ANL-78-65
Part II

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RADIOLOGICAL AND ENVIRONMENTAL RESEARCH DIVISION ANNUAL REPORT

Center for Human Radiobiology

July 1977—June 1978

ARGONNE NATIONAL LABORATORY, ARGONNE, ILLINOIS

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Center for Human Radiobiology

July 1977 through June 1978

R. E. Rowland, Division Director
A. F. Stehney, Section Head

Preceding Report: ANL-77-65, Part II, July 1976–June 1977

FOREWORD

The first paper in this Annual Report describes five recently discovered cases of malignancy attributed to radium in patients with measured body burdens of radium. Included is a case of bone sarcoma diagnosed in 1977, the second since the Center was established in 1969. During the same period, seven cases of carcinoma of the mastoid air cells were diagnosed. The great importance of malignant tumors of head sinuses as a manifestation of radium toxicity is indicated by publication of a comprehensive review of these cases (cf. paper 2) and study of the tissues at risk (paper 9). At the same time, new data are being obtained on cells possibly at risk for induction of osteosarcoma (paper 5). In an analysis of dose-response relationships (paper 3), only the contribution from ^{226}Ra and its daughters was included in the dose for head carcinomas, whereas dose contributions from both ^{226}Ra and ^{228}Ra were included for bone sarcomas. For other reports from the Center on the possible health effects of radium, the reader is referred to papers 4 and 8 and to the list of publications at the back of this Report.

Among new results in the study of persons injected with plutonium in the 1940's, it is of interest to note that measurable amounts of plutonium were found in the hair of a woman who died 1.4 years after injection and that the lengthwise distribution may reflect the relative concentration in the blood as a function of time (paper 20). Paper 27 is a report on the radioactivity content of plants and animals sampled in the vicinity of tailings piles from uranium mills; this is part of a cooperative study with Argonne's Division of Environmental Impact Studies.

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Center for Human Radiobiology

RECENT CASES OF RADIUM-INDUCED MALIGNANCY

A. M. Brues

Five cases of malignant disease attributed to radium in patients with measured body burdens have been discovered since 1974, including three bone sarcomas and two mastoid carcinomas. Pertinent findings in these cases are summarized here.

A previous report[1] listed six cases under surveillance by the Center for Human Radiobiology in which a diagnosis of mastoid carcinoma had been made in the period between 1969 and 1974. This report describes five cases of presumed radium-related malignancy discovered since 1974. Three of the five were included in tabulations in our 1976 and 1977 reports[2,3] and two others are tabulated in this report (Appendix B) for the first time; one of the latter had been lost to follow-up since 1935. The five cases include three bone sarcomas and two mastoid carcinomas. Brief summaries of these cases are given here.

Case 01-051

This woman was born in 1904 and was employed as a dial painter in a Connecticut plant for three years between 1923 and 1926. She regularly tipped the brush between her teeth and lips. She married in 1926 and had a miscarriage at two months in 1927. In the following year she gave birth to a daughter, by caesarean section, in the course of which she is said to have sustained an injury to her coccyx, a source of chronic distress, which was removed surgically in 1941. She had been found to have a significant radium burden by electroscope measurement in 1928, and x-ray evidence of skeletal radium effects was seen in 1947. In 1963 she was found to have severe hypertension, myocardial disease, and diabetes mellitus in the course of hospitalization for duodenal ulcer. Generalized vascular disease progressed and she became bedridden. In February 1972 circulatory insufficiency in the right leg necessitated amputation at knee level, followed by further amputation at a higher

level. At that time, an osteolytic lesion detected by x ray in the lower third of the left tibia was biopsied and identified as "chronic inflammation in dense fibrous tissue." Six months later she entered another hospital because of ischemic symptoms in the left leg and insisted on a mid-thigh amputation for relief. A biopsy of the tibial region taken at that time was interpreted as plasma cell myeloma. These sections were subsequently reviewed by Dr. Margaret Littman and by Dr. Shields Warren, who separately made the diagnosis of osteosarcoma with areas of plasma cell infiltration. This case was added to the list of bone sarcomas in the 1976 report.[2] She died in May 1977, and was autopsied. The estimated total skeletal radiation dose ($^{226}Ra + ^{228}Ra$) was 4260 rads as of 1972. The death certificate indicated arteriosclerotic heart disease with coronary artery disease.

Case 01-087

This patient was born in 1905 and worked as a dial painter in the same establishment for eight years between 1920 and 1928. Although there are few details of her early medical history, she evidently was heavily exposed to radium and mesothorium. Prior to 1935, osteomyelitis of the mandible was seen, and there is evidence that she had at least four pathologic fractures, including both femora and an ankle, in the course of the next ten or fifteen years. In that period she also developed bilateral cataracts and iridocyclitis as well as a chronic discharge from the ears. In 1957, a polyp in the left external auditory canal was removed and identified as a squamous cell carcinoma extending from the middle ear: this area was treated by deep radiotherapy, 5000 R in ten doses. She is still living without recurrence in 1978, totally blind and partially deaf. The estimated total skeletal dose accumulated to 1957 is 18,110 rads. This case was added to the list of head carcinomas in 1977[3] and is considered to have its origin in the mastoid.[4]

Case 02-676

This is a female patient on whom almost no history has been obtained directly. From collateral data, it is known that she was employed in an

Illinois plant as a dial painter for a period that included late 1924. In reply to a questionnaire, she stated that she painted dials for one month in 1925 and never pointed the brush in her mouth. A breath sample was collected in 1961 and analyzed at Argonne, from which an estimate of 1.2 μCi total burden was derived. A whole-body gamma count done in 1963 under suboptimal conditions, led to an estimate of 1.7 μCi. In 1976, we learned that she had a neoplasm at the tip of the left mastoid bone, which was discovered following a biopsy of a neoplastic cervical lymph node. The histologic diagnosis, confirmed at the Center, was epidermoid carcinoma, grade II, moderately well differentiated. She died in August 1977 and there was no autopsy. The estimated total skeletal dose was 6430 rads (1976). The death certificate indicated arteriosclerosis with cerebrovascular and cardiovascular disease.

Case 03-106

This woman, born in 1876, entered a mental hospital in July 1930, with a diagnosis of involutional melancholia. She is one of a group who in that period was being given a therapeutic trial of intravenous radium chloride,[5] and received 160 μg of radium in a series of 10 μg injections between March and July, 1931. She was discharged in the custody of her daughter in 1935, and was then lost to follow-up until recently. As the result of an exhaustive interstate search, evidence of remarriage in 1948 and a 1959 death certificate were obtained, showing that death was caused by a bone sarcoma of the left leg, metastatic to the liver and abdomen. Skeletal x rays had been obtained from Elgin in 1951 and reported as negative for radium effects on a patient bearing this patient's previous name, apparently a different, younger individual. The best estimate of mean skeletal dose to onset of the bone sarcoma (1957) is 1320 rads.

Case 05-953

This female patient, born in 1902, had for a long time declined to participate in studies, as she had lost two sisters with malignant disease who also had been dial painters, and was in quite good health. Records indicate

that she worked as a dial painter in New Jersey for 14 months in 1918–1919. In 1977 a malignant tumor of the right leg and hip was discovered. Amputation was refused at first, and she was treated by chemotherapeutic agents. Biopsy of a mass in the lower third of the femur showed a well-differentiated fibrosarcoma, and a mass in the right ilium with identical histologic appearance was considered metastatic. An amputation was carried out in November 1977 for palliative purposes. The patient died in June 1978 and there was no autopsy. Based on extrapolation from measurements of the amputated leg, the total skeletal dose is estimated as 6590 rads in 1977.

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RADIUM-INDUCED MALIGNANT TUMORS OF THE MASTOID AND PARANASAL SINUSES*

M. S. Littman, I. E. Kirsh, and A. T. Keane

In the records of 5058 persons with therapeutic or occupational exposure to radium, 21 patients with carcinoma of the mastoid and 11 with malignant tumors of the paranasal sinuses were identified. Induction time for mastoid tumors was 21 to 50 years, median 33; for paranasal sinus tumors it was 10 to 52 years, median 34. Dosimetric data are given for the patients whose body burdens of radium have been measured.

Observations and theoretical considerations strongly suggest that radon in the air of sinuses may be an important or perhaps a major factor in the induction of paranasal sinus malignancies in the radium cases. Given the relatively thick paranasal sinus mucosa, we suspect that radium-induced carcinomas probably begin primarily in the mucosa of poorly ventilated sinuses. If a radium-induced carcinoma had arisen in a well-ventilated sinus, it would probably have had its origin in the submucosal glands.

On the other hand, the epithelium of the mastoid air cavities is probably sufficiently close to bone to receive an oncogenic dose from radioactivity deposited in bone at the levels found in the cases in this study. In case 03-423 the mean bone-to-mucosa thickness of the mastoid air cells, measured with calibrated optics and a 10× objective lens, was 17 μm. Thus, the simple squamous and cuboidal mastoid mucosal cells are within α-particle range of bone.

We found a high proportion of mucoepidermoid carcinomas, comprising 38% of the mastoid and 36% of the paranasal sinus tumors. Three patients had antecedent bone sarcoma (20, 11, and 5 years), and in a fourth patient a bone sarcoma was discovered at autopsy. Radiographic changes in the mastoid and paranasal sinuses were similar to those seen in malignant tumors not associated with radium exposure.

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*Abstract of a paper to be published in the Am. J. Roentgenol.

More than 800 known persons exposed to radium before 1930 and another group of unknown size who received radium water or injections of radium from physicians are still alive and at risk of developing malignant tumors of the mastoid and paranasal sinuses.

DOSE-RESPONSE RELATIONSHIPS FOR FEMALE RADIUM DIAL WORKERS*

R. E. Rowland, A. F. Stehney, and H. F. Lucas, Jr.

Among 1474 women employed in the U.S. radium dial painting industry before 1930, there are 61 known cases of bone sarcoma and 21 cases of carcinoma of the paranasal sinuses or the mastoid air cells ("head carcinomas"). The relative effectiveness of ^{226}Ra and ^{228}Ra and dose-incidence relationships were examined for the 759 of these women whose radium body burden has been determined; there are 38 cases of bone sarcoma and 17 cases of head carcinoma in this group. Incidence (I) was expressed as tumor cases per person-year and the dose parameter (D) was the quantity (μCi) of radium that entered the blood during the period of exposure. To the observed data for each type of tumor were fitted equations that can be formulated from the general form $I = (C + \alpha D + \beta D^2)e^{-\gamma D}$, where C, the natural incidence for this population, was about 10^{-5} per person-year. For each equation, the best values of the dose coefficients were found by a least-squares fitting procedure. An equation of the form $I = (C + \beta D^2)e^{-\gamma D}$ provided the best fit for the bone sarcomas, when the dose was expressed as μCi of ^{226}Ra plus 2.5 times μCi of ^{228}Ra. An acceptable fit to the head carcinoma data was provided by the linear equation $I = C + \alpha D$, with D equal to μCi ^{226}Ra. As a test of bias due to selection of cases with known symptoms of malignancy, the analyses were repeated after removal of all cases for whom radium was determined only after exhumation, and no significant changes in the fitted coefficients were found. The dose-incidence equations obtained when the dose was expressed as average skeletal dose in rads are also given.

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*Abstract of a paper accepted for publication in Radiation Research.

SURVIVAL TIMES OF WOMEN RADIUM DIAL WORKERS FIRST EXPOSED BEFORE 1930*

A. F. Stehney, H. F. Lucas, Jr., and R. E. Rowland

Life table methods were applied to survival data on U.S. women radium dial workers in order to compare observed and expected deaths as a function of time after exposure to radium. The study population consisted of 1235 workers employed in the industry before 1930 for whom age and year of death, withdrawal, or loss from the study were known. Expected deaths were estimated from age- and time-specific death rates for U.S. white females. The closing year for analysis was 1976, so observation times of 45 to 60 years were possible. For all causes, 529 deaths before age 85 were observed vs. 461 expected, and the cumulative survival of the population was significantly less than expected at 10 and more years after first employment. Estimates were made of the net survival probabilities after elimination of risk due to the well-known radium-related malignancies, i.e., bone sarcomas and carcinomas of the paranasal sinuses and the mastoid air cells. There were 455 observed deaths from other causes vs. 460 expected, and there was no significant difference between observed and expected cumulative net survival at one-year intervals from zero to 59 years after first employment. These findings indicate that only the known radium-related malignancies contributed significantly to life shortening of the exposed population as a whole, but the presence of other radium-related causes of death may yet be detectable by examination of specific risks as a function of dose.

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* Abstract of a paper presented at the International Symposium on the Late Biological Effects of Ionizing Radiation, 13–17 March 1978, Vienna. The complete paper will appear in the Conference Proceedings to be published by the International Atomic Energy Agency.

THE GEOMETRY OF FLATTENED CELLS ON ENDOSTEAL SURFACES OF HUMAN BONE—IMPLICATIONS FOR THE INDUCTION OF OSTEOSARCOMA AND THE SHAPE OF THE DOSE-RESPONSE RELATIONSHIP

E. L. Lloyd, C. B. Henning, and M. A. Gemmell

Cells lining bone surfaces are thought to be the cells at risk for the production of bone tumors by alpha particle emitters, such as ^{226}Ra and ^{239}Pu. Hence, precise measurement of the size and location of these cells relative to bone mineral is necessary for meaningful dose vs. risk estimates in man. The nuclear dimensions of these lining cells in normal adult human bone were measured in the present study by means of techniques which left the surface layer of cells well preserved and in contact with normal undecalcified bone. The cells, which were found to be flattened, had an average length (± S.E.) of 7.84 ± 0.22 μm and an average width of 1.8 ± 0.8 μm. This particular geometry in conjunction with the direction of the radiation arising from bone mineral may favor malignant transformations over cell killing and may profoundly affect the shape of the dose-response relationships. As shown by others, the distended nature of these cells may provide proliferative advantages for the eventual development of tumors. These results taken together provide further evidence that the direct action of alpha radiation on the nuclei of flattened cells at bone surfaces appears to be the most likely initiating event for the induction of bone tumors.

Introduction

The high risk of bone tumors in persons with long-term burdens of more than 1 μCi of ^{226}Ra or ^{228}Ra has been well documented. Other alpha particle emitters, such as ^{239}Pu, which deposit on bone surfaces have also been shown to produce tumors in animals. The cells lining bone surfaces have been considered to be the cells at risk for the formation of these bone tumors.[1] Hence, a knowledge of the cell geometry at bone surfaces is a prerequisite for understanding the basic mechanisms of bone tumor induction. In a previous report[2] cells in culture have been shown to be transformed to become malignant by the direct action of 5.6 MeV α particles from a Tandem Van de Graaff machine. The cells which were irradiated were greatly flattened on the surfaces of Petri dishes. Although it has long been known that many bone surface cells have a similar flattened appearance, their dimensions were largely unknown, and hence, the extent to which our in vitro experiments simulated the in vivo geometry was uncertain.

We know of only one previous report on the dimensions of surface cells in human bone.[3] The author acknowledged that these preliminary results were severely limited in accuracy because of (1) poor fixation as used in the conventional histological techniques used to prepare celloidin sections and (2) the use of decalcified bone. In the present study, care was taken to minimize shrinkage of the cells by use of glutaraldehyde as a fixative and to leave the undecalcified bone intact with the surface cells. The shrinkage with this procedure was estimated to be less than 10%.

Materials and Methods

The bone used in this study was obtained fresh from the femur of a 65-year-old male whose leg was amputated because of an embolism. Two one-inch napkin rings of bone were taken from a portion 15 cm above the right knee (Figure 1).

The rings of bone were rinsed in phosphate buffered saline (PBS) before being placed in a 3% glutaraldehyde solution buffered with PBS at pH 7.2. Slices approximately 2 mm thick were cut from one of the bone rings with a Stryker saw. From these pieces, ~ 2 mm cubes were selected from the endosteum with the endosteal cell layer attached to the bone. The cubes were processed as described elsewhere for electron microscope studies,[4] but the time in each processing solution was increased. The samples were taken from the 3% buffered glutaraldehyde and rinsed three times in PBS, then post-fixed for 2 hr with 1.5% osmium tetroxide in 0.2 M s-collidine buffer. After a

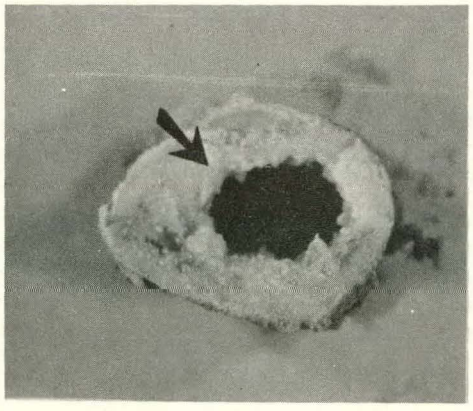

FIG. 1.--Cross section of human adult femur. The arrow indicates the endosteal bone surface where cell measurements were made.

rapid water wash, the samples were dehydrated in 50%, 75%, 95% (2 changes), and 100% (2 changes) ethanol for 10 to 60 min—the time increasing as the concentration of alcohol increased. For the final dehydration step, three changes of propylene oxide for 20 min each were used, and the samples were then placed in a mixture of araldite-epon in propylene oxide (1:1) overnight to start the infiltration procedure. The next day, this resin solution was replaced by a mixture of two parts araldite-epon to one part propylene oxide and left overnight. The samples were flat embedded in the desired positions and polymerized in fresh 100% epon-araldite in a 35°C oven overnight, followed by 8 hr in a 45°C oven and 48 hr in a 60°C oven. Then, they were removed from the flat embedding mold, trimmed, and sectioned on a Huxley ultramicrotome with a diamond knife. For the light microscope studies, sections ~ 1.5 μm thick were cut. These were transferred by brush to water droplets on a light microscope slide and allowed to dry and adhere on a warming plate. These slides were stained with crystal violet and safranin, and coverslipped for photography with a Leitz Labolux light microscope. For the electron microscope, sections 0.1 to 1.0 μm thick were cut from the same blocks as those used in the light microscope studies. Sections were transferred to copper grids, stained with uranyl acetate, and examined with the Siemens type 1A Elmiskop microscope.

Results

Light Microscope

Figure 2 shows a portion of the endosteal surface of bone with three flattened cells whose nuclei are clearly distinguishable at the bone-marrow interface. A similar pattern is also seen in Figure 3 where the flattened cells are associated with a bone trabecula. Figure 4 shows one of these cells at a higher magnification (× 1600), at which most of the measurements were made.

Measurements of cell nuclear thickness and length were made after checking the magnification using a micrometer stage photographed through the microscope. The distribution of lengths and widths of the cell nuclei of the 103 cells measured in this way are shown in Figures 5 and 6. The average

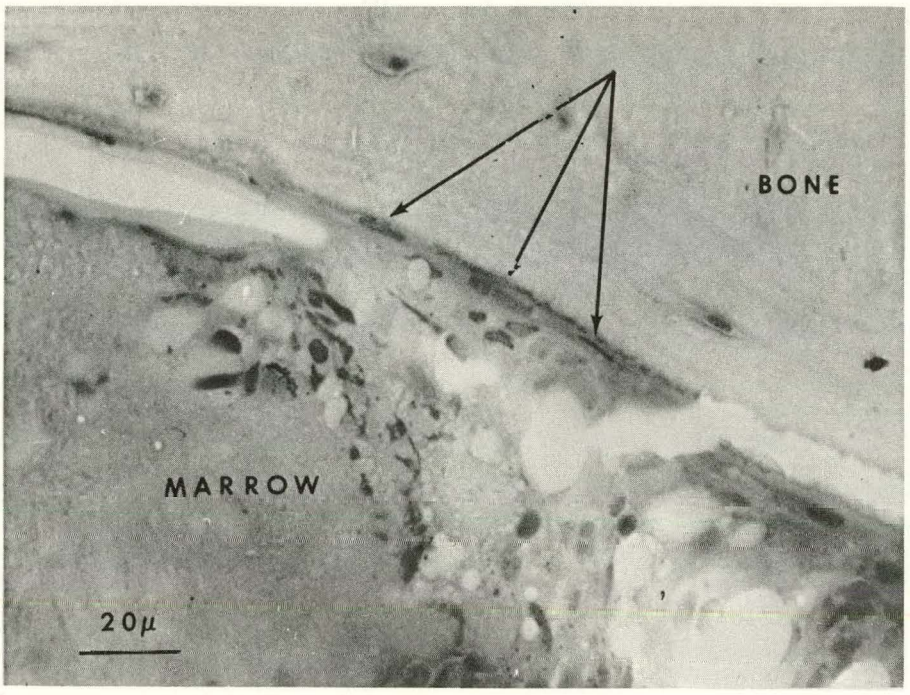

FIG. 2.--1.5 μm thick section of bone with endosteal cells attached. Arrows indicate the nuclei of three flattened cells.

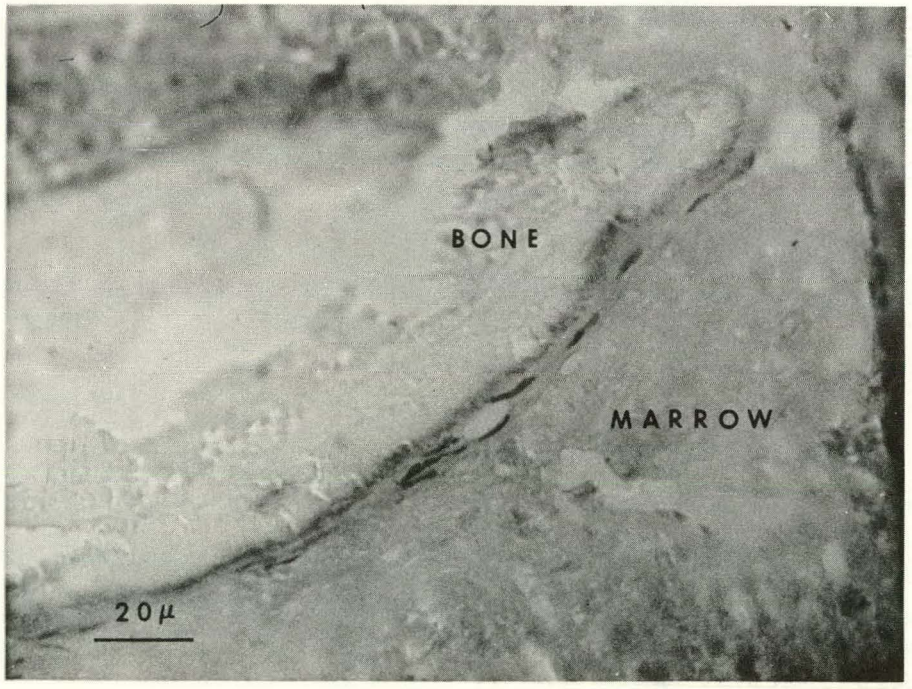

FIG. 3.--1.5 μm thick section showing a bone trabecula lined with flattened cells whose nuclei are clearly visible.

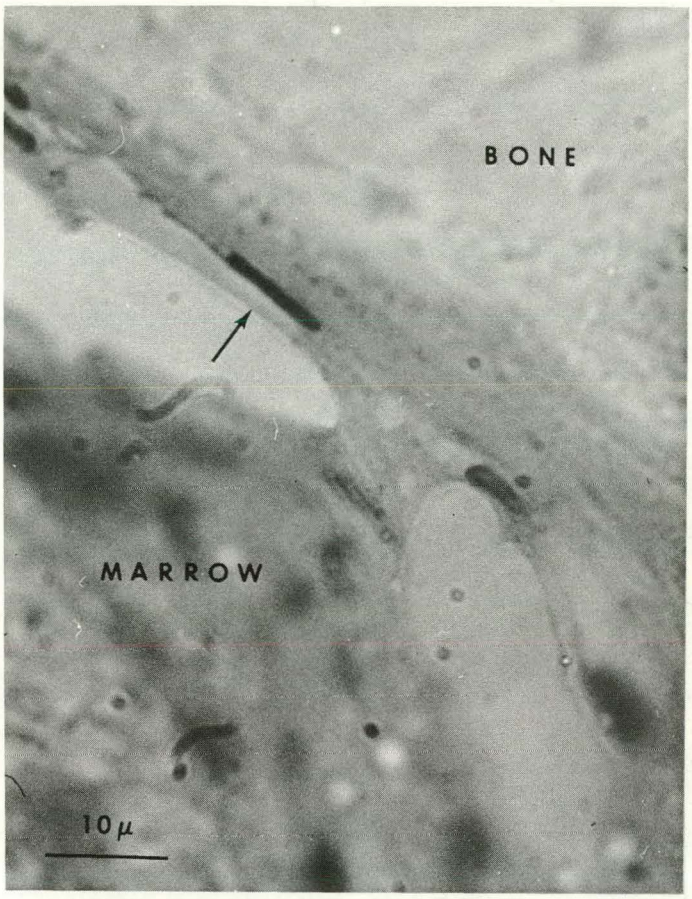

FIG. 4.--A single flattened cell nucleus shown at the magnification at which most of the cell nuclei were measured.

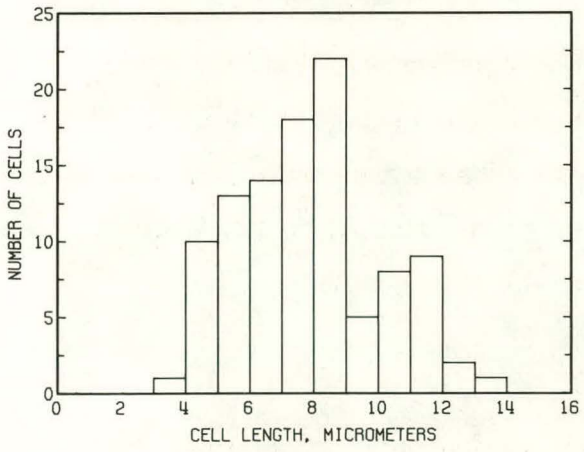

FIG. 5.--Histogram showing the distribution in lengths of the 103 cells measured.

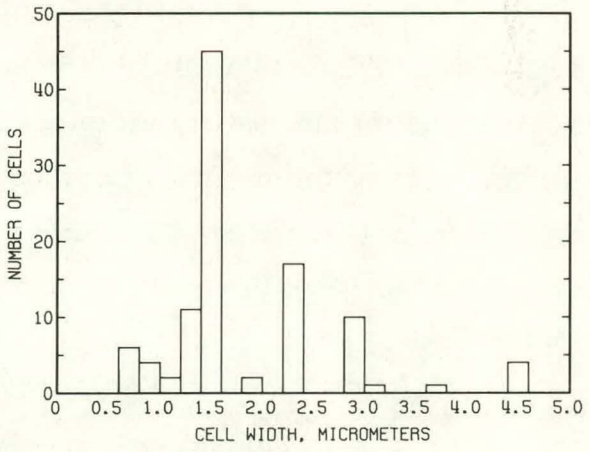

FIG. 6.--Histogram showing the distribution in widths of the 103 cells measured.

length with the standard error was found to be 7.84 ± 0.22 μm with the average width 1.8 ± 0.08 μm. The median and modal lengths were 7.4 and 7.35 μm, respectively, and the median and modal widths 1.5 μm and 1.4 μm.

If the lengths measured are considered as mean chord lengths of a circular area, the average nuclear diameter would be $\frac{4}{\pi} \times 7.84 = 9.98$ μm, and the nuclear area would then be 78.3 μm^2. Considering the nuclei to be flat cylinders of average height, 1.8 μm, this would give a nuclear volume of 141 ± 8 μm^3.

Electron Microscope

Figure 7 shows the appearance of part of the endosteal surface as studied with the electron microscope. Four distinct regions can be seen here—bone mineral, osteoid with collagen fibrils, and two cell layers. The flattened cell adjacent to the collagen layer is unusually long and thin (~22 μm long and ~1.5 μm wide). Collagen fibers can be seen on both sides of this cell and are most prominent at the membrane junctions at the middle and right-hand side of the picture. This suggests that the cell is in a state of active secretion even though very little cytoplasm appears to be associated with the cell as seen here. Figure 8, taken from a position more centrally located in the marrow cavity than that seen in Figure 7, shows the typical appearance of marrow cells. Many red blood cells and granulocytes with rounded nuclei are clearly visible. The spaces around the red blood cells at the top right of the picture correspond to fatty areas which were removed during processing. Often these fatty areas, together with marrow cells, were in direct contact with the bone surface without any lining cells.

Discussion

Although nuclear dimensions of only the flattened cells lining bone surfaces have been measured in the present study, it is of course recognized that other marrow cells lie within the range of alpha particles deposited in bone and may also be important for the production of bone tumors. However, there appears to be increasing evidence that cell geometry is important in regulating cell growth and that the flattened geometry of cells which are

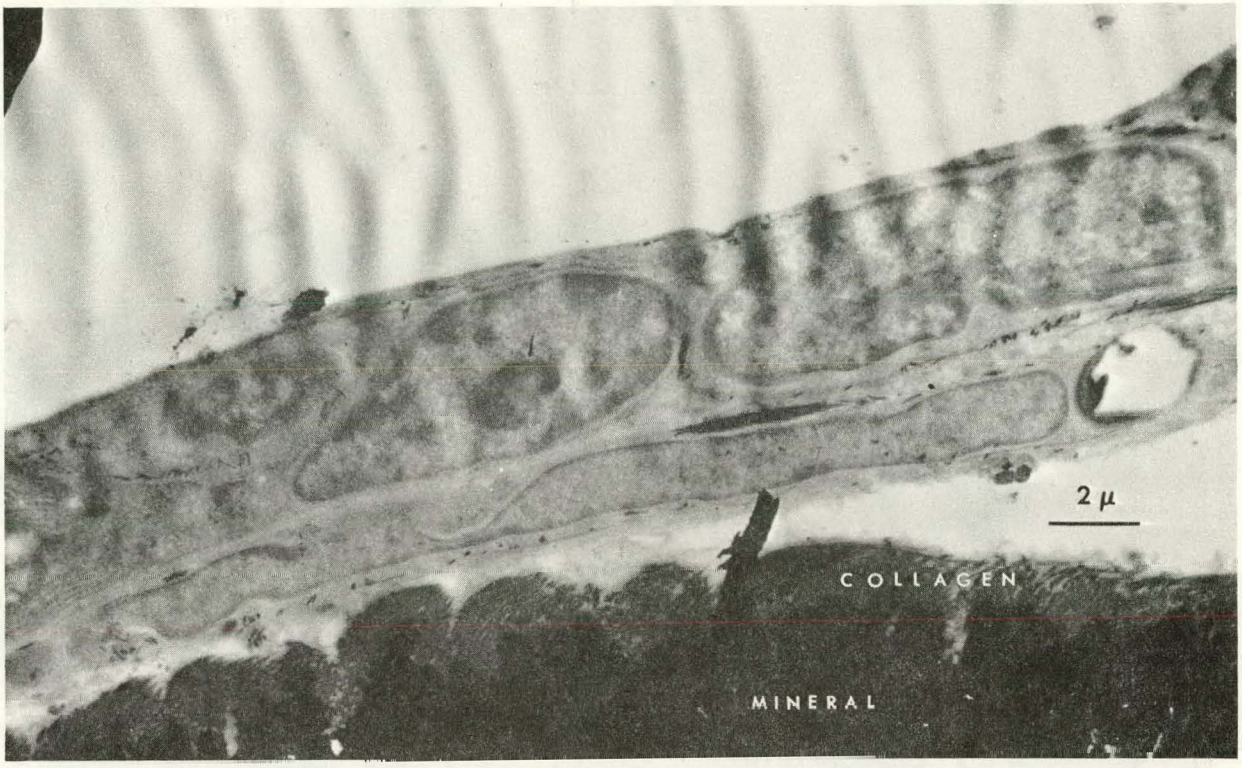

FIG. 7.--Electronmicrograph of the endosteal surface of the femur of a 65-year-old male. Two layers of cells are seen with prominent nuclei but little cytoplasm lining the bone surface. Collagen fibrils are clearly visible covering the bone mineral as well as at intercellular junctions between the two layers.

attached to a layer of collagen on bone surfaces might actually favor or be responsible for the proliferative activity of this surface layer as discussed by Folkman and Moscona.[5,6] It has been suggested that in most systems, cells need to tense themselves in order to divide, and that tension stimulates the cell cycle.[7]

In many early reports, the flattened cells against bone surfaces were considered to be mainly resting cells, but more recently Kimmel and Jee[8] have shown in dog bone that the uptake of DNA in flattened cells is similar to that observed in the better recognized osteoprogenitor cells found within 5 to 10 μm of bone surfaces. Traditionally, estimates of carcinogenic risks have been based on dosimetric measurements. A difficulty for those calculating

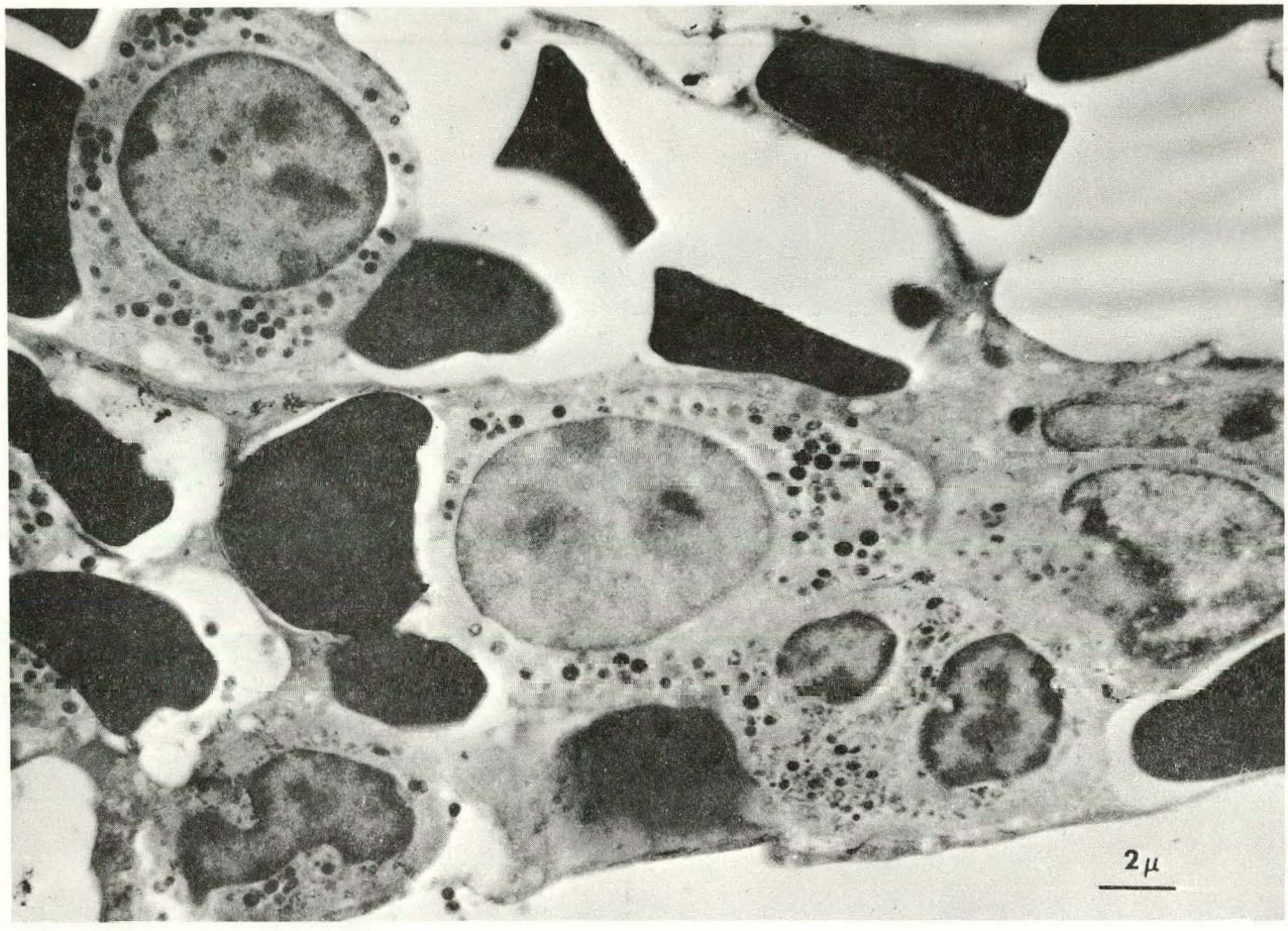

FIG. 8.--Electronmicrograph of a section of marrow tissue from the same bone, and close to the same site as that shown in Figure 7.

doses for alpha emitters which deposit in bone, has always been a lack of knowledge of the distance of the sensitive cells from the sites of radioactive deposition,[1] since dose decreases rapidly with distance from the source. If radium and plutonium in bone are deposited only in the mineral, as is generally supposed, and the sensitive cells for the production of bone tumors are the flattened cells, the distance in question (mineral to the center of the layer of sensitive cells) would appear to be of the order of 1 to 10 μm from our measurements. Further measurements of our electron microscope pictures, where the collagen covering bone mineral is well resolved, should give us more accurate figures for this distance than have previously been obtained.

