place after John Marshall joined Argonne in 1955 and Jennifer Jowsey arrived in 1956 on the first of two multiyear visits from Oxford University. Two parallel concepts were investigated: the damage done by radium deposited in bone (Rowland et al. 1958, 1959b) and the relationship between the level of a radioactive isotope in blood and the level of the subsequent deposition in bone. The latter investigation required, in addition to the straightforward radiochemical measurement of radioactivity in blood (Lucas et al. 1970), measurement of both the mass of mineral and the level of

radioactivity in a microscopic volume of bone. Quantitative microradiography was developed to address the first requirement (Rowland et al. 1959a), while several different types of quantitative autoradiography addressed the second (Marshall et al. 1959b; Rowland and Marshall 1959).

These studies provided an understanding of the mechanism by which radium and other alkaline earth elements are lost from bone. That the radium body content of an individual decreased with time was well known. The loss was thought to be due to resorption of bone, a process that removed discreet volumes of bone by an active cellular process. However, the results showed that resorption accounted for only a small portion of radium loss from the skeleton; the major loss at early times after uptake was the consequence of an exchange process by which radium was slowly but steadily removed from the entire volume of bone. That is, uptake of radium causes the entire bone volume to be labeled with radium; years later, all the bone that remains is still labeled, but at a lower concentration (Rowland 1961).

The loss of radium from bone by exchange processes was first demonstrated by an experiment in which bone from a dog given weekly injections of equal quantities of radium was examined at two different times. Such an injection schedule provides a uniform diffuse distribution and broad "hot spots" in locations of new bone growth, both of which are areas of radium-labeled bone ideal for quantitative alpha-track analysis. An amputation, shortly after the series of radium injections, provided the first bone sample for an evaluation of the hot-spot and diffuse levels. At sacrifice, a year later, the remaining bones were obtained for comparison. A definite loss of activity was observed from both distributions in regions where no bone resorption had taken place. These early bone studies were summarized, in part, by Marshall et al. (1959a-d) and Rowland et al. (1959a).

In an attempt to verify that the same kind of radium loss took place in humans, bones were examined from some of the Elgin State Hospital patients, each of whom was considered to have received identical radium injections at weekly intervals for various periods of time. This type of administration causes wide bands of newly formed bone to be uniformly labeled with radium, just as in the dog experiment described above. Samples from several deceased individuals had been obtained and examined, but the variation in survival times was too small to verify the concept. What was needed was a bone sample from an individual suffering an early death. An exhumation of a patient who had died early would provide the necessary sample, and such exhumations might in addition provide data on early radium cases for the radium toxicity program.

Thus, the bone metabolism study led to the first Argonne exhumation in a radium case. On October 13, 1959, the body of an Elgin State Hospital patient, Case 03-140, was exhumed. This individual had been injected with radium in 1933 and had died in 1937 at 32 years of age. Autoradiography revealed that both the hot-spot and the diffuse distributions were smoother and more uniform than in fresh bone samples. The conclusion was that the embalming process led to a leaching and redistribution of radium in bone after burial. Thus, although exhumations might be useful for determinations of total body radium, they were not useful for microscopic bone studies. Quantitative autoradiographic studies of radium in bone would have to depend upon well-preserved bone samples. Exhumation, however, proved to be valuable for the study of radium cases, and ultimately more than 100 exhumations were performed.

In this period little progress was made in finding and measuring additional radium cases. Two dial painters and one individual who had received therapeutic radium were remeasured in 1956. Two more measurements were made on dial painters in the first half of 1957. Also in 1957, levels of ^{137}Cs in 11 Central and South Americans were measured for comparison with levels in North Americans. (Levels were lower in the Central and South Americans.) In addition, seven inhabitants of the Marshall Islands were counted to examine their levels of fallout fission products.

Thorium was another radioactive element of interest, particularly to Marinelli. In 1957 the first worker from a nearby thorium refinery was brought in for measurement. He contained about 50 mg of ^{232}Th, indicating that a study could be made of employees from this plant. In addition, a patient injected with Thorotrast in 1939 was studied. Other radionuclides measured during this period were ^{235}U and ^{233}Pa. However, very few new radium cases were measured.

The New Jersey Radium Research Project

The New Jersey Radium Research Project (NJRRP), in the New Jersey State Department of Health, was initiated in November 1957 as a feasibility study and was designated an ongoing epidemiologic study in March 1958. This project was funded by a contract from the Division of Biology and Medicine of the AEC.

For its first three years, the efforts of the NJRRP were directed toward the identification and location of former dial painters and radium workers in New Jersey and the metropolitan areas of New York City and Philadelphia. In subsequent years the project concentrated on the collection of medical, dental,

and laboratory data for individuals willing to participate in the study. The project was terminated in 1967, with the case files being transferred to MIT and ultimately to the Center for Human Radiobiology at Argonne.

Located in West Orange, New Jersey, the project was organized under an administrative group consisting of a project director, a medical director, a radiologist, and a dentist. Lester A. Barrer served as the project director until October 1963. Dr. A.A. Florin then served as acting project director until Dr. Samuel C. Ingraham was appointed project director in July 1964. Ingraham continued in that capacity until the project was terminated.

Dr. Hyman W. Fisher served as the medical director from October 1959 until the end of project. Similarly, Drs. Carye-Belle Henle (from November 1959) and Robert Bonda (from December 1958) served as project radiologist and dentist, respectively, until 1967.

No facilities or personnel were available for many of the scientific studies required; these were contracted out to other organizations. The whole-body counting, radon breath analyses, and some radiochemical analyses were performed for several years at the New York University Medical Center by Professor Merril Eisenbud and his staff. For interproject comparisons, whole-body counting and radon breath analyses on selected subjects were also performed in the Department of Physics at MIT by Evans and his staff. Special excretion studies were undertaken at New York University and in the RPY Division at Argonne.

General pathology studies were conducted by Dr. Hugh Grady, who provided histopathologic interpretation of soft tissues and bone, first at the Seton Hall College of Medicine and Dentistry and later at the New Jersey College of Medicine and Dentistry, here with assistance from Dr. William Sharpe. Oral pathology studies were conducted at Georgetown University School of Dentistry, where Drs. Joseph Bernier and Joseph Belzile provided histopathologic evaluations of oral specimens. Statistical evaluation and consultation on data processing came from Professors Ellis Ott and Thomas Hayton of the Rutgers University Statistics Center.

When the project started, the names of two individuals thought to be living in the area were provided by the U.S. Radium Corporation. A list containing 83 of this corporation's employees, dated 1921, was obtained from files that had been transferred by the corporation to the radium program at Argonne. Using this list provided by Argonne, public health nurses made the initial contacts with the former employees, interviewed them, and solicited their cooperation. From information supplied by these original contacts, the nurses located and gained the cooperation of other former workers.

The first progress report, published three years after the formation of the project (NJRRP 1962a), stated that more than 900 workers from the radium industry had been identified. Former employees of the U.S. Radium Corporation, some 800, constituted the largest group. Some 520 workers were located; of these, 340 were alive, and the remaining 180 were deceased. Approximately 200 of the 340 living cases were currently undergoing comprehensive medical, radiographic, dental, clinical laboratory, and radiologic studies.

By the end of the project, 978 names were in the files. (Of these cases, 23 were already under study by either MIT or Argonne, so the newly discovered cases totaled 955.) Of these, 328 were alive, and 269 were dead; the remaining 381 were not found and existed only as names in the files. Of the 978, 520 had been employed as dial painters, 80 as laboratory workers, and the remaining 378 in other occupations.

A core population, consisting of 161 cooperative cases with relatively complete records, was used by several investigators and consultants in evaluating the findings. An age- and sex-matched control population, consisting of 99 siblings and spouses of the subjects, was set up to furnish comparative information for clinical, laboratory, history, and other studies.

During the life of this project eight progress reports were published (NJRRP 1962a-b, 1963a-c, 1964, 1965, 1966). At the project's termination a two-volume summary of its findings was also published (NJRRP 1967).

Argonne Cancer Research Hospital and the Argonne Radium Studies

A search for dial painters in the area of Ottawa, LaSalle, and Peru, Illinois, was started in 1956 by Miller, on his own initiative. Miller was aided subsequently by Irene Sweet, Rose's secretary. In 1957 Miller stated that he had identified about 300 former dial painters who appeared willing to participate in a study at Argonne. These women were found by interviewing known former painters and asking them to recall their coworkers. Some former workers produced personal Christmas card lists or company photographs that were quite helpful. Miller was remarkably successful in this effort, and much of the credit for the later success of the Argonne program is due to his early efforts to find dial painters and win their cooperation.

The semiannual reports of the RPY Division from this period indicated that radium-exposed individuals were now coming to Argonne for body burden measurements and clinical examinations. At the time of the above

estimate of 300 located cases, 42 cases were reported as measured. In the first half of 1958, 11 dial painters and 2 other radium cases were measured, and a total of 68 cases were said to have been measured and 36 X-rayed. In the second half of 1958, 37 dial painters were reported to be measured, making a total of 92 measured to date in addition to the 6 reported in the first half of 1957. The accounting represents the first suggestion that Miller was separating the cases he had located from those previously identified and measured at Argonne.

In an article titled "Status of the Radium Dial Project" in the RPY semiannual report for the period July-December 1959, Miller described the locations and dates of operation of four "radium arts studios" that operated in Illinois. Miller stated that these plants were owned by the Radium Chemical Company of Pittsburgh, Pennsylvania, but operated under the name of the Radium Dial Company. (It is probable that the radium used was supplied by the former and that both had the same officers, but there is no evidence that Radium Dial was owned by Radium Chemical.) For the plant in Town C, probably Ottawa, 191 names were obtained from city directories. Of these, 87 had been measured as of December 1959. For Town B, probably Peru, 48 names had been obtained and 23 measured. For Town A, probably Chicago, 14 names had been obtained, but no effort had been made to trace individuals. No figures were given for Town D, probably Streator, but some cases from this town had been measured.

The philosophy of Miller's search, stated in this article, resulted in a bias in the radium program while he was responsible for the search for additional cases. He had observed that the individuals who started work after July 1925 had low radium body burdens, usually less than 10^{-8} Ci. Thus, his search was limited to women who painted watches before July 1925. This policy did not change until the Center for Human Radiobiology was formed in 1969 under the direction of Rowland and Stehney. The new policy recognized that all employees in the dial industry were equally important for epidemiologic studies and therefore emphasized that employment, not body content, was the critical factor.

The same semiannual report (July-December 1959) contained the first article by the team of Finkel, Miller, and Hasterlik. They reported having studied approximately 200 persons suspected of having appreciable body contents of radium. This team had correlated radiographic changes in the skeleton attributable to radium deposition with the measured body contents for the 175 subjects for whom X-rays had been made and showed that such changes did increase with radium body content.

Also in this semiannual report Miller reported on another remeasurement of nine Elgin State Hospital patients. These new measurements were compared with those made in 1951 and 1953. Four of these patients were found to have body burdens higher than those reported in 1951, and three of these were higher than those reported in 1953. Miller felt that patient movement during the 1959 measurements could have produced erroneous values for these four subjects, because the average loss in the remaining five was as predicted by the Norris retention function. These results illustrated the difficulties of using these unique cases with known radium insults.

This semiannual report of the RPY Division for the second half of 1959 was the last one for several years to contain reports of new radium cases. Subsequent issues reported radiochemical and bone studies and cooperative studies on radium-related problems with other laboratories, but they contained no mention of studies of new radium cases.

The freeze on the reporting of studies of new radium cases ultimately ended after Miller transferred on January 1, 1964, to Argonne's Health Division, under Finkel, with the subsequent retention of the radium case files in that division. Miller continued to make all of the whole-body radium measurements in Building 203, although later in 1964 the iron room he used was moved from the A-Wing basement to a new location in the C-Wing basement.

Miller refused to report on the search for and measurement of new radium cases in the RPY semiannual reports, because he believed that these studies should be reported only by the Health Division. Nevertheless, radium in humans continued to occupy the attention of other staff members. In 1961 detailed descriptions of autoradiographic evidence of leaching and translocation of radium within bone stored in formalin in museum jars appeared in the semiannual report. Here, for the first time, was mentioned an awareness that the rate of intake of radium, in μCi/wk, could be determined by comparison of hot-spot intensities with those found in the Elgin cases. Since the latter received a known rate of 10 μCi/wk, the rate of intake of a dial painter or other subject could be obtained from the ratio of the hot spots as determined by autoradiography. Lacking, however, was knowledge of the discrimination against radium by the gut. The hot-spot ratio reflected the ratio in the blood of two subjects, but the relation between ingested radium and systemic radium was yet to be determined. As indicated previously in the review of the work at MIT by Evans, this discrimination was measured in a classic experiment in 1967 and found to be about 5:1; that is, only 20% of the ingested radium reached the circulating fluids.

Also in 1961, Miller, who was willing to report on his nonradium-related activities in the RPY reports, described the continuing accumulation of ^{137}Cs in the general population. These results were based on annual measurements of a number of Argonne staff members. In 1961 the levels were falling, having reached a peak in 1959.

A large underground vault proposed in 1961 was subsequently constructed at the west end of B Wing, Building 203. Rose gave Lucas responsibility for the design of this facility. The vault, when completed in 1964, contained an experimental area 30 ft long and 16 ft wide, with a 15-ft ceiling. The concrete roof was 3 ft thick, 5 ft below the surface, with an additional 6 ft of dirt mounded over it. The interior walls were lined with overlapping steel sheets, soldered together to seal out radon. Circulating air within the vault was first cleared of radon, then via an air lock maintained at a pressure slightly greater than atmospheric, to reduce the influx of radon from the outside and thus to keep the background at the lowest possible level. The radon level in the vault is on the order of 0.3 fCi/L. The vault ultimately was equipped with three shielded whole-body counting facilities.

Several personnel changes took place during this period. Stehney had already transferred to the Chemistry Division, and Rowland resigned in 1962 to pursue a Ph.D. degree at the University of Rochester. Rose asked in June 1963 to be relieved of the duties of division director and was replaced by his associate director, Marinelli. While Marinelli was well qualified to be director of research, the role he had previously played in the division, he was not comfortable as an administrator and disliked the role. One of his first moves was to contact Rowland in Rochester and request that on the completion of his degree he return to the division as the RPY associate division director. Rowland returned in the summer of 1964 to assume his new duties. In many ways this was a unique administrative team, because Marinelli turned over much of the routine office work to Rowland, who served as the division's contact with Argonne administration, while Marinelli continued to focus on research efforts.

The annual reports issued by the RPY Division during the 1960s indicated that the division was expanding both in its range of scientific interests and in the number of personnel. The annual report for the period July 1966 through June 1967 was the first to recognize the three major research areas within the division. The report was divided into three sections, titled "Radiological and Health Physics," "Toxicity of Radioelements," and "Meteorological Studies."

In an unexpected move, Marinelli resigned as director of the RPY Division in 1967, and Rowland was appointed to that position in October of

1967. Subsequently, P.F. Gustafson was transferred in from the BIM Division to be associate division director of RPY, bringing with him a fourth research area for the division, called "Bio-Environmental Studies." However, the requirement, following the Calvert Cliffs decision, that Argonne devote considerable effort to the preparation of environmental impact statements resulted in the assignment of Gustafson to this effort in 1972, with his subsequent transfer to head a new division where these activities were centered. The environmental research activities remained, however, within the RPY Division, resulting in a change in the divisional name to the Radiological and Environmental Research (RER) Division. The first annual report of the RER Division covered the period July 1971 through June 1972.

As mentioned previously, Miller transferred to Argonne's Health Division at the end of 1963. This transfer was motivated in part by Miller's concern that Marinelli, who was then his division director, was too interested in Miller's day-to-day results. Miller felt that such interest would result in shared authorship. However, Miller had always taken the time to report on and discuss his results with Marinelli and Rose, perhaps not recognizing that by this practice he was continuing to involve them in his studies. On the other hand, both Hasterlik, at the University of Chicago's Argonne Cancer Research Hospital, and Finkel, at Argonne, were physicians involved in clinical studies on the radium cases, and they naturally described their results in their own reports. Whatever the reasons, the request for the transfer was not opposed, and the Health Division, a service division, suddenly had an ongoing research project in cooperation with the Argonne Cancer Research Hospital.

The Health Division eventually published three Argonne reports and two joint reports with the Argonne Cancer Research Hospital. The first of the Argonne reports (Argonne 1964) covered the period January-June 1964. The following statement appeared in the introduction:

> The human gamma-ray spectroscopy group, consisting of Charles E. Miller and supporting staff, was transferred from the Radiological Physics Division to the Health Division on January 1, 1964. The principal purpose of this transfer was to consolidate the management of the study of long-term effects of radium in man, currently under investigation as a cooperative project of Asher J. Finkel and Charles E. Miller (Health Division) and Robert J. Hasterlik (Argonne Cancer Research Hospital).

This first report also contained the following progress report on the radium studies:

> The current set of studies has been under way since 1957, during which time approximately 250 patients have been examined. Of these 82% are former radium dial painters, 9% were given radium therapeutically by personal physicians, 7% were Elgin State Hospital (Illinois) patients, and 2% were radium chemists.

In this report, Miller also described the extension of his studies of ^{137}Cs in his RPY colleagues. The maximum ^{137}Cs level, observed in 1959, was followed by a decrease to a minimum in 1961, when a marked increase was observed, apparently peaking in 1964 at a level more than twice the 1959 value. This was the last of Miller's publications on ^{137}Cs in man from bomb testing.

The next Health Division report covered the period July 1964 through June 1965 (Argonne 1965). It contained a long article reviewing the measurements made on the Elgin patients between 1931 and 1964 but no update on the ongoing radium studies.

The final Health Division report covered the period July 1965 through June 1968 (Argonne 1968). It contained two articles of interest to the radium program. The first of these was a paper presented at the Sun Valley symposium held in September 1967 (Finkel et al. 1969). The following excerpt from that publication summarizes the work:

> We have acquired the names of approximately 250 individuals who worked at radium dial painting in Illinois before 1930. Of these persons who were exposed to radium occupationally, we have studied 185 by whole-body gamma-ray spectroscopy and by skeletal radiography. We have examined only a few of the 200 or so persons who started work after 1930 since all those that have been measured have small radium burdens. The iatrogenic group of cases resulted from the administration of radium orally or by intravenous or intramuscular injections up to 1933 for treatment of a variety of diseases. . . . One series of at least 41 patients was treated in this way from 1931 to 1933 in a state mental hospital not far from Chicago. We have studied or have other pertinent knowledge of 36 of these cases found by a deliberate search of the records. In addition, we have accumulated another series of 36 cases who received radium from personal physicians. Many of these cases have come to our attention because of pathological

changes that they developed, and they represent a very small sample out of the several thousand persons presumed to have been so treated by their doctors.

The second article of interest in this final report was a transcription of a series of letters written by Arthur L. Miller to Rose in 1959. Miller had worked from 1914 until 1929 for the Standard Chemical Company, where he was involved in the final extraction and purification of radium. He apparently was involved in the preparation of some 45 g of radium. His letters provide a colorful description of the process of radium preparation in the first U.S. plant to successfully mine, extract, and prepare radium for the market.

The first joint report with the Argonne Cancer Research Hospital, dated January 1969 (Argonne 1969a), was a summary in a database format of 454 radium cases. The following statement is from the introduction to that report:

> For the past 20 years a continuing investigation of the long-term effects of radium deposition has been conducted at Argonne National Laboratory and Argonne Cancer Research Hospital. These studies were expanded greatly in 1957 after the discovery of a photograph of 98 dial painters taken in August 1924. The identification and tracing of these and many other persons occupationally exposed to radium, along with those iatrogenically exposed, made possible the collection of a substantial body of data regarding exposure, radium body content, retention patterns, and the medical consequences of such exposure. . . .
>
> The corpus of information condensed and summarized in this report represents an information retrieval dated January 1, 1969. The material summarized here has been developed primarily by the present investigators. Some information gathered on these same patients by other investigators . . . has been included, along with some more recent data from autoradiographic and micro-radiographic analyses of bone specimens . . . and radiochemical analyses of bone and teeth. . . .
>
> The determinations of radium body content were made principally by Charles E. Miller, utilizing the human gamma-ray spectrometer of the Argonne National Laboratory Health Division and the measurement techniques that he developed. . . .

The final joint report, dated April 1969, was titled *Computed Radiological Indices* (Argonne 1969b). It provided some basic data for each of the measured cases in the preceding document and calculations of a number of parameters, including estimated maximum radium burden, total radium absorbed during time of exposure, dose rates to the skeleton, and mean daily dose rate to the skeleton. The introduction stated the following:

> In a previous report we recorded the essential data that had been collected for approximately 300 patients who had or were suspected of having a measurable radium burden. In the present report we present a number of calculated radiobiological indices for those patients of the previous series who acquired their radium burdens by ingestion during occupational exposure. This group consisted of 208 patients who worked in the radium industry, principally as dial painters.

The publication of these documents and the halt in the publication of Health Division annual reports after the middle of 1968 may be assumed to be a consequence of action in Washington to consolidate all of the radium studies into one organization. As indicated previously, Evans had proposed such a unification in 1967 at the Sun Valley symposium, and the concept was under consideration at AEC headquarters. The consolidation was expected to be approved, with Rowland heading the program.

In August 1968, Dr. John Totter, Director of the Division of Biology and Medicine of the AEC, sent a letter to the director of the Argonne National Laboratory inviting the Laboratory to express an interest in carrying on the radium program at Argonne. Dr. Duffield, the Argonne director, responded that a center to carry out the suggested program would be part of the RPY Division. Subsequently, Rowland, director of the RPY Division, sent a more detailed proposal to Totter, outlining the required personnel, budget, and facilities. On November 20, 1968, Totter, in a letter to Duffield, announced that the project would be assigned to Argonne and that Rowland would have the scientific and administrative responsibility for it.

Formal acceptance on the part of Argonne was documented in a letter, Duffield to Totter, dated February 24, 1969. (In this letter Duffield reported that C.E. Miller had left Argonne to join the Loyola Medical School and that Hasterlik had resigned from the University of Chicago. Finkel resigned from Argonne somewhat later.) The Center for Human Radiobiology (CHR) was formed within the RPY Division on September 1, 1969. Thus, Evans's efforts to ensure the continuation of the study of radium in man as a single research program were successful, and, to a larger extent than ever before, this study

was made an ongoing responsibility of the Division of Biology and Medicine of the AEC.

Unfortunately, the transition was not without difficulties, but not with the program at MIT. The newly formed CHR agreed to accept all personnel at MIT who wished to continue working on the radium program, and four staff members from MIT did transfer to Argonne. In addition, two staff members, Miss Shanahan and Professor Evans himself, constituted an Argonne satellite activity at MIT, and later, after their retirements, in Arizona. All of the radium case records were copied and sent to Argonne. Similarly, the case records from the terminated New Jersey Radium Research Project, which had been transferred to MIT, were sent to Argonne. An Argonne office was maintained and staffed in Orange, New Jersey, to maintain contact with the dial painters in that area.

The difficulties occurred at Argonne. Miller, Finkel, and Hasterlik were offered roles in the new CHR, but they refused. Finkel then stated that as physicians they could not turn over their medical records without written approval of each of the living subjects, but he did agree to turn over the records of the deceased individuals.

The Chairman of the Joint Committee on Atomic Energy, Congressman Holifield, wrote to Rowland in 1969, asking for a report on the transfer of patients from the Hasterlik, Finkel, Miller group to the CHR. After a meeting between Rowland and Finkel to determine the status of the transfer, Finkel took it upon himself to answer Holifield directly. In a letter to Holifield dated May 8, 1969, Finkel indicated his displeasure with the assignment of the responsibility of the radium program.

In a periodic report to Dr. Spofford English, General Manager for Research and Development, AEC, Rowland reported on December 11, 1970, that files for all 131 deceased individuals and for 87 of the 287 living subjects in Finkel's possession had been received by the CHR. As a consequence of a meeting between administrators from the University of Chicago and Argonne, to resolve the conflict about patient records, Finkel had agreed to write to each living radium subject for whom he had medical records. He was to tell the subjects about the formation of the CHR and ask if they would allow their records to be transferred to this new organization. What Rowland did not know was that Miller had written to many of the subjects before Finkel's letter was sent, asking them not to allow their records to be transferred.

Fortunately, after the Illinois radium cases had been identified and located, little difficulty was encountered in obtaining the required permission to transfer the files. This was undoubtedly because of the high-quality team

that had been assembled within the CHR to locate and maintain contact with the living radium subjects. As a consequence, most of the records of the surviving cases were transferred to the CHR during the early 1970s, and all were eventually received. Only when some of the women to whom Miller had written brought to the CHR his letters asking them not to cooperate did his attempt to prevent the radium cases from being included in the CHR files come to light.

While these problems were being solved, some very significant scientific work was underway within the program. In 1968 the International Commission on Radiological Protection (ICRP) set up a task group to provide quantitative information on the distribution of radioactive materials in bone. John Marshall of the CHR was asked to head this group, which took its charge to include the alkaline earth elements and addressed calcium, strontium, barium, and radium and considered the known data on both bone and soft-tissue retention of the radioelements of these elements in man. The group's work was first published as a research report (Marshall et al. 1973) and subsequently as a report from the ICRP (1973).

The task group derived a whole-body retention function of the form

$$R = (1 - p)e^{-mt} + p\varepsilon^{b}(t + \varepsilon)^{-b}\,[be^{-r\lambda t} + (1 - b)e^{-\sigma r\lambda t}], \qquad (1)$$

where R is the fraction of the injected activity in the whole body after a single intravenous injection at time t, in days. The values of several of the remaining parameters depend on the radioisotope in question. In the discussion that follows, this retention function for radium is referred to as the *ICRP 20 retention function.*

This combination of an exponential term and a power function has similarities to the simple function of Norris et al. (1955), but it contains important metabolic parameters that the Norris function does not. For example, λ is the rate of apposition and resorption of compact bone, and σ is the ratio of the turnover rates in cancellous and compact bone. Thus, the ICRP 20 retention function accounts for the turnover of various portions of the bone itself within the skeleton in its prediction of retention of an isotope as a function of time. This function has become the standard for any discussion of the retention of the alkaline earth radioelements in bone.

Significant improvements were later made to the ICRP 20 retention function. Schlenker et al. (1982), after an examination of data on the distribution of radium in soft tissue, found that the ICRP 20 function did not describe the actual distribution of radium between soft tissues and bone in the human body. They modified the values of certain parameters in the equation

so that a much better fit was obtained. This modification is here called the *Schlenker retention function.* This modification made essentially no change in the whole-body retention predictions of the function, but it changed the distribution of radium between soft tissues and bone.

Somewhat later Rundo et al. (1985), Keane and Schlenker (1987), and Keane et al. (1988) analyzed the long-term retention of radium after low-level radium exposure and observed that 30-60 years after acquisition, these subjects lost radium more rapidly than predicted by the ICRP 20 retention function. Rowland (1993) examined their data and the predictions of the ICRP 20 function and pointed out a contradiction. This retention function contains the quantity λ, the rate of apposition and resorption in compact bone, which is defined in the ICRP (1973) publication as 2.5% per year. The ICRP (1973) document states that this quantity is a property of bone itself rather than a property of one of the radioelements in bone. Nevertheless, when the parameters were defined for the retention of radium, 1.5% per year was employed for λ. This value was used to fit the retention function to the Elgin cases, who received injections of radium sufficiently large to induce several malignancies.

Rowland (1993) changed the parameters of the Schlenker retention function to retain the distribution between soft tissue and bone and yet incorporate the higher bone turnover rate. This modification resulted in a retention function that closely parallels the ICRP 20 model for the first five years, then predicts a slightly more rapid loss that fits the observed data for radium cases with initial systemic intakes of less than 30 μCi of ^{226}Ra, as calculated previously by the Norris function.

Figure 9 illustrates the retention predictions of the Norris function, the ICRP 20 function, and the modification obtained by changing the value of λ from 1.5% per year to the more appropriate value of 2.5% per year. Figure 9 shows the fractional retention predicted by each of these retention functions from 1 year to 70 years after a single injection of radium, neglecting radioactive decay. The differences are not great, but it must be remembered that the body contents of all the radium subjects were measured many years after exposure. Stehney et al. (1978) stated that the average date of first measurement for the early dial painters was some 40 years after initial exposure to radium. Both the total dose and the initial systemic intake are calculated by first extrapolating back along the retention curve to the body content at day one. In the past, all such extrapolation was by means of the Norris function. The values in this document have been obtained by extrapolating back along a retention curve appropriate to the body content. For the low-level cases, the "new" curve in Figure 9 will give a content at day

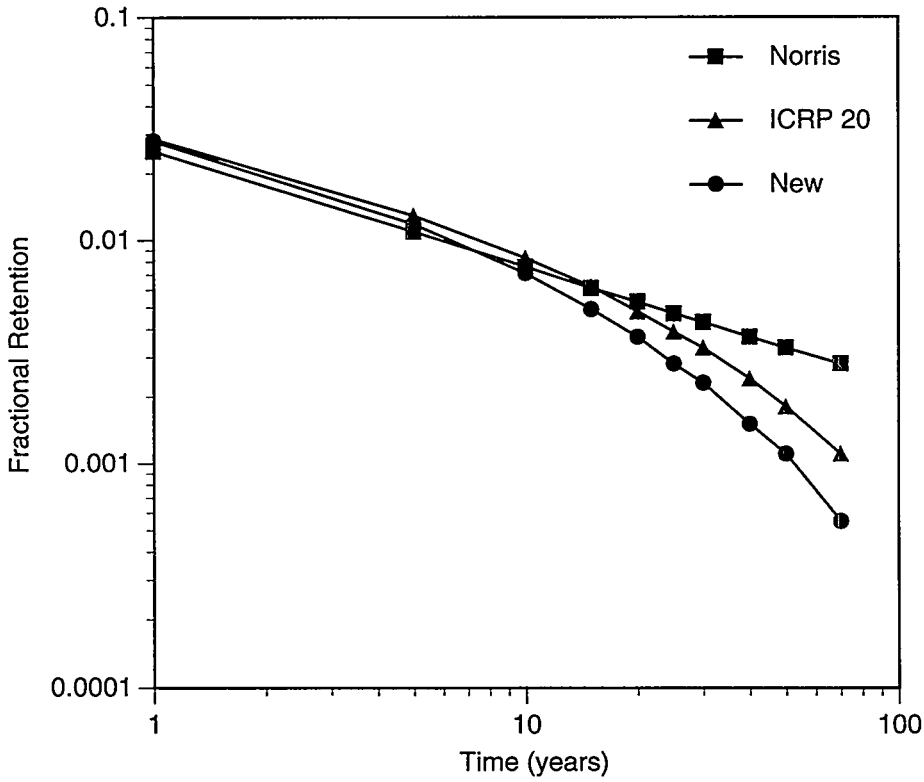

FIGURE 9 Three radium retention functions are plotted for comparison.

one that is higher than a result obtained from the Norris function. Thus, the low-level cases have been found to have higher initial systemic intakes and doses than previously thought.

Lucas and Keane (unpublished data) extended this analysis so that correct intake values could be calculated for cases with large radium intakes, where radiation damage results in slower losses of radium at long times after acquisition. These techniques have produced corrected initial systemic intakes for all of the measured radium cases; these new values have been used in this report. The values are summarized in the appendix, which lists all of the measured radium cases.

The Center for Human Radiobiology

The formation of the CHR in 1969 brought many changes to the RPY Division. So many papers were submitted for the annual report that separate

volumes for each of the four sections within the division were required. The first annual report of the new CHR, for the period July 1969 through June 1970, listed Stehney as section head and incorporated the staff that had previously reported under "Toxicity of Radioelements." Stehney, who originally had joined the division in 1950 and subsequently had transferred to the Chemistry Division, rejoined the RPY Division in September 1969 to head the new section. In 1976 he was appointed an associate director of the RPY Division.

Another new name appearing in this annual report was that of John Rundo. Rundo, formerly a staff member at the United Kingdom's Harwell Laboratory, had also joined the CHR in September 1969 to assume the responsibility for the whole-body counting activities. He prepared a description for this annual report of the construction of three whole-body counting rooms in the underground vault attached to Building 203. The steel for these rooms came from the two iron rooms previously built by RPY and from the one room that had been constructed in Building 202.

An article in this first annual report, which attempted to fit dose-response functions to the data for the measured radium cases, stated that 777 such cases had been obtained by combining the MIT cases with the Argonne and Argonne Cancer Research Hospital cases transferred to the CHR. Of these, 474 case files were obtained from MIT and 316 from the two Argonne sources; three measured cases had files at both MIT and Argonne.

The second annual report from the CHR, for July 1970 through June 1971, showed several more changes. With the arrival of the case records at Argonne, the staff submitted 20 manuscripts related to the radium cases, in comparison to only 3 such papers in the first annual report. The emphasis of the group had shifted markedly, and the enthusiasm with which the staff tackled the new program was obvious. In addition, for the first time data on all of the measured radium cases (now 955) were included in the annual report. This practice was continued as long as such reports were produced, so that a listing of the cases, with a measure of their radiation doses, was available to all interested readers.

In this second report, Stehney introduced a time-invariant measure of the radium dose, which he called the *initial systemic intake*. When the skeletal dose for a living radium case is expressed in centigrays, the value increases each year. Consequently, two subjects who initially acquired identical levels of radium would have vastly different doses if one lived only a short time after radium acquisition, while the other lived out a full life span. The initial systemic intake, the quantity of radium that entered the circulating fluids, circumvents this problem and gives our two hypothetical cases identical

measures of risk as a function of intake. In all listings of the radium cases published by the CHR, both the skeletal dose in rads (centigrays) and the initial systemic intake have been listed. The initial systemic intake may be expressed as activity per unit of skeletal mass, nCi/g, or as the total systemic activity, μCi. In this document the latter format is used.

Another problem that arose for the new CHR concerned space. With increased funding and a charge to take over the study of all U.S. radium cases, acquiring offices for new staff and storage space for the case records created a difficult problem for the small RPY Division. The JCAE came to the aid of the CHR at this time, because it was particularly interested in this study of exposed humans.

Rowland was twice called to testify at JCAE hearings on the adequacy of funding and facilities at Argonne for the program. One of the many concerns had to do with an appropriate area in which exhumed bodies could be studied. Such studies were then housed in a metal building attached to one of the wings of Building 203. Unfortunately, this structure was also the passageway to the relatively new underground counting facility. One end of the building was used for a waiting room for visiting radium subjects who were awaiting their turn to enter the whole-body counter. The only entrance to the temporary morgue was through this waiting room, and a continuing fear was that the door would be opened at an inappropriate time.

Testimony as to the unsatisfactory nature of facilities fell on receptive ears, and funding was authorized in Fiscal Year 1972 to add a new wing to Building 203. This new facility for the CHR housed a complete morgue in the basement, with access for delivery of cadavers through an adjacent, enclosed, out-of-sight area. On the main floor was a specially designed room for record storage, with adjacent office space for employees who worked with these records. Division office space was also included, freeing office and laboratory space for others. This new wing solved the vexing problems of space and also provided facilities actually designed for the type of work underway, resulting in a remarkable increase in staff efficiency.

The CHR was deeply grateful for the efforts of two members of the JCAE who were instrumental in making this facility available. These were Congressman Holifield from California and Congressman Price from Illinois. These legislators appeared truly interested in studies of the effects of radiation on humans, seeing such studies as having immediate applications to the protection of workers in AEC facilities.

The examination of radium patients by the new CHR began in July of 1971. In the next 12 months, 159 patients came to Argonne for examination.

Each patient received a comprehensive physical examination, gamma-ray and radon breath measurements of body radioactivity, a measurement of bone mineral mass for evidence of accelerated bone resorption, and an examination of chromosomes from peripheral blood leukocytes. The case load was increased during this period from two persons per week to six per week. During this year an additional 83 cases were examined at the satellite laboratory at MIT.

The effort to locate and maintain contact with potential new radium cases was assigned to an entirely new group within the CHR, the medical assistants. Headed by B.R. Patten, this group had the responsibility of finding previously unmeasured subjects and encouraging them to come to Argonne for radium measurements. Besides paying all patient expenses, a complete report of each examination, with an analysis of the risk associated with the measured radium content, was sent to the subject's designated private physician, and each individual was advised that Argonne would pay for a visit to that physician so that he or she could transmit the results to the subject. In addition, a participation allowance was provided, and reimbursement for lost pay was available. Further, for those who wished, the expenses of a traveling companion were paid.

Visiting patients were housed in a local motel, where they were met by one of the medical assistants to ensure that they were comfortable. All procedures at Argonne were explained to the patients in advance, and they were always escorted to the various locations where the examinations and measurements were performed. A booklet about the CHR, with photographs and descriptions of the various procedures, was mailed to the participants before they visited Argonne.

The medical assistants maintained contact with each patient through telephone calls and holiday greetings to reflect the Argonne staff's concern for each subject's well-being and to learn about any change in health status or mailing address. Schedules were subsequently developed to determine when each patient would be invited to return to Argonne for additional measurements. The schedule for these reexaminations depended on the subjects' measured body contents; those with the highest radium levels were brought back with the greatest frequency.

Many individuals exposed to radium had to be convinced of the value of a trip to Argonne. They were increasing in age, with the usual infirmities associated with aging, and most had experienced no effects from their radium exposures and thus saw no need to participate in the study. In many such cases the medical assistants performed above and beyond expectations, as they convinced the subjects that learning as much as possible about the effects (or

lack of effects) of radium in humans would benefit both themselves and society as a whole.

To be sure, many individuals never did permit examinations to be made. Some did not want to be reminded of their radium exposure, having read too many gloomy predictions in the newspapers. A small number of these, unfortunately, later developed malignancies, at which time they contacted the CHR for help. It was often possible to arrange treatment for these individuals through the University of Chicago Hospitals.

The medical assistants did much to ensure the success of the CHR in expanding the number of radium cases examined and in maintaining the good will of the subjects. A measure of their success is the growth in the number of measured cases in the files of the CHR. Table 4 illustrates the accomplishments of the CHR as measured by the growth of the measured radium cases from those originally transferred, when the CHR was formed, until 1983, when budgetary restrictions slowed and then stopped the active

TABLE 4 Growth in the Number of Measured Radium Cases

Year	Number of Measured Cases in the CHR Files at the End of the Year	Number of Malignancies in the Measured Cases	
		Sarcomas	Carcinomas
1969	777	51	20
1970	955	51	20
1971	1,032	51	20
1972	1,346	51	20
1973	1,568	54	27
1974	1,740	54	27
1975	1,832	55	27
1976	1,933	56	29
1977	2,072	58	29
1978	2,164	60	29
1979	2,223	60	29
1980	2,259	60	30
1981	2,282	60	31
1982	2,312	61	31
1983	2,400	61	32
1990	2,403	64	32

study of radium cases. Table 4 also includes the number of radium-induced malignancies in the measured cases; the number of new malignancies was not large over this time period, but measured cases with malignancies were added as a result of the exhumation program.

In Table 4 the numbers for 1969 reflect cases in the file after the MIT cases were transferred to the CHR. The numbers through 1983 are taken from the published annual reports, the last of which appeared in 1984. The 1990 figures were obtained from a recent update of the files of the CHR. The total of 2,403 measured cases includes 20 offspring of radium-exposed subjects. Without these, a total of 2,383 persons were directly exposed to radium.

Examination of Table 4 shows that, in the ten-year period from 1970 to 1980, the number of newly measured cases increased by about 100 each year; more than 1,200 previously unmeasured cases were added to the files in this period. In addition, previously measured cases were being remeasured each year, on a schedule based on body content. Thus, the productivity of the CHR during this period was amazingly high, as more new radium cases were added to the files than had previously been found and measured by all other studies since the beginning of the radium era.

More than 4,200 additional identified cases are in the files, mostly unlocated and many deceased. These unmeasured cases deserve special mention. The files of the unmeasured cases range from merely the name of an individual or a death certificate to an extensive file. Many of the latter type are patients with whom the CHR has had continuing contact but who have been unwilling or unable to participate in the study, or who died before they could be measured. Extensive files also exist on some individuals who died long before radium study programs were initiated. Among the group of unmeasured cases, documentation exists to show that 21 died with bone sarcomas and 5 with carcinomas of the paranasal sinuses or mastoid air cells.

However, simply increasing the number of measured cases was not enough. Meaningful epidemiologic studies required knowledge of the number of people exposed to radium by each route. For those exposed as a consequence of medical treatment by various physicians, such studies were impossible, because most records of treatment with radium were lost or perhaps destroyed. Likewise, for those who drank radium water, available over the counter, no records at all existed. The names of patients who received radium as a treatment in a hospital, for example at the Elgin State Hospital, were more likely to be available. Another midwestern institution where radium was given to patients also provided a list to the CHR. However,

as Table 5 suggests, neither of these sources was of great epidemiologic value, and both suffered from a common fault. Although the quantity of radium injected was usually recorded, evidence suggests that some individuals may have received further, unrecorded radium treatments. Thus, in these cases the recorded treatments must be taken as lower limits of the actual radium injection levels.

Three significant differences distinguish the patients at the two institutions in Table 5. First, the Elgin patients were, on the average, at least ten years younger than the clinic patients. Second, the Elgin patients received,

TABLE 5 Comparison of Patients Treated at Two Midwestern Institutions

Point of Comparison	Elgin	Clinic
Dates of treatment	1931-1933	1922-1928
Number of radium-treated patients	41	116
Males:females	17:24	74:42
Age at treatment, years (± s.d.)	33.5 ± 14.0	46.6 ± 15.0
Age range, years	16-72	15-81
Number of patients with known ages	41	114
Average radium dose, μCi (± s.d.)	189.3 ± 99.9	43.0 ± 31.6
Dose range, μCi	70-450	10-210+
Number of patients with known doses	30	108
Average survival after radium, years (± s.d.)	30.6 ± 14.3	15.6 ± 16.3
Survival range, years	4-52.6	0.02-57.8
Number of patients still living (1990)	1	0
Number of deaths verified by death certificate	28	69
Number of patients with measured body burdens	31	9
Number measured while alive	30	4
Number measured after exhumation	1	5
Number of radium-induced malignancies		
Bone sarcomas	3	0
Head carcinomas	5	0

on the average, more than four times as much radium as the clinic patients. Third, the Elgin patients, in spite of their higher radium doses, survived more than twice as long as the clinic patients.

The Elgin patients' treatment was described in the literature in 1933 (Schlundt et al. 1933). Most were still institutionalized in the 1950s and thus were easily located when the radium studies were initiated at Argonne. In contrast, eight clinic patients were treated with radium water (doses not recorded) in 1922, and the remainder received intravenous injections in 1925-1928. An account of some of the latter treatments, for hypertension and pain, was published by Allen et al. (1927), but this publication was unknown to CHR personnel until the 1970s. The patient records were not obtained until 1972 or later, when Professor Evans, then retired and working for the CHR, received approval to examine the clinic's files. By this time most of the patients were deceased, and those still living were very difficult to locate.

The Elgin patients received radium injections that we now know were within a range making the probability of radium-induced malignancy high, but the injections were not large enough to produce any somatic effects. These patients received radium in an attempt to alleviate a mental problem, not a physical disease. The reduced survival of the clinic patients probably had nothing to do with their radium exposures, because they were already sick at the time of their treatments. Fourteen of them survived less than a year after their treatment at the clinic.

Industrial experience was more likely to yield useful lists of those potentially exposed. The MIT group obtained an excellent list of workers at the Waterbury Clock Company, in Waterbury, Connecticut. C.E. Miller had obtained photographs of employees of the Radium Dial Company plant in Ottawa, Illinois, from which identities of almost all workers in the pictures were established. The medical assistants searched through the city directories of the Illinois cities in which dial painting plants were located to find the names of employees. From such sources some lists were developed, and it is of interest to see how many of the employees on such lists have been studied. Table 6 shows, for four of the largest dial painting plants, the number of female dial workers identified from employment lists, city directories, or photographs and the fraction of the workers studied.

A number of employees from the Luminous Processes plant are apparently still alive and unmeasured; they could be added to the totals in Table 6, but only a few still survive from the other listed plants. More than 10% of the total number of workers in the Luminous Processes plant started

TABLE 6 Female Dial Workers Named on Lists

Dial Company	Number on Lists	Number Studied	Percent Studied	Number of Unmeasured Workers Known to Be Dead by 1990
Radium Dial Company, Illinois plants	219	145	66.2	60
Waterbury Clock, Waterbury, Connecticut	404	254	62.9	91
U.S. Radium Corporation, Orange, New Jersey	73	33	45.2	30
Luminous Processes, Ottawa, Illinois	608	434	71.4	68

work in the 1950s and 1960s and are therefore younger than workers in the other three plants, where all employees started work long before 1950.

Many studied subjects were examined only after death. The exhumation program carried out by the CHR made it possible to measure the skeletal radium contents of some individuals who died before the radium studies were initiated. Many of these subjects were exhumed because of the possibility that they died with a radium-induced malignancy. Such cases were studied whenever possible to determine radium contents. More than 100 exhumations were performed. In addition, about two dozen willed bodies were received; these have provided unique opportunities for detailed pathologic and radiologic studies.

Review, *Ad Hoc*, and Advisory Committees

As indicated previously, when the subcommittee of the AEC Advisory Committee for Biology and Medicine recommended that the radium studies program be assigned to Argonne, it also recommended that an advisory committee to the CHR be appointed and that Evans be a member. That committee held its first meeting at Argonne on November 16-17, 1972. The

members were Evans, Chairman; L.H. Hempelmann; G.B. Hutchison; C.R. Richmond; and A.C. Upton.

The committee was informed at its first meeting that the RPY Division already had a scientific review committee that evaluated the scientific work in terms of quality and relevance. Thus, the CHR Advisory Committee could best serve the CHR by giving guidance on policies and advice on specific problems. The committee should also concern itself with matters that it considered should be brought to the attention of the CHR.

For the first meeting, the CHR identified in a letter to the committee chairman four areas on which guidance was sought:

- A policy on skeletal X-rays was requested, to determine whether skeletal surveys were justified for all patients.
- Help was sought in identifying diagnostic procedures likely to be of special value in screening for radiation damage.
- The committee was asked to consider whether the CHR should be funded to provide therapy for subjects with apparent radiation damage.
- Aid in identifying feasible goals was solicited.

Subsequently, Evans, in his role as chairman, sent a letter to the members suggesting that they address the following topics during their meeting:

- Clinical aspects and diagnostic procedures
- Feasible epidemiologic goals
- Nonepidemiologic goals
- Long-range (30-year) objectives
- Selective exhumations
- Selective ($\geq$ 0.1 μCi of radium) or random sampling of World War II dial painters
- Sampling of people currently employed in radium dial painting

- Human experimentation on inhalation of mock dial paint dust
- Policy on skeletal X-rays
- Policy on radionuclides other than radium and thorium
- Plans for reports and for subsequent meetings

The report of the CHR Advisory Committee addressed each of these points. The major conclusions were that complete skeletal X-rays should be obtained on all patients; a rigorous exhumation program should be undertaken; no therapy should be provided; and feasible, defensible epidemiologic goals should be established. The goals, however, were not defined.