In our in vitro transformation studies[2,9-11] the nuclear thickness of the cells which we irradiated was about 2 μm, in good agreement with the values obtained in the present study. In those studies in which a parallel beam of 5.6 MeV alpha particles perpendicular to the cells was used we found the cross section for cell killing was about 23 μm. This would correspond to the traversal of 3.4 alpha particles on average through a typical 78 μm^2 nucleus as measured in the present work. As discussed previously,[9-11] this flattened geometry allows for the distinct possibility that a cell whose nucleus has been hit by a single alpha particle may survive to become malignant.

The geometry of the cell might also be expected to influence the shape of the dose-response curve. In our in vitro experiments, transformation frequencies were found to increase with about the cube of the dose. However, when the angular distribution of incident particles, such as those arising from bone mineral, are considered, Marshall and Groer[12] showed that a possible linear-square dose response for the induction of bone tumors might be expected as a result of the flattened cell geometry shown here. On the other hand, carcinomas of the paranasal sinuses and mastoids in the radium cases arise from epithelial cells, which have more spherical nuclei. If osteosarcomas arise from flattened surface cells irradiated at right angles to their plane and carcinomas arise from spherical epithelial cells, these differences may help to explain the more nearly linear dose-response relationship observed for the carcinomas than for osteosarcomas in female radium dial workers.[14] Further in vitro work is planned to determine the dependence of dose response on cell geometry and angle of radiation.

Conclusion

Measurement of the dimensions of the cells lining bone surfaces, as presented here, not only gives us more insight into the likely mechanism of initiation of bone cancer by alpha irradiation, but suggests a possible reason for the different dose responses for the induction of osteosarcomas and carcinomas in the human radium population. In addition, more accurate doses can now be calculated to what appear to be the most important sensitive sites for bone tumor induction in man.

Acknowledgements

The authors wish to thank J. H. Marshall and R. E. Toohey for the computer printouts of the distribution of the lengths and widths of the cells measured. We also acknowledge the help of A. F. Stehney and R. E. Rowland who read the manuscript and made helpful suggestions.

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SUPPRESSION OF TRANSFORMED FOCI, INDUCED BY ALPHA RADIATION OF C3H 10T1/2 CELLS, BY UNTRANSFORMED CELLS

E. L. Lloyd, M. A. Gemmell, and C. B. Henning

The C3H 10T1/2 CL8 cell line obtained from a mouse embryo has been widely used for screening chemical carcinogens. Transformed foci are easily distinguishable in this system as crisscrossed, piled-up cells which stain more deeply than the surrounding untransformed cells. When these foci are ring-cloned and subcultured, they have been shown to give rise to malignant tumors in C3H immunodepressed mice. Previous work showed that such malignant transformations, which occurred with a dose dependent frequency, could be induced by alpha particle irradiation. The present study, in turn, demonstrates that the expression of these transformations can be completely suppressed by co-cultivating the transformed cells with a large number of untransformed cells. The precise ratio of the number of untransformed cells to transformed cells to give complete suppression was found to vary in different experiments. Maximum effects were seen when a small number of transformed cells in low passage were used. These experiments may provide at least a partial explanation for the greatly increased frequency of transformations per cell irradiated in vitro, compared with the number of tumors observed after irradiation of the same number of cells in vivo. In addition, if conditions could be optimized whereby transformed foci could reproducibly be eliminated by the use of a known number of untransformed cells, this might have important applications in the prevention and treatment of certain human cancers.

Introduction

The C3H 10T1/2 CL8 cell line (10T1/2) which is highly sensitive to postconfluent inhibition of cell division in culture has been widely used as a quantitative system for carcinogenic studies. Formation of morphologically and malignantly transformed foci of cells is readily scored by visual and microscopic examination. This cell line (10T1/2) developed by Reznikoff et al.[1] has now been shown to be capable of being transformed by many different physical and chemical carcinogens. Last year we reported, for the first time, transformations in this system using alpha particle irradiation.[2] Transformed foci appeared with a dose dependent frequency, and on isolation by ring cloning, these transformed colonies gave rise to fibrosarcomas when subcultured and injected into immunosuppressed mice.

During the course of these experiments, it was noted that the frequency with which transformations occurred was inversely related to the number of cells irradiated. This work, together with similar observations made by others using different carcinogenic agents,[3-9] suggested the need for investigating the mechanisms by which the presence of increased numbers of apparently untransformed cells inhibited the expression of the transformed foci in the same culture. This finding has important implications for our understanding of mechanisms of induction of human tumors and perhaps even for treatment.

Screening of carcinogens using the 10T1/2 system has generally been carried out at low cell densities (about 1000 cells/60 mm dish) where the cells are in the logarithmic phase of growth. The test is most sensitive at these low densities but simulates poorly the in vivo situation where cells generally exist in intimate contact with each other. This difference may be an important factor in explaining the greatly increased frequency of transformation per irradiated cell observed in vitro compared with the in vivo situation.

Agents which reduce the expression of transformations in vitro might also be expected to reduce the expression of malignancy in vivo if methods could be found for their effective application; e.g., Vitamin A and its analogs have been shown to be similarly effective in reducing transformed foci in 10T1/2 cell cultures[10] and are being considered for clinical trials as prophylactic agents in high risk cancer patients.[11] It does not, therefore, seem outside the bounds of possibility that normal cells from a person carrying a tumor might be useful in effecting the retardation of growth of malignant cells in the same individual, where cell rejection would not be expected to be a factor. Cell therapy for the treatment of cancer using mostly embryonic cells from other species has been practiced in European clinics[12] for several decades. These studies have been met with skepticism, usually because of exaggerated claims and lack of proper controls. However, if in vitro experiments could establish the effectiveness of normal cells on limiting the growth of malignant cells in cultures, a scientific basis at least for using one's own normal cells might be established. Of course, the effectiveness of such a treatment could ultimately only be validated in vivo.

There have been several reports in the literature of transformed cells having been added either before or after untransformed cells and allowed to grow together in culture.[4,8] In these experiments, the cells which were first added would appear to have a selective advantage for attachment and growth. In these experiments, although the untransformed cells were shown to diminish the number of transformed foci, as far as is known, there has been no previous report of complete suppression of foci as seen in the present study. In our experiments, the transformed cells were mixed with the untransformed cells before plating to prevent any selective advantage due to the order of plating. A given number of transformed cells was mixed with increasing numbers of untransformed cells and allowed to grow together in culture for 3 or 5 weeks, when they were stained and scored.

Methods and Materials

Stock cultures of the 10T1/2 cell line were maintained as described by Reznikoff et al.[1] The transformed cells used in this study were obtained from a transformed focus (94,2b) induced by alpha particle irradiation.[2] The focus used was a Type III focus which was subcultivated and shown to produce tumors in immunosuppressed C3H mice at passage 5. At the time of injection, the cells (P5) were split into three fractions and treated as follows: (1) Cells were used as described in the present experiment, (2) cells were frozen in liquid nitrogen at this passage for future use, (3) cells were further subcultivated for future use. The untransformed cells were used in passages between 15 and 20.

The transformed and untransformed cells, after counting, were made up separately in complete medium[1] to give the required number, using 0.5 ml per dish. Before plating, the transformed and untransformed cells were thoroughly mixed, care being taken to ensure a single cell suspension. One milliliter of the mixed cells was plated on each 60 mm diameter Falcon petri dish, to which 4 ml of medium was subsequently added. The cultures were maintained in a humidified incubator in an atmosphere of 5% CO_2 in air at 37°C. The medium was changed every 5 days until the completion of the experiment at 3 or 5 weeks, when the cells were fixed and stained as described.[2]

Results

Figure 1 shows the appearance of 20 transformed cells (passage 5) (top left) and 100,000 untransformed cells (top right) growing alone after 5 weeks in culture. The other petri dishes pictured in Figure 1 were obtained 5 weeks after plating 20 (left-hand side) and 200 (right-hand side) transformed cells with different numbers of untransformed cells. In the successive plates shown in each column, the number of untransformed cells was increased by a factor of ten from 10^2 to 10^5.

When 20 transformed cells were used, the transformed foci were seen to disappear completely when mixed with 10,000 untransformed cells. However, surprisingly, at the highest concentration, when 100,000 untransformed cells were used, the transformed foci reappeared and four transformed foci are clearly visible at the bottom left of Figure 1. A similar pattern was observed when 200 transformed cells were used (RHS, Figure 1), but although the number of foci was greatly reduced by using increasing numbers of untransformed cells, the foci failed to disappear completely at any of the cell concentrations used.

Since the plating efficiency of the transformed cells at this passage was found to be greater than 40%, if a single transformed cell gave rise to each individual focus, one would expect a constant number of about 80 foci per dish when 200 transformed cells were plated. In the dish to which 10,000 untransformed cells were added, the number of transformed foci was less than the expected value by an order of magnitude, although the number of foci increased when 100,000 untransformed cells were used with both 20 and 200 transformed cells.

The dishes shown in Figure 1 were part of a larger experiment which is illustrated in Figure 2. This shows the reproducibility of the result using 3 dishes at each cell concentration. In addition, the effect of using 500 transformed cells with different numbers of untransformed cells is seen. A similar pattern was observed when increasing numbers of untransformed cells were added.

Many investigators have shown that the potential malignancy of transformed cells increases with their time in culture following the initial

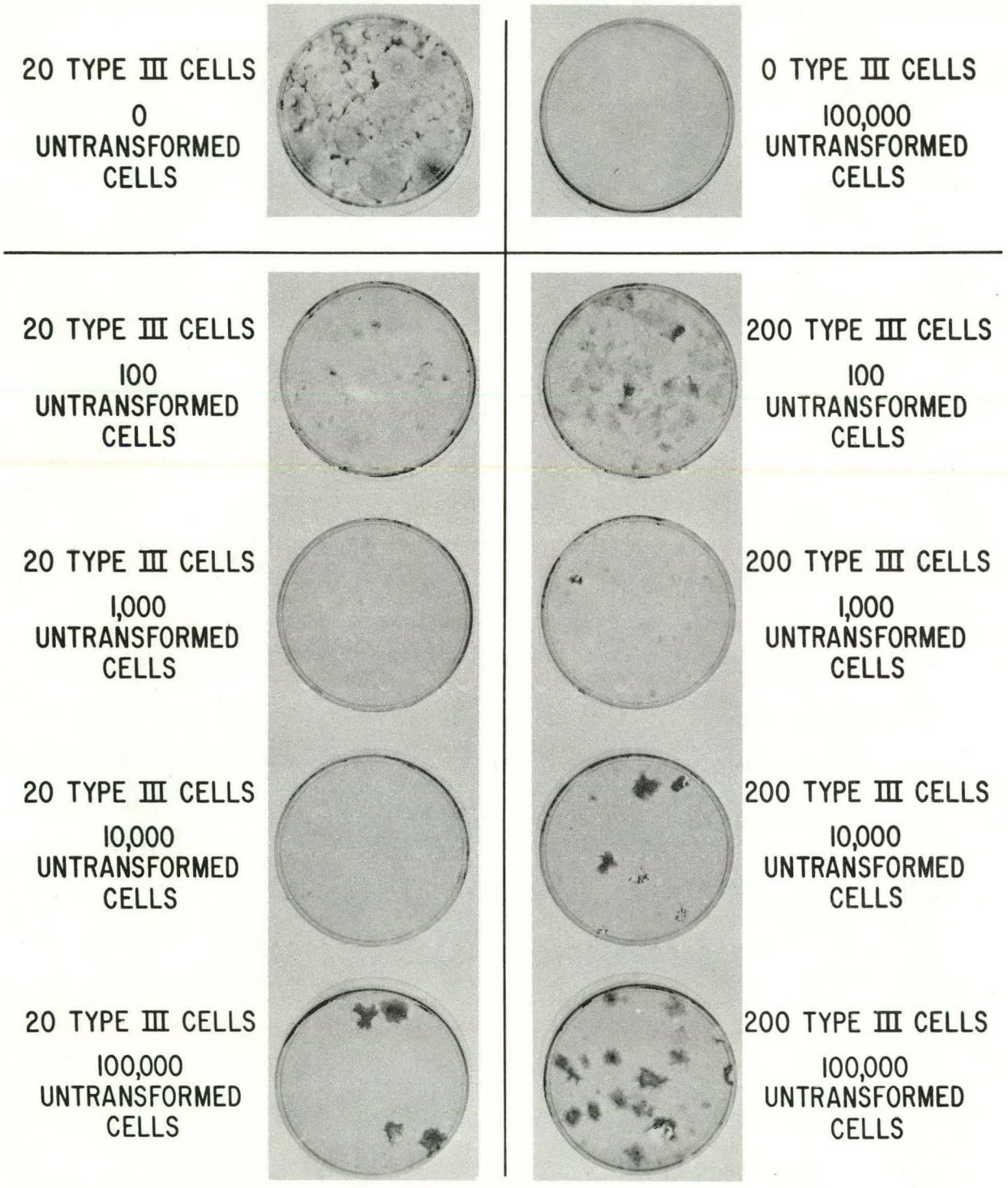

FIG. 1.--Control plates showing the appearance of 20 transformed cells and 100,000 untransformed cells growing separately for 5 weeks are shown at the top of the picture. The plates shown below illustrate the effects of different numbers of untransformed cells on the expression of transformed foci using 20 and 200 transformed cells in passage 5. (ANL Neg. 149-77-530)

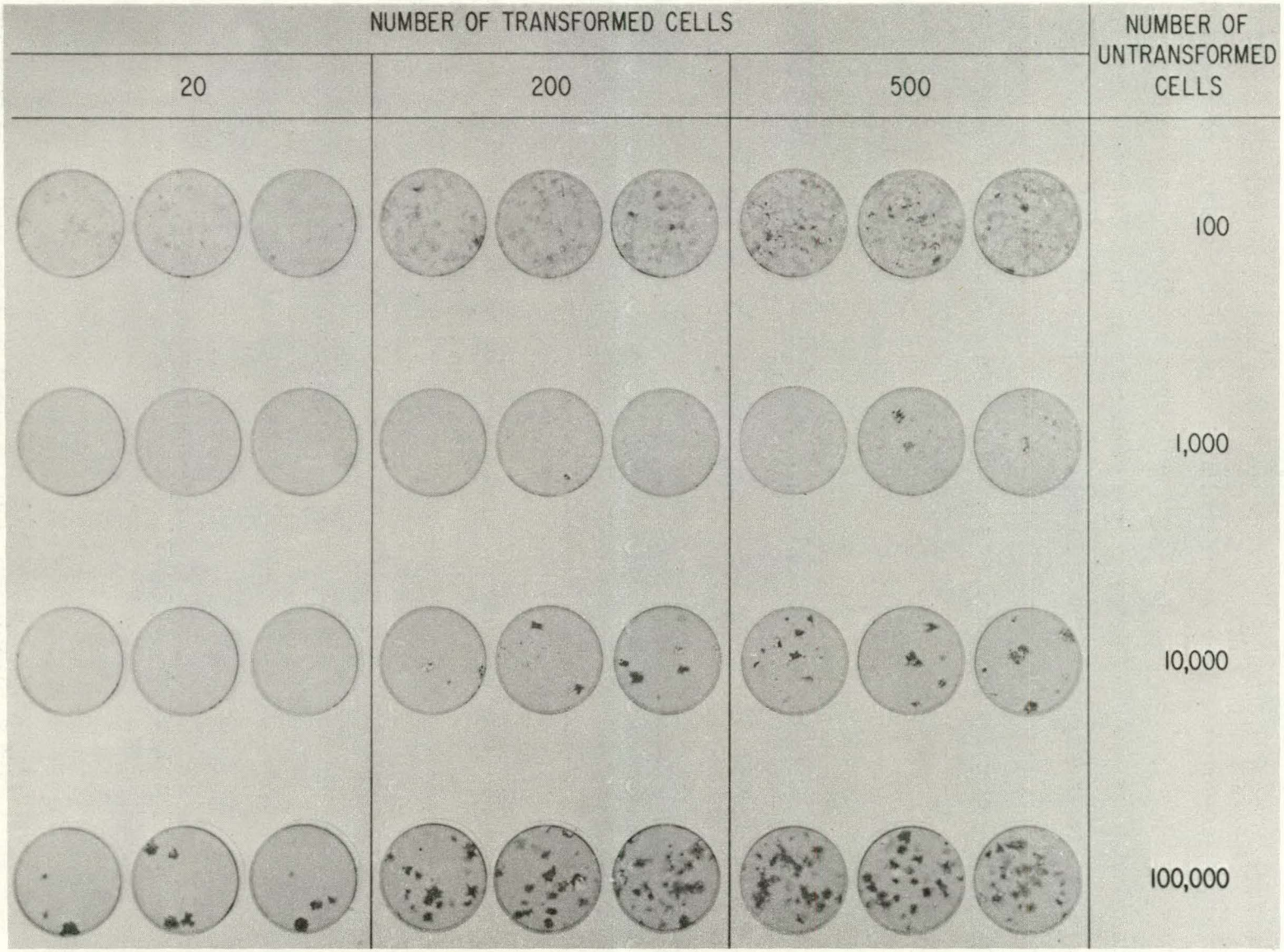

FIG. 2.--Plates showing the reproducibility of the results of a larger experiment from which the plates shown in Figure 1 were taken. In addition, the effect of different numbers of untransformed cells mixed with 500 transformed cells are shown. (ANL Neg. 149-78-124)

transformation event. Hence, the test was repeated with the same cells in passage 24 to compare with the results obtained when the cells were in their 5th passage. Figures 3 and 4 show the results of this experiment (using P24 cells) when the cultures were terminated at 3 weeks and 5 weeks, respectively. The number of foci observed was greatly increased at all cell concentrations compared with the number seen when the cells were in their 5th passage. The decision to terminate half of the cultures at 3 weeks was taken to avoid the loss of foci. These foci were easily dislodged on feeding and either lifted off the plate completely or gave rise to secondary foci. The bare patches, due to loss of piled-up foci, are clearly seen in Figure 4 (top left) when 20 Type III

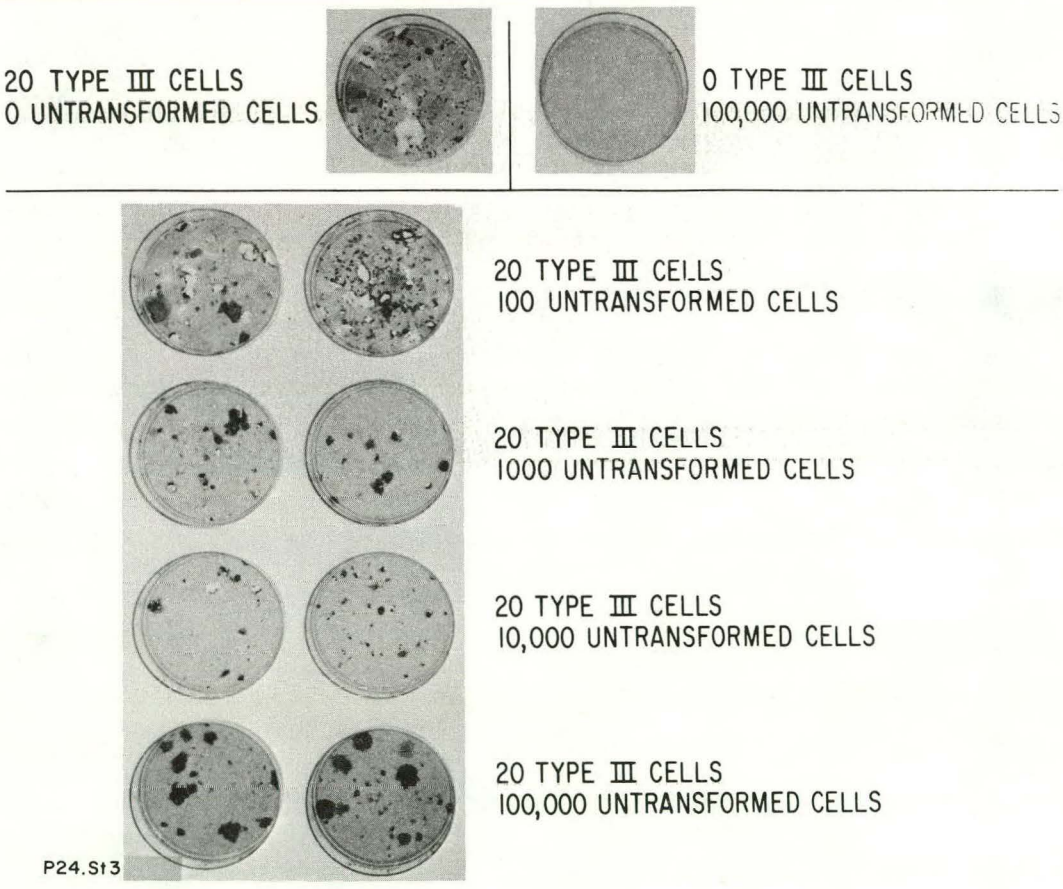

FIG. 3.--The effect of different numbers of untransformed cells on transformed foci using the transformed cells in passage 24 and stained at 3 weeks. (ANL Neg. 149-78-197)

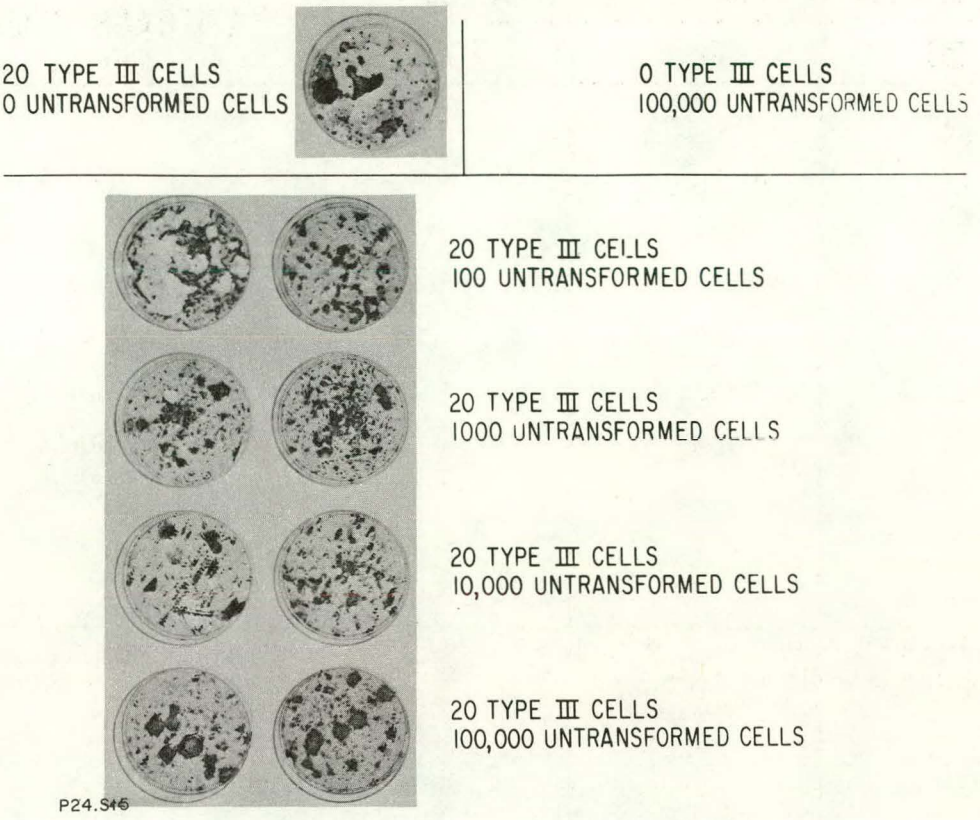

FIG. 4.--The effect of different numbers of untransformed cells on transformed foci using the transformed cells in passage 24 and stained at 5 weeks. (ANL Neg. 149-78-198)

transformed cells and no untransformed cells were used.

The effect of the increased number of passages from the time of formation of the original foci (from which all the subsequent cells were derived) was further confirmed by comparing the results seen in Figure 3 with an earlier experiment (Figure 5). In this case, the cells, which were treated as already described, were used in passage 5 prior to being frozen as part of the stock used in subsequent experiments. At this early stage, the use of 2,000 transformed cells and 170,000 untransformed cells resulted in only 9 visible foci, and no foci were visible when 200 transformed cells were mixed with either 17,000 or 170,000 untransformed cells.

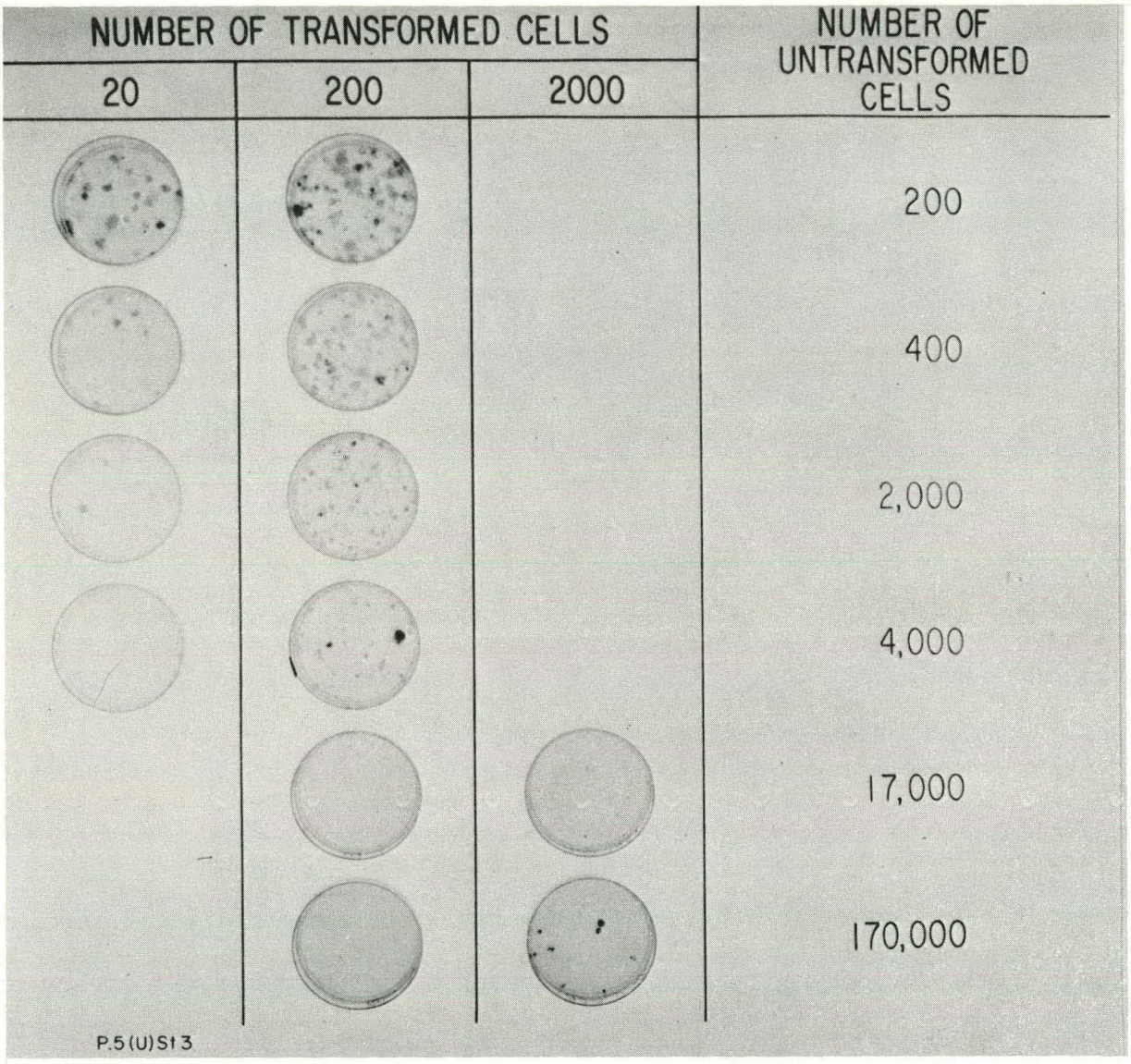

FIG. 5.--The effect of different numbers of untransformed cells on transformed foci in our original pilot experiment with the cells used in passage 5 before storing in liquid nitrogen (stained at 3 weeks).
(ANL Neg. 149-78-200)

Discussion

The results reported here are difficult to interpret. Because of the inverse relationship between the frequency of transformation and the number of cells exposed to alpha irradiation or other carcinogens, the reduction in the number of transformed foci by the addition of untransformed cells was to be

expected. However, as far as is known, this is the first time that complete suppression of foci has been reported. A further unexpected feature of the results was the reversal to more prominent foci when the number of untransformed cells was increased to 100,000.

The 10T1/2 cell line was originally used for detection of chemical carcinogens because of its postconfluent inhibition of growth of untransformed cells. Transformed cells, by contrast, have the ability to grow without attachment and are easily distinguishable in this system only after confluence, when they start to pile up. Cell contact, which has often been advanced as a mechanism for inhibiting further cell growth, may play a part in inhibiting the growth of the transformed cells at the intermediate cell densities. It is, however, difficult to understand why unrestricted growth of transformed cells appears to take place at the highest cell densities when greater cell contact would be expected.

An alternative to the cell contact theory is the possibility that critical amounts of secretory substances are necessary to repress the transformed state. The total amount of any substance elaborated by the cells would be expected to be dependent both on the number of cells present and on the number which are actively dividing. At the lowest cell density of untransformed cells used here (100 cells) the number of cells present may be inadequate, while at the highest cell densities the number of cell divisions may be insufficient. The number of cell doublings before the cells become confluent and supposedly stop growing is inversely related to the cell density. For the cell densities used, the approximate number of doublings before confluence vary between about 17, for 20 cells plated, to about 5, when 100,000 cells are plated (assuming 30% plating efficiency). Hence, for the highest cell densities used here, only 4 or 5 doublings would be expected to occur. This would be complete after a few days, a very short time compared with the 5 weeks used for the test. Another factor which may influence the results at the high densities is the possibility of continued growth after confluence, albeit at a very much slower rate. Some evidence for this is seen in the more darkly stained background of the plates seeded with the highest densities. This increased cell density

might in turn give the transformed cells selective advantages as the transformed cells can pile up on top of each other.

For the results observed here, the number of untransformed cells used and the cell ratio untransformed/transformed appear to be critical. The best results were obtained when only a small number of transformed cells were used and when they were in an early passage. However, it should be noted that at the earliest passage used here (P 5) these transformed cells produced tumors in mice. Other experiments carried out both in our own laboratory and by other workers have shown that transformed cells in later passages become more malignant. At this stage very little regression of foci was observed in the present experiment.

In conclusion, it is evident from this work that under some conditions, transformed cells can be made to alter their phenotypic expression to appear normal in culture as the result of the addition of untransformed cells. If these conditions could be reproduced in vivo, the use of normal cells might become an effective method for treatment of some cancers in man, particularly if diagnosed at a very early stage.

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TRANSFORMATIONS OF C3H 10T1/2 CELLS BY BENZO(A)PYRENE AND SUBSEQUENT ATTEMPTS AT SUPPRESSION OF TRANSFORMED FOCI BY UNTRANSFORMED CELLS AND VITAMIN A

E. L. Lloyd and M. A. Gemmell

The mouse embryo cell line (C3H 10T1/2 CL8) has been shown here, in agreement with findings by others, to be transformed by benzo(a)pyrene (BP). Transformation studies were carried out at two different concentrations (0.25 μg BP/ml and 2.5 μg BP/ml) and two different cell densities (200 and 1000 cells/60 mm dish). Transformation frequencies per surviving cell were found to be greatest when the higher concentration of BP was used with the lower cell density. A comparison of these results with our earlier alpha-irradiation experiments demonstrated the greater effectiveness of BP as a transforming agent in this cell system, although the foci produced by the two agents were morphologically similar.

Attempts made to eliminate the expression of BP transformed foci by two different techniques were unsuccessful, although one of these had previously been shown to be effective with cells transformed by alpha particle irradiation. The two systems tested were (1) treatment with retinyl acetate, a common nutritional form of Vitamin A and (2) the previously successful technique—growth of transformed cells with large numbers of untransformed cells. The differences between the BP-induced transformations and those induced by alpha particle irradiation may result from (1) intrinsic differences in the mechanism of action of the two carcinogenic agents, (2) differences in the number of cell generations between the induction of the transformed foci and the subsequent treatment of the cells, (3) genetic differences between different foci.

A common feature of all the systems tested was the progression towards malignancy with each succeeding generation, as cells passed from an environmentally constrained state to one of indefinite proliferation. Further studies of factors that inhibit this natural sequence of events may provide the most significant clues to the prevention of human cancers.

Introduction

The cell line C3H 10T1/2 (hereafter designated as 10T1/2) developed by Reznikoff et al.[1] has been widely used in screening tests for carcinogenic agents. We previously reported the first alpha-particle-induced oncogenic transformations with this cell line.[2] The experiment described here was undertaken to compare transformations by alpha particles with those induced by benzo(a)pyrene (BP).

As an extension of this work, attempts were made to inhibit BP-induced transformations. Two different inhibition techniques were used: (1) dilutions of transformed cells with untransformed cells and (2) treatment with retinyl acetate, one of the common nutritional forms of vitamin A shown by others to inhibit certain malignancies in animals.[3]

The results reported here emphasize quantitative differences in the transforming ability of different carcinogenic agents, and when considered together with other work, demonstrate important transition stages between normal cells and the cancerous state. When treatment is introduced at a sufficiently early stage in this progression, it would appear that expression of the transformed state can be inhibited, at least for some carcinogenic agents.

Materials and Methods

Treatment with BP for Transformation and Survival Studies

A total of 65 plates (Falcon 60 mm) were used in this study. Fifteen of these were plated with 200 cells per dish for colony counts and used in survival studies. The remainder were used for transformation experiments; 25 plates were seeded with 200 cells per dish and 25 with 1000 cells per dish. The cells (passage 15) were plated in 3 ml of Eagle's basal medium supplemented with 10% heat-inactivated fetal calf serum and 1% gentamicin. A stock solution of BP was made up by dissolving 10 mg in 10 ml of acetone (spectrograde) and diluting 0.5 ml of this solution with 49.5 ml of complete medium. This was stored at 4°C in the dark and used within 24 hr. All steps in the experimental procedure with BP were carried out in a minimum amount of light. Two concentrations of BP in complete medium, 0.25 μg/ml and 2.5 μg/ml, were used with each of the two cell densities of 200 and 1000 cells per dish. The cells were exposed to BP for 24 hr. Subsequently, the medium containing the BP was withdrawn and replaced with the usual growth medium described above. Thereafter, the medium was changed twice weekly until the cells reached confluence and weekly thereafter until termination of the experiment in the sixth week. At this time, many foci were observed, and a Type III transformed colony according to the criteria of Reznikoff et al.[1] was removed

from one of the plates for further subcultivation. The remainder of the cells was fixed and stained as described previously.[2]

Inhibition Experiments with Untransformed Cells and with Retinyl Acetate

Figure 1 shows the appearance of the transformed focus in the living culture, which was subsequently cloned and subcultured to supply the stock culture of transformed cells used in our inhibition experiments. This focus appeared in a dish treated with 2.5 μg/ml of BP. Transfer of untransformed (contact inhibited) cells lying outside the focus was avoided by use of a cloning ring which fitted well inside the perimeter of the piled-up cells. After 4 subsequent passages in culture, these cells were used in the experiments illustrated in Figs. 2 and 3. Transformed cells were mixed with different numbers of untransformed cells before plating as outlined in the preceding paper.

Six mg of retinyl acetate (kindly provided by Dr. Michael Sporn, Nat. Cancer Inst.) were dissolved in 6 ml of dimethyl sulphoxide (DMSO) under

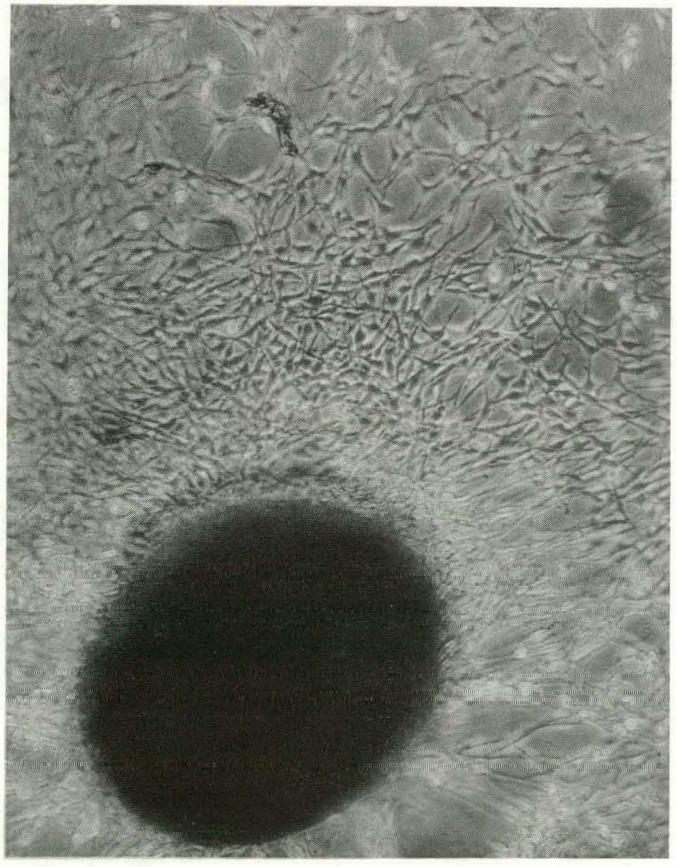


FIG. 1.--Focus of transformed cells induced by 2.5 μg BP/ml which was subsequently ring cloned and subcultivated to provide a stock of transformed cells.
(ANL Neg. 149-78-280)

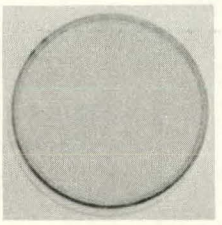
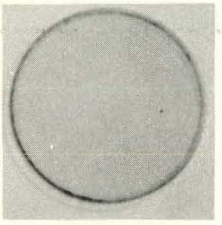
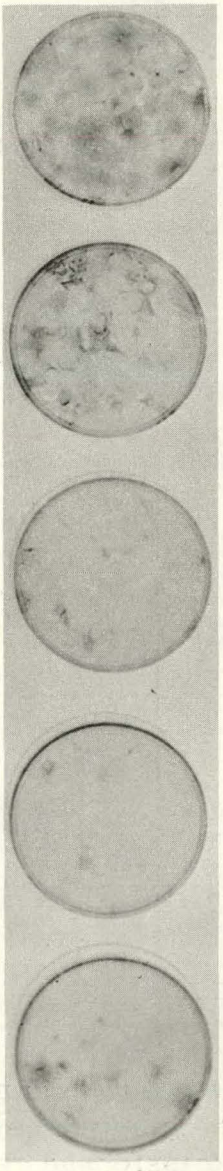
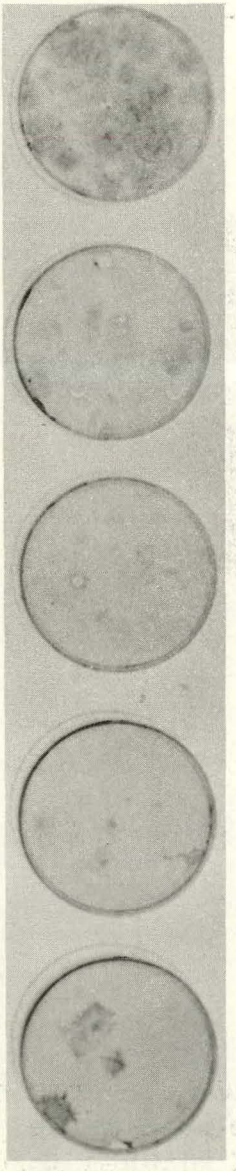
NUMBER OF BP TRANSFORMED CELLS	NUMBER OF UNTRANSFORMED CELLS	WITHOUT RETINYL ACETATE	WITH RETINYL ACETATE (0.5 μg/ml)
0	100,000		
20	0		
20	100		
20	1,000		
20	10,000		
20	100,000		

FIG. 2.--Transformed and untransformed cells with and without retinyl acetate. The cells were stained 6 weeks after seeding.

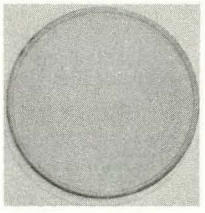
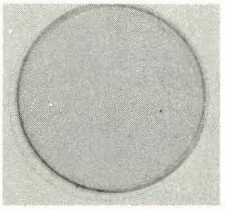
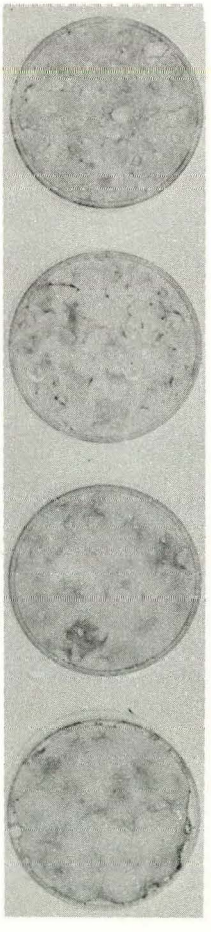
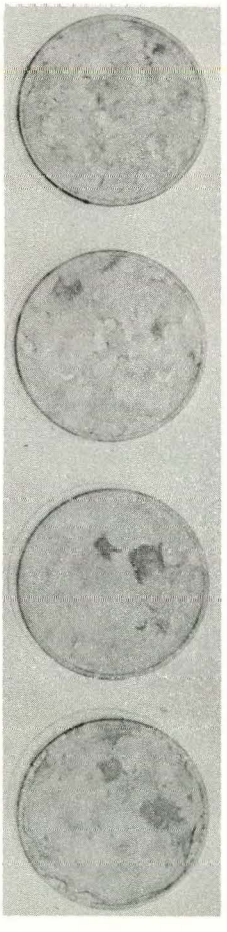
NUMBER OF BP TRANSFORMED CELLS	NUMBER OF UNTRANSFORMED CELLS	WITHOUT RETINYL ACETATE	WITH RETINYL ACETATE (0.5 μg/ml)
0	100,000		
200	0		
200	100		
200	10,000		
200	100,000		

FIG. 3.--Appearance of transformed and untransformed cells with and without retinyl acetate. The pictures below the horizontal line show the appearance of mixtures of 200 transformed cells with different numbers of untransformed cells with and without retinyl acetate. The cells were stained 6 weeks after seeding. (ANL Neg. 149-78-311)

minimum illumination. Two subsequent dilutions were made in modified Dulbecco's medium, made up with 10% fetal calf serum (FCS) to give a final concentration of 0.5 μg/ml of complete medium. The following cultures were treated with retinyl acetate: (1) cells subcloned and cultivated from the BP transformed colony as described above, (2) untransformed control cells, and (3) a mixture of transformed and untransformed cells in different ratios. Four weeks after seeding the cells were incubated with retinyl acetate for 6 days, after which the cultures were terminated. Control cultures were treated with medium containing the same concentrations of DMSO as those used in the retinyl acetate experiment (0.5 μg/ml medium).

Scoring of Transformed Foci

Foci were stained and scored by microscopic examination of cells as previously described.[2] Only type II and type III were scored (according to the criteria of Reznikoff et al.[1]), since type I is not usually considered to be malignant.

Results

Toxicity and Transformations in BP Treated Cells

Table 1 shows the toxicity of BP at the two concentrations used (0.25 μg/ml and 2.5 μg/ml). Table 2 compares the transformation frequencies observed at the same concentrations of BP. At the lower concentration (0.25 μg/ml), 15 of the 20 treated dishes contained foci, compared with 13 out of 20 at the higher concentration (2.5 μg/ml). In the controls, one type II transformed focus was observed in 10 plates, each of which had 0.5 μg/ml acetone added.

Effects of Untransformed Cells and Retinyl Acetate on Transformed Foci

Figure 2 illustrates the appearance of the cells stained 6 weeks after the plating of 20 transformed cells with different numbers of untransformed cells. The effects with and without retinyl acetate are also shown. Figure 3 illustrates the same experiment as Fig. 2 except 200 instead of 20 transformed cells were used; because of the large number of foci per dish and the uncertainty of the number of secondary foci arising from the detachment and reseeding of the transformed cells, no attempt was made to count individual foci.

Table 1. Toxicity of BP at Two Different Concentrations*

No. Cells Seeded Per Dish	Concentration of BP μg/ml	Surviving Colonies	Plating Efficiency	Surviving Colonies % of Control
200	0	88 ± 3	44 ± 1	100
200	0.25	74 ± 4	37 ± 2	84 ± 5
200	2.5	12 ± 1	6 ± 0.5	14 ± 2

*Five dishes were used at each concentration of BP and the mean ± S.E. is shown.

Table 2. Frequency of Transformed Foci After Treatment with BP

BP conc., μg/ml	Cell number seeded per dish	Number of dishes	Number of type III foci	Number of type II foci	% Frequency foci (II + III) per exposed cell	% Frequency foci (II + III) per surviving cell
0.25	1000	10	7	18	0.25	0.29
0.25	200	10	2	10	0.6	0.71
2.5	1000	10	6	12	0.18	1.3
2.5	200	10	3	9	0.6	4.2

Discussion

Cell Survival and Transformations Following Treatment with BP

The cell survival following treatment with BP found here—84% of control for 0.25 μg BP/ml—is in good agreement with that found by Mondal et al.[4] who reported 85% survival for the same cell system and the same concentration of BP. Terzaghi and Little,[5] who also used the 10T1/2 system, reported 46% survival with a dose of 1 μg BP/ml. This value lies between the value we obtained for 0.25 μg BP/ml and the 14% survival we found for 2.5 μg BP/ml.

As seen in Table 2, about three times as many type II as type III transformed foci were observed independent of the cell density or the concentration of BP. Although the total number of transformations (type II + type III) was smaller when 200 cells were seeded compared with 1000 cells, the percentage transformed was two to threefold greater than with the larger cell density. This effect has been noted previously for all carcinogens tested with this system and is discussed at length in the preceding paper.

No significant difference in the transformation frequency per treated cell was observed between the two concentrations of BP when either 200 cells or 1000 cells were seeded. However, the frequency of transformation per surviving cell was about 5 times greater with the higher concentration. Under the experimental conditions used here, the maximum transformation frequency per surviving cell observed was 4.2%. This was obtained when 200 cells were plated and treated with 2.5 μg BP/ml. In other systems, transformation frequencies per surviving cell have been shown to increase linearly with concentration up to 10 μg BP/ml.[6] In another report Terzaghi and Little[5] reported a value of 2.5 to 4 $\times 10^{-3}$ for the transformation frequency per surviving cell when about 1000 cells per dish survived a dose of 2.5 or 3 μg BP/ml. Based on figures of about 20% plating efficiency and 14% survival, this would correspond to about 35,000 cells initially seeded. The increased cell seeding density used by Terzaghi and Little compared with the 200 and 1000 cells seeded in the present experiment may well account for the different transformation frequencies observed in the two laboratories.

Effects of Untransformed Cells and Retinyl Acetate

As seen in Fig. 2, the use of increasing numbers of untransformed cells (without retinyl acetate) mixed with 20 transformed cells appears to inhibit the appearance of the transformed foci until the highest concentration of 100,000 untransformed cells is used. At this concentration, a reversal to more prominent foci is apparent. The use of retinyl acetate (0.5 μg/ml) did not significantly affect this finding. Basically, the same pattern was seen using 200 transformed cells (Fig. 3) except that the foci were always more pronounced.

Comparison of Transformation Induced by BP and Alpha Irradiation

Figure 4 demonstrates the similarity in morphology of transformed foci obtained with BP and with alpha irradiation. There are, however, many important differences between the two carcinogenic agents. In the present experiment the maximum transformation per surviving cell (4.2%) as a result of treatment by BP was obtained when 14% of the cells were still alive, compared with 1% survival when the maximum transformation was observed in the alpha irradiation experiment. Moreover, as already indicated, a much higher maximum transformation frequency might have been expected if we had used higher concentrations of BP than those used here—values up to 23% have been reported for 40 μg BP/ml.[6] By contrast, in the alpha irradiation experiment[2] the maximum transformation frequency of ~4% was obtained by generating a dose-response curve which was shown to reach a plateau at this observed maximum frequency. Further increase in dose did not result in higher frequencies of transformation. From these results it would appear that BP is much more efficient at transforming cells in culture than any of the radiation treatments so far employed. The difference in the shape of the dose response for the two transforming agents is also significant. For BP the transformation frequency has been reported to be linearly related to concentration at the lowest levels tested,[6] whereas an approximately cubic relationship was found for transformation frequencies as a function of alpha radiation dose.[2]

In our alpha radiation experiment the number of passages from the time of formation of the original foci had a profound effect on the ability of untransformed cells to inhibit focus formation (preceding paper). In the fifth passage, the appearance of transformed foci was completely suppressed, but in the 24th passage little or no suppression was observed. In the experiment described here, the BP transformed cells in their 4th passage behaved like the alpha particle transformed cells in the later passages. These findings are similar to those reported for inhibition of 3-methylcholanthrene (MCA) transformed foci by retinyl acetate. Merriman and Bertram[7] found they could inhibit focus formation when retinyl acetate at a concentration of 0.1 μg/ml was added to their cultures seven days after the addition of the carcinogen, but "established"

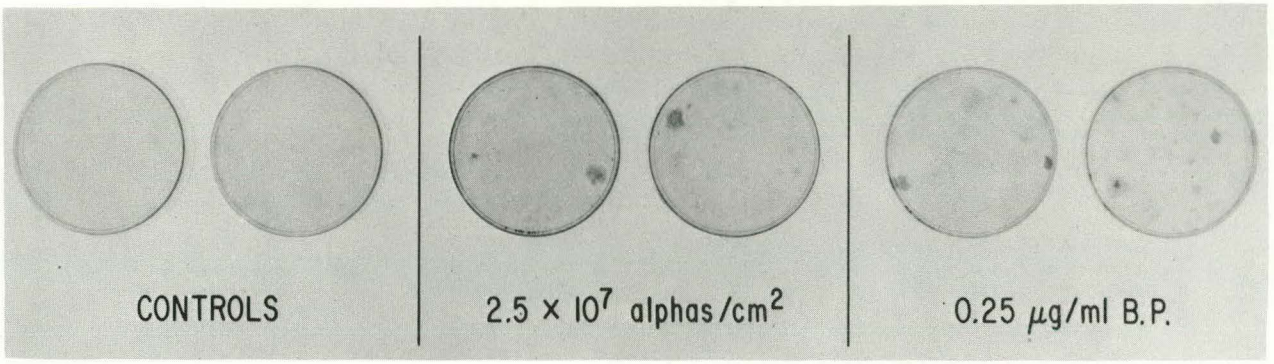


FIG. 4.--This shows the similarity in appearance of cells transformed by alpha irradiation and by benzo(a)pyrene. (ANL Neg. 149-78-123)

MCA transformed foci could not be inhibited even with higher doses of retinyl acetate.

The work described here, together with other reports describing attempts to inhibit expression of transformations both in vitro and in vivo,[3] underscores the fact that there are different stages of carcinogenesis. The 10T1/2 cell line, as used here, would appear to be a useful test system for further studies of this kind, which may hold the key to the prevention of tumors in man.

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LYMPHOBLASTOGENESIS IN RESPONSE TO PHYTOHEMAGGLUTININ IN RADIUM PATIENTS: QUANTITATIVE ASPECTS AND REPRODUCIBILITY

C. S. Serio, C. B. Henning, and E. L. Lloyd

Lymphocyte stimulation by different concentrations of phytohemmagglutinin (PHA) provided a profile of lymphocyte responsiveness in 5 normal subjects and 40 radium dial painters. This profile will, hopefully, improve detection of lymphocyte defects in patients with impairment of cellular immunity. When a suboptimal dose (0.15 μg PHA-protein) was used, decreased lymphocyte responsiveness was observed in patients with high body burdens of ^{226}Ra (> 0.1 μCi) compared with patients with low body burdens (< 0.1 μCi) and normal subjects. At all other PHA concentrations, the lymphocyte responsiveness was not significantly different in these three groups. A decrease in lymphocyte response was found with age in both normal controls and in the radium patients, but the decrease appeared to be somewhat greater in the radium patients above 50 years of age. To test the significance of these findings, larger numbers of control subjects in the higher age groups and low body burden radium patients known not to be on any type of medication will have to be examined.

Introduction

Lymphocyte transformation by phytohemagglutinin (PHA), an in vitro correlate of cellular immunity, has encountered wide acceptance in measuring impaired lymphocyte responses of patients with congenital[1,2] and acquired immune deficiencies.[3] Nonspecific stimulation in vitro by mitogen-induced lymphocyte DNA synthesis was utilized in the present experiments to measure the immunocompetence of 40 patients who had been exposed to ^{226}Ra either by injection for therapeutic purposes or by ingesting during occupational processes. These lymphocyte stimulation assays with a spectrum of PHA concentrations (from minimum to maximum stimulatory concentrations) provide a profile of lymphocyte responsiveness. By utilizing serial sampling of lymphocytes with various PHA concentrations this profile may improve detection of lymphocyte defects in patients with impaired cellular immunity and provide a quantitation of the impairment. Previous experiments[4] in our laboratory have demonstrated a reproducible microtechnique (< 10% variability within the same sample) for measuring stimulation of human lymphocytes by PHA. We have extended these studies as a means of determining if immunological impairment

is a precursor to tumor development in patients predisposed to neoplastic disease because of alpha particle emission from absorbed radium in the skeletal tissue.

The average age of the patients measured in these experiments is 60 ± 14 yr. In the interpretation of the data a discrete distinction must be made between impaired lymphocyte function due to a diseased state rather than alterations due to age-related effects, since there is well-documented evidence of impaired lymphocyte function in aged humans.[5-8] Although both male and female subjects were utilized in these experiments, no sex differences were noticed in lymphocyte responsiveness.

Materials and Methods

Study Population

Forty radium patients were investigated. Body burdens of these patients ranged from < 0.003 to 0.240 μCi of radium. Five patients had body burdens greater than 0.1 μCi ^{226}Ra, and 4 out of the 5 were in the 70 to 80-year-old age group, while the other patient was in the 40 to 50-year-old age group. The remaining low body burden patients ranged from ages 23 to 78. One of 5 approximately age-matched controls (unexposed to radium) was assayed on the same day as the radium patients. To avoid circadian influences, all work was carried out at the same time of day, beginning with venipuncture at approximately 9:00 a.m.

Lymphocyte Separation and Cultures

The preparation of human lymphocytes for PHA stimulation was performed according to a previously described method[4] with slight modifications. Briefly, lymphocyte-rich plasma was obtained from 20 ml of freshly drawn heparinized (preservative-free heparin, Connaught Lab., Toronto) blood. The blood was centrifuged for 10 min at 170 g, the plasma was aspirated, and the remaining cells diluted with 10 ml of RPMI 1640 containing 15 ml of a 1 M HEPES buffer (Gibco) and 0.5 ml of a 10 mg/ml gentamicin reagent solution per 500 ml of RPMI 1640. Red blood cells were sedimented with 3 ml of a 2% methyl cellulose solution in normal saline for 40 min at room temperature. The supernatant was

removed and centrifuged at 800 g for 10 min. To the resulting pellet 5 ml of an ammonium chloride solution was added containing: 0.8% NH_4Cl, 0.1% EDTA Na_3, 0.01% KH_2PO_4, pH 7.0 to lyse any contaminating erythrocytes. The resulting population of leukocytes was washed with phosphate buffered saline (PBS), counted with the aid of a hemocytometer and adjusted to a concentration of 4×10^6 cells/ml.

Leukocyte cultures of 2×10^5 cells/well in 0.2 ml culture media were prepared in flat-bottomed microculture plates (Costar). The stock culture medium was made up as follows: 10 ml of medium containing 2 ml of heat-inactivated fetal bovine serum (FBS), 0.1 ml solution of 0.5 M 2-mercaptoethanol buffer solution (Sigma Chemical Company) diluted 1:50 in RPMI 1640 and 7.9 ml of RPMI 1640. One hundred microliters of this solution were dispensed per well to give a final concentration of 2×10^{-5} M mercaptoethanol and 10% FBS. Starting with a stock solution of 166 μg PHA-protein (Difco), serial twofold dilutions with RPMI 1640 were utilized to give the desired concentrations of phytohaemagglutinin. Each test was performed in 2 to 4 replicate wells with a total incubation period of 72 hr. At the start of the last 18 hr, cultures were pulsed with 0.5 μCi/well of tritiated thymidine (5 mCi/ml, New England Nuclear) in 50 μl of RPMI. Cells were harvested on glass filter strips with the aid of a multiple automated sample harvester (MASH II). Filters were cut, suspended in 10 ml of scintillation fluid (Econofluor, New England Nuclear), and subsequently counted in a Packard Tri-Carb Liquid Scintillation Counter.

Preparation of Leukocyte Subpopulations

Lymphocytes were separated from whole blood, adjusted to 2×10^7 cells/ml in RPMI 1640, and layered on top of a Ficoll discontinuous gradient consisting of various percentages of Ficoll (i.e., 10–20, 20–25, and 25–28%) prepared by dissolving a 40% stock solution of Ficoll with appropriate percentages of calcium- and magnesium-free phosphate buffered saline. This preparation was centrifuged at 1200 g for 30 min at 4°C. The resulting lymphocyte subpopulations which locate at the various Ficoll interfaces were aspirated from the gradient, washed three times in RPMI 1640, and assayed for their response to PHA stimulation as previously described.

Results

Reproducibility between Normal Control Patients

In order to determine the reproducibility between the same and different control subjects, 5 normal individuals were tested repeatedly over an 8-month period. All volunteers showed maximum stimulation in the range of 0.1 to 10 μg PHA-protein per culture (Figure 1). The magnitude of lymphocyte responses to PHA varied from two to tenfold within each normal individual measured on different occasions. This intrapersonal variation occurred at each level of PHA stimulation as illustrated in Figure 1. Interpersonal variation could also be noticed, and in particular, an age-dependent factor seems to be suggested, especially between the youngest and oldest subjects. This age factor is best seen at the maximum response when 2.5 μg PHA-protein/culture was used.

Effects of Different Sera

In comparing the effects of autologous and fetal bovine serum on normal lymphocyte responseiveness to a variety of PHA concentrations, we found an enhanced lymphocyte response to FBS at both very low and very high concentrations of PHA but not at intermediate concentrations between 2.5 and 40 μg PHA-protein/culture (Figure 2). At the lowest concentration (0.15 μg PHA), lymphocytes in the presence of 15% FBS were enhanced in their responsiveness more than tenfold over those cells incubated with autologous serum alone. At concentrations of 0.6 μg and 160 μg of PHA, a twofold increase could be noticed.

The effect of heterologous serum from high and low body burden patients with and without tumor was also studied. When lymphocytes were incubated with heterologous serum from patients with tumors, either osteosarcoma or lung carcinoma, a decrease in lymphocyte responsiveness was observed at both high and low PHA concentrations, but not at the intermediate (2.5 to 10 μg PHA-protein) concentrations (Figure 3). Heterologous serum from the higher body burden patient without tumor and from a normal patient (not shown) did not differ significantly from autologous serum in its ability to increase or decrease lymphocyte responsiveness.

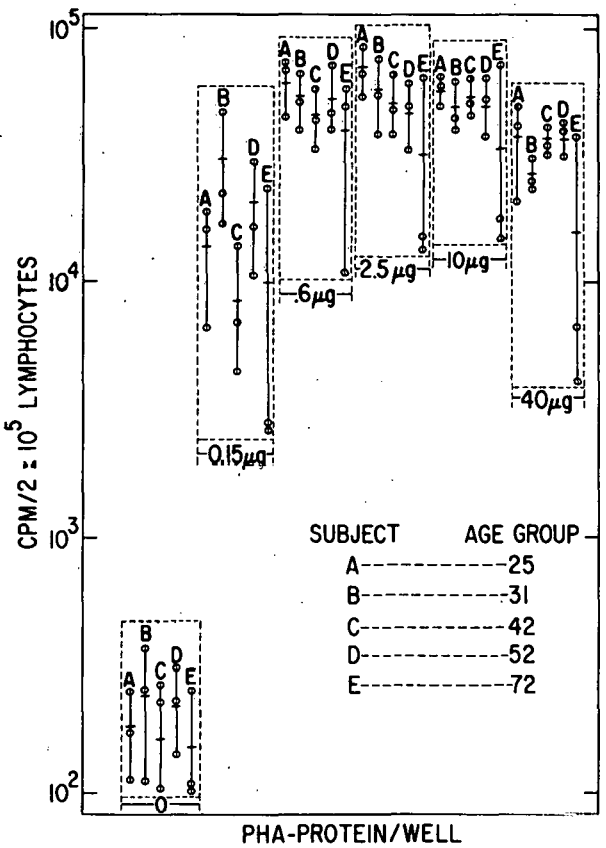

FIG. 1.--Reproducibility of the PHA stimulation of peripheral blood lymphocytes by ^{3}H-thymidine uptake. Five controls between the ages 25 and 72 were tested with 0 to 40 µg PHA.

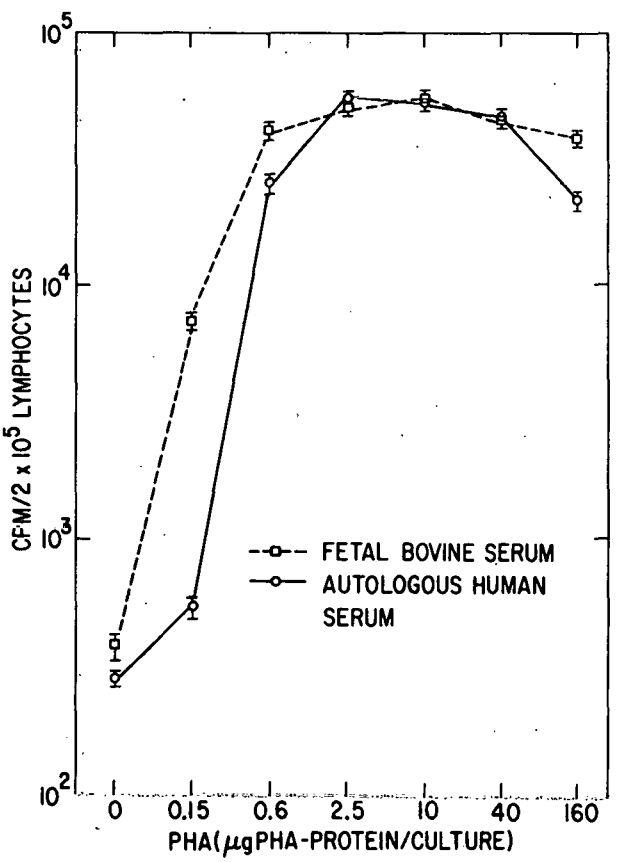

FIG. 2.--Comparison of the effects of autologous serum and fetal bovine serum on lymphocyte stimulation by PHA using ^{3}H-thymidine uptake. Mean cpm with the standard errors for triplicate samples are shown for each PHA concentration.

Variables Influencing PHA Stimulation Assays

In order to assess the quantitative differences encountered in PHA stimulation assays we examined various parameters of this assay to determine the effects of cell numbers, PHA incubation time, ^{3}H-thymidine pulse time, and number of wash cycles. We consistently received maximum stimulation with 2×10^{5} lymphocytes/culture when incubated with PHA for 42 hr followed by a 14 hr ^{3}H-thymidine pulse time. In harvesting the cell cultures no apparent difference could be noticed between 5 and 20 washes.

Expression of Lymphocyte Heterogeneity in Response to PHA Stimulation

The thymus-derived T cell is the most responsive cell in the peripheral blood to PHA stimulation. It is now obvious that various T cell-dependent immunological functions are mediated by subpopulations of T cells.[9–15] Characterization of these T cell subsets can increase our understanding of how T cells function. To this end we utilized velocity sedimentation in Ficoll discontinuous gradients to obtain lymphocyte subpopulations to study the response to PHA stimulation. Figure 4 demonstrates the division of these lymphocytes into 3 distinct subpopulations, each of which shows a characteristic lymphocyte response. Lymphocytes which were located in the 20/25% Ficoll interface maintained a higher degree of stimulation when compared with 10/20% or 25/28% Ficoll interface.

Effect of Age on Lymphocyte Responsiveness to PHA Stimulation

Forty radium patients and 5 healthy normal age-matched subjects all showed increasing degrees of lymphocyte transformation up to and including the maximum response with 2.5 μg PHA-protein/culture after which a decrease in activity was observed. The effect of age on lymphocyte response is shown in Table 1, for a concentration of 5 μg PHA-protein. In all groups, decreased lymphocyte reactivity was observed in the older subjects. Values obtained in the younger age groups (i.e., 20–29 to 40–49) show no significant difference between the normal control and radium patients, but there appears to be a difference in the above-50 age group between the normal control and both high- and low-level radium patients. However, to confirm this finding, it would be necessary to test several controls in these age groups. Medication-related

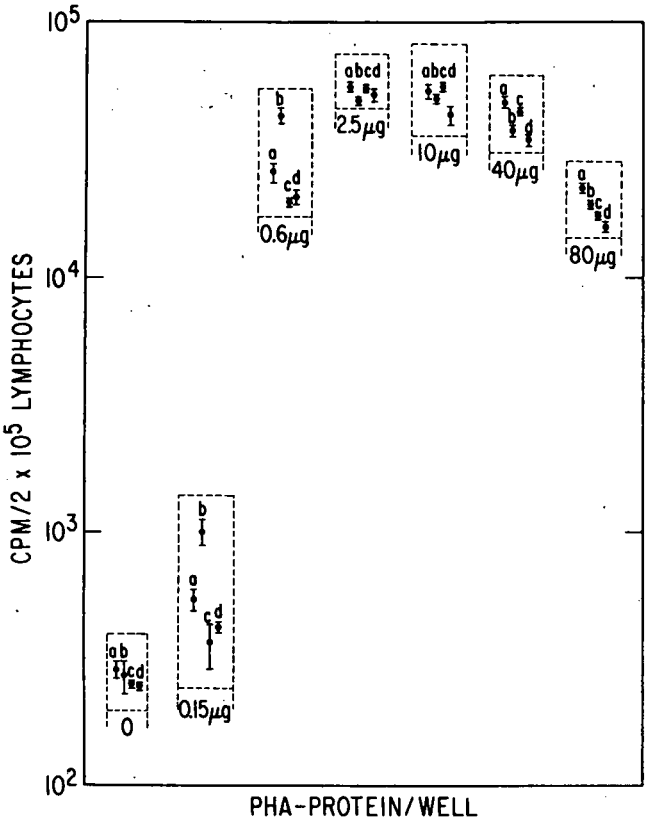

FIG. 3.--Effects of autologous and heterologous serum on PHA stimulation of lymphocytes from a normal 31-year-old subject. The values plotted are the mean cpm ± S.E. for triplicate samples measured at each concentration of PHA. a) normal lymphocytes + autologous serum; b) normal lymphocytes + heterologous serum from a patient with 0.65 μCi body burden of ^{226}Ra without osteosarcoma; c) normal lymphocyte + heterologous serum from a patient with 0.45 μCi body burden of ^{226}Ra with osteosarcoma; d) normal lymphocyte + heterologous serum from a patient with lung carcinoma.

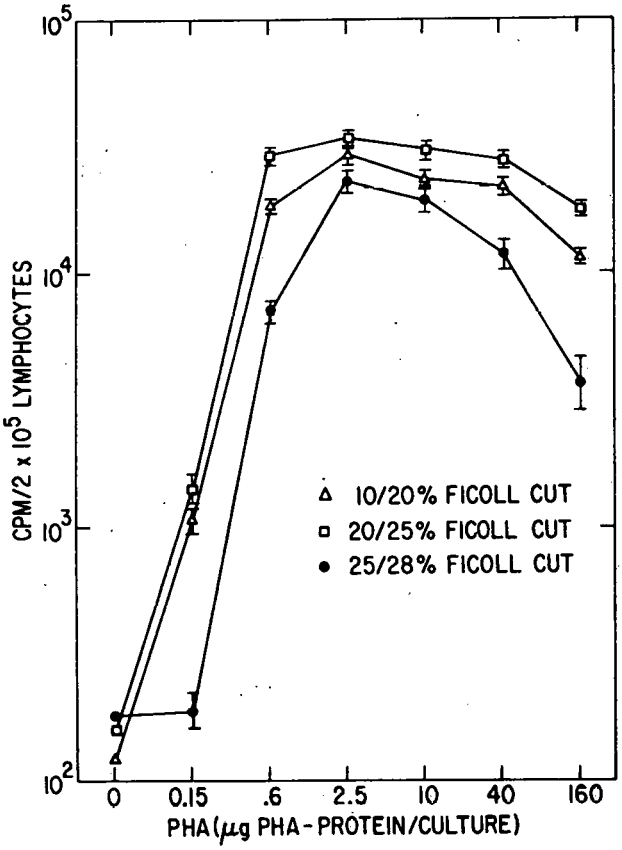

FIG. 4.--Expression of lymphocyte heterogeneity among subpopulations of peripheral blood lymphocytes when assayed for blastogenic activity by PHA stimulation.

Table 1. Effects of Age on Lymphocyte Responses to 5 μg PHA-protein

	Counts per minute per 2×10^5 lymphocytes (Mean ± S.E.)		
Age group	Normal control	Low body burden radium patients	High body burden radium patients
20—29	69790 ± 8870 (5)[a]	56390 ± 8110 (4)[b]	ND
30—39	56780 ± 10550 (9)	ND	
40—49	54060 ± 5700 (9)	54730 ± 4510 (3)	43370 + 1750 (1)[b]
50—59	54420 ± 9120 (4)	24080 ± 5770 (12)	ND
60—69	ND	21800 ± 8010 (7)	ND
70—79	40160 ± 14480 (3)	20040 ± 4340 (9)	16360 ± 13740 (4)

[a] Represents the number of times each individual was measured for lymphocyte responsiveness to PHA stimulation.

[b] Represents the number of radium patients tested for lymphocyte responsiveness to PHA stimulation.

effects should be considered, since they might account for the differences that were observed.

Comparison of Low and High Body Burden Radium Patients

To eliminate the age effect in order to determine if there was any significant difference between lymphocyte stimulation from patients with high and low body burdens, groups of 4 (> 0.1 μCi body burden) and 9 (< 0.1 μCi body burden) radium patients were examined from the 70 to 80-year-old group using the total spectrum of PHA concentrations. Table 2 demonstrates the results of this experiment in which a decrease in lymphocyte response is noted in the high body burden patients at PHA concentrations of 0.15, 0.6, and 2.5 μg. Significant differences were not detected at concentrations above 2.5 μg PHA-protein/culture.