A second meeting of this committee was held at Argonne on October 4-5, 1973. Present was a representative of the Division of Biomedical and Environmental Research of the AEC (DBER/AEC), Dr. Sidney Marks. The agenda for this meeting was limited to three topics, submitted to Evans by Rowland in preparation for the meeting. These topics were the following:

- Skeletal X-ray examination of all subjects
- The goals and intensity of the exhumation program
- The epidemiologic design for the study of several subgroups

The first item above appeared again, Rowland explained, because he was worried about possible criticisms of the CHR's policy of obtaining skeletal X-rays on all patients in view of current concerns about unnecessary X-rays, particularly if the criteria of risk for X-ray exposures changed at a later date. (After-the-fact judgments have not yet surfaced about this policy, but fortunately the CHR staff had ignored the first recommendation on human experimentation on inhalation of mock dial paint dust. The study of mock dial paint at MIT [Maletskos et al. 1969] was later selected as an example of "the use of human subjects as guinea pigs for radiation experiments" by a subcommittee of the U.S. House of Representatives. This experiment, which "did not benefit them as individuals" and "exceeded presently recognized limits for occupational exposure" was included in the subcommittee's published report titled *American Nuclear Guinea Pigs* [U.S. House of Representatives 1986]).

The CHR Advisory Committee concluded that complete skeletal X-rays were justified by the additional protection that early diagnosis provided for

the patient. The exhumation program was to concentrate on three types of cases: those that might provide information on the toxicity ratio of mesothorium to radium, those that might clarify the retention of radium at early times after acquisition, and those given plutonium in the 1940s. Also important were radium cases from Waterbury and Ottawa, because such cases would increase the number of subjects known as a consequence of their employment. No recommendations were made about epidemiologic goals, although Stehney presented tables summarizing the current population of measured radium cases and progress that had been made in the follow-up of specific subgroups of this population.

This was the last meeting of the CHR Advisory Committee. Its next scheduled meeting was postponed until the question of an epidemiologic plan was settled. This issue was not settled until early in 1976. No records are available to suggest why further meetings of the CHR Advisory Committee were not held.

Shortly after this CHR Advisory Committee meeting, and before its recommendations were received, a DBER/AEC site review visit was made to the CHR. The site review team criticized the design of the CHR studies of human populations and requested that an *ad hoc* committee of epidemiologists be formed to establish rigorously formulated objectives and biometric methods for the continuation of the program. The CHR was directed to prepare a plan that would meet these objectives, for submission to the *ad hoc* committee.

A plan was put together by Stehney, largely incorporating ideas suggested by Marshall that were directed toward the statistical problems involved in differentiating between squared and linear dose-response functions. The resulting 160-page document covered the origins of the radium program, examined the core populations under study, introduced plans for both a prospective and a retrospective study, discussed the statistical weaknesses of each type of study, reviewed the medical procedures and the data collection and storage systems, and concluded with the current list of measured radium cases. This plan was completed in May 1974 and was submitted to the *ad hoc* committee of epidemiologists in July 1974.

The *ad hoc* committee consisted of Hutchison (a member of the CHR Advisory Committee), Marks (DBER/AEC), S. Jablon, P. Meier, R. Miller, and E. Tomkins. The report of this committee, received in June 1975, found the plan as submitted to be unsatisfactory and requested that "a general protocol of the radium project should be drawn up . . . with broad objectives and a broad outline of the study plan. Specific protocols should be prepared for limited projects within the total study."

This request was honored by the submission of a design for epidemiologic studies, dated October 1975. This document was prepared by A.P. Polednak, who had joined the CHR in May 1975 as an epidemiologist. In it he described two specific cohort studies of dial painters, outlined a study of birth characteristics of offspring of female radium dial workers, and included a design for an epidemiologic study of former thorium workers. This document was subsequently approved in 1976.

The Study of Plutonium and Thorium Cases

Durbin (1972), in a review of patients of presumed short life expectancy given plutonium in 1945-1946 (Langham et al. 1950), deduced that some of these patients might still be alive. They had been injected to determine a relationship between the quantity of plutonium in the body and its subsequent elimination. The relationship determined was to be used to estimate the body content of accidentally exposed plutonium workers. The patients received injections of ^{238}Pu (2 cases) and ^{239}Pu (16 cases) containing 0.1-5.9 μCi. With Durbin's help and encouragement, the CHR found 4 of the original 18 patients to be still living in 1975. Three of these patients were hospitalized in a metabolic ward, and all urine and feces were collected for at least eight days. Blood samples from two of the patients were analyzed for plutonium.

In these efforts the CHR was not at first encouraged by the staff of the DBER/AEC, who had no objections to the analysis of the data from the published report of Langham but did object to direct contact with the still-living subjects. Rowland argued that the value of human experience with plutonium more than justified the difficulties that might be experienced in obtaining the information. Ultimately he obtained approval to attempt to exhume deceased subjects and to locate living individuals, as long as the CHR did not indicate to the subjects or their families that the study dealt with individuals containing plutonium or identify the university hospitals in which the radioactive materials were administered. Rowland agreed to these restrictions but did not get them in writing.

These restrictions later embarrassed the CHR, because the Division of Inspection of the AEC located an internal CHR memo that cautioned the staff not to use the term plutonium in regard to these cases. Quite correctly, since the CHR was operating under the restrictions placed upon it by DBER/AEC, the inspectors pointed out that the CHR did not have valid informed consent from these patients for its actions. The DBER/AEC was then required to contact each of the patients and inform them that they had been injected with plutonium in the period 1945-1946. Subsequently, this failure of the CHR to

obtain informed consent was included in the report titled *American Nuclear Guinea Pigs* (U.S. House of Representatives 1986).

The study of the retention and distribution of plutonium in human subjects attracted a good deal of attention, and it still provides the only long-term data on the subject at these relatively high levels. The CHR staff published several papers on these measurements, including the following: Larsen et al. (1976), Rowland and Durbin (1976), Rundo et al. (1976), Rundo and Holtzman (1976), and Schlenker et al. (1976). Examination of this small group of patients gave no indication that they had experienced any detrimental effects from the plutonium injections.

The study of thorium workers, mentioned in Polednak's revision of the epidemiologic plan for the CHR, was of particular interest to the U.S. Nuclear Regulatory Commission, which subsequently aided in funding the study. The Lindsay Chemical Company, later known as the American Potash Chemical Company and finally as a division of Kerr-McGee Corporation, was formed in Chicago in 1902. It originally used thorium nitrate in the manufacture of incandescent mantles. The company moved to West Chicago in the late 1930s, where it remained until it closed in 1973. By 1952 mantle production was limited, and the production of rare earths and thorium chemicals constituted the major activities.

Copies of all employment records were obtained, covering the period from the mid 1920s until 1973. A potential study group of 4,478 workers at the West Chicago plant contained 3,538 males and 940 females. Exposure data were provided by an unpublished industrial hygiene survey made in 1952 (Kelvin and Fresco 1953) and by AEC inspections in 1956-1973. Radiation levels reported in the 1952 survey were 0.5-5 mR/hr at locations where thorium chemicals were processed or stored. Airborne thorium levels for workers directly involved in the thorium extraction process were somewhat higher than the present limits.

Three published reports examined mortality rates for a cohort of 3,039 men (Polednak et al. 1978; Rundo et al. 1979; Stehney et al. 1980). An excess of deaths had occurred (511 versus 486.8 expected), but only two causes were statistically significant at the 95% level: diseases of the circulatory system, with 205 deaths versus 249.5 expected (significantly less than expected), and motor vehicle accidents, with 38 deaths versus 23.2 expected. The authors concluded, after further examining mortality results based on year of first employment, duration of employment, and job classification, that the results were not consistent with a strong relationship between employment at this plant and subsequent mortality from respiratory diseases (as might have been expected from the inhalation of airborne

radioactivity). The most complete description of the entire thorium study is found in Stehney et al. (1980).

In a second follow-up, Liu et al. (1992) reported on 3,796 men and women from the plant. Increased mortality ratios for males were reported for all causes, for all malignant neoplasms, for lung cancer, and for external causes. Female workers, in contrast, had death rates below those of the general U.S. population.

4 The Hazards of Internally Deposited Radium

The information in the following sections on the health status and mortality of radium-exposed individuals was derived from three sources. First, the cause of death for each deceased radium subject was coded according to the eighth revision of the *International Classification of Diseases, Adapted* (U.S. Public Health Service 1965). A death certificate has been obtained for almost every radium subject known to be deceased. Each certificate has been submitted to the National Center for Health Statistics, whose nosologists have provided the appropriate International Classification of Diseases (ICD) code number. Every subsequent reference to a coded cause of death refers to the cause designated by the ICD code number.

The second source of information is known as the SNOP (Systematized Nomenclature of Pathology [College of American Pathologists 1965]) file. The SNOP file is a very large database containing coded medical information. The coding process was described by Littman et al. (1973). Briefly, information from primary sources (autopsy, surgical reports, physical examination, death certificates) and secondary sources (physician letters, journal reports, family histories) is included. The source for each item of information is coded, as is the information itself. Each case may have any number of entries, depending on the number of sources of information available. Some cases do not appear in the SNOP file, while others have 1-100 or more entries. The file has entries for almost 5,700 radium, thorium, and other cases. Each medical event recorded is listed in a single record; more than 200,000 records are in this file. In spite of its size, this database is not suitable for epidemiologic surveillance of the population, because the uniformity of data gathering is unsatisfactory. However, the SNOP file does contain a wealth of information, and it may show trends that are suggestive even if they cannot be rigorously quantified.

The third source of information applies only to subjects with the known radium-induced malignancies (bone sarcomas and carcinomas arising in the paranasal sinuses and the mastoid air cells). These malignancies have been identified primarily by pathologic analysis of tissues or, in a few cases, by gross radiologic evidence from the primary cancer site. The subjects known to have these malignancies are identified specifically by a date of first diagnosis; they are included in the appropriate malignancy listing regardless of the coded cause of death.

Some of the several causes of death summarized here have been positively associated with internal radium by statistical analysis; these include bone sarcomas and head carcinomas. Other causes have yet to be proven to be associated with radium, but they have appeared in excess of expected

numbers; these causes include multiple myeloma and breast cancer. Leukemias have been expected by many to be elevated in this population, but they are not. Life shortening is not a cause of death, but it is included because the lack of life shortening came as a surprise to many.

Bone Sarcomas

A total of 85 subjects with diagnosed bone sarcomas are known or suspected to have had radium deposited within their bodies in the form of ^{226}Ra or ^{228}Ra or both. Five individuals with a diagnosed bone sarcoma also had a diagnosed head carcinoma. These 85 subjects represent about 1.3% of the total exposed population. For the smaller population with measured radium burdens, 64 of a total of 2,383, or 2.7%, developed bone sarcomas. As will be discussed in a later section, the incidence of bone sarcoma increases rapidly with increasing radium intake.

As a producer of bone sarcomas, ^{228}Ra has been found to be more effective than ^{226}Ra. Rowland et al. (1978) found an effectiveness ratio (^{228}Ra to ^{226}Ra) of 1.5 when average skeletal doses were used and 2.5 when initial systemic intake was used as the measure of risk. When the measured radium cases are arranged in order of increasing initial systemic intake, expressed as μCi of ^{226}Ra plus 2.5 times μCi of ^{228}Ra, the lowest-dose case with a bone sarcoma is number 2,102 on the list of 2,383 cases. That is, no bone sarcomas occurred in the 2,101 cases with lower combined intakes than this case. The value of the combined systemic intake for this case was 100 μCi, from 81.53 μCi of ^{226}Ra and 7.42 μCi of ^{228}Ra. This lowest-dose sarcoma case was a female dial painter who started work in 1918 and died in 1983 of a bone sarcoma diagnosed in 1981. The distribution of the bone sarcoma cases by sex is summarized in Table 7. Table 8 compares the distribution of bone sarcomas for dial workers and all other radium subjects.

An examination of the appearance of the bone sarcomas with the passage of time after first exposure to radium is of interest. The first bone sarcoma was observed 5 years after first exposure to radium. The bone sarcomas seemed to appear quickly after a 5-year latent period. After about 50 years the frequency fell markedly. However, the diagnosis of a bone sarcoma 63 years after first exposure to radium is evidence that this malignancy can appear at any time in life, given a sufficiently large initial systemic intake of radium. The subject with this latest appearance time had a combined intake of 100 μCi, the lowest intake associated with this malignancy. Figure 10 shows the number of bone sarcomas diagnosed in each 5-year period after exposure.

TABLE 7 Bone Sarcomas by Sex

Case	Number of Persons		Number of Bone Sarcomas	
	Female	Male	In Females	In Males
Measured cases	1,903	480	61	3
Unmeasured cases	2,781	1,511	19	2
Total	4,684	1,991	80	5

TABLE 8 Distribution of Bone Sarcomas between Dial Workers and Other Radium-Exposed Subjects

Case	Number of Persons[a]		Number of Bone Sarcomas[b]	
	Female	Male	In Females	In Males
Measured cases				
Dial workers	1,747	161	46	0
Other cases	156	319	15	3
Total	1,903	480	61	3
Unmeasured cases				
Dial workers	1,910	315	18	0
Other cases	871	1,196	1	2
Total	2,781	1,511	19	2
Sum	4,684	1,991	80	5

[a] Total persons = 6,675.

[b] Total bone sarcomas = 85.

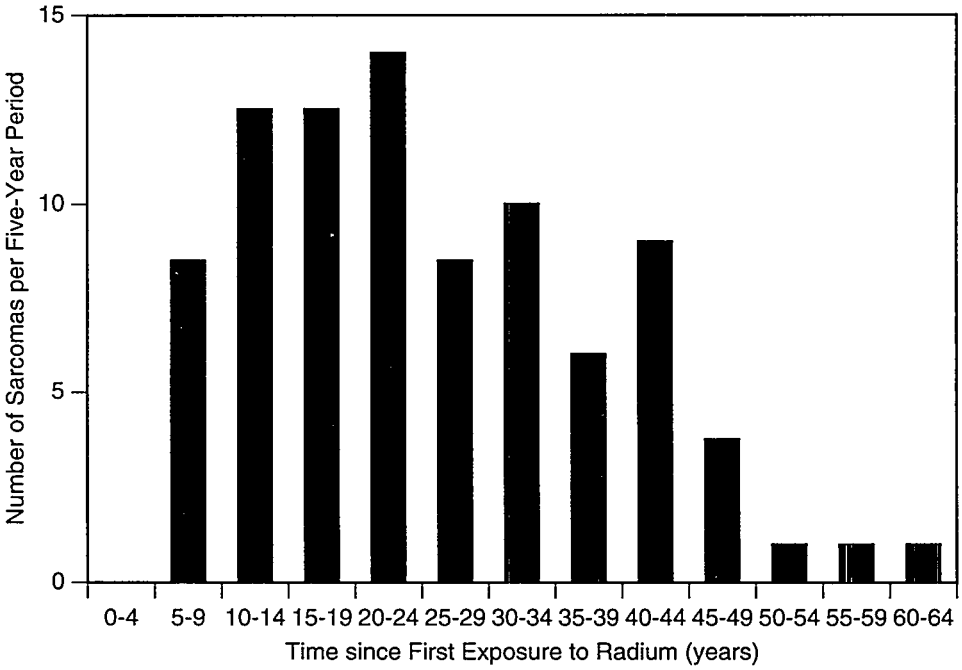

FIGURE 10 Appearance times of bone sarcomas are grouped by five-year periods.

The marked dropoff in the number of bone sarcomas appearing 50 or more years after first exposure could well reflect the fact that few subjects with high intakes were still alive that long after exposure. All of the high intakes in the dial workers occurred before 1925. Since the average age of those entering the industry then was about 20 years, only workers surviving into their 70s would have appearance times greater than 50 years. Subjects who received radium medically often were considerably older than the dial workers. Therefore, 50 years after intake, they would have been in their 80s or 90s.

Figure 11 shows the relationship between time of appearance (diagnosis) of the bone sarcomas and the initial systemic intake. The initial systemic intake here is the sum of μCi of ^{226}Ra plus 2.5 times μCi of ^{228}Ra. The one point omitted from the plot in Figure 11 represents a bone sarcoma in a subject with an intake twice any of those plotted (6,331 μCi). Diagnosed 7 years after first exposure to radium, this bone sarcoma occurred in the dial worker with the highest calculated intake of any radium case.

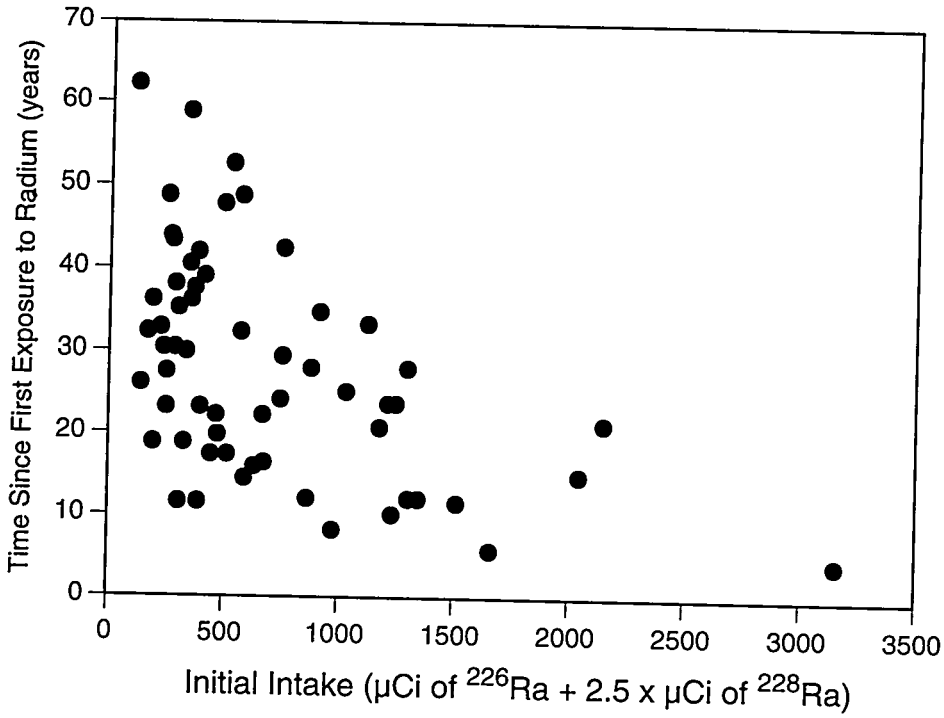

FIGURE 11 This plot of appearance time versus initial systemic intake for radium-induced bone sarcomas does not support the practical threshold hypothesis.

Figure 11 indicates that subjects with low radium intakes have experienced bone sarcomas at short times as well as long times after radium intake. This observation is in contrast to the practical threshold hypothesis, which implies that lower doses are correlated with larger appearance times. Few sarcomas occurred at long appearance times in individuals with high radium intakes, possibly reflecting the fact that only 17 measured cases had combined intakes exceeding 2,000 μCi. Four of these individuals developed bone sarcomas, in agreement with the observation that about one-third of the cases at any intake level ever developed bone sarcomas.

Table 9 summarizes all of the measured individuals considered to have radium-induced bone sarcomas. This table gives the year of first exposure to radium and the year in which the sarcoma was diagnosed. Also listed are initial systemic intake and skeletal dose values for both radium isotopes.

TABLE 9 Measured Radium Cases with Bone Sarcomas

Case	Sex	Route[a]	Year of First Exposure	Year Diagnosed	Initial Systemic Intake		Skeletal Dose at Death	
					μCi ^{226}Ra	μCi ^{228}Ra	cGy ^{226}Ra	cGy ^{228}Ra
00-003	F	DP	1917	1927	603.65	2,290.80	3,404	24,410
00-004	F	DP	1917	1930	908.13	163.53	6,721	2,138
00-005	F	DP	1917	1939	169.12	203.48	1,614	2,964
00-006	F	DP	1918	1930	242.72	507.86	1,556	6,021
00-007	F	DP	1919	1934	112.64	195.59	894	2,650
00-019	F	DP	1917	1946	325.60	393.98	3,966	6,082
00-023	F	DP	1917	1929	690.14	71.51	4,604	874
00-027	F	DP	1918	1942	317.90	356.12	3,474	5,397
00-028	F	DP	1917	1930	1,012.34	132.04	7,549	1,726
01-007	F	RI	1926	1948	466.12	0	5,110	0
01-009	F	DP	1918	1944	876.14	63.72	10,678	985
01-011	F	RW	1919	1936	668.26	0	5,811	0
01-024	F	DP	1916	1956	178.72	68.01	2,444	1,027
01-025	F	RI	1924	1950	227.30	5.81	2,501	85
01-026	F	DP	1925	1955	135.14	76.65	1,547	1,104
01-031	F	DP	1925	1934	79.06	359.03	443	3,786
01-032	F	DP	1924	1940	154.37	750.65	1,255	10,303
01-033	F	DP	1923	1930	198.15	1,181.87	990	11,186
01-046	F	DP	1920	1942	66.30	438.99	660	6,489
01-051	F	DP	1923	1972	34.97	208.20	569	3,133
01-059	F	DP	1920	1962	38.22	221.84	593	3,383
01-073	F	DP	1921	1969	27.20	185.69	379	2,707
01-079	F	DP	1920	1942	92.16	822.00	962	12,326

TABLE 9 (Cont.)

Case	Sex	Route[a]	Year of First Exposure	Year Diagnosed	Initial Systemic Intake		Skeletal Dose at Death	
					μCi ^{226}Ra	μCi ^{228}Ra	cGy ^{226}Ra	cGy ^{228}Ra
01-099	F	DP	1924	1942	29.86	169.19	260	2,319
01-103	F	DP	1922	1946	50.43	271.97	530	4,060
01-105	F	DP	1921	1945	59.71	469.94	672	7,156
01-112	F	DP	1924	1954	24.99	116.26	242	1,578
01-172	F	DP	1916	1968	339.87	71.74	6,409	1,131
01-179	F	RI	1924	1943	330.23	0	5,267	0
01-239	F	DP	1917	1955	201.94	35.17	2,773	523
01-268	F	DP	1917	1959	42.95	134.59	558	1,903
01-389	F	DP	1923	1930	78.58	631.40	360	5,436
01-439	F	RW	1922	1949	135.98	0	1316	0
01-520	F	RD	1930	1967	163.72	68.32	2,162	1,008
01-562	F	DP	1920	1931	948.49	116.46	5,976	1,365
01-613	F	DP	1923	1935	72.27	125.72	419	1,389
03-106	F	RI	1931	1957	134.42	0	1,239	0
03-110	F	RI	1931	1963	175.72	0	1,943	0
03-118	F	RI	1931	1955	390.53	0	4,329	0
03-201	F	RW	1922	1962	483.48	0	7,956	0
03-209	M	RI	1925	1958	215.21	0	1,767	0
03-210	M	RI	1926	1956	250.16	9.19	2,236	98
03-212	F	RW	1927	1951	226.85	5.26	2,276	75
03-213	F	RI	1925	1954	886.99	0	11,802	0
03-215	M	RI	1925	1957	558.63	0	7,146	0

TABLE 9 (Cont.)

Case	Sex	Route[a]	Year of First Exposure	Year Diagnosed	Initial Systemic Intake		Skeletal Dose at Death	
					μCi ^{226}Ra	μCi ^{228}Ra	cGy ^{226}Ra	cGy ^{228}Ra
03-216	F	RI	1922	1959	183.97	0	2,148	0
03-227	F	RI	1930	1949	193.49	0	1,724	0
03-234	F	RI	1915	1964	252.61	0	4,031	0
03-401	F	DP	1923	1962	370.99	0	5,797	0
03-402	F	DP	1923	1953	275.17	8.53	5,154	132
03-429	F	DP	1923	1967	273.69	0	4,722	0
03-455	F	DP	1922	1934	170.60	54.34	2,859	800
03-584	F	DP	1923	1958	916.33	0	13,868	0
03-619	F	DP	1922	1962	282.07	45.63	4,362	710
03-648	F	DP	1922	1956	730.71	155.28	10,290	2,435
03-649	F	DP	1924	1953	233.73	0.29	2,651	4
03-658	F	DP	1922	1938	625.13	0	4,832	0
03-671	F	DP	1922	1952	540.54	84.61	7,338	1,322
03-680	F	DP	1924	1943	498.73	0	5,307	0
03-848	F	DP	1922	1958	294.01	0	4,149	0
05-215	F	DP	1917	1960	253.43	200.36	4,707	3,166
05-281	F	DP	1916	1956	165.00	84.14	2,551	1,270
05-917	F	DP	1918	1981	81.53	7.42	933	229
05-953	F	DP	1918	1977	282.62	23.67	5,547	368

[a] Routes of exposure: DP, dial painter; RI, radium injection; RD, Radithor; RW, radium water.

Individuals with unknown or uncertain radium contents who had probable or confirmed bone sarcomas are listed in Table 10, along with the year of first exposure and the year of sarcoma diagnosis or appearance.

Head Carcinomas

Table 11 summarizes the subjects with diagnosed head carcinomas (of the paranasal sinus and mastoid air cell carcinomas) observed in the radium-exposed population, with distributions by sex and between the measured and unmeasured cases. Fewer of these malignancies occurred than bone sarcomas, 37 versus 85. Five of the 37 persons with head carcinomas also had diagnosed bone sarcomas. These numbers indicate that radium-exposed individuals developed more than twice as many bone sarcomas as head carcinomas.

Table 12 compares the head carcinomas in dial workers and in all other radium-exposed subjects.

When the time of appearance is examined, a significant difference is apparent between head carcinomas and bone sarcomas; the head carcinomas seem to appear much later. Figure 12 shows the times of appearance, in intervals of 5 years after first exposure to radium, for head carcinomas. The first head carcinoma was diagnosed 19 years after exposure to radium.

In Figure 13, the time of appearance of head carcinomas is plotted against the initial systemic intake. Here the initial systemic intake refers to the value for ^{226}Ra alone, because these malignancies are induced by radon (^{222}Rn) trapped within the air spaces in bone. Therefore, the ^{226}Ra body content is the significant parameter, because the very short half-life (55 seconds) of ^{220}Rn, formed in the decay scheme of ^{228}Ra, precludes its migration into these cavities.

When the measured cases are arranged in order of increasing initial systemic intake, the head carcinoma occurring at the lowest dose (Figure 13) is found to be number 2,024 in the list of 2,383 cases, with a calculated intake of 25.63 μCi of ^{226}Ra. As indicated below, this intake value is incorrect; the true value is larger, but how much larger is unknown.

The subject with the head carcinoma occurring at the lowest dose, Case 03-225, was given radium as radium water daily for approximately two years when he was six to eight years old. Looney et al. (1955) gave the following account of the treatment in their description of another case, the

TABLE 10 Unmeasured Cases with Bone Sarcomas

Case	Sex	Route[a]	Year of First Exposure	Year Diagnosed
00-011	F	DP	1917	1935
00-013	F	DP	1917	1933
00-030	F	DP	1918	1923
00-031	F	DP	1920	1938
00-035	F	DP	1917	1941
01-088	F	DP	1923	1931
01-107	F	DP	1923	1935
01-108	F	DP	1924	1947
01-117	F	DP	1922	1931
01-387	F	RD	1918	1943
01-465	M	RD	1925	1943
01-695	F	DP	1923	1935
03-660	F	DP	1923	1935
03-661	F	DP	1922	1934
03-665	F	DP	1924	1929
03-759	F	DP	1924	1930
03-800	F	DP	1924	1944
03-806	F	DP	1922	1956
05-534	F	DP	1917	1937
05-987	F	DP	1918	1962
09-087	M	CR	1912	1933

[a] Routes of exposure: CR, radium chemist; DP, dial painter; RD, Radithor.

TABLE 11 Head Carcinomas by Sex

	Number of Persons			Number of Head Carcinomas		
Case	Female	Male	Total	In Females	In Males	Total
Measured cases	1,903	480	2,383	27	5	32
Unmeasured cases	2,781	1,511	4,292	5	0	5

TABLE 12 Distribution of Head Carcinomas in Dial Workers and Other Radium-Exposed Subjects

Case	Number of Persons[a]		Number of Head Carcinomas[b]	
	Female	Male	In Females	In Males
Measured cases				
Dial workers	1,747	161	19	0
Other cases	156	319	8	5
Total	1,903	480	27	5
Cases not measured				
Dial workers	1,910	315	5	0
Other cases	871	1,196	0	0
Total	2,781	1,511	5	0
Sum	4,684	1,991	32	5

[a] Total persons = 6,675.

[b] Total head carcinomas = 37.

subject's older (by two years) brother (Case 03-220), identified at that time as Patient 120:

> On questioning, the patient did not at first recall ever having received radium, and because of his age it was difficult to conceive that he had been given radium therapeutically at the height of its popularity as a medicament. A perusal of the old files of Schlundt revealed, however, that the patient's father, a physician, had been a patient of Dr. [Findley John] and had been given radium by him. The patient then remembered that for a period of about two years, when he was between the ages of eight and ten, his father had his brother, mother, and himself each drink a glassful of "radium water" daily as a tonic.

At the ages of these two brothers, which averaged seven years and nine years during the period when they received radium from their father, bones are growing rapidly in size and shape. Thus, some of the radium deposited in the bones was probably removed rapidly. Because of the very rapid clearance of radium from human blood, little resorbed radium is

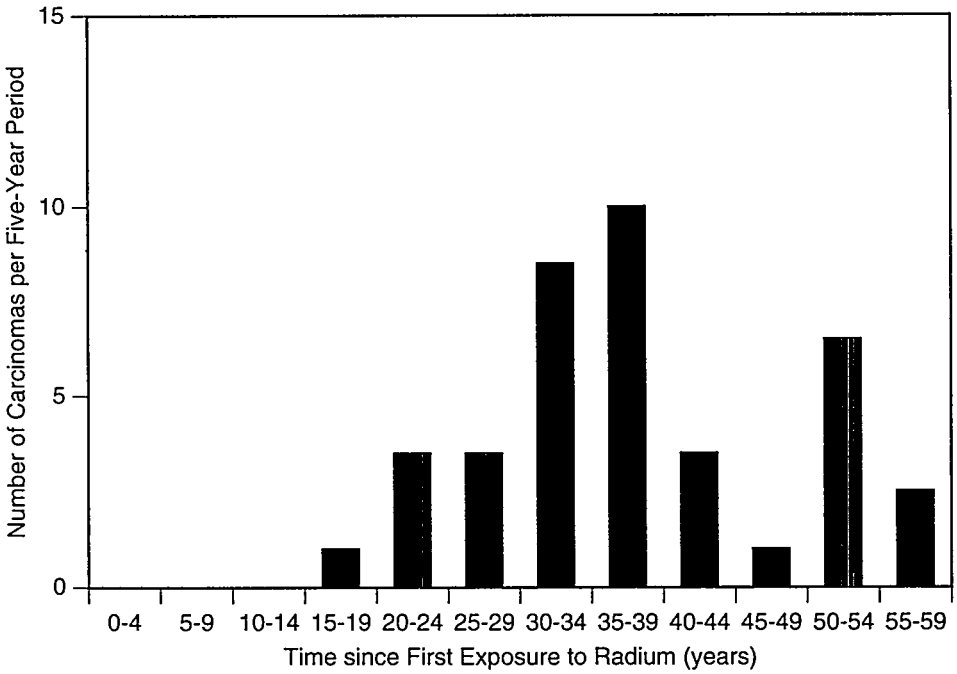

FIGURE 12 Appearance times for radium-induced head carcinomas are grouped by five-year periods.

redeposited. Thus, rapid growth would deplete radium from the body much faster than the radium retention functions, which are based on adult models, would predict. This principle is demonstrated by the results of measurements of the radium content of the older brother, Case 03-220, whose intake is listed as 83.73 μCi of ^{226}Ra. Thus, the two-year difference in age of the two brothers, who apparently received equal doses of radium, caused their intake values to differ by more than a factor of three. Even the value for the older brother is probably too low, but it may not be far from the true value. The quantity of radium given each day was probably about 1 μCi of ^{226}Ra, which would total 730 μCi in two years, resulting in an intake of 20% of 730 μCi or about 146 μCi. This case was discussed in some detail by Keane and Mays (1987).

Aside from this case, the carcinoma case at the next lowest dose is number 2,138 on the ranked list of 2,383 measured cases, with a value of 77.96 μCi of ^{226}Ra. The case at the highest dose is number 2,371, with an intake of 998.09 μCi of ^{226}Ra.

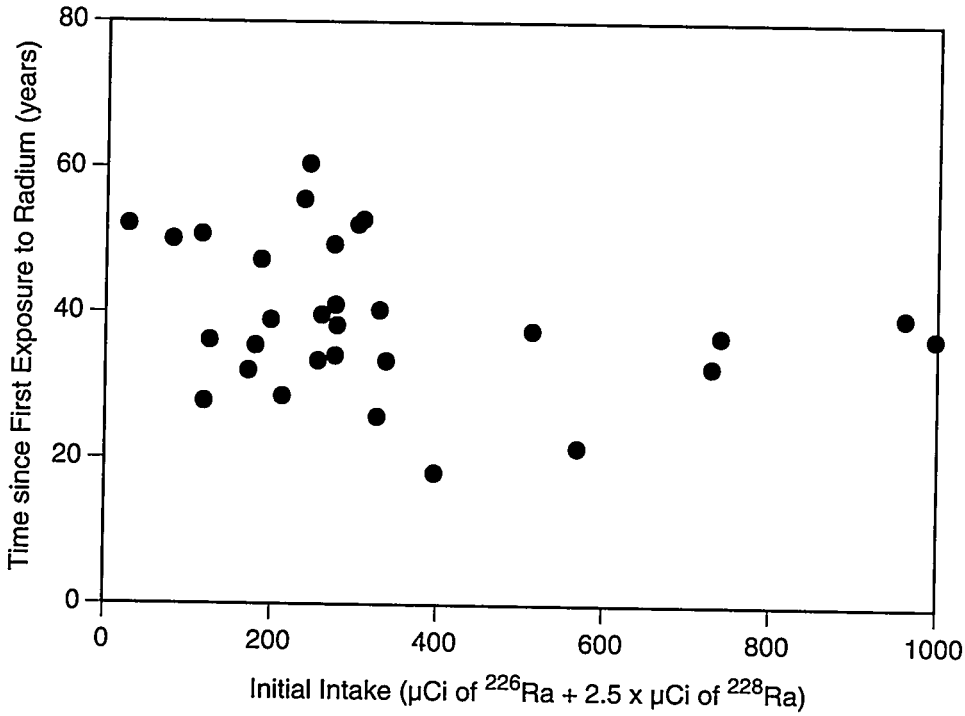

FIGURE 13 Appearance times for radium-induced head carcinomas are plotted against initial systemic intake levels.

Table 13 summarizes the measured individuals considered to have radium-induced carcinomas of the paranasal sinus or mastoid. Given for each case are the year of first exposure to radium and the year of diagnosis of the malignancy. The initial systemic intake is provided only for ^{226}Ra on the assumption that ^{228}Ra was not effective in inducing these malignancies. Similarly, the skeletal dose from only ^{226}Ra is included. Table 13 also lists the five individuals with unknown or uncertain radium contents who had probable or confirmed diagnoses of these malignancies.

Multiple Myeloma

Multiple myeloma is more prevalent among the radium cases than might be expected. Cuzik (1981) reported eight deaths from myeloma in a registry of 3,600 radium-exposed persons. These deaths, he stated, were identified by examination of death certificates, and all occurred after 1959. Six of these deaths occurred among dial painters who had worked in the industry before 1929; five were in women. The other two deaths with myeloma included a woman who started dial painting in 1944 and a male chemist.

TABLE 13 Radium Cases with Head Carcinomas

Case	Sex	Route[a]	Year of First Exposure	Year Diagnosed	Initial Systemic Intake (μCi ^{226}Ra)	Skeletal Dose at Death (cGy ^{226}Ra)
Measured Cases						
01-006	F	DP	1919	1938	398.69	3,463
01-008	F	DP	1917	1958	962.18	15,752
01-014	F	DP	1916	1949	338.74	4,591
01-022	F	DP	1917	1951	177.44	1,909
01-087	F	DP	1921	1957	125.46	2,569
01-145	F	DP	1918	1957	998.09	15,815
01-149	F	DP	1919	1958	261.06	4,236
01-171	M	RI	1914	1966	301.43	4,251
01-179	F	RI	1924	1965	330.38	5,267
01-573	F	DP	1916	1945	121.33	1,291
03-101	F	RI	1931	1970	278.28	4,106
03-105	M	RI	1931	1957	326.98	2,709
03-110	F	RI	1931	1963	175.74	1,943
03-126	F	RI	1931	1965	257.32	3,320
03-141	M	RI	1933	1963	213.03	1,664
03-206	M	RI	1936	1974	515.63	5,862
03-214	F	RI	1925	1959	277.71	4,164
03-225	M	RW	1929	1981	25.63	189
03-232	F	RI	1917	1956	743.81	12,118
03-235	F	RI	1928	1965	264.18	3,813
03-240	F	RI	1930	1953	571.43	6,670
03-402	F	DP	1923	1964	275.18	5,154
03-407	F	DP	1923	1959	275.58	3,882
03-417	F	DP	1924	1962	197.12	2,459
03-423	F	DP	1923	1971	186.44	2,431
03-429	F	DP	1923	1973	273.66	4,722
03-433	F	DP	1924	1983	243.47	4,180
03-459	F	DP	1924	1980	238.38	3,876
03-488	F	DP	1922	1973	77.96	862
03-648	F	DP	1922	1955	730.71	10,290
03-676	F	DP	1924	1976	311.05	5,708
05-284	F	DP	1919	1970	115.62	1,422

TABLE 13 (Cont.)

Case	Sex	Route[a]	Year of First Exposure	Year Diagnosed	Initial Systemic Intake (μCi ^{226}Ra)	Skeletal Dose at Death (cGy ^{226}Ra)
Unmeasured Cases						
01-587	F	DP	1919	1943	-	-
03-675	F	DP	1922	1959	-	-
03-760	F	DP	1924	1946	-	-
03-772	F	DP	1922	1953	-	-
03-785	F	DP	1925	1953	-	-

[a] Routes of exposure: DP, dial painter; RI, radium injection; RW, radium water.

Cuzik (1981) apparently deduced from Polednak et al. (1978) that fewer than 0.86 deaths from myeloma would be expected among all workers in radium dial plants (not more than 2,000 workers before 1929). Polednak et al. (1978) had stated that, among 634 female dial workers identified from employment lists or similar sources who were employed before 1930, no deaths occurred from causes identified as "lymphatic and myeloma" (ICD numbers 200-203), while the expected number was 0.81. Cuzik (1981) assumed that 35% of the deaths in this group (ICD numbers 200-203) would be myelomas; by a proportional extrapolation he determined the expected number of 0.86 deaths in a population of 2,000 women with 5 deaths observed. Thus, his observed number implies a statistically significant death rate for myeloma.

Stebbings et al. (1984) examined the methodology of Polednak et al. (1978) and concluded that Cuzik's expected value was in error because of an error in the code numbers used by Polednak et al. (1978). Stebbings et al. (1984) reported 6 deaths with multiple myelomas among female dial workers and calculated an expected number of 2.15, which is still a significant result ($p = 0.045$). However, one of these deaths was found to have been improperly certified as multiple myeloma when it was actually mycosis fungicides. If this discrepancy is taken into account, the observed number is not significant (5 observed, 2.15 expected, $p = 0.135$).

Stebbings et al. (1984) also pointed out that of the five women who actually died with multiple myeloma, one was never measured, while the four who were measured had low radium intakes. However, all had worked for 40 or more weeks at their jobs, suggesting that if the malignancy was indeed a consequence of their employment, it might be a consequence not of their radium body burden but of the length of their employment, that is, of their extended period of exposure to external gamma radiation.

As of 1990, 11 cases in the files had deaths coded to ICD 203, multiple myeloma. These cases are listed in Table 14; in 6 cases the radium intakes were calculated from a radium body content measurement, while the remaining 5 were never measured.

Seven of the cases in Table 14 were first exposed by 1922. The earliest death from multiple myeloma among these cases occurred 42 years after exposure. The ^{226}Ra intakes were not high, and there was no intake of ^{228}Ra. All of these subjects worked one or more years in contact with radium. The last four individuals, all unmeasured, worked in the 1940s or early 1950s. No measured worker from this period has yet been found with a high radium intake, so these cases probably also had very little radium intake. Interestingly, the time between exposure to radium and the appearance of myelomas was somewhat shorter for this unmeasured group.

Examination of the SNOP files identified an additional six cases coded as having multiple myeloma but with deaths coded to a different cause. These cases are summarized in Table 15, along with two cases still alive at last contact. These individuals survived long periods after first exposure to radium, the minimum being 40 years. The year of diagnosis was not always known for the cases in Table 15, but in each case the first entry about malignant myeloma was only a few years before death. For the cases indicated as still living, the entries were dated in the early 1980s.

Breast Cancer

Several studies have examined deaths attributed to breast cancer among female workers in the U.S. dial painting industry. Polednak et al. (1978) examined causes of death for 634 women who entered the industry between 1915 and 1929 and whose names were obtained from employment lists or similar sources; they found 9 deaths from breast cancer when 9.45 were expected. When this cohort was restricted to those whose body content had been measured in 1954 or later, 3 deaths from breast cancer were found among the 302 women with radium intakes of less than 50 μCi

TABLE 14 Radium Case Deaths Coded to Multiple Myeloma

Case	Sex	Exposure Route[a] and Year	Duration of Exposure (weeks)	Year of Death	Systemic Intake of ^{226}Ra (μCi)	Systemic Intake of ^{228}Ra (μCi)	Time from Exposure until Death (years)
Measured Cases							
01-217	M	DP 1914	208	1971	3.73	0	57
01-282	M	CR 1916	156	1973	41.42	0	57
03-642	F	DP 1922	52	1978	31.18	0	56
05-116	F	DP 1917	52	1959	13.21	0	42
05-310	F	DP 1916	78	1965	4.35	0	49
09-020	F	DP 1917	52	1968	0.79	0	51
Unmeasured Cases							
01-673	F	DH 1918		1964			46
12-116	F	DP 1943		1983			40
12-697	F	DP 1944		1965			21
14-396	F	WC 1950		1983			33
14-732	F	WC 1951		1977			26

[a] Exposure routes: CR, radium chemist; DH, dial handler; DP, dial painter; WC, U.S. Radium Corporation, Bloomsburg, Pennsylvania.

TABLE 15 Radium Subjects with Multiple Myeloma Who Were Alive at Last Contact or Whose Deaths Were Coded to Another Cause

Case	Sex	Exposure Route[a] and Year	Duration of Exposure (weeks)	Year of Death	Systemic Intake of ^{226}Ra (μCi)	Systemic Intake of ^{228}Ra (μCi)	Time from Exposure until Death (years)
01-051	F	DP 1923	162	1977	35.00	208.40	54
01-141	M	RD 1928	130	1978	10.57	7.49	50
01-740	F	DP 1916	NA[b]	1974	-	-	58
03-488	F	DP 1922	26	1975	77.96	11.29	53
03-586	F	DP 1926	82	1968	231.46	0	42
11-104	F	DH 1942	43	1985	0.57	0	43
14-201	M	DB 1950	235	Living	1.99	0	> 40
14-776	F	DP 1952	312	Living	0	0	> 38

[a] Exposure routes: DB, dial worker who did not handle radium; DH, dial handler; DP, dial painter; RD, radium water.

[b] NA, not available.

(2.79 expected), while 1 death was found in the 98 with intakes greater than 50 μCi (0.62 expected). Neither of these results is significant at the 5% level.

In contrast to these results, Adams and Brues (1980) found a statistically significant increase in breast cancer mortality and incidence at high intake levels. They examined the data for 1,180 radium dial workers who entered the industry before 1930, 779 of whom had been measured. In the measured cases of this cohort, 13 breast cancer deaths occurred (16.96 expected), while in the 401 unmeasured cases 23 deaths occurred (6.81 expected). When entry into the study was defined as occurring at the time of first measurement and the breast cancers diagnosed before first measurement were excluded, 11 deaths remained in 736 measured cases. Six of these deaths were in the group whose radium intake was greater than 50 μCi (1.04 expected); 5 deaths were in the group with intakes less than 50 μCi (7.8 expected).

Adams and Brues (1980) also examined the incidence of breast cancer in their measured cohort. They found 9 incident cases in the group exposed to more than 50 μCi (2.72 expected) and 16 incident cases in the group exposed to less than 50 μCi (20.68 expected). Both mortality and incidence data were said to be significant (chi-square p values < 0.001) for the group with intakes above 50 μCi.

Subsequently, Stebbings et al. (1984) published results that made the Adams and Brues (1980) result suspect. The Stebbings study differed from both earlier studies (Polednak et al. [1978] and Adams and Brues [1980]) in that it took into account the observation of Keane and Schlenker (1983) that the soft-tissue alpha-particle dose from ^{228}Ra at short times after radium intake was nearly six times that from ^{226}Ra. The previous two studies defined intake as the sum of the intakes of the two radium isotopes, but Stebbings et al. (1984) took the intake to be the sum of the μCi of ^{226}Ra and six times the μCi of ^{228}Ra. Further, they took for their study population all ascertained dial workers, not just those from employment and other lists.

Stebbings et al. (1984) found, as had Adams and Brues (1980), that the standard mortality ratios for breast cancer increased with increasing radium intake in the pre-1930 cohort. However, they also found an increase in the 1930-1949 cohort, even though the radium intakes in this period were much lower than in the pre-1930 era. Thus, the 1930-1949 cohort showed a significant increase in breast cancer deaths for an intake range that had fewer deaths than expected in the pre-1930 cohort. Next, Stebbings et al. found, when the study population was divided into three workplace locations, that more breast cancer deaths than expected were found in two locations: Orange, New Jersey, and Ottawa, Illinois. However, in Waterbury, Connecticut,

the observed deaths were only 15% of the expected number. No reason for these differences is known.

The studies mentioned above examined radium dial workers. In contrast, Rowland et al. (1989) examined breast cancer mortality in a cohort of female dial painters. (The term *dial workers* includes not only those who painted dials but also those who worked at other tasks in the dial painting workplace. The term *dial painters* refers only to those who actually painted the dials.) Rowland et al. identified a population of 1,261 female dial painters that did not include any cases with the well-known radium-induced malignancies, the bone sarcomas or the head carcinomas. This population started work before 1950 and had been measured for radium while living; the length of employment was also known for each case. This last requirement was introduced to test the effect of external gamma radiation on the induction of breast cancer, because the total external dose would be proportional to the length of exposure or the period of employment.

This defined population contained 924 painters still alive at last contact or at the end of 1985 and 377 known to be deceased. Of the 377 deaths, 26 were attributed to breast cancer (16 expected); this difference is significant at the $p < 0.05$ level by the chi-square test. However, when the external dose to the whole body was estimated from the employment history, the mortality from breast cancer did not increase with increasing whole-body dose. The authors suggested that the actual breast dose might be the significant parameter. Because of the placement of a supply of radium paint directly in front of each painter, the actual breast dose would be greater than the estimated whole-body dose from all the sources in the workplace. An estimate of 15 cGy/yr to the breast was made for the average painter, but this dose was recognized to depend strongly on the work habits of each dial painter. Some may have placed the paint supply near their bodies as they sat at their work benches, while others may have placed the paint supply farther away.