Table 2. Comparison of Lymphocyte Responsiveness from 9 Low (<0.1 μCi) and 4 High (>0.1 μCi) Body Burden Radium Patients in the 70—80 Year Old Age Group

PHA concentration, μg/culture	Low body burden patients (0.001 μCi—0.075 μCi)	High body burden patients (0.139 μCi—0.240 μCi)
0	193 ± 61[a]	252 ± 18
0.15	5250 ± 1780	1010 ± 220
0.6	62780 ± 9980	30930 ± 3230
2.5	64910 ± 5420	41740 ± 2910
10	56790 ± 7140	45900 ± 2850
40	46580 ± 6670	40630 ± 2310
160	36620 ± 9430	25500 ± 7110

[a]Figures represent counts per minute ± S.E. in response to 2×10^5 lymphocytes/well at each concentration of PHA.

Discussion

Lymphocyte transformation due to phyto-mitogens, as well as to specific antigens, has been extensively investigated in recent years.[16] Of the various morphologic, biochemical, and radiological methods, the one favored for assessing lymphoblastogenesis is the measurement of ^{3}H-thymidine incorporation into DNA. Most peripheral blood lymphocytes are thymus-dependent, PHA-responsive cells[17] that form the basis of cellular immunity.[9] It is implied, therefore, that impaired in vitro lymphocyte responses to PHA reflect abnormalilities of in vivo expressed immunity.

Many attempts have been made to standardize the PHA stimulation assay in order to achieve uniformity of results and facilitate the highest lymphocyte responses.[18] Several investigators have attempted to establish a normal profile of lymphocyte transformation by exposing cells to several concentrations of PHA.[17,19]. This approach has uncovered subnormal responses

to submaximal doses of PHA in certain diseases.[20] In support of these findings, it has recently been reported that lymphocyte sensitivity (the response to the minimal stimulatory concentration of PHA) was abnormal in patients with disseminated Hodgkin's disease and metastatic solid tumors, while in most patients the maximum lymphocyte transformation remained normal.[9]

The results presented here also show maximum differences at suboptimal doses of PHA. We find these differences in the high and low body burden patients when the effects of sera from patients with and without tumor were compared, although no statistically significant differences at optimal PHA concentrations could be detected (Figure 3). The stimulation of normal lymphocytes reflected these same differences at the submaximum dose when comparing the effects of FBS versus autologous serum, cell number variations, PHA incubation time, ^{3}H-thymidine pulse, and heterogeneity of T cell subclass. Because of the steep dose response at suboptimal concentrations of PHA one would expect a greater variation in the results at low levels than at concentrations stimulating maximum responses. Like others,[21] we found that the intrapersonal variation was as great as interpersonal variation. These variations were most predominant at the lower concentrations of PHA. Since immunologic deficiencies are quantitative rather than all-or-none phenomena, it is to be expected that various degrees of response would be elicited according to the intensity of the stimulus.[22]

Our experiments on T cell heterogeneity showed that three subclasses of lymphocytes were distinct in their response to PHA stimulation. Further study of these subclasses could lead to the detection of immunologically incompetent lymphocytes which otherwise might be masked by the presence of whole populations.

In conclusion, the data suggest that a deficiency in the immunocompetence of high body burden radium patients can be detected when compared with patients with low body burdens and normal controls used in this present study, regardless of intra- and interpersonal variations. However, drug-related effects cannot, at this time, be ruled out as a possible cause for some of these differences. Finally, variations in cell numbers, serum, PHA incubation

time and ^{3}H-thymidine pulse time must be carefully monitored to obtain reproducible results.

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QUANTITATIVE HISTOLOGY OF HUMAN PARANASAL SINUS AND MASTOID AIR CELL EPITHELIA

M. J. Harris and R. A. Schlenker

A study was made of epithelial cell populations in the paranasal sinuses and mastoid air cells and their relationship to bone surface to provide a basis for practical microdosimetric calculations.

Introduction

Among radium cases, there is a high risk for carcinomas in the paranasal sinuses and in the mastoid air cells.[1-3] Little is known, however, of the dose delivered to the epithelial cells at risk because there are few quantitative data on the spatial arrangement and dimensions of potential targets of the radiation from radium and its daughters. The medical literature contains a number of studies of normal and pathological paranasal sinus and mastoid air cell epithelium,[4-7] but preparation methods or the scope of interest limit the usefulness of these studies for the kind of quantitative analysis needed to make them applicable as a base for dosimetric study. This study presents preliminary data on the normal relationships of sinus and air cell epithelium to underlying bone in nontumorous cases from the CHR postmortem collection.

Materials and Methods

Paranasal sinus and mastoid tissues were samples from the cases listed in Table 1. Material was fixed in buffered 10% neutral formalin or buffered 3% glutaraldehyde. Some samples were preserved by deep freezing, but they were found to be unsatisfactory because of ice crystal induced damage and unknown treatment prior to freezing. Other samples were of unknown preservation history and had significant autolysis, rendering them unsatisfactory for demonstration of epithelial structures. The useful specimens were those taken immediately postmortem and fixed well in formalin or glutaraldehyde.

A modification of the Woodruff and Norris[8] method for embedding was used, with care taken to achieve expeditious infiltration of the samples to minimize artifacts. Five-micrometer sections were cut from the embedded

Table 1. CHR and NWU[a] Cases Surveyed for Histological Suitability

Case No.	Site	Preservation method	Condition
01-051	Frontal, maxillary sinuses	Freezing	Poor cellular detail
01-141	Frontal, maxillary, spheroidal sinuses; mastoid, ethmoidal air cells	Buffered Glutaraldehyde	Good cellular detail
05-044	Frontal, sphencidal, maxillary sinuses; mastoid air cells	Buffered neutral formalin	Good cellular detail
A75 229[a]	Mastoid air cells	Buffered neutral formalin	Good cellular detail
10-247	Mastoid air cells	Buffered neutral formalin	Some cellular detail
01-543	Mastoid air cells	Freezing	Not studied
03-206[b]	Mastoid air cells	Freezing	Unsatisfactory
03-488[b]	Mastoid air cells	Freezing	Unsatisfactory

[a] From Northwestern University Medical School.

[b] Mucoepidermoid ca. cases.

samples with a Jung sledge microtome. Sections were mounted on gelatin-coated microscope slides and allowed to adhere overnight and stained with a hematoxylin and eosin combination.[9] The sections were coverslipped for microscopic examination and photographed with Polaroid Type 55 P/N film. The negatives were enlarged in a positive format convenient for measurement.

Tissue Shrinkage

The fixation and embedding necessary for histological work causes tissue shrinkage. In order to estimate the degree of shrinkage, the diameters of red blood cells in our sections were compared with published values of red blood cell diameters. Leeson and Leeson[10] stated that "in sectioned material the diameter (of an average RBC) is ... about 7 μm." They contrast this diameter to the 7.6 μm diameter for dried smears and to the 8.5 μm diameter of living undehydrated RBC's. Documenta Geigy Scientific Tables[11] report mean values for RBC diameter of 8.56 μm for wet preparations, 7.11 μm for dry preparations, and 8.70 μm for rouleaux formations.

We measured 25 RBC's from representative sections of paranasal sinus and mastoid air cell tissues and obtained an average diameter of 5.96 ± 1.44 μm. Using the value given by Leeson and Leeson of 8.5 μm for RBC diameter in living material, we found the shrinkage averaged 30%. We assume a similar distortion for other soft tissues in the specimen and believe that the measured values for epithelial dimensions, presented in the next section, may be low by 30%.

Results and Discussion

The sinus and air cell mucosa consist of two layers, the lamina propria, and the epithelium. The lamina propria is a connective tissue layer which lies between the bone surface and the epithelium giving structural support to the latter. The bone, lamina propria, and epithelium can all be seen clearly in Figs. 1–4, except in the mastoid air cell from case 01-141 (Fig. 3a) where no lamina propria is apparent. The morphological classification of the epithelial membrane is given for each illustration. Our classifications confirm those

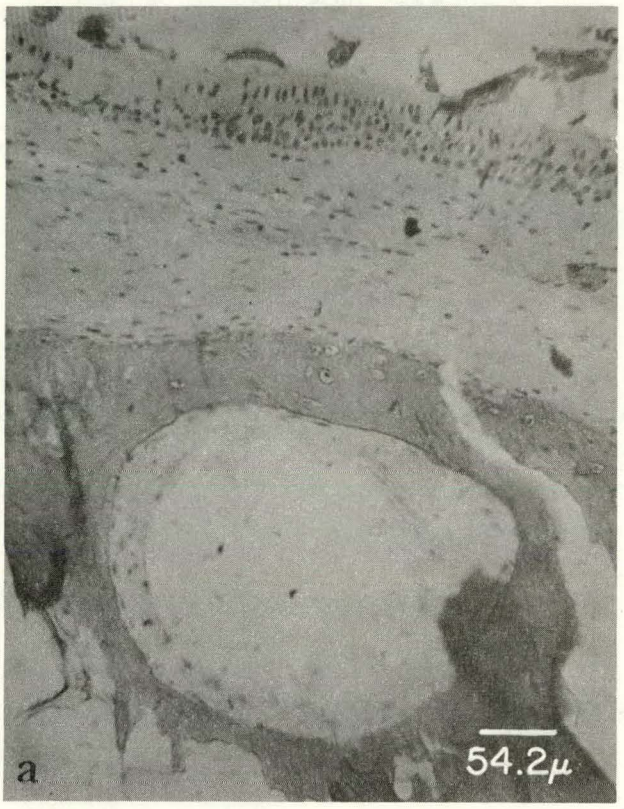
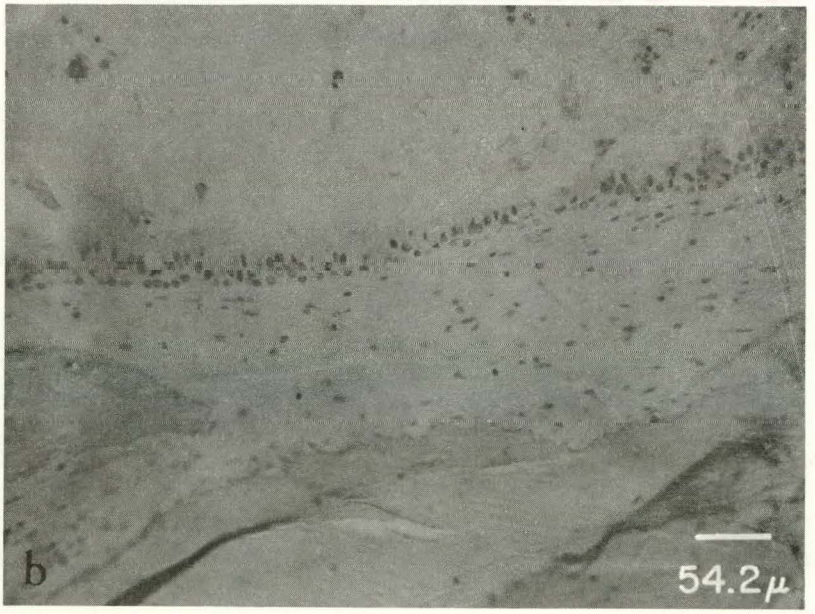


FIG. 1.--CHR case 05-044; pseudostratified, ciliated columnar epithelium; Mayer's hematoxylin.eosin Y. a, Frontal sinus; b, sphenoidal sinus.



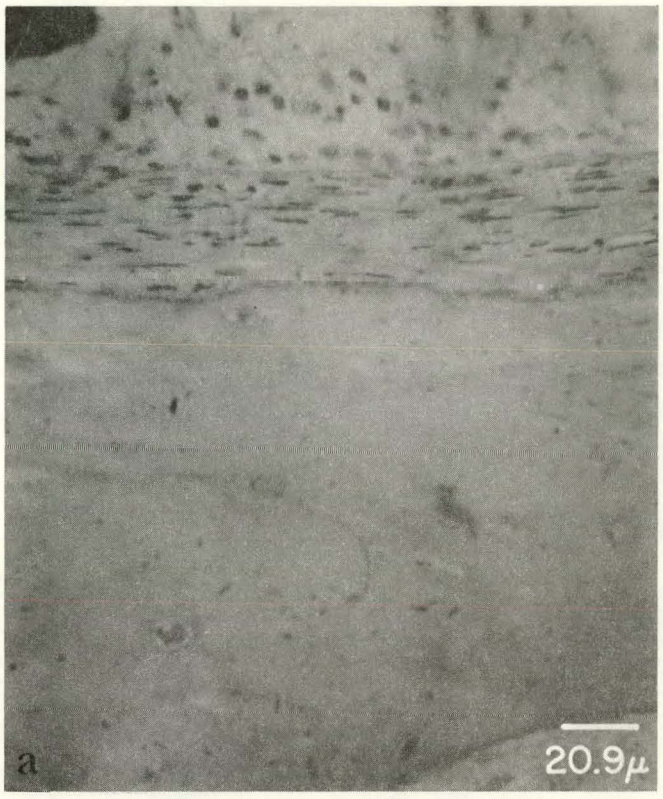
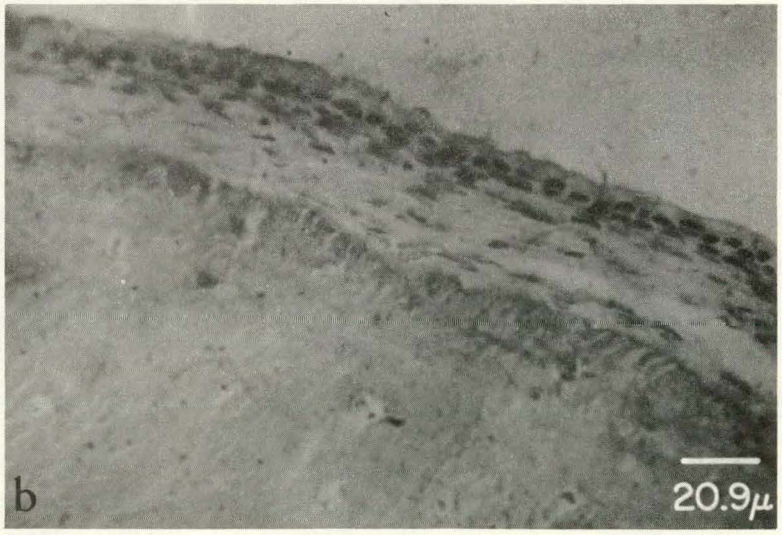


FIG. 2.--CHR case 01-141; Mayer's hematoxylin/ eosin Y. a, R. frontal sinus; pseudostratified, ciliated columan epithelium; b, L. frontal sinus; stratified cuboidal epithelium.



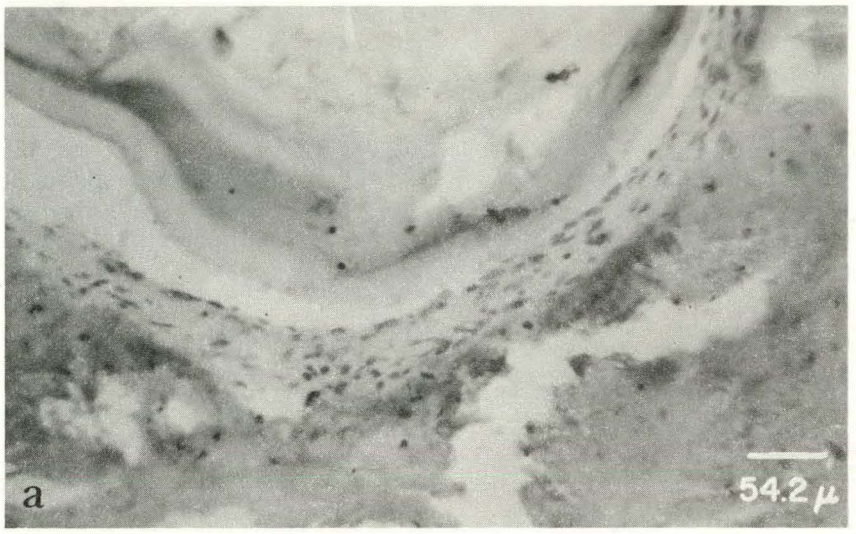
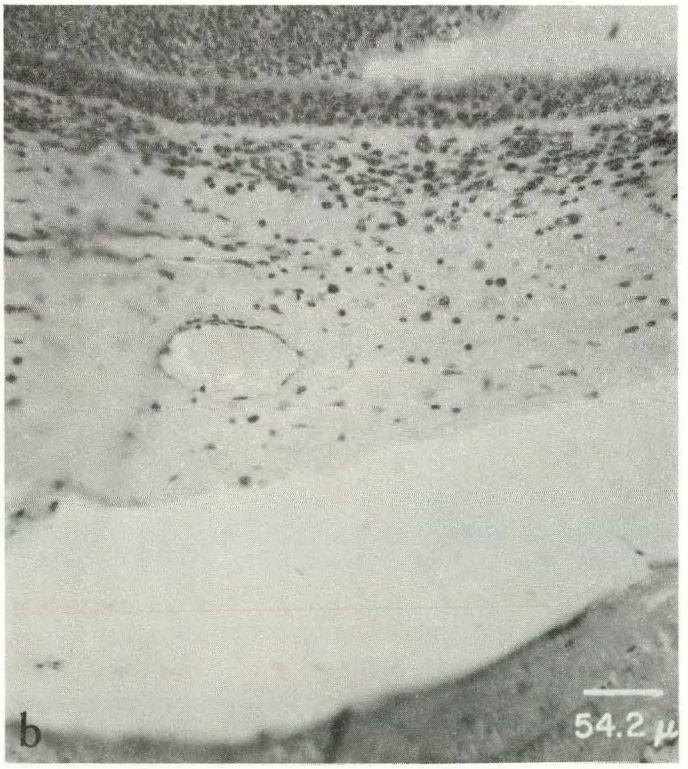


FIG. 3.--CHR case 01-141; stratified cuboidal epithelium; Mayer's hematoxylin/eosin Y. a, Mastoid air cell; b, sphenoidal sinus.



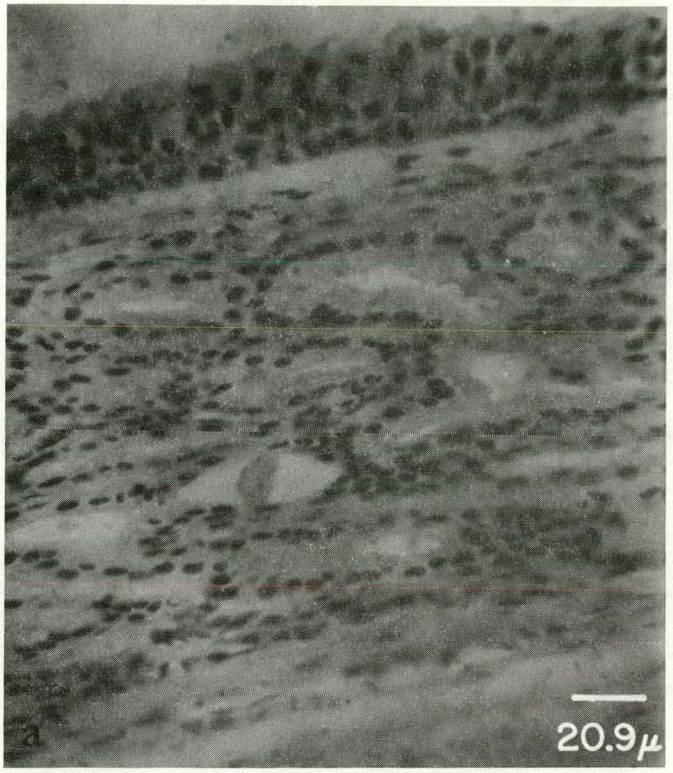
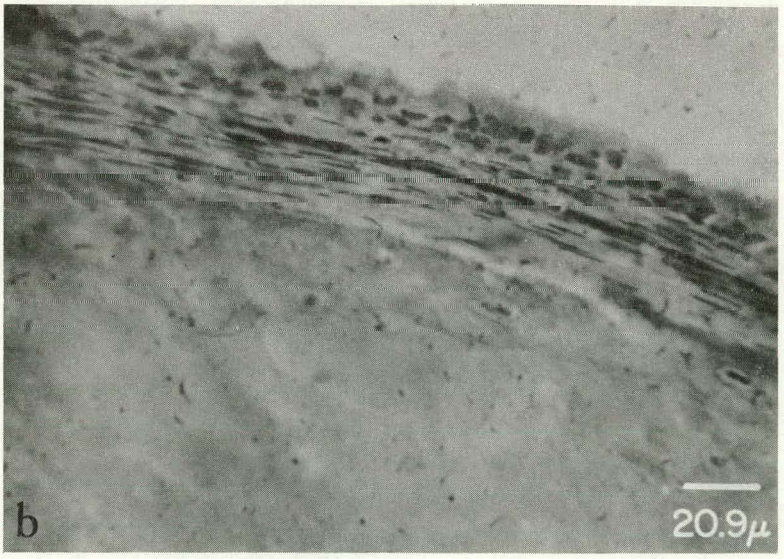


FIG. 4.--CHR case 01-141; Mayer's hematoxylin/eosin Y. a, Ethmoidal air cell; pseudostratified (possibly ciliated) columan epithelium. Note the numerous glands in the lamina propria; b, Maxillary sinus, stratified columnar epithelium (possibly ciliated).



reported in earlier work in that the paranasal sinus epithelium is, for the most part, a pseudostratified, ciliated cuboidal to columnar epithelium. Because of the problems of obtaining sufficient specimens immediately postmortem and technical problems with undecalcified specimens, it is not possible to state with confidence whether the mastoid air cell epithelium is ciliated as reported by other laboratories. We have observed, however, the presence of squamous epithelium similar to that reported by Sade[6] and Hentzer[7] in mastoid air cells.

We have collected data on two quantities which affect risk, tissue thickness, and the population of target nuclei. Tissue thickness is important because the alpha particles must penetrate the tissue before they can strike the target nuclei. A thick tissue layer shields the nuclei from the alpha particles. The population of target nuclei is important in assessing relative risk. Areas which have few target nuclei are potentially at lower risk than areas which have many.

We envision four possible sources of alpha particles for irradiation of the epithelial cells: (1) radium and its daughter products contained in bone, (2) radon in the sinus and air cell cavities, (3) radon daughter products on the mucosal surfaces, and (4) radon dissolved in the mucosa and its decay products. Thus, the epithelial cells could be irradiated from beneath or above, and with this in mind, we have collected data on the thicknesses of both the lamina propria and the epithelium, since both thicknesses would enter into dosimetric calculations.

The population of target nuclei is expressed as the percentage of the epithelium occupied by nuclei. To determine this we select a portion of the epithelium for study and measure its area as seen on a photographic enlargement of the section. Then we determine the average area per nucleus by measuring the areas of ten nuclei in the study region. We count the total number of nuclei in the study region and multiply by the area per nucleus to obtain the total area of nuclei. The population of target nuclei is finally expressed as

$$\frac{\text{Area of nuclei}}{\text{Area of epithelium}} \times 100 .$$

Results of our measurements are presented in Tables 2 and 3. At all of the sites but one, two or more regions of the mucosa were studied, and the data for each are listed separately. The ranges of radium series alpha particles in water lie between about 30 and 70 μm, and this serves as a standard of comparison for the thickness data reported. Of the twenty values reported for the thickness of epithelium, 8 are less than 30 μm and 18 are less than 70 μm. If we assume that these dimensions are only 70% of normal due to tissue shrinkage and correct them upward by a factor of 1.42, 4 of the corrected values are less than 30 μm and 16 of them are less than 70 μm. This implies that many alpha particles entering the epithelium through its surface will stop before they completely penetrate the epithelium and that a substantial fraction of the epithelial cell nuclei will be outside the ranges of these alpha particles. This suggests that the dose to target nuclei from alphas which enter through the surface will be a sensitive function of the thickness of the epithelium and will vary strongly as a function of depth beneath the surface. It further points to the need to determine the number of target nuclei as a function of depth beneath the surface, so that dosimetric calculations can be made properly, rather than the total number of target nuclei, as has been done for this report.

Nineteen values are reported for the thickness of the lamina propria. Of these, 4 are less than 30 μm and 7 are less than 70 μm. If a correction is made for tissue shrinkage by multiplying by 1.42, 2 of the corrected values are less than 30 μm and 6 of the corrected values are less than 70 μm. Thus, in most cases, the lamina propria is so thick that no alpha particles emitted from the bone will enter the epithelium. In some cases, it is thin enough so that alpha particles from all of the radium series nuclides will enter the epithelial layer. In some regions, then, the underlying bone could make a significant contribution to the epithelial dose, and the bone cannot be ignored as a source of dosage. Furthermore, the thickness of the lamina propria is so variable that it is very likely that all of the paranasal sinuses contain regions in which the epithelial cells lie within range of the bone surface. As in the case of the dose from alpha particles entering through the epithelial layer surface, determination of the number of target nuclei as a function of distance above the

Table 2. Measurements of Paranasal Sinus and Mastoid Air Cell Epithelium in Normal Postmortem Specimens from Two CHR Cases

Case	Site	Sample No.	Thickness of epithelium, μm[a]	Thickness of lamina propria, μm[a]
01-141, 92-year-old male, 0.017 μCi ^{226}Ra	R. frontal sinus	1	37.6 ± 8.1	38.6 ± 4.8
		2	43.5 ± 0.8	36.6 ± 2.4
	L. frontal sinus	1	11.0 ± 1.5	13.6 ± 1.8
		2	12.4 ± 1.7	26.1 ± 3.2
	R. mastoid air cells	1	45.3 ± 8.5	none
	Sphenoidal sinus	1	29.8 ± 2.8	341 ± 77
		2	34.2 ± 2.9	224 ± 28
	Ethmoidal air cells	1	24.8 ± 2.0	112 ± 9
		2	29.1 ± 1.4	170 ± 2
	L. maxillary sinus	1	17.2 ± 2.8	29.2 ± 2.9
		2	15.4 ± 1.5	61.6 ± 2.8
05-044, 80-year-old male, 0.002 μCi ^{226}Ra	Frontal sinus	1	54.7 ± 7.5	307 ± 20
		2	64.6 ± 5.0	not measured
	Sphenoidal sinus	1	29.9 ± 4.0	140 ± 18
		2	37.5 ± 2.0	155 ± 2
		3	46.8 ± 4.4	140 ± 5
		4	34.0 ± 6.2	133 ± 4
		5	36.4 ± 3.2	111 ± 9
	R. maxillary sinus	1	136 ± 17	541 ± 68
		2	133 ± 21	528 ± 40

[a]Based on 10 measurements at different locations on photographic enlargement of slide. Average ± standard deviation.

Table 3. Data on the Population of Target Nuclei in the Paranasal Sinus and Mastoid Air Cell Epithelium in Normal Postmortem Specimens from Two CHR Cases

Case	Site	Sample	Area[a] of epithelium studied, μm^2	Average area[b] of cell nucleus, μm^2	Number of nuclei in area studied	Total area of nuclei, μm^2	$\frac{\text{Area of nuclei}}{\text{Area of epithelium}} \times 100$
01-141	R. frontal sinus	1	6830	16.8 ± 4.1	54	907	13.3
		2	3830	14.0 ± 4.7	33	462	12.1
	L. frontal sinus	1	1780	13.3 ± 5.5	37	492	27.6
		2	2540	13.9 ± 4.3	62	862	33.9
	R. mastoid air cells	1	35100	65.4 ± 38.3	146	9500	27.1
	Sphencidal sinus	1	4410	41.3 ± 15.8	23	950	21.5
		2	9600	36.5 ± 8.5	100	3650	38.0
	Ethmoidal air cells	1	4430	20.7 ± 5.9	69	1430	32.3
		2	5500	17.0 ± 4.7	89	1510	27.5
	L. maxillary sinus	1	3850	15.7 ± 4.9	70	1100	28.6
		2	2520	18.7 ± 5.1	39	730	29.0
05-044	Frontal sinus	1	15500	39.3 ± 13.1	82	3220	20.8
		2	6880	44.8 ± 10.0	42	1880	27.3
	Sphenoidal sinus	1	4960	30.2 ± 16.1	24	725	14.6
		2	5690	30.6 ± 3.7	18	551	9.7
		3	5650	46.3 ± 11.2	24	1110	19.6
		4	6000	36.2 ± 12.3	32	1160	19.3

[a] Measured on photograph using planimeter; 3 repetitions

[b] Based on planimeter measurements of 10 photographically enlarged nuclei; 5 repetitions. Average ± standard deviation.

interface between the epithelial layer and the lamina propria is essential for proper dosimetric calculations to be carried out.

According to Table 3, nuclei constitute from 9.7% to 38% of the epithelial layer in the regions studied. Regions with high nuclear content are expected to be more radiosensitive than regions with low nuclear content since an alpha particle entering the former region has a greater chance of striking nuclear material than an alpha particle entering the latter region. As data accumulate we expect to be able to determine whether or not the greater number of head carcinomas in the mastoid air cells compared to the paranasal sinuses is due, in part, to a greater population of nuclei in the mastoid.[1] This will require a determination of the total amount of epithelium in the sinuses and mastoid air cells so that the total amount of nuclear material in each can be determined from percentages such as those given in Table 3.

To date we have given little consideration to glands in the lamina propria, principally because they have been absent from most of the sections we have studied. A second reason is that the epithelial cells of glands are more highly differentiated than those of epithelial lining membranes and, therefore, should have less proliferative potential and be less at risk of malignant expression. Indeed, when new cells are needed for exocrine glands they are obtained by division and differentiation of the epithelial lining of the ducts. From a dosimetric standpoint the cells of glands are closer to the bone surface and farther from the epithelial surface than are the cells of the lining membrane so that alpha particles entering the membrane surface should be less a factor and alpha particles from the bone surface should be more a factor in their dosimetry. Thus, the doses received by these cells will most likely be different from the doses received by the cells of the membrane. At present we plan to include gland cells in future studies by determining the frequency with which they appear in our sections relative to the frequency with which membrane lining cells appear. Should they be more highly abundant than we now believe, we would expand our study to include them on an equal basis with the membrane lining cells.

We have observed, in addition, that the epithelium is apparently totally lacking in a significant portion of the mastoid air cells in case 01-141. This may be due in part to the delay in postmortem fixation and subsequent sloughing of the mucosal cells or to an undefined artifact of tissue preparation; or perhaps this condition occurs normally in air cell tissues of older or moribund patients.

The observation that epithelial cells from the paranasal sinuses of a particular case are well preserved might lead one to suspect that equal preservation could be expected in the inner labyrinth of the temporal bone. In fact, there are examples of mastoid preparations which indeed do have epithelial linings in portions of the air cells. This observation would tend to support the hypothesis of senile changes.

Another plausible explanation relates to the anatomical structure and physiology of the mastoid air cells. Low turnover of air and/or fluid in these spaces would permit the accumulation of lytic factors postmortem which could denude the air cells of their epithelium. Because of the intricate series of channels in the temporal bone, penetration of fixative could be so slow that sufficient time would be allowed for cell lysis or sloughing to occur.

Under ideal conditions, perfusion of the specimen immediately postmortem with the desired fixative followed by collection of the appropriate sample would assure the best results.

Application to Radioprotection

Until now very little quantitative information has been available on the locations of target cells in the sinus epithelium relative to the sources of alpha particles. The one figure quoted for humans by the ICRP,[12] of 50 to 100 μm for the total thickness of the mucosal layer is consistent with all of our data to within an order of magnitude but has not been useful for dosimetric calculations because it does not tell where the cells lie relative to the sources of radiation. The absence of information may be partly responsible for the failure to make a clear distinction between bone sarcomas and head carcinomas and between their microdosimetry. This failure is clearly evident in the latest

recommendations of the ICRP,[13] where there is no explicit mention of the two types of radium induced cancers and where the distinction between the microdosimetry of endosteal bone cells and epithelial cells is blurred. The statement in ICRP-26, paragraph 47 reads: "The radiosensitive cells in bone have been identified as the endosteal cells and epithelial cells on bone surfaces (see ICRP Publication 11). The Commission recommends that, where possible, dose equivalent in bone should apply to the endosteal cells and cells on bone surfaces, and should be calculated as an average over tissue up to a distance of 10 μm from the relevant bone surfaces." It is clear from our data that the epithelial cells are not on bone surfaces but distant from them, and that taking the epithelial cell dose as the dose averaged over a tissue layer 10 μm thick adjacent to the bone surfaces would grossly overestimate the epithelial cell dose from radionuclide deposits in bone. Although our study is in its early stages, the data we have gathered so far clearly establish that there is a vast difference between the microdosimetry of endosteal bone cells and epithelial cells and this difference should be recognized by the organizations which establish radiation protection guidelines.

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REDUCTION OF PATIENT DOSE DELIVERED BY CHR DIAGNOSTIC X-RAY EXAMINATIONS

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Three changes in technique have been made which reduce the x-ray dose delivered by diagnostic examinations of patients of the Center for Human Radiobiology (CHR): (1) Kodak Lanex Regular screens and Kodak Ortho G film have been substituted for DuPont Cronex Parspeed screens and DuPont Cronex 4 film for five projections in the MIT examinations; (2) 3 mm Al added filtration is now used in place of 1 mm Al added filtration in the ANL examination; (3) improvements in collimation for the ANL examination have been made. Use of the new screen-film combination at MIT has reduced the mean dose to the active marrow of the female RANDO phantom from 606 ± 69 mrad to 235 + 16 mrad; it has reduced the ovary dose from 606 ± 40 mrad to 291 ± 19 mrad and has left the breast dose unchanged at 333 ± 103 mrad. The change from 1 mm Al to 3 mm Al added filtration at ANL, without changes in collimation, would reduce the mean marrow dose in the phantom from 232 ± 14 mrad to 175 ± 26 mrad, reduce the ovary dose from 243 ± 25 mrad to 162 ± 38 mrad and reduce the breast dose from 388 ± 35 mrad to 226 ± 9 mrad. The changes in collimation at ANL should reduce these doses even further but the quantitative effect has not been ascertained.

Introduction

Last year the doses delivered to the marrow, ovaries, and breasts of a female RANDO phantom[1] by the diagnostic x-ray examinations given here and by the Cambridge satellite laboratory (MIT) were reported. The report recommended three changes in x-ray technique to reduce patient dose: (1) use of a faster screen-film combination with the MIT examination; (2) use of 3 mm Al added filtration instead of 1 mm Al added filtration with the ANL examination; (3) improved collimation on exposures of the breast at ANL and possible improved collimation on other exposures.

All three recommendations were followed by the radiologists in charge. The principal purpose of this report is to give estimates of the doses delivered by the MIT examination using a faster screen-film combination and to describe the estimation procedure.

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Before proceeding we shall comment on the other changes. According to dose values reported last year[1] for 1 mm Al and 3 mm Al added filtration at ANL, the change from 1 to 3 mm would result in the following reductions in dose if the collimations were unchanged: (1) mean marrow dose would be reduced from 232 ± 14 mrad to 175 ± 26 mrad; (2) ovary dose would be reduced from 243 ± 25 mrad to 162 ± 38 mrad; (3) breast dose would be reduced from 388 ± 35 mrad to 226 ± 9 mrad. Improvements in collimation should reduce the dose even further, but the quantitative effect of these improvements has not been measured or estimated.

Change of Screens and Films at MIT

In order to reduce patient dose a faster screen-film combination was adopted for five projections of the torso which contribute substantially to the marrow and ovary dose. The older combination, DuPont Cronex Parspeed screen with DuPont Cronex 4 film has been replaced by Eastman Kodak Lanex Regular screens with Eastman Kodak Ortho G film when the AP thoracic spine, lateral thoracic spine, AP lumbar spine, lateral lumbar spine, and AP pelvis exposures are made. This had led to a considerable reduction in patient exposure as outlined in Table 1, where typical technical factors for the Parspeed-Cronex 4 and Lanex-Ortho G combinations are given, along with exposure doses in air at 40" from the tube focus and a reduction factor which equals the ratio of the exposure doses. The technical factors are those which were used most frequently among 23 patients examined shortly before and 23 different patients examined shortly after the changeover. The exposure dose was obtained by multiplying the mAs values shown in the table by measured values of the mR/mAs.