The studies reviewed above clearly show that breast cancer was elevated in some but not all of the workplaces. However, no clear evidence indicates that this elevation was due to the radium acquired internally or to the external gamma-ray dose.

At the end of 1990 the radium case files contained data for 1,903 women who had their radium body contents measured at least once. Of these, 815 were known to be deceased. A total of 37 deaths were coded to breast cancer, and 32 of these were dial painters. This measured group included 1,541 dial painters or 81% of the cases, and they experienced 86% of the breast cancer deaths.

In addition, 2,781 women were identified as being exposed to radium but were never measured. Of this group 893 are known to be deceased. Among the deceased are 49 deaths coded to breast cancer. Thirty-five of these deaths, or 71%, were among dial painters. In this unmeasured group, 1,544 women (56%) were known to have been dial painters. However, an additional 755 women are not identified as to the type of radium exposure; they had 9 of the 49 breast cancer deaths. Thus, the true numbers of dial painters and breast cancers among dial painters are not known. (This unidentified group is from the U.S. Radium Corporation plant at Bloomsburg, Pennsylvania.) Nevertheless, the dial painters apparently had more breast cancer deaths than would be expected by their number in the exposed population.

The observations on female breast cancer can be extended to include incident cases by means of the SNOP file. The file contains information on a total of 265 radium-exposed individuals who were diagnosed with breast cancer. Of these, 245 were dial workers; of the dial workers, 212 were dial painters. The total number, 265, is three times the number (86) of measured and unmeasured breast cancer deaths. Without doubt, breast cancer is elevated among the radium cases, but the cause is unknown.

As indicated earlier, the SNOP file is incomplete. For example, death certificate records indicate that 67 dial painters died as a consequence of breast cancer (32 measured and 35 unmeasured cases), but only 28 of these are listed in the SNOP file. If less than half (28 out of 67) of the individuals who died from breast cancer are in the SNOP file, then the true number of incident cases may be much greater than the 265 cases discussed above. With time and manpower, all of the medical files of each of the more than 6,000 radium cases could be examined to bring the SNOP file up to date, not just for breast cancer but for all conditions listed.

Leukemias

Radiation-induced leukemias are known to appear shortly after exposure, are known to be easily induced when red marrow is the target, and have been an expected consequence of radium deposition in bone. However, they have not been seen in excess in any cohort of radium cases. The definitive study of leukemia incidence in female radium dial workers was published by Spiers et al. (1983), who examined a population of 2,696 female dial workers who started work before 1970. Nine leukemias were found, while the expected number from natural causes was 7.97.

Spiers et al. (1983) also demonstrated that the risk estimates given by the ICRP (1977), which were based on dose calculations with a quality factor of 20 for alpha particles, were not applicable for internally deposited radium. For a cohort of 693 women whose radium burdens were measured while they were living, the number predicted from the alpha-particle dose and the risk estimate was 2.63. The number expected from natural causes was 2.05. The sum of the expected numbers, 4.68, was more than twice the observed number of 2 leukemias in this cohort.

Other studies led to the same conclusion. Polednak et al. (1978) found 3 leukemia deaths in a population of 634 women identified from employment lists and equivalent sources entering the dial painting industry before 1930. The expected number was 1.41. Subsequently, Stebbings et al. (1983), using a population of 1,285 pre-1930 dial workers, found 3 leukemia deaths when 4.1 were expected; for 1,185 women who entered the industry between 1930 and 1949, 4 leukemia deaths were found when 1.8 were expected. The contradiction here is that the women employed between 1930 and 1949 acquired far lower radium burdens and were exposed to lower gamma-ray doses in the workplace than those employed before 1930. Thus, these results do not lead to a conclusion that the small excesses noted were due to the radium exposure.

Among radium cases, 21 deaths were coded to leukemia; 7 of these individuals were dial painters. The SNOP files identified 32 diagnosed cases of leukemia among the radium cases; 10 of these individuals had the cause of death coded to leukemia. There is no indication that leukemias are elevated in this population.

Life Shortening in the Dial Worker Population

Polednak et al. (1978), in a study of 634 women who entered the dial painting industry between 1915 and 1929, found a significantly greater number of deaths (240) than expected (188.5). Yet, they concluded that there was "no apparent increase in the number of deaths from various chronic diseases other than cancer. . . ."

This subject was examined in detail by Stehney et al. (1978), who applied life table methods to survival data for female radium dial workers, examining a cohort of 1,235 women employed before 1930 for whom age, year of death, and withdrawal/loss from the study were known. The closing year for this study was 1976, making observation times of 45-60 years possible.

The entire population of 1,235 women was examined by comparing the observed and expected deaths per calendar year, starting with the year of first exposure to radium and ending in 1976. Observed deaths numbered 529, while only 461.2 were expected, yielding an observed-to-expected ratio of 1.15 ($p < 0.005$). However, removal of the deaths from radium-induced malignancies (bone sarcomas and head carcinomas) reduced the observed-to-expected ratio to 0.99 (455 deaths versus 460.3 expected). When the cumulative net survival was plotted against the cumulative net expected survival, the two curves were almost identical.

Stehney et al. (1978) summarized their findings as follows:

> This study has demonstrated that when the radium tumor deaths are removed, the average survival of the dial worker population is indistinguishable from estimates of the survival of contemporary white females of the same age. This is a remarkable result, for it implies that, to the precision obtainable with a population of some 1000 persons, the life expectancy of the remaining population was unaffected by radium burden.

Some of the earliest dial painters did die of causes other than bone sarcoma or head carcinoma shortly after leaving their work. Without doubt, these women had very large radium burdens. None of them survived long enough to be in the population studied by Stehney et al. (1978). Their radium intakes were thought to be much higher (and in cases measured after death, intakes were found to be higher) than those of any of the women in the above study. People with very large radium burdens will certainly experience radiation damage and probably will not survive long enough for a malignancy to develop.

After the radium program at Argonne had ended, Stehney (in press) presented a second paper on life shortening at an international seminar on radium and thorium (in Heidelberg, April 1994). In this publication, which extended the study to the end of 1989, Stehney used the revised estimates of systemic radium intake and enlarged the study population to include 1,301 women first employed before 1930 (the early group) and 1,242 first employed in 1930-1949 (the late group). In the early group, 85 deaths from radium-induced malignancies were observed, but only 724 deaths occurred from all other causes versus 755 expected. Life shortening ($\pm$ standard error) of 1.8 ± 0.5 years was calculated for the entire group before the cases with radium-induced malignancies were removed. The late group experienced no radium-induced malignancies, and 350 deaths were observed versus 343 expected. This work, therefore, validated the conclusion from the original

study that the life expectancy of the population remaining after removal of radium-induced malignancies was unaffected by radium burden.

Bone Changes

Since the radium isotopes are deposited in bone mineral, seeing the effects of these radioactive isotopes in bone is not surprising. The bone sarcomas are the most obvious effect, but subtler effects are also well known. Martland (1929) pointed out some of the major effects on bone of internally deposited radium. Discussing five female dial painters still living at the time, he stated the following:

> One girl has had a spontaneous fracture through the upper third of the femur producing extensive crippling.
>
> The disease is now present in a milder form, undoubtedly as the result of the natural uninfluenceable decay of the mesothorium, which formed the largest percentage of the original deposits. There is a constant diminution in the amount of irritative radiation. The main symptoms now are crippling bone lesions, the result of radiation osteitis.

Today one might argue that the milder appearance of the disease was observed because the then living cases had lower total radium intakes than Martland's earlier cases, not because of the decay of mesothorium in the body. Nevertheless, bone changes were evidently taking place in these early, high-level radium cases.

Subsequently, Aub et al. (1952), in their review of 30 patients who had carried radium as long as 25 years, stated the following:

> The fundamental lesion observed as a late effect of internally deposited radium (and presumably, to a lesser extent, mesothorium) occurs in the skeletal system. The bony changes, which can be visualized in roentgenograms before symptoms develop, correspond to the "radiation osteitis" described by Martland in his early reports of radium poisoning. The first abnormality of bone noted in roentgenograms is a coarsening of the trabeculae.
>
> In later stages of the disease, this type of bone destruction can also be seen in the cortical portions of the bones.

> Pathologic fracture is another form of injury which results from the bone destruction produced by bone-fixed radioactive materials.

Not surprisingly, Evans (1966), in his study of the radium cases at MIT, and subsequently Finkel et al. (1969), in their study at Argonne, developed a quantitative measure of the radium changes visible in radiographs, which they called the *X-ray score*. This scoring system was described (Evans 1966) as follows:

> In order to quantify the effect on the bones, Dr. John E. Gary, in collaboration with Dr. Robert J. Hasterlik of the Argonne Cancer Research Hospital and Dr. Asher J. Finkel of the Argonne National Laboratory developed a reproducible X-ray scoring system several years ago. The body is divided into 20 radiographic areas, and 34 films covering these areas and including both lateral and antero-posterior views of several areas are obtained.

In his publication, Evans (1966) plotted the X-ray score against various measures of skeletal dose to demonstrate how the scores were essentially zero ("no symptoms") until the dose level was reached at which spontaneous fractures and malignancies were first seen. The cases in this particular study included ". . . about 270 individuals for whom complete, whole-body X-ray scores have been obtained." In 26 of these individuals, 29 bone sarcomas had occurred, and some 20 subjects had suffered 53 spontaneous bone fractures.

Evans (1966) felt that the primary cause of osteomyelitis and dense-bone necrosis was injury to the circulation in bone. He stated that

> the primary deposit of radium or mesothorium in the bone serves to irradiate the cells of the circulatory system in bone and thus to destroy a portion of the circulation. Such a situation would then be followed by necrosis of the areas thus deprived of their circulation.

Rowland et al. (1959b) showed, by using microradiographs of thin sections of undecalcified bone, that bone from high-level radium cases was characterized by mineral plugs in the vascular spaces of Haversian systems. These plugs, which obliterated the small blood vessels in these systems, increased in number with increasing radium body content. Since bone from individuals exposed to very high levels of radium often had up to 25% of the Haversian canals plugged, a marked disruption of blood flow through such

bones was assumed to have occurred and to be a primary cause of the observed bone necrosis.

One consequence of the disruption of the flow of blood through bone was the death of osteocytes, the cells in bone lacunae that are assumed to provide the mechanism for the flow of fluids through compact bone and thus to play a role in the exchange of minerals between bone and blood. Rowland et al. (1959b) found that often many lacunae in bone containing high levels of radium were filled with mineral, obliterating the osteocytes and thus eliminating the blood-bone exchange processes in these regions.

Keane et al. (1983) described a study of X-ray scores of 201 women who had been radium dial workers and were exposed primarily to ^{226}Ra and 159 similar women who were exposed primarily to ^{228}Ra. The authors considered the severity of the changes to be related to the numerical scores as follows: scores of 0.5-8 indicated minimal changes; 8.5-16, mild changes; 16.5-25, moderate changes; above 25, advanced changes. This study had 120 matched controls, 16 of whom had scores in the minimal-change range (0.5-8). For those exposed primarily to ^{226}Ra, only one case (out of 164) with initial systemic intake under 50 μCi of ^{226}Ra had a score greater than 8, and no case under 100 μCi had a score greater than 16. Similarly, for those exposed primarily to ^{228}Ra, no case with an intake less than 50 μCi of ^{228}Ra had a score greater than 8, and no case under 100 μCi had a score greater than 16. Above 100 μCi of either radium isotope, scores above 16 were quite common.

These results indicate that intake levels as large as 50 μCi of either radium isotope produce bone changes that cannot be distinguished from changes sometimes appearing in unexposed individuals. Only when the intake levels are significantly larger do bone changes appear that clearly indicate the presence of radium within the body.

As mentioned previously, all of the values of initial systemic intake and skeletal dose have been recalculated. This recalculation became necessary upon the discovery that low-level radium cases lost radium more rapidly than predicted by the Norris retention function (Norris et al. 1955). All of the references to values of initial systemic intake or skeletal dose in this manuscript refer to the newly calculated values. However, the values published by Keane et al. (1983) and quoted above are examples of the old values. To compare them with the new values, ratios of new values to old values were calculated. For example, 85 dial workers with intake values greater than 5 μCi and less than 15 μCi of ^{226}Ra yielded a new-to-old ratio of 2.4. Thus, a case with an old intake of 10 μCi of ^{226}Ra would now be listed as having about 24 μCi. Similar calculations at other intake levels

demonstrated that old values of about 50 μCi of ^{226}Ra would now be about 85 μCi, while old values near 100 μCi ^{226}Ra would still be near 100 μCi, and thus little changed by the new calculation.

Since the lowest ^{226}Ra intake level associated with a malignancy (a head carcinoma) is 78 μCi ^{226}Ra (new dose calculations), malignancies have apparently occurred at intake levels that may or may not cause changes in bone that are observable on X-ray images. Radium-induced malignancies may well occur at intake levels accompanied by no other diagnostic symptom.

5 Risk Estimates for Radium-Induced Malignancies

When the Argonne radium studies were initiated, the maximum permissible body burden, 0.1 μCi, was the reference value used to determine whether a radium-exposed individual might be at risk. This value had been adopted in 1941. Its adoption was reviewed by Evans (1980, 1981).

During the early years of the Argonne program, the emphasis was on enlarging the population under study and on improving the measurement techniques. Little attention was directed toward dose-response relationships. Hasterlik (1956) did call attention to the finding that radiographic changes increased in number and severity with increasing radium body content. Subsequently, Finkel et al. (1969) created dose-response curves for their data by plotting radium burden on a logarithmic scale versus incidence of radium-induced malignancies on a linear scale. The curves they drew suggested that a threshold existed. These workers examined the incidence of malignancies against both preterminal body content and estimated maximum radium burdens. No attempt was made to express the resulting curves algebraically; malignancies included both bone sarcomas and head carcinomas.

Meanwhile, at MIT, where more cases were available for study, Evans (1966) also examined dose-response functions. Using the available population of measured cases, he pointed out that at least 12 radiation dose parameters could be used with some 16 different response parameters. Graphic representations of 9 of these combinations were included in this 1966 publication. Evans pointed out that for residual ^{226}Ra burdens of 0.5-60 μCi, the fractional incidence of osteogenic sarcomas plus carcinomas of the paranasal sinuses or of the mastoids was about 40%; this value appeared to be independent of the residual body burden. Evans also suggested that time to tumor appearance increased substantially as the residual body burden decreased, so that for small residual body burdens the time to tumor appearance might exceed the human life span, providing a practical threshold below which these malignancies might not appear.

Other publications during this period used data from the radium programs to derive alternate dose-response functions for radium-induced malignancies (Hems 1967; Goss 1970; Gofman and Tamplin 1971). These analyses usually forced linear functions through the data without testing the resulting curves for goodness of fit.

Hems (1967) found that the data could be described by a linear nonthreshold function that predicted a tumor risk of 1 in 20 at an initial radium content of 100 μCi. Evans et al. (1968), in a letter to the editor of the journal that published Hems's paper, challenged the manner in which the data had been selected from the quoted sources. Hems, for example, selected only portions of

the Finkel et al. (1964) data, omitting 117 cases below 0.1 μCi, where no tumors were seen. Similarly, only portions of the MIT data were used in Hems's analysis.

Goss (1970) took a different approach. He suggested that a zero incidence of tumors in the low dose range was highly unlikely and suggested that a linear dose-response relationship existed in the region between zero dose and the apparent "practical threshold." Goss tested the Evans et al. (1969) data against a natural tumor incidence rate of 0.09% at zero dose and a rate of 1.09% at 138 cGy (his calculation of the dose after a daily intake of radium that would result in a body content of 0.1 μCi after 50 years). This function predicted 3.41 tumors in the 406 subjects below 1,200 cGy, among whom no tumors were seen; Goss stated that the probability of seeing none if the prediction was true was 3.1%. He then tested this function against the Evans et al. (1969) data combined with the Finkel et al. (1969) data, obtaining about twice as many cases with no tumors up to a maximum dose of 450 cGy (the Argonne tumor case at the lowest dose). This data set yielded a probability of only 1.9% of seeing none instead of the predicted number. Goss concluded that neither of these probabilities was small enough to reject the linear hypothesis.

Gofman and Tamplin (1971) tested individual dose ranges from the data of Evans et al. (1969) and Finkel et al. (1969) against a linear expression of the form

$$I = I_0 (1 + 0.1D), \quad (2)$$

where I = expected cancer incidence,

I_0 = spontaneous cancer incidence, and

D = dose in cGy.

Gofman and Tamplin (1971) used a spontaneous tumor incidence of 0.0006 and calculated the expected incidence in each set of data for each dose range where no bone malignancies were seen. Their expression yielded a value that did not lie outside the range of 50-90% probability for any dose range tested. They concluded that the data for the radium cases neither supported a threshold hypothesis nor rejected a linear model of radiation carcinogenesis. They also pointed out, however, that their analysis did not prove that the linear model was correct or disprove the existence of a safe threshold.

Subsequently, Evans (1974) expounded on his threshold concept and provided dose-response plots of cumulative tumor incidence versus cumulative skeletal cGy for more than 500 epidemiologically suitable cases. He found tumor incidence for these cases to be 28 ± 6% at 1,000-50,000 cGy, and zero below

1,000 cGy. His population consisted of 503 cases below 1,000 cGy, none with tumors and all "epidemiologically suitable," and another 102 cases above 1,000 cGy, only 67 of which (with 19 tumors) were suitable. (The 35 unsuitable cases had 24 tumors.)

Evans (1974) also addressed other studies that had used the radium data to demonstrate that linear nonthreshold dose-response functions could be fitted to the data. His complaint was that none of the authors had tested their alternative models for goodness of fit. He demonstrated his objection by addressing the paper by Gofman and Tamplin (1971). Using the chi-squared test for goodness of fit over the portion of the data they had selected, Evans found a probability of less than 1 in 200,000,000 that differences from Gofman and Tamplin's linear model as large as or larger than those observed could be due to chance. Evans concluded that the linear model of Gofman and Tamplin was thus not supportable.

When the radium cases were transferred to Argonne's CHR, the now enlarged population was used to examine dose-response relations. Three studies were published in the annual reports (Rowland et al. 1970, 1971a, 1971b). The first two were identical studies; the second was undertaken because some changes in assigned dose values had been made in the previous year, and it was thought appropriate to see whether these changes were sufficient to change the results. The third used initial systemic intake for the measure of radium insult, while the first two had used average skeletal dose.

These studies used all of the available cases, a total of 777 cases at that time, for two reasons. First, it was not clear at that time that cases found as a consequence of their symptoms could be readily identified. Second, including all of the cases would probably bias the results in the direction of enlarging the radium risk, which was thought to be an acceptable practice. In the first study, all tumors were considered together, as had been done in the past; however, the sarcomas and carcinomas were also considered separately, because the tumors and their appearance times were quite different. The following three equations were fitted to the data and tested for goodness of fit:

$$\text{Incidence} = KD \tag{3}$$

$$\text{Incidence} = K_1 D e^{-D/D_1} \tag{4}$$

$$\text{Incidence} = K_2 D^2 e^{-D/D_2} \tag{5}$$

Here K_S and D_S are constants to be determined, incidence is expressed as tumors per person for each dose group, and D is the dose. In each case the sarcoma data were best fitted by the D^2-exponential function, while the carcinoma data were

best fitted by the linear D-exponential function. A linear function through the origin was also fitted for the cases in the dose range in which no tumors had been observed; this function was the one with the largest slope for which the probability P was not less than 0.05. This last fitting was done on the premise that to predict the effects of radium at low doses by using a linear function, one should use a linear function that realistically fits the existing low-dose data.

A number of years were to pass before dose-response functions for the radium cases were published in the open literature by the Argonne group. Rowland et al. (1978) reported functions derived for a population limited to female dial workers employed before 1930. This cohort consisted of 759 women who had experienced 38 bone sarcomas and 17 head carcinomas. Initial systemic intake was used for the dose parameter, and incidence was in units of tumor cases per person year. The fitted functions were derived from an equation of the form

$$\text{Incidence} = (C + \alpha D + \beta D^2)\, e^{-\gamma D}. \quad (6)$$

Here C is the natural incidence of the malignancy for the population under study, and α, β, and γ are parameters to be determined by the least-squares fitting process. Six functions were tested: the general form shown above and five simplifications obtained by omitting the D, the D^2, the exponential term $e^{-\gamma D}$, and the exponential term plus the D or D^2 term. Each of the equations obtained was tested for goodness of fit with a chi-squared statistic. The fitted values and the value of the chi-squared statistic were shown for each trial. Acceptable fits were those for which the confidence level, p, was ≥ 0.05.

To test for possible bias introduced by including individuals with malignancies whose body contents were determined only after exhumation, these cases were removed with little effect on the fitted equations. For each cohort the best fit for the sarcoma data was given by the equation of the form

$$\text{Incidence} = (C + \beta D^2)\, e^{-\gamma D}, \quad (7)$$

and an acceptable fit was provided by the complete general expression.

When the carcinoma cases were tested, the best fit was given by the expression

$$\text{Incidence} = C + \alpha D, \quad (8)$$

but the complete expression and three other simplifications of it also provided acceptable fits. The small number of carcinomas (17) allowed less discrimination between the proposed equations than the larger number (38) of sarcomas.

The next Argonne publication on dose-response (Rowland et al. 1983) used data for female dial painters who started work before 1950. Two cohorts were examined to find dose-response functions for the induction of bone sarcomas. The first cohort included all measured subjects who survived at least five years after first employment, while the second limited the analysis to those surviving two years after the first measurement of their body contents. The five-year requirement for the first cohort was based on an assumed five-year minimum bone sarcoma induction period, while the two-year survival for the second cohort was chosen to eliminate individuals who might have been measured because of a diagnosed bone sarcoma. The first cohort contained 1,468 women who experienced 42 bone sarcomas; the second was limited to 1,257 women with only 13 bone sarcomas. The striking reduction in the number of sarcomas in the second group reflects the fact that the study, and thus the measurement of most of the dial painters, took place long after they started work and often as a consequence of their symptoms. For this analysis, incidence was in units of bone sarcomas per person year, and dose (D) was in initial systemic intake, where intake was expressed as the sum of μCi of ^{226}Ra plus 2.5 times μCi of ^{228}Ra.

Rowland et al. (1983) used the same procedure as in their 1978 publication and again found the best fit to be given by a function of the form

$$\text{Incidence} = (C + \beta D^2)\, e^{-\gamma D} \qquad (9)$$

for the first cohort. The second cohort (defined by the time of first measurement) was found to be fitted both by the above function and by the simple linear function.

Rowland et al. (1983) used the dose-squared function, as found for the first cohort (defined by time of entry into dial painting), to predict the occurrence of bone sarcomas in two other populations of measured radium cases. These individuals, who acquired radium from sources such as medical usage and laboratory exposure, included all of the other measured radium cases. One group consisted of 138 women who experienced 15 bone sarcomas. The derived function predicted 17.3 sarcomas for this population, in remarkable agreement with the observed number. The second group contained 347 men who experienced 3 sarcomas. From the distribution of systemic intakes derived from the measured body burdens for this group, 10.9 sarcomas were predicted. This is an unlikely result, indicating either that the population of men in the CHR files is not representative of radium-exposed males or that the dose-response function derived from observation of female dial painters does not apply to males.

In an attempt to address the risk from radium and uranium isotopes in drinking water, Mays et al. (1985) predicted the bone sarcoma risk from ^{226}Ra with the linear function from the second cohort (defined by time of first

measurement) of Rowland et al. (1983). For the carcinomas, Mays et al. used the function derived by Rowland et al. (1978), which again was linear. Since drinking water normally contains low levels of radium, lifetime accumulations in the body remain very low, and the use of linear dose-response functions for the very low dose range was judged to be appropriate. Mays et al. found that, for an intake of 5 pCi per day over a lifetime of 75 years, about 9 radium-induced sarcomas and 12 radium-induced carcinomas would occur among 1,000,000 exposed individuals. The same population would experience about 750 bone sarcomas and 375 carcinomas due to natural causes.

These calculations certainly indicate that exposure to the stated level of ^{226}Ra is relatively safe, but the use of linear dose-response functions implies that, at very low radium levels, the carcinomas of the paranasal sinuses and mastoid air cells are more likely to be observed than the bone sarcomas. Since this implication is in direct opposition to the observations made on a large number of radium cases (at much, much higher radium levels), the conclusion should be viewed with suspicion. The dose-squared function, which appears to fit the sarcoma data so well, would predict even fewer bone sarcomas at these very low doses, suggesting that the true description of radium-induced carcinomas might fall off much faster than linearly at the lower doses. With so few of these rare carcinomas as a basis for a dose-response function, this question will probably remain unanswered.

After the radium program at Argonne had ended, Rowland (in press) presented an invited paper on dose-response relationships at an international seminar on radium and thorium (in Heidelberg, April 1994). This new analysis considered all female dial workers with body burden measurements who entered the industry before 1950, a total of 1,530 individuals. In this cohort, 46 women had bone sarcomas, and 19 had head carcinomas; 3 women experienced both a bone sarcoma and a head carcinoma.

Incorporation of the revised estimates of systemic intake into the new calculations significantly changed the dose-response functions. Rowland (in press) found no satisfactory fits with the previously used form of the dose-response function for bone sarcomas (Equations 6 and 9) when the very small constant term indicating the natural rate of sarcomas in an unexposed population was ignored. However, changing the exponent on the systemic intake term (D in Equation 9) from 2 to 3.15 (a parameter determined from the data) produced a good fit. When the exponent was specified instead, satisfactory fits were obtained with exponents of 2.7-4.1. Alternatively, when a threshold was assumed for bone sarcomas and the exponent was set at 3.15, a solution was obtained with a threshold of 79 μCi. The mechanistic meaning of the exponent value of 3.15 is not obvious, suggesting that, at least for radium-induced bone sarcomas in humans, a threshold hypothesis is as good as any other.

In the same publication, Rowland (in press) also examined dose-response functions for head carcinomas. As in previous studies, the linear, linear exponential, and dose-squared exponential functions all fitted the data satisfactorily. The reason lies in the scarcity of head carcinomas. With only 19 of these malignancies observed in the cohort examined, the data are insufficient to discriminate between alternate formulations.

The records indicate that bone sarcomas occurred about twice as often as head carcinomas in the radium-exposed populations. The total known population had 85 bone sarcomas and 37 head carcinomas, while the total measured population had 64 bone sarcomas and 32 head carcinomas. Among the female dial workers, considered the best cohort for dose-response analyses, 46 bone sarcomas and 19 head carcinomas were observed. Because the number of head carcinomas is not sufficient to allow unequivocal determination of the best form for a dose-response function, the best predictor of their incidence at a given intake level is the predicted number of bone sarcomas at that level, divided by two.

At the same international seminar in Heidelberg, Thomas (in press) presented work in which he used lognormal data analysis and the newly calculated skeletal dose values (presented in the appendix of this document) to reach the conclusion that a threshold exists for radium-induced malignancies. Thomas calculated threshold values of 390-620 cGy and suggested the adoption of a value of 1,000 cGy as the threshold for radium-induced malignancies in humans, the same value Evans (1974) had derived 20 years earlier.

Among the unanswered questions that remain is the striking paucity of bone tumors in males. Is this a defect in the accumulated data, or is it a real effect? If it is real, it may be indicative of some male-female differences. Another question is whether other malignancies, such as multiple myeloma or breast cancer in females, are induced by radium. Much remains to be learned from radium-exposed humans, but little time remains before they are all gone.

6 The Termination of the Radium Studies Program

In 1981, Dr. W.K. Sinclair resigned his position as Argonne's Associate Laboratory Director (ALD) for Biomedical and Environmental Research to accept the presidency of the National Council on Radiation Protection and Measurement. (Sinclair had held this Argonne position since it was established in April 1974. Previously the biology program had been under the ALD for Physical Research.) Argonne's director, Dr. Walter Massey, appointed Dr. R.E. Rowland the interim ALD, effective September 1, 1981, and asked him to form a committee to search for a permanent ALD. Rowland took this opportunity to indicate that he would not accept the position of ALD on a permanent basis and that he would not return to the RER Division as its director. He retired in February 1983. Dr. Harvey Drucker was hired to fill the position; he arrived in August 1983.

In May 1983 the RER Division was merged with the Environmental Impact Studies Division to form the Environmental Research (ER) Division, with Dr. P.F. Gustafson as the acting division director. Subsequently, in the fall of 1984, the CHR was transferred to the BIM Division, and the medical records and many of the staff ultimately moved from Building 203 to Building 202. Over the next decade several different staff members in turn took on the administrative responsibility for the program. Because of these changes and budgetary uncertainties, many of the staff elected to transfer to more stable programs elsewhere at Argonne.

With the continual reduction in funding and the transfer of personnel and program responsibility, the character of the program changed markedly. Very few patients were brought to the Laboratory after 1984. However, the staff continued to record the deaths of known cases, and periodically death certificates were obtained.

At the beginning of Fiscal Year 1991 the U.S. Department of Energy shifted management for the project (now called the Internal Emitter Program) from the Office of Health and Environmental Research of the Office of Energy Research (OER), where it had long resided, to the Assistant Secretary for Environment, Safety, and Health (ESH). The epidemiology programs that had been funded by OER were now placed in ESH under the Office of Epidemiology and Health Surveillance, under Dr. Robert Goldsmith. In January 1991, a letter from Dr. Harry Pettingill, Deputy Assistant Secretary for Health, to Drucker requested that responsibility for the Internal Emitter Program be transferred from the BIM Division back to the ER Division. In addition, Pettingill requested that Dr. Robert Thomas of the ER Division assume management responsibility for the project, beginning in October 1991.

In June 1991, Thomas called a meeting of a few non-Argonne scientists plus a small contingent of past radium program staff members, some supported in other divisions and others retired but still associated with Argonne through special-term appointments. These included Stehney, Lucas, Rowland, Schlenker, Keane, and Toohey; two representatives of the Office of Epidemiology and Health Surveillance, John Peeters and Michael Ginevan, also attended. The objective was to propose a plan that would best suit the needs of the new U.S. Department of Energy management. The meeting identified five important tasks for the final stages of the long-standing radium program. These tasks were the following:

- Perform a predictive analysis of the detectable level of risk from exposure to radium.
- Update the database by means of the ORACLE system.
- Complete a history of the program.
- Interact with scientists in other epidemiologic programs to broaden the perception of the importance of the radium data.
- Inventory and archive the skeletal and soft-tissue samples accumulated from autopsy material.

These five tasks were accepted by the U.S. Department of Energy program manager as an acceptable approach.

During Fiscal Year 1992 the morgue in Building 203 at Argonne was cleared of all equipment and supplies and was decontaminated and sterilized for unrestricted use. The frozen tissue samples from autopsies were transported to Washington State University, under the direction of Dr. Ronald Kathren.

In September 1992 a letter from Dr. Terry Thomas, Director of Health Communication and Coordination, ESH, was sent to Robert Thomas, directing that the project be terminated on September 30, 1993, and that no copying of medical records or radiographs take place. In December 1992 a meeting was called by Mr. William LeFurgy, now manager of the Internal Emitter Program in the Office of Epidemiology and Health Surveillance, to direct that all medical records be digitally copied and that the radiographs be copied next if time and funds permitted. In March 1993 this order was rescinded by letter from Goldsmith to Dr. Christopher Reilly, Director of Argonne's ER Division.

As this volume goes to press, the Argonne program has been terminated, but no decision has been made about the disposition of the individual case records.

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Appendix:

Measured Radium Cases

Appendix:

Measured Radium Cases

Table A.1 in this appendix summarizes data on 2,403 radium-exposed individuals for whom satisfactory measurements of radium body content had been made by the end of 1990. This total is 20 more than the number listed in the tables in the text; the additional cases are the offspring of females exposed to radium. Twelve of these offspring had no measurable radium contents; among the remaining eight, the highest calculated intake was 7.7 μCi of ^{226}Ra.

The cases are listed in order of identification number, followed by the sex and the year of birth. The fourth column ("Live") contains the year of last contact with the live subject, if that subject has been lost to the study. Most living subjects are not considered lost because of frequent contacts; for these cases the year 1990 is listed, even though the individual may not actually have been contacted in 1990. The date in this column has been used to calculate the skeletal dose for cases not known to be deceased. No entry is in this column when the individual is known to be deceased; in this case the year of death is listed in the next column. The sixth column contains the code number for the cause of death according to the eighth revision of the International Classification of Diseases.

The seventh column ("Exp. Type") contains a two-letter code that signifies the type of exposure to radium. These codes are identified in Table A.2.

The eighth column in Table A.1 ("Year First Exp.") contains the year of first exposure to radium, and the ninth column contains the duration of the exposure in weeks. A value of zero means that the exposure was a single event or had a duration of less than one week. However, "+0" means that the duration of exposure is unknown; in these cases zero duration was used in the calculation of dose. For a dial painter whose first exposure was before 1926 but whose period of exposure extended into 1926 or beyond, the duration used in calculating the dose corresponds to the exposure terminating in 1926. The remaining columns contain information on body content and dose calculations. The first of these ("Year of Meas.") contains the year of the measurement on which the remaining data have been based, and the next column, "^{226}Ra (nCi)," contains the total body ^{226}Ra measured at that time. The 12th column, "^{228}Ra to ^{226}Ra Ratio," contains the calculated ratio of ^{228}Ra to ^{226}Ra, at the time when the radium body content was measured. The next two columns contain the initial systemic intake, first for ^{226}Ra and next for ^{228}Ra, as calculated with the revised ICRP 20 retention function discussed

in the text and with an appropriate bone turnover rate. The final two columns contain the skeletal dose (cGy) at death, last contact, or the end of 1990, first for ^{226}Ra and finally for ^{228}Ra.

TABLE A.1 Exposure Data for Radium Subjects to the End of 1990, Calculated with the Modified ICRP 20 Retention Function

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
00-001	M	1883		1928	9261E	CR	1913	780	1967	13000	0.00700	807.9	1036.9	2729	6149
00-002	F	1896		1922	5280	DP	1917	223	1966	16000	0.00110	797.8	232.7	1912	785
00-003	F	1894		1927	1704	DP	1917	104	1966	7000	0.01200	603.7	2290.8	3404	24410
00-004	F	1900		1931	9261E	DP	1917	88	1963	9000	0.00080	908.1	163.5	6721	2138
00-005	F	1901		1939	1707	DP	1917	300	1963	1400	0.00700	169.1	203.4	1614	2964
00-006	F	1903		1930	1951	DP	1918	128	1969	2610	0.00536	242.7	507.9	1556	6021
00-007	F	1903		1935	481	DP	1919	104	1963	1000	0.01000	112.5	195.4	894	2650
00-008	M	1890		1938	1621	CR	1915	598	1972	3045	0.00288	341.1	412.5	2179	4167
00-009	F	1900		1928	6510	DP	1918	266	1969	2650	0.00490	209.3	333.0	1025	3050
00-017	F	1899		1924	9261E	DP	1917	156	1970	17000	0.00069	1189.4	391.8	4693	2776
00-019	F	1895		1946	1991	DP	1917	260	1976	2400	0.00140	325.6	394.0	3966	6082
00-020	M	1888		1925	284	CR	1912	676	1969	920	0.00228	62.2	42.7	171	214
00-022	F	1889		1925	2810	DP	1917	377	1960	10000	0.01000	579.2	594.2	1886	3219
00-023	F	1900		1929	1951	DP	1917	65	1978	7214	0.00007	690.1	71.5	4604	874
00-027	F	1902		1942	9261E	DP	1918	130	1970	2500	0.00256	317.9	356.1	3474	5397
00-028	F	1902		1933	946E	DP	1917	279	1969	10000	0.00036	1012.3	132.0	7549	1726
00-029	F	1900		1990		DP	1917	409	1969	17	0.0	14.7	0.0	158	0
00-033	M	1868		1922	2810	CR	1919	156	1970	6	0.00300	0.2	0.2	0	0
00-034	F	1882		1943	582	DH	1917	232	1979	1	0.00060	0.2	0.2	2	2
01-001	F	1878		1949	4123	RI	1922	+0	1972	15400	0.0	2099.5	0.0	25929	0
01-002	F	1906		1939	2079	DP	1922	676	1936	18000	0.01950	1719.3	137.6	13969	1886
01-003	M	1888		1956	1621	RI	1925	304	1967	12800	0.00037	1763.3	69.2	15986	767
01-004	F	1869		1953	946E	RW	1918	+0	1941	10500	0.0	1323.7	0.0	19641	0
01-005	M	1877		1939	5264	RD	1927	12	1939	5000	0.50000	477.0	992.8	2345	8840
01-006	F	1899		1938	946E	DP	1919	260	1970	3590	0.00144	398.7	189.5	3463	2674

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-007	F	1886		1949	1704	RI	1926	+0	1967	3620	0.0	466.1	0.0	5110	0
01-008	F	1900		1958	946E	DP	1917	78	1960	6000	0.00067	962.2	102.5	15752	1620
01-009	F	1898		1945	1707	DP	1918	52	1960	6500	0.00050	876.1	63.7	10678	985
01-010	M	1882		1956	5311	RW	1926	+0	1967	5200	0.0	738.5	0.0	7017	0
01-011	F	1872		1937	1621	RW	1919	156	1975	6000	0.0	668.3	0.0	5811	0
01-012	F	1867		1956	4109	RI	1922	+0	1970	5800	0.0	869.6	0.0	12641	0
01-014	F	1901		1949	946E	DP	1916	156	1968	2240	0.00036	338.7	51.9	4591	808
01-015	M	1888		1967	4329	DP	1917	1664	1961	0	0.0	0.0	0.0	0	0
01-016	F	1891		1966	174	DP	1921	208	1973	1940	0.00245	321.9	314.9	5513	4980
01-017	F	1883		1976	4123	RD	1926	156	1977	1120	0.00156	216.6	128.2	3871	2008
01-018	M	1889		1958	1621	CR	1911	2340	1950	1250	0.0	126.3	0.0	920	0
01-019	F	1903		1936	1700	DP	1922	253	1965	240	0.02958	31.7	126.2	193	1458
01-020	F	1905		1956	4319	RI	1923	5	1950	1500	0.0	256.5	0.0	3250	0
01-021	F	1887		1973	1519	DP	1916	104	1965	1250	0.0	280.8	0.0	5134	0
01-022	F	1900		1951	946E	DP	1917	110	1968	600	0.0	177.4	0.0	1909	0
01-024	F	1901		1956	1709	DP	1916	308	1943	1140	0.02190	178.9	68.1	2444	1027
01-025	F	1886		1952	9320E	RI	1924	+0	1951	1200	0.00100	227.3	5.8	2501	85
01-026	F	1905		1958	1709	DP	1925	156	1950	700	0.03000	135.0	76.6	1547	1104
01-027	M	1889		1957	1621	CR	1912	1040	1960	500	0.0	144.9	0.0	1165	0
01-028	M	1879		1965	9109E	CR	1912	260	1953	250	0.0	113.0	0.0	925	0
01-029	M	1876		1958	342	CR	1902	+0	1950	300	0.0	155.2	0.0	1402	0
01-030	M	1882		1952	070	RA	1936	0	1950	20	0.0	3.8	0.0	17	0
01-031	F	1906		1934	946E	DP	1925	4	1975	910	0.01130	79.1	359.0	443	3786
01-032	F	1908		1940	1709	DP	1924	201	1968	1450	0.02800	154.4	750.6	1255	10303
01-033	F	1908		1931	1704	DP	1923	42	1963	2472	0.05153	198.2	1181.9	990	11186

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-034	F	1913	1990			DP	1929	18	1965	8	0.01000	4.5	3.3	47	42
01-035	F	1901		1972	1531	DP	1920	19	1971	0	0.01860	0.0	0.0	0	0
01-037	F	1908	1990			DP	1928	26	1974	0	0.00327	0.0	0.0	0	0
01-038	F	1910	1990			DP	1927	111	1959	8	0.02000	3.7	3.0	39	38
01-039	F	1915	1990			DH	1934	1092	1972	1	0.0	0.4	0.0	4	0
01-040	F	1907		1929	946E	DP	1923	60	1963	4300	0.05209	299.4	1766.3	1172	12427
01-041	F	1909	1990			DP	1927	22	1971	0	0.00470	0.0	0.0	0	0
01-043	F	1912	1990			DP	1927	8	1958	9	0.02200	4.2	3.7	43	48
01-044	F	1904	1990			DP	1924	22	1959	4	0.08000	2.1	11.1	23	142
01-045	F	1889		1980	5193	DP	1922	237	1959	0	0.08000	0.0	0.0	0	0
01-046	F	1903		1943	946E	DP	1920	657	1963	551	0.05607	66.3	439.2	660	6489
01-047	F	1896		1984		DP	1920	367	1962	80	0.05700	25.9	154.5	377	2203
01-048	F	1900		1979	1621	DP	1920	206	1957	140	0.09290	31.9	195.4	517	2903
01-049	F	1903		1937	946E	DP	1920	1	1960	1000	0.07300	112.0	993.0	995	14117
01-050	F	1911		1986	4123	DP	1925	10	1976	1	0.00258	1.0	1.2	10	15
01-051	F	1904		1977	4123	DP	1923	162	1977	120	0.01100	35.0	208.4	569	3133
01-052	F	1910		1930	946E	DP	1924	144	1965	2000	0.03500	134.3	568.4	495	3672
01-054	F	1909		1937	946E	DP	1924	202	1965	2100	0.03714	203.5	915.3	1410	11473
01-055	F	1907		1988		DP	1925	85	1976	4	0.01024	3.7	16.3	39	210
01-056	F	1904		1978	1991	DP	1920	364	1965	134	0.03432	35.9	185.3	570	2746
01-057	F	1908		1931	9261E	DP	1924	81	1963	4900	0.05163	360.9	1824.2	1560	14568
01-059	F	1905		1967	1959	DP	1920	299	1964	180	0.04277	38.2	221.6	593	3383
01-060	F	1909	1990			DH	1928	20	1974	0	0.00330	0.0	0.0	0	0
01-063	F	1911		1979	1621	DP	1927	213	1976	34	0.00154	26.5	11.3	271	146
01-066	F	1904	1990			DP	1925	0	1980	0	0.00159	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-069	F	1905		1989		DP	1922	107	1976	0	0.01024	0.0	0.0	0	0
01-070	F	1910		1989		DP	1927	63	1973	1	0.00370	0.8	0.7	8	9
01-071	F	1908		1967	1538	DP	1927	6	1958	0	0.02300	0.0	0.0	0	0
01-072	F	1899	1990			DP	1921	130	1954	100	0.10000	30.0	134.6	419	1876
01-073	F	1900		1969	1959	DP	1921	122	1966	87	0.03563	27.2	185.7	379	2707
01-074	F	1909		1990		DP	1927	47	1979	4	0.00172	4.0	3.3	42	43
01-075	F	1902		1990		DP	1922	52	1979	4	0.00713	4.4	27.5	48	354
01-078	F	1909	1990			DP	1925	50	1978	3	0.00194	3.1	3.3	33	42
01-079	F	1901		1943	1707	DP	1920	176	1960	750	0.09070	92.2	822.0	962	12326
01-080	F	1902	1990			DP	1921	204	1968	106	0.02075	36.1	164.6	573	2382
01-081	F	1907	1990			DP	1923	11	1959	7	0.08000	3.8	22.6	41	290
01-082	F	1902		1935	946E	DP	1919	230	1963	1030	0.03786	105.7	592.3	808	7863
01-084	F	1904	1990			DP	1923	712	1974	46	0.01297	24.9	122.3	331	1682
01-085	F	1913	1990			DP	1927	47	1958	6	0.02200	2.7	2.4	29	30
01-086	F	1907		1966	5719	DP	1925	4	1959	0	0.08000	0.0	0.0	0	0
01-087	F	1905		1979	2509	DP	1921	344	1964	780	0.03690	125.5	587.2	2569	9312
01-090	F	1910	1990			DP	1927	90	1977	5	0.00218	4.6	3.6	48	47
01-091	F	1907	1990			DP	1927	264	1979	0	0.00179	0.0	0.0	0	0
01-092	F	1906		1976	4443	DP	1922	24	1971	2	0.01860	1.8	11.6	18	148
01-093	F	1904	1990			DP	1926	8	1971	0	0.00460	0.0	0.0	0	0
01-094	F	1888		1966	402	DP	1921	128	1964	11	0.04400	6.9	45.4	69	590
01-095	F	1907		1977	4109	DP	1922	34	1975	6	0.01163	5.6	36.6	59	473
01-096	F	1909	1990			DP	1927	310	1960	27	0.01800	11.6	7.4	122	95
01-097	F	1905		1986	174	DP	1921	110	1963	122	0.03852	34.9	182.2	567	2669
01-099	F	1905		1945	1707	DP	1924	18	1963	164	0.05365	29.9	169.1	260	2319

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-100	F	1905		1967	7969	DP	1924	36	1957	34	0.13200	13.4	88.9	141	1184
01-101	F	1905	1990			DP	1924	4	1959	0	0.08000	0.0	0.0	0	0
01-103	F	1903		1946	946E	DP	1922	172	1978	374	0.00800	50.5	272.2	530	4060
01-105	F	1898		1945	1709	DP	1921	21	1963	460	0.05217	59.7	470.0	672	7156
01-106	F	1902		1977	1621	DP	1924	155	1959	10	0.08000	5.0	23.6	51	304
01-110	F	1909		1986		DP	1925	93	1979	1	0.00172	1.1	1.1	11	15
01-111	F	1910	1990			DP	1927	16	1980	1	0.00152	1.3	1.1	13	14
01-112	F	1908		1955	1959	DP	1924	835	1960	80	0.07000	25.0	116.3	242	1578
01-113	F	1912		1983	4329	DP	1928	5	1959	3	0.02000	1.4	1.1	14	15
01-115	F	1908		1944	946E	DP	1924	330	1963	472	0.03093	65.0	191.4	585	2713
01-116	F	1899		1965	4339	DP	1920	459	1955	290	0.10000	49.8	225.6	769	3467
01-118	F	1909		1971	4122	DP	1923	13	1959	0	0.08000	0.0	0.0	0	0
01-119	F	1899		1966	4002	DP	1920	14	1958	5	0.09000	2.9	24.7	29	318
01-120	F	1910	1990			DP	1925	125	1959	10	0.02000	5.1	5.7	54	73
01-122	F	1912	1990			DP	1927	49	1981	9	0.00141	8.8	7.7	93	98
01-123	F	1889		1980	174	DP	1923	11	1976	0	0.01024	0.0	0.0	0	0
01-124	F	1909	1990			DP	1927	64	1979	41	0.00180	34.2	29.3	382	382
01-125	F	1911		1988		DP	1927	6	1979	0	0.00179	0.0	0.0	0	0
01-126	F	1903		1969	1830	DP	1922	416	1969	150	0.02667	36.5	213.3	558	3225
01-127	F	1908		1988		DP	1927	9	1974	1	0.00330	0.9	0.8	9	10
01-128	F	1910	1990			DP	1927	4	1959	2	0.02000	1.0	0.9	10	12
01-129	F	1906		1934	2201	DP	1923	4	1977	2	0.00907	0.4	2.1	2	22
01-130	F	1909	1990			DP	1926	196	1964	11	0.01140	6.2	5.3	65	68
01-132	F	1908		1944	9261E	DP	1923	76	1966	1327	0.03496	160.1	891.8	1613	13268
01-133	F	1910	1990			DP	1926	65	1958	13	0.03000	6.0	7.8	64	100

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-136	F	1907	1990			DP	1923	185	1978	30	0.00674	21.7	89.7	266	1205
01-137	F	1901		1984	1621	DP	1923	714	1977	5	0.00902	4.8	23.6	51	304
01-138	F	1883		1963	354	RW	1919	4	1959	6	0.0	3.7	0.0	35	0
01-139	M	1881		1964	4339	RD	1928	130	1962	1270	0.01417	200.6	144.1	2049	1600
01-140	F	1890		1986		DP	1919	78	1975	0	0.0	0.0	0.0	0	0
01-141	M	1886		1978	1590	RD	1928	130	1979	12	0.00180	10.6	7.5	75	69
01-142	F	1899	1990			DP	1917	52	1969	0	0.0	0.0	0.0	0	0
01-143	F	1904		1986		DP	1921	65	1976	1	0.0	1.1	0.0	12	0
01-144	F	1897		1973	7123	RW	1922	26	1971	694	0.0	225.7	0.0	3357	0
01-145	F	1900		1957	946E	DP	1918	60	1966	6331	0.00077	998.1	227.5	15815	3591
01-146	F	1882		1967	4379	RD	1927	156	1968	100	0.00870	48.6	48.1	491	638
01-147	F	1902	1990			DP	1917	26	1965	52	0.0	41.5	0.0	465	0
01-148	F	1907	1965			NU	1936	364	1958	40	0.0	9.9	0.0	78	0
01-149	F	1888		1959	946E	DP	1919	26	1969	1630	0.00533	261.1	545.1	4236	8609
01-150	F	1881		1979	486	RW	1930	104	1970	3	0.0	1.9	0.0	19	0
01-151	F	1905	1990			NU	1927	52	1981	0	0.0	0.0	0.0	0	0
01-152	F	1904		1989		DP	1920	17	1977	2	0.00159	2.4	3.5	26	45
01-153	M	1890		1964	4109	CR	1920	104	1963	280	0.00036	130.5	7.2	1043	70
01-154	M	1896		1968	4109	CR	1923	+0	1959	0	0.01500	0.0	0.0	0	0
01-156	F	1900		1959	5718	DP	1918	156	1959	40	0.0	24.9	0.0	234	0
01-157	F	1894		1982	2000	RD	1925	13	1975	49	0.00139	39.4	21.8	428	285
01-158	F	1901		1977	2901	NU	1920	52	1959	1	0.0	0.6	0.0	6	0
01-159	F	1915	1990			DP	1935	220	1980	5	0.0	3.3	0.0	33	0
01-160	F	1873		1965	4339	RD	1925	+0	1959	130	0.02000	52.1	61.6	542	824
01-161	F	1896		1973	1533	DP	1918	17	1959	1	0.0	0.7	0.0	7	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-162	M	1898		1966	1621	CR	1920	364	1959	95	0.01000	43.9	29.7	320	279
01-163	F	1903		1983	929	DP	1920	26	1972	2	0.00360	2.0	3.6	21	46
01-164	F	1900		1972	486	DP	1918	39	1959	9	0.0	6.1	0.0	62	0
01-165	F	1904	1990			DP	1922	22	1981	13	0.0	16.0	0.0	171	0
01-166	F	1897		1969	4122	DP	1916	26	1959	0	0.0	0.0	0.0	0	0
01-168	F	1895		1989		NU	1919	468	1966	1	0.0	0.7	0.0	8	0
01-169	F	1918	1990			DP	1936	69	1975	0	0.0	0.0	0.0	0	0
01-170	M	1893		1966	492	RI	1940	0	1959	2	0.0	0.5	0.0	3	0
01-171	M	1895		1975	4410	RI	1914	6	1958	1500	0.0	301.4	0.0	4251	0
01-172	F	1898		1968	1709	DP	1916	136	1961	1960	0.00112	339.9	71.7	6409	1131
01-173	M	1881		1959	4419	CR	1917	1300	1958	70	0.0	25.2	0.0	153	0
01-175	F	1900		1966	4123	RD	1927	13	1965	1710	0.00760	272.7	195.2	4318	3072
01-176	F	1893		1969	1890	DP	1917	104	1969	0	0.0	0.0	0.0	0	0
01-177	M	1915	1974			CR	1936	312	1969	61	0.0	25.9	0.0	167	0
01-178	M	1939	1990			RA	1958	0	1973	2	0.0	0.3	0.0	2	0
01-179	F	1890		1966	149	RI	1924	58	1959	2000	0.0	330.4	0.0	5267	0
01-180	F	1900	1990			DP	1918	26	1971	3	0.0	3.2	0.0	34	0
01-181	M	1913		1963	1530	CR	1940	130	1959	220	0.0	49.6	0.0	269	0
01-182	M	1902		1959	4409	RD	1936	+0	1959	7	0.02600	2.3	0.9	12	8
01-183	F	1901		1969	595	DP	1915	78	1969	203	0.0	133.1	0.0	1591	0
01-184	M	1887		1969	4123	RI	1922	10	1968	48	0.0	36.5	0.0	261	0
01-185	M	1881		1962	1540	CR	1912	+0	1959	40	0.0	32.0	0.0	232	0
01-186	M	1925	1990			CR	1943	416	1976	19	0.0	8.0	0.0	54	0
01-187	M	1917	1990			CR	1943	78	1980	23	0.0	12.8	0.0	89	0
01-188	F	1886		1979	4123	RW	1933	3	1959	4	0.0	1.5	0.0	14	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-189	M	1921	1990			RA	1958	0	1973	0	0.0	0.0	0.0	0	0
01-190	F	1927	1990			RA	1958	0	1973	0	0.0	0.0	0.0	0	0
01-191	M	1897		1966	402	CR	1913	78	1959	4	0.0	3.2	0.0	23	0
01-192	F	1902		1962	493	DP	1925	52	1959	34	0.0	17.1	0.0	156	0
01-193	F	1886		1960	4109	CR	1917	156	1974	31	0.0	21.1	0.0	201	0
01-194	M	1898		1982	4123	DP	1916	676	1972	0	0.0	0.0	0.0	0	0
01-195	F	1893		1958	174	CR	1912	520	1959	1	0.0	0.7	0.0	6	0
01-196	M	1907	1990			RD	1930	20	1972	69	0.00540	40.9	33.3	327	312
01-197	F	1883		1965	4123	RW	1916	+0	1958	16	0.0	11.3	0.0	112	0
01-198	F	1888		1972	4109	MI	1913	+0	1959	0	0.0	0.0	0.0	0	0
01-200	F	1910		1987		DP	1925	220	1977	3	0.00914	2.9	13.0	31	167
01-201	F	1911	1990			DP	1925	55	1959	26	0.02100	12.8	15.0	138	192
01-203	F	1908		1983	493	DP	1923	1	1973	0	0.01470	0.0	0.0	0	0
01-204	F	1901		1980	450	DP	1917	22	1959	5	0.0	3.6	0.0	37	0
01-205	M	1921		1974	4109	CR	1951	52	1972	7	0.0	2.1	0.0	11	0
01-206	M	1896		1982	4123	CR	1918	17	1981	11	0.0	15.1	0.0	114	0
01-207	F	1909		1967	4109	DP	1927	9	1959	4	0.02000	1.9	1.8	18	23
01-208	M	1901		1972	1621	CR	1939	1144	1974	818	0.0	146.8	0.0	928	0
01-209	F	1908		1975	450	DP	1926	16	1959	0	0.02700	0.0	0.0	0	0
01-210	M	1878		1971	4123	CR	1918	2028	1959	12	0.0	2.5	0.0	15	0
01-216	F	1903		1963	2839	DP	1924	4	1959	0	0.08000	0.0	0.0	0	0
01-217	M	1894		1971	203	DP	1914	208	1959	5	0.0	3.7	0.0	27	0
01-218	M	1924	1990			CR	1950	780	1974	0	0.0	0.0	0.0	0	0
01-219	F	1910	1990			DP	1927	10	1976	0	0.00246	0.0	0.0	0	0
01-220	F	1907	1990			DP	1924	26	1959	2	0.07100	1.1	5.0	11	64

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-221	M	1892		1970	492	CR	1916	520	1967	10	0.00320	8.0	5.8	57	53
01-222	F	1910		1988		DP	1925	17	1964	4	0.04400	2.5	11.6	26	149
01-223	F	1912	1990			DP	1927	7	1963	0	0.01200	0.0	0.0	0	0
01-225	F	1906		1990		DP	1931	35	1959	0	0.0	0.0	0.0	0	0
01-226	F	1911		1987		DP	1927	22	1976	0	0.00258	0.0	0.0	0	0
01-227	F	1908	1990			DH	1933	2184	1975	0	0.0	0.0	0.0	0	0
01-228	F	1906		1989		DP	1926	61	1972	6	0.00420	4.8	4.7	50	60
01-229	F	1903		1985	428	DP	1923	2	1959	8	0.08000	4.3	26.0	46	334
01-230	F	1913		1989		DP	1927	19	1978	0	0.00203	0.0	0.0	0	0
01-231	F	1910		1969	174	DP	1930	84	1959	0	0.0	0.0	0.0	0	0
01-232	F	1909		1961	9509E	RW	1926	43	1959	0	0.0	0.0	0.0	0	0
01-233	F	1912		1973	1533	DP	1927	145	1959	2	0.02000	0.9	0.7	9	9
01-234	F	1913		1966	1579	DP	1927	1	1959	0	0.02000	0.0	0.0	0	0
01-235	F	1908		1990		DP	1925	8	1959	1	0.08000	0.5	2.5	6	32
01-236	F	1910		1976	9500E	DP	1927	9	1965	1	0.01000	0.6	0.6	6	7
01-237	F	1907	1990			DP	1927	8	1979	0	0.00179	0.0	0.0	0	0
01-238	F	1896		1967	4109	DP	1920	2	1959	1	0.08000	0.6	5.5	6	70
01-239	F	1901		1958	1708	DP	1917	78	1957	830	0.00157	202.0	35.2	2773	523
01-240	F	1910		1990		DP	1927	13	1971	7	0.00450	5.3	4.6	56	59
01-243	M	1873		1959	4339	CR	1905	520	1958	15	0.0	12.9	0.0	91	0
01-244	F	1901		1979	4123	DP	1927	18	1975	1	0.00307	0.9	0.9	9	11
01-245	F	1920	1990			DP	1957	30	1969	0	0.0	0.0	0.0	0	0
01-246	F	1885		1970	4124	NU	1915	39	1967	3	0.0	3.0	0.0	31	0
01-247	M	1901	1990			CR	1923	689	1981	4	0.00107	4.0	1.8	30	16
01-248	F	1903		1990		DP	1917	208	1976	21	0.0	23.8	0.0	260	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-249	M	1928	1975			OF	1928	39	1967	2	0.02700	1.3	3.5	9	32
01-250	M	1894	1990			CR	1916	520	1975	0	0.0	0.0	0.0	0	0
01-251	M	1890		1965	4339	CR	1912	156	1974	11	0.0	10.9	0.0	78	0
01-252	F	1898		1985	4369	DP	1917	104	1976	22	0.0	25.7	0.0	278	0
01-253	F	1898		1964	1538	DP	1916	104	1959	22	0.0	15.1	0.0	148	0
01-254	F	1910		1982	425	DP	1927	2	1971	1	0.00460	0.8	0.7	8	9
01-255	F	1920		1990		DP	1942	52	1975	0	0.0	0.0	0.0	0	0
01-256	M	1919		1982	4109	CR	1949	208	1959	7	0.0	0.8	0.0	5	0
01-257	M	1885		1962	4109	CR	1941	624	1959	0	0.0	0.0	0.0	0	0
01-258	M	1903		1985	4121	CR	1923	1092	1969	17	0.0	9.0	0.0	64	0
01-259	F	1910		1990		NU	1927	416	1977	0	0.0	0.0	0.0	0	0
01-260	F	1891		1960	481	RW	1918	50	1959	8	0.0	5.1	0.0	48	0
01-261	F	1909		1969	4100	DP	1927	2	1959	0	0.02000	0.0	0.0	0	0
01-262	F	1895		1987		NU	1918	0	1969	22	0.0	20.8	0.0	225	0
01-263	F	1897		1976	4109	DP	1917	17	1976	9	0.0	11.3	0.0	117	0
01-264	M	1906		1967	954E	DP	1944	770	1964	90	0.0	14.1	0.0	63	0
01-265	F	1902	1990			DP	1919	2	1959	3	0.08000	1.9	18.7	21	240
01-266	F	1904		1961	1533	DP	1923	3	1959	1	0.08000	0.6	3.4	5	44
01-267	F	1904	1990			DP	1926	104	1966	45	0.0	27.4	0.0	293	0
01-268	F	1901		1968	1990	DP	1917	208	1967	100	0.01000	43.0	134.7	558	1903
01-269	M	1911	1990			CR	1932	624	1979	2	0.0	1.4	0.0	10	0
01-270	F	1901		1988		DP	1943	32	1976	4	0.0	2.0	0.0	19	0
01-271	F	1899	1990			DP	1917	86	1979	2	0.0	2.8	0.0	30	0
01-272	M	1888	1959			CR	1956	130	1959	39	0.0	1.7	0.0	2	0
01-273	F	1907	1990			DP	1924	1	1959	2	0.08400	1.1	6.1	12	78

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-274	F	1906		1980	4109	DP	1922	5	1978	0	0.00799	0.0	0.0	0	0
01-275	M	1930	1972			CR	1959	+0	1959	23	0.0	0.3	0.0	2	0
01-276	M	1930		1962	7123	CR	1945	208	1959	30	0.0	4.9	0.0	22	0
01-277	F	1909		1986		DP	1925	6	1978	5	0.00828	4.6	21.9	48	281
01-278	F	1904		1976	5193	NU	1925	0	1969	10	0.0	7.7	0.0	76	0
01-279	M	1901		1969	491	CR	1928	1404	1966	0	0.0	0.0	0.0	0	0
01-280	F	1905	1990			DP	1926	7	1971	0	0.00460	0.0	0.0	0	0
01-282	M	1893		1973	203	CR	1916	156	1972	42	0.0	41.4	0.0	313	0
01-283	F	1895		1971	1977	DH	1918	52	1959	3	0.0	2.1	0.0	20	0
01-284	M	1892		1970	4349	CR	1943	780	1959	5	0.0	0.5	0.0	3	0
01-285	F	1900		1984	4123	DP	1923	1	1960	4	0.07100	2.3	14.0	24	178
01-287	F	1908		1986		DP	1927	674	1977	2	0.00232	1.5	0.6	15	7
01-288	F	1894		1970	4109	DP	1926	2	1960	2	0.02400	1.1	1.5	10	19
01-289	F	1899		1975	402	DP	1919	80	1971	4	0.01860	3.6	31.6	38	408
01-291	F	1910		1969	4449	DP	1928	17	1960	5	0.01800	2.4	2.0	22	25
01-293	F	1911		1984	5739	DP	1924	11	1978	0	0.00805	0.0	0.0	0	0
01-294	F	1912	1990			DP	1927	52	1971	3	0.00450	2.3	1.9	24	24
01-295	F	1910	1990			DP	1927	14	1981	0	0.00141	0.0	0.0	0	0
01-296	F	1908	1990			DP	1927	5	1960	0	0.01800	0.0	0.0	0	0
01-297	F	1901	1990			DP	1921	122	1960	16	0.09375	8.1	70.7	94	930
01-299	F	1896		1989		DP	1917	104	1968	3	0.0	2.9	0.0	31	0
01-301	F	1904	1990			RI	1926	5	1969	17	0.0	12.4	0.0	131	0
01-302	F	1899		1966	4123	RI	1927	10	1968	2850	0.0	461.2	0.0	7302	0
01-303	M	1919		1990		DP	1940	104	1974	0	0.0	0.0	0.0	0	0
01-305	M	1925		1968	1621	CR	1946	1040	1972	87	0.0	11.7	0.0	44	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-306	M	1928	1990			CR	1955	364	1979	22	0.0	6.1	0.0	37	0
01-307	M	1930	1990			CR	1957	104	1975	4	0.0	0.9	0.0	6	0
01-308	M	1918		1957	2070	CR	1943	728	1958	1200	0.0	85.0	0.0	240	0
01-309	F	1908		1973	453	DP	1923	2	1961	2	0.06200	1.2	7.3	12	92
01-310	F	1928	1976			OF	1928	39	1975	0	0.01148	0.0	0.0	0	0
01-311	F	1911	1990			DP	1927	2	1961	1	0.01500	0.5	0.5	6	6
01-312	F	1907	1990			DP	1925	13	1976	0	0.0	0.0	0.0	0	0
01-313	M	1892		1981	485	CR	1911	624	1961	3	0.0	2.3	0.0	17	0
01-314	F	1909	1990			DP	1924	0	1961	1	0.06200	0.6	3.1	6	40
01-324	F	1907	1990			DP	1923	15	1962	1	0.05700	0.6	3.9	7	49
01-326	F	1896		1972	4299	RD	1925	156	1966	100	0.01100	48.2	60.2	526	807
01-327	F	1908	1990			DP	1927	1	1965	0	0.01000	0.0	0.0	0	0
01-330	M	1915		1988		CR	1942	364	1976	66	0.0	28.4	0.0	196	0
01-331	M	1901		1982	5999	RD	1927	+0	1966	80	0.01100	41.1	48.8	328	460
01-332	F	1912		1971	149	DP	1927	52	1965	0	0.01000	0.0	0.0	0	0
01-333	F	1905		1990		DP	1924	10	1981	1	0.00589	1.3	7.2	14	92
01-335	F	1899		1987		DP	1917	78	1980	3	0.0	4.7	0.0	50	0
01-336	M	1899	1990			CR	1945	1092	1979	41	0.0	12.3	0.0	78	0
01-341	M	1883		1980	4123	CR	1943	176	1961	5	0.0	1.1	0.0	7	0
01-342	M	1897		1990		CR	1944	56	1961	1	0.0	0.2	0.0	2	0
01-343	F	1873		1954	4123	RW	1927	+0	1963	0	0.0	0.0	0.0	0	0
01-344	F	1904		1976	4339	DP	1922	19	1962	7	0.05700	4.3	29.3	45	378
01-345	F	1910		1977	4442	DP	1924	1	1962	4	0.05700	2.4	13.1	24	168
01-346	F	1911	1990			DP	1927	17	1962	44	0.01700	21.9	24.4	239	315
01-347	M	1896		1968	4109	CR	1926	1872	1962	14	0.0	2.0	0.0	10	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-348	F	1902		1973	4109	DP	1924	19	1966	112	0.03482	34.2	180.2	482	2619
01-349	F	1907		1967	4109	DP	1924	10	1966	93	0.03225	32.7	161.3	409	2294
01-350	F	1898		1973	486	DP	1923	108	1962	0	0.05700	0.0	0.0	0	0
01-351	F	1906	1990			DP	1923	3	1962	0	0.05700	0.0	0.0	0	0
01-352	M	1922		1983	1621	CR	1940	338	1962	191	0.0	45.3	0.0	312	0
01-356	M	1912		1973	5609	CR	1937	572	1969	23	0.0	8.5	0.0	52	0
01-357	F	1907		1970	4360	DH	1927	408	1962	0	0.01400	0.0	0.0	0	0
01-358	F	1906		1978	4124	DH	1923	168	1962	0	0.05700	0.0	0.0	0	0
01-359	F	1908	1990			DP	1925	55	1962	25	0.05600	12.5	55.8	142	730
01-360	F	1911	1990			DP	1928	34	1962	0	0.01400	0.0	0.0	0	0
01-361	F	1907		1976	4100	DP	1924	20	1974	1	0.01323	0.9	4.9	9	63
01-362	F	1906		1990		DP	1923	5	1962	0	0.05700	0.0	0.0	0	0
01-363	F	1888		1978	4123	DP	1918	260	1962	7	0.05700	4.5	36.2	48	468
01-364	F	1911	1990			DH	1927	440	1964	6	0.01140	3.0	1.6	31	21
01-365	F	1901		1986		DP	1924	40	1962	10	0.05700	5.7	29.5	61	381
01-367	F	1899		1985	1538	DP	1920	221	1976	4	0.01024	4.0	26.0	43	335
01-368	M	1925	1986			CR	1947	65	1979	35	0.0	16.2	0.0	107	0
01-369	F	1906	1990			DP	1923	33	1975	0	0.01043	0.0	0.0	0	0
01-370	F	1904	1990			DP	1927	21	1962	0	0.01500	0.0	0.0	0	0
01-371	F	1912	1990			DH	1928	39	1979	3	0.00180	2.9	2.3	30	30
01-372	F	1911		1975	491	DP	1927	1	1962	7	0.01470	3.8	3.7	37	48
01-373	F	1910		1988		DP	1927	84	1962	2	0.01400	1.1	0.9	11	12
01-374	F	1910	1990			DP	1927	+0	1962	12	0.01470	6.5	6.4	68	81
01-376	F	1907		1973	5621	DP	1927	33	1963	2	0.01300	1.1	1.1	11	14
01-377	F	1915	1990			DR	1929	208	1979	1	0.0	0.9	0.0	9	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-378	F	1907	1990			DP	1925	94	1981	2	0.00141	1.8	2.0	19	26
01-379	F	1909		1981	4123	DP	1926	7	1975	18	0.00281	15.4	15.5	160	199
01-380	F	1910		1980	1538	DP	1927	3	1972	0	0.00420	0.0	0.0	0	0
01-381	M	1887		1978	2301	RD	1927	1	1964	5	0.01400	3.0	3.5	21	32
01-382	F	1900	1990			DP	1920	320	1963	43	0.01000	24.9	29.3	281	382
01-383	F	1907		1990		DP	1923	2	1976	0	0.01006	0.0	0.0	0	0
01-384	F	1905	1990			DP	1923	1	1975	0	0.01177	0.0	0.0	0	0
01-385	F	1906		1971	4123	DP	1924	11	1963	5	0.05000	3.1	16.5	30	211
01-386	F	1904	1990			DP	1927	15	1963	9	0.01300	5.1	4.9	53	62
01-388	F	1873		1944	4123	RD	1928	+0	1965	2580	0.01027	282.8	246.4	2403	3437
01-389	F	1910		1930	1707	DP	1923	26	1963	1029	0.06812	78.6	631.4	360	5436
01-390	F	1887		1931	988E	RD	1925	260	1965	7400	0.02527	403.5	873.7	1125	3747
01-391	F	1914		1969	174	KH	1950	520	1964	1	0.0	0.1	0.0	1	0
01-392	M	1913		1972	5719	KH	1950	520	1964	1	0.0	0.1	0.0	1	0
01-393	M	1937	1990			KH	1950	520	1972	2	0.0	0.5	0.0	3	0
01-394	F	1944	1990			KH	1950	520	1982	0	0.0	0.0	0.0	0	0
01-395	F	1945	1990			KH	1950	520	1972	5	0.0	1.1	0.0	10	0
01-396	M	1947	1990			KH	1950	520	1982	0	0.0	0.0	0.0	0	0
01-397	F	1950	1990			KH	1950	498	1973	4	0.0	1.0	0.0	9	0
01-398	M	1951	1990			KH	1951	429	1982	0	0.0	0.1	0.0	1	0
01-399	F	1953	1990			KH	1953	350	1982	1	0.0	0.2	0.0	2	0
01-400	M	1903		1982	0388	KH	1961	156	1964	2	0.0	0.1	0.0	0	0
01-401	F	1910		1986	4109	KH	1961	156	1964	3	0.0	0.1	0.0	1	0
01-402	F	1898		1983	5193	DP	1920	18	1963	0	0.05000	0.0	0.0	0	0
01-403	F	1912		1980	535	RD	1926	+0	1971	27	0.01838	17.2	69.9	190	923

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-404	M	1875		1945	869E	CR	1912	1716	1965	2800	0.0	248.9	0.0	1442	0
01-405	F	1885		1957	4410	CR	1912	1716	1965	52	0.0	17.8	0.0	147	0
01-406	M	1902		1969	4123	CR	1916	260	1963	18	0.0	13.8	0.0	98	0
01-407	M	1912		1977	1978	CR	1930	416	1963	38	0.0	15.7	0.0	107	0
01-408	F	1918	1990			CR	1934	416	1978	14	0.0	9.2	0.0	92	0
01-409	F	1914	1990			CR	1930	13	1975	34	0.0	25.7	0.0	271	0
01-410	F	1920	1990			CR	1940	156	1978	33	0.0	18.6	0.0	185	0
01-411	M	1915		1978	5710	CR	1935	200	1973	8	0.0	4.6	0.0	31	0
01-412	M	1915		1970	4124	RD	1929	+0	1963	1	0.01600	0.5	0.5	4	5
01-413	F	1901		1965	3480	DP	1924	229	1964	11	0.04400	6.5	30.8	62	397
01-414	F	1897	1990			CR	1931	78	1979	2	0.0	1.9	0.0	20	0
01-415	M	1898		1979	955E	CR	1921	520	1964	0	0.0	0.0	0.0	0	0
01-416	F	1908	1990			DP	1924	2	1963	9	0.04900	5.4	28.5	59	367
01-417	F	1907		1989		DP	1923	1	1963	0	0.05000	0.0	0.0	0	0
01-418	M	1900		1972	4419	CR	1919	104	1963	6	0.0	4.5	0.0	32	0
01-419	M	1895		1965	4339	CR	1916	260	1963	9	0.0	7.0	0.0	49	0
01-420	F	1903		1967	1951	CR	1920	65	1963	2	0.0	1.5	0.0	14	0
01-421	F	1887		1976	4459	CR	1915	312	1963	8	0.0	6.4	0.0	65	0
01-423	M	1897		1990		CR	1919	260	1973	22	0.0	21.0	0.0	162	0
01-424	F	1882		1979	4123	RI	1924	+0	1979	350	0.0	182.5	0.0	2452	0
01-425	M	1933	1990			KH	1961	104	1964	0	0.0	0.0	0.0	0	0
01-426	F	1930	1990			KH	1961	104	1964	5	0.0	0.2	0.0	2	0
01-427	F	1960	1990			KH	1961	104	1964	5	0.0	0.2	0.0	2	0
01-428	F	1957	1990			KH	1961	104	1964	2	0.0	0.1	0.0	1	0
01-429	F	1897		1987		CR	1922	208	1979	1	0.0	1.1	0.0	12	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-430	M	1880		1969	4210	RD	1930	+0	1966	41	0.02195	20.9	34.5	143	320
01-431	F	1901		1975	2509	RI	1922	52	1971	765	0.0	232.6	0.0	3610	0
01-432	M	1895		1973	4339	CR	1915	520	1964	17	0.0	12.7	0.0	92	0
01-434	M	1880		1932	5264	RD	1927	156	1965	6126	0.02189	348.8	599.9	704	1850
01-435	F	1907	1990			DP	1925	5	1977	0	0.00228	0.0	0.0	0	0
01-436	F	1895		1976	1519	DP	1927	180	1964	8	0.01140	4.4	3.4	43	44
01-437	F	1910		1971	174	CR	1931	104	1965	1	0.0	0.5	0.0	5	0
01-438	M	1867		1940	4219	RD	1925	208	1965	1850	0.01372	186.8	241.5	974	2231
01-439	F	1880		1953	1959	RW	1922	8	1968	406	0.0	136.0	0.0	1316	0
01-440	F	1908		1989		DP	1924	204	1965	0	0.03900	0.0	0.0	0	0
01-443	F	1911		1987		DP	1927	74	1978	8	0.00200	7.5	6.3	78	80
01-447	F	1909	1990			DP	1925	110	1965	3	0.01000	1.9	2.1	20	26
01-448	F	1907	1990			DP	1925	5	1964	25	0.01140	15.0	18.3	163	236
01-449	F	1899	1990			DP	1922	2	1965	7	0.03900	4.8	32.6	53	420
01-450	M	1877		1936	1621	CR	1912	364	1966	0	0.0	0.0	0.0	0	0
01-451	F	1908		1978	5321	DP	1924	4	1977	14	0.00907	11.9	61.9	129	812
01-453	F	1899		1963	4409	DP	1920	20	1979	4	0.00780	2.8	25.5	27	327
01-454	F	1880		1970	4123	DP	1920	884	1974	1990	0.00034	356.7	53.7	6397	847
01-456	M	1878		1948	1579	RD	1928	26	1965	74	0.03648	18.1	54.3	95	489
01-457	F	1904	1990			CR	1920	78	1964	8	0.0	6.0	0.0	64	0
01-459	M	1886		1971	4379	CR	1921	52	1964	10	0.0	7.3	0.0	51	0
01-460	M	1882		1966	694	CR	1912	104	1964	0	0.0	0.0	0.0	0	0
01-461	M	1914		1970	4123	CR	1930	26	1964	9	0.0	4.7	0.0	31	0
01-464	F	1908	1990			DP	1927	4	1970	4	0.00540	3.0	2.8	31	35
01-466	F	1902		1946	1830	DP	1920	52	1965	0	0.03800	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-468	F	1910		1987		DP	1927	0	1978	0	0.00209	0.0	0.0	0	0
01-469	M	1894		1983	4109	CR	1918	52	1965	4	0.0	3.4	0.0	25	0
01-470	F	1912		1984	4109	DP	1927	70	1965	0	0.01000	0.0	0.0	0	0
01-472	F	1896		1969	4339	CR	1919	156	1965	7	0.0	5.5	0.0	54	0
01-474	F	1904		1985	4109	DH	1921	100	1979	0	0.00637	0.0	0.0	0	0
01-475	F	1901		1986	4123	DP	1928	4	1974	0	0.00330	0.0	0.0	0	0
01-476	F	1909		1981	4109	DH	1927	71	1972	4	0.00420	3.1	2.7	31	34
01-477	F	1897		1978	4123	RD	1925	+0	1965	1240	0.00475	218.2	126.0	4117	1972
01-478	F	1914		1986	4123	DP	1935	24	1965	0	0.0	0.0	0.0	0	0
01-479	F	1912		1986	1538	DP	1927	1	1978	2	0.00209	2.4	2.3	24	29
01-480	F	1915	1990			DP	1927	1	1965	38	0.01000	21.5	20.6	234	266
01-481	F	1909	1990			DP	1927	14	1965	0	0.01000	0.0	0.0	0	0
01-482	F	1912	1990			DP	1927	6	1979	1	0.00181	1.0	1.0	11	12
01-483	M	1907	1990			DP	1922	104	1975	0	0.01184	0.0	0.0	0	0
01-484	F	1908		1974	1830	DP	1926	0	1965	0	0.01000	0.0	0.0	0	0
01-485	M	1870		1951	4319	RI	1911	1300	1965	340	0.0	92.3	0.0	601	0
01-486	F	1907		1987		DP	1923	6	1974	0	0.01318	0.0	0.0	0	0
01-487	F	1911	1990			DH	1927	565	1976	0	0.00257	0.0	0.0	0	0
01-489	F	1910	1981			DP	1926	348	1965	225	0.01000	82.1	56.9	1004	782
01-490	F	1908	1990			DP	1924	17	1974	2	0.01318	1.9	9.8	20	125
01-491	F	1922		1966	2001	DP	1943	728	1963	7	0.0	1.2	0.0	7	0
01-492	F	1900		1985	4123	CR	1921	260	1973	1	0.0	0.9	0.0	10	0
01-493	M	1893		1975	4379	CR	1927	1820	1973	4	0.0	1.4	0.0	8	0
01-494	M	1906		1966	4109	CR	1926	999	1966	0	0.0	0.0	0.0	0	0
01-495	F	1908	1990			DP	1924	4	1965	0	0.03900	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-496	F	1918	1990			DH	1934	106	1966	3	0.0	1.4	0.0	14	0
01-497	F	1902		1978	4379	DP	1921	8	1966	13	0.03400	8.6	64.1	95	840
01-498	F	1897		1982	174	CR	1920	104	1981	0	0.0	0.0	0.0	0	0
01-501	M	1867		1937	1538	RD	1926	156	1966	2500	0.00760	221.9	169.7	924	1330
01-503	M	1936	1973			OF	1936	39	1966	0	0.0	0.0	0.0	0	0
01-504	F	1913	1990			DP	1927	2	1975	0	0.0	0.0	0.0	0	0
01-505	F	1902	1990			DP	1927	1	1966	9	0.00880	5.7	5.4	60	69
01-506	F	1897	1990			RW	1923	4	1966	7	0.0	5.2	0.0	55	0
01-507	F	1909	1990			DP	1927	22	1982	6	0.00119	6.4	5.5	67	70
01-508	F	1906		1968	5718	DP	1944	52	1966	30	0.0	8.9	0.0	68	0
01-509	F	1943	1977			OF	1943	39	1967	0	0.0	0.0	0.0	0	0
01-510	F	1897		1990		DP	1927	12	1966	38	0.00880	22.2	20.8	242	269
01-511	F	1908	1990			DH	1927	9	1979	0	0.00181	0.0	0.0	0	0
01-512	F	1895		1976	4369	RW	1912	13	1973	0	0.0	0.0	0.0	0	0
01-514	F	1904	1990			DH	1924	2184	1981	2	0.00097	0.8	0.0	7	0
01-515	F	1886		1980	4274	RI	1940	0	1966	4	0.0	1.5	0.0	14	0
01-516	F	1907		1976	5621	DP	1927	2	1967	7	0.00780	4.6	4.4	46	56
01-518	M	1912		1988		RI	1949	+0	1977	0	0.0	0.0	0.0	0	0
01-519	M	1919	1990			CR	1937	260	1967	13	0.0	5.2	0.0	37	0
01-520	F	1882		1969	1701	RD	1930	+0	1967	670	0.00492	163.7	68.3	2162	1008
01-525	M	1923	1990			CR	1943	104	1968	17	0.0	5.7	0.0	40	0
01-526	M	1921	1990			CR	1945	38	1979	29	0.0	14.8	0.0	102	0
01-529	M	1920	1990			CR	1943	260	1975	14	0.0	6.1	0.0	42	0
01-530	M	1920		1971	1621	CR	1943	104	1968	52	0.0	17.2	0.0	100	0
01-531	M	1918		1988		CR	1941	354	1974	13	0.0	5.6	0.0	38	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-533	F	1903		1978	1541	RW	1911	+0	1969	4	0.0	5.0	0.0	53	0
01-534	M	1920	1990			CR	1944	154	1976	1	0.0	0.5	0.0	3	0
01-536	M	1916	1990			CR	1943	286	1968	17	0.0	5.2	0.0	36	0
01-537	M	1917		1971	2072	CR	1944	208	1968	59	0.0	17.6	0.0	99	0
01-540	M	1890		1981	4123	DB	1940	260	1968	0	0.0	0.0	0.0	0	0
01-543	M	1920		1976	1990	CR	1943	167	1975	19	0.0	8.5	0.0	52	0
01-544	F	1879		1953	4121	RD	1930	+0	1968	93	0.00430	28.4	11.7	219	148
01-546	F	1897		1980	4123	DP	1914	52	1967	0	0.0	0.0	0.0	0	0
01-547	F	1897		1989		RA	1920	104	1979	4	0.0	5.0	0.0	53	0
01-548	M	1917		1988	1618	RD	1930	+0	1972	5	0.00200	3.6	1.1	26	10
01-552	M	1907		1984	4109	CR	1936	104	1967	20	0.0	8.8	0.0	62	0
01-553	F	1910	1967			DP	1948	988	1967	0	0.0	0.0	0.0	0	0
01-554	F	1928	1967			DP	1952	780	1967	490	0.0	37.3	0.0	150	0
01-555	F	1894	1990			DP	1921	2	1975	0	0.01155	0.0	0.0	0	0
01-556	F	1910	1990			DP	1927	0	1967	0	0.00780	0.0	0.0	0	0
01-557	F	1908		1988		DP	1925	35	1975	2	0.00293	1.9	2.1	20	27
01-558	M	1913	1990			RD	1927	130	1981	292	0.00042	143.7	33.8	1443	340
01-562	F	1901		1931	946E	DP	1920	52	1970	10300	0.00032	948.5	116.5	5976	1365
01-565	F	1892		1957	4123	RI	1925	26	1970	1600	0.0	280.3	0.0	3602	0
01-567	M	1885		1949	188	RD	1925	+0	1970	1100	0.00400	158.7	140.6	1233	1500
01-568	M	1907		1928	201	RI	1926	+0	1969	4900	0.0	207.9	0.0	226	0
01-569	F	1896		1980	4369	DH	1922	282	1978	4	0.00804	4.0	18.8	42	242
01-570	F	1908	1990			DP	1926	260	1968	10	0.0	6.5	0.0	68	0
01-571	F	1911	1990			DP	1928	44	1979	0	0.00181	0.0	0.0	0	0
01-573	F	1892		1945	1600	DP	1916	312	1970	670	0.00195	121.3	104.4	1291	1520

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-574	F	1885		1937	1519	RI	1924	77	1968	2730	0.0	285.9	0.0	1957	0
01-575	M	1910		1977	4100	DP	1950	1196	1973	2	0.0	0.2	0.0	1	0
01-576	F	1930	1984			DP	1946	780	1968	160	0.0	28.3	0.0	244	0
01-578	F	1904		1930	201	RI	1926	17	1969	3700	0.0	235.3	0.0	701	0
01-579	F	1928		1928	7700	OF	1928	26	1973	2	0.00289	0.0	0.0	1	0
01-580	F	1894		1986	2901	DP	1918	52	1972	1	0.0	1.1	0.0	11	0
01-581	M	1918		1988		CR	1946	52	1968	10	0.0	3.0	0.0	20	0
01-582	F	1893		1988	4123	CR	1917	24	1979	1	0.0	1.4	0.0	15	0
01-583	M	1890		1969	4409	CR	1918	104	1968	0	0.00250	0.0	0.0	0	0
01-584	F	1908		1975	1579	DP	1926	260	1968	10	0.0	6.5	0.0	63	0
01-585	F	1906		1969	4319	DP	1925	26	1968	0	0.00450	0.0	0.0	0	0
01-586	F	1879		1973	4379	RI	1924	+0	1968	130	0.0	81.1	0.0	866	0
01-588	F	1908	1990			DP	1929	104	1968	5	0.0	3.1	0.0	32	0
01-589	M	1907	1990			CR	1927	78	1978	1	0.0	1.0	0.0	7	0
01-590	M	1929	1978			OF	1929	39	1976	0	0.01062	0.0	0.0	0	0
01-591	F	1891		1975	4123	DP	1918	52	1973	0	0.00016	0.0	0.0	0	0
01-592	F	1903		1971	4123	DP	1917	6	1968	0	0.0	0.0	0.0	0	0
01-594	M	1926	1990			DP	1962	34	1975	2	0.0	0.3	0.0	2	0
01-595	F	1897		1988		DP	1917	130	1969	5	0.0	4.9	0.0	52	0
01-597	F	1923	1990			DP	1940	364	1982	0	0.0	0.0	0.0	0	0
01-598	M	1879		1953	517	CR	1941	572	1952	400	0.0	25.6	0.0	69	0
01-599	F	1909	1990			DP	1927	7	1978	0	0.00203	0.0	0.0	0	0
01-601	F	1902		1990		DP	1918	6	1969	0	0.00020	0.0	0.0	0	0
01-603	F	1894		1985	1538	DP	1915	676	1968	7	0.00450	5.9	7.1	63	91
01-604	F	1896	1990			DP	1914	52	1971	1	0.0	1.2	0.0	13	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-607	F	1907	1990			DH	1927	+0	1978	0	0.00203	0.0	0.0	0	0
01-608	F	1906		1976	4109	DP	1927	11	1974	0	0.00330	0.0	0.0	0	0
01-609	F	1906		1988		DP	1926	366	1978	1	0.0	0.9	0.0	9	0
01-610	M	1904		1969	4109	CR	1919	208	1968	10	0.00450	8.3	10.3	58	94
01-612	F	1859		1936	428	DP	1923	255	1972	18	0.00680	2.7	4.8	14	49
01-613	F	1906		1936	1706	DP	1923	265	1972	658	0.00680	72.4	125.9	419	1389
01-614	M	1882		1922	284	CR	1920	+0	1974	24	0.0	1.2	0.0	2	0
01-617	M	1922		1990		OF	1922	39	1973	1	0.00020	1.0	0.1	7	1
01-619	F	1909		1978	4109	DP	1927	52	1969	0	0.0	0.0	0.0	0	0
01-621	F	1908	1990			DP	1924	2	1978	8	0.00791	7.7	39.6	85	513
01-625	F	1911		1982	4109	DP	1927	468	1968	6	0.0	3.5	0.0	34	0
01-626	F	1932	1976			OF	1932	39	1971	0	0.0	0.0	0.0	0	0
01-627	F	1897		1986		DP	1917	52	1970	0	0.0	0.0	0.0	0	0
01-628	F	1908	1990			DP	1925	312	1975	0	0.00200	0.0	0.0	0	0
01-629	F	1892		1977	4123	DP	1926	260	1969	12	0.0	8.1	0.0	79	0
01-633	F	1878		1926	2059	RI	1925	4	1970	2600	0.0	92.2	0.0	98	0
01-635	M	1880		1937	404	CR	1918	312	1973	1900	0.0	241.3	0.0	1378	0
01-636	F	1879		1930	582	DP	1919	1	1979	1	0.00075	0.1	0.1	1	1
01-640	F	1908	1990			DP	1924	21	1969	34	0.00420	24.2	22.0	268	285
01-653	F	1910	1990			DP	1925	78	1969	7	0.00420	5.3	4.1	55	52
01-659	F	1912	1990			DP	1928	26	1969	11	0.0	7.5	0.0	78	0
01-660	F	1881		1957	4122	RD	1932	+0	1970	15	0.0	5.3	0.0	42	0
01-661	M	1874		1934	4109	CR	1914	572	1974	2	0.0	0.3	0.0	1	0
01-663	M	1927		1984	4109	OF	1927	39	1969	11	0.0	7.7	0.0	57	0
01-665	M	1923	1975			OF	1923	39	1969	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-667	F	1918	1990			DP	1941	234	1972	0	0.0	0.0	0.0	0	0
01-668	M	1933	1990			KH	1964	+0	1974	1	0.0	0.2	0.0	1	0
01-669	F	1917	1990			DP	1934	130	1980	0	0.0	0.0	0.0	0	0
01-670	M	1897	1975			RW	1928	+0	1969	0	0.0	0.0	0.0	0	0
01-671	F	1923	1990			DP	1941	260	1972	2	0.0	0.8	0.0	8	0
01-674	M	1908	1990			DP	1931	1716	1980	1	0.0	0.2	0.0	1	0
01-681	F	1904		1978	4369	DH	1920	4	1972	0	0.00320	0.0	0.0	0	0
01-684	F	1894		1974	486	DP	1917	1	1973	0	0.0	0.0	0.0	0	0
01-688	M	1868		1948	1419	DR	1920	+0	1972	0	0.00320	0.0	0.0	0	0
01-690	M	1878		1940	1420	RW	1918	+0	1970	21	0.0	6.5	0.0	34	0
01-691	F	1913		1974	1990	RW	1935	0	1971	0	0.0	0.0	0.0	0	0
01-692	M	1885		1974	4442	RD	1925	+0	1970	30	0.00680	21.1	31.8	156	295
01-694	M	1886		1953	4109	RI	1928	+0	1971	10000	0.0	1316.5	0.0	10997	0
01-701	M	1892		1974	188	CR	1916	312	1970	0	0.0	0.0	0.0	0	0
01-706	F	1908	1990			DR	1923	100	1980	1	0.00629	1.5	7.9	16	101
01-707	F	1908		1974	1830	DP	1927	1	1971	0	0.00470	0.0	0.0	0	0
01-710	F	1901		1986	5932	DP	1925	289	1978	0	0.00141	0.0	0.0	0	0
01-711	F	1905		1982	4124	DP	1925	312	1970	0	0.00370	0.0	0.0	0	0
01-715	F	1907		1990		DP	1927	5	1976	0	0.00258	0.0	0.0	0	0
01-717	M	1910	1990			RD	1927	13	1979	3	0.00230	3.0	3.6	23	32
01-727	F	1908	1990			DP	1920	1	1982	4	0.00092	5.7	8.9	61	113
01-728	F	1912		1989		DP	1927	6	1978	0	0.00203	0.0	0.0	0	0
01-731	F	1905	1990			DP	1926	8	1979	3	0.00200	3.1	3.6	33	46
01-733	F	1911	1990			DP	1927	61	1978	45	0.00200	35.9	30.5	403	398
01-736	F	1907		1931	670	DP	1923	52	1977	1	0.00170	0.1	0.1	0	1