Method of Dose Estimation for Active Marrow

As in our previous work,[1] the body is divided into three anatomical regions, "cranial," "center," and "caudal" in order to observe the nonuniformity of marrow dosage. The cranial region consists of the head and cervical vertebrae. The center region consists of the upper limb girdle, sternum, ribs, and

Table 1. Comparison of technique and exposure dose with the two screen-film combinations

	Typical technique and exposure dose for patients, kVp/mAs/mA/mR		Exposure dose reduction factor
Projection	Parspeed-Cronex 4	Lanex-Ortho G	
Thoracic spine, AP	75/100/100/300	65/30/100/60	0.20
Thoracic spine, lat.	65/500/200/1000	65/50/200/100	0.10
Lumbar spine, AP	75/150/300/480	75/35/100/100	0.21
Lumbar spine, lat.	75/500/200/1600	75/70/200/220	0.14
Pelvis, AP	75/100/200/320	75/25/100/75	0.23

thoracic vertebrae and the caudal region consists of the lumbar vertebrae, sacrum, and lower limb girdle.

Values of the mean marrow dose, $\overline{D_P}$, and the integral dose for each of these regions in the RANDO phantom when the Parspeed-Cronex 4 combination was used for all films were reported last year.[1] To estimate the mean marrow dose, $\overline{D_L}$, and integral dose in each of these regions when the Lanex-Ortho G combination is used for five of the projections, we develop an expression for the dose ratio $\overline{D_L}/\overline{D_P}$.

Let D_i be the mean dose absorbed by active marrow within the useful beam when the i^{th} projection is made using the Parspeed-Cronex 4 combination and let m_i be the mass of active marrow exposed. Then the mean dose $\overline{D_P}(k)$ within the k^{th} anatomical region is given by

$$\overline{D_P}(k) = \frac{\sum D_i m_i}{M_k} \quad , \tag{1}$$

where M_k is the total mass of active marrow in the k^{th} region and the summation extends only over those projections which expose the k^{th} region. When the Lanex-Ortho G combination is used, the analogous dose $\overline{D_L}(k)$ is given by

$$\overline{D_L}(k) = \frac{\sum r_i D_i m_i}{M_k} \quad , \tag{2}$$

where r_i is the exposure dose reduction factor. Thus

$$\frac{\overline{D_L}(k)}{\overline{D_P}(k)} = \frac{\sum r_i D_i m_i}{\sum D_i m_i} \quad . \tag{3}$$

The absorbed dose D_i is unknown but may be replaced by a quantity which is approximately known, the entrance exposure dose, X_i. D_i differs from X_i by factors which account for tissue geometry, penetration, and conversion of units of measurement. Let the combination of these factors be represented by c_i. Then $D_i = c_i X_i$ and

$$\frac{\overline{D_L}(k)}{\overline{D_P}(k)} = \frac{\sum r_i c_i X_i m_i}{\sum c_i X_i m_i} \quad . \tag{4}$$

If we assume that the value of c_i is about the same for all projections, then

$$\frac{\overline{D_L}(k)}{\overline{D_P}(k)} \cong \frac{\sum r_i X_i m_i}{\sum X_i m_i} \quad . \tag{5}$$

To evaluate the numerator and denominator of Eq. 5 , values are needed for r_i, X_i, and m_i. The values of r_i are obtained from Table 1 for the five projections in which the Lanex-Ortho G combination has been substituted for the Parspeed-Cronex combination; otherwise $r_i = 1$. Values of m_i are obtained by examining the x-ray films to determine which bones of the skeleton are exposed by each projection and then using the tables of Ellis,[2] normalized to 1000 g total active marrow to determine the number of grams marrow exposed by each projection. The values are given in Table 2 for the three anatomical regions. For X_i we use the values of exposure dose in air at 40" or 72" (for the PA chest, lateral chest, and lateral cervical spine) determined from values of the mAs for each projection and measured values of mR/mAs. These exposure doses differ from the entrance exposure dose by backscatter factors and inverse square law corrections, but are nearly proportional to the entrance exposure doses. Table 3 gives values

Table 2. Marrow exposed by x-ray projections in the MIT examination of the RANDO phantom

Anatomical region	Projections	Bones exposed[a]	Active marrow[b] exposed, g
Cranial (contains 165 g active marrow)	Skull, AP	Head	131
	Skull, lat.	Head, CV1, CV2, and CV3	146
	Sphenoid, PA	Head	131
	Skull, mod. Waters, PA	Head	131
	Mandible, r., tang.	Mandible and 25% cranium	42
	Mandible, l., tang.	Mandible and 25% cranium	42
	Stenver's view of mastoids, r., tang.	25% cranium, CV1 and CV2	41
	Stenver's view of mastoids, l., tang.	25% cranium, CV1 and CV2	41
	Cervical spine, AP	All CV	34
	Cervical spine, lat.	Mandible and all CV	46
Center (contains 326 g active marrow)	Cervical spine, AP	TV1, TV2, TV3, and TV4	33
	Chest, PA	Upper limb girdle, all ribs, sternum, and all TV	326
	Chest, lat.	Upper limb girdle, all ribs sternum, and all TV	326
	Shoulder, r., AP	Right scapula and head of right humerus	34
	Shoulder, l., AP	Left scapula and head of left humerus	34
	Thoracic spine, AP	Sternum and all TV	164
	Thoracic spine, lat.	Upper limb girdle, all ribs, sternum and all TV	326
	Lumbar spine, AP	Left and right ribs 9, 10, 11, 12, TV 10, TV 11, and TV 12	69
	Lumbar spine, lat.	Left and right ribs 9,10, 11, 12, TV 10, TV 11, and TV 12	69
Caudal (contains 509 g active marrow)	Chest, PA	LV1, LV2, LV3, and LV4	86
	Chest, lat.	LV1, LV2, LV3, and LV4	86
	Lumbar spine, AP	All LV, sacrum and lower limb girdle	509
	Lumbar spine, lat.	All LV and 50% lower limb girdle	240
	Femur, r. & l., AP	Right and left femur diaphysis	0
	Pelvis, AP	LV4, LV5, sacrum and lower limb girdle	446
	Hip, r., lat.	Right half of lower limb girdle	131
	Hip, l., lat.	Left half of lower limb girdle	131

[a]Bone designations are those used by Ellis.[2] CV, TV, and LV are abbreviations for cervical certebrae, thoracic vertebrae, and lumbar vertebrae.

[b]Normalized to 1000 g total active marrow.

Table 3. Technical factors and exposure doses in air at 40" from tube focus during the x-ray examination of the RANDO phantom at MIT

Projection	kVp	mA	mAs	mR/mAs	mR[a]
Skull, AP	83	200	100	4.4	440
Skull, lat.	70	200	100	2.8	280
Sphenoid, PA	88	200	100	5.2	520
Skull, mod. Waters, PA	78	200	100	3.6	360
Mandible, r., tang.	80	300	30	3.3	99
Mandible, l., tang.	80	300	30	3.3	99
Stenver's view of mastoids, r., tang.	78	200	100	3.6	360
Stenver's view of mastoids, l., tang.	78	200	100	3.6	360
Cervical spine, AP	68	200	70	2.5	175
Cervical spine, lat.	78	300	40	0.96	38
Chest, PA	96	300	10	1.5	15
Chest, lat.	116	300	15	2.5	38
Shoulder, r., AP	65	100	35	2.1	74
Shoulder, l., AP	65	100	35	2.1	74
Thoracic spine, AP	80	100	100	3.6	360
Thoracic spine, lat.	60	200	500	1.5	750
Lumbar spine, AP	75	300	90	3.1	279
Lumbar spine, lat.	75	200	500	2.9	1450
Femur, r. & l., AP	60	200	100	1.8	180
Pelvis, AP	80	200	100	3.8	380
Hip, r., lat.	80	200	100	3.8	380
Hip, l., lat.	80	200	100	3.8	380

[a] At 40" from the tube focus except for the lateral cervical spin, PA chest, and lateral chest which are at 72" from the focus. The mR/mAs is a function of kVp, mA, and mAs; hence all three values are quoted.

of the exposure factors used with each projection and of the exposure dose in mR at 40" or at 72".

Marrow Dose Values for the Lanex-Ortho G Combination

Cranial Region

The projections which use the Lanex-Ortho G combination do not expose the cranial region directly. Nevertheless, there should be a reduction of marrow dose due to a reduction of radiation scattered into this region from exposures of the center and caudal regions. The only data we have on this

effect is for the lateral lumbar spine projection which contributes 1 g · rad to the integral dose in the cranial region when the Parspeed-Cronex 4 combination is used.[1] The exposure dose using the Lanex-Ortho G combination for the lateral lumbar spine projection is only 14% of that using the Parspeed-Cronex 4 combination (Table 1), and use of the Lanex-Ortho G combination should also reduce the integral dose contribution to the cranial region from scattered radiation to 0.14 g · rad. If this reduction is taken into account, the integral dose of 117 ± 11 g · rad reported for the cranial region last year becomes 116 ± 11 g · rad, and the mean marrow dose to the cranial region is reduced from 709 ± 64 mrad to 703 ± 64 mrad. Additional slight reductions in dose are expected to result from reductions in radiation scattered into the cranial region by the AP thoracic spine, lateral thoracic spine, AP lumbar spine, and AP pelvis projections. The combined reduction from these four projections is expected to be about the same as the reduction from the lateral lumbar spine projection since the combined exposure dose from the four views is 1769 mR (Table 3) compared with 1450 mR for the lateral lumbar spine.

Center Region

Table 4 deals with the center region. The new screen and film combination reduces the integral dose and the mean dose absorbed by the active marrow by a factor of 0.183 or to about 18% of the dose with the Parspeed-Cronex 4 combination. These values had been 212 ± 22 g · rad and 650 ± 69 mrad.[1] The new values for the center region are 38.8 ± 4.1 g · rad and 119 ± 13 mrad as shown at the bottom of Table 4.

Caudal Region

Table 5 deals with the caudal region. The new screen and film combination reduces the integral dose and the mean dose absorbed by active marrow by a factor of 0.290 or to 29% of the dose with the Parspeed-Cronex 4 system. These values had been 278 ± 37 g · rad and 545 ± 73 mrad. Nearly one-half of the new dose to the caudal region is from the right and left lateral hip views, for which the Parspeed-Cronex 4 combination is used for better resolution. The new values for the caudal region are 80.6 ± 19.7 g · rad and 158 ± 21 mrad.

Table 4. RANDO center region: Integral dose and dose when Lanex-Ortho G combination is used for AP and lateral thoracic and lumbar spine films

Projection	Marrow mass (m_i), g	Parspeed-Cronex 4 Exposure dose (X_i), mR	Parspeed-Cronex 4 $X_i m_i$ kg · mR	Lanex-Ortho G Reduction factor (r_i)	Lanex-Ortho G $r_i X_i m_i$ kg · mR
Cervical spine, AP[a]	33	175	5.8	1	5.8
Chest, PA	326	15	4.9	1	4.9
Chest, lat.	326	38	12.4	1	12.4
Shoulder, r., AP	34	74	2.5	1	2.5
Shoulder, l., AP	34	74	2.5	1	2.5
Thoracic spine, AP	164	360	59.0	0.20	11.8
Thoracic spine, lat.	326	760	344.5	0.10	34.5
Lumbar spine, AP[b]	69	279	19.3	0.21	4.1
Lumbar spine, lat.[b]	69	1450	100.1	0.14	14.0
			$\sum X_i m_i = 451.0$		$\sum r_i X_i m_i = 82.5$

$$\frac{\overline{D_L}}{\overline{D_P}} = \frac{82.5}{451.0} = 0.183$$

Integral dose with Lanex-Ortho G combinationL $0.183 \times (212 \pm 22) = 38.8 \pm 4.1$ g · rad

Mean dose with Lanex-Ortho G combination: $0.183 \times (650 \pm 69) = 119 \pm 13$ mrad

[a]Overlap of thoracic vertebrae 1–4 into center region from film taken in cranial region.

[b]Overlap of l. and r. ribs 9–12 and thoracic vertebrae 10–12 into the center region from films taken in the caudal region.

Total Marrow

Data from Tables 2, 4, and 5 are drawn together in Table 6 where integral dose and mean absorbed dose values are presented for the three anatomical regions for the total marrow. The mean dose value when the Lanex-Ortho G combination is used is 235 ± 16 mrad in comparison with 606 ± 69 mrad when the Parspeed-Cronex 4 combination is used for all films. Thus the substitution of the Lanex-Ortho G combination for five projections effects a 61% reduction in total dose.

Ovary and Breast Doses for the Lanex-Ortho G Combination

The ovaries are exposed by four projections: the AP thoracic spine and the AP pelvis which expose both ovaries, and the lateral left hip and the

Table 5. RANDO caudal region: Integral dose and dose when Lanex-Ortho G combination is used for AP pelvis and for AP and lateral thoracic and lumbar spine films

Projection	Marrow mass (m_i), g	Parspeed-Cronex 4		Lanex-Ortho G	
		Exposure dose (X_i), mR	$X_i m_i$, kg · mR	Reduction factor (r_i)	$r_i X_i m_i$ kg · mR
Lumbar spine, AP	509	279	142.0	0.21	29.8
Lumbar spine, lat.	240	1450	348.0	0.14	48.7
Pelvis, AP	446	380	169.5	0.23	39.0
Femur, r. & l., AP	0	180	0	1	0
Hip, r., lat.	131	380	49.8	1	49.8
Hip, l., lat.	131	380	49.8	1	49.8
Chest, PA[a]	87	15	1.3	1	1.3
Chest, lat.[a]	87	38	3.3	1	3.3
			$\sum X_i m_i = 763.7$		$\sum r_i X_i m_i = 221.7$

$$\frac{\overline{D_L}}{\overline{D_P}} = \frac{221.7}{763.7} = 0.290$$

Integral dose with Lanex-Ortho G combination: $0.290 \times (278 \pm 37) = 80.6 \pm 10.7$ g · rad

Mean dose with Lanex-Ortho G combination: $0.290 \times (545 \pm 73) = 158 \pm 21$ mrad

[a]Overlap of lumbar vertebrae 1–4 into the caudal region from chest films taken in the center region.

Table 6. Integral dose and mean absorbed dose to RANDO active marrow when Lanex-Ortho G combination is used for AP pelvis and for AP and lateral thoracic and lumbar spine films, and Parspeed-Cronex 4 combination for all other films

Region	Marrow mass, (M_k), g	Parspeed-Cronex 4		Lanex-Ortho G	
		Integral dose, g · rad	Absorbed dose, mrad	Integral dose, g · rad	Absorbed dose, mrad
Cranial	165	117 ± 11	709 ± 64	116 ± 11	703 ± 64
Center	326	212 ± 22	650 ± 69	3.8 ± 4.1	119 ± 13
Caudal	509	278 ± 37	545 ± 73	80.6 ± 10.7	158 ± 21
Total marrow	1000	606 ± 69	606 ± 69	235 ± 16	235 ± 16

lateral right hip, each of which exposes one ovary. Equation 5 may be applied to this exposure situation as well if m_i is assumed to be the mass of ovary tissue exposed, and if $\overline{D_L}$ and $\overline{D_P}$ are interpreted as mean ovary doses rather than mean marrow doses. Then,

$$\frac{\overline{D_L}}{\overline{D_P}} = \frac{(0.20)(360)m + (0.23)(380)m + (1)(380)(m/2) + (1)(380)(m/2)}{360m + 380m + 380(m/2) + 380(m/2)}$$

$$= 0.48 , \qquad (6)$$

where m is the mass of both ovaries. Therefore,

$$\overline{D_L} = 0.48 \times (606 \pm 40) = 291 \pm 19 \text{ mrad} .$$

The breasts are not directly exposed by any of the projections which use the Lanex-Ortho G combination. Therefore, the breast dose should remain the same as previously reported,[1] 333 ± 103 mrad, except for small dose reductions due to reduction of scattered radiation.

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ANALYSIS OF THICK SOURCE ALPHA PARTICLE SPECTRUM FROM RADIUM AND ITS DAUGHTERS IN BONE

L. F. Mausner and R. A. Schlenker

The alpha particle energy spectrum of ^{226}Ra and its four alpha emitting daughters in an ashed, ground bone sample has been resolved into its components using a computerized spectrum stripping algorithm. These calculated results have been compared to direct measurements of the ^{226}Ra and ^{214}Po distributions obtained by alpha-gamma coincidence techniques. The ability of the calculation to deconvolute the total spectrum into its five alpha components implies that straightforward alpha counting may be used instead of the very low efficiency ^{226}Ra alpha-gamma coincidence method. From knowledge of the actual ^{226}Ra distribution, along with suitable detector energy and efficiency calibrations, one could determine endosteal cell dose rate empirically.

Introduction

It is now believed that the cells at risk for the induction of bone cancer are those on endosteal bone surfaces. The alpha particles which irradiate these cells are emitted from a surface layer of bone one alpha particle range thick. At present, calculations of absorbed dose to the endosteal cells are based on measurements of the skeletal radium content and the assumption that radium is uniformly distributed in bone right up to the surface. Autoradiographic techniques generally do not offer sufficient spatial resolution for measurement of the radium concentration in bone surface layer so we are developing a method based on thick source alpha particle spectrometry, which will measure radionuclide concentrations to a depth of one alpha particle range. We use silicon surface barrier detectors to measure directly the energies of alpha particles escaping from bone surfaces. Using the measured energies and the known initial energies of the alpha particles, we can determine the energy lost by the alphas as they travel through bone and translate this into the concentration as a function of depth using a range-energy relationship.

Figure 1 displays the alpha particle energy spectrum of ^{226}Ra and its daughters in a thick (compared to a typical alpha range of 40 μm) sample of ground and ashed bone. The process of grinding the original intact sample thoroughly mixes surface with interior bone and guarantees a uniform distribution

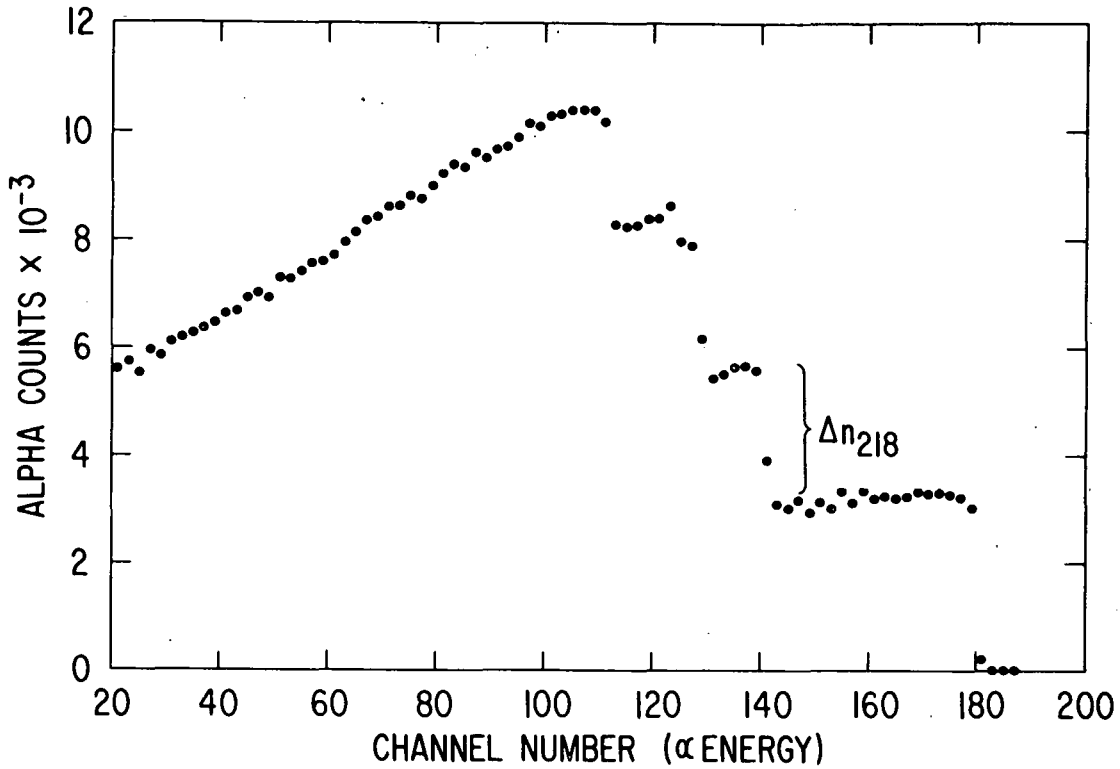

FIG. 1.--Alpha spectrum of ^{226}Ra in ground, ashed bone (every other point plotted). The discontinuity at the maximum energy of alpha particles emitted by ^{218}Po is indicated as Δn_{218}. (ANL Neg. 149-78-296)

of radionuclides. The spectrum consists of portions which increase linearly with energy and end in step-like discontinuities. This shape is characteristic of uniformly distributed sources, and deviations from it imply a nonuniform radionuclide distribution. For example, if the spectrum shows a sharp non-linear increase (reminiscent of a spectral peak from a thin alpha particle source) just to the low energy side of a discontinuity, the concentration of one of the radionuclides is much greater just below the surface than it is deeper in the source. Or, if the spectrum slopes off gradually where a discontinuity is expected, one of the radionuclides is less heavily concentrated near the source surface than it is deeper down. Both these features are present in the spectrum of alpha particles from a sample of intact skull bone (Figure 2). Thus uniformity or nonuniformity of the distribution can be decided by inspection of the spectrum shape, but a quantitative determination of the distribution requires a more elaborate analysis.

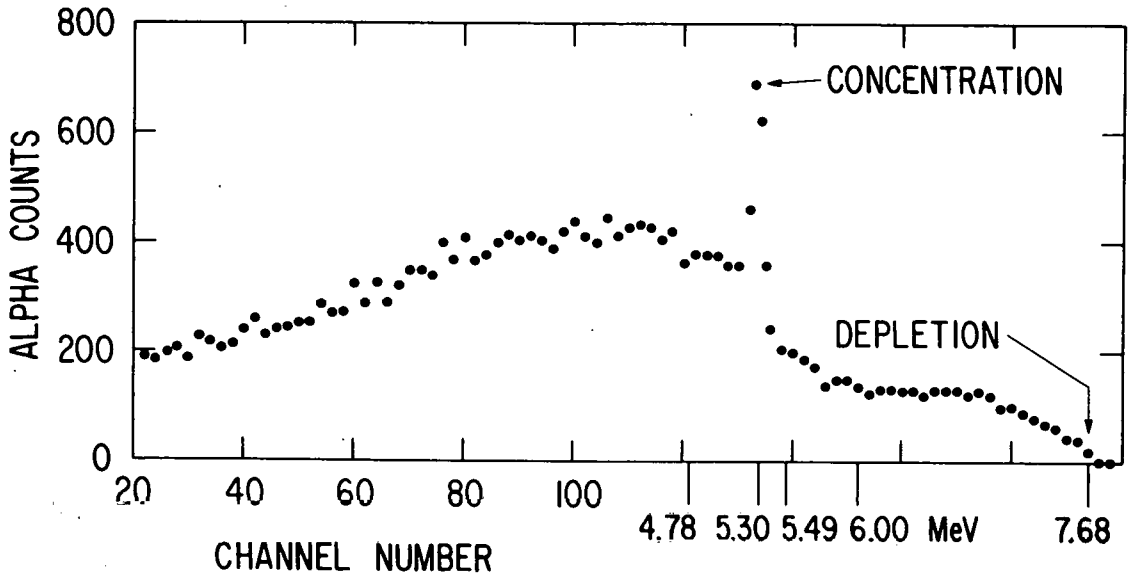

FIG. 2.--Spectrum showing both surface concentration and surface depletion features in a sample of intact skull bone (every other point plotted). (ANL Neg. 149-78-298)

The energy spectrum in Figure 1 is the sum of five overlapping components each of which is produced by one of the five alpha emitters, ^{226}Ra, ^{222}Rn, ^{218}Po, ^{214}Po, and ^{210}Po, in the radium decay series. Each component begins at zero energy and ends at one of the five discontinuities. In order to obtain the distribution of each radionuclide, its component must be separated from the total spectrum and analyzed using range-energy and stopping power relationships. We shall describe below a method to perform this separation with the aid of a computer.

Two of the spectrum components, those corresponding to ^{226}Ra and to ^{214}Po (RaC'), can be measured separately by alpha-gamma coincidence techniques. This involves electronically vetoing all alpha counts except those in coincidence with a single gamma ray which is associated with the radionuclide of interest. However, the counting efficiency is the product of the alpha and gamma efficiencies, leading to much lower coincidence count rates than alpha count rates and often to impractically long counting periods for the low levels of activity in our bone samples. Further, the equipment necessary is significantly more costly than straightforward alpha counting with surface barrier

detectors. This severely limits the number of different samples that can be run concurrently. We have performed these coincidence measurements to compare with the computer analysis of the composite alpha spectrum. Besides providing a test of the accuracy of computer analyses, coincidence techniques are useful when the composite spectrum can not be analyzed easily by computer.

In this report we limit our attention to computer and coincidence method analyses for the uniformly distributed powdered bone source. Although radium in most natural bone samples is nonuniformly distributed, it has proved easier to develop computer analysis techniques first for the uniformly distributed source, and then to modify them for nonuniformly distributed natural bone than to develop the techniques for nonuniform sources directly.

Experimental Methods

A section of humerus from case 00-017 was ashed for 24 hr at 600°C and ground to powder with a mortar and pestle. A layer of powder was spread in a small brass cup to form a source thicker than the alpha range of any of the nuclides in the radium decay series. As little powder was used as practical to minimize gamma background from radium daughter products. The brass cup was then placed in a small vacuum chamber 2.6 cm from the face of an uncollimated 900 mm^2 silicon surface barrier detector. This whole assembly fit into the $2\frac{1}{4}$ in × $2\frac{1}{4}$ in well of a 5 in × 4 in NaI well crystal. The high gamma background in this large detector was reduced by a factor of 30 by surrounding it with two inches of lead shielding. Alpha spectra were recorded on a multichannel analyzer whose energy calibration was made using thin ^{228}Th and ^{241}Am sources.

The composite alpha spectrum, shown in Figure 1, was measured without the use of coincidence circuitry. The typical count rate for the ashed bone sample in the geometry described above was 130 cpm.

The ^{226}Ra component was obtained directly by counting only alphas which are in coincidence with the 186 keV gamma ray that follows the radium decay 3.3% of the time. The data were collected using a standard coincidence system which yielded a low count rate (about 0.8 cpm for the ashed bone source) because of the small gamma-ray branch.

A modification of the coincidence system was required in order to measure the ^{214}Po component. Here, the gamma ray (609 keV, 47%) precedes the alpha decay of the ^{214}Po ground state which has a half-life of 164 μs. The coincidence system used to measure the ^{226}Ra component has a resolving time much shorter than this and would yield a count rate much lower than that obtained for ^{226}Ra. This difficulty was overcome through the use of a time-to-amplitude converter, which serves the function of a coincidence circuit with very long resolving time (in this case 200 μs). With the modified system, we record 57% of the ^{214}Po alpha particles which strike the detector and veto the alphas from the other emitters which are not time correlated with the 609 keV gamma ray. The resolving time of 200 μs was chosen as a compromise between maximizing the ^{214}Po count rate, which exponentially approaches an asymptotic value as the resolving time is increased, and minimizing the accidental count rate, which increases linearly without limit as the resolving time increases. The actual coincidence count rate was 3 cpm for ^{214}Po in the ashed bone sample.

Theoretical Analysis

The ultimate goal of the data analysis is to determine the distribution of each radionuclide as a function of depth beneath the surface of the source. This is a two-step process in which the five components are first separated from the composite energy spectrum and second, are translated into a spatial distribution by using a stopping power relationship. In general, this second step requires that we measure only alpha particles emitted perpendicularly to the source surface. However, collimating the detector and increasing the source-detector distance to meet this condition significantly reduces the count rate. Since, in this case, the source was known to be uniform, collimation was unnecessary and step two was not performed. The more general procedure for collimated conditions is outlined below. Following that is a detailed description of the deconvolution method and the assumptions on which it is based.

Briefly, a spectrum such as shown in Figure 1 is the sum of the five components shown schematically in Figure 3. Each component is the differential energy spectrum, $dn/d\epsilon$, of one of the radionuclides. When we have resolved the components from one another, each may be multiplied by the stopping power, $d\epsilon/d\rho$, to obtain the differential range spectrum

$$\frac{dn}{d\rho} = \left(\frac{dn}{d\epsilon}\right)\left(\frac{d\epsilon}{d\rho}\right) \quad . \tag{1}$$

The quantity $(dn/d\rho)d\rho$ is the number of detected alpha particles which have a residual range ρ (in the interval $d\rho$) in the source material. A particle with residual range ρ has traveled a distance $R-\rho$ in the source, where R is the particle range. Thus, when the detector is well collimated, $(dn/d\rho)d\rho$ gives the number of detected particles which have traveled a distance $R-\rho$ (in the interval $d\rho$) in the source material. This is the distribution of the radionuclide

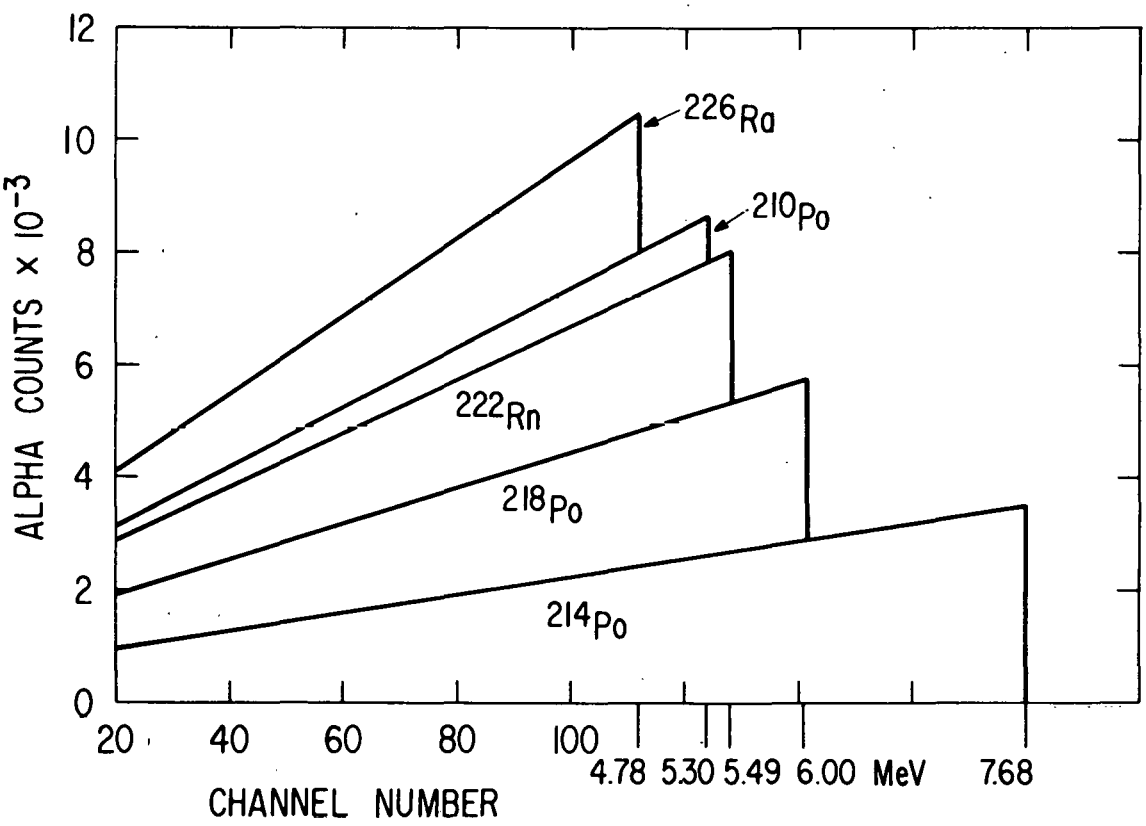

FIG. 3.--Schematic spectrum depicting the separate components in Figure 1 and their sum. (ANL Neg. 149-78-297)

as a function of depth, R-ρ, beneath the bone surface and is the quantity we seek. Additionally, by determining the efficiency calibration for the alpha particle source-detector geometry, we could determine the absolute concentration of the radionuclide as a function of depth, and we could, therefore, make a calculation of the dose rate at the source surface based purely on experimental information. Lacking measurements of the geometrical efficiency, dose-rate calculations require that the concentration be assumed. We plan to develop the appropriate calibration methods in the future.

Resolution of the total spectrum into its components is impossible without knowing beforehand how the distributions of radionuclides differ. This problem is solved by assuming that they are directly proportional to one another. There is no a priori reason for this to be true, and in fact, it cannot be precisely true because the nuclear recoil which accompanies alpha decay causes a slight shift in the location of daughter nuclides relative to their parents and thus introduces a slight nonproportionality, which is undetectable by our methods. Redistribution of the alpha emitters relative to one another could also be caused by diffusion of any of the radium daughter products. However, close consideration of the chemical and decay properties of this series suggests that this may not be a serious complication.

In particular, radium decays to gaseous radon (t= 3.82 days), some of which is retained in bone. The retention is thought to occur when radon atoms recoiling from alpha decay become firmly lodged in bone mineral crystals. Thus, we assume that this ^{222}Rn fraction is retained within a recoil range of its site of production and that the spatial concentrations of ^{226}Ra and retained ^{222}Rn are proportional to one another.

Following ^{222}Rn in the chain are ^{218}Po, ^{214}Pb, ^{214}Bi, and ^{214}Po with half lives of 3.05 min, 26.8 min, 19.7 min, and 164 μs, respectively. These are heavy metal atoms that one expects to be biologically immobile, certainly on the time scale set by their short lifetimes. Thus, we assume no migration and assume that the concentrations of retained ^{222}Rn, ^{218}Po, ^{214}Pb, ^{214}Bi, and ^{214}Po are all directly proportional to one another.

Next in the decay series are ^{210}Pb, ^{210}Bi, and ^{210}Po, with half lives of 22.4 years, 5.01 days, and 138.4 days, respectively. Translocation of ^{210}Pb appears to have ample time to occur. Nevertheless, Holtzman[1] has found that in 44 bone samples taken from 13 persons, the ratios of ^{210}Pb specific activities to those of ^{226}Ra were fairly uniform. It was concluded that little ^{210}Pb migrated or that ^{226}Ra and ^{210}Pb were metabolized similarly. In either case, we can assume that the concentration of ^{210}Po is proportional to that of radium.

With the assumption of proportionality as an aid, we can proceed with separating the components from the total spectrum. From Figure 3, it is clear that in the energy region 6.00 to 7.68 MeV only the ^{214}Po component contributes to the spectrum. Therefore, this portion of the total spectrum can be used as a model to strip out the separate contributions of the other alpha emitters. In general, we want to transform the energy spectrum of one component, $(dn/d\epsilon)_i$, which we take as a model, into the energy spectrum of a second component, $(dn/d\epsilon)_j$, and then subtract this second component from the composite spectrum. Recalling the proportionality assumptions we made above we have

$$\left(\frac{dn}{d\rho}\right)_i d\rho = C_{ij}\left(\frac{dn}{d\rho}\right)_j d\rho \quad , \tag{2}$$

where C_{ij} is the proportionality constant and the distributions are evaluated for the same path length in bone $R_i - \rho_i = R_j - \rho_j$. Applying Eq. 1, we have

$$\left(\frac{dn}{d\epsilon}\right)_i = \frac{(dn/d\rho)_i}{(d\epsilon/d\rho)_i} \quad \text{and} \quad \left(\frac{dn}{d\epsilon}\right)_j = \frac{(dn/d\rho)_j}{(d\epsilon/d\rho)_j} \quad . \tag{3}$$

We can relate these expressions through Eq. 2. Thus,

$$\left(\frac{dn}{d\epsilon}\right)_j = \frac{1}{C_{ij}} \quad \frac{\left(\frac{dn}{d\epsilon}\right)_i \left(\frac{d\epsilon}{d\rho}\right)_i}{\left(\frac{d\epsilon}{d\rho}\right)_j} \quad . \tag{4}$$

The quantities $(dn/d\epsilon)_i$ and $(d\epsilon/d\rho)_i$ are evaluated at $R_i - \rho_i$ or at the corresponding

alpha energy ϵ_i and the quantities $(dn/d\epsilon)_j$ and $(d\epsilon/d\rho)_j$ are evaluated at $R_j - \rho_j$ or at the corresponding alpha energy ϵ_j. The energies and ranges are related to one another through the equation $R_i - \rho_i = R_j - \rho_j$.

The stopping powers have been obtained using the simple empirical approximation

$$\frac{d\epsilon}{d\rho} = \frac{1}{K_1 \epsilon + K_2} \tag{5}$$

developed by Kolenkow and Manly[2] for alpha particles in bone. K_1 and K_2 are constants specific to bone. This relation is valid between 2 and 9 MeV and is adequate for our purposes. Integrating the inverse of Eq. 5 yields a simple range-energy relationship

$$(R - \rho) = \frac{K_1}{2} (E^2 - \epsilon^2) + K_2 (E - \epsilon) , \tag{6}$$

where E is the initial energy of the alpha particle. Equation 6 may be used to find the relationship between ϵ_i and ϵ_j.

Initial estimates of the proportionality constants C_{ij} are calculated from decay systematics, ^{222}Rn retention information and estimates of the retention of ^{210}Po in bone, assuming that ^{222}Rn and ^{226}Ra are retained according to the Norris function. These estimates may well be quite different from the final values, since ^{222}Rn retention in microscopic volumes of individual bone samples does not necessarily obey the Norris function.

Operationally, we first strip out the ^{218}Po component between 5.49 MeV and 6.00 MeV, using ^{214}Po between 6.00 MeV and 7.68 MeV as a model. We begin by smoothing out statistical fluctuations of the data by the method of cubic splines. Then starting at the highest integral channel number equivalent to $\epsilon_{218} \leq 6.00$ MeV, we find the corresponding depth in bone from Eq. 6. Turning Eq. 6 around, we solve for ϵ_{214} at this depth. We evaluate $(dn/d\epsilon)_{214}$ and $(d\epsilon/d\rho)_{214}$ at ϵ_{214}, and evaluate $(d\epsilon/d\rho)_{218}$ at ϵ_{218}. Then $(dn/d\epsilon)_{218}$ is determined from Eq. 4. This procedure is repeated for all data channels between 5.49 and 6.00 MeV, and $(dn/d\epsilon)_{218}$ is subtracted point by point from the composite spectrum. The remainder is that portion of $(dn/d\epsilon)_{214}$ which

overlapped the initial part of $(dn/d\epsilon)_{218}$. The calculated residual of $(dn/d\epsilon)_{214}$ should be continuous with the known section between 6.00 MeV and 7.68 MeV. The proportionality constant $C_{214-218}$ can be adjusted if necessary to achieve continuity. This entire procedure is then repeated at lower energies for all the alpha emitters.

Results

Shown in Figure 4 are the stripped components underlying the relevant section of the composite spectrum. It is, of course, necessary to check this deconvolution procedure quantitatively. A parameter which can give a meaningful comparison between theory and this empirical fit is the ratio $\Delta n_i/\Delta n_{226}$, where Δn_i is the size of the discontinuity of the i^{th} component of the spectrum (see Figure 1). In terms of spectrum parameters

$$\Delta n_i = \left(\frac{dn}{d\epsilon}\,\Delta\epsilon\right)_{\epsilon = E_i} , \tag{7}$$

where $\Delta\epsilon$ is the channel width i.e., the energy difference between adjacent channels in the multichannel analyzer. Using Eqs. 1 and 5 we get

$$\frac{dn}{d\epsilon} = \left(\frac{dn}{d\rho}\right)(K_1\epsilon + K_2) . \tag{8}$$

Since the radionuclides are uniformly distributed throughout the source, $(dn/d\rho) = C$, a constant. Thus,

$$\frac{dn}{d\epsilon} = C(K_1\epsilon + K_2) . \tag{9}$$

Integrating over energy,

$$\int_0^{E_i} \frac{dn}{d\epsilon}\,d\epsilon = C\int_0^{E_i} (K_1\epsilon + K_2)\,d\epsilon$$

$$n(E_i) = C\left(\frac{K_1}{2}E_i^{\,2} + K_2E_i\right) , \tag{10}$$

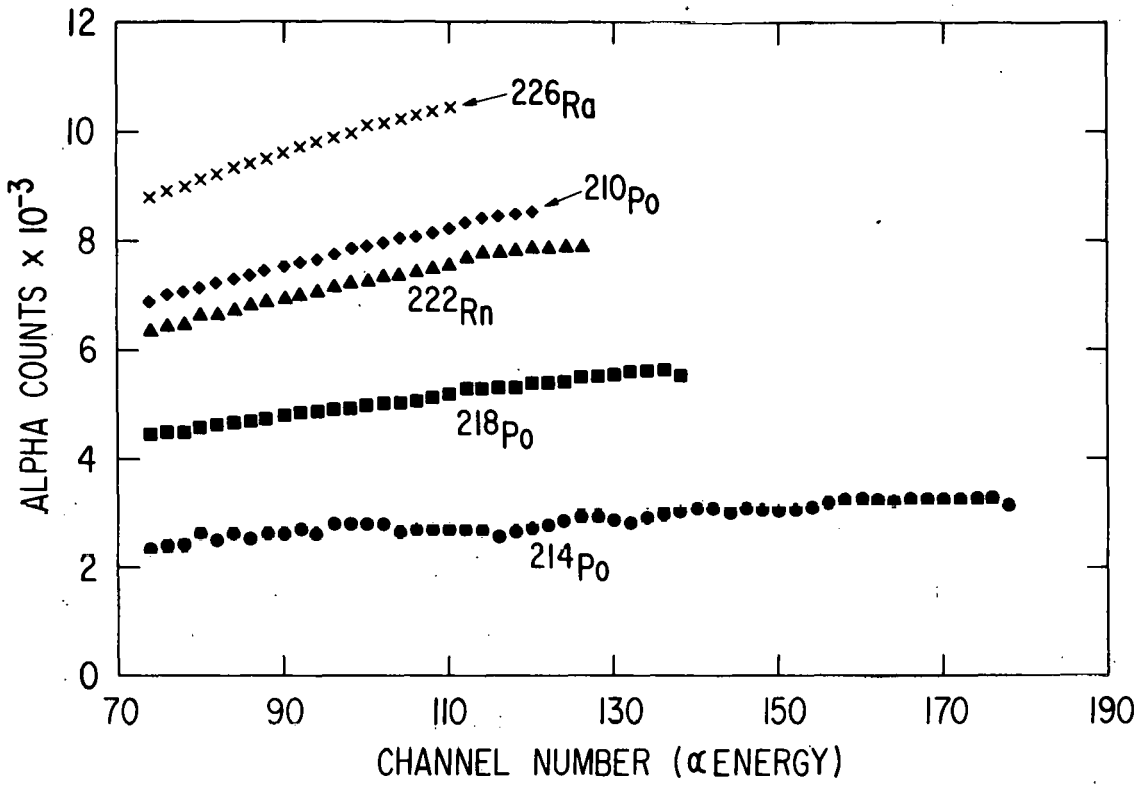

FIG. 4.--The composite spectrum and the components determined by the methods described in the text. For each component, the number of counts is the difference between the points plotted above and below the symbol for the nuclide. Note that components are shown only to channel 70 rather than to channel 20, which is the beginning of the spectrum of Figure 1. (ANL Neg. 149-78-295)

where $n(E_i)$ represents the total number of counts from component i. We can solve for C and then obtain

$$\left(\frac{dn}{d\epsilon}\right)_i = \frac{n(E_i)\,[K_1\epsilon + K_2]}{\frac{K_1}{2}E_i^{\,2} + K_2E_i} \quad . \tag{11}$$

The ratio $\Delta n_i/\Delta n_{226}$ is then given by

$$\frac{\Delta n_i}{\Delta n_{226}} = \frac{n(E_i)}{n(E_{226})} \left\{ \frac{[K_1E_i + K_2]/\left[\frac{K_1}{2}E_i^{\,2} + K_2E_i\right]}{[K_1E_{226} + K_2]/\left[\frac{K_1}{2}E_{226}^{\,2} + K_2E_{226}\right]} \right\} \quad . \tag{12}$$

Let us now consider the quantity $n(E_i)$. It is proportional to the integral of $dn/d\rho$, evaluated over the source volume and integrated over the solid angle subtended by the detector. For a standard coordinate system centered on the bone surface, it is straightforward to express $n(E_i)$ as:

$$n(E_i) = \int N dA \int d\rho_i \int_{\theta} \int_{\phi} G d\theta d\phi \quad . \tag{13}$$

N is the number of particles emitted from a unit of source volume at distance $R_i - \rho_i$ from the element of surface area dA. G is a geometry factor defined by the source detector geometry in terms of the angles θ and ϕ of a standard spherical coordinate system, and R_i is the range of the alpha particle in bone. For a sample with a uniform distribution of emitters, the geometry factor should be the same for the parent and all the daughters. Thus, the ratio $n(E_i)/n(E_{226})$ is given by

$$\frac{n(E_i)}{n(E_{226})} = \frac{R_i}{R_{226}} \quad \frac{N_i}{N_{226}} \quad . \tag{14}$$

If we assume that the radioactive equilibrium exists between ^{226}Ra and its daughter products, then $(N_i/N_{226}) = 1.0$. Using computed ranges in dry bone,[3] Eq. 12 can now be evaluated. Table 1 displays the results of this theoretical calculation along with the empirical values of $\Delta n_i/\Delta n_{226}$ obtained from the individual components shown in Fig. 4. The values in Table 1 have been normalized to a value of 1.00 for ^{226}Ra. There is good agreement between theory and experiment for ^{222}Rn, ^{218}Po, and ^{214}Po, indicating that these three daughter products are in equilibrium with ^{226}Ra, as assumed. The disagreement between theory and experiment for ^{210}Po indicates that it is not in equilibrium with ^{226}Ra. In most bone samples some ^{222}Rn escapes so that equilibrium between ^{222}Rn and ^{226}Ra is not commonly encountered. The complete retention of ^{222}Rn in this sample is explicable by the fact that the bone was ashed. In such cases, ^{222}Rn retention usually increases due to a growth in the size of the bone crysfals. On a time scale of a few weeks, an increase in ^{222}Rn retention would produce a disequilibrium between ^{222}Rn and

Table 1. Comparison of Theoretical and Empirical Values of $\Delta n_i/\Delta n_{226}$

Nuclide	E_α MeV	Theory[a]	Empirical Fit
^{226}Ra	4.782	1.00	1.00
^{222}Rn	5.490	1.11	1.06 ± 0.03
^{218}Po	6.002	1.17	1.14 ± 0.03
^{214}Po	7.687	1.40	1.40 ± 0.04
^{210}Po	5.310	1.08	0.28 ± 0.02

[a]The calculation assumes 100% retention of ^{222}Rn.

^{210}Po, even though they might have been in equilibrium before ashing. This is due to the fact that the parent of ^{210}Po is ^{210}Pb, which has a 22.4 year half-life and could not undergo the same sudden increase in activity as its ancestor ^{222}Rn. Furthermore, it is likely that ^{210}Po would be driven off by the ashing procedure, thus lowering its activity with respect to ^{226}Ra and ^{226}Rn.

Although ^{222}Rn retention was assumed to be completely retained in this example, empirical values of ^{222}Rn retention, N_{222}/N_{226}, can be determined from the observed value of $\Delta n_{222}/\Delta n_{226}$ by substituting Eq. 14 into Eq. 12, solving for N_{222}/N_{226}, and evaluating the equation.

Figures 5a and 5b are plots of the ^{226}Ra and ^{214}Po alpha-gamma coincidence spectra, respectively. As expected for a uniformly distributed sample, the energy spectra are simple ramps with a sharp cutoff at the alpha decay energy. The cutoff is not perfectly sharp owing to finite detector resolution. Although it is not readily visible from Figure 5a, the ^{226}Ra cutoff is shifted downward slightly, relative to its position in the singles spectrum since the energy of the alpha actually in coincidence with the 186 keV gamma ray is 4.60 MeV instead of 4.78 MeV. The spectral region below channel 20 is not of interest because of increasing detector noise and nonlinearity of the range-energy relation at low energy.

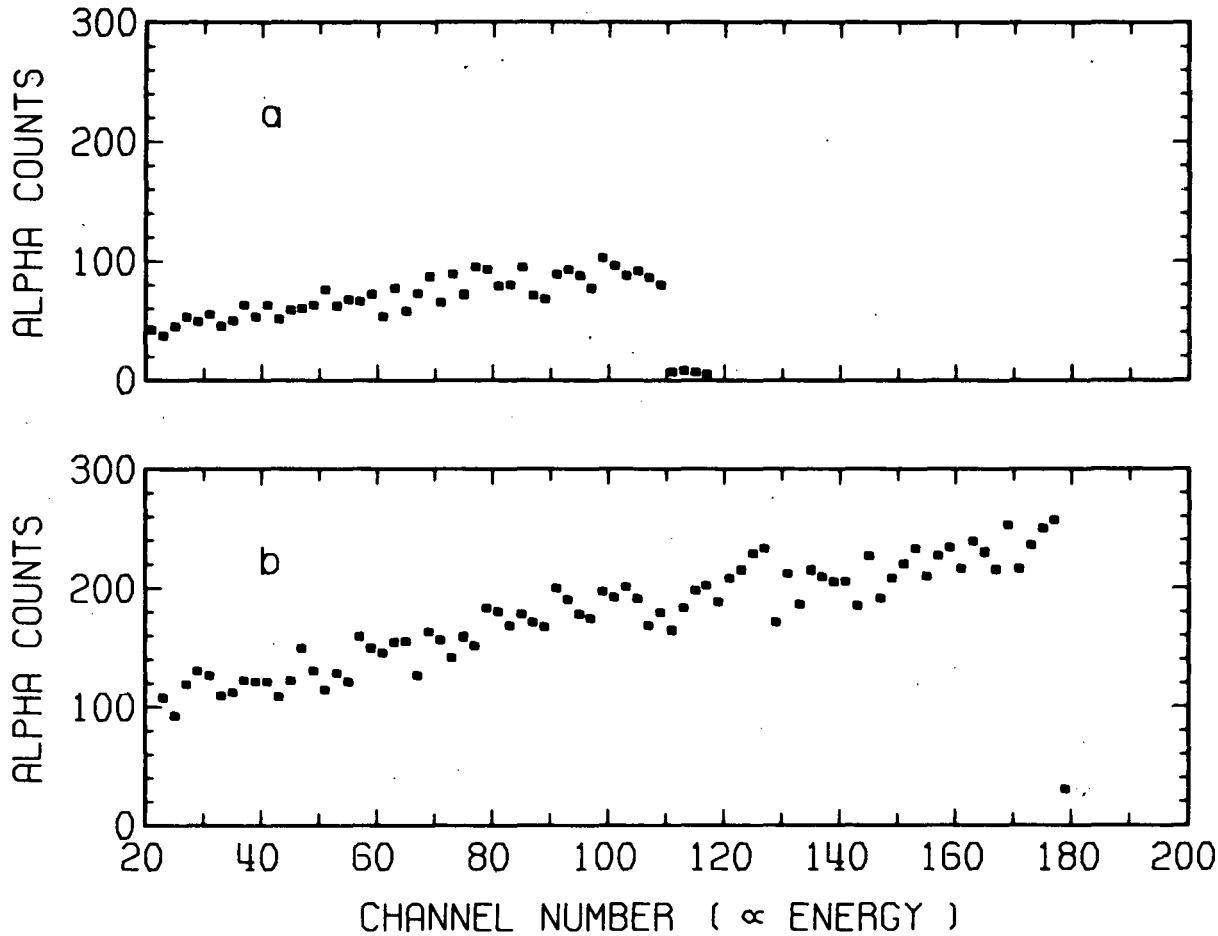

FIG. 5.--Alpha-gamma coincidence spectrum a) ^{226}Ra and b) ^{214}Po (every other point plotted).

Because no attempt has been made to measure the absolute efficiency of these coincidence systems, it is not meaningful to compare the detected number of counts to the empirical fits of Figure 4. However, the slopes of straight lines fitted to the ramps can be compared if we correct by a scale factor obtained from the ratio of singles to coincidence count at the cutoff energy. The slope of the measured ^{226}Ra component is 0.323 ± 0.126 (counts per channel) and 0.743 ± 0.044 for the measured ^{214}Po component. This compares to corrected slopes of 0.379 ± 0.062 and 0.613 ± 0.075 for ^{226}Ra and ^{214}Po determined from the deconvoluted curves. The uncertainties are statistical for the coincidence measurements but also include a systematic uncertainty of 10% for the stripped components. The agreement is reasonably

good. Further fine tuning of the proportionality constants C_{ij} could have improved these results somewhat.

Therefore, the ability of the analysis method described here to deconvolute a composite spectrum accurately into its components could have great practical significance in our program of measuring endosteal dose rates in terms of the time necessary to acquire each spectrum and the number of samples which can be measured concurrently. Further work will be done to extend this method to nonuniform radionuclide distributions and to test its accuracy with samples of lower specific activity.

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RADIOCHEMICAL DETERMINATION OF ^{226}Ra IN BONE: A PRELIMINARY ANALYSIS OF RESULTS

J. Rundo, R. B. Holtzman, and J. Y. Sha

Data on ^{226}Ra and calcium in samples of bone from 41 females who died from 1 to 53 years after exposure to radium have been subjected to preliminary analysis. The data for 86 samples from 26 femora show that there is a tendency for the $^{226}Ra/Ca$ ratio (normalized to the terminal ^{226}Ra body content) in any part of the femur to increase with time of death after exposure, but no progressive changes of distribution between cancellous bone (at the ends) and cortical bone (shaft) can be demonstrated. Direct comparisons are made between predictions of the ICRP model of alkaline earth metabolism and the data for compact bone (femoral shaft) and for cancellous bone (vertebra). Although the agreement is not good in either type of bone, the data provide some support for the model for samples from those cases in which the average absorbed dose to the skeleton was less than 10^4 rad (100 Gy). There is some indication of an effect of radiation on the metabolism of radium in bone, but this conclusion is based on the assumption that the ICRP model provides a true description of the rate of loss of radium from the different types of bone.

Introduction

Since the formation of the Center for Human Radiobiology in 1969, numerous samples of tissues, excreta and diet from patients have been analyzed for radium and for calcium, the chemical analogue of radium among the essential elements of the body. The data have been the subjects of interpretative papers on such topics as the excretion rate of radium at late times after acquisition, the metabolic parameters of radium in man, and the concentration of radium and calcium in the human eye.[1-7] Reports dealing with radium in bone have been confined largely to such studies as distribution in an individual skeleton or bone[2-4] or estimation of the content of the whole skeleton,[1,5] with little emphasis on the detailed metabolism of radium in bone.[1] We now have the analytical results for about 400 samples of bone in the CHRIS file CHEM and we present here a preliminary analysis of some of these data. In particular, we have examined the extent to which the data provide support for the ICRP model of alkaline earth metabolism,[8] and we have looked for evidence of an effect of radiation on the metabolism of radium in man.

Materials and Methods

Samples for these studies were obtained from exhumed and willed bodies, from surgical specimens, and from autopsies. Exhumation was the largest source. In mid-1972, the sampling of bone was standardized; when sufficient bone is available, the set of samples consists of seven pieces of a femur, one piece of a rib (mid-section), one-half of a vertebra, and one-half of the iliac crest. These are shown in Figure 1, for the samples from a body which was exhumed in 1972 after a 12-yr interment. The samples which were taken for chemical analysis are identified by the adjacent letters RC and by the arrows. The pieces of bone with the adjacent letters MA were used for microradiography and autoradiography; the remaining material was stored for possible future use.

The samples were either ashed overnight at 600°C and dissolved in nitric acid, or wet-ashed with nitric and perchloric acids. Once in solution, the samples were analyzed for ^{226}Ra by the emanation method and for calcium by atomic absorption spectrophotometry.

The results of the chemical analyses are stored in the file CHEM of the computerized CHR Information System (CHRIS) and they can be retrieved and manipulated as desired, alone or in combination with data on the same cases from other CHRIS files. Restrictions and selections of any kind are thus possible.

Treatment of Data

To make comparisons of data from many subjects we must attempt to allow for all the variables inherent in the samples and the subjects from whom they came. The most obvious confounding factor is the total body content of ^{226}Ra, which ranges from less than 5 nCi (185 Bq) to 10 μCi (370 kBq) or more; other factors are sex, age of the subject at first exposure, duration of exposure, duration of interment (which might relate to differential leaching by ground water of radium versus calcium), radiation dose-rate (including the effect of the 5.75-yr ^{228}Ra content of the material absorbed) and biological variability between individuals. Taking the last point first, we have shown[9] that there is considerable variability in the gross distribution of radium in vivo; while some 45% of 40 subjects investigated were in a category where a maximum of

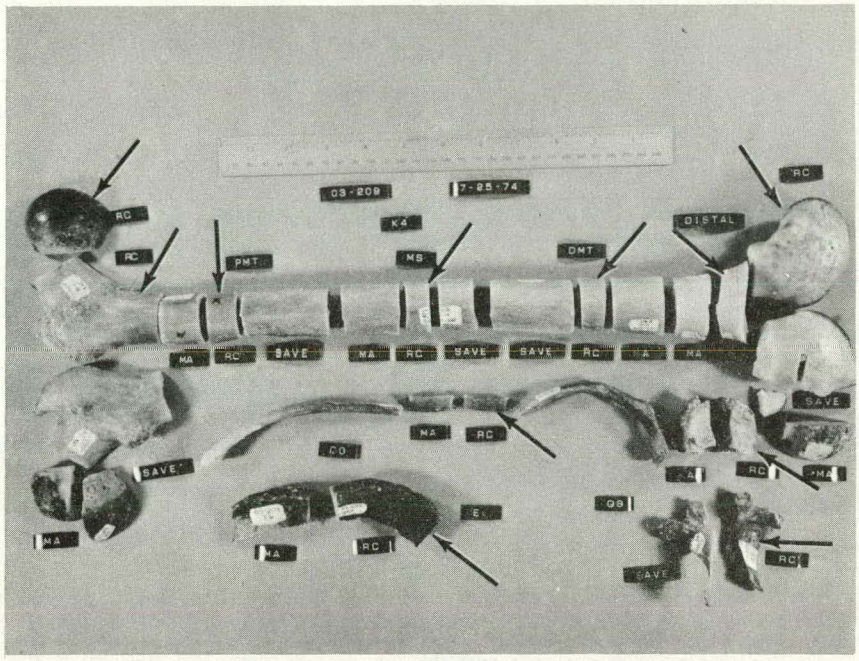

FIG. 1.--The bones selected for examination in the standard sampling. The arrows identify the parts submitted for chemical analysis. (Case 03-209) (ANL Neg. 149-78-285)

activity was observed in the lowest third of the (erect) body, in 30% of the subjects the highest activity was in the uppermost third of the body. These differences are of particular importance because the femur is heavily represented in the standardized sampling and they must be borne in mind when a comparison is made of results for femur from many subjects. Variations of a factor of about two on either side of the mean are, therefore, to be expected.

We have handled the problem of the wide range of terminal radium contents as follows. All the data are presented and manipulated as the ratio of radium content in pCi to calcium content in g (1 pCi/g = 37 Bq/kg), divided by the total radium content in μCi. Although the result, which we call the normalized ^{226}Ra/Ca ratio, has the dimension of mass^{-1} we shall still refer to it in units of pCi/g, it being understood that this is for a total radium content of 1 μCi. Use of this ratio (rather than radium concentration in dry or wet bone) puts all the data on a common basis relative to the mineral content of the bone, and eliminates variations caused by different pretreatment or condition

(cleaning, drying, presence of fat, etc.).[10] Furthermore, we make no mention of analytical errors in the data because they are always quite negligible (2% to 5%) compared with the biological variability, even at the lowest levels and for the smallest samples.

At the present time, the file CHEM contains the analytical results of at least one sample of bone from 45 females and 18 males who contained more than 5 nCi (185 Bq) ^{226}Ra at death. This lower limit was chosen because the uncertainty in the determination of the body content below 5 nCi is considerable (> 30%), even though the sensitivity of the radiochemical method for individual bones is adequate at far lower levels. A few of these 63 cases were not considered to be suitable because the uncertainty in the body content exceeded 20%, even though the estimated content was substantial. This was the case for four females and three males. Because of the preponderance of females, we shall here consider only the data obtained from them, thus eliminating the possible complication of differences due to sex.

Femur

In the remaining group of 41 females, we have results for 86 samples from 26 femora; many of these were received before the standardized sampling was introduced, which explains partly why the average number of samples per femur is so much less than seven.

Consideration of the normalized ^{226}Ra/Ca ratio at different times after exposure should be useful in demonstrating changes in the distribution of radium in the skeleton. One of the tenets of the ICRP model of alkaline earth metabolism is that the rate of turnover of cancellous bone is four times that of compact bone.[8] Since both types of bone are present in the femur, we have examined the data to see if such an effect could be demonstrated. The 26 subjects from whom the samples of femur were obtained had died from 1 to 53 yr after the mid-point of the exposure period. The results were therefore divided into four groups covering 1 to 4.5 (0–5) yr, 5.1 to 10.0 (5–10) yr, 10.2 to 16.8 (10–20) yr, and 20 or more years after the mid-point of the exposure period, and they were averaged for each section of bone within these groups.

The results are plotted in Figure 2, where it will be noted that there are data for only six sampling locations instead of the seven indicated in Figure 1. This is because the head and trochanter were not separated in six of the 12 femora for which analyses of this bone part have been completed. The results for head and trochanter were therefore combined and referred to simply as "head." In assessing the plots of Figure 2, it must be borne in mind that some of the different groups (time periods as well as sections of bone) are represented by very few samples. In fact, only one sample of condyle was available for both the 0 to 5 yr and the 10 to 20 yr groups. Examination of Figure 2 shows that there is a tendency for the average value of the normalized $^{226}Ra/Ca$ ratio to increase with time since the mid-point of the exposure period, although there are several irregularities and the increase is not pronounced for any bone part except the distal portion of the distal metaphysis (dist) where it amounts to almost a factor of two.

It can also be seen that there is a tendency for the normalized $^{226}Ra/Ca$ ratio to decrease along the length of the shaft from the proximal metaphysis (PMT) to the distal metaphysis (DMT), although there are again irregularities. The average of all three samples may thus give the "best" value for the whole shaft in an individual case.

We also note that the ratio is markedly higher at the ends of the femur, where there is a substantial amount of cancellous bone, than in the shaft, which is predominantly cortical; however, the data do not demonstrate a progressive change in the distribution of radium between cancellous and cortical bone, possibly because the number of sites sampled was insufficient.

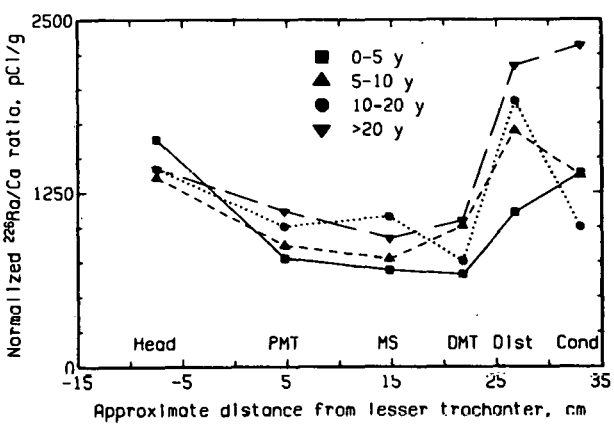

FIG. 2.--Summary of analytical results for 86 samples of femur from 26 females. PMT-proximal metaphysis, MS-midshaft, DMT-distal metaphysis, Dist-distal part of DMT, Cond-condyle (see Figure 1).

Comparison with the Predictions of the ICRP Model

The ICRP model of alkaline earth metabolism[8] contains mathematical functions which describe the fractional retention following intravenous injection of each of the four elements in a number of compartments of bone as well as in the whole body, soft tissue and plasma. Of particular interest in the present context are the retention functions for radium in the whole body, in compact bone, and in cancellous bone. These are designated R, $R_{COMPVOLUME}$, and $R_{CANVOLUME}$, respectively. Compact and cancellous bone are defined in relation to the surface/volume ratio; the shaft of a long bone is associated with the compact bone of the model and the whole of vertebra with cancellous bone, despite the presence of some trabecular bone in the former and of some cortical bone in the latter. We can therefore compare the average of the analytical results for the three samples from the shaft of femur, PMT, MS, and DMT, with the function $R_{COMPVOLUME}$ and the results for vertebra with the function $R_{CANVOLUME}$, both functions being divided by R and by the appropriate calcium content to give the normalized $^{226}Ra/Ca$ ratio. For compact bone, which is considered in the ICRP model to contain 80% of skeletal calcium, the expression which predicts the ratio is

$$\frac{10^6 R_{COMPVOLUME}}{0.8\ CR}$$

where C is the total calcium content of the body, and the factor of 10^6 allows for the fact that our ratio is expressed as pCi ^{226}Ra/g Ca per μCi total ^{226}Ra content. A value for C has been taken from the results of Cohn et al.[11] and rounded to 800 g. In an individual, the total calcium content depends on stature and on age, and an average value of 800 g is certainly too high for females who were much older than 55 years at the time of death; although the age of each of our subjects was known, in most cases we have no information on the height and weight. Therefore, it was not possible to adjust the measured $^{226}Ra/Ca$ ratio to allow for the not inconsiderable variations with stature. The coefficient of variation in Cohn's data ranged from 4.5% to 16% for ages ranging from 30 to 78 yr.

The expression for the model's predictions of the normalized $^{226}Ra/Ca$ ratio in compact bone then becomes

$$\frac{1562.5\ R_{COMPVOLUME}}{R}$$

while that for cancellous bone is

$$\frac{6250\ R_{CANVOLUME}}{R}$$

since 20% of the total calcium content is considered to be in cancellous bone. In these expressions, no allowance has been made for the radium deposited on bone surfaces, but this is negligible for times of one year or more. More serious may be the fact that the expression for R includes the soft tissue component (which is estimated in the ICRP model to be 40% of the total body content at one year, 20% at three years, and 9% at 10 years), whereas most of our values for total body content were based on measurements of γ radiation from the exhumed skeleton with no soft tissue. Allowance can be made for this by replacing R in the above expressions by the sum of the retentions in compact and cancellous bone.

In Figure 3, the results for femoral shaft are plotted as a function of time since mid-exposure, and they may be compared with the predictions of the ICRP model, drawn as a continuous curve. The dashed curve shows the effect of replacing the whole body retention by the skeletal retention. The solid circles represent the mean values for the ten femora for which all three samples (PMT, MS, and DMT) have been analyzed. Only one or two samples from the remaining 16 femora have been analyzed, and the data are plotted as open circles.

The agreement between the data and either set of predictions of the model cannot be described as good, but it is particularly interesting to note that the continuous curve seems to describe an upper limit to the data points. It is also clear that the presence of the malignancies identified as radium-related (osteosarcoma, or carcinoma of the mastoid or paranasal sinuses) is not the reason for the substantial differences between the observed and predicted values of the normalized $^{226}Ra/Ca$ ratio. For about half of the points, there is reasonable

agreement between the observed and predicted ratios. If we assume that the model provides a correct description of the metabolism of radium in bone at this level of detail, we can ask if the deviations from the continuous curve are a consequence of an effect of radiation on the metabolism. To do this, we plot the ratio of the observed to predicted normalized ^{226}Ra/Ca ratios as a function of average skeletal dose in rad in Figure 4. The line is based on the results of a linear unweighted least squares fit to the data. The correlation coefficient, r, was -0.50 which was significantly different from zero ($p \approx 0.01$, d.f. = 23). Inspection of Figure 4 suggests that the four points at the highest absorbed doses are crucial to the correlation, and if they are omitted, r becomes -0.34 which is not significant ($p > 0.10$). Thus, there is some evidence for an effect of the alpha-radiation on the metabolism of radium. However, it must be remembered that this demonstration of a possible effect of radiation is based entirely on the assumption that the ICRP model provides a true description of the metabolism of radium in bone. In this connection, it may be noted that the intercept of the fitted line (0.887 ± 0.044) is barely significantly different from unity. The straight line fit to the data should not be construed as representing a conclusion as to the form of a dose-response curve, which the data are inadequate to predict.

Vertebra and the ICRP Model

Analytical results are available for 24 vertebrae from the 41 females who met the criteria already detailed. For seven of these subjects, the vertebra was divided into arch and body, and the two parts were analyzed separately. For one case, two vertebrae were so treated. There was considerable variability in the results, but no consistent difference between the two parts. Thus, in five vertebrae (from four subjects), the ^{226}Ra/Ca ratio was higher in the arch than in the body, while the reverse was the case in the other three pairs of samples. The ratio of the ^{226}Ra/Ca ratio in the body to that in the arch ranged from 0.55 to 1.40, while the mean was 0.92. For these subjects, we have therefore used the mean of the results for body and arch as the value for vertebra.

The normalized $^{226}Ra/Ca$ ratio is plotted in Figure 5 as a function of time since the mid-point of the exposure period and the predictions of the ICRP model are again shown as continuous and dashed curves (compare with Figure 3). The 12 arrowed points are again for samples from patients with radium-related malignancies. As was the case for the femur, the agreement between observed and predicted ratios is not good, although it should be noted that only seven of the 24 points deviate from the curve by more than a factor of two. It is also noteworthy that the curve appears to describe a lower limit of the points, the opposite of the situation for femoral shaft (Figure 3). To determine whether the largest deviations are a consequence of a radiation effect, we again plot the ratio of the observed to predicted normalized $^{226}Ra/Ca$ ratios as a function of average skeletal dose (Figure 6). The continuous line represents the results of an unweighted linear least squares fit to all the data; the correlation coefficient was +0.48, which was significant ($p \approx 0.02$, d.f. = 22). As was the case in Figure 4, the straight line does not represent a conclusion as to the shape of the dose response curve. Inspection of both Figures 4 and 6 shows that the scatter is such as to preclude detection of a significant effect of radiation at average absorbed doses to the skeleton of less than about 10 krad (100 Gy). We note that the intercept of the line drawn in Figure 6 (1.49 ± 0.22) is hardly significantly different from 1.0, the value expected if the ICRP model provides a true description of the metabolism of radium.

Discussion and Conclusions

The demonstration of a possible effect of radiation on the metabolism of radium in bone is of considerable interest, although the data are inconclusive as yet because of the small number of cases with sufficiently high absorbed doses to show an effect that is greater than the scatter. The reason that the effect in compact bone is in the opposite direction from that in cancellous bone may be understood from the following argument. The effect is expected to manifest itself as a greater retention in bone due to the inhibition of resorption. This effect should be more marked in bone that turns over more rapidly, i.e., cancellous bone. This is confirmed by the data in Figure 6; cancellous bone,

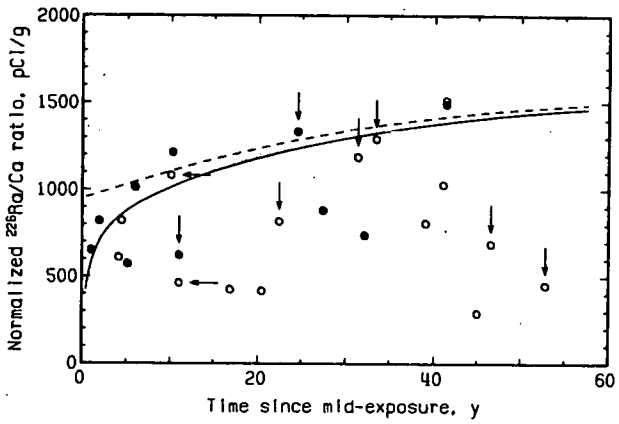

FIG. 3.--Comparison of the observed (points) and predicted (curves) normalized ^{226}Ra/Ca ratios for 25 samples from femoral shaft. Shaded circles are mean values of PMT, MS, and DMT; unshaded circles are values for one or two samples only (see text). Arrowed points are for samples from subjects with malignancies.