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
01-739	F	1856		1928	201	RI	1926	7	1972	11500	0.0	524.2	0.0	961	0
03-005	M	1917		1978	1729	RA	1948	+0	1973	0	0.0	0.0	0.0	0	0
03-008	F	1934		1990	4121	OF	1934	39	1971	0	0.0	0.0	0.0	0	0
03-009	F	1918	1990			DP	1941	104	1972	1	0.0	0.4	0.0	3	0
03-101	F	1908		1971	1732	RI	1931	15	1963	1580	0.0	278.3	0.0	4106	0
03-102	M	1908		1976	4124	RI	1931	15	1973	628	0.0	205.1	0.0	1927	0
03-103	F	1868		1952	428	RI	1931	15	1951	420	0.0	97.6	0.0	755	0
03-104	F	1880		1945	5719	RI	1931	15	1931	13900	0.0	400.1	0.0	3051	0
03-105	M	1903		1957	2262	RI	1931	16	1951	2600	0.0	327.0	0.0	2709	0
03-106	F	1876		1959	1707	RI	1931	16	1931	4600	0.0	134.4	0.0	1239	0
03-107	F	1884		1957	4123	RI	1931	16	1931	3600	0.0	105.5	0.0	909	0
03-108	F	1875		1953	428	RI	1931	16	1932	1900	0.0	72.3	0.0	560	0
03-109	F	1904		1957	188	RI	1931	18	1953	630	0.0	143.3	0.0	1290	0
03-110	F	1899		1967	4109	RI	1931	20	1964	584	0.0	175.7	0.0	1943	0
03-111	F	1909	1978			RI	1931	20	1973	879	0.0	234.0	0.0	3428	0
03-112	F	1899		1968	4109	RI	1931	26	1960	5310	0.0	737.0	0.0	11311	0
03-113	F	1914		1946	0119	RI	1931	38	1932	7800	0.0	284.6	0.0	2200	0
03-114	F	1901		1968	4109	RI	1931	36	1964	949	0.0	221.0	0.0	2756	0
03-115	F	1911	1990			RI	1931	26	1973	745	0.0	219.3	0.0	3415	0
03-116	F	1907		1983	4123	RI	1931	25	1983	931	0.0	255.7	0.0	4205	0
03-117	M	1898		1957	963E	RI	1931	45	1953	1540	0.0	240.0	0.0	1818	0
03-118	F	1898		1955	1621	RI	1931	41	1953	3090	0.0	390.7	0.0	4329	0
03-119	F	1880		1960	485	RI	1931	7	1959	1038	0.0	217.9	0.0	2366	0
03-120	F	1879		1937	402	RI	1931	11	1931	5300	0.0	155.7	0.0	603	0
03-121	F	1911		1972	5931	RI	1931	9	1964	371	0.0	133.5	0.0	1446	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-122	M	1907		1981	5719	RI	1931	10	1931	6500	0.0	97.7	0.0	768	0
03-123	M	1914		1937	0189	RI	1931	9	1931	9700	0.0	147.0	0.0	405	0
03-124	M	1910		1983	0092	RI	1931	9	1981	235	0.0	138.6	0.0	1180	0
03-125	F	1913		1976	4510	RI	1931	11	1973	556	0.0	194.8	0.0	2490	0
03-126	F	1910		1965	1608	RI	1931	20	1965	1300	0.0	257.3	0.0	3320	0
03-127	F	1908		1989	4109	RI	1931	26	1962	565	0.0	166.0	0.0	2182	0
03-135	M	1905		1983	1538	RI	1931	+0	1973	1431	0.0	289.6	0.0	3647	0
03-139	M	1908		1980	485	RI	1933	11	1973	373	0.0	154.2	0.0	1305	0
03-140	M	1905		1937	0119	RI	1933	11	1961	500	0.0	36.3	0.0	74	0
03-141	M	1906		1963	1959	RI	1933	11	1962	961	0.0	213.0	0.0	1664	0
03-201	F	1909		1963	1709	RW	1922	+0	1962	2968	0.0	483.5	0.0	7956	0
03-202	M	1892		1984	5969	RI	1925	+0	1960	1800	0.0	310.4	0.0	4342	0
03-203	F	1903		1973	402	RI	1933	+0	1959	84	0.0	29.9	0.0	283	0
03-204	F	1896		1970	4123	RW	1922	+0	1960	21	0.0	12.8	0.0	126	0
03-205	F	1900		1979	4124	RI	1929	15	1968	291	0.0	129.4	0.0	1496	0
03-206	M	1914		1975	1700	RI	1936	4	1973	3297	0.0	515.6	0.0	5862	0
03-207	F	1879		1969	4339	RW	1922	416	1960	755	0.0	193.2	0.0	2537	0
03-209	M	1894		1960	1709	RI	1925	572	1973	1105	0.0	215.3	0.0	1767	0
03-210	M	1906		1958	1708	RI	1926	+0	1957	1350	0.00089	250.2	9.2	2236	98
03-211	M	1890	1960			RI	1923	20	1960	10	0.0	5.9	0.0	38	0
03-212	F	1902		1951	1959	RW	1927	+0	1951	1300	0.00130	226.9	5.3	2276	75
03-213	F	1892		1955	1706	RI	1925	+0	1952	6570	0.0	887.0	0.0	11802	0
03-214	F	1895		1966	1601	RI	1925	+0	1964	1382	0.0	277.7	0.0	4164	0
03-215	M	1896		1971	7960	RI	1925	+0	1961	3630	0.0	558.6	0.0	7146	0
03-216	F	1907		1961	1709	RI	1922	+0	1961	530	0.0	184.0	0.0	2148	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-217	M	1912		1974	4310	RI	1921	+0	1963	460	0.0	178.6	0.0	1672	0
03-218	M	1908	1990			RI	1924	+0	1972	3	0.0	3.0	0.0	22	0
03-219	F	1888		1961	4319	RW	1919	+0	1951	60	0.0	27.8	0.0	267	0
03-220	M	1920		1983	161	RW	1928	208	1976	130	0.0	83.7	0.0	661	0
03-221	M	1908		1963	1419	RI	1924	+0	1957	620	0.0	182.4	0.0	1516	0
03-222	M	1872		1954	4109	RI	1922	+0	1951	1600	0.0	271.4	0.0	2474	0
03-223	F	1886		1968	4379	RI	1929	156	1951	4200	0.0	507.5	0.0	7865	0
03-224	M	1869		1960	1578	RI	1922	364	1951	5400	0.0	710.6	0.0	7449	0
03-225	M	1922		1984	1601	RW	1929	+0	1980	28	0.0	25.6	0.0	189	0
03-226	M	1874		1953	284	RI	1934	39	1951	10700	0.0	1175.6	0.0	7966	0
03-227	F	1878		1952	1709	RI	1930	+0	1952	1000	0.0	193.5	0.0	1724	0
03-228	M	1900		1955	4123	RI	1927	+0	1951	5600	0.0	719.6	0.0	6511	0
03-230	F	1899		1982	486	RI	1927	+0	1976	438	0.0	189.4	0.0	2594	0
03-231	F	1879		1973	428	RI	1939	+0	1952	60	0.0	10.6	0.0	94	0
03-232	F	1898		1957	1732	RI	1917	+0	1956	4700	0.0	743.8	0.0	12118	0
03-233	F	1879		1947	4109	RI	1922	+0	1947	4000	0.0	526.5	0.0	6138	0
03-234	F	1890		1965	1707	RI	1915	+0	1965	920	0.0	252.6	0.0	4031	0
03-235	F	1900		1968	1700	RI	1928	+0	1965	1290	0.0	264.2	0.0	3813	0
03-236	F	1880		1961	4123	RI	1927	+0	1951	500	0.0	129.8	0.0	1297	0
03-237	F	1890		1983	4379	RW	1923	156	1961	3	0.0	1.8	0.0	18	0
03-238	M	1883		1954	4109	RI	1926	+0	1951	13900	0.0	1815.6	0.0	16435	0
03-239	F	1883		1953	1829	RI	1925	+0	1970	10000	0.0	1381.1	0.0	17502	0
03-240	F	1916		1955	1601	RI	1930	+0	1973	4320	0.0	571.4	0.0	6670	0
03-401	F	1900		1963	1621	DP	1923	95	1960	2287	0.0	370.9	0.0	5797	0
03-402	F	1905		1982	1601	DP	1923	260	1974	1223	0.00008	275.2	8.5	5154	132

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-403	F	1915		1964	1830	DP	1935	572	1957	8	0.0	1.7	0.0	13	0
03-404	F	1897		1985	4123	DP	1923	195	1975	577	0.0	209.9	0.0	3272	0
03-405	F	1904		1984	1830	DP	1924	273	1962	625	0.0	183.3	0.0	2602	0
03-406	F	1914	1990			DP	1935	484	1980	3	0.0	2.3	0.0	23	0
03-407	F	1905		1961	1601	DP	1923	1196	1958	1545	0.00019	275.6	2.9	3882	44
03-408	F	1908		1959	191	DP	1924	676	1957	160	0.0	67.4	0.0	626	0
03-409	F	1923	1990			DP	1942	78	1972	8	0.0	3.6	0.0	35	0
03-410	F	1895		1974	4109	DP	1923	104	1957	60	0.0	28.8	0.0	292	0
03-411	F	1908	1990			DP	1931	572	1976	1	0.0	0.9	0.0	9	0
03-412	F	1894		1983	4274	DP	1922	134	1977	227	0.0	142.1	0.0	1804	0
03-413	F	1917		1978	4109	DP	1939	169	1972	4	0.0	1.7	0.0	15	0
03-414	F	1921	1990			DP	1946	557	1972	3	0.0	0.8	0.0	7	0
03-415	F	1911		1973	4109	DP	1930	780	1957	15	0.0	3.8	0.0	34	0
03-416	F	1907		1983	1538	DP	1923	65	1981	1097	0.0	281.1	0.0	5303	0
03-417	F	1909		1966	1990	DP	1924	60	1964	617	0.0	197.1	0.0	2459	0
03-418	F	1896		1980	486	DP	1926	602	1972	5	0.0	3.1	0.0	31	0
03-419	F	1906		1988		DP	1924	208	1962	679	0.0	199.0	0.0	3007	0
03-420	F	1906		1960	1538	DP	1922	212	1957	18	0.0	9.0	0.0	81	0
03-421	F	1908	1990			DP	1924	117	1979	3	0.0	3.3	0.0	35	0
03-422	F	1907	1990			CR	1925	104	1981	4	0.0	4.8	0.0	50	0
03-423	F	1907		1972	1601	DP	1923	641	1962	591	0.0	186.4	0.0	2431	0
03-424	F	1905	1990			DP	1923	186	1978	245	0.0	147.3	0.0	1953	0
03-425	F	1916	1990			DP	1935	293	1980	3	0.0	1.8	0.0	18	0
03-426	F	1906		1985	4123	DP	1924	1196	1981	131	0.0	105.3	0.0	1243	0
03-427	F	1906	1990			DP	1925	823	1973	12	0.0	10.7	0.0	113	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-428	F	1908		1989		DP	1925	164	1974	493	0.0	196.8	0.0	2939	0
03-429	F	1908		1976	191	DP	1923	208	1974	1169	0.0	273.7	0.0	4722	0
03-430	F	1922	1990			DP	1941	468	1971	4	0.0	1.6	0.0	15	0
03-431	F	1901		1982	4123	DP	1922	156	1963	1297	0.0	267.1	0.0	4894	0
03-432	F	1902		1990		DP	1923	112	1980	23	0.0	25.5	0.0	276	0
03-433	F	1904		1984	1732	DP	1924	117	1964	1052	0.0	243.5	0.0	4180	0
03-434	F	1920	1990			DP	1941	125	1975	5	0.0	2.4	0.0	24	0
03-435	F	1912		1986	4109	DP	1934	104	1971	3	0.0	1.5	0.0	15	0
03-436	F	1910	1990			DP	1926	619	1975	8	0.0	6.2	0.0	63	0
03-437	F	1906	1990			DP	1926	52	1957	55	0.0	24.1	0.0	258	0
03-438	F	1908	1990			DP	1925	8	1957	0	0.0	0.0	0.0	0	0
03-439	F	1906	1990			DP	1925	56	1957	0	0.0	0.0	0.0	0	0
03-440	F	1908	1990			DP	1925	3	1979	1	0.0	0.8	0.0	8	0
03-441	F	1905		1981	4123	DP	1925	528	1957	56	0.0	25.5	0.0	265	0
03-442	F	1904	1990			DP	1924	13	1976	4	0.0	4.0	0.0	42	0
03-443	F	1914	1990			DP	1935	268	1980	0	0.0	0.0	0.0	0	0
03-444	F	1907	1990			DP	1925	56	1980	12	0.0	13.2	0.0	140	0
03-445	F	1905		1974	174	DP	1922	260	1966	1367	0.0	278.7	0.0	4782	0
03-446	F	1903	1990			DP	1922	260	1980	56	0.0	55.1	0.0	619	0
03-447	F	1906		1981	4319	DP	1924	4	1958	2	0.0	1.1	0.0	11	0
03-448	F	1903		1963	1519	DP	1924	19	1958	25	0.0	12.9	0.0	119	0
03-449	F	1905		1974	4109	DP	1922	1456	1964	1135	0.0	250.9	0.0	4078	0
03-450	F	1910	1990			DP	1924	697	1979	8	0.0	8.1	0.0	86	0
03-451	F	1922	1990			DP	1940	524	1982	0	0.0	0.0	0.0	0	0
03-452	F	1909		1988	4109	DP	1925	728	1983	16	0.0	15.3	0.0	157	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-453	F	1907	1990			DP	1924	8	1981	3	0.0	3.8	0.0	39	0
03-454	F	1914		1982	1712	CR	1934	548	1958	48	0.0	11.8	0.0	113	0
03-455	F	1906		1988		DP	1922	56	1975	491	0.00059	170.5	54.3	2859	800
03-456	F	1921		1965	174	DP	1942	470	1958	33	0.0	5.1	0.0	35	0
03-457	F	1915	1990			DP	1939	520	1972	1	0.0	0.4	0.0	4	0
03-458	F	1925	1990			DP	1946	1664	1982	20	0.0	4.3	0.0	34	0
03-459	F	1906		1980	1608	DP	1924	43	1976	774	0.0	238.4	0.0	3876	0
03-460	F	1905		1990	2000	DP	1923	19	1977	4	0.0	4.1	0.0	44	0
03-461	F	1896		1989	4123	DP	1922	6	1958	6	0.0	3.3	0.0	34	0
03-462	F	1906	1990			DP	1922	2912	1981	217	0.0	143.5	0.0	1900	0
03-463	F	1918		1966	4309	DP	1942	832	1958	33	0.0	2.7	0.0	17	0
03-464	F	1907	1990			DP	1923	104	1974	0	0.0	0.4	0.0	4	0
03-465	F	1908	1990			DP	1925	8	1981	0	0.0	0.5	0.0	5	0
03-466	F	1904	1990			DP	1924	10	1981	3	0.0	3.2	0.0	33	0
03-467	F	1911	1990			DP	1926	416	1981	7	0.0	6.6	0.0	69	0
03-468	F	1908	1990			DP	1926	121	1958	29	0.0	13.2	0.0	139	0
03-469	F	1903		1960	4109	DP	1925	30	1958	10	0.0	5.0	0.0	44	0
03-470	F	1926	1990			DP	1943	247	1971	3	0.0	1.2	0.0	12	0
03-471	F	1908		1981	4123	DP	1926	91	1958	13	0.0	6.1	0.0	61	0
03-472	F	1922		1989	4109	DP	1941	247	1972	5	0.0	2.2	0.0	21	0
03-473	F	1904		1965	4109	DP	1922	156	1962	1170	0.0	252.5	0.0	3685	0
03-474	F	1909	1990			DP	1925	21	1958	19	0.0	9.5	0.0	100	0
03-475	F	1903		1962	583	DP	1921	65	1958	0	0.0	0.0	0.0	0	0
03-476	F	1895		1970	4270	DP	1927	6	1958	0	0.0	0.0	0.0	0	0
03-477	F	1911	1990			DP	1925	11	1982	1	0.0	1.3	0.0	14	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-478	F	1907	1990			DP	1924	8	1958	5	0.0	2.6	0.0	28	0
03-479	F	1908	1990			DP	1924	52	1981	29	0.00001	31.3	0.3	340	4
03-480	F	1909		1987		DP	1924	10	1980	0	0.0	0.0	0.0	0	0
03-481	F	1922	1990			DP	1942	481	1982	10	0.0	5.6	0.0	53	0
03-482	F	1927	1990			DP	1945	83	1972	3	0.0	1.0	0.0	9	0
03-483	F	1901		1987	174	DP	1922	177	1980	1	0.0	0.6	0.0	6	0
03-484	F	1888		1966	4109	DP	1919	156	1962	1622	0.0	306.6	0.0	5118	0
03-485	F	1909		1977	4123	DP	1929	364	1958	0	0.0	0.0	0.0	0	0
03-486	F	1909	1990			DP	1925	156	1977	208	0.0	131.8	0.0	1666	0
03-487	F	1907		1964	2041	DP	1924	676	1958	367	0.00251	126.3	16.4	1370	224
03-488	F	1907		1975	1601	DP	1922	26	1958	170	0.00201	78.0	11.3	862	149
03-489	F	1911		1964	1519	DP	1926	73	1958	120	0.0	51.2	0.0	484	0
03-490	M	1904		1981	4109	DH	1925	177	1973	5	0.0	3.9	0.0	28	0
03-491	F	1908		1984		DP	1924	2	1979	19	0.0	20.3	0.0	213	0
03-492	F	1928	1990			DP	1946	325	1973	5	0.0	1.5	0.0	14	0
03-493	F	1893		1990		DP	1920	199	1980	4	0.0	5.1	0.0	54	0
03-494	F	1902		1982	4109	DP	1924	177	1959	4	0.0	2.1	0.0	22	0
03-495	F	1910		1980	5932	DP	1923	7	1976	0	0.0	0.4	0.0	4	0
03-496	F	1907		1988		DP	1923	8	1981	1	0.0	1.0	0.0	11	0
03-497	F	1903		1970	7824	DP	1923	260	1959	16	0.0	8.5	0.0	82	0
03-498	F	1905	1990			DH	1923	1040	1976	2	0.0	1.3	0.0	13	0
03-499	F	1905	1990			DP	1924	56	1978	185	0.00175	89.4	95.9	1302	1358
03-500	F	1901		1959	2509	DP	1922	8	1959	0	0.0	0.0	0.0	0	0
03-501	F	1912	1990			DP	1928	8	1959	7	0.0	3.3	0.0	34	0
03-502	F	1887		1964	422	DP	1918	156	1959	170	0.0	88.1	0.0	925	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-503	F	1894		1960	4109	DP	1922	112	1959	125	0.0	62.0	0.0	590	0
03-504	F	1905	1990			DP	1922	30	1981	8	0.0	10.1	0.0	107	0
03-505	F	1907		1976	4310	DP	1923	1300	1975	169	0.0	111.9	0.0	1279	0
03-506	F	1917	1990			DP	1935	1872	1975	9	0.0	2.2	0.0	19	0
03-507	F	1907		1962	2251	DP	1923	6	1959	12	0.0	6.8	0.0	63	0
03-508	F	1905		1963	4109	DP	1923	8	1959	10	0.0	5.7	0.0	53	0
03-509	F	1907	1990			DP	1924	2548	1973	28	0.0	23.4	0.0	251	0
03-510	F	1907		1977	174	DP	1923	2028	1962	729	0.0	205.1	0.0	2953	0
03-511	F	1910		1979	4339	DP	1946	673	1959	10	0.0	0.8	0.0	6	0
03-512	F	1906	1990			DP	1925	26	1959	11	0.0	5.7	0.0	60	0
03-513	F	1908	1990			DP	1925	48	1974	73	0.0	57.1	0.0	637	0
03-514	F	1909	1990			DP	1925	208	1959	26	0.0	13.2	0.0	140	0
03-515	F	1908	1990			DP	1925	91	1983	4	0.0	4.8	0.0	50	0
03-516	F	1911		1989		DP	1925	290	1981	4	0.0	4.8	0.0	51	0
03-517	F	1922	1990			DP	1943	260	1972	1	0.0	0.2	0.0	2	0
03-518	F	1921	1990			DP	1940	464	1972	8	0.0	3.0	0.0	29	0
03-519	F	1903		1987	4109	DP	1924	8	1959	98	0.0	48.7	0.0	534	0
03-520	F	1907	1990			DP	1925	780	1974	112	0.0	80.1	0.0	924	0
03-521	F	1907		1961	4123	DP	1925	39	1959	10	0.0	5.2	0.0	46	0
03-522	F	1898		1989		DP	1921	52	1981	85	0.0	83.4	0.0	978	0
03-523	F	1900	1990			DP	1923	30	1980	8	0.0	8.9	0.0	94	0
03-524	F	1903	1990			DP	1925	260	1972	48	0.0	36.9	0.0	401	0
03-525	F	1911		1976	3959	DP	1931	2132	1959	19	0.0	2.2	0.0	16	0
03-526	F	1896		1981	4109	DP	1925	52	1959	0	0.0	0.0	0.0	0	0
03-527	F	1909	1990			DP	1925	130	1959	5	0.0	2.6	0.0	27	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-528	F	1904		1988	1713	DP	1922	524	1959	1630	0.00327	270.2	57.6	5713	900
03-529	F	1902		1989		DP	1921	104	1980	73	0.0	71.5	0.0	823	0
03-530	F	1907		1965	398	DP	1923	91	1963	474	0.0	172.2	0.0	2024	0
03-531	F	1906		1990		DP	1925	403	1959	41	0.0	20.5	0.0	219	0
03-532	F	1910	1990			DP	1926	190	1980	44	0.0	40.2	0.0	436	0
03-533	F	1908	1990			DP	1925	260	1979	12	0.0	12.4	0.0	131	0
03-534	F	1910	1990			DP	1925	104	1976	3	0.0	3.2	0.0	34	0
03-535	F	1907	1990			DP	1922	21	1964	227	0.0	116.4	0.0	1443	0
03-536	F	1910	1990			DP	1925	7	1959	35	0.0	17.9	0.0	191	0
03-537	F	1900		1987		DH	1916	52	1977	1	0.0	1.5	0.0	16	0
03-538	F	1909		1976	4100	DP	1927	13	1959	61	0.0	28.1	0.0	282	0
03-539	F	1900		1984	4123	DP	1922	20	1979	5	0.0	5.6	0.0	59	0
03-540	F	1904	1990			DP	1923	364	1973	1605	0.0	321.9	0.0	6857	0
03-541	F	1913	1990			DP	1935	178	1978	0	0.0	0.0	0.0	0	0
03-542	F	1904	1990			DP	1922	13	1981	22	0.0	26.3	0.0	285	0
03-543	F	1918	1990			DP	1947	117	1982	0	0.0	0.2	0.0	1	0
03-544	F	1906		1975	4379	DP	1922	26	1959	5	0.0	3.0	0.0	30	0
03-545	F	1898		1981	7963	DP	1920	208	1959	0	0.0	0.0	0.0	0	0
03-546	F	1903		1981	4360	DP	1925	52	1959	95	0.0	45.0	0.0	476	0
03-547	F	1907		1962	1540	DP	1923	108	1959	19	0.00310	10.2	2.1	94	27
03-548	F	1906	1990			DP	1922	17	1971	80	0.0	62.0	0.0	704	0
03-549	F	1910	1990			DP	1925	936	1980	41	0.0	40.9	0.0	447	0
03-550	F	1900	1990			DP	1917	104	1980	8	0.0	11.4	0.0	123	0
03-551	F	1903		1989	486	DP	1922	338	1973	1077	0.0	261.7	0.0	5047	0
03-552	F	1904	1990			DP	1924	108	1978	114	0.0	90.1	0.0	1060	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-553	F	1904		1986	4389	DP	1924	13	1979	6	0.0	7.2	0.0	75	0
03-554	F	1899		1977	2509	DP	1924	433	1961	2000	0.0	335.2	0.0	6224	0
03-555	F	1913		1978	2022	DP	1928	364	1972	2	0.0	1.6	0.0	16	0
03-556	F	1911	1990			DP	1928	100	1981	0	0.0	0.4	0.0	4	0
03-557	F	1910		1978	4109	DP	1925	3	1959	0	0.0	0.0	0.0	0	0
03-558	F	1904		1971	174	DP	1923	13	1959	115	0.02173	47.4	76.3	535	1029
03-559	F	1907		1975	4100	DP	1922	21	1959	17	0.0	9.9	0.0	100	0
03-561	F	1909	1990			DP	1924	416	1959	67	0.0	33.2	0.0	360	0
03-562	F	1908		1980	4409	DP	1927	520	1972	4	0.0	2.4	0.0	23	0
03-563	F	1909		1985	2509	DP	1924	10	1980	4	0.0	5.1	0.0	53	0
03-564	F	1906	1990			DP	1923	3	1981	2	0.0	1.9	0.0	20	0
03-565	F	1913		1979	4272	DP	1930	676	1978	7	0.0	5.1	0.0	48	0
03-566	F	1910	1990			DP	1930	624	1978	2	0.0	1.5	0.0	15	0
03-567	F	1900		1990		DP	1922	104	1972	26	0.0	22.5	0.0	242	0
03-568	F	1905		1977	1621	DP	1922	260	1959	120	0.0	57.8	0.0	616	0
03-569	F	1901		1973	174	DP	1922	312	1959	144	0.0	67.5	0.0	712	0
03-570	F	1908	1990			DP	1925	43	1981	1	0.0	1.6	0.0	17	0
03-571	F	1909	1990			DP	1925	52	1981	798	0.0	246.5	0.0	4437	0
03-572	F	1906	1990			DP	1924	56	1977	62	0.0	55.3	0.0	617	0
03-573	F	1900		1979	4109	DP	1925	52	1977	16	0.0	15.1	0.0	154	0
03-574	F	1904	1990			DP	1920	624	1976	1	0.0	0.6	0.0	7	0
03-575	F	1913	1990			DP	1931	52	1973	0	0.0	0.0	0.0	0	0
03-576	F	1909		1987		DP	1925	156	1981	5	0.0	5.7	0.0	60	0
03-577	F	1901		1961	4109	DP	1921	104	1959	81	0.0	43.9	0.0	418	0
03-578	F	1909		1980	4109	DP	1924	30	1976	8	0.0	8.0	0.0	82	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-579	F	1905	1990			DP	1922	13	1959	30	0.0	17.4	0.0	187	0
03-580	F	1904		1984	4123	DP	1923	4	1959	2	0.0	1.2	0.0	12	0
03-581	F	1904		1986	4360	DP	1922	10	1959	13	0.0	7.7	0.0	80	0
03-583	M	1893		1962	4419	DR	1930	+0	1959	50	0.0	20.7	0.0	129	0
03-584	F	1905		1959	1709	DP	1923	+0	1959	6000	0.0	916.3	0.0	13868	0
03-585	F	1894		1982	4409	DP	1918	260	1966	74	0.0	52.3	0.0	576	0
03-586	F	1908		1968	174	DP	1926	82	1967	900	0.0	231.5	0.0	3173	0
03-587	F	1906	1990			DP	1925	34	1959	13	0.0	6.7	0.0	71	0
03-588	F	1901		1967	924E	DP	1922	229	1962	316	0.0	131.5	0.0	1465	0
03-589	F	1906		1969	4379	DP	1924	21	1959	77	0.0	38.9	0.0	385	0
03-590	F	1900		1982	174	DP	1922	26	1965	104	0.0	65.3	0.0	723	0
03-591	F	1907	1990			DP	1926	2340	1980	5	0.0	1.8	0.0	16	0
03-592	F	1905	1990			DP	1922	78	1979	70	0.0	66.8	0.0	763	0
03-593	F	1905	1990			DP	1922	10	1981	12	0.0	14.5	0.0	155	0
03-594	F	1905		1968	4123	DP	1922	52	1959	41	0.0	23.0	0.0	225	0
03-595	F	1902		1989	1539	DP	1923	52	1980	0	0.0	0.0	0.0	0	0
03-596	F	1904	1990			DP	1922	8	1979	10	0.0	12.2	0.0	130	0
03-597	F	1903	1990			DP	1925	1300	1972	74	0.0	33.7	0.0	355	0
03-598	M	1890		1981	1541	DR	1933	4	1971	1	0.0	0.5	0.0	3	0
03-599	F	1906		1975	174	DP	1922	26	1959	9	0.0	5.3	0.0	53	0
03-600	F	1902		1986		DH	1926	988	1972	0	0.0	0.0	0.0	0	0
03-601	F	1893		1969	5741	DP	1925	260	1960	6	0.0	3.2	0.0	31	0
03-602	F	1899		1979	1830	DP	1925	104	1960	4	0.0	1.9	0.0	19	0
03-603	F	1888		1979	4123	DP	1924	520	1960	0	0.0	0.0	0.0	0	0
03-604	F	1899		1990		DP	1916	572	1981	4	0.0	4.6	0.0	49	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-605	F	1900		1986	7340	DP	1921	364	1972	1	0.0	0.5	0.0	6	0
03-606	F	1903		1986	7123	DP	1924	6	1971	2	0.0	1.6	0.0	17	0
03-607	F	1906	1990			DP	1922	26	1981	91	0.0	86.4	0.0	1017	0
03-608	F	1895		1976	4123	DP	1917	104	1960	19	0.0	13.4	0.0	138	0
03-609	F	1896		1974	4309	DP	1923	4	1960	0	0.0	0.0	0.0	0	0
03-610	F	1917	1990			DP	1935	104	1973	1	0.0	0.8	0.0	8	0
03-611	F	1893		1969	1541	DP	1916	208	1960	3	0.0	2.2	0.0	21	0
03-612	F	1892		1968	4339	DP	1918	234	1960	500	0.0	175.2	0.0	2243	0
03-613	F	1905	1990			DP	1925	95	1972	2	0.0	1.4	0.0	14	0
03-614	F	1909	1990			DP	1924	56	1975	94	0.0	73.4	0.0	842	0
03-615	F	1905	1990			DP	1923	107	1975	14	0.0	13.8	0.0	146	0
03-617	F	1902		1951	946E	DP	1921	312	1963	7000	0.0	956.5	0.0	11964	0
03-618	F	1893		1969	1830	DP	1920	43	1960	10	0.0	6.5	0.0	64	0
03-619	F	1903		1962	1707	DP	1922	34	1962	1576	0.00139	282.1	45.6	4362	710
03-620	F	1923	1990			DP	1942	208	1971	5	0.0	1.9	0.0	18	0
03-621	F	1916	1990			DP	1944	208	1971	4	0.0	1.4	0.0	14	0
03-622	F	1910	1990			DP	1926	104	1960	0	0.0	0.0	0.0	0	0
03-623	F	1902		1978	4379	DP	1924	+0	1960	4	0.0	2.3	0.0	23	0
03-624	F	1905		1959	4319	DP	1923	156	1959	1000	0.0	227.5	0.0	2825	0
03-625	F	1901	1990			DP	1923	13	1981	2	0.0	2.6	0.0	28	0
03-626	F	1906	1984			DP	1924	208	1960	200	0.0	88.8	0.0	1016	0
03-627	F	1905		1966	174	DP	1924	208	1960	50	0.0	26.2	0.0	249	0
03-628	F	1905		1974	1619	DP	1921	34	1962	0	0.0	0.0	0.0	0	0
03-629	F	1903		1969	5710	DP	1922	+0	1960	0	0.0	0.0	0.0	0	0
03-630	F	1908	1990			DP	1924	17	1974	19	0.0	17.0	0.0	181	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-632	F	1905		1975	4123	DP	1922	+0	1960	0	0.0	0.0	0.0	0	0
03-633	F	1902	1990			DP	1922	780	1960	20	0.0	11.2	0.0	119	0
03-634	F	1909		1961	4109	DP	1924	+0	1960	3	0.0	1.7	0.0	16	0
03-635	F	1907		1986	1621	DP	1925	+0	1960	47	0.0	24.7	0.0	262	0
03-636	F	1903		1990		DP	1924	186	1981	6	0.0	6.6	0.0	70	0
03-637	F	1906		1990		DP	1924	6	1979	39	0.0	39.7	0.0	435	0
03-638	F	1902		1972	4109	DP	1924	+0	1960	7	0.0	4.0	0.0	39	0
03-639	F	1912	1990			DP	1925	156	1960	67	0.0	33.8	0.0	367	0
03-640	F	1902	1990			DP	1924	60	1960	5	0.0	2.8	0.0	29	0
03-641	F	1904		1984	4100	DP	1922	26	1979	9	0.0	10.5	0.0	110	0
03-642	F	1905		1978	203	DP	1922	52	1976	31	0.0	31.2	0.0	325	0
03-643	F	1909		1979	4123	DP	1926	156	1975	10	0.0	8.3	0.0	83	0
03-645	F	1906	1990			DP	1924	312	1959	56	0.0	28.1	0.0	303	0
03-646	F	1888		1981	4270	DP	1926	+0	1960	0	0.0	0.0	0.0	0	0
03-647	F	1901	1990			DP	1925	5	1960	35	0.0	18.6	0.0	199	0
03-648	F	1903		1956	1601	DP	1922	155	1956	5000	0.00433	730.7	155.3	10290	2435
03-649	F	1906		1954	1706	DP	1924	1352	1951	1300	0.00005	233.6	0.3	2651	4
03-658	F	1903		1938	946E	DP	1922	468	1984	6000	0.0	625.1	0.0	4832	0
03-666	F	1905		1929	032	DP	1923	347	1978	24812	0.00024	1592.4	231.6	5424	1332
03-671	F	1906		1953	1959	DP	1922	8	1952	3820	0.00433	540.5	84.6	7338	1322
03-672	F	1899		1984	4369	DP	1924	+0	1960	3	0.0	1.7	0.0	18	0
03-673	F	1909	1990			DP	1926	8	1960	35	0.0	17.9	0.0	190	0
03-674	F	1908		1977	4109	DP	1925	43	1976	2	0.0	2.1	0.0	20	0
03-676	F	1897		1977	4124	DP	1924	+0	1963	1700	0.0	311.1	0.0	5708	0
03-677	M	1899		1965	8120E	CR	1924	+0	1961	232	0.0	105.8	0.0	785	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-678	M	1919	1990			DH	1953	988	1982	3	0.0	0.6	0.0	4	0
03-679	F	1910	1990			DP	1930	10	1977	1	0.0	1.2	0.0	12	0
03-680	F	1906		1946	1704	DP	1924	+0	1984	3930	0.0	498.7	0.0	5307	0
03-681	F	1906	1962			DP	1922	6	1962	1	0.0	0.4	0.0	4	0
03-682	F	1907		1988		DP	1925	60	1978	2	0.0	1.9	0.0	20	0
03-683	F	1906		1979	4123	DP	1923	0	1961	0	0.0	0.0	0.0	0	0
03-684	F	1907	1990			DP	1927	17	1982	0	0.0	0.0	0.0	0	0
03-685	F	1902	1990			DP	1921	65	1979	86	0.0	80.3	0.0	940	0
03-686	F	1904	1990			DP	1923	1040	1983	12	0.0	14.4	0.0	153	0
03-687	F	1900		1974	174	DP	1925	43	1961	51	0.0	27.3	0.0	273	0
03-688	F	1918	1990			DP	1935	367	1972	3	0.0	1.3	0.0	13	0
03-689	F	1903	1990			DP	1923	208	1982	70	0.0	68.0	0.0	776	0
03-690	F	1909		1967	447	DP	1924	290	1959	380	0.0	137.8	0.0	1527	0
03-692	M	1887		1976	4124	DH	1920	+0	1961	6	0.0	4.2	0.0	30	0
03-693	F	1920	1952			DP	1942	520	1952	14	0.0	0.9	0.0	3	0
03-695	F	1921	1990			DP	1942	44	1982	1	0.0	0.7	0.0	7	0
03-696	F	1932	1990			DP	1950	52	1963	0	0.0	0.0	0.0	0	0
03-697	F	1902		1981	0389	DP	1924	34	1967	181	0.0	100.8	0.0	1159	0
03-701	F	1907	1990			DP	1924	9	1977	0	0.0	0.0	0.0	0	0
03-703	F	1921	1990			DP	1946	282	1980	1	0.0	0.6	0.0	6	0
03-710	F	1907		1981	4100	DP	1924	728	1977	3	0.0	3.4	0.0	34	0
03-712	F	1922	1990			DP	1942	62	1977	7	0.0	3.8	0.0	37	0
03-713	F	1921	1990			DP	1941	1456	1971	2	0.0	0.3	0.0	2	0
03-714	F	1923	1990			DP	1942	364	1971	3	0.0	1.2	0.0	12	0
03-716	F	1920		1976	1830	DP	1941	104	1971	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-717	F	1906		1977	174	DP	1922	156	1977	150	0.0	109.5	0.0	1262	0
03-720	F	1910		1985	4109	DP	1926	52	1976	6	0.0	5.2	0.0	53	0
03-722	F	1905	1990			DP	1924	4	1977	3	0.0	2.8	0.0	29	0
03-726	F	1905		1972	4123	DP	1922	186	1968	574	0.0	197.7	0.0	2690	0
03-727	F	1906		1977	5193	DP	1923	988	1972	165	0.0	103.8	0.0	1179	0
03-729	F	1926	1990			DP	1943	208	1973	1	0.0	0.5	0.0	5	0
03-730	M	1894		1963	492	CR	1923	+0	1961	7	0.0	4.3	0.0	29	0
03-732	F	1924	1990			DP	1942	78	1973	2	0.0	0.7	0.0	7	0
03-736	F	1896		1990		DP	1919	22	1980	0	0.0	0.0	0.0	0	0
03-741	F	1908	1990			DP	1925	260	1975	4	0.0	3.4	0.0	36	0
03-748	F	1910	1990			DP	1927	+0	1977	5	0.0	4.6	0.0	48	0
03-752	F	1904	1990			DP	1922	15	1980	7	0.0	8.6	0.0	92	0
03-753	F	1906	1990			DP	1922	+0	1980	12	0.0	15.0	0.0	161	0
03-757	F	1902		1987		DP	1923	91	1978	10	0.0	11.1	0.0	117	0
03-761	F	1901	1990			DP	1927	1144	1980	17	0.0	11.5	0.0	116	0
03-763	F	1901	1990			DP	1931	52	1976	0	0.0	0.0	0.0	0	0
03-764	F	1908		1987		DP	1926	364	1981	1	0.0	0.6	0.0	6	0
03-771	F	1900	1990			DP	1923	13	1981	108	0.0	96.1	0.0	1146	0
03-774	F	1909	1990			DP	1924	3	1977	1	0.0	0.6	0.0	7	0
03-775	F	1922	1990			DP	1942	52	1974	4	0.0	1.8	0.0	18	0
03-778	F	1904		1987	3499	DP	1923	104	1973	54	0.0	44.6	0.0	488	0
03-779	F	1905		1942	1713	DP	1922	+0	1979	1835	0.0	260.9	0.0	2436	0
03-782	F	1908	1990			DP	1923	5	1981	0	0.0	0.4	0.0	4	0
03-784	F	1905		1986	4100	DP	1923	178	1954	750	0.0	187.0	0.0	2711	0
03-788	F	1905	1990			DP	1926	104	1976	1	0.0	0.7	0.0	8	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
03-795	F	1897		1944	1570	DP	1926	78	1944	8	0.0	1.9	0.0	12	0
03-796	F	1907		1990	5193	DP	1925	2	1972	0	0.0	0.2	0.0	2	0
03-798	F	1915	1990			DP	1935	280	1978	2	0.0	1.0	0.0	10	0
03-801	F	1906		1986		DP	1924	13	1981	1	0.0	1.2	0.0	13	0
03-807	F	1923	1985			DP	1954	780	1973	0	0.0	0.0	0.0	0	0
03-810	F	1919	1990			DP	1934	312	1972	2	0.0	1.0	0.0	10	0
03-817	F	1907	1990			DP	1926	13	1978	0	0.0	0.0	0.0	0	0
03-818	F	1902		1984	174	DP	1927	62	1975	4	0.0	3.6	0.0	37	0
03-825	F	1906		1985	4123	DP	1922	4	1981	1	0.0	0.9	0.0	10	0
03-828	M	1915	1990			DH	1950	936	1980	0	0.0	0.0	0.0	0	0
03-834	F	1907		1984	402	DP	1925	+0	1976	1	0.0	1.3	0.0	13	0
03-836	F	1908		1980	1621	DP	1924	23	1967	0	0.0	0.0	0.0	0	0
03-838	F	1928	1990			DP	1947	130	1975	2	0.0	0.9	0.0	9	0
03-842	F	1910		1983	486	DP	1926	416	1976	3	0.0	2.7	0.0	27	0
03-845	F	1908	1990			DP	1927	104	1979	0	0.0	0.0	0.0	0	0
03-848	F	1903		1958	1707	DP	1922	+0	1984	1600	0.0	294.0	0.0	4149	0
03-850	F	1923	1990			DP	1942	78	1979	7	0.0	4.3	0.0	42	0
05-001	F	1900		1987		DP	1919	52	1981	57	0.00027	59.0	25.7	693	339
05-002	F	1903		1973	887E	DP	1917	104	1971	2	0.0	2.1	0.0	22	0
05-003	F	1900		1959	1579	DP	1917	8	1958	0	0.0	0.0	0.0	0	0
05-004	F	1904	1990			DP	1920	104	1959	12	0.01600	7.2	11.0	78	140
05-005	F	1901		1980	4109	DP	1916	13	1960	0	0.0	0.0	0.0	0	0
05-007	F	1896	1990			DP	1920	95	1967	23	0.00600	17.2	26.0	192	336
05-008	M	1894		1964	4510	DR	1916	104	1963	4	0.0	3.3	0.0	23	0
05-010	F	1901		1974	4109	DP	1921	34	1961	4	0.01200	2.6	3.7	26	47

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
05-011	F	1902		1981	4123	DP	1917	52	1959	12	0.0	8.4	0.0	88	0
05-012	F	1901		1959	209	DP	1917	52	1979	16	0.0	11.5	0.0	108	0
05-014	F	1900	1990			DP	1916	208	1981	105	0.00051	76.3	74.6	1056	1032
05-015	F	1891		1986	4339	DP	1916	67	1978	4	0.0	5.0	0.0	53	0
05-016	M	1891		1965	5192	CR	1916	100	1958	15	0.0	10.3	0.0	72	0
05-017	F	1894		1980	450	DP	1919	+0	1968	5	0.00520	4.5	8.4	47	107
05-018	M	1886		1979	4123	CR	1918	156	1971	4	0.00180	4.0	3.4	29	31
05-019	F	1885		1968	4123	DP	1921	2	1960	0	0.01400	0.0	0.0	0	0
05-020	F	1898		1980	4109	DP	1917	52	1959	3	0.0	2.1	0.0	22	0
05-022	F	1900		1969	2509	DH	1916	32	1964	4	0.0	3.5	0.0	35	0
05-023	F	1899		1960	423	DP	1918	104	1960	38	0.00320	24.4	10.7	234	137
05-024	M	1890		1965	5719	CR	1916	208	1961	4	0.01200	2.9	6.1	21	55
05-025	F	1893		1983	1538	DP	1917	78	1971	86	0.00020	71.3	8.5	825	112
05-037	F	1898		1977	2509	DP	1916	260	1971	2	0.0	2.1	0.0	21	0
05-038	F	1901		1986		DH	1916	156	1972	99	0.0	83.3	0.0	984	0
05-039	F	1899	1990			DH	1917	156	1980	18	0.00043	21.9	15.1	244	194
05-040	F	1899	1990			DP	1917	54	1971	10	0.0	10.6	0.0	114	0
05-042	F	1918	1990			DP	1940	130	1972	1	0.0	0.5	0.0	5	0
05-043	M	1888		1960	1459	CR	1919	208	1965	0	0.00430	0.0	0.0	0	0
05-044	M	1895		1975	4339	CR	1915	468	1971	2	0.0	2.0	0.0	15	0
05-045	F	1899		1960	4124	DP	1917	60	1965	5	0.0	3.7	0.0	35	0
05-049	F	1905	1990			DP	1923	13	1965	6	0.0	4.3	0.0	45	0
05-072	M	1891		1950	1538	DR	1919	13	1976	0	0.00100	0.0	0.0	0	0
05-088	F	1886		1990		DP	1917	4	1959	4	0.0	2.9	0.0	31	0
05-089	F	1900		1982	4123	DP	1916	78	1971	13	0.0	14.0	0.0	148	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
05-092	F	1901		1982	4409	DP	1916	104	1959	6	0.0	4.3	0.0	45	0
05-093	F	1897		1974	1541	DP	1915	78	1961	6	0.0	4.9	0.0	50	0
05-094	F	1927	1990			DP	1946	39	1973	6	0.0	2.3	0.0	22	0
05-096	F	1901		1971	174	DP	1918	26	1962	234	0.00050	119.8	11.4	1417	155
05-097	M	1892		1976	4109	CR	1918	26	1961	4	0.00050	3.0	0.3	22	2
05-100	F	1907		1984	4100	DP	1919	156	1968	4	0.00520	3.5	5.3	36	68
05-101	F	1902	1990			DP	1924	6	1964	4	0.00850	2.7	2.7	28	35
05-102	F	1901		1982	2699	DP	1915	364	1960	6	0.00350	4.2	2.0	44	26
05-103	F	1906	1990			DP	1923	4	1959	1	0.01600	0.6	0.7	6	9
05-104	F	1900	1990			DP	1918	13	1964	4	0.00040	3.3	0.3	35	4
05-105	M	1903		1959	943E	DR	1918	30	1959	0	0.00070	0.0	0.0	0	0
05-111	M	1895		1977	5193	DR	1920	312	1970	5	0.00660	4.2	7.6	30	69
05-116	F	1898		1959	203	DP	1917	52	1972	19	0.0	13.2	0.0	125	0
05-117	M	1887		1968	4109	CR	1915	208	1964	4	0.0	3.4	0.0	24	0
05-118	F	1901	1990			DP	1917	65	1977	2	0.0	2.6	0.0	28	0
05-119	F	1905		1986	1621	DP	1924	212	1977	10	0.00175	9.8	8.8	103	113
05-120	F	1890		1985	4109	DH	1919	6	1959	5	0.00770	3.3	3.1	35	39
05-121	F	1906		1990		DP	1921	26	1980	8	0.00117	9.6	12.9	104	166
05-122	M	1879		1962	485	DR	1922	208	1979	5	0.00144	3.1	3.3	20	30
05-123	F	1897		1972	4109	DP	1918	1	1960	4	0.00060	2.9	0.3	29	3
05-125	F	1902		1976	4123	DH	1916	104	1959	26	0.0	18.1	0.0	189	0
05-126	M	1889		1970	887E	DP	1921	52	1970	0	0.0	0.0	0.0	0	0
05-127	M	1893		1986		CR	1918	999	1967	20	0.0	12.3	0.0	91	0
05-129	F	1900		1969	1519	DH	1917	104	1960	4	0.0	2.9	0.0	29	0
05-130	F	1920	1990			DP	1940	78	1972	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
05-132	F	1898		1982	5932	DH	1918	52	1969	0	0.00020	0.0	0.0	0	0
05-133	M	1903		1967	4822	DH	1918	13	1959	0	0.00070	0.0	0.0	0	0
05-134	F	1900	1990			DP	1917	6	1959	9	0.0	6.4	0.0	69	0
05-135	F	1919	1990			DP	1941	106	1976	0	0.0	0.0	0.0	0	0
05-136	M	1896		1966	4100	CR	1917	78	1959	94	0.0	57.2	0.0	423	0
05-138	F	1917	1990			DP	1941	104	1968	5	0.0	1.9	0.0	18	0
05-139	F	1891		1966	4123	DP	1919	70	1962	4	0.00540	2.9	2.5	28	32
05-140	F	1897		1960	4339	DP	1917	168	1978	670	0.00082	137.8	139.6	2098	2142
05-142	F	1904	1990			DP	1919	39	1960	11	0.00680	7.4	6.5	79	84
05-143	F	1899		1962	4121	DH	1918	+0	1961	4	0.00050	3.0	0.3	28	3
05-145	M	1883		1961	4123	DH	1916	572	1961	4	0.00150	2.6	0.4	17	4
05-146	M	1897		1984	1579	CR	1920	286	1968	2	0.00490	1.6	1.8	12	16
05-150	F	1899		1969	8147E	DH	1917	6	1960	45	0.0	31.3	0.0	321	0
05-151	F	1897		1988		DP	1924	95	1963	7	0.00960	4.3	4.0	45	51
05-154	F	1900		1978	4122	DP	1916	11	1970	0	0.0	0.0	0.0	0	0
05-155	F	1898		1965	8147E	DH	1916	28	1963	4	0.0	3.4	0.0	34	0
05-160	F	1917	1990			DP	1942	156	1969	0	0.0	0.0	0.0	0	0
05-161	M	1901		1979	4379	CR	1918	9	1971	0	0.00016	0.0	0.0	0	0
05-162	F	1914	1990			DH	1942	+0	1960	29	0.0	7.1	0.0	69	0
05-163	M	1912		1970	4100	DR	1941	104	1960	35	0.0	8.5	0.0	50	0
05-165	F	1899		1964	188	DP	1919	13	1972	1	0.0	0.6	0.0	6	0
05-172	F	1907		1960	2201	DP	1934	999	1960	24	0.0	4.8	0.0	30	0
05-174	F	1902		1990		DP	1919	130	1977	0	0.00126	0.0	0.0	0	0
05-179	F	1921		1982	1830	DP	1940	182	1974	0	0.0	0.0	0.0	0	0
05-181	F	1901		1990		DP	1918	4	1970	0	0.00018	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
05-184	M	1901		1974	1621	DP	1922	156	1964	5	0.0	3.4	0.0	24	0
05-185	F	1912	1990			DP	1941	208	1972	2	0.0	0.9	0.0	8	0
05-186	F	1922	1990			DP	1941	208	1982	0	0.0	0.0	0.0	0	0
05-188	M	1889		1964	4100	DH	1917	104	1961	4	0.0	3.0	0.0	21	0
05-189	M	1890		1972	5710	DR	1921	104	1964	4	0.00850	2.9	3.7	20	34
05-194	F	1902		1965	1990	DP	1926	5	1975	31	0.0	19.3	0.0	179	0
05-197	M	1898		1981	929E	DR	1919	7	1973	0	0.00140	0.0	0.0	0	0
05-199	F	1901	1990			DP	1917	2	1967	0	0.0	0.0	0.0	0	0
05-201	F	1919		1982	174	DP	1941	221	1976	6	0.0	3.0	0.0	28	0
05-203	F	1898		1985		DP	1919	52	1960	0	0.00680	0.0	0.0	0	0
05-204	M	1880		1961	4339	DR	1918	78	1978	0	0.00037	0.0	0.0	0	0
05-205	F	1907		1981	0389	DP	1924	208	1961	4	0.0	2.3	0.0	23	0
05-206	F	1894		1981	4379	DP	1922	52	1971	2	0.00360	1.8	2.2	19	28
05-207	M	1893		1980		CR	1917	+0	1962	6	0.0	4.8	0.0	36	0
05-210	F	1899		1971	4123	DP	1916	158	1977	1060	0.0	269.0	0.0	4702	0
05-212	F	1903	1990			DH	1918	8	1965	4	0.00030	3.4	0.3	37	4
05-215	F	1886		1968	4123	DP	1917	208	1969	1410	0.00198	253.5	200.4	4707	3166
05-237	M	1896		1969	4100	CR	1920	364	1961	4	0.0	2.4	0.0	17	0
05-246	F	1884		1969	4412	NU	1911	728	1962	4	0.0	3.1	0.0	30	0
05-251	F	1896		1981	4123	DP	1917	34	1965	13	0.0	11.3	0.0	119	0
05-252	F	1890		1976	1533	DP	1917	52	1964	4	0.0	3.4	0.0	35	0
05-255	M	1886		1966	4109	DR	1920	104	1964	5	0.00850	3.7	5.4	25	50
05-257	F	1895		1975	4409	DP	1932	1248	1972	3	0.0	1.1	0.0	10	0
05-258	F	1901	1990			DP	1917	1	1970	0	0.0	0.0	0.0	0	0
05-259	F	1900		1983	4369	DH	1917	52	1960	6	0.0	4.4	0.0	46	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
05-260	F	1898		1980	4124	DH	1917	32	1960	0	0.0	0.0	0.0	0	0
05-261	F	1892		1977	5740	DP	1943	104	1960	4	0.0	0.9	0.0	8	0
05-262	F	1917	1990			DP	1942	260	1982	6	0.0	3.6	0.0	35	0
05-263	M	1883		1967	4109	DR	1919	104	1962	4	0.00800	2.9	3.5	20	32
05-264	M	1903		1983	4339	DH	1917	5	1961	4	0.0	3.1	0.0	23	0
05-265	M	1884		1963	4109	DH	1916	104	1962	4	0.0	3.2	0.0	22	0
05-266	M	1881		1970	481	DR	1918	130	1964	4	0.00200	3.2	1.4	23	12
05-268	F	1893	1990			DP	1918	39	1960	4	0.00060	2.8	0.3	30	3
05-269	M	1887		1971	4109	DR	1918	52	1964	4	0.00040	3.3	0.3	23	3
05-270	M	1901	1990			DH	1916	52	1961	8	0.0	6.3	0.0	48	0
05-272	M	1895		1986		CR	1918	65	1972	0	0.00014	0.0	0.0	0	0
05-273	F	1889		1968	4109	DP	1918	104	1960	4	0.01400	2.7	5.3	27	67
05-274	F	1903	1990			DH	1920	4	1970	0	0.0	0.0	0.0	0	0
05-276	F	1906		1988		DP	1921	75	1961	4	0.01200	2.6	3.5	27	44
05-277	M	1894		1973	1532	CR	1918	104	1960	4	0.00320	2.8	1.2	20	11
05-278	F	1893		1965	0122	DP	1917	52	1964	37	0.0	29.3	0.0	292	0
05-279	F	1896		1979	4109	DP	1917	1820	1969	0	0.0	0.0	0.0	0	0
05-281	F	1898		1964	4109	DP	1916	148	1963	660	0.00216	165.0	84.2	2551	1270
05-282	F	1898		1983	4272	DP	1917	34	1964	8	0.0	6.8	0.0	71	0
05-284	F	1899		1973	4123	DP	1919	156	1969	218	0.00080	115.6	30.9	1422	426
05-286	M	1901		1963	1541	CR	1916	104	1965	1	0.0	0.4	0.0	3	0
05-287	M	1889		1970	4124	DR	1917	390	1965	4	0.00420	3.1	2.5	22	23
05-288	F	1897	1990			DP	1918	10	1960	4	0.00060	2.9	0.3	31	3
05-290	F	1898		1967	342	DP	1918	52	1960	8	0.00060	5.6	0.5	55	6
05-291	F	1902		1974	5322	DP	1920	8	1968	4	0.00540	3.5	6.0	36	76