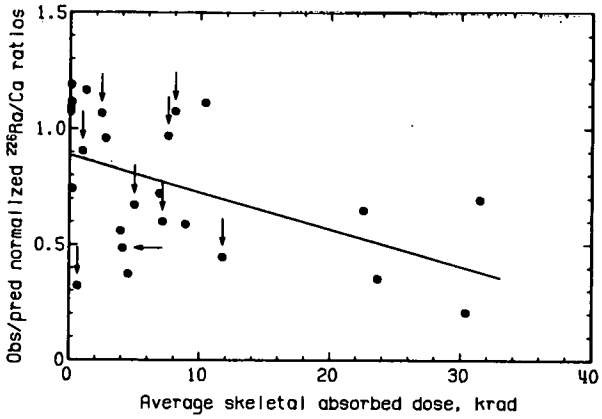

FIG. 4.--Ratio of the observed normalized ^{226}Ra/Ca ratio of femoral shaft to the value predicted by the ICRP model (continuous curve of Figure 3), as a function of average skeletal dose. Arrowed points are for samples from subjects with malignancies. The straight line represents the results of a least squares fit to the data.

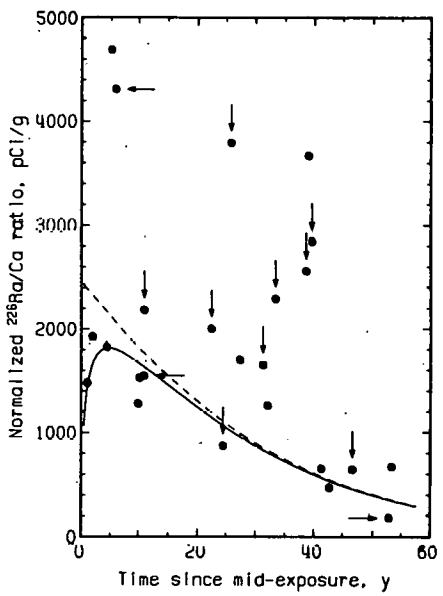

FIG. 5.--As for Figure 3, but for 24 samples of vertebra. Arrowed points are for samples from subjects with malignancies.

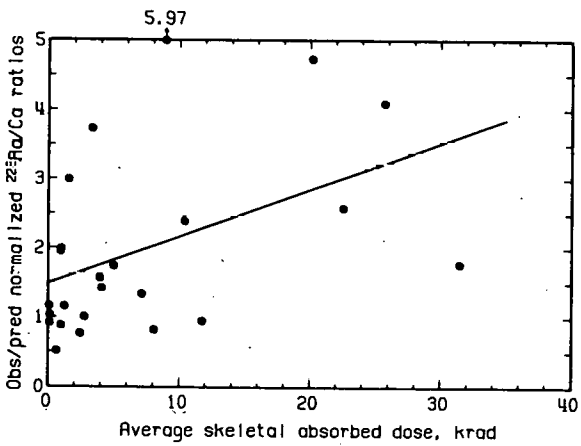

FIG. 6.--As for Figure 4, but for vertebrae. The straight line represents the results of a least squares fit to the data.

therefore, contains a larger fraction of the whole body content at late times than predicted by the ICRP model. The fraction of the total content in compact bone is, therefore, smaller than predicted and this produces the decrease with dose seen in Figure 4. The situation is complicated by the fact that the whole body retention function, R, obviously takes no account of radiation effects, and it therefore predicts a retention which is lower than the retention would be at high doses if resorption were inhibited. The deviations of the data for the femoral shaft from the ICRP model are thus enhanced by this, while those for the vertebra are reduced.

The data for cases with "low" doses certainly provide some support for the details of the ICRP model despite the scatter evident in Figures 3 and 5. Thus, for femoral shaft, the normalized ^{226}Ra/Ca ratio was within a factor of two of the predicted ratio for 16 of the 19 subjects who received less than 10^4 rad, and it was within 25% for nine of these. For vertebra, 18 subjects received less than 10^4 rad, and the ratio was within a factor of two of the predicted value for 15 of these and within 25% for seven. In view of the variability of the gross distribution of radium observed in vivo,[9] this must be regarded as quite good agreement for both bone types. It is particularly interesting that the predictions for the normalized ^{226}Ra/Ca ratio in cancellous bone at times later than 40 years after exposure, agree to within a factor of two (Figure 5 and Table 1) with the observed values in the five cases for which we have data. On the

TABLE 1. Comparison of observed and predicted normalized ^{226}Ra/Ca ratios for five very long term cases.

Time since mid-exposure y	Case Number	Average skeletal dose, rad	Tumor	Obs/pred normalized ^{226}Ra/Ca ratios Vertebra	Femur
41.3	05-116	64	-	1.16	1.08
42.7	01-100	975	-	0.88	-
46.6	05-281	4099	Sarcoma	1.42	0.49
52.8	03-488	621	Carcinoma	0.51	0.31
53.3	01-183	917	-	1.95	-

other hand, in two of the three cases for which we also have values for femoral shaft, the agreement is poor; it may be noteworthy that both cases had malignancies. For case 05-281, the deviations from unity are in the direction expected for an effect of radiation on the metabolism of radium in both types of bone; the average skeletal dose was substantial, although less than 10^4 rad.

Of the several variables enumerated earlier, we have taken no account of the age of the subject at first exposure, the duration of the exposure (except insofar as we have used the mid-point of the exposure period as the instant at which the radium entered the blood), or the radiation dose rate. By restricting our analysis to bones from females, we have eliminated the possible effect of sex; biological variability was all too evident in the results as plotted in Figures 3 and 6. We intend to investigate the influence of the other variables, possibly using multivariate regression analysis.

Some of the discrepancies between our data and the predictions of the ICRP model, other than those which may possibly be attributed to effects of radiation could result from incorrect assumptions in the model. We have examined the consequences of changes in the values of two of the parameters, viz., λ, the rate of apposition and resorption in compact bone and σ, the ratio of the turnover rates of cancellous and compact bone. In the model the value of λ was taken as 2.5%/yr for calcium and strontium, but as 1.5%/yr for radium. When the value of λ is taken as 2.5%/yr for radium, there is little effect on the predicted normalized ^{226}Ra/Ca ratio for compact bone; there is a small increase, amounting to no more than about 10%, and most noticeable at times between 15 and 40 yr after intake. The effect on the ratio for cancellous bone is dramatic. The value of the ratio is lowered by about 25% at 10 yr and by more than a factor of four at 50 yr after intake. The agreement with the data of Figures 3 and 5 is slightly worse for femur shaft and much worse for vertebra. Thus, the data offer no support for a value for λ of 2.5%/yr and a value of 4 for σ.

The effects of a smaller value of σ (with λ maintained at 1.5%/yr) were also investigated. With $\sigma = 2$, the predicted ^{226}Ra/Ca ratios were lower at all times than the values in the curve of Figure 3 for compact bone and higher than

the values in the curve of Figure 5 for cancellous bone. The decrease for compact bone was progressive with time but not great, amounting to 18% at 50 yr after intake. On the other hand, the increase for cancellous bone was quite dramatic, amounting to 22% at 10 yr and a factor of 3.6 at 50 yr. While the agreement with the data in Figure 5 was improved for times up to 32 yr, it was very poor for the five points at long times (41–53 yr), which were all for cases with relatively low absorbed doses. The entries in column 5 of Table 1 were decreased by a factor of 2.7 to 3.6. Thus, a decrease in the value of σ is only partially supported by the data.

The fact that the intercepts of the fitted lines in Figures 4 and 6 are both close to unity shows that the assumption that 80% and 20% of total skeletal calcium are in compact and cancellous bone, respectively, is about right.

Despite the detail into which we have gone, we still regard this as a preliminary analysis. There are seven cases for which we have results for bones other than femur or vertebra which might fall neatly into one of the two categories we have considered. Thus, shaft of tibia or humerus might be a suitable substitute for shaft of femur, and innominate might replace vertebra. This needs investigation.

A most useful consequence of making the analysis has been its revealing of where we need more data. Thus, we have identified samples for analysis for ^{226}Ra and calcium from six cases with average skeletal doses in excess of 10^4 rad. The results may help to confirm our tentative conclusion of an effect of radiation on the metabolism of radium in bone.

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^{226}Ra, ^{210}Pb, ^{210}Po, AND CALCIUM IN THE EYES OF LONG-TERM RADIUM CASES*

R. B. Holtzman, J. Y. Sha, and N. J. Plondke

It is known that dogs injected with radium accumulate a significant fraction of this element in the eye, with consequent radiation damage. We are interested in determining whether there are similar accumulations in the human eye. An eye from each of two radium patients (cases 01-144 and 01-017), who died about 50 yr after intake with terminal body burdens of 675 and 1160 nCi ^{226}Ra, respectively, was analyzed for ^{226}Ra, ^{210}Pb, ^{210}Po, and calcium.

The total amounts in the eyes were 1.80 pCi ^{226}Ra, 1.3 pCi ^{210}Pb and 2.3 mg Ca for case 01-144, and 5.4 pCi ^{226}Ra, 2.1 pCi ^{210}Pb, 5.0 pCi ^{210}Po, and 14.2 mg Ca for case 01-017. The eye from the latter case was dissected and showed the highest concentrations (on a wet weight basis) of ^{226}Ra in the choroid and of ^{210}Po in the iris and choroid (Table 1).

The ^{226}Ra concentrations in the choroid, sclera, and iris of case 01-017 were appreciably greater than in soft tissues from this subject. These concentrations ranged from 0.005 pCi/g in liver to 0.2 pCi/g in the thyroid. The radium contents of the eyes were 2.7×10^{-4}% and 4.7×10^{-4}% of the total body contents for cases 01-144 and 01-017, respectively. The ^{226}Ra/Ca ratios in the whole eye were 1/3 to 1/4 those in bone, but that for the choroid of the dissected eye (case 01-017) was about the same as for bone (1620 pCi ^{226}Ra/g Ca), while those of the sclera and cornea were about 360 pCi/g. The ^{210}Pb/Ca and ^{210}Po/Ca ratios in the eyes were comparable to those of the whole body, which indicates that the eye concentrates these nuclides relative to ^{226}Ra.

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*Summary of a paper presented at the Twenty-Third Annual Health Physics Society Meeting, Minneapolis, Minnesota, June 18–23, 1978.

Table 1. ^{226}Ra, ^{210}Pb, ^{210}Po and calcium concentrations in tissues of the eye (case 01-017).

Tissue	Wet Weight, g	^{226}Ra, pCi/g	^{210}Pb, pCi/g	^{210}Po, pCi/g	Ca, mg/g
Choroid	0.14	3.8	2.0	6.3	2.4
Sclera	1.39	3.4	1.1	2.4	9.4
Iris	0.035	2.9	2.7	6.8	0.0
Ciliary body	0.12	0.71	0.74	4.0	0.25
Retina	0.16	0.093	-	-	~ 0.0
Lens	0.28	0.05	0.28	0.31	0.42
Cornea	0.074	0.29	0.8	-	0.81
Vitreous humor	4.1	0.00	0.02	0.0	0.07

For case 01-017 the mean terminal dose rate to the skeleton was about 60 rad/yr, while that to the choroid was estimated as about 1 rad/yr from the ^{226}Ra and ^{210}Po combined.

We wish to thank the many people who contributed to acquisition of these samples, especially Professor R. D. Evans, who made the arrangements, and to Drs. Rodrigues and Weinreb, who did the micro-dissection in the eyes of one subject shortly after death.

FIELD MEASUREMENTS OF RADIUM IN THE HUMAN BODY

R. E. Toohey and H. A. May

Two whole body counting systems have been developed and employed for field measurements. The radium contents of nine previously unmeasured cases have been determined during three field trips. Future trips are being scheduled to make body radioactivity measurements on a specific subpopulation of CHR radium cases.

Introduction

The need has existed for some time for a method of obtaining reliable body radioactivity measurements of radium cases who are unwilling or unable to visit the CHR whole body counting facilities. Due to the variability noted in radon exhalation rate by any given individual[1] and to sampling difficulties, the field collection of radon breath samples does not meet this need. A preferred method of determining at least the retained fraction of body radium content is by γ-ray spectrometry using an NaI(Tl) detector. Two such detectors have been readied as part of complementary systems. The first consists of a shadow-shield whole body counter mounted in a semitrailer, and the second consists of a crystal, supporting stand, and framework which are transportable by "carryall" or similar heavy-duty vehicle. This latter system is intended for measurement of those cases who cannot be counted in the trailer, e.g., a bed-ridden patient, and it uses the same detector and associated equipment prepared for the detection of radium in buried remains.[2]

The chief target population for these field measurements are those CHR cases who worked as dial painters prior to 1930 and who have never had a body radioactivity measurement. Our files contain 263 cases of this type, of whom 207 have been located. Of these, 81 are currently unwilling or unable to visit CHR but may not object to being counted near their homes. The cases are concentrated in three geographical areas corresponding to the principal locations of the radium dial industry, namely Ottawa, Illinois, Orange, New Jersey, and Waterbury, Connecticut.

The Mobile Counter

The mobile counter consists of a shadow shield essentially identical to that described by Palmer,[3] installed in a 7.6 m (24 ft) long trailmobile semitrailer.* The base and sides of the shield are 4.1 m long, assembled from standard lead bricks placed on edge. This gives a thickness of 102 mm (4") under the bed and of 51 mm (2") at the sides, which are spaced 0.6 m apart. The thickness of the central 0.53 m of the shield (that portion which supports the detector housing and most directly "shadows" the detector) is increased to 204 mm. The detector crystal is mounted within a cubical housing placed over the center of the base; shielding of 204 mm is provided to the sides and top. The entire assembly is held together with steel straps and tie rods fastened to the heavy oak floor of the trailer, and covered with a formed stainless steel enclosure, which is welded into a single housing. The bed on which the patient reclines is moved underneath the detector by a chain drive mechanism, so that each measurement is an integrated profile scan.

The radiation detector is a 204-mm diameter by 102 mm-thich NaI(Tl) crystal, coupled to three photomultiplier tubes. A capacitative mixing circuit is used to combine and equalize the signals from the three tubes. We have encountered intermittent instability and gain shift in the resistive adders which are currently used on all the detectors with multiple photomultiplier tubes in the CHR whole-body counting facilities.[4] This is usually caused by high resistance contacts developing within the trimmer potentiometers used for gain setting. Since greatly increased incidence of such problems was anticipated in the more severe environment encountered by a mobile system, we replaced the adders by a capacitative mixer which eliminates all sliding contacts (Figure 1). Variable capacitors C_1 are glass or ceramic dielectric units, with a range of 5 to 50 pF. Fixed capacitors C_2 are silver mica or Mylar types, with values chosen to give rough equality of signals at the summing junction. The charge sensitive preamplifier presently in use was selected because it has a minimum number of components and could be easily

*The shield and trailer were purchased used from Helgeson Nuclear Services.

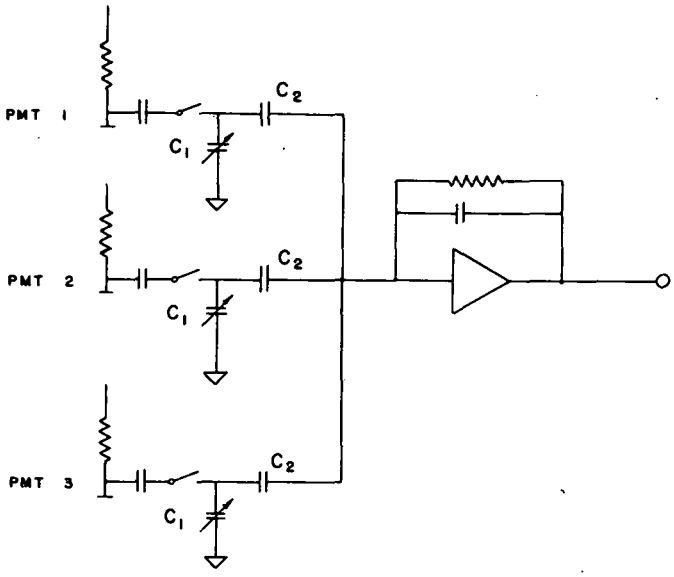

FIG. 1.--The capacitative mixing circuit used with the NaI(Tl) crystal in the mobile counter. (ANL Neg. 149-78-278)

built on a small printed circuit board adjacent to the tube sockets without any compromise in performance.* The entire mixer and preamplifier occupy approximately 50 cm^2; any contemporary charge-sensitive preamplifier would be usable.

The γ-ray spectra are accumulated with a multichannel analyzer and punched onto paper tape for later analysis. All electronics units are mounted in a double width rack cabinet, which is isolated from road vibration and shock with vertical and lateral shock mounts. In an attempt to obtain maximum isolation, we first employed a system of six vertical and three horizontal air-filled mounts (Barry Type SLM-1), which gave a natural frequency of around 4 to 5 Hz. Despite the manufacturers' claim that no external snubbers or lateral supports were required for the mounts, they failed on the first road trip ($\sim$100 miles, much of it interstate highway) and gave physical evidence of having grossly exceeded their elastic limit. A second type of all-elastomer mount (Barry Type 507-1BC) with much greater internal damping has been installed, with greater attention being given to matching shock mount position to the actual load distribution. The system now has a natural frequency of about 11 Hz and is protected against excess displacement in all planes. Actual over-the-road performance remains to be evaluated.

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*The preamplifier was designed by Irving Sherman of the Electronics Division.

The trailer contains two 1.5 kW electric heaters and an 18,000 BTU (19 MJ) air conditioner for climate control. It is wired for 230 V, single phase power, with ground fault interrupters to give extra safety to all concerned. Two hundred feet of heavy duty cable are provided, so that commercial power may normally be utilized. When this is impossible, an auxiliary gasoline-driven alternator mounted underneath the trailer is employed. Many small standby-type generators, primarily designed for contractors or recreational vehicle use, were tested and proved to be seriously deficient in frequency stability under variable loading, and in wave form. For satisfactory operation of electronic equipment having synchronous motors and/or regulating-type power transformers, wave forms and stability approaching that provided by the power company are essential. The unit finally chosen (Onan Model 2.5AJ, 2.5 kW capacity, electric start) can satisfactorily power one of the heaters and the electronics units without significant effects introduced by cyclic switching of the heater load. However, considerably larger capacity would have to have been provided to meet the transient loads imposed by the air conditioner.

The counter was calibrated in the field by radioactivity measurements of case 03-637, who had previously been counted in the reclining chair at ANL. The calibration factor for the mobile counter is 1.2 nCi ^{214}Bi/count/min. This is approximately twice that of the reclining chair, as was expected from considerations of crystal size and counting geometry. In addition, the higher and more variable background in the field results in a limit of detection for the mobile counter (3σ of background) of 9 nCi (333 Bq) ^{214}Bi. The calibration factor for ^{212}Bi (^{228}Ra series) can be derived by correcting the ^{214}Bi factor for differences in detector efficiency and γ-ray abundance, and is equal to 0.6 nCi ^{212}Bi/cpm. The background in the ^{212}Bi band (2.52–2.72 MeV) is approximately a factor of three lower than that in the ^{214}Bi band (1.66–1.86 MeV), and consequently the limit of detection for ^{212}Bi is estimated to be 3 nCi (111 Bq).

The results for the cases measured in the mobile counter during the past year, and their radium contents, are given in Table 1. The ^{226}Ra contents

Table 1. Cases measured in the mobile counter.

Case No.	Where measured	^{214}Bi content, nCi ± 1σ	^{226}Ra content, nCi ± 1σ
03-701	Ottawa, Ill.	-1.4 ± 4.0	-3.8 ± 10.9
05-007	Orange, N.J.	10.4 ± 3.6	28.1 ± 10.7
05-174	Orange, N.J.	-1.9 ± 2.7	-5.1 ± 7.3
05-303	Arlington, Va.	0.5 ± 3.8	1.4 ± 10.6
05-395	Berkeley Hts., N.J.	-2.8 ± 2.8	-7.7 ± 7.7
05-574	Berkeley Hts., N.J.	-1.6 ± 2.7	-4.3 ± 7.3
10-438	Ottawa, Ill.	5.0 ± 3.1	13.5 ± 8.7
10-595	Ottawa, Ill.	2.2 ± 2.8	5.9 ± 7.6

have been derived from the ^{214}Bi contents with the assumption of 38% radon retention. Case 05-007 was counted at MIT in 1967 and was found to have a ^{214}Bi content of 10 ± 1 nCi (370 ± 37 Bq); there should have been about a 10% decrease in the content since that measurement. The recent measurements in the trailer gave 10.4 ± 3.6 nCi (385 ± 133 Bq), in excellent agreement for a measurement so near the limit of detection.

Future improvements to the mobile counter include a new multichannel analyzer with region-of-interest sums displayed and magnetic tape output. Studies are under way to relate subject background to body size and to improve the calibration by counting more cases with known body burdens who visit ANL as part of the regular program of radium studies.

The Portable Counter

The portable counter consists of a 102-mm diameter by 102-mm thick NaI(Tl) crystal and a supporting stand. The stand is a slide projector table

with reinforced legs which are locked into a steel frame which rests on the floor. The frame contains an array of lead bricks which is 0.7 m wide by 1.2 m long by 51 mm thick. The face of the detector is 745 mm above the lead and the detector is surrounded by lead rings, forming a cylinder with 70-mm thick walls. The shield weighs 295 kg (13 rings); the single heaviest ring is 30 kg (64 lbs). The total weight of the system is 880 kg.

This detector was used in the field to measure the radium content of case 03-717 some 30 hr after death. Counts were made with the crystal centered over the entire body, over mid-sternum, and finally over the knees. The detector was calibrated with a point source of radium placed on the lead bricks, and the mean counting rate was then corrected for geometry (extended source), absorption in the body and the closer distance of the mid-plane of the body to the detector. The body content was determined to be 67 ± 20 nCi (2.5 ± 0.7 kBq) ^{214}Bi. The fractional retention of radon at death was assumed to be 0.38, and if there were no loss of radon from the body after death, the fractional retention would have grown to 0.51 at the time of the measurements. The best estimate of body radium content was obtained with a bias towards the figure assuming no loss of radon; it was calculated to be 150 ± 60 nCi.

It is difficult to establish a limit of detection for this counter because of the dependence of its calibration factor on body size and position under the counter. The above measurements indicate a limit of detection of approximately 50 nCi (1.85 kBq) ^{214}Bi for a counting time of 1 hr.

These measurements also indicated the need for more easily transportable electronic equipment. The present configuration, along with the lead shielding, requires more than a single automobile for transport. Our goal is to reduce the bulk and weight of the counter so that, omitting the lead bricks, the system would be easily transportable by a single individual, even on a commercial airplane if necessary.

Finally, it should be noted that the purpose of this detector and also that of the mobile counter is not to distinguish among burdens of tens of nCi of ^{214}Bi, but rather between burdens of tens of nCi and those of 100 nCi or more.

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POSTPRANDIAL CHANGES IN THE EXHALATION RATE OF RADON PRODUCED IN VIVO*

J. Rundo, F. Markun, and J. Y. Sha

The rate of exhalation of radon by persons with long-standing radium burdens increases about twofold shortly after a meal. The increase is short-lived and "normal" values are regained in 1.5 to 2 hours. The effect may account in part for the poor reproducibility in estimates of the freely emanating part of the radium content.

*Abstract of a paper published in Science 199, 1211–1212 (1978).

POSTPRANDIAL CHANGES IN THE EXHALATION OF RADON FROM THE ENVIRONMENT

J. Rundo, F. Markun, and N. J. Plondke

The exhalation of radon originally inhaled from the home environment and dissolved in body fluids and tissues has been studied serially for periods of several hours in six persons. The observation of a pronounced postprandial peak in the rate of exhalation of radon shows that the similar peak observed in the exhalation of radon produced from radium in vivo, results from the flushing of a reservoir in soft tissue, and not from a change in the fraction lost from bone.

Introduction

Our observation of a postprandial peak in the rate of exhalation of radon by persons with long-standing burdens of radium,[1] raised the question as to the origin of the "excess" radon exhaled during the period of digestion. If it were the result of a transient change in the fraction of radon lost from bone, the dosimetric consequences would be trivial. On the other hand, if it were the result of the flushing of a reservoir of radon dissolved in soft tissue, it could be important to identify the reservoir which might be the recipient of chronic irradiation at levels greater than many soft tissues. This question would be resolved by the demonstration of a postprandial peak in the exhalation of radon which had been inhaled previously.

Because radon emanates from radium in building materials, the concentration of radon in the air of a house is commonly higher than in outdoor air, especially during the heating season when there is minimum ventilation.[2-4] We have previously observed relatively high levels of radon in the breath of some persons, which were not due to the presence of radium in vivo. Instead they could be attributed to exposure to elevated levels of radon in the subjects' homes.[5] We therefore decided to exploit this situation by determining serially the exhalation rate of radon in the breath of ourselves and some colleagues, paying particular attention to the period following a meal.

Methods and Results

Samples of air were collected from the houses of the subjects to determine the concentration of radon to which they had been exposed for periods of about 14 hr. Data on the six subjects are shown in Table 1.

Four of the subjects brought a breakfast of choice and went to the underground low background laboratory ("vault") as soon as possible after arriving at Argonne. One or two 10-min samples of breath were taken for determination of the initial exhalation rate of radon, and the subject then ate breakfast. Breath sampling was started immediately after the end of the meal and at first was almost continuous: serial 10-min collections were made during a period of one to two hours, with a two- or three-minute interval between each

Table 1. Data on the six subjects and the concentrations of airborne radon in their houses.

Case No.	Sex	Height, m	Weight, kg	Age, yr	Rn concentration[a] in house air, pCi/L
50-002	M	1.68	78.2	51	1.55 ± 0.11
50-009[b]	M	1.78	77.3	52	1.59 ± 0.11
50-009[b]	M	1.78	77.3	52	1.39 ± 0.11
50-026	F	1.58	94.5	29	20.2 ± 0.56
50-070	M	1.83	82.3	32	0.28 ± 0.08
50-109	M	1.71	84.1	38	1.24 ± 0.06
50-148	M	1.74	69.5	52	4.13 ± 0.17

[a] Means of pairs of consistent results on samples taken the night before and on the morning of the test, except for subjects 50-026 and 50-070, where the house air samples were taken two days later. 1 pCi/L = 37 Bq/m^3.

[b] Subject 50-009 was tested on two occasions, a week apart.

collection while the radon trap (charcoal) was being changed. After this time less frequent collections were made. The amount of radon exhaled in each 10-min period was determined, and the exhalation rate was plotted as a function of time after leaving home. The results were somewhat variable, but the common features were that a postprandial peak was present and that it was superimposed on a declining baseline. The most detailed (and also the most spectacular) results, obtained on two subjects who were tested for six and seven hours and who ate lunch as well as breakfast in the underground vault, are plotted in Figure 1 on a logarithmic scale. These were the subjects whose houses had the highest concentrations of radon; the exhalation rates reflected this. Error bars ($\pm$ 1σ) are not shown because for all points they were no greater than the heights of the symbols. In both cases there was a pronounced postprandial peak after breakfast, although the increase started later after the meal for subject 50-026 than for subject 50-148. Also, there was a definite peak after lunch for the former case, and this was not complete at the time sampling was discontinued. For the latter case, the evidence for a second postprandial peak was less conclusive. The dashed lines drawn under the first peaks represent assumed exponential baselines to permit the calculation of the "excess" radon exhaled during the period of the peak.

We wished to determine if the postprandial peak could be deferred until after lunch. Accordingly, two subjects ate no breakfast; the exhalation rate of radon was determined at intervals during the morning and continued more frequently after lunch which was eaten in the vault. The results are plotted in Figure 2. Error bars ($\pm$ 1σ) are again within the heights of the symbols. The postprandial peaks are quite unambiguous in both cases, but the details are very different. For subject 50-109, the straight line represents the results of a least squares fit of an exponential function to the data from 143 to 295 min, and the dashed extrapolation was used as the baseline for the peak. This procedure was not possible for subject 50-070; the results suggest strongly that sampling of the breath was discontinued before the exhalation rate of radon had recovered from the elevated values.

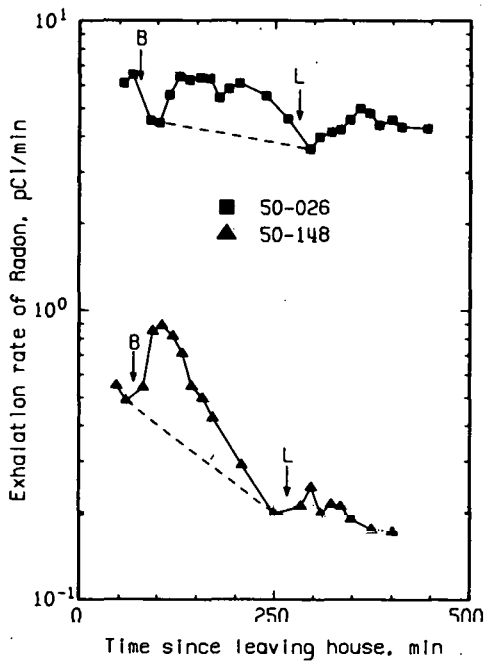

FIG. 1.--Exhalation rate of radon (logarithmic scale) as a function of time since leaving home, for two subjects: Arrows indicate mid-point of meal. B, breakfast; L, lunch. (1 pCi/min = 37 mBq/min).

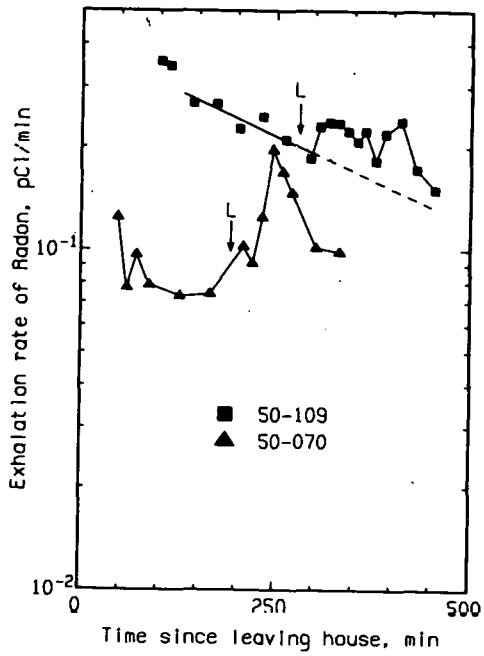

FIG. 2.--As for Figure 1, but for two subjects who ate no breakfast.

The total amount of radon exhaled during the period of the peak was calculated for each case. The amount which was exhaled during the time between successive pairs of samples was assumed to be at a rate equal to the average of the rates before and after. These amounts were calculated and added to the sum of the amounts found in the sampling periods. The estimated amount of exhaled radon which was not collected ranged among the subjects from 30 to 41% of the total, except for the first test on subject 50-009 for whom it was 69% of the total. This was an exploratory test and gamma-ray measurements were also being made in vivo during the course of the morning.

For each subject it was assumed that had there been no postprandial peak, the rate of exhalation of radon would have followed an exponential decline as exemplified by the dashed lines in Figures 1 and 2. The total amount under the baseline was determined by integration of the exponential function between appropriate limits. This quantity was then subtracted from

the total radon exhaled during the period of the peak; the results are set out in Table 2. The uncertainties shown are based on the statistical errors of counting; however, in the calculation of the error on the total amount exhaled, the estimated error on the amount exhaled between successive pairs of samples was doubled before propagation with those for the samples, to allow for some biological variability. For each subject the uncertainty on the area under the baseline was derived from the error on the ratio of the exhalation rates at the beginning and end of the period of the peak.

In order to compare the data for subjects who had been exposed to widely differing concentrations of radon (but for comparable periods of about 14 to 16 hr), the net area in each peak was converted from pCi to an equivalent volume of room air (EVRA)[6] by dividing by the appropriate concentration in the house air. The results are in the penultimate column of Table 2. The range of values for the peak areas is reduced from about 70:1 to about 4:1. As a measure of the relative effect, the ratio of the net peak area to the area under the baseline was calculated, and the results are given in the final column of Table 2. They show a range of about 3:1.

Discussion

The observation of the postprandial peak in the exhalation of radon from the environment proves conclusively that the source of the similar peak in the exhalation of radon produced in vivo is a reservoir of radon dissolved in body fluids or soft tissue, and not a change in the fraction released from radium in bone. It was hoped that the area of the peak, when expressed as the equivalent volume of room air, might give some clue as to the identity of the reservoir. However, there is no obvious correlation between the area of the peak and any of the parameters in Table 1. The identity of the reservoir remains a mystery, although one possibility is the capillary bed. More data are needed before any conclusion can be drawn.

The detailed behavior of the exhalation rate during the period of the peak merits comment. For subject 50-026 (Figure 1) and 50-109 (Figure 2) there is a suggestion of a splitting or doubling of the peak. When first observed

Table 2. Total, baseline, and net amounts of radon exhaled during the period of the postprandial peak.

Case No.	Total radon exhaled during postprandial peak, pCi	Area under baseline, pCi	Net peak area		Peak area / Baseline area
			pCi	EVRA[a], liter	
50-002	44.1 ± 0.4	33.7 ± 1.0	10.4 ± 1.1	6.7 = 0.8	0.31
50-009	50.5 ± 0.9	43.1 ± 1.1	7.4 ± 1.4	4.7 ± 1.0	0.17
50-009	30.3 ± 0.3	25.2 ± 0.7	5.1 ± 0.8	3.7 ± 0.7	0.20
50-026	1178 ± 8	890 ± 18	288 ± 20	11.0 ± 0.8	0.32
50-070	15.3-16.9[b]	11.5-11.9[b]	3.8-5.0[b]	13.6-17.8[b]	0.33-0.42[b]
50-109	35.6 ± 0.3	27.9 ± 1.4	7.7 ± 1.4	6.2 ± 1.2	0.28
50-148	89.8 ± 0.7	55.4 ± 1.4	34.4 ± 1.6	8.3 ± 0.5	0.62

[a]Equivalent Volume of Room Air--obtained by dividing the peak area in pCi by the concentration in house air in pCi/L (Table 1).

[b]Values depend on starting time assumed for peak and on how baseline is drawn. See Figure 2.

(in subject 50-109) this was thought merely to be biological variation, but its observation in subject 50-026 tended to confirm it as a real phenomenon, even though its existence depends on the validity of one low point in each case. The peak for subject 50-148 does not show a doubling, but there is a marked asymmetry of the peak; because of variations in respiratory minute volume, there was a small increase in the concentration of radon in the breath at 158 min. Similarly the results for subject 50-070 (Figure 2) suggest that there may have been a "step" in the declining portion of the peak which was not observed because breath sampling was discontinued too early. These observations warrant further study. It is possible that this noninvasive technique may offer a tool to physiologists for the study of some aspects of the digestive process not otherwise amenable to investigation.

It should be pointed out that an exhalation rate of 5 pCi/min (subject 50-026, Figure 1) would correspond to an emanating ^{226}Ra content of about 40 nCi. The observation of a similar exhalation rate for a subject known to have been exposed to radium, would be interpreted as a consequence of a radium burden unless it were not confirmed by gamma-ray measurements in vivo. The highest exhalation rate we have so far seen in a radium patient who probably contained very little radium, corresponded to an emanating content of 13.3 nCi[5], about one third of the level for subject 50-026. It is known that the latter contains no detectable radium and that the radon exhaled was due solely to the high level in the house. The results of further studies of radon in this subject, including gamma-ray measurements in vivo, and of studies planned for her husband, will be described elsewhere.

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DESIGN, CONSTRUCTION, AND OPERATION OF A NEW RADON TRANSFER SYSTEM

R. B. Holtzman, F. Markun, and J. E. Miranda, Jr.*

A new radon transfer line was constructed from a design modified from the earlier one of Lucas. A roller-type peristaltic transfer pump and a pressure-sensitive transducer were substituted for the previously used equipment. The electrical and electronic equipment, and operating switches are consolidated in a single control panel. Black or amber pure gum, latex dipped tubing were shown to have a minimum "memory" effect for radon, and a silicone tubing also appears to be suitable. The precision of measurement was about 2.7% (S.D.), based on eight replicate measurements with a single standard solution of radium. During the course of these test, it was found that part of the variability was due to leakage of radon from the flask that contained the radium.

Introduction

A new radon transfer system was built to increase the analytical capacity of our laboratory for the determination of ^{226}Ra and ^{222}Rn. This apparatus transfers ^{222}Rn, adsorbed on charcoal contained in a trap, to an alpha-scintillation radon counter.[1] The system is based on the design of Lucas,[2,3] which has been in use in our laboratory since 1964. A similar apparatus was described by Nelson and Rust.[4] The new system contains modifications that utilize newer equipment and materials to improve the performance and convenience.

A photograph of the apparatus is shown in Figure 1 and a diagram in Figure 2. The major changes to the original design are replacement of the large Sigma transfer pump by a smaller peristaltic roller-type tubing pump with a variable speed drive, and replacement of the mercury manometer control by a small pressure-sensitive transducer and indicator-controller that interrupts the transfer pump on completion of the transfer cycle. The control switches were modified for more convenient operation, and the switches, controls, and meters were consolidated in a single control panel. The

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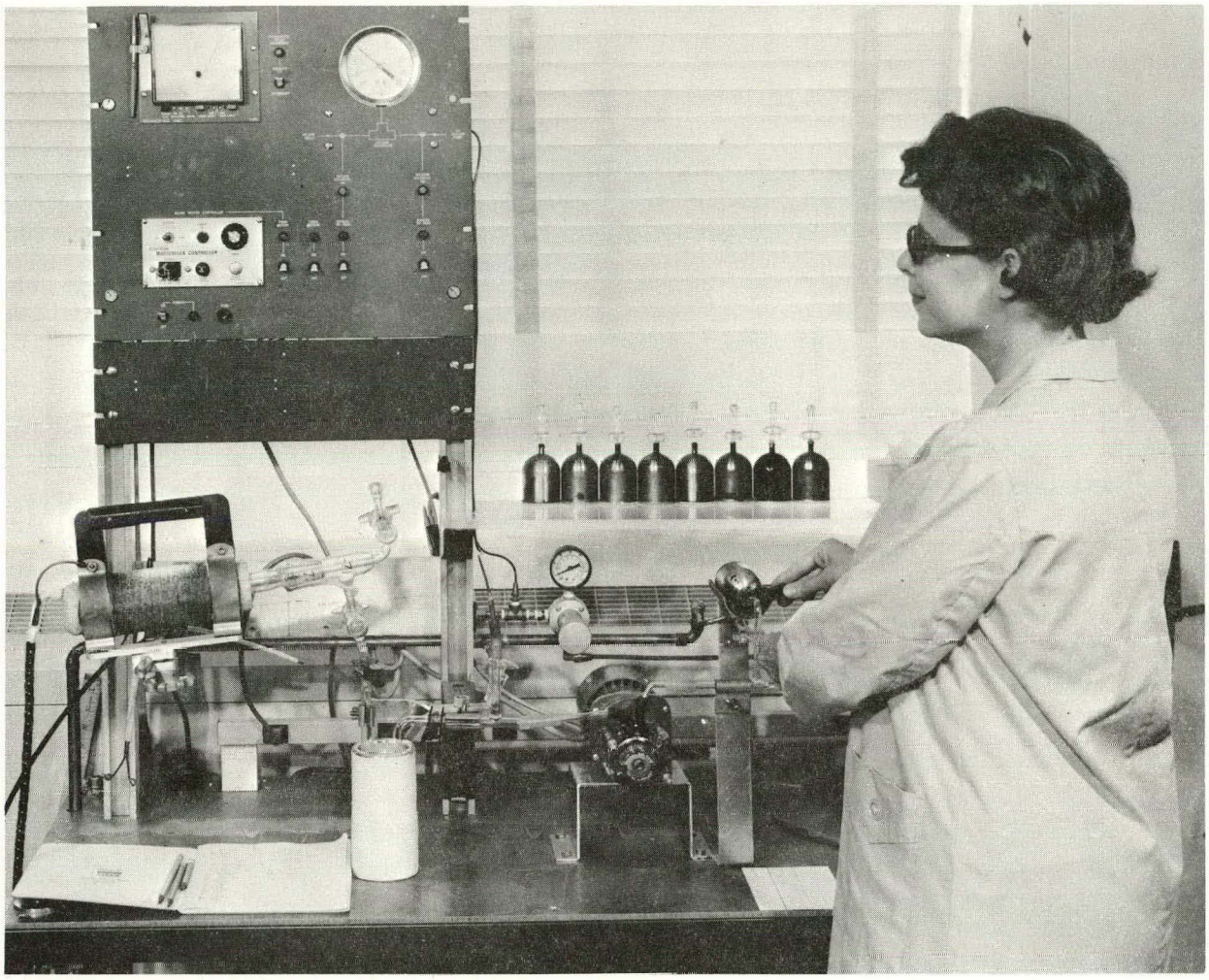

FIG. 1.--Photograph of the radon transfer apparatus in use. (ANL Neg. 149-77-297)

operation of the radon transfer system, as described in the Appendix, is very similar to that of the earlier apparatus. Special equipment and materials are listed in Table 1, along with possible manufacturers and suppliers.

Description and Operation of the Apparatus

Transfer Pump

The transfer pump is a variable speed (30 to 600 rpm) Masterflex pump with a Model 7015 pump head. The variable speed was essential to obtain optimum conditions, but for subsequent installations a fixed-speed drive, which

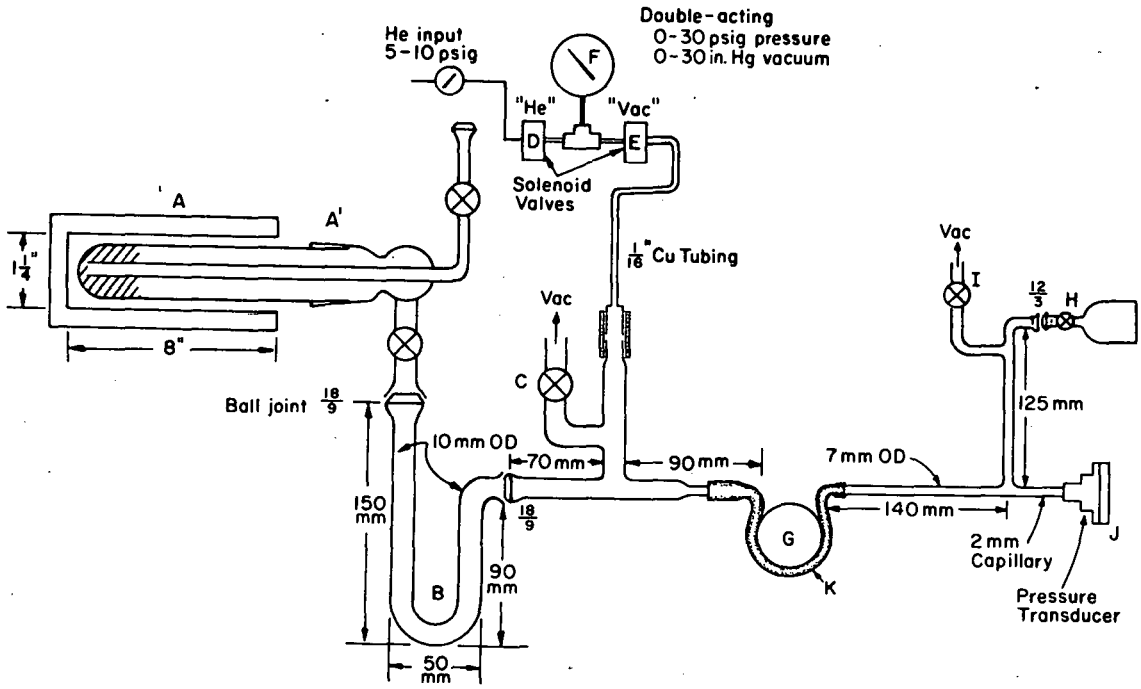

FIG. 2.--Diagram of radon transfer system: (A) trap heater (furnace) at 500°C with charcoal trap (A'); (B) water trap at -80°C; (C) stopcock to vacuum pump; (D) solenoid valve to helium reservoir; (E) solenoid valve to transfer line; (F) vacuum-pressure gauge; (G) transfer pump; (H) radon counting chamber; (I) stopcock to vacuum; (J) pressure transducer; (K) rubber tubing. (ANL Neg. 149-78-292 Rev.)

normally operates at 600 rpm, would probably be suitable. While this head nominally requires tubing with dimensions of 0.1925 in i.d. × 0.3920 in o.d. (4.89 × 9.96 mm, from Cole-Parmer), we have satisfactorily used a larger diameter black pure gum rubber tubing, 3/16 in i.d. × 7/16 in o.d. (4.76 × 11.11 mm). The use of gum rubber is necessary to minimize the "memory" effect, i.e., the retention of radon by diffusion into the tubing during one run, and diffusion back into the line during subsequent runs. Amber tubing of the same type and silicone rubber tubing were also found to be suitable. A thick wall is necessary if the tubing is to operate under vacuum.

The tubing and pump-head rollers were lubricated with castor oil to prevent abrasion and to minimize heating of the rubber. The memory effect was also minimized by the use of the shortest possible length of tubing (about

Table 1. Special equipment and materials and possible sources of supply.

Transfer pump	Masterflex pump with solid state controller, variable speed drive, 30 to 600 rpm, Model 7545, Cole-Parmer Instrument Co., Chicago, Ill., 60648.
Pump head	Model 7015, from above company.
Furnace	Semicylindrical heating unit Model 50022 Type 76-KS, 350 W, 57.5 V, nominal i.d. 1-1/4 in, length 8 in. Note that this model has the heating coils embedded in a ceramic cement; a lower temperature model has exposed heating coils. Two units are combined to form a cylinder. The coils are connected in series to operate at a nominal voltage of 115 V ac, Lindberg, Watertown, Wis.
Timer	Four cam 1 rpm, Foundation Type MC2, Gear assembly A-12 (for 60 s cycle), Industrial Timer Corporation, e.g., from Newark Electronics, Chicago, Ill., 60624.
Solenoid valves	Model V5 D 570S FP5, 3/32 in orifice, Skinner Electric Valve Div., New Britain, Conn.
Pressure-vacuum gauge	Double Acting Gauge, 3-1/2 in, 0 to 30 psig and 0 to 30 in Hg vacuum, e.g., Ashcroft.
Pressure transducer	Model TP1 controller, Model G (0.250 in dia.) transducer Range 0 to 15 psia, with limit option to turn off timer when pressure reaches atmospheric (adjustable limit) Sensotec, Inc., 1400 Holly Ave., Columbus, Ohio 43212.
Rubber tubing	Black or amber pure gum rubber, latex dipped, 7/16 in o.d. × 3/16 in i.d., 1/8 in wall, e.g., VWR Scientific, Inc. No. 56430-048, or SGA Scientific Inc., No. R8480 amber pure latex.
Furnace coating materials	Glass Cloth Electrical Tape, Scotch Brand, No. 27.
	Ceramic coating material for furnace, Dylon Super Bond Cement, Grade C3, Dylon Industries, 14430 Indian Creek, Cleveland, Ohio, 44130.
Furnace support arm	Panavise Model 300, Colbert Industries, 10107 Adella Ave., South Gate, Calif., 90280.

200 mm). The pump should operate at an optimum rate of about 500 to 600 rpm; this is fast enough to transfer the gas in 50 s, but slow enough to minimize heating. The transfer rate is adjusted so that, with a trap connected and open to the line, the latter will be pumped from a vacuum of 24 in (610 mm) Hg to 29 in (740 mm) Hg within 50 s.

The results of tests on the memory effects for various tubings are shown in Table 2. For these tests a sample of radon in a charcoal trap was transferred to a counting chamber, and the trap remained open to the line for subsequent transfers. The memory is then the amount in the second and subsequent transfers relative to the total activity.* The black pure gum latex dipped rubber tubing had the least memory. The similar amber tubing had essentially identical characteristics, while the smaller amber tubing was somewhat worse. This effect was probably the result of decreased pumping speed due to the smaller bore. For silicone tubing the memory effect is somewhat larger than that of the gum rubber, but small enough that this tubing appears to be suitable for use.

The smallest memory effect observed on this system, 0.24%, may be compared to the 0.6% found for one test on the older transfer system routinely used in the laboratory.

Electrical Control System

The transfer is automated to add carrier helium gas, and to start and stop the transfer at the proper times. A schematic diagram of the electrical circuits is shown in Figure 3.

The system uses a solid-state pressure sensor, rather than the mercury manometer and photocell-operated controller previously used. The solid state device should improve the reliability and reduce maintenance problems associated with dust and corrosion in the mercury. The transducer is attached to the vacuum line by a short section of rubber tubing, but because it is sensitive

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*While part of the effect attributed to tubing "memory" may be due to residual radon not transferred on the first cycle, adsorbed in the charcoal and dissolved in stopcock grease, we estimate these sources to contribute less than 0.1% of the total to the value of the memory.

Table 2. Memory effect of radon in tubing.

Tubing	Dimensions i.d., in	Dimensions Wall, in	Memory,[a] % of total
Pure gum rubber latex			
Black (SGA R-8480)	3/16	1/8	0.27
Amber (SGA R-8480)	3/16	1/8	0.31
Amber (SGA R-8480)	1/8	1/8	2/9
Silicone, Cole-Parmer	1/8	1/16	0.5

[a]Memory is the ratio of the radon transferred from trap to bottle in 2nd, 3rd, and 4th transfer cycles to the total radon transferred (sum of all transfers). The trap was flushed with helium five times during each transfer cycle.

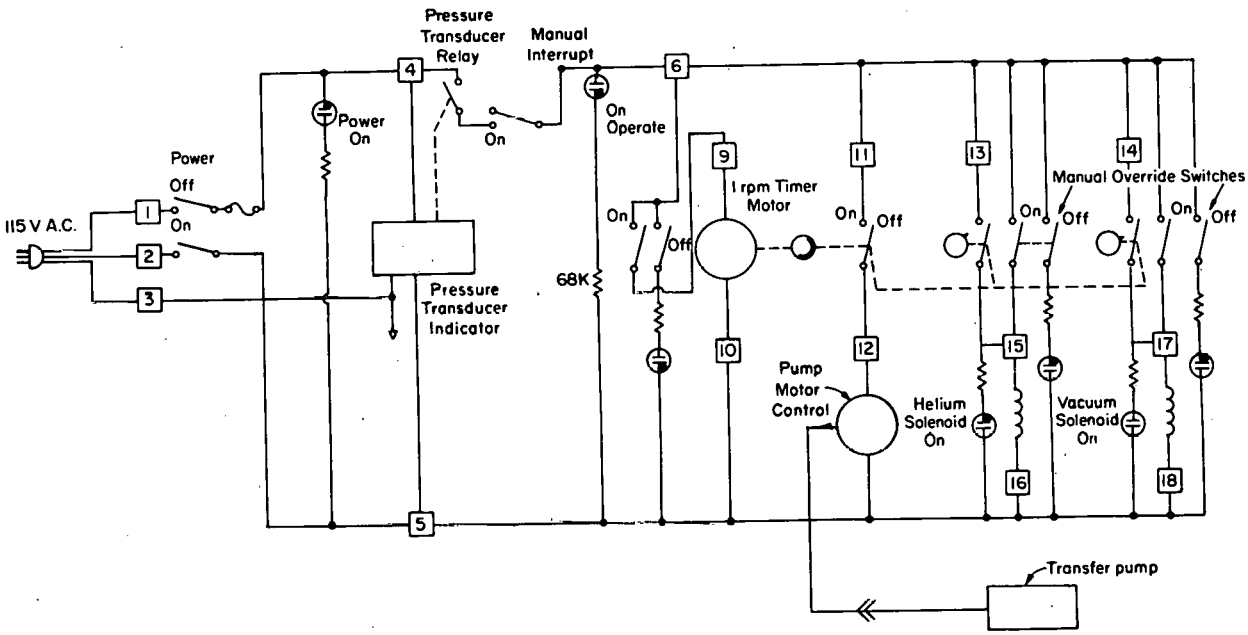

FIG. 3.--Schematic diagram of the electrical circuitry of the radon transfer apparatus. The numbered squares are terminals on terminal strips. (ANL Neg. 149-78-306)

to pressures on the barrel, a special holder was designed to hold it so that the forces from the holder would be exerted only on the mounting flange rather than the barrel of the transducer (Figure 4).

Flushing and pumping cycles are controlled by three cams of a timer, which has a one-minute cycle time. The first cam is adjusted to open momentarily solenoid valve D (Figure 2) ("helium solenoid" on Figure 3) to fill the chamber between D and E with gas. After D closes, valve E ("vacuum solenoid") opens momentarily to fill the line and trap with gas. The transfer pump is then activated by the third cam for 50 s to move the helium to the counting chamber. As the pressure in the chamber reaches atmospheric (after 5 additions of helium to the system), the controller on the pressure transducer interrupts the timer and pump.

The timer may be stopped at any point in its cycle by a manual interrupt switch. The timer cams must be set carefully to insure that solenoid D closes before E opens, and that the latter is open only momentarily (less than a few seconds). The cam operating the pump must be set to operate for at least 50 s, without overlapping the cycles of the other cams.

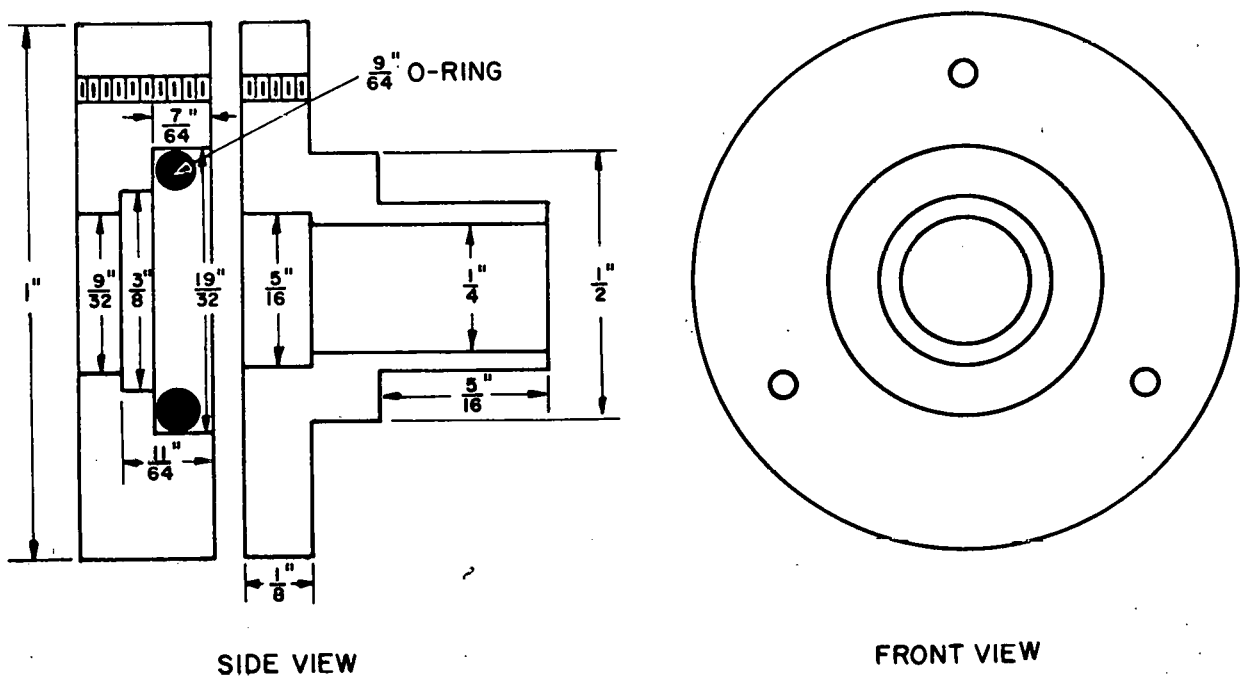

FIG. 4.--Diagram of holder for pressure transducer (brass construction). (ANL Neg. 149-78-307).

Trap Heater (Furnace)

The furnace for the traps is a cylindrical heater about 8 in long and 1-1/4 in i.d. constructed from two Lindberg semicylindrical 350 W heating units (Table 1) held together by glass cloth tape. The tape is then covered by ceramic furnace coating. The coils are connected in series electrically to give a nominal voltage rating of 115 V. Power is supplied by a variable autotransformer adjusted so that the temperature of the thermocouple in the center of the cylinder is 500°C.

Calibration of the System

The system was calibrated against a modified ^{226}Ra NBS standard,* and the results compared to those of the older system, which was calibrated similarly. When the radon counting chambers are filled on the older system, the overall transfer and counting efficiency is 5.32 cpm per pCi ^{222}Rn. Chambers filled on the new system had about a 5% greater overall efficiency. This was due to a smaller dead volume on the chamber side of the transfer line, G to H in Figure 2, so that in the new system a larger fraction of the transferred radon was in the chamber. Because the other system was in routine use, and to avoid the possible confusion of having several calibration factors, we increased the dead-volume of the new system by addition of a small glass bulb to the vacuum line on the chamber side of the transfer pump. The volume was adjusted to give the same calibration factor for both systems.

A test of the system with the modified 396 pCi ^{226}Ra standard showed a mean of 388 ± 11 pCi (S.D., n=8). The precision was good, with a coefficient of variation of 2.7% from eight measurements made over a period of three months (June 1 to August 29, 1977) as shown in Table 3. The variability includes counting statistics, counter variability, and the uncertainties in deemanating and collecting the radon in the trap. In addition, there appears to

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*This standard was from a 0.4986 aliquot fraction of NBS Standard Reference Material 4950-B, which contained a total mass of $8.06_q \times 10^{-10}$ g ± 1.00% ^{226}Ra in March 1968. On the basis of a half-life of 1600 yr for ^{226}Ra, the calculated activity in our standard solution at the time of measurement (August 1977) was 396 ± 4 pCi.

Table 3. Replicate analyses of a 396 pCi NBS ^{226}Ra standard, based on an overall efficiency factor of 5.32 cpm/pCi.

Date of measurement	^{226}Ra, pCi	Radon growth time,[a] days
6/1/77	392.6	4.84
6/7	389.0	6.02
7/6	375.7	29.93
8/8	375.3	32.02
8/10	394.8	2.02
8/16	386.6	6.01
8/29	385.7	13.02
8/29	407.9	0.0222

[a]Time since last de-emanation.

be a systematic error, attributed to slight leakage of radon from the emanation flask; the amount calculated is strongly linearly (negatively) correlated with storage time ($r = -0.88$, d.f. $= 6$, $p < 0.01$). The intercept at $t = 0$ of the linear regression curve, which represents the value with no leakage, is 397 ± 6 pCi, a value essentially identical to that of the standard. After correction for radon leakage, the precision of measurement is also improved, the coefficient of variation now being 1.5%. The estimated half-life of the loss of radon is about 120 days or about 1/2% per day. This effect is being studied.

Conclusion

The new transfer system is suitable for routine operation and has characteristics similar to those of the older Lucas system. The operational experience on the older system has allowed us to make some improvements in equipment and in convenience of operation. From repeated measurements of radon from a standard solution of radium, the precision of transfer, including experimental errors inherent in the measurement, was found to be about 2.7%, which can probably be improved if loss of radon from emanation flasks is prevented.

Acknowledgements

Thanks are due to the participants in this project, particularly R. Selman, R. Smith, and E. Fudala from Central Shops on the construction, and to H. F. Lucas, Jr. for many helpful suggestions and discussions.

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APPENDIX: Operation of the Radon Transfer System

1. An evacuated radon counting chamber is connected at point H, Figure 2.
2. The evacuated charcoal trap A' containing the sample is connected to the water trap B cooled with dry ice. The trap heater (furnace) A operated at about 500°C is then placed over the trap.
3. The lines are evacuated by opening the stopcocks to the vacuum pump at C.
4. The lines and counting chamber are flushed with helium at least 3 times by opening the solenoid valves D and E and operating the transfer pump G.
5. The system and counting chamber are evacuated again, and the stopcocks at C and I are closed.
6. The charcoal trap is opened to the system, and about 15 to 20 ml of He is added to the system. This is done by opening D to a helium reservoir maintained at about 5 to 10 psig (tank of helium connected to a gas regulator valve). D is then closed, and E is opened to the system.
7. The transfer pump is activated for 50 s to transfer the helium with entrained

radon into the counting chamber. The pumping rate is such that in 50 s the gas on the trap side of the line will be transferred almost completely, so that a "good" vacuum of about 29 in (740 mm) Hg is attained.

8. Steps 6 and 7 are repeated four additional times until the pressure in the chamber side of the system is about one atmosphere. The pressure of the helium and the transfer rate are adjusted to fill the counting chamber in approximately 5 cycles. On opening solenoid valve E to the evacuated line, the pressure on gauge F should be about 24 to 24-1/2 in (610 to 622 mm) Hg. Note that the pressure is appreciable because the gauge chamber now contains the helium for flushing.
9. On completion of the cycle, when the pressure in the chamber is approximately atmospheric, the timer is interrupted, the stopcock on the counting chamber is closed, and the chamber is removed for counting.

THE PLUTONIUM INJECTION CASES: AN UPDATE TO 1977*

R. E. Rowland and Patricia W. Durbin†

Several hospitalized individuals of relatively short life expectancy were given intravenous injections of plutonium in 1945–1947, and excreta were collected and measured for as long as they remained hospitalized.[1] Following the discovery by Durbin[2] that some of these individuals were still living, she and, subsequently, the Center for Human Radiobiology at the Argonne National Laboratory made an effort to trace all of these unique cases. This search resulted in a published report which documented the doses of plutonium administered, the survival of the recipients, the causes of death, and the doses accumulated by liver and bone in each case.[3]

The injections of plutonium contained from about two to more than 100 times the maximum permissible body burden of 40 nCi; two contained ^{238}Pu, while the remainder contained only ^{239}Pu. Seven of those injected succumbed to their illness within one year after receiving plutonium. Three more died in less than two and a half years. The remaining eight cases survived at least eight years, and four were still alive in 1975 when the Rowland and Durbin report was prepared.

The surviving cases are of considerable interest, for each carries a high level of plutonium in their liver and skeleton. One case died in November of 1975, identified as HP-8, of causes that do not seem to be related to injected plutonium. Three remain alive at this time, having carried plutonium for more than 30 years. Two are considered to have body burdens about seven times the maximum permissible level.

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*Summary of an oral presentation at the Scientific Group Meeting on Long-Term Effects of Radium and Thorium in Man, Geneva, 12–16 September 1977 (World Health Organization).

†Lawrence Berkeley Laboratory, Berkeley, California 94720.

Table 1 updates the calculated average organ doses accumulated by the members of this group to death or to 31 July 1977. Table 2 lists the causes of death, contributory causes, and durations of illnesses, as determined from death certificates. As far as we can determine, the internally deposited plutonium was not responsible for or related to the death of any of these cases.

Table 1. Calculated bone and liver doses

Case	Injection, μCi		Dates		Survival, days	Calculated α-ray doses, rads	
	^{238}Pu	^{239}Pu	Injection	Death		Bone	Liver
Cal-I	3.5	0.046	5/14/45	1/9/66	7545	580	1460
Cal-II	-	0.169	4/26/46	1/6/47	255	2.5	8.7
Cal-III	0.095	-	7/18/47	Living	10971[a]	11	27
Chi-I	-	0.40	4/26/45	10/3/45	160	1.5	4.2
Chi-II	-	5.9	12/27/45	1/13/46	17	3.5	8.5
Chi-III	-	5.9	12/27/45	Unknown	~ 170	24	70
HP-1	-	0.28	10/16/45	1/12/60	5201	33	85
HP-2	-	0.31	10/23/45	4/4/48	894	6.5	18
HP-3	-	0.30	11/27/45	Living	11569[a]	110	230
HP-4	-	0.30	11/27/45	4/29/47	518	5.4	13
HP-5	-	0.31	11/30/45	4/29/46	150	1.1	3.1
HP-6	-	0.33	2/1/46	Living	11503[a]	81	190
HP-7	-	0.39	2/8/46	10/27/46	261	3.5	8.6
HP-8	-	0.40	3/9/46	11/22/75	10850	140	290
HP-9	-	0.39	4/3/46	7/2/47	455	4.2	12
HP-10	-	0.38	7/16/46	6/2/57	3974	34	91
HP-11	-	0.40	2/20/46	2/26/46	6	0.06	0.16
HP-12	-	0.29	4/10/45	4/13/53	2925	20	52

[a]To 31 July 1977

Table 2. Causes of death as given on death certificates

Case	Sex	Date of birth	Age at injection	Age at death	Cause of death Contributory causes
Cal-I	M	10/11/86	58	79	Cardio-respiratory failure (12 hr) Pulmonary edema (2 day); arteriosclerotic heart disease with decompensation (1 wk)
Cal-II	M	5/26/41	4	5	Sarcoma of knee (9 mo)[a]
Cal-III	M	1/26/11	36	--	(Living)
Chi-I	M	3/20/77	68	68	Recurrence of cancer of chin and metastasis to lungs (1 yr)[a]
Chi-II	F	1/25/89	56	56	Metastatic carcinoma of aberrant breast tissue (5 mo)[a]
Chi-III	M	Unknown	Unknown	Unknown	(Lost to study after ~ 170 day)
HP-1	M	5/20/78	67	81	Bronchopneumonia (3 day) General arteriosclerosis (unknown interval)
HP-2	M	8/5/97	48	50	Hypertensive encephyalopathy (days) ? Cerebral hemorrhage (days); hypertensive cardiovascular disease (years);[a] hemophilia (years)[a]
HP-3	F	4/1/97	48	--	(Living)
HP-4	F	2/9/27	18	20	Cushing syndrome (6 yr)[a] Adenoma-pituitary (6 yr)
HP-5	M	1/4/89	56	57	Bronchopneumonia (2 day) Amyotrophic lateral sclerosis (2 yr, 6 mo)[a]
HP-6	M	11/14/01	44	--	(Living)
HP-7	F	5/7/86	59	60	Pulmonary failure Pneumonia; left base rheumatic heart; thyrotoxic goiter
HP-8	F	9/9/04	41	71	Malnutrition and electrolyte imbalance (several weeks) Carcinoma of larynx (2 yr); scleroderma; arteriosclerotic heart disease
HP-9	M	8/10/01	64	65	Terminal bronchopneumonia (days) Laryngeal edema and debilitation (days); dermatomyositis (years)[a]
HP-10	M	10/31/93	52	63	Arteriosclerotic heart disease[a]
HP-11	M	1/29/77	69	69	Bronchopneumonia (days) Cirrhosis of liver (years); congestion of viscera (days); arteriosclerosis (years)
HP-12	M	3/17/90	55	63	Heart failure (3 wk) Auricular fibrillation (3 wk)

[a] Diagnosed illness at time of injection.

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BLOOD CONTENT AND EXCRETORY PLASMA CLEARANCE OF PLUTONIUM 10^4 DAYS AFTER INJECTION*

J. Rundo and F. H. Ilcewicz†

Blood samples were taken from two subjects who had been injected intravenously with about 0.3 μCi ^{239}Pu(IV) as the citrate 10^4 days previously; the subjects were on a metabolic ward, and all urine and feces were collected. Plutonium was determined by alpha-spectrometric isotope dilution in the samples of excreta[1,2] and in separated plasma and red cells.[3] The concentration of ^{239}Pu in the plasma of each subject was four or five times that in the red cells; the ^{239}Pu in the latter was thought to be due to entrained plasma.

By dividing the daily excretion rate (urinary or urinary plus fecal) by the contemporary concentration in the plasma, we obtain the urinary or total excretory plasma clearance. The results summarized in Table 1 show that while the excretion rate is higher for the female than for the male, the reverse is true for the plasma clearance.

Concentrations of ^{239}Pu in excreta and in whole blood shortly after the injections were reported by Langham et al.[4] In order to calculate the excretory clearance at early times for comparison with our results, we had to make certain assumptions about the hematocrit and the density of plasma.[3] With these assumptions we obtained the results plotted in Figures 1 and 2. For both subjects our results are very similar to those at two or three days after injection, including the higher value for the male. The difference between the excretion rates of the two subjects may be a consequence of the higher degree of osteoporosis to be expected in a female although case 40-009 did not have clinical osteoporosis. However, this would not explain the lower excretory plasma clearance.

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†Deceased.

Table 1. Excretion rates and plasma clearance of ^{239}Pu 10^4 days after intravenous injection in two subjects.

Case number (sex)	40-009 (F)	40-012 (M)
Urinary excretion, pCi/day[a]	7.60 ± 0.21	4.68 ± 0.17
Fecal excretion, pCi/day[a]	3.17 ± 0.09	1.77 ± 0.01
Plasma concentration, pCi/L	97 ± 5	39 ± 2
Urinary plasma clearance, L/day[c]	0.078 ± 0.004	0.12 ± 0.01
Total excretory plasma clearance, L/day[c]	0.111 ± 0.006	0.17 ± 0.01

[a] 1 pCi/d = 37 mBq/d

[b] 1 pCi/L = 37 Bq/m^3

[c] 1 L/d = 0.001 m^3/d

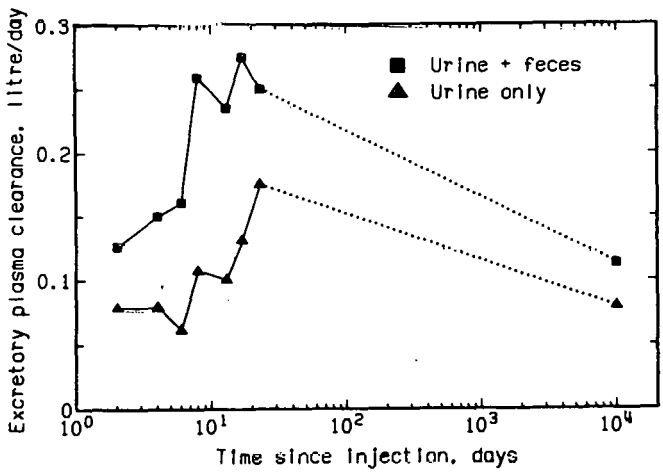

FIG. 1.--Excretory plasma clearance as a function of time on a logarithmic scale for case 40-009, a female, age 49 years at the time of the injection and 77 years 10^4 days later.

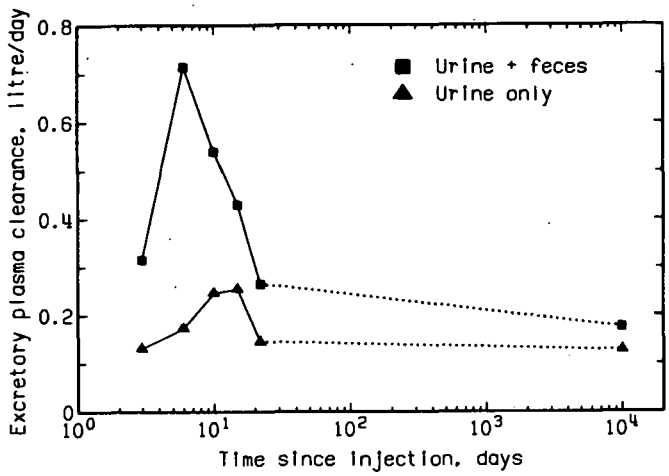

FIG. 2.--As for Figure 1, but for case 40-012, a male, age 45 years at the time of the injection and 73 years 10^4 days later.

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DISTRIBUTION OF INJECTED PLUTONIUM IN THE SKELETON AND CERTAIN SOFT TISSUES

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The burden and macrodistribution of plutonium in the skeleton and its concentration in certain soft tissues have been determined in the remains of a woman who received plutonium by injection. The method of analysis was alpha spectrometric-isotopic dilution. The skeletal burden was 54 ± 2% of the amount injected. As expected, the concentrations in bones that are primarily trabecular were much higher than those that are primarily cortical: The ratio of the concentration in the bodies of the thoracic vertebrae to that in the diaphysis of the tibia was 40. The bones of the skull and the clavicle are quite representative of the entire skeleton. The concentrations in soft tissue are a factor of about 100 lower than those in bone. The concentration in hair increases markedly with the distance from the scalp.

The burden and macrodistribution of plutonium in the skeletal remains of a woman who in 1945 received 11 kBq (0.3 μCi) of plutonium-239 by injection have been determined. At the time of injection she was suffering from Cushing's syndrome, hypertension