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
05-292	M	1904		1974	1621	DR	1918	+0	1965	4	0.00033	3.5	0.3	25	3
05-304	F	1897	1990			DP	1921	26	1962	4	0.01100	2.7	4.0	29	51
05-306	F	1903		1983	1621	DP	1921	156	1981	4	0.00107	5.0	5.9	52	76
05-307	F	1920	1990			DP	1944	73	1982	0	0.0	0.2	0.0	2	0
05-308	M	1893		1964	1419	DR	1916	208	1962	4	0.00130	3.1	0.8	21	7
05-310	F	1894		1965	203	DP	1916	78	1964	5	0.0	4.3	0.0	42	0
05-311	M	1887		1961	1519	CR	1920	156	1960	4	0.01400	2.5	3.5	17	32
05-312	M	1886		1961	4119	DP	1919	34	1961	2	0.00610	1.4	1.3	10	12
05-318	M	1901		1961	2051	DH	1918	+0	1965	4	0.00030	3.0	0.3	20	2
05-321	F	1899		1990		DP	1916	208	1966	16	0.00330	13.3	13.8	147	177
05-322	M	1900		1975	4123	DH	1917	312	1973	4	0.0	4.2	0.0	31	0
05-323	F	1899		1961	402	DP	1915	26	1961	2	0.0	1.6	0.0	15	0
05-349	F	1884		1956	4100	DP	1919	+0	1979	7	0.00075	4.3	4.4	39	56
05-351	F	1891		1983	404	DP	1917	30	1968	23	0.0	21.5	0.0	230	0
05-352	M	1900		1963	485	DR	1917	40	1964	1	0.0	0.8	0.0	6	0
05-353	M	1900		1985	5193	DH	1915	13	1978	0	0.0	0.1	0.0	1	0
05-357	F	1890		1978	887E	DR	1917	104	1972	3	0.0	3.3	0.0	34	0
05-360	M	1892		1968	4100	DP	1914	+0	1963	4	0.0	3.7	0.0	27	0
05-363	F	1899		1980	4339	DH	1917	9	1964	4	0.0	3.4	0.0	36	0
05-368	F	1901		1990	4369	DR	1917	104	1977	0	0.0	0.0	0.0	0	0
05-369	F	1901		1986	4123	DH	1919	26	1978	1	0.00077	1.3	1.1	14	15
05-370	F	1895	1990			DP	1920	26	1965	4	0.00760	3.2	5.1	34	66
05-372	F	1888		1970	2509	DP	1916	104	1968	14	0.0	13.5	0.0	136	0
05-374	F	1905	1990			DP	1923	8	1980	0	0.00124	0.0	0.0	0	0
05-377	F	1895		1974	4123	DP	1916	15	1969	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
05-380	F	1904		1970	2509	DH	1925	104	1962	4	0.01100	2.3	1.9	22	24
05-383	F	1901		1982	2315	CR	1917	165	1973	73	0.00060	60.7	24.7	706	327
05-387	M	1902	1990			CR	1918	9	1975	0	0.00010	0.0	0.0	0	0
05-395	F	1911	1990			DP	1927	728	1977	0	0.0	0.0	0.0	0	0
05-397	F	1900		1976	1570	IR	1918	13	1962	4	0.0	3.1	0.0	32	0
05-399	M	1892		1982		DR	1916	104	1961	4	0.0	3.1	0.0	23	0
05-401	M	1898		1986	4109	CR	1917	169	1971	5	0.00170	5.1	4.6	39	42
05-407	F	1898	1990			DP	1916	9	1978	0	0.0	0.0	0.0	0	0
05-409	F	1900		1983	4123	DR	1918	61	1974	0	0.00011	0.0	0.0	0	0
05-410	F	1899		1990		DP	1916	26	1980	2	0.0	2.6	0.0	28	0
05-413	F	1900		1971	4360	DP	1916	39	1969	18	0.0	18.1	0.0	185	0
05-420	F	1889		1935	5110	DP	1917	104	1970	50	0.0	11.6	0.0	77	0
05-437	F	1888		1985		DR	1923	26	1971	3	0.00350	2.6	2.9	28	36
05-438	F	1907	1990			DP	1926	13	1961	4	0.0	2.2	0.0	23	0
05-439	F	1898		1970	492	DP	1916	104	1967	200	0.0	124.5	0.0	1465	0
05-440	F	1896		1975	4124	DP	1922	1	1971	0	0.00360	0.0	0.0	0	0
05-442	F	1888		1989		DH	1917	6	1962	8	0.0	6.4	0.0	68	0
05-443	F	1922		1986		DH	1941	52	1972	3	0.0	1.4	0.0	13	0
05-444	M	1899		1963	4109	CR	1917	43	1961	4	0.0	3.1	0.0	21	0
05-446	M	1888		1971	4123	RI	1925	+0	1964	4	0.0	2.6	0.0	18	0
05-447	F	1902	1990			DP	1916	9	1970	2	0.0	2.2	0.0	24	0
05-448	F	1903	1990			DP	1916	1	1961	4	0.0	3.2	0.0	35	0
05-449	F	1892		1961	4109	DP	1919	52	1961	4	0.00610	2.8	2.5	26	32
05-450	F	1903	1990			DH	1918	117	1971	1	0.00090	1.0	0.5	11	6
05-459	F	1917	1974			DP	1933	208	1961	8	0.0	3.0	0.0	27	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
05-460	F	1898		1979	4109	DH	1916	182	1961	4	0.0	3.0	0.0	31	0
05-464	F	1895		1969	4109	DP	1917	+0	1968	5	0.0	4.9	0.0	49	0
05-473	M	1899		1970	4109	CR	1921	26	1962	4	0.01100	2.7	4.0	19	36
05-528	F	1892		1981	4369	DP	1917	52	1967	0	0.0	0.0	0.0	0	0
05-541	F	1913	1990			DP	1937	884	1972	0	0.0	0.0	0.0	0	0
05-546	F	1902	1990			DH	1918	52	1973	1	0.00012	1.1	0.1	12	1
05-551	F	1895		1981	4123	DP	1918	9	1970	15	0.00018	14.8	1.4	156	17
05-555	F	1898		1965	1519	DH	1917	27	1975	1	0.0	0.9	0.0	9	0
05-560	M	1894		1965	4121	DR	1921	260	1962	4	0.01100	2.5	2.7	17	25
05-574	F	1904		1985	1538	DP	1918	1	1977	0	0.00008	0.0	0.0	0	0
05-580	M	1904		1975	442	DH	1919	6	1968	4	0.00260	3.7	3.4	27	31
05-602	M	1899		1986	5330	CR	1925	1300	1975	0	0.0	0.0	0.0	0	0
05-611	F	1900		1938	887E	DP	1914	156	1974	0	0.0	0.0	0.0	0	0
05-631	F	1897		1976	481	DP	1917	17	1970	0	0.0	0.0	0.0	0	0
05-639	M	1906		1962	4100	CR	1922	39	1964	1	0.00850	0.7	0.8	4	8
05-674	M	1922	1990			CR	1946	156	1965	4	0.0	1.0	0.0	6	0
05-688	F	1921		1976	398	DP	1939	130	1965	5	0.0	1.8	0.0	16	0
05-736	F	1898		1954	4310	CR	1918	156	1972	150	0.00410	50.0	110.8	534	1535
05-737	M	1895		1957	1539	CR	1918	156	1971	10	0.00463	5.9	13.1	39	119
05-742	F	1898		1975	4109	DP	1916	30	1969	0	0.0	0.0	0.0	0	0
05-751	F	1901		1933	6379	DP	1920	+0	1969	0	0.00500	0.0	0.0	0	0
05-765	F	1900	1990			DH	1916	117	1964	4	0.0	3.4	0.0	37	0
05-802	F	1893		1980	4123	DP	1918	+0	1972	1	0.00014	0.6	0.1	6	1
05-818	F	1901		1969	4109	DP	1918	52	1967	25	0.00026	21.5	1.9	217	24
05-873	F	1894		1984	4409	DH	1917	286	1962	39	0.00350	25.6	13.9	280	179

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
05-880	F	1921	1990			DP	1939	520	1974	2	0.0	0.9	0.0	9	0
05-882	F	1917		1965	1940	DP	1935	468	1964	13	0.0	4.4	0.0	35	0
05-885	F	1917		1984		DP	1939	572	1969	0	0.0	0.0	0.0	0	0
05-892	F	1904		1982	4119	DP	1917	4	1969	160	0.0	112.7	0.0	1371	0
05-897	F	1899		1968	4319	DP	1917	69	1968	1310	0.0	292.5	0.0	5099	0
05-898	F	1919	1990			DP	1936	468	1972	0	0.0	0.0	0.0	0	0
05-900	F	1919		1973	4309	DP	1936	312	1972	3	0.0	1.5	0.0	13	0
05-901	F	1918		1984	929	DP	1934	468	1972	2	0.0	1.0	0.0	10	0
05-902	F	1919	1990			DP	1936	988	1962	6	0.0	1.2	0.0	12	0
05-905	F	1916	1990			DH	1937	156	1972	0	0.0	0.0	0.0	0	0
05-906	F	1913	1990			DP	1935	624	1972	2	0.0	0.9	0.0	9	0
05-907	F	1915		1984	1541	DP	1935	260	1972	3	0.0	1.6	0.0	16	0
05-911	M	1886		1982	1732	DR	1923	6	1972	0	0.00310	0.0	0.0	0	0
05-912	M	1877		1951	1579	DR	1918	26	1969	0	0.00020	0.0	0.0	0	0
05-917	F	1902		1983	1713	DP	1918	39	1982	76	0.00010	78.8	17.2	933	229
05-920	M	1895		1963	404	CR	1917	43	1962	4	0.0	3.2	0.0	22	0
05-921	F	1896		1990		DP	1916	30	1969	67	0.0	59.2	0.0	682	0
05-942	M	1901		1988		CR	1918	9	1975	0	0.00010	0.0	0.0	0	0
05-949	M	1899		1974	491	CR	1921	422	1968	0	0.0	0.0	0.0	0	0
05-953	F	1902		1978	1719	DP	1918	65	1977	1200	0.00008	282.7	23.7	5547	368
05-962	F	1894		1977	1990	DP	1918	84	1964	47	0.00200	33.6	15.2	362	197
05-974	F	1900		1984	4109	DH	1918	104	1970	0	0.00100	0.0	0.0	0	0
05-979	F	1897		1989		DP	1917	4	1982	124	0.0	119.5	0.0	1513	0
05-993	M	1902		1972	453	DH	1917	6	1971	0	0.0	0.0	0.0	0	0
05-994	F	1886		1985	4123	DP	1922	26	1967	9	0.00570	7.0	8.5	73	109

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
05-998	F	1902		1986		DP	1918	3	1980	0	0.00005	0.0	0.0	0	0
09-001	F	1901	1990			DP	1917	39	1971	4	0.0	4.3	0.0	46	0
09-002	F	1902		1970	4109	DP	1917	17	1959	10	0.0	7.1	0.0	71	0
09-003	M	1892		1963	1621	CR	1914	572	1959	410	0.0	150.3	0.0	1288	0
09-004	F	1890		1961	4109	DP	1912	416	1960	550	0.0	187.5	0.0	2475	0
09-006	F	1898		1971	4109	DP	1917	65	1963	1	0.0	0.8	0.0	8	0
09-007	F	1901		1965	4109	DP	1917	104	1960	33	0.0	22.7	0.0	224	0
09-008	F	1898	1990			DP	1917	8	1960	20	0.0	14.5	0.0	158	0
09-009	F	1893		1969	450	DP	1915	78	1960	2	0.0	1.6	0.0	16	0
09-010	F	1897		1964	4109	DP	1914	+0	1960	10	0.0	8.2	0.0	82	0
09-013	F	1900		1976	4349	DP	1917	13	1971	4	0.0	4.4	0.0	45	0
09-015	M	1890		1972	4109	RW	1914	52	1960	0	0.0	0.0	0.0	0	0
09-019	F	1903		1981	4100	DP	1917	18	1975	0	0.0	0.0	0.0	0	0
09-020	F	1897		1968	203	DP	1917	156	1963	1	0.0	0.8	0.0	8	0
09-024	M	1873		1960	4121	CR	1915	+0	1960	0	0.0	0.0	0.0	0	0
09-026	F	1902	1990			DP	1917	48	1978	16	0.0	20.6	0.0	225	0
09-028	F	1897		1976	8121E	DP	1916	78	1975	60	0.0	61.9	0.0	682	0
09-029	F	1901		1962	2000	DP	1917	13	1960	16	0.0	11.7	0.0	113	0
09-031	F	1897		1990		DH	1913	364	1960	286	0.0	134.5	0.0	1824	0
09-032	F	1902		1969	1621	DP	1917	52	1969	97	0.0	77.2	0.0	835	0
09-038	F	1903		1988		DP	1919	1	1960	0	0.0	0.0	0.0	0	0
09-041	M	1889		1952	4109	CR	1914	260	1965	114	0.0	56.3	0.0	376	0
09-043	F	1898		1976	174	DP	1917	26	1971	3	0.0	3.3	0.0	34	0
09-044	F	1900		1955	174	DP	1917	13	1975	17	0.0	10.4	0.0	95	0
09-046	F	1902		1965	4109	DP	1917	104	1960	10	0.0	7.1	0.0	70	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
09-049	F	1902		1983	1621	DP	1915	+0	1969	14	0.0	14.9	0.0	159	0
09-051	F	1900		1971	402	DP	1917	104	1960	50	0.0	33.4	0.0	345	0
09-052	F	1900		1971	1532	DP	1916	52	1960	20	0.0	14.8	0.0	151	0
09-053	M	1874		1966	4124	RW	1919	+0	1960	81	0.0	50.1	0.0	363	0
09-057	F	1890		1973	4123	DP	1917	52	1960	0	0.0	0.0	0.0	0	0
09-058	F	1899		1989		DP	1917	39	1960	4	0.0	3.0	0.0	32	0
09-059	F	1903		1972	8120E	DP	1917	1	1971	2	0.0	2.2	0.0	22	0
09-060	F	1899		1975	1538	DP	1917	65	1969	43	0.0	38.9	0.0	412	0
09-061	F	1892	1990			DP	1914	208	1970	0	0.0	0.0	0.0	0	0
09-062	F	1901		1985	4123	DP	1918	52	1972	4	0.0	4.3	0.0	45	0
09-064	F	1891		1985	4122	DP	1916	9	1973	1	0.0	1.2	0.0	13	0
09-065	F	1887		1975	2509	IR	1914	78	1960	1	0.0	0.8	0.0	8	0
09-066	F	1899		1983	4272	DP	1917	8	1972	2	0.0	2.3	0.0	24	0
09-070	M	1875		1967	4123	CR	1913	208	1960	3	0.0	2.4	0.0	17	0
09-071	F	1897		1977	4100	DP	1917	104	1975	2	0.0	2.4	0.0	25	0
09-072	F	1893		1974	4360	DP	1917	39	1972	2	0.0	2.5	0.0	25	0
09-073	M	1886		1963	4109	CR	1916	468	1962	0	0.0	0.0	0.0	0	0
09-074	F	1892		1976	4123	DP	1920	104	1962	13	0.0	8.9	0.0	91	0
09-075	M	1893		1967	4109	CR	1913	884	1963	1	0.0	0.7	0.0	5	0
09-076	M	1882		1966	8147E	CR	1913	1872	1964	14	0.0	6.1	0.0	38	0
09-077	M	1894		1985	4123	CR	1914	520	1972	2	0.0	2.1	0.0	16	0
09-078	M	1883		1966	4124	CR	1911	832	1963	3	0.0	2.3	0.0	16	0
09-079	M	1891		1981	4123	CR	1916	570	1962	0	0.0	0.0	0.0	0	0
09-080	M	1886		1982	4124	CR	1919	312	1962	5	0.0	3.3	0.0	25	0
09-082	M	1892		1981	486	CR	1916	312	1979	6	0.0	7.9	0.0	59	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
09-083	M	1889		1964	4109	CR	1915	17	1962	5	0.0	4.3	0.0	30	0
09-084	M	1888		1927	2050	CR	1912	676	1965	382	0.0	40.8	0.0	131	0
09-086	M	1895		1979	4123	CR	1921	78	1974	1	0.0	1.0	0.0	8	0
09-088	M	1900	1990			CR	1922	338	1971	18	0.0	14.4	0.0	109	0
09-089	M	1890		1973	2509	CR	1915	78	1959	64	0.0	43.5	0.0	331	0
09-090	M	1888		1971	4123	CR	1913	78	1963	0	0.0	0.0	0.0	0	0
09-095	M	1894		1975	4123	CR	1918	416	1975	0	0.0	0.0	0.0	0	0
09-096	M	1892		1978	5631	CR	1919	17	1963	9	0.0	6.9	0.0	51	0
09-097	M	1896		1983	5193	IR	1916	988	1974	1	0.0	0.9	0.0	7	0
09-098	M	1902		1971	4123	CR	1921	104	1963	14	0.0	9.6	0.0	68	0
09-099	M	1898		1971	4123	CR	1913	208	1963	1	0.0	0.9	0.0	7	0
09-100	M	1888		1980	0389	CR	1918	364	1963	9	0.0	6.3	0.0	46	0
09-101	M	1884		1964	4123	CR	1920	39	1963	6	0.0	4.4	0.0	30	0
09-102	M	1882		1951	5621	CR	1915	1	1964	150	0.0	73.2	0.0	498	0
09-103	M	1895		1971	4379	CR	1918	416	1965	1	0.0	0.7	0.0	5	0
09-104	M	1880		1967	470	CR	1906	364	1965	42	0.0	42.1	0.0	322	0
09-105	M	1886		1928	1959	CR	1912	832	1966	1390	0.00093	103.9	15.2	328	87
09-106	M	1901		1990		CR	1919	156	1979	0	0.0	0.0	0.0	0	0
09-107	M	1897		1974	4412	CR	1913	104	1965	1	0.0	1.0	0.0	7	0
09-108	M	1891		1981	402	CR	1915	104	1965	4	0.0	3.7	0.0	28	0
09-109	M	1895		1982	492	CR	1914	104	1965	4	0.0	3.8	0.0	29	0
09-110	M	1900		1985	4272	CR	1914	52	1965	7	0.0	6.8	0.0	52	0
09-111	M	1874		1944	491	MD	1913	520	1967	0	0.0	0.0	0.0	0	0
09-112	M	1898		1979	342	CR	1940	416	1966	84	0.0	24.5	0.0	158	0
09-115	M	1893		1985	4123	CR	1920	52	1969	3	0.0	2.7	0.0	20	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
09-117	F	1899		1987		DP	1917	24	1971	4	0.0	4.4	0.0	46	0
09-118	F	1901	1990			DR	1921	+0	1970	50	0.0	41.6	0.0	461	0
09-120	M	1889		1945	4310	CR	1918	104	1974	1	0.0	0.5	0.0	3	0
09-123	M	1890		1985	4109	CR	1917	156	1979	0	0.0	0.0	0.0	0	0
10-007	F	1916	1990			DP	1934	1144	1981	6	0.0	3.1	0.0	30	0
10-008	F	1904	1990			DP	1918	13	1981	1	0.00005	1.5	0.1	16	2
10-010	F	1895		1975	4123	RI	1930	+0	1971	8600	0.0	1389.0	0.0	24592	0
10-012	M	1886		1941	5369	RI	1925	+0	1972	0	0.0	0.0	0.0	0	0
10-018	F	1920	1990			DP	1952	416	1975	1	0.0	0.3	0.0	2	0
10-024	M	1914	1990			CR	1936	1612	1971	50	0.0	10.4	0.0	68	0
10-025	M	1937	1990			DH	1963	416	1971	7	0.0	0.4	0.0	2	0
10-026	M	1948	1990			DH	1968	200	1971	2	0.0	0.1	0.0	0	0
10-027	F	1928	1990			DP	1946	156	1972	0	0.0	0.0	0.0	0	0
10-028	M	1886		1976	2050	CR	1918	156	1976	0	0.0	0.0	0.0	0	0
10-031	F	1928	1990			DP	1946	52	1979	3	0.0	1.5	0.0	14	0
10-032	M	1937	1990			DR	1961	156	1972	0	0.0	0.0	0.0	0	0
10-033	F	1927	1990			DP	1946	264	1974	3	0.0	1.2	0.0	12	0
10-034	F	1919	1990			DP	1943	202	1973	9	0.0	3.6	0.0	35	0
10-035	F	1922	1990			DP	1942	674	1982	7	0.0	3.5	0.0	33	0
10-036	F	1920	1990			CR	1945	208	1972	0	0.0	0.0	0.0	0	0
10-037	F	1927	1990			DP	1951	52	1976	3	0.0	1.1	0.0	10	0
10-038	F	1929	1990			DP	1947	78	1974	1	0.0	0.2	0.0	2	0
10-039	F	1922	1990			DH	1942	260	1972	4	0.0	1.6	0.0	15	0
10-040	F	1917		1989	4109	DP	1946	+0	1972	0	0.0	0.0	0.0	0	0
10-041	F	1924	1990			DP	1943	13	1972	1	0.0	0.4	0.0	4	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-042	F	1927	1990			DP	1947	125	1982	0	0.0	0.1	0.0	0	0
10-043	F	1919	1990			RI	1941	8	1975	0	0.0	0.0	0.0	0	0
10-044	F	1925	1990			DH	1943	273	1982	7	0.0	4.2	0.0	40	0
10-045	F	1923	1990			DP	1946	13	1982	1	0.0	0.5	0.0	5	0
10-046	F	1927	1990			DP	1947	208	1975	0	0.0	0.0	0.0	0	0
10-047	F	1924	1990			DP	1942	52	1974	10	0.0	4.6	0.0	45	0
10-048	F	1894		1986	4123	CR	1917	156	1977	0	0.0	0.0	0.0	0	0
10-049	F	1926	1990			DP	1946	104	1972	0	0.0	0.0	0.0	0	0
10-050	F	1920	1990			DP	1943	114	1982	6	0.0	3.9	0.0	38	0
10-051	M	1914	1990			CR	1931	468	1979	1	0.0	1.0	0.0	7	0
10-053	F	1926	1990			DP	1946	267	1982	1	0.0	0.3	0.0	2	0
10-054	F	1926	1990			DP	1946	304	1972	1	0.0	0.4	0.0	4	0
10-055	M	1922	1975			OF	1922	39	1972	0	0.00040	0.0	0.0	0	0
10-056	M	1924	1975			OF	1924	39	1972	2	0.00040	1.8	0.2	13	2
10-057	F	1929	1990			DP	1946	52	1972	1	0.0	0.5	0.0	5	0
10-058	F	1923	1990			DP	1941	208	1982	5	0.0	3.2	0.0	31	0
10-059	F	1917	1990			DP	1954	143	1980	1	0.0	0.4	0.0	3	0
10-060	F	1919	1990			DP	1943	104	1972	0	0.0	0.0	0.0	0	0
10-061	F	1923	1990			DH	1942	164	1972	6	0.0	2.5	0.0	24	0
10-062	F	1920	1990			DP	1939	182	1972	1	0.0	0.6	0.0	6	0
10-063	F	1911	1990			DP	1928	624	1981	1	0.0	0.7	0.0	7	0
10-064	F	1921		1983	880	DR	1943	156	1972	0	0.0	0.0	0.0	0	0
10-065	F	1920	1990			DP	1941	260	1972	0	0.0	0.1	0.0	1	0
10-066	F	1924		1978	174	DP	1942	104	1972	12	0.0	5.0	0.0	44	0
10-067	F	1923	1990			DP	1942	468	1972	8	0.0	2.9	0.0	28	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-068	F	1918	1990			DP	1942	78	1972	0	0.0	0.0	0.0	0	0
10-069	F	1923	1990			DP	1947	1300	1972	12	0.0	1.3	0.0	11	0
10-070	F	1921	1990			DP	1945	1352	1982	6	0.0	1.7	0.0	14	0
10-071	F	1924	1990			DP	1943	1716	1983	5	0.0	1.3	0.0	11	0
10-072	F	1924	1990			DP	1947	1560	1983	5	0.0	1.1	0.0	9	0
10-075	F	1929	1990			DP	1949	277	1982	4	0.0	1.7	0.0	16	0
10-076	F	1923	1990			DP	1951	52	1972	0	0.0	0.0	0.0	0	0
10-077	F	1920	1990			DP	1951	17	1972	1	0.0	0.2	0.0	2	0
10-078	F	1923	1990			DP	1941	715	1980	10	0.0	4.9	0.0	46	0
10-079	F	1920	1990			DP	1940	624	1978	8	0.0	3.8	0.0	36	0
10-080	F	1913		1983	4309	CR	1943	1508	1972	5	0.0	0.6	0.0	5	0
10-081	F	1916		1990		DP	1946	104	1980	3	0.0	1.6	0.0	15	0
10-082	F	1915	1990			DP	1951	758	1972	5	0.0	0.9	0.0	7	0
10-083	F	1924	1990			DP	1943	104	1972	5	0.0	2.1	0.0	20	0
10-084	F	1928	1990			DP	1946	82	1972	0	0.0	0.0	0.0	0	0
10-085	M	1946	1990			DP	1964	17	1972	0	0.0	0.0	0.0	0	0
10-086	F	1915	1990			DP	1943	156	1979	3	0.0	1.4	0.0	14	0
10-087	F	1920		1978	1740	DP	1942	1560	1972	19	0.0	2.4	0.0	16	0
10-088	F	1923	1990			DP	1946	282	1982	1	0.0	0.3	0.0	2	0
10-089	F	1921	1990			DP	1942	13	1972	0	0.0	0.2	0.0	2	0
10-090	F	1922	1990			DP	1941	78	1972	1	0.0	0.6	0.0	6	0
10-091	M	1883		1952	4109	RI	1930	+0	1974	423	0.0	107.6	0.0	615	0
10-094	M	1905		1974	4123	DR	1919	104	1972	0	0.00240	0.0	0.0	0	0
10-095	F	1927	1990			DP	1946	260	1972	5	0.0	1.7	0.0	16	0
10-096	F	1930	1990			DP	1951	832	1982	1	0.0	0.4	0.0	4	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-097	F	1919	1990			DP	1943	364	1972	4	0.0	1.3	0.0	12	0
10-098	F	1917	1990			DP	1935	208	1972	4	0.0	2.2	0.0	22	0
10-099	F	1924	1990			DP	1942	104	1980	12	0.0	7.0	0.0	68	0
10-100	F	1924	1990			CR	1942	78	1972	7	0.0	3.2	0.0	31	0
10-101	F	1925	1990			DP	1943	208	1982	2	0.0	1.2	0.0	11	0
10-102	F	1926	1990			DP	1944	60	1972	1	0.0	0.3	0.0	3	0
10-103	F	1912	1990			DP	1946	104	1978	0	0.0	0.0	0.0	0	0
10-104	F	1929	1990			DP	1948	258	1982	4	0.0	1.9	0.0	18	0
10-105	F	1927	1990			DP	1946	417	1982	0	0.0	0.0	0.0	0	0
10-106	F	1926	1990			DP	1946	104	1982	3	0.0	1.5	0.0	15	0
10-107	F	1909	1990			DP	1926	9	1972	0	0.0	0.0	0.0	0	0
10-108	F	1916		1990		RW	1950	+0	1972	3	0.0	0.9	0.0	8	0
10-109	F	1951	1990			DR	1969	78	1972	0	0.0	0.0	0.0	0	0
10-110	F	1917	1990			DP	1946	520	1972	0	0.0	0.1	0.0	1	0
10-111	F	1906		1988	4109	DP	1923	2	1981	5	0.0	5.9	0.0	62	0
10-112	M	1902		1980	4272	DP	1923	+0	1976	3	0.0	3.2	0.0	23	0
10-113	F	1924	1990			DP	1942	75	1982	0	0.0	0.3	0.0	3	0
10-114	F	1937	1990			DP	1970	390	1982	0	0.0	0.0	0.0	0	0
10-115	F	1921	1990			DH	1970	130	1972	1	0.0	0.0	0.0	0	0
10-116	F	1924	1990			DP	1969	312	1976	5	0.0	0.3	0.0	2	0
10-117	F	1924	1990			DP	1967	208	1972	2	0.0	0.1	0.0	1	0
10-118	F	1924	1990			DP	1945	1820	1982	8	0.0	1.6	0.0	12	0
10-119	F	1952	1990			DP	1971	82	1972	2	0.0	0.0	0.0	0	0
10-120	F	1950	1990			DP	1971	98	1974	4	0.0	0.2	0.0	1	0
10-121	F	1926		1982	3969	DP	1946	7	1972	1	0.0	0.2	0.0	2	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-122	F	1921	1990			ME	1921	+0	1972	0	0.0	0.0	0.0	0	0
10-125	F	1903		1981	4389	DP	1917	8	1975	1	0.0	1.3	0.0	13	0
10-126	F	1927	1990			DP	1946	13	1982	1	0.0	0.5	0.0	4	0
10-128	F	1923	1990			DP	1942	364	1972	6	0.0	2.4	0.0	23	0
10-129	F	1923	1990			DP	1942	269	1975	9	0.0	4.0	0.0	39	0
10-130	F	1922	1990			DP	1942	147	1981	14	0.0	8.2	0.0	80	0
10-131	F	1917	1990			DR	1941	260	1972	1	0.0	0.4	0.0	4	0
10-132	F	1929	1990			DH	1970	126	1982	0	0.0	0.0	0.0	0	0
10-133	F	1910		1989		DP	1941	1248	1981	2	0.0	0.7	0.0	6	0
10-134	F	1913	1990			DP	1932	1768	1978	1	0.0	0.3	0.0	2	0
10-135	F	1922	1990			DP	1939	130	1972	6	0.0	2.9	0.0	29	0
10-136	F	1920	1990			DP	1941	26	1972	0	0.0	0.0	0.0	0	0
10-137	F	1918	1990			DP	1935	117	1972	1	0.0	0.3	0.0	3	0
10-139	F	1922		1985	1538	DP	1942	130	1972	3	0.0	1.2	0.0	11	0
10-140	F	1935	1990			DH	1956	17	1972	2	0.0	0.4	0.0	3	0
10-141	F	1918	1990			DP	1945	104	1972	0	0.0	0.0	0.0	0	0
10-142	F	1922	1990			DP	1942	156	1972	2	0.0	0.9	0.0	9	0
10-144	F	1926	1990			DP	1945	156	1972	0	0.0	0.0	0.0	0	0
10-145	F	1928	1990			DH	1946	130	1976	6	0.0	2.4	0.0	22	0
10-146	F	1921	1990			DP	1940	364	1972	4	0.0	1.5	0.0	14	0
10-147	F	1927		1990	1726	DP	1946	156	1972	2	0.0	0.7	0.0	7	0
10-148	F	1913	1990			DP	1935	13	1978	2	0.0	1.6	0.0	17	0
10-149	F	1924	1990			DP	1943	114	1972	4	0.0	1.8	0.0	17	0
10-150	F	1889		1976	4123	DP	1919	13	1972	0	0.0	0.0	0.0	0	0
10-151	M	1887		1979	4124	CR	1915	520	1974	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-152	F	1923	1990			DP	1941	52	1972	2	0.0	0.9	0.0	9	0
10-153	F	1921		1983	1519	DP	1941	234	1972	1	0.0	0.4	0.0	4	0
10-160	F	1921		1985	174	DP	1941	208	1976	20	0.0	10.1	0.0	96	0
10-162	F	1931	1990			DP	1951	13	1974	3	0.0	1.0	0.0	9	0
10-164	F	1915		1988	4100	DP	1937	156	1974	0	0.0	0.1	0.0	1	0
10-165	F	1919	1990			DP	1942	416	1972	2	0.0	0.8	0.0	7	0
10-170	F	1923	1990			DP	1941	290	1980	20	0.0	11.0	0.0	107	0
10-171	F	1924	1990			DP	1942	156	1974	3	0.0	1.3	0.0	12	0
10-172	F	1930		1977	1729	DR	1948	60	1974	3	0.0	1.2	0.0	10	0
10-173	F	1915		1977	9520E	DP	1948	123	1973	0	0.0	0.0	0.0	0	0
10-180	F	1919	1990			DP	1941	728	1974	9	0.0	3.2	0.0	30	0
10-181	F	1912	1990			DP	1931	287	1978	2	0.0	1.2	0.0	12	0
10-190	F	1921	1990			DP	1951	106	1982	0	0.0	0.0	0.0	0	0
10-191	F	1940	1990			DP	1971	17	1972	2	0.0	0.1	0.0	0	0
10-192	F	1924	1990			DP	1942	78	1974	3	0.0	1.3	0.0	12	0
10-193	F	1921	1990			DP	1941	104	1972	3	0.0	1.2	0.0	12	0
10-195	F	1920	1990			DP	1937	1560	1982	9	0.0	3.6	0.0	32	0
10-198	F	1920	1990			DP	1946	378	1977	8	0.0	3.3	0.0	31	0
10-201	F	1918		1988	4249	DP	1946	1352	1972	9	0.0	1.0	0.0	8	0
10-202	F	1925	1990			DP	1942	53	1974	2	0.0	0.8	0.0	8	0
10-203	F	1926	1990			DP	1946	0	1974	2	0.0	0.7	0.0	6	0
10-204	F	1950	1990			DH	1971	43	1972	6	0.0	0.2	0.0	1	0
10-205	F	1923	1990			DP	1942	39	1972	1	0.0	0.5	0.0	5	0
10-206	F	1924	1990			DP	1943	230	1972	6	0.0	2.4	0.0	23	0
10-207	F	1923	1990			DP	1942	197	1982	5	0.0	3.4	0.0	32	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-208	F	1922	1990			DP	1942	7	1972	1	0.0	0.3	0.0	3	0
10-209	F	1920	1990			DP	1942	71	1982	7	0.0	4.4	0.0	42	0
10-210	F	1909		1989		DP	1926	1040	1972	17	0.0	9.0	0.0	91	0
10-212	M	1950	1990			DH	1971	55	1973	1	0.0	0.0	0.0	0	0
10-213	M	1951	1990			DH	1971	45	1973	1	0.0	0.0	0.0	0	0
10-214	F	1942	1990			DR	1972	30	1974	0	0.0	0.0	0.0	0	0
10-215	F	1921	1990			DP	1943	208	1972	1	0.0	0.4	0.0	3	0
10-216	F	1916	1990			DP	1946	1456	1973	2	0.0	0.2	0.0	2	0
10-218	F	1915	1990			DP	1934	492	1973	0	0.0	0.1	0.0	1	0
10-219	F	1916	1990			DP	1937	364	1979	10	0.0	6.3	0.0	62	0
10-221	F	1917	1990			DP	1941	676	1981	0	0.0	0.0	0.0	0	0
10-222	F	1919	1990			DP	1941	234	1972	0	0.0	0.0	0.0	0	0
10-225	F	1911	1990			DP	1933	1872	1981	4	0.0	1.6	0.0	14	0
10-226	F	1923	1990			DP	1941	1612	1972	3	0.0	0.3	0.0	3	0
10-227	M	1912		1990		DH	1928	2548	1977	6	0.0	1.1	0.0	7	0
10-228	F	1912		1981	3960	DP	1940	1508	1980	0	0.0	0.0	0.0	0	0
10-229	F	1920	1990			DP	1941	260	1972	1	0.0	0.5	0.0	4	0
10-230	F	1929	1990			DP	1948	13	1973	0	0.0	0.0	0.0	0	0
10-231	M	1968	1990			OF	1968	39	1972	1	0.0	0.1	0.0	0	0
10-232	M	1969	1990			OF	1969	39	1972	0	0.0	0.0	0.0	0	0
10-233	F	1919	1990			DP	1942	92	1976	2	0.0	1.0	0.0	10	0
10-234	F	1928		1972	8540E	DH	1959	676	1972	0	0.0	0.0	0.0	0	0
10-236	F	1919	1990			DP	1949	156	1974	0	0.0	0.0	0.0	0	0
10-237	F	1910	1990			DP	1940	156	1977	2	0.0	1.4	0.0	13	0
10-239	M	1908		1979	491	CR	1934	1300	1976	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-240	M	1906	1990			CR	1931	884	1981	0	0.0	0.0	0.0	0	0
10-241	F	1904		1978	2303	DP	1922	17	1972	0	0.0	0.0	0.0	0	0
10-242	F	1947	1990			DR	1966	156	1974	1	0.0	0.1	0.0	0	0
10-244	F	1916		1985	4272	DP	1943	1	1981	3	0.0	1.7	0.0	16	0
10-245	M	1914		1978	4123	DH	1941	104	1972	0	0.0	0.0	0.0	0	0
10-247	M	1915		1976	4121	DR	1948	364	1972	1	0.0	0.2	0.0	1	0
10-249	M	1943	1990			DH	1962	126	1973	1	0.0	0.1	0.0	0	0
10-250	F	1938	1990			DH	1956	23	1982	0	0.0	0.0	0.0	0	0
10-251	F	1923	1990			DP	1941	65	1974	2	0.0	0.9	0.0	9	0
10-252	F	1919	1990			DP	1935	273	1982	5	0.0	3.9	0.0	39	0
10-254	F	1905	1990			DH	1953	832	1976	0	0.0	0.0	0.0	0	0
10-256	F	1917	1990			DP	1940	78	1972	1	0.0	0.5	0.0	5	0
10-257	F	1932	1990			DH	1951	56	1982	1	0.0	0.2	0.0	2	0
10-258	F	1923	1990			DP	1943	26	1972	3	0.0	1.2	0.0	12	0
10-260	F	1913		1988		DP	1928	52	1978	2	0.0	1.6	0.0	17	0
10-261	F	1922	1990			DP	1941	28	1972	3	0.0	1.3	0.0	13	0
10-262	F	1919		1984	201	DP	1941	104	1973	2	0.0	0.8	0.0	7	0
10-263	F	1921	1990			DP	1941	130	1972	2	0.0	0.9	0.0	9	0
10-266	F	1905		1987		DP	1926	2236	1978	1	0.0	0.5	0.0	5	0
10-269	F	1925	1990			DP	1945	17	1972	0	0.0	0.0	0.0	0	0
10-270	F	1926	1990			DP	1946	104	1972	1	0.0	0.2	0.0	2	0
10-272	F	1915	1990			DP	1935	60	1979	5	0.0	4.0	0.0	41	0
10-273	F	1929	1990			DP	1948	22	1973	2	0.0	0.7	0.0	6	0
10-274	F	1924	1990			DP	1947	62	1973	3	0.0	1.2	0.0	12	0
10-276	F	1932	1990			DP	1951	6	1973	1	0.0	0.2	0.0	1	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-277	F	1915		1981	1579	DP	1946	154	1973	1	0.0	0.2	0.0	2	0
10-278	F	1908		1984	1621	DP	1929	1872	1981	2	0.0	0.7	0.0	7	0
10-279	F	1937	1990			DP	1955	728	1973	2	0.0	0.2	0.0	2	0
10-280	F	1904		1988		DH	1921	1768	1981	1	0.0	0.3	0.0	3	0
10-281	F	1931	1990			DP	1950	416	1973	1	0.0	0.1	0.0	1	0
10-282	F	1921		1974	1621	DP	1941	22	1974	2	0.0	1.0	0.0	8	0
10-283	F	1918		1986	402X	DP	1937	208	1974	0	0.0	0.1	0.0	1	0
10-284	F	1918	1990			DP	1936	1456	1974	3	0.0	0.9	0.0	8	0
10-285	M	1917	1990			DH	1935	81	1973	0	0.0	0.0	0.0	0	0
10-286	F	1937	1990			DH	1956	124	1973	0	0.0	0.1	0.0	1	0
10-287	F	1923	1990			DP	1944	2	1973	1	0.0	0.5	0.0	5	0
10-291	F	1916	1990			DP	1934	104	1981	2	0.0	1.6	0.0	16	0
10-292	F	1913		1975	174	DP	1934	102	1973	6	0.0	3.9	0.0	36	0
10-293	F	1938	1990			DH	1970	24	1973	0	0.0	0.0	0.0	0	0
10-294	F	1916	1990			DP	1934	416	1974	2	0.0	0.9	0.0	9	0
10-295	M	1923	1990			DR	1946	282	1973	2	0.0	0.5	0.0	3	0
10-296	F	1930	1990			DP	1948	50	1973	0	0.0	0.1	0.0	1	0
10-297	F	1929		1973	3039	DH	1969	66	1973	0	0.0	0.0	0.0	0	0
10-299	F	1923	1990			DP	1942	43	1973	6	0.0	2.9	0.0	29	0
10-300	F	1911	1990			DP	1940	1612	1977	0	0.0	0.1	0.0	1	0
10-301	M	1930	1990			DR	1948	74	1973	0	0.0	0.1	0.0	1	0
10-302	F	1917	1990			DH	1933	312	1973	0	0.0	0.0	0.0	0	0
10-304	F	1926	1990			DP	1950	364	1973	2	0.0	0.6	0.0	6	0
10-306	F	1907	1990			DP	1923	4	1981	4	0.0	5.4	0.0	57	0
10-307	F	1893		1948	1892	RI	1930	+0	1974	85	0.0	20.6	0.0	141	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-309	F	1925	1990			DP	1943	28	1973	2	0.0	0.8	0.0	7	0
10-310	F	1916		1989		DP	1935	53	1973	2	0.0	1.1	0.0	11	0
10-311	F	1919	1990			DP	1942	16	1973	0	0.0	0.1	0.0	1	0
10-312	F	1923	1990			DP	1942	16	1973	2	0.0	0.7	0.0	7	0
10-313	F	1924	1990			DP	1942	202	1973	9	0.0	3.9	0.0	38	0
10-314	F	1918	1990			DP	1943	119	1973	4	0.0	1.7	0.0	16	0
10-316	M	1946	1990			DH	1965	167	1973	2	0.0	0.2	0.0	1	0
10-318	M	1908		1990		DR	1970	364	1977	0	0.0	0.0	0.0	0	0
10-319	F	1912	1990			DH	1934	832	1973	6	0.0	2.6	0.0	26	0
10-320	M	1918	1990			DH	1939	1352	1973	1	0.0	0.3	0.0	2	0
10-321	F	1910	1990			DP	1942	1456	1981	0	0.0	0.0	0.0	0	0
10-322	F	1904	1990			DH	1936	1768	1981	2	0.0	0.5	0.0	5	0
10-323	F	1951	1990			DR	1973	52	1979	2	0.0	0.2	0.0	1	0
10-324	F	1912	1990			DP	1926	13	1978	0	0.0	0.0	0.0	0	0
10-325	M	1952	1990			DR	1970	22	1974	1	0.0	0.1	0.0	1	0
10-326	F	1954	1990			DH	1973	39	1974	0	0.0	0.0	0.0	0	0
10-327	M	1953	1990			DR	1973	52	1977	1	0.0	0.1	0.0	0	0
10-329	F	1914		1989		DH	1938	884	1979	0	0.0	0.0	0.0	0	0
10-330	F	1921	1990			DH	1945	520	1973	0	0.0	0.0	0.0	0	0
10-331	F	1911	1990			DH	1934	162	1981	2	0.0	1.7	0.0	17	0
10-332	F	1901		1986	4124	DP	1927	0	1978	0	0.00204	0.0	0.0	0	0
10-333	F	1915		1984	4123	DP	1941	208	1973	1	0.0	0.4	0.0	4	0
10-334	F	1921	1990			DP	1943	26	1973	0	0.0	0.0	0.0	0	0
10-335	F	1939	1990			DH	1969	24	1973	0	0.0	0.0	0.0	0	0
10-336	F	1923	1990			DH	1943	1092	1973	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-337	M	1892		1971	4109	CR	1913	260	1974	1	0.0	0.8	0.0	6	0
10-339	F	1902		1988		DP	1925	1	1976	0	0.00260	0.0	0.0	0	0
10-340	F	1920	1990			CR	1942	104	1974	6	0.0	2.8	0.0	27	0
10-341	F	1919	1990			DP	1939	312	1973	1	0.0	0.5	0.0	5	0
10-347	M	1947	1990			OF	1947	39	1973	1	0.0	0.4	0.0	2	0
10-348	F	1921	1990			DP	1941	104	1974	0	0.0	0.0	0.0	0	0
10-350	F	1924	1990			DP	1941	27	1973	1	0.0	0.4	0.0	4	0
10-351	M	1931	1990			DR	1964	14	1973	1	0.0	0.2	0.0	1	0
10-352	F	1926	1990			DR	1947	104	1974	1	0.0	0.3	0.0	3	0
10-353	F	1922	1990			DP	1942	21	1973	1	0.0	0.2	0.0	2	0
10-356	F	1915	1990			DH	1948	46	1980	1	0.0	0.6	0.0	5	0
10-357	F	1923	1990			DP	1942	68	1973	3	0.0	1.5	0.0	14	0
10-358	F	1920	1990			DP	1946	16	1973	3	0.0	1.1	0.0	10	0
10-359	M	1950	1990			DR	1971	32	1973	3	0.0	0.1	0.0	1	0
10-360	F	1919	1990			DP	1941	46	1975	0	0.0	0.0	0.0	0	0
10-362	F	1922	1990			DP	1941	364	1973	4	0.0	1.7	0.0	16	0
10-365	F	1920	1990			DP	1939	260	1973	0	0.0	0.1	0.0	1	0
10-367	F	1919	1990			DP	1940	260	1973	1	0.0	0.3	0.0	3	0
10-369	F	1921		1981	513	DP	1941	104	1978	1	0.0	0.3	0.0	3	0
10-370	F	1916	1990			DP	1934	312	1981	1	0.0	0.9	0.0	9	0
10-375	F	1924	1990			DP	1943	20	1973	1	0.0	0.5	0.0	5	0
10-377	F	1898		1983	1531	DH	1923	1976	1981	0	0.0	0.0	0.0	0	0
10-378	F	1906	1990			DH	1946	520	1981	0	0.0	0.0	0.0	0	0
10-379	F	1917	1990			DP	1941	89	1983	21	0.0	14.0	0.0	138	0
10-381	F	1927	1990			DP	1946	27	1973	6	0.0	2.2	0.0	21	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-382	F	1923	1990			DP	1942	119	1973	5	0.0	2.4	0.0	23	0
10-384	F	1919	1990			DP	1943	884	1973	1	0.0	0.4	0.0	4	0
10-385	F	1921	1990			DH	1964	16	1973	0	0.0	0.0	0.0	0	0
10-386	F	1933	1990			DP	1954	56	1973	1	0.0	0.4	0.0	3	0
10-387	F	1928	1990			DP	1947	15	1973	0	0.0	0.1	0.0	1	0
10-388	F	1927	1990			DP	1945	51	1983	0	0.0	0.0	0.0	0	0
10-389	F	1919	1990			DP	1943	24	1973	0	0.0	0.0	0.0	0	0
10-390	F	1923	1990			DP	1942	38	1973	3	0.0	1.4	0.0	14	0
10-392	F	1903	1990			DH	1932	520	1973	0	0.0	0.0	0.0	0	0
10-393	F	1907		1985	4109	DP	1925	208	1981	1	0.0	1.4	0.0	14	0
10-394	F	1907		1976	5719	DP	1923	728	1974	1	0.0	0.5	0.0	5	0
10-395	F	1908	1990			DP	1925	260	1981	0	0.0	0.0	0.0	0	0
10-397	F	1927	1990			DP	1946	16	1973	1	0.0	0.3	0.0	3	0
10-398	F	1918	1990			DP	1951	624	1973	1	0.0	0.2	0.0	1	0
10-409	F	1921	1990			DP	1943	118	1973	0	0.0	0.0	0.0	0	0
10-410	F	1926	1990			DP	1946	5	1973	0	0.0	0.0	0.0	0	0
10-411	F	1920		1981	4123	DP	1942	14	1973	3	0.0	1.4	0.0	12	0
10-412	F	1908		1990		DP	1925	13	1976	1	0.0	0.7	0.0	7	0
10-414	F	1926	1990			DP	1944	511	1973	1	0.0	0.4	0.0	4	0
10-415	F	1943	1990			DR	1973	8	1974	0	0.0	0.0	0.0	0	0
10-416	F	1953	1990			DP	1972	290	1979	0	0.0	0.0	0.0	0	0
10-419	M	1913	1990			CR	1936	2184	1978	2	0.0	0.4	0.0	2	0
10-432	F	1920	1990			DP	1940	104	1975	0	0.0	0.2	0.0	2	0
10-438	F	1907		1985	5193	DP	1925	17	1977	14	0.0	13.4	0.0	140	0
10-439	F	1925	1990			DP	1943	20	1973	2	0.0	0.9	0.0	9	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-440	F	1920	1990			DP	1948	1	1973	0	0.0	0.0	0.0	0	0
10-442	F	1932	1990			DP	1951	8	1973	0	0.0	0.0	0.0	0	0
10-443	F	1899	1990			DP	1926	234	1979	34	0.0	30.7	0.0	329	0
10-444	F	1927	1990			DP	1949	4	1973	1	0.0	0.2	0.0	2	0
10-445	F	1924	1990			DP	1943	2	1973	2	0.0	0.9	0.0	9	0
10-446	F	1920	1990			DP	1940	3	1973	1	0.0	0.4	0.0	4	0
10-447	F	1929	1990			DP	1947	5	1973	6	0.0	2.2	0.0	21	0
10-449	F	1923	1990			DP	1943	0	1976	4	0.0	1.8	0.0	18	0
10-451	F	1921	1990			DP	1943	3	1973	0	0.0	0.1	0.0	1	0
10-453	F	1927	1990			DP	1943	1	1973	0	0.0	0.1	0.0	1	0
10-454	F	1926	1990			DP	1942	5	1973	0	0.0	0.2	0.0	2	0
10-455	F	1909	1990			DP	1928	104	1977	0	0.0	0.2	0.0	2	0
10-457	F	1921	1990			DP	1941	65	1973	1	0.0	0.6	0.0	6	0
10-458	M	1927	1990			DP	1954	1040	1973	24	0.0	2.1	0.0	12	0
10-459	F	1923	1990			DP	1956	832	1973	0	0.0	0.0	0.0	0	0
10-460	F	1936	1990			DP	1959	676	1973	0	0.0	0.0	0.0	0	0
10-464	M	1940	1990			DR	1961	12	1973	0	0.0	0.0	0.0	0	0
10-465	F	1924	1990			DP	1942	8	1973	0	0.0	0.0	0.0	0	0
10-470	F	1924	1990			DP	1942	179	1973	0	0.0	0.0	0.0	0	0
10-471	F	1924	1990			DP	1943	34	1973	3	0.0	1.2	0.0	12	0
10-472	F	1928	1990			DP	1947	12	1973	0	0.0	0.0	0.0	0	0
10-473	F	1926	1990			DP	1945	18	1973	0	0.0	0.2	0.0	2	0
10-474	F	1921	1990			DP	1946	77	1974	2	0.0	0.8	0.0	8	0
10-475	F	1927	1990			DR	1946	90	1973	0	0.0	0.0	0.0	0	0
10-476	F	1928	1990			DP	1946	12	1973	1	0.0	0.2	0.0	2	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-477	F	1924	1990			DP	1944	42	1975	2	0.0	1.1	0.0	11	0
10-478	F	1922	1990			DP	1942	11	1973	0	0.0	0.0	0.0	0	0
10-479	F	1926		1985	174	DP	1946	11	1973	0	0.0	0.0	0.0	0	0
10-480	F	1924	1990			DP	1943	4	1973	0	0.0	0.2	0.0	2	0
10-481	F	1925	1990			DP	1942	5	1973	1	0.0	0.6	0.0	5	0
10-482	F	1925	1990			DP	1943	28	1973	4	0.0	1.9	0.0	19	0
10-483	M	1934	1990			DH	1951	5	1973	2	0.0	0.5	0.0	3	0
10-485	F	1918	1990			DP	1948	4	1973	0	0.0	0.1	0.0	1	0
10-486	F	1919	1990			DP	1942	32	1973	0	0.0	0.1	0.0	1	0
10-487	F	1924	1990			DP	1943	220	1973	0	0.0	0.1	0.0	1	0
10-488	F	1921	1990			DP	1942	20	1973	0	0.0	0.0	0.0	0	0
10-490	F	1922	1990			DP	1943	20	1974	8	0.0	3.6	0.0	35	0
10-492	F	1925		1981	1621	DP	1951	326	1973	2	0.0	0.4	0.0	4	0
10-494	F	1913		1989		DP	1939	312	1973	1	0.0	0.3	0.0	3	0
10-495	F	1924	1990			DP	1942	312	1973	0	0.0	0.0	0.0	0	0
10-496	F	1922	1990			DP	1940	108	1975	0	0.0	0.0	0.0	0	0
10-501	F	1928	1990			DP	1946	15	1973	2	0.0	0.7	0.0	7	0
10-502	F	1928	1990			DP	1946	13	1973	2	0.0	0.7	0.0	7	0
10-505	F	1933	1990			DP	1951	3	1973	2	0.0	0.6	0.0	6	0
10-506	F	1920		1986		DR	1946	4	1973	0	0.0	0.2	0.0	1	0
10-510	F	1924	1990			DR	1942	26	1973	1	0.0	0.6	0.0	5	0
10-511	F	1923	1990			DP	1943	12	1973	5	0.0	2.2	0.0	22	0
10-512	F	1936	1990			DP	1965	1	1973	0	0.0	0.0	0.0	0	0
10-518	F	1905		1989		NU	1928	1196	1978	1	0.0	0.6	0.0	6	0
10-520	F	1924	1990			DP	1942	5	1973	1	0.0	0.5	0.0	5	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-521	F	1923	1990			DP	1955	416	1973	1	0.0	0.3	0.0	2	0
10-523	F	1922	1990			DP	1942	17	1973	0	0.0	0.0	0.0	0	0
10-525	F	1928	1990			DP	1947	1	1973	1	0.0	0.3	0.0	3	0
10-530	F	1952	1990			DH	1971	52	1973	3	0.0	0.1	0.0	1	0
10-531	F	1924	1990			DP	1946	1	1973	2	0.0	0.7	0.0	7	0
10-532	F	1916	1990			DP	1942	2	1973	1	0.0	0.6	0.0	5	0
10-533	F	1925	1990			DP	1943	5	1973	2	0.0	0.9	0.0	8	0
10-534	F	1925		1985	4109	DP	1946	54	1973	2	0.0	0.8	0.0	7	0
10-535	F	1927	1990			DP	1946	16	1973	1	0.0	0.4	0.0	4	0
10-536	F	1927	1990			DP	1942	1	1973	1	0.0	0.5	0.0	5	0
10-538	M	1896		1978	1621	DH	1938	2028	1977	1	0.0	0.2	0.0	1	0
10-540	M	1917		1978	1621	DP	1939	1768	1973	2	0.0	0.3	0.0	1	0
10-543	M	1891	1990			CR	1916	26	1973	3	0.0	3.6	0.0	28	0
10-546	F	1907		1985	2699	DH	1929	208	1979	6	0.0	5.2	0.0	52	0
10-549	F	1919	1990			DP	1941	62	1973	4	0.0	2.0	0.0	20	0
10-550	F	1914	1990			DP	1965	230	1979	1	0.0	0.2	0.0	1	0
10-557	F	1921	1990			DP	1942	43	1974	4	0.0	1.9	0.0	18	0
10-558	M	1927	1990			RA	1951	+0	1973	5	0.0	1.5	0.0	10	0
10-559	F	1919	1990			DP	1941	69	1973	2	0.0	0.8	0.0	8	0
10-560	F	1923	1990			DP	1942	96	1973	4	0.0	1.6	0.0	16	0
10-561	M	1906		1986	4123	CR	1927	52	1978	2	0.0	2.0	0.0	14	0
10-566	M	1914		1977	1621	RD	1930	13	1976	5	0.00334	4.1	3.4	29	31
10-567	F	1913	1987			NU	1931	572	1963	2	0.0	0.6	0.0	6	0
10-569	F	1925	1990			DP	1946	1	1975	0	0.0	0.0	0.0	0	0
10-570	M	1907		1983	185	CR	1934	780	1977	2	0.0	0.9	0.0	6	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-573	F	1922	1990			DP	1944	14	1973	3	0.0	1.2	0.0	11	0
10-574	M	1908		1987		DP	1930	2236	1980	2	0.0	0.8	0.0	5	0
10-575	F	1930	1990			DP	1948	1040	1973	4	0.0	0.7	0.0	6	0
10-582	F	1938	1990			DP	1965	416	1973	1	0.0	0.0	0.0	0	0
10-583	M	1918	1990			CR	1939	1352	1973	0	0.0	0.0	0.0	0	0
10-584	F	1925	1990			DP	1942	3	1973	1	0.0	0.4	0.0	4	0
10-585	M	1908	1990			CR	1930	52	1978	1	0.0	1.1	0.0	8	0
10-587	M	1946	1990			DH	1966	416	1973	1	0.0	0.1	0.0	0	0
10-588	F	1910	1990			DP	1927	2	1974	0	0.00330	0.0	0.0	0	0
10-589	M	1938	1990			DR	1971	78	1973	2	0.0	0.1	0.0	0	0
10-590	M	1912		1979	2901	CR	1948	728	1979	0	0.0	0.0	0.0	0	0
10-592	M	1899		1984	4122	CR	1923	1300	1978	1	0.0	0.7	0.0	5	0
10-594	F	1917		1984	174	DP	1943	5	1973	5	0.0	2.3	0.0	21	0
10-595	F	1908		1986	471X	DP	1928	104	1977	6	0.0	5.3	0.0	54	0
10-596	F	1909	1990			DP	1927	6	1973	6	0.0	4.6	0.0	48	0
10-597	F	1911	1990			DP	1928	17	1976	2	0.0	1.8	0.0	19	0
10-598	F	1914		1979	4123	DP	1934	156	1973	1	0.0	0.5	0.0	5	0
10-601	M	1920		1981	185	RA	1951	0	1975	0	0.0	0.0	0.0	0	0
10-606	F	1910	1990			DH	1928	468	1975	0	0.0	0.0	0.0	0	0
10-608	F	1917	1990			DP	1939	14	1975	1	0.0	0.3	0.0	3	0
10-609	F	1925	1990			DP	1943	42	1973	2	0.0	0.7	0.0	7	0
10-610	F	1920		1984	4109	DP	1941	22	1975	2	0.0	0.9	0.0	8	0
10-611	F	1924	1990			DP	1942	13	1973	2	0.0	0.9	0.0	9	0
10-613	F	1919	1990			DP	1945	12	1973	0	0.0	0.0	0.0	0	0
10-614	F	1915		1986	2049	DP	1942	30	1975	1	0.0	0.5	0.0	4	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-616	F	1929	1990			DP	1948	15	1973	2	0.0	0.6	0.0	6	0
10-617	F	1922	1990			DP	1942	182	1974	10	0.0	4.5	0.0	44	0
10-618	F	1923	1990			DP	1944	54	1975	0	0.0	0.1	0.0	1	0
10-621	M	1905	1990			CR	1925	1716	1979	1	0.0	0.4	0.0	3	0
10-623	M	1917	1990			CR	1938	1144	1973	1	0.0	0.3	0.0	2	0
10-627	M	1911	1990			IR	1928	208	1974	4	0.00420	3.1	2.5	23	23
10-628	M	1906	1990			CR	1927	156	1976	0	0.0	0.0	0.0	0	0
10-630	F	1915	1990			DP	1937	13	1981	0	0.0	0.0	0.0	0	0
10-631	F	1929	1990			DP	1946	26	1974	0	0.0	0.0	0.0	0	0
10-635	F	1922	1990			DP	1943	156	1973	3	0.0	1.0	0.0	10	0
10-643	M	1853		1928	4459	RI	1928	0	1978	316	0.0	4.2	0.0	1	0
10-644	M	1870		1927	4123	RI	1927	0	1975	5300	0.0	36.1	0.0	2	0
10-645	F	1930	1990			DH	1948	90	1973	0	0.0	0.0	0.0	0	0
10-648	F	1923	1990			DP	1942	30	1974	2	0.0	0.8	0.0	8	0
10-649	F	1921	1990			DP	1942	15	1973	2	0.0	0.7	0.0	7	0
10-650	F	1926	1990			DP	1946	59	1973	8	0.0	2.9	0.0	28	0
10-651	F	1923	1990			DP	1942	260	1974	0	0.0	0.0	0.0	0	0
10-653	F	1926		1979	4510	DP	1946	16	1973	0	0.0	0.0	0.0	0	0
10-655	F	1922	1990			DP	1947	2	1978	2	0.0	1.0	0.0	10	0
10-656	F	1923	1990			DP	1942	20	1973	1	0.0	0.3	0.0	3	0
10-657	F	1922		1976	4109	DP	1943	13	1973	1	0.0	0.5	0.0	4	0
10-658	F	1906	1990			DP	1927	208	1974	6	0.0	4.7	0.0	49	0
10-659	F	1904		1980	4123	DP	1927	52	1974	0	0.0	0.3	0.0	3	0
10-660	F	1924	1990			DP	1942	172	1973	18	0.0	7.8	0.0	76	0
10-661	F	1926		1973	2050	DP	1945	23	1977	10	0.0	4.1	0.0	33	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-662	F	1909	1990			DP	1930	13	1977	2	0.0	2.0	0.0	20	0
10-664	F	1925	1990			DP	1943	1	1973	3	0.0	1.3	0.0	13	0
10-665	F	1927	1990			DP	1946	104	1973	1	0.0	0.4	0.0	4	0
10-666	F	1924	1990			DP	1943	13	1974	1	0.0	0.3	0.0	3	0
10-667	F	1908		1974	191	DP	1925	52	1973	7	0.0	5.7	0.0	56	0
10-668	F	1925	1990			DP	1943	19	1973	1	0.0	0.4	0.0	3	0
10-671	M	1932	1990			CS	1957	988	1982	1	0.0	0.2	0.0	1	0
10-672	M	1916		1980	1560	CR	1936	1040	1974	0	0.0	0.0	0.0	0	0
10-673	M	1911		1976	4329	CR	1932	364	1973	0	0.0	0.0	0.0	0	0
10-675	F	1921	1990			RW	1929	26	1982	4	0.0	4.0	0.0	41	0
10-681	M	1922	1990			IM	1941	1508	1982	1	0.0	0.2	0.0	1	0
10-683	F	1924	1990			DP	1942	14	1973	0	0.0	0.0	0.0	0	0
10-684	M	1927		1987	4123	DH	1950	104	1974	1	0.0	0.4	0.0	2	0
10-688	F	1923		1976	174	DP	1942	12	1974	4	0.0	2.1	0.0	19	0
10-689	F	1919		1982	1621	DP	1943	26	1974	3	0.0	1.2	0.0	11	0
10-696	F	1911	1990			DP	1929	15	1977	8	0.0	7.1	0.0	73	0
10-714	F	1908	1990			DP	1925	57	1979	1	0.00126	1.1	0.8	11	11
10-718	F	1910		1979	450	DP	1925	0	1979	7	0.0	7.6	0.0	77	0
10-723	F	1911	1990			DP	1929	15	1982	2	0.0	1.9	0.0	20	0
10-725	M	1927	1990			RA	1952	1	1973	5	0.0	1.5	0.0	9	0
10-728	F	1923	1990			DP	1946	2	1974	0	0.0	0.0	0.0	0	0
10-729	F	1902	1990			CR	1920	832	1973	1	0.0	0.8	0.0	8	0
10-730	F	1907		1988		DP	1928	260	1979	1	0.0	0.8	0.0	8	0
10-731	M	1921	1990			DH	1951	1196	1974	2	0.0	0.2	0.0	1	0
10-732	M	1924	1990			DH	1950	1300	1974	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-736	F	1929	1990			DP	1948	9	1974	0	0.0	0.0	0.0	0	0
10-738	M	1923		1990		DR	1965	6	1974	3	0.0	0.4	0.0	2	0
10-739	F	1931	1990			DP	1951	7	1974	1	0.0	0.2	0.0	1	0
10-741	F	1927		1989		DP	1945	60	1977	1	0.0	0.5	0.0	5	0
10-742	F	1929	1990			DH	1946	1	1974	2	0.0	0.7	0.0	7	0
10-744	F	1890		1978	5699	RI	1925	0	1975	120	0.0	88.1	0.0	973	0
10-754	F	1881		1977	4123	RI	1925	0	1975	12	0.0	11.3	0.0	113	0
10-786	F	1866		1928	0389	RI	1927	0	1976	1360	0.0	37.7	0.0	26	0
10-807	M	1894		1976	4100	RI	1925	1	1976	388	0.0	183.3	0.0	1711	0
10-825	M	1904		1985	185	RI	1927	0	1978	941	0.0	256.3	0.0	3196	0
10-831	M	1879		1926	4003	RI	1925	+0	1977	786	0.0	32.2	0.0	30	0
10-840	M	1869		1926	1519	RI	1925	0	1976	390	0.0	8.9	0.0	3	0
10-850	F	1925	1990			DP	1943	0	1974	1	0.0	0.5	0.0	5	0
10-851	F	1921	1990			DP	1951	139	1974	0	0.0	0.0	0.0	0	0
10-852	F	1905		1980	4272	DP	1923	13	1974	0	0.01300	0.0	0.0	0	0
10-853	F	1919	1990			DP	1947	1300	1974	1	0.0	0.2	0.0	1	0
10-854	M	1909	1990			CM	1928	104	1979	1	0.0	1.0	0.0	7	0
10-855	F	1928	1990			DP	1946	28	1976	7	0.0	2.9	0.0	28	0
10-856	F	1952	1990			DP	1973	6	1974	1	0.0	0.0	0.0	0	0
10-859	F	1951	1990			DH	1973	0	1974	0	0.0	0.0	0.0	0	0
10-860	F	1925	1990			DH	1962	7	1974	7	0.0	1.2	0.0	10	0
10-861	F	1954	1990			DP	1973	22	1974	1	0.0	0.0	0.0	0	0
10-862	F	1928	1990			DP	1946	10	1974	0	0.0	0.0	0.0	0	0
10-864	M	1906		1990		DP	1949	1560	1979	0	0.0	0.0	0.0	0	0
10-866	F	1900		1990		DP	1920	12	1979	8	0.00775	7.9	71.6	92	942

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-867	F	1915		1983	4100	DH	1929	209	1974	0	0.0	0.0	0.0	0	0
10-869	F	1902	1990			DP	1927	132	1979	2	0.00181	2.0	1.6	20	20
10-870	F	1911		1978	1970	DH	1944	650	1974	0	0.0	0.0	0.0	0	0
10-874	F	1924	1990			DP	1942	728	1974	4	0.0	1.4	0.0	13	0
10-880	M	1912	1990			CR	1935	156	1974	0	0.0	0.0	0.0	0	0
10-883	F	1883		1935	7130	RD	1930	+0	1975	27	0.0	2.3	0.0	8	0
10-890	F	1912		1990		DP	1927	2	1979	0	0.00181	0.0	0.0	0	0
10-893	F	1926		1987		DP	1943	78	1977	5	0.0	2.7	0.0	26	0
10-894	F	1924	1990			DP	1942	38	1974	1	0.0	0.3	0.0	3	0
10-895	F	1925	1990			DP	1943	9	1974	2	0.0	0.7	0.0	7	0
10-896	F	1923	1990			DP	1941	8	1974	0	0.0	0.1	0.0	1	0
10-897	F	1930		1989		DR	1951	208	1975	3	0.0	0.8	0.0	7	0
10-901	F	1910	1990			DP	1924	3	1975	0	0.01160	0.0	0.0	0	0
10-902	M	1905		1990		CR	1928	17	1982	0	0.0	0.0	0.0	0	0
10-903	F	1909	1990			DP	1943	2	1976	0	0.0	0.2	0.0	2	0
10-905	F	1928	1990			DP	1946	10	1974	0	0.0	0.1	0.0	1	0
10-906	F	1921		1980	1579	DH	1969	0	1976	1	0.0	0.1	0.0	0	0
10-907	F	1910		1989		DP	1946	5	1979	1	0.0	0.5	0.0	4	0
10-908	F	1928	1990			DP	1946	4	1974	1	0.0	0.4	0.0	4	0
10-909	F	1919	1990			DP	1941	4	1974	2	0.0	1.2	0.0	11	0
10-911	F	1928	1990			DP	1947	2	1974	2	0.0	0.7	0.0	7	0
10-915	F	1931	1990			DP	1953	0	1974	1	0.0	0.2	0.0	2	0
10-916	F	1915	1990			DP	1946	2	1974	0	0.0	0.0	0.0	0	0
10-918	F	1907	1990			DP	1923	0	1981	2	0.00547	1.9	11.3	21	144
10-919	F	1924	1990			DP	1943	8	1974	2	0.0	0.7	0.0	7	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-920	F	1929	1990			DP	1947	4	1977	0	0.0	0.0	0.0	0	0
10-921	F	1905		1983	4270	DP	1923	1	1977	0	0.00907	0.0	0.0	0	0
10-928	M	1918		1988		RA	1948	0	1958	1	0.0	0.1	0.0	1	0
10-931	M	1911	1990			DP	1946	1040	1979	4	0.0	1.3	0.0	8	0
10-932	M	1903		1988		RA	1919	208	1979	15	0.0	17.9	0.0	137	0
10-933	F	1924	1990			DP	1943	3	1974	2	0.0	0.9	0.0	9	0
10-938	F	1952	1990			DP	1971	8	1974	0	0.0	0.0	0.0	0	0
10-940	F	1939	1990			DR	1958	4	1974	1	0.0	0.2	0.0	1	0
10-941	F	1928	1990			DP	1948	13	1974	1	0.0	0.2	0.0	2	0
10-944	F	1922	1990			DP	1951	6	1974	0	0.0	0.0	0.0	0	0
10-945	F	1915	1990			DP	1943	12	1979	4	0.0	2.2	0.0	22	0
10-948	F	1923	1990			DP	1943	3	1974	0	0.0	0.2	0.0	2	0
10-949	F	1925	1990			DP	1943	0	1974	2	0.0	0.9	0.0	9	0
10-950	F	1922	1990			DP	1943	1	1974	5	0.0	2.2	0.0	21	0
10-951	F	1916	1990			DP	1943	4	1980	2	0.0	1.4	0.0	14	0
10-952	F	1911	1990			DP	1927	10	1980	1	0.00160	1.4	1.3	14	16
10-953	F	1908		1989		DP	1923	49	1979	15	0.00770	12.8	77.2	151	1022
10-955	F	1922	1990			DP	1942	104	1974	1	0.0	0.5	0.0	5	0
10-957	F	1922	1990			DP	1941	130	1974	1	0.0	0.5	0.0	5	0
10-958	F	1931	1990			DP	1951	13	1975	3	0.0	1.0	0.0	10	0
10-959	F	1929	1990			DP	1946	2	1974	4	0.0	1.5	0.0	14	0
10-962	F	1916	1990			DH	1934	27	1978	0	0.0	0.0	0.0	0	0
10-963	F	1901		1986	2381	DP	1919	10	1975	647	0.00170	139.3	194.1	3024	3034
10-966	F	1908	1990			DP	1929	4	1974	0	0.0	0.0	0.0	0	0
10-967	F	1924	1990			DP	1943	2	1974	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
10-969	M	1920		1981	4109	DH	1969	52	1976	0	0.0	0.0	0.0	0	0
10-970	F	1955	1990			DH	1973	22	1974	2	0.0	0.1	0.0	0	0
10-971	F	1952	1990			DP	1973	22	1975	1	0.0	0.0	0.0	0	0
10-972	F	1926	1990			DP	1947	5	1974	0	0.0	0.1	0.0	1	0
10-974	F	1924	1990			DP	1941	48	1974	0	0.0	0.0	0.0	0	0
10-975	F	1929	1990			DP	1947	13	1974	0	0.0	0.0	0.0	0	0
10-977	F	1923	1990			DP	1943	38	1974	6	0.0	2.8	0.0	27	0
10-978	M	1927	1985			DH	1943	1612	1974	4	0.0	0.5	0.0	3	0
10-979	F	1925	1990			DP	1943	13	1974	1	0.0	0.3	0.0	3	0
10-980	F	1926		1989	1621	DR	1945	1	1974	1	0.0	0.4	0.0	4	0
10-981	F	1928	1990			DR	1946	0	1974	0	0.0	0.0	0.0	0	0
10-987	F	1926	1990			DP	1946	26	1974	1	0.0	0.6	0.0	5	0
10-988	M	1952		1974	890E	DH	1973	22	1974	0	0.0	0.0	0.0	0	0
10-989	F	1927		1989	1538	DH	1958	3	1975	1	0.0	0.1	0.0	1	0
10-990	F	1920	1990			DH	1943	20	1974	0	0.0	0.0	0.0	0	0
10-991	M	1901		1985	4389	DR	1941	1716	1979	2	0.0	0.6	0.0	3	0
10-992	F	1919	1990			DP	1942	39	1974	0	0.0	0.0	0.0	0	0
10-993	F	1904		1981	2509	DH	1942	4	1979	3	0.0	1.8	0.0	16	0
10-996	F	1900		1985	4270	DH	1943	260	1979	1	0.0	0.5	0.0	5	0
10-997	F	1926	1990			DH	1945	572	1979	0	0.0	0.0	0.0	0	0
10-998	F	1909	1990			DH	1942	988	1978	0	0.0	0.0	0.0	0	0
11-002	F	1919	1990			DP	1941	728	1979	0	0.0	0.0	0.0	0	0
11-003	F	1919	1990			DH	1942	+0	1974	3	0.0	1.5	0.0	14	0
11-004	M	1924		1984	4109	DP	1946	702	1979	1	0.0	0.4	0.0	2	0
11-005	M	1926	1990			DP	1948	1612	1979	3	0.0	0.4	0.0	2	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
11-009	F	1913		1985	1621	DH	1942	884	1979	0	0.0	0.0	0.0	0	0
11-010	F	1922	1990			DH	1942	598	1979	0	0.0	0.0	0.0	0	0
11-015	F	1907		1984	486X	DP	1925	2	1976	0	0.01000	0.0	0.0	0	0
11-016	F	1906	1990			DP	1924	17	1978	24	0.00803	18.0	92.6	219	1239
11-017	F	1906		1989		DP	1923	1	1977	0	0.00907	0.0	0.0	0	0
11-018	F	1908		1981	1621	DP	1925	5	1974	0	0.00330	0.0	0.0	0	0
11-021	F	1907	1990			DH	1931	282	1978	0	0.00203	0.0	0.0	0	0
11-023	F	1911	1990			DP	1927	2	1975	0	0.00290	0.0	0.0	0	0
11-026	F	1916		1989		DP	1941	52	1981	0	0.0	0.0	0.0	0	0
11-027	F	1910		1979	4123	DP	1948	312	1978	0	0.0	0.0	0.0	0	0
11-028	F	1925		1987		DP	1944	78	1974	0	0.0	0.0	0.0	0	0
11-030	F	1928	1990			DH	1951	112	1975	0	0.0	0.0	0.0	0	0
11-032	M	1931		1986		CR	1956	936	1974	3	0.0	0.3	0.0	1	0
11-033	M	1951	1990			CR	1973	104	1975	0	0.0	0.0	0.0	0	0
11-034	M	1915	1990			CR	1934	2184	1977	51	0.0	9.8	0.0	61	0
11-035	M	1949	1990			IR	1973	60	1977	0	0.0	0.0	0.0	0	0
11-036	M	1914	1990			CR	1946	1716	1979	6	0.0	0.8	0.0	5	0
11-038	M	1914	1990			IR	1940	1456	1979	11	0.0	3.4	0.0	21	0
11-040	M	1915	1990			CR	1939	2132	1980	6	0.0	0.9	0.0	5	0
11-042	M	1923	1990			CR	1946	1456	1974	5	0.0	0.6	0.0	3	0
11-045	M	1915		1976	1621	CR	1943	1560	1974	27	0.0	4.1	0.0	19	0
11-049	F	1908	1990			DP	1923	13	1981	2	0.00563	2.4	14.1	26	180
11-053	F	1905	1990			DP	1923	0	1977	0	0.00907	0.0	0.0	0	0
11-056	F	1908		1985	4109	DP	1927	40	1974	2	0.00330	1.7	1.5	17	19
11-059	F	1925	1990			DP	1943	13	1974	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
11-065	F	1928	1990			DH	1943	13	1974	0	0.0	0.0	0.0	0	0
11-070	F	1924		1990	8147E	DP	1945	26	1974	1	0.0	0.2	0.0	2	0
11-071	F	1935	1990			DH	1967	2	1974	2	0.0	0.2	0.0	1	0
11-081	M	1921	1990			DH	1941	1300	1978	1	0.0	0.4	0.0	2	0
11-086	F	1919	1990			DP	1941	208	1977	2	0.0	1.0	0.0	10	0
11-087	M	1923	1990			DH	1941	52	1977	3	0.0	1.7	0.0	12	0
11-089	F	1920		1980	4109	DP	1942	182	1978	2	0.0	1.2	0.0	11	0
11-092	F	1911	1990			DP	1943	52	1977	0	0.0	0.0	0.0	0	0
11-103	F	1918	1990			DP	1942	100	1980	2	0.0	0.9	0.0	9	0
11-104	F	1905		1985	174	DH	1942	43	1978	1	0.0	0.6	0.0	5	0
11-107	F	1916	1990			DP	1942	52	1977	0	0.0	0.2	0.0	2	0
11-108	F	1923	1990			DH	1941	208	1977	1	0.0	0.4	0.0	4	0
11-112	F	1916	1990			DP	1943	52	1977	1	0.0	0.4	0.0	4	0
11-115	F	1909	1990			DP	1942	104	1979	1	0.0	0.5	0.0	5	0
11-118	F	1920	1990			DP	1942	260	1979	0	0.0	0.0	0.0	0	0
11-119	F	1918	1990			DP	1941	117	1976	0	0.0	0.0	0.0	0	0
11-120	F	1919	1990			DP	1943	39	1979	1	0.0	0.5	0.0	4	0
11-121	F	1909	1990			DP	1950	520	1977	0	0.0	0.1	0.0	1	0
11-129	F	1923	1990			DP	1942	182	1978	0	0.0	0.0	0.0	0	0
11-131	F	1933	1990			DP	1952	104	1978	0	0.0	0.0	0.0	0	0
11-143	F	1923		1989		DP	1940	104	1977	0	0.0	0.2	0.0	2	0
11-147	F	1907	1990			DP	1943	52	1981	0	0.0	0.1	0.0	1	0
11-161	F	1921	1990			DP	1940	130	1976	0	0.0	0.0	0.0	0	0
11-166	F	1917	1990			DP	1942	137	1978	2	0.0	0.9	0.0	8	0
11-168	F	1918	1990			DP	1942	90	1979	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
11-173	F	1911		1980	7960	DH	1943	104	1980	0	0.0	0.0	0.0	0	0
11-176	F	1915		1987		DP	1942	208	1977	2	0.0	1.1	0.0	11	0
11-184	F	1919	1990			DP	1941	260	1978	2	0.0	1.0	0.0	10	0
11-190	F	1921	1990			DP	1942	156	1978	1	0.0	0.5	0.0	5	0
11-192	F	1924	1990			DH	1943	104	1977	1	0.0	0.5	0.0	4	0
11-196	F	1916		1982	1621	DH	1941	208	1977	1	0.0	0.3	0.0	3	0
11-207	M	1917	1990			DP	1939	208	1974	0	0.0	0.0	0.0	0	0
11-223	F	1917	1990			DH	1943	104	1978	2	0.0	1.0	0.0	9	0
11-230	F	1904		1988		DR	1942	104	1981	0	0.0	0.0	0.0	0	0
11-231	F	1912		1986		DP	1942	34	1980	0	0.0	0.0	0.0	0	0
11-232	F	1919	1990			DR	1942	156	1978	1	0.0	0.3	0.0	3	0
11-246	F	1916	1990			DH	1942	78	1977	1	0.0	0.3	0.0	3	0
11-247	F	1923	1990			DH	1944	104	1978	1	0.0	0.3	0.0	3	0
11-262	F	1913	1990			DP	1933	208	1981	1	0.0	1.2	0.0	12	0
11-264	F	1915	1990			DP	1934	130	1976	0	0.0	0.1	0.0	1	0
11-285	F	1915	1990			DH	1946	154	1980	0	0.0	0.0	0.0	0	0
11-290	F	1917	1990			DP	1946	412	1978	2	0.0	0.8	0.0	7	0
11-291	F	1919	1990			DP	1951	164	1974	3	0.0	0.8	0.0	7	0
11-293	M	1942	1990			DR	1965	15	1980	0	0.0	0.0	0.0	0	0
11-294	M	1943		1990		DR	1968	6	1974	0	0.0	0.0	0.0	0	0
11-296	M	1923	1990			DH	1961	156	1978	2	0.0	0.4	0.0	2	0
11-302	F	1901	1990			DP	1924	0	1976	0	0.01000	0.0	0.0	0	0
11-304	F	1912		1982	4270	DH	1928	150	1978	0	0.0	0.0	0.0	0	0
11-329	F	1915		1981	5193	DH	1933	156	1978	0	0.0	0.0	0.0	0	0
11-361	F	1910	1990			DP	1925	23	1982	2	0.00126	2.3	2.6	24	33

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
11-368	F	1910		1983	4109	DP	1927	1	1977	0	0.00230	0.0	0.0	0	0
11-389	F	1908		1980	4109	DP	1924	7	1976	3	0.01150	2.9	17.2	30	220
11-411	F	1905		1982	2509	DH	1922	345	1979	33	0.00713	22.4	96.5	270	1307
11-453	F	1923	1990			DP	1942	13	1976	0	0.0	0.0	0.0	0	0
11-521	F	1910	1990			DP	1927	4	1974	0	0.00330	0.0	0.0	0	0
11-531	F	1894		1978	4409	DP	1918	54	1977	7	0.00134	8.4	12.6	88	161
11-534	F	1918	1990			DP	1941	52	1978	3	0.0	1.6	0.0	16	0
11-561	F	1910		1981	174	DP	1925	2	1976	0	0.00260	0.0	0.0	0	0
11-565	F	1911	1990			DP	1927	76	1974	2	0.00330	1.7	1.4	18	18
11-584	F	1904		1981	4123	DP	1922	15	1977	4	0.0	4.5	0.0	46	0
11-637	M	1902	1990			CR	1934	52	1975	0	0.0	0.0	0.0	0	0
11-803	F	1905		1983	4100	NU	1942	13	1976	0	0.0	0.0	0.0	0	0
11-859	F	1923	1984			DP	1941	208	1978	1	0.0	0.6	0.0	5	0
11-861	F	1922	1990			DP	1941	364	1977	0	0.0	0.0	0.0	0	0
11-863	F	1916	1990			DP	1942	52	1977	0	0.0	0.0	0.0	0	0
11-865	F	1920		1984	4123	DP	1952	260	1978	2	0.0	0.7	0.0	5	0
11-866	F	1907	1990			DP	1942	156	1977	0	0.0	0.0	0.0	0	0
11-867	F	1924	1990			CR	1945	76	1980	3	0.0	1.5	0.0	14	0
11-869	F	1915	1990			DP	1943	3	1981	1	0.0	0.7	0.0	7	0
11-871	F	1925	1990			DP	1940	276	1977	5	0.0	2.7	0.0	26	0
11-875	F	1923	1990			DP	1941	364	1977	1	0.0	0.5	0.0	5	0
11-916	F	1918	1990			DP	1941	108	1975	1	0.0	0.5	0.0	5	0
11-923	F	1924	1990			DP	1942	208	1976	1	0.0	0.3	0.0	3	0
11-924	F	1920	1990			DP	1941	104	1978	1	0.0	0.4	0.0	4	0
11-925	F	1920	1990			DP	1941	78	1975	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
11-938	F	1931	1990			DP	1951	56	1975	0	0.0	0.0	0.0	0	0
11-943	F	1924	1990			DP	1941	78	1981	1	0.0	0.7	0.0	6	0
11-947	F	1925	1990			DP	1947	260	1975	4	0.0	1.5	0.0	13	0
11-948	F	1917	1990			DP	1943	104	1981	0	0.0	0.0	0.0	0	0
11-949	F	1921		1984	1990	DP	1941	78	1981	0	0.0	0.0	0.0	0	0
11-950	F	1925	1990			DH	1944	52	1981	0	0.0	0.1	0.0	1	0
11-957	F	1925	1990			DP	1942	78	1979	1	0.0	0.6	0.0	6	0
11-959	F	1912	1990			DP	1941	208	1982	0	0.0	0.1	0.0	1	0
11-960	F	1924	1990			DP	1942	31	1975	0	0.0	0.0	0.0	0	0
11-962	F	1922	1990			DP	1942	130	1979	0	0.0	0.0	0.0	0	0
11-964	F	1925	1990			DP	1945	52	1980	0	0.0	0.1	0.0	1	0
11-971	F	1923	1990			DP	1944	52	1979	0	0.0	0.0	0.0	0	0
11-973	F	1917		1985	4379	DP	1950	108	1975	1	0.0	0.3	0.0	3	0
11-974	F	1917	1990			DP	1944	40	1977	0	0.0	0.0	0.0	0	0
11-978	F	1920	1990			DP	1942	82	1980	0	0.0	0.2	0.0	2	0
11-982	F	1922	1990			DP	1942	208	1976	0	0.0	0.0	0.0	0	0
11-989	F	1921		1986		DP	1943	35	1977	0	0.0	0.0	0.0	0	0
11-991	F	1924	1990			DP	1942	6	1976	2	0.0	1.1	0.0	10	0
11-993	F	1919	1990			DP	1944	104	1980	2	0.0	1.0	0.0	10	0
11-999	M	1907		1985	1621	DP	1941	160	1980	0	0.0	0.0	0.0	0	0
12-002	F	1918	1990			DP	1941	52	1976	0	0.0	0.0	0.0	0	0
12-008	F	1916		1988		DP	1943	52	1982	2	0.0	1.0	0.0	10	0
12-016	F	1919	1990			DP	1941	111	1977	0	0.0	0.0	0.0	0	0
12-022	F	1924	1990			DP	1942	156	1978	0	0.0	0.0	0.0	0	0
12-025	F	1924	1990			DP	1951	182	1975	1	0.0	0.3	0.0	3	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
12-026	F	1914	1990			DP	1942	166	1976	0	0.0	0.0	0.0	0	0
12-027	F	1916		1990		DP	1942	181	1981	5	0.0	2.9	0.0	28	0
12-033	F	1925	1990			DH	1950	52	1975	3	0.0	1.0	0.0	10	0
12-034	F	1931	1990			DP	1952	1456	1981	0	0.0	0.1	0.0	0	0
12-038	F	1923	1990			DP	1943	26	1980	0	0.0	0.0	0.0	0	0
12-040	F	1921	1990			DP	1942	156	1976	3	0.0	1.5	0.0	14	0
12-041	F	1911	1990			DR	1943	52	1981	0	0.0	0.0	0.0	0	0
12-043	F	1921	1990			DP	1942	182	1978	2	0.0	1.1	0.0	10	0
12-045	F	1925	1990			DP	1942	160	1977	0	0.0	0.0	0.0	0	0
12-059	F	1920	1990			DP	1942	52	1980	0	0.0	0.1	0.0	1	0
12-061	F	1920	1990			DP	1942	182	1975	1	0.0	0.5	0.0	5	0
12-063	F	1916	1990			DH	1943	104	1981	0	0.0	0.2	0.0	2	0
12-064	F	1924	1990			DP	1942	156	1979	0	0.0	0.0	0.0	0	0
12-066	F	1918	1990			DH	1948	43	1981	0	0.0	0.0	0.0	0	0
12-073	F	1920	1990			DP	1942	104	1980	1	0.0	0.4	0.0	4	0
12-074	F	1923	1990			DP	1943	104	1977	1	0.0	0.5	0.0	5	0
12-075	F	1923	1990			DP	1941	208	1977	1	0.0	0.5	0.0	5	0
12-081	F	1913	1990			DP	1941	52	1981	0	0.0	0.1	0.0	1	0
12-083	F	1920	1990			DP	1943	104	1981	0	0.0	0.2	0.0	2	0
12-086	F	1925	1990			DR	1942	156	1977	2	0.0	1.0	0.0	10	0
12-088	F	1921	1990			DP	1942	52	1981	0	0.0	0.0	0.0	0	0
12-089	F	1928	1990			DP	1943	52	1974	0	0.0	0.0	0.0	0	0
12-094	F	1929	1990			DP	1946	4	1975	3	0.0	1.1	0.0	10	0
12-095	F	1927	1990			DP	1947	1	1974	0	0.0	0.1	0.0	1	0
12-096	F	1921	1990			DP	1946	22	1978	2	0.0	1.2	0.0	11	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
12-098	F	1930	1990			DP	1951	52	1974	1	0.0	0.2	0.0	2	0
12-099	F	1929		1982	3452	DH	1951	18	1976	0	0.0	0.0	0.0	0	0
12-102	F	1951	1990			DH	1972	0	1978	1	0.0	0.1	0.0	0	0
12-106	F	1921	1990			DP	1943	4	1980	0	0.0	0.0	0.0	0	0
12-108	F	1915	1990			DP	1942	23	1980	0	0.0	0.0	0.0	0	0
12-110	F	1927	1990			DP	1946	1	1976	0	0.0	0.1	0.0	1	0
12-111	F	1929	1990			DP	1947	19	1974	4	0.0	1.6	0.0	15	0
12-113	F	1915		1984	5193	DP	1942	19	1980	1	0.0	0.4	0.0	3	0
12-115	F	1953	1990			DH	1972	52	1975	0	0.0	0.0	0.0	0	0
12-117	F	1914	1990			DP	1943	3	1979	0	0.0	0.0	0.0	0	0
12-118	F	1932	1990			DP	1954	2	1977	1	0.0	0.4	0.0	4	0
12-119	F	1938	1990			DP	1967	41	1975	1	0.0	0.1	0.0	1	0
12-123	F	1924	1990			DP	1945	17	1976	1	0.0	0.6	0.0	5	0
12-127	F	1917		1980	5193	DP	1941	17	1975	0	0.0	0.0	0.0	0	0
12-128	F	1920		1990		DP	1943	30	1978	2	0.0	1.3	0.0	12	0
12-129	F	1927	1990			DP	1946	4	1976	0	0.0	0.1	0.0	1	0
12-130	F	1924	1990			DP	1947	2	1976	5	0.0	2.0	0.0	19	0
12-133	F	1926		1990		DP	1946	7	1976	1	0.0	0.5	0.0	5	0
12-134	F	1927	1990			DP	1946	1	1975	0	0.0	0.0	0.0	0	0
12-135	F	1913	1990			DP	1943	6	1980	0	0.0	0.1	0.0	1	0
12-136	F	1928	1990			DH	1966	4	1975	1	0.0	0.1	0.0	1	0
12-141	F	1925	1990			DP	1943	3	1978	3	0.0	1.9	0.0	18	0
12-142	F	1922	1990			DP	1942	8	1976	0	0.0	0.0	0.0	0	0
12-143	F	1924	1990			DP	1942	56	1975	1	0.0	0.3	0.0	3	0
12-145	F	1921	1990			DP	1941	35	1976	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
12-146	F	1920	1990			DP	1943	32	1977	0	0.0	0.2	0.0	2	0
12-148	F	1925	1990			DP	1946	4	1975	0	0.0	0.0	0.0	0	0
12-150	F	1919	1990			DP	1943	104	1976	6	0.0	2.9	0.0	28	0
12-155	F	1929		1987	1621	DP	1954	39	1976	0	0.0	0.1	0.0	1	0
12-163	F	1920	1990			DP	1942	78	1974	4	0.0	1.8	0.0	18	0
12-164	F	1920		1986		DP	1943	13	1976	0	0.0	0.2	0.0	1	0
12-165	F	1917	1990			DP	1947	78	1974	3	0.0	1.3	0.0	12	0
12-168	F	1926	1990			DP	1946	13	1975	1	0.0	0.4	0.0	4	0
12-171	F	1921	1990			DP	1940	4	1976	2	0.0	0.9	0.0	9	0
12-173	F	1930	1990			DP	1951	0	1974	2	0.0	0.6	0.0	5	0
12-174	F	1924	1990			DP	1948	18	1976	0	0.0	0.0	0.0	0	0
12-175	F	1927		1988	1830	DP	1946	39	1975	1	0.0	0.2	0.0	2	0
12-178	F	1925	1990			DP	1943	8	1976	0	0.0	0.2	0.0	2	0
12-179	F	1924	1990			DP	1943	9	1976	1	0.0	0.4	0.0	4	0
12-182	F	1922	1990			DP	1942	26	1977	0	0.0	0.2	0.0	2	0
12-185	F	1920	1990			DP	1943	52	1975	0	0.0	0.0	0.0	0	0
12-186	F	1927		1984	4100	DP	1945	4	1974	8	0.0	3.2	0.0	29	0
12-188	F	1936	1990			DH	1965	1	1976	1	0.0	0.1	0.0	1	0
12-190	F	1927	1990			DP	1947	3	1975	0	0.0	0.0	0.0	0	0
12-192	F	1921		1990		DP	1946	52	1976	1	0.0	0.4	0.0	4	0
12-193	F	1925	1990			DP	1942	1	1974	1	0.0	0.6	0.0	6	0
12-194	F	1924		1978	4272	DP	1946	5	1977	1	0.0	0.6	0.0	5	0
12-195	F	1925	1990			DP	1945	2	1976	1	0.0	0.5	0.0	5	0
12-197	F	1906	1990			DP	1922	13	1979	2	0.0	2.3	0.0	24	0
12-198	M	1909		1990		DH	1929	520	1981	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
12-199	M	1913	1990			DB	1934	104	1982	2	0.0	1.4	0.0	10	0
12-203	F	1913		1990		DP	1943	108	1980	0	0.0	0.0	0.0	0	0
12-204	M	1918	1990			CR	1941	104	1977	0	0.0	0.0	0.0	0	0
12-206	F	1914	1990			DP	1942	130	1977	1	0.0	0.3	0.0	3	0
12-212	M	1930	1990			DP	1958	988	1977	2	0.0	0.2	0.0	1	0
12-214	F	1937	1990			DP	1967	26	1977	0	0.0	0.0	0.0	0	0
12-215	F	1936	1990			DP	1958	936	1977	0	0.0	0.0	0.0	0	0
12-216	F	1931		1979	470	DP	1957	104	1977	0	0.0	0.0	0.0	0	0
12-218	M	1937	1978			DP	1955	17	1977	0	0.0	0.0	0.0	0	0
12-221	F	1914	1990			DH	1954	572	1977	1	0.0	0.2	0.0	1	0
12-223	F	1923	1990			DH	1963	728	1977	0	0.0	0.0	0.0	0	0
12-224	F	1927	1990			DP	1963	738	1977	0	0.0	0.0	0.0	0	0
12-225	F	1942	1990			DP	1962	17	1980	0	0.0	0.0	0.0	0	0
12-226	F	1926	1990			DH	1961	520	1977	0	0.0	0.0	0.0	0	0
12-228	F	1935	1990			DP	1959	22	1977	0	0.0	0.0	0.0	0	0
12-229	F	1921	1990			DP	1955	676	1977	1	0.0	0.1	0.0	1	0
12-231	F	1925	1990			DP	1958	104	1981	0	0.0	0.1	0.0	1	0
12-236	F	1928	1990			DP	1960	130	1977	1	0.0	0.2	0.0	1	0
12-237	F	1936	1990			DP	1954	52	1977	0	0.0	0.1	0.0	1	0
12-238	F	1936	1990			DP	1955	117	1981	1	0.0	0.2	0.0	2	0
12-239	F	1922	1990			DP	1956	104	1977	2	0.0	0.5	0.0	5	0
12-252	F	1920	1990			DP	1943	104	1979	1	0.0	0.4	0.0	4	0
12-258	F	1923		1981	150	DP	1943	78	1978	2	0.0	1.0	0.0	9	0
12-259	F	1920	1990			DP	1943	104	1979	1	0.0	0.8	0.0	7	0
12-260	F	1915	1990			DP	1943	52	1979	3	0.0	1.8	0.0	17	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
12-262	F	1921	1990			DP	1942	52	1975	0	0.0	0.2	0.0	1	0
12-270	F	1919	1990			DP	1943	18	1975	0	0.0	0.1	0.0	1	0
12-271	F	1920	1990			DP	1943	77	1980	0	0.0	0.0	0.0	0	0
12-289	F	1921	1990			DP	1943	52	1978	0	0.0	0.0	0.0	0	0
12-294	F	1906		1985	2001	DP	1936	360	1980	2	0.0	1.2	0.0	12	0
12-296	F	1895		1983	4123	DP	1941	104	1982	4	0.0	2.3	0.0	22	0
12-297	F	1923	1990			DP	1943	26	1978	0	0.0	0.2	0.0	2	0
12-299	F	1921	1990			DP	1943	104	1979	0	0.0	0.0	0.0	0	0
12-300	F	1915	1990			DP	1943	104	1980	0	0.0	0.0	0.0	0	0
12-302	F	1914	1990			DP	1943	1	1980	0	0.0	0.1	0.0	1	0
12-304	F	1923	1990			DP	1943	52	1975	0	0.0	0.0	0.0	0	0
12-308	F	1900		1985	1978	DP	1942	52	1980	1	0.0	0.4	0.0	3	0
12-330	M	1928	1990			DH	1944	63	1974	1	0.0	0.4	0.0	3	0
12-331	M	1930		1990		DH	1944	65	1974	0	0.0	0.0	0.0	0	0
12-333	M	1932	1990			CR	1955	728	1974	3	0.0	0.4	0.0	2	0
12-334	F	1908	1990			DP	1924	17	1980	3	0.0	3.9	0.0	41	0
12-342	F	1915	1990			DP	1942	780	1979	7	0.0	3.0	0.0	28	0
12-343	F	1900		1976	4410	DH	1918	208	1974	0	0.00630	0.0	0.0	0	0
12-344	F	1908	1990			DH	1930	104	1974	0	0.0	0.0	0.0	0	0
12-346	F	1908	1990			DP	1926	3	1975	3	0.0	3.0	0.0	31	0
12-350	F	1906		1983	4339	DP	1923	39	1979	1	0.0	0.9	0.0	10	0
12-352	F	1906		1990		NU	1928	416	1975	1	0.0	1.0	0.0	10	0
12-358	F	1913	1990			DP	1940	520	1976	7	0.0	3.3	0.0	31	0
12-359	F	1914	1990			DP	1940	52	1979	1	0.0	0.8	0.0	8	0
12-364	F	1927	1984			DP	1968	364	1975	1	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
12-365	F	1931	1985			DP	1952	520	1975	1	0.0	0.1	0.0	1	0
12-368	F	1923	1985			DP	1958	884	1975	2	0.0	0.1	0.0	1	0
12-370	F	1908	1990			DH	1924	104	1974	0	0.01300	0.0	0.0	0	0
12-375	F	1917	1985			DP	1958	312	1975	0	0.0	0.0	0.0	0	0
12-376	M	1945	1984			DH	1964	520	1977	0	0.0	0.0	0.0	0	0
12-377	F	1920	1985			DP	1961	676	1975	0	0.0	0.0	0.0	0	0
12-383	F	1909	1990			DP	1923	988	1977	0	0.00159	0.0	0.0	0	0
12-384	F	1913	1990			DP	1929	75	1980	15	0.0	14.1	0.0	147	0
12-385	F	1909		1981	4123	DP	1942	182	1979	8	0.0	4.5	0.0	41	0
12-390	F	1905		1988		DP	1929	7	1979	17	0.0	15.7	0.0	164	0
12-392	F	1923	1990			DP	1942	52	1978	0	0.0	0.1	0.0	1	0
12-395	F	1921	1990			DP	1943	104	1982	1	0.0	0.4	0.0	4	0
12-397	M	1916		1990		CR	1947	520	1979	15	0.0	5.9	0.0	38	0
12-405	F	1942	1990			DP	1958	104	1982	1	0.0	0.3	0.0	2	0
12-421	M	1940	1985			CR	1968	260	1978	2	0.0	0.2	0.0	1	0
12-422	F	1907	1990			DP	1937	39	1975	0	0.0	0.0	0.0	0	0
12-425	M	1938	1990			CR	1960	6	1975	0	0.0	0.0	0.0	0	0
12-426	M	1923	1990			IR	1946	18	1975	1	0.0	0.4	0.0	3	0
12-428	F	1907	1990			DP	1922	13	1982	123	0.0	109.4	0.0	1339	0
12-429	F	1922	1990			DP	1945	13	1975	0	0.0	0.0	0.0	0	0
12-430	F	1927	1990			DP	1941	26	1975	1	0.0	0.3	0.0	3	0
12-432	M	1937	1990			CR	1959	572	1977	1	0.0	0.1	0.0	0	0
12-436	F	1896		1979	174	DP	1918	26	1975	1	0.0	0.8	0.0	9	0
12-437	F	1926	1990			DP	1943	104	1975	1	0.0	0.7	0.0	6	0
12-438	M	1942	1990			CR	1964	122	1977	1	0.0	0.2	0.0	1	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
12-442	M	1945	1990			CR	1971	56	1978	1	0.0	0.1	0.0	0	0
12-443	M	1919		1978	4109	CR	1945	13	1976	1	0.0	0.4	0.0	3	0
12-444	M	1950	1990			CR	1972	70	1980	2	0.0	0.2	0.0	1	0
12-447	M	1918	1990			CR	1940	260	1976	6	0.0	3.3	0.0	23	0
12-448	M	1923	1990			CR	1968	624	1979	1	0.0	0.1	0.0	0	0
12-450	M	1911	1990			IR	1946	20	1982	0	0.0	0.0	0.0	0	0
12-451	M	1949	1990			CR	1970	13	1977	0	0.0	0.0	0.0	0	0
12-452	M	1948	1990			CR	1970	52	1977	1	0.0	0.1	0.0	1	0
12-453	M	1914	1990			CR	1939	156	1979	9	0.0	5.3	0.0	38	0
12-455	M	1943	1990			CR	1970	87	1979	3	0.0	0.3	0.0	1	0
12-456	M	1918		1980	4339	CR	1938	364	1976	249	0.0	101.3	0.0	741	0
12-460	M	1923	1990			DP	1945	1092	1975	0	0.0	0.0	0.0	0	0
12-499	F	1908	1990			DP	1925	8	1980	0	0.0	0.3	0.0	4	0
12-502	F	1924	1990			DP	1945	13	1975	0	0.0	0.0	0.0	0	0
12-508	F	1937	1990			DP	1957	884	1975	0	0.0	0.0	0.0	0	0
12-509	F	1918	1990			DP	1941	160	1977	0	0.0	0.0	0.0	0	0
12-510	F	1923	1990			DP	1941	364	1977	1	0.0	0.5	0.0	5	0
12-515	F	1917	1990			DP	1941	52	1978	0	0.0	0.0	0.0	0	0
12-516	F	1918	1990			DP	1941	4	1979	3	0.0	1.8	0.0	18	0
12-518	M	1899		1986		DH	1941	104	1979	0	0.0	0.0	0.0	0	0
12-522	F	1921	1990			DP	1941	30	1977	0	0.0	0.2	0.0	2	0
12-523	F	1923	1990			DP	1941	104	1977	0	0.0	0.1	0.0	1	0
12-528	F	1917	1990			DP	1941	156	1979	1	0.0	0.6	0.0	6	0
12-529	F	1920	1990			DP	1941	104	1977	0	0.0	0.2	0.0	2	0
12-530	M	1920		1984	955	DH	1958	364	1976	3	0.0	0.6	0.0	3	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
12-532	M	1905	1990			DH	1929	2132	1980	1	0.0	0.5	0.0	3	0
12-533	F	1952	1990			DH	1970	260	1975	2	0.0	0.1	0.0	0	0
12-536	F	1929	1990			DP	1951	1664	1983	2	0.0	0.2	0.0	2	0
12-544	F	1921	1990			DP	1941	534	1975	4	0.0	1.7	0.0	16	0
12-545	F	1920	1990			DP	1937	706	1982	12	0.0	7.5	0.0	72	0
12-547	F	1918	1990			DP	1942	1508	1975	3	0.0	0.6	0.0	5	0
12-548	F	1919	1990			DP	1939	832	1975	1	0.0	0.4	0.0	4	0
12-549	F	1917	1990			DP	1943	604	1975	2	0.0	0.7	0.0	7	0
12-552	F	1922	1990			DP	1940	338	1975	7	0.0	3.4	0.0	33	0
12-553	F	1922	1990			DP	1950	260	1976	0	0.0	0.0	0.0	0	0
12-556	F	1922	1990			DP	1942	213	1975	3	0.0	1.4	0.0	13	0
12-557	F	1919	1990			DP	1936	676	1976	2	0.0	1.2	0.0	12	0
12-559	F	1919	1990			DP	1939	104	1976	1	0.0	0.5	0.0	5	0
12-561	F	1917	1990			DP	1942	243	1975	0	0.0	0.0	0.0	0	0
12-563	F	1913	1990			DP	1940	289	1979	1	0.0	0.6	0.0	6	0
12-569	F	1922	1990			DP	1941	208	1978	0	0.0	0.0	0.0	0	0
12-570	F	1912	1990			DP	1941	29	1980	1	0.0	0.3	0.0	3	0
12-572	F	1914		1982	4309	DP	1941	78	1978	0	0.0	0.0	0.0	0	0
12-576	F	1921	1990			DP	1941	208	1978	1	0.0	0.6	0.0	6	0
12-579	F	1921	1990			DP	1941	208	1977	0	0.0	0.0	0.0	0	0
12-582	F	1914	1990			DP	1941	26	1977	0	0.0	0.0	0.0	0	0
12-583	M	1923	1976			OF	1923	39	1976	0	0.0	0.0	0.0	0	0
12-584	F	1907	1990			DH	1926	1820	1979	0	0.0	0.0	0.0	0	0
12-620	F	1928	1990			DP	1966	169	1981	2	0.0	0.4	0.0	3	0
12-623	F	1934	1990			DP	1967	102	1977	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
12-624	F	1939	1990			DP	1965	312	1976	0	0.0	0.0	0.0	0	0
12-628	F	1945	1990			DP	1969	38	1981	0	0.0	0.0	0.0	0	0
12-635	F	1938	1990			DH	1967	156	1978	2	0.0	0.3	0.0	2	0
12-640	F	1946	1985			DH	1964	9	1977	0	0.0	0.0	0.0	0	0
12-643	F	1933	1985			DP	1957	126	1977	0	0.0	0.0	0.0	0	0
12-644	F	1934	1985			DP	1972	52	1977	1	0.0	0.1	0.0	0	0
12-645	F	1944	1984			DP	1963	156	1977	1	0.0	0.1	0.0	1	0
12-646	F	1946	1987			DP	1965	260	1977	0	0.0	0.0	0.0	0	0
12-650	F	1931	1985			DP	1949	1456	1977	2	0.0	0.2	0.0	1	0
12-652	F	1931	1985			DP	1953	56	1977	2	0.0	0.5	0.0	5	0
12-654	M	1942	1985			DR	1962	43	1977	3	0.0	0.6	0.0	3	0
12-656	M	1944	1985			DP	1962	104	1976	2	0.0	0.3	0.0	2	0
12-657	M	1924	1985			CR	1950	520	1977	6	0.0	1.9	0.0	11	0
12-660	M	1926	1985			DP	1955	39	1977	2	0.0	0.5	0.0	3	0
12-661	F	1946	1985			DP	1965	13	1977	0	0.0	0.0	0.0	0	0
12-665	F	1925	1985			DR	1971	260	1977	1	0.0	0.1	0.0	0	0
12-669	M	1957	1985			DR	1974	22	1977	0	0.0	0.0	0.0	0	0
12-670	M	1929	1985			DP	1951	52	1977	1	0.0	0.4	0.0	3	0
12-672	F	1920	1990			DP	1942	89	1979	2	0.0	1.2	0.0	11	0
12-675	F	1921	1990			DP	1952	30	1978	0	0.0	0.1	0.0	1	0
12-688	F	1917	1990			DP	1944	17	1977	1	0.0	0.4	0.0	4	0
12-693	F	1922	1990			DP	1942	0	1979	0	0.0	0.1	0.0	1	0
12-694	F	1931	1990			DP	1949	13	1976	0	0.0	0.0	0.0	0	0
12-695	F	1926	1990			DP	1951	133	1979	1	0.0	0.4	0.0	4	0
12-700	F	1929	1990			DP	1952	52	1978	2	0.0	0.7	0.0	7	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
12-702	F	1918	1990			DP	1942	160	1977	1	0.0	0.5	0.0	5	0
12-709	F	1925	1990			DP	1952	121	1976	0	0.0	0.0	0.0	0	0
12-710	F	1911		1985	4123	DP	1952	104	1981	0	0.0	0.0	0.0	0	0
12-712	F	1906	1990			DP	1952	30	1981	0	0.0	0.1	0.0	1	0
12-729	F	1904		1986		DP	1944	6	1978	0	0.0	0.0	0.0	0	0
12-738	F	1922	1990			DP	1949	32	1979	0	0.0	0.0	0.0	0	0
12-739	F	1914	1990			DP	1954	17	1978	3	0.0	0.8	0.0	8	0
12-746	F	1913	1990			DP	1942	124	1976	0	0.0	0.0	0.0	0	0
12-748	F	1911	1990			DP	1944	13	1978	0	0.0	0.0	0.0	0	0
12-757	F	1922	1990			DP	1941	104	1976	1	0.0	0.6	0.0	6	0
12-764	F	1924	1990			DP	1952	104	1977	1	0.0	0.5	0.0	4	0
12-765	F	1921	1990			DP	1949	1352	1976	0	0.0	0.0	0.0	0	0
12-771	F	1930	1990			DP	1949	936	1976	0	0.0	0.0	0.0	0	0
12-773	F	1922	1990			DP	1944	17	1980	0	0.0	0.0	0.0	0	0
12-775	F	1916	1990			DP	1945	104	1981	0	0.0	0.0	0.0	0	0
12-777	F	1924		1989		DP	1942	2	1980	1	0.0	0.6	0.0	5	0
12-779	F	1929	1990			DP	1952	52	1976	0	0.0	0.1	0.0	1	0
12-782	F	1923	1990			DP	1945	8	1982	1	0.0	0.5	0.0	5	0
12-784	F	1930	1990			DP	1953	17	1977	1	0.0	0.2	0.0	2	0
12-788	F	1918	1990			DP	1951	160	1979	0	0.0	0.0	0.0	0	0
12-791	F	1920	1990			DP	1943	17	1979	2	0.0	0.9	0.0	8	0
12-795	F	1918	1990			DP	1949	17	1977	1	0.0	0.2	0.0	2	0
12-797	F	1922	1990			DP	1951	184	1979	2	0.0	0.6	0.0	5	0
12-802	F	1906	1990			DP	1943	14	1978	2	0.0	1.2	0.0	12	0
12-810	F	1910	1990			DP	1943	104	1977	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
12-815	F	1910	1990			DP	1943	8	1980	0	0.0	0.1	0.0	1	0
12-826	F	1906	1990			DP	1943	8	1977	2	0.0	1.1	0.0	10	0
12-829	F	1922	1990			DP	1949	18	1977	0	0.0	0.0	0.0	0	0
12-830	F	1917	1990			DP	1943	2	1981	1	0.0	0.7	0.0	7	0
12-831	F	1918		1979	174	DP	1940	207	1978	1	0.0	0.6	0.0	5	0
12-841	F	1922	1990			DP	1952	1	1977	0	0.0	0.0	0.0	0	0
12-843	F	1916	1990			DP	1952	30	1979	0	0.0	0.0	0.0	0	0
12-849	F	1916	1990			DP	1941	208	1977	3	0.0	1.5	0.0	14	0
12-850	F	1917	1990			DP	1951	26	1977	0	0.0	0.0	0.0	0	0
12-857	F	1926	1990			DP	1951	208	1977	0	0.0	0.0	0.0	0	0
12-858	F	1917		1990		DP	1951	22	1978	0	0.0	0.0	0.0	0	0
12-863	F	1929	1990			DP	1953	11	1978	2	0.0	0.5	0.0	5	0
12-864	F	1919	1990			DP	1952	34	1978	0	0.0	0.0	0.0	0	0
12-872	F	1924	1990			DP	1943	8	1980	0	0.0	0.0	0.0	0	0
12-875	F	1921	1990			DP	1952	15	1979	0	0.0	0.0	0.0	0	0
12-878	F	1920		1986		DP	1949	237	1976	1	0.0	0.4	0.0	3	0
12-880	F	1917	1990			DP	1950	52	1977	1	0.0	0.5	0.0	5	0
12-884	F	1911	1990			DP	1944	27	1980	0	0.0	0.2	0.0	2	0
12-885	F	1918	1990			DP	1945	4	1978	2	0.0	1.1	0.0	11	0
12-887	F	1925	1990			DP	1942	78	1977	0	0.0	0.0	0.0	0	0
12-889	F	1924		1986		DP	1947	260	1976	1	0.0	0.4	0.0	3	0
12-891	F	1920	1990			DP	1952	8	1979	3	0.0	1.0	0.0	9	0
12-894	F	1914	1990			DP	1951	73	1980	0	0.0	0.0	0.0	0	0
12-901	F	1915	1990			DP	1951	13	1977	0	0.0	0.0	0.0	0	0
12-904	F	1922		1985	4449	DP	1952	17	1980	2	0.0	0.7	0.0	6	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
12-905	F	1914		1986	4123	DP	1949	312	1981	1	0.0	0.3	0.0	2	0
12-908	F	1923	1990			DP	1952	87	1976	0	0.0	0.0	0.0	0	0
12-916	F	1921	1990			DP	1942	676	1977	2	0.0	0.8	0.0	8	0
12-918	F	1918	1990			DP	1940	208	1977	1	0.0	0.5	0.0	5	0
12-924	F	1904	1990			DP	1950	17	1982	0	0.0	0.0	0.0	0	0
12-927	F	1919	1990			DP	1942	13	1977	2	0.0	0.9	0.0	9	0
12-929	F	1911	1990			DW	1942	4	1982	0	0.0	0.0	0.0	0	0
12-933	F	1923	1990			DP	1944	52	1979	2	0.0	0.9	0.0	8	0
12-941	F	1925	1990			DP	1944	2	1980	0	0.0	0.0	0.0	0	0
12-942	F	1898		1981	485	DP	1944	+0	1977	2	0.0	0.9	0.0	8	0
12-943	F	1917	1990			DP	1952	52	1976	1	0.0	0.4	0.0	4	0
12-961	F	1913	1990			DP	1940	200	1980	6	0.0	4.0	0.0	39	0
12-963	F	1920	1990			DP	1942	104	1979	1	0.0	0.8	0.0	8	0
12-965	F	1924		1986		DP	1945	52	1977	2	0.0	0.9	0.0	8	0
12-967	F	1913	1990			DP	1953	12	1979	0	0.0	0.0	0.0	0	0
12-977	F	1920	1990			DP	1943	7	1978	1	0.0	0.4	0.0	4	0
12-978	F	1919		1982	7123	OF	1919	39	1976	0	0.0	0.0	0.0	0	0
12-981	F	1907	1990			DP	1923	19	1977	0	0.00907	0.0	0.0	0	0
12-983	F	1921	1990			DP	1940	1040	1976	6	0.0	2.2	0.0	20	0
12-985	M	1934	1976			OF	1934	39	1976	1	0.0	0.7	0.0	5	0
12-986	M	1932		1976	4272	OF	1932	39	1976	2	0.0	1.5	0.0	10	0
12-987	F	1920	1990			DP	1940	65	1982	0	0.0	0.1	0.0	1	0
13-002	F	1901	1990			DP	1923	468	1977	0	0.0	0.0	0.0	0	0
13-007	M	1911	1990			CR	1951	676	1981	1	0.0	0.4	0.0	3	0
13-010	F	1923	1990			DP	1942	26	1977	2	0.0	0.9	0.0	9	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
13-011	F	1924	1990			DP	1942	39	1979	0	0.0	0.0	0.0	0	0
13-015	F	1910		1979		DP	1954	884	1976	1	0.0	0.1	0.0	1	0
13-019	F	1915	1990			DP	1942	104	1977	0	0.0	0.0	0.0	0	0
13-021	F	1914		1986		DP	1941	104	1979	0	0.0	0.0	0.0	0	0
13-022	F	1920	1990			DP	1942	69	1979	0	0.0	0.2	0.0	2	0
13-025	F	1914		1989		DP	1940	32	1977	0	0.0	0.0	0.0	0	0
13-026	F	1921	1990			DP	1941	26	1977	0	0.0	0.0	0.0	0	0
13-027	F	1922	1990			DP	1942	156	1977	1	0.0	0.7	0.0	7	0
13-030	F	1920	1990			DP	1940	416	1982	0	0.0	0.0	0.0	0	0
13-031	F	1923	1990			DP	1941	26	1982	1	0.0	1.0	0.0	9	0
13-037	F	1913	1990			DP	1941	52	1982	0	0.0	0.0	0.0	0	0
13-044	F	1954	1990			RA	1977	+0	1977	0	0.0	0.0	0.0	0	0
13-050	M	1932	1990			RA	1977	+0	1977	1	0.0	0.0	0.0	0	0
13-051	F	1878		1962	4123	RW	1925	+0	1949	700	0.0	163.5	0.0	1789	0
13-055	F	1908	1990			DH	1923	11	1978	0	0.00800	0.0	0.0	0	0
13-056	M	1958	1985			CR	1976	52	1977	3	0.0	0.1	0.0	0	0
13-057	F	1922	1985			DR	1976	104	1978	0	0.0	0.0	0.0	0	0
13-058	M	1956	1980			DP	1976	62	1977	0	0.0	0.0	0.0	0	0
13-059	M	1910	1990			DR	1933	2184	1978	1	0.0	0.2	0.0	1	0
13-063	F	1908		1989		DH	1933	1976	1978	0	0.0	0.0	0.0	0	0
13-064	F	1912	1990			DH	1959	102	1978	0	0.0	0.0	0.0	0	0
13-067	F	1917	1990			DP	1942	39	1978	0	0.0	0.0	0.0	0	0
13-071	F	1923	1985			DP	1942	78	1978	1	0.0	0.6	0.0	5	0
13-073	F	1924		1988		DP	1942	1352	1978	0	0.0	0.0	0.0	0	0
13-078	F	1908	1990			DH	1942	1300	1978	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
13-080	F	1921	1990			DH	1939	312	1978	0	0.0	0.0	0.0	0	0
13-082	F	1920	1990			DP	1942	52	1978	2	0.0	1.1	0.0	11	0
13-085	F	1918	1990			DH	1942	936	1982	1	0.0	0.4	0.0	3	0
13-087	F	1925	1990			DP	1942	8	1978	0	0.0	0.0	0.0	0	0
13-088	F	1922	1990			DP	1942	8	1978	0	0.0	0.0	0.0	0	0
13-089	F	1923	1990			DP	1942	104	1978	0	0.0	0.0	0.0	0	0
13-092	F	1917	1990			DH	1952	1196	1979	0	0.0	0.0	0.0	0	0
13-102	F	1912	1990			DP	1928	936	1979	4	0.0	2.8	0.0	28	0
13-107	F	1904	1990			DP	1936	1820	1978	5	0.0	1.4	0.0	12	0
13-108	F	1907	1990			DH	1926	2444	1978	2	0.0	0.6	0.0	5	0
13-109	F	1910	1990			DP	1943	1	1979	7	0.0	4.0	0.0	39	0
13-113	F	1906		1988		DP	1926	2080	1978	2	0.0	0.8	0.0	7	0
13-115	F	1923	1990			DH	1959	52	1980	0	0.0	0.1	0.0	1	0
13-127	F	1914	1990			DH	1942	260	1978	1	0.0	0.5	0.0	5	0
13-132	F	1905	1990			DH	1932	1976	1978	3	0.0	0.9	0.0	9	0
13-136	F	1908		1982	1410	DH	1942	130	1978	0	0.0	0.0	0.0	0	0
13-138	F	1907		1986		DH	1942	520	1979	0	0.0	0.0	0.0	0	0
13-139	F	1922	1990			DP	1944	130	1978	4	0.0	2.0	0.0	19	0
13-145	F	1920	1990			DP	1937	468	1978	2	0.0	1.2	0.0	11	0
13-146	F	1921	1990			DP	1942	52	1978	1	0.0	0.6	0.0	5	0
13-147	F	1900		1988		DH	1922	1664	1979	1	0.0	1.1	0.0	12	0
13-148	F	1926	1990			DP	1946	52	1982	1	0.0	0.5	0.0	4	0
13-151	F	1904	1990			DH	1927	936	1978	1	0.0	0.7	0.0	7	0
13-152	M	1901	1990			DH	1941	208	1978	3	0.0	1.7	0.0	11	0
13-153	M	1908	1990			DH	1939	1352	1978	1	0.0	0.3	0.0	2	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
13-154	F	1905	1990			DH	1941	1248	1978	0	0.0	0.0	0.0	0	0
13-158	F	1920	1990			DP	1944	52	1979	1	0.0	0.5	0.0	5	0
13-160	F	1948	1990			DP	1966	52	1982	1	0.0	0.1	0.0	1	0
13-161	F	1948	1985			DP	1969	8	1978	2	0.0	0.2	0.0	1	0
13-165	F	1917	1990			DP	1943	104	1979	1	0.0	0.7	0.0	7	0
13-167	F	1928	1990			DH	1958	260	1979	0	0.0	0.0	0.0	0	0
13-169	M	1928		1990		DP	1953	988	1981	1	0.0	0.1	0.0	1	0
13-170	F	1923		1988		DP	1943	104	1979	0	0.0	0.0	0.0	0	0
13-175	F	1923	1990			DP	1944	52	1982	0	0.0	0.0	0.0	0	0
13-178	F	1935	1990			DP	1967	832	1983	0	0.0	0.0	0.0	0	0
13-179	F	1937	1990			DP	1968	676	1983	0	0.0	0.0	0.0	0	0
13-180	M	1910		1981	1621	DB	1978	98	1983	3	0.0	0.2	0.0	0	0
13-181	M	1925		1980	2051	DH	1977	109	1983	3	0.0	0.1	0.0	0	0
14-001	F	1933	1990			DP	1951	83	1982	0	0.0	0.0	0.0	0	0
14-002	F	1931	1990			DP	1951	26	1982	0	0.0	0.0	0.0	0	0
14-007	F	1930	1990			DW	1949	86	1983	0	0.0	0.1	0.0	1	0
14-009	F	1933	1990			DP	1952	16	1983	0	0.0	0.0	0.0	0	0
14-010	F	1929	1990			DP	1948	645	1983	1	0.0	0.2	0.0	2	0
14-013	F	1928	1990			DP	1949	1040	1983	3	0.0	0.8	0.0	7	0
14-014	M	1938	1990			CS	1959	530	1983	0	0.0	0.0	0.0	0	0
14-022	F	1894		1980	4369	DP	1917	2184	1977	1	0.0	1.6	0.0	17	0
14-023	F	1932	1990			DP	1951	96	1983	0	0.0	0.0	0.0	0	0
14-026	F	1921	1990			DP	1950	988	1983	0	0.0	0.0	0.0	0	0
14-027	M	1917	1990			DB	1956	706	1982	1	0.0	0.2	0.0	1	0
14-029	F	1933	1990			DP	1953	112	1983	3	0.0	1.3	0.0	12	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
14-032	F	1936	1990			DB	1955	31	1983	0	0.0	0.0	0.0	0	0
14-040	F	1930	1990			DP	1951	4	1983	1	0.0	0.3	0.0	3	0
14-045	M	1937	1990			DB	1956	9	1983	1	0.0	0.5	0.0	3	0
14-049	F	1932	1990			DB	1955	29	1983	1	0.0	0.3	0.0	3	0
14-060	M	1932	1990			CS	1956	273	1983	1	0.0	0.2	0.0	1	0
14-062	F	1941	1990			DH	1959	173	1983	0	0.0	0.0	0.0	0	0
14-072	F	1935	1990			DB	1955	755	1983	0	0.0	0.0	0.0	0	0
14-077	M	1932	1990			CS	1952	9	1983	0	0.0	0.0	0.0	0	0
14-083	M	1938	1990			DW	1956	2	1983	1	0.0	0.3	0.0	2	0
14-088	F	1936	1990			DW	1955	35	1983	1	0.0	0.2	0.0	2	0
14-091	M	1935	1990			DB	1955	176	1983	0	0.0	0.0	0.0	0	0
14-092	M	1937	1990			DB	1956	191	1983	0	0.0	0.0	0.0	0	0
14-093	M	1932	1990			DB	1955	18	1983	1	0.0	0.4	0.0	2	0
14-096	F	1935	1990			CS	1965	194	1983	0	0.0	0.0	0.0	0	0
14-099	M	1917	1990			DH	1951	56	1983	1	0.0	0.2	0.0	2	0
14-100	M	1937	1990			CS	1955	233	1983	0	0.0	0.1	0.0	1	0
14-102	F	1932	1990			DH	1951	148	1983	0	0.0	0.1	0.0	1	0
14-111	F	1935	1990			DB	1955	649	1983	0	0.0	0.0	0.0	0	0
14-115	F	1937	1990			DP	1955	68	1983	0	0.0	0.0	0.0	0	0
14-124	F	1934	1990			DB	1954	17	1983	0	0.0	0.0	0.0	0	0
14-125	F	1931	1990			DP	1951	3	1983	0	0.0	0.0	0.0	0	0
14-127	M	1934	1990			DB	1953	13	1983	0	0.0	0.0	0.0	0	0
14-131	M	1945	1990			CS	1965	202	1983	0	0.0	0.0	0.0	0	0
14-132	M	1934	1990			CS	1959	20	1983	0	0.0	0.0	0.0	0	0
14-134	M	1918	1990			DB	1950	16	1983	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
14-138	F	1936	1990			DB	1963	32	1983	0	0.0	0.0	0.0	0	0
14-144	F	1915	1990			DB	1955	24	1983	0	0.0	0.0	0.0	0	0
14-145	M	1932	1990			CS	1966	113	1983	2	0.0	0.5	0.0	3	0
14-147	M	1927	1990			DW	1955	714	1983	0	0.0	0.1	0.0	0	0
14-149	M	1920	1990			DM	1968	60	1983	0	0.0	0.0	0.0	0	0
14-155	M	1910		1981	4299	CR	1942	1404	1979	21	0.0	6.1	0.0	34	0
14-156	M	1945	1990			DP	1964	98	1983	0	0.0	0.0	0.0	0	0
14-162	M	1914	1990			CR	1934	1820	1976	9	0.0	2.4	0.0	15	0
14-170	M	1925		1990	2381	DW	1955	728	1982	1	0.0	0.3	0.0	2	0
14-180	F	1940	1990			DH	1961	120	1974	1	0.0	0.1	0.0	1	0
14-185	F	1930	1990			DH	1950	217	1983	3	0.0	1.2	0.0	11	0
14-201	M	1923	1990			DB	1950	235	1983	4	0.0	2.0	0.0	13	0
14-209	M	1933	1990			DP	1956	683	1983	0	0.0	0.0	0.0	0	0
14-218	M	1929	1990			DH	1952	4	1983	0	0.0	0.0	0.0	0	0
14-249	F	1938	1990			DH	1959	532	1983	0	0.0	0.0	0.0	0	0
14-252	M	1929	1990			DB	1953	11	1983	0	0.0	0.0	0.0	0	0
14-261	M	1932	1990			DB	1956	642	1982	3	0.0	0.7	0.0	4	0
14-262	M	1912		1985	486	DB	1954	99	1983	0	0.0	0.0	0.0	0	0
14-301	F	1932	1990			DP	1951	927	1983	0	0.0	0.0	0.0	0	0
14-327	M	1930	1990			DB	1955	30	1982	0	0.0	0.0	0.0	0	0
14-332	M	1921	1990			CS	1950	988	1979	21	0.0	5.2	0.0	31	0
14-338	F	1928	1990			DP	1948	346	1976	5	0.0	1.6	0.0	15	0
14-340	F	1944	1990			DP	1966	144	1983	1	0.0	0.1	0.0	1	0
14-341	M	1930	1990			DB	1954	2	1982	1	0.0	0.4	0.0	2	0
14-352	M	1926		1979	4109	DB	1948	1092	1973	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
14-377	F	1930	1990			DP	1951	887	1973	3	0.0	0.4	0.0	4	0
14-395	M	1921	1990			DH	1955	410	1983	1	0.0	0.2	0.0	1	0
14-408	F	1928	1990			DP	1948	13	1983	1	0.0	0.7	0.0	6	0
14-410	M	1927	1990			CS	1951	936	1973	8	0.0	1.3	0.0	8	0
14-422	M	1928	1990			DH	1952	676	1983	0	0.0	0.0	0.0	0	0
14-440	F	1929	1990			DP	1952	84	1983	0	0.0	0.0	0.0	0	0
14-442	M	1933	1990			DB	1955	92	1983	1	0.0	0.2	0.0	1	0
14-445	M	1905		1988		CR	1928	2028	1982	215	0.0	67.8	0.0	530	0
14-450	M	1926	1990			DM	1955	754	1983	4	0.0	1.1	0.0	6	0
14-467	F	1931	1990			DM	1955	728	1983	0	0.0	0.1	0.0	0	0
14-469	M	1914		1973	4109	CR	1945	138	1968	1	0.0	0.3	0.0	2	0
14-512	F	1930	1990			DH	1948	5	1983	0	0.0	0.0	0.0	0	0
14-513	F	1936	1990			DH	1955	20	1983	0	0.0	0.0	0.0	0	0
14-514	M	1932		1989		CS	1955	742	1974	2	0.0	0.2	0.0	1	0
14-522	F	1924		1986	354X	DP	1952	884	1982	1	0.0	0.2	0.0	1	0
14-558	F	1936	1990			DP	1955	232	1983	1	0.0	0.4	0.0	4	0
14-569	F	1922	1990			DP	1952	396	1983	1	0.0	0.2	0.0	2	0
14-575	F	1944	1990			DB	1965	202	1982	0	0.0	0.1	0.0	0	0
14-582	M	1946	1990			DB	1965	156	1983	3	0.0	0.6	0.0	3	0
14-590	F	1941	1990			DB	1965	200	1983	0	0.0	0.0	0.0	0	0
14-620	M	1919		1985	4123	CS	1950	884	1983	2	0.0	0.5	0.0	3	0
14-623	F	1924	1990			DP	1951	936	1974	0	0.0	0.0	0.0	0	0
14-627	M	1929	1990			DB	1953	101	1983	0	0.0	0.0	0.0	0	0
14-636	F	1931	1990			DP	1951	39	1983	0	0.0	0.2	0.0	2	0
14-650	F	1924	1990			DP	1950	988	1983	5	0.0	1.4	0.0	12	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
14-652	F	1928	1990			DP	1951	936	1983	0	0.0	0.0	0.0	0	0
14-661	F	1932	1990			DW	1950	320	1983	1	0.0	0.6	0.0	6	0
14-675	M	1922	1990			CS	1953	156	1976	1	0.0	0.4	0.0	2	0
14-687	M	1919	1990			CS	1953	832	1972	0	0.0	0.0	0.0	0	0
14-726	M	1933	1990			DB	1966	72	1983	0	0.0	0.0	0.0	0	0
14-728	F	1928	1990			DP	1948	278	1983	1	0.0	0.3	0.0	3	0
14-729	M	1930	1990			DB	1953	17	1983	1	0.0	0.3	0.0	2	0
14-735	M	1891		1964	4122	CR	1915	1248	1959	82	0.00700	33.2	6.4	224	60
14-736	F	1935	1990			DP	1955	415	1983	1	0.0	0.5	0.0	4	0
14-769	M	1922	1990			DB	1955	681	1983	0	0.0	0.0	0.0	0	0
14-776	F	1931	1990			DP	1952	711	1983	0	0.0	0.0	0.0	0	0
14-804	F	1925	1990			DP	1952	45	1983	1	0.0	0.2	0.0	2	0
14-811	M	1917		1987	1709	CR	1942	1144	1968	30	0.0	5.2	0.0	32	0
14-812	F	1932	1990			DP	1955	728	1983	0	0.0	0.0	0.0	0	0
14-833	M	1929		1983	4109	CS	1965	160	1982	0	0.0	0.0	0.0	0	0
14-836	F	1929	1990			DP	1951	59	1983	1	0.0	0.3	0.0	3	0
14-854	F	1928	1990			DP	1951	25	1983	0	0.0	0.0	0.0	0	0
14-883	F	1927	1990			CR	1951	350	1978	0	0.0	0.0	0.0	0	0
14-887	M	1915	1990			DH	1955	717	1983	0	0.0	0.1	0.0	0	0
14-891	M	1925	1990			CS	1948	1092	1973	10	0.0	1.6	0.0	10	0
14-908	F	1931	1990			DP	1952	24	1982	0	0.0	0.0	0.0	0	0
14-955	M	1926	1990			DH	1948	1092	1976	6	0.0	1.2	0.0	7	0
14-961	M	1929		1988	191	DB	1956	402	1982	0	0.0	0.1	0.0	0	0
15-046	M	1925	1990			DB	1959	540	1974	0	0.0	0.0	0.0	0	0
15-177	F	1938	1990			DB	1960	489	1983	0	0.0	0.0	0.0	0	0

TABLE A.1 (Cont.)

Case	Sex	Born	Live	Died	Cause	Exp. Type	Year First Exp.	Exp. Dur. (wk)	Year of Meas.	^{226}Ra (nCi)	^{228}Ra to ^{226}Ra Ratio	Intake ^{226}Ra (μCi)	Intake ^{228}Ra (μCi)	Dose ^{226}Ra (cGy)	Dose ^{228}Ra (cGy)
15-392	M	1933	1990			DB	1959	520	1983	3	0.0	0.7	0.0	4	0
15-641	F	1920	1990			DB	1959	537	1983	0	0.0	0.0	0.0	0	0
15-679	F	1939	1990			DB	1960	489	1983	0	0.0	0.1	0.0	1	0

TABLE A.2 Codes Describing Types of Exposure to Radium

Exposure Code	Number of Cases	Code Description
CM	1	Chemist, mesothorium (^{228}Ra). Includes physicists and laboratory technicians, but not persons working with ^{228}Th or ^{232}Th.
CR	238	Chemist, radium (^{226}Ra). Includes physicists and laboratory technicians.
CS	17	Chemist, radium plus other nuclides.
DB	38	A blue-collar worker in a plant where others handled radium (includes watchmen, maintenance personnel, and other production workers).
DH	213	Dial handler. Routinely handled radium or products incorporating exposed radium.
DM	3	A dial plant worker exposed to radium plus other nuclides.
DP	1,575	Dial painter.
DR	72	Dial remote, not otherwise specified as white or blue collar. (Worked in a plant where others handled radium.)
DW	7	Dial remote, white collar. A supervisor, secretary, accountant, office boy, etc., in a plant where others handled radium.
IM	1	Industrial (nonluminizing facility) exposure to radium plus other nuclides.
IR	8	Industrial (nonluminizing facility) exposure to radium.
KH	16	Kabakjian house (a home contaminated with radium).
MD	1	Physician.
ME	1	Miscellaneous exposure to radium (^{228}Ra or ^{226}Ra).

TABLE A.2 (Cont.)

Exposure Code	Number of Cases	Code Description
MI	1	Mesothorium injection.
NU	13	Nurse (radium).
OF	20	Offspring born to female subjects after they had been exposed to radium (^{228}Ra or ^{226}Ra).
RA	13	Radium accident.
RD	33	Radithor.
RI	101	Radium injection.
RW	31	Radium water or radium-barium capsule.

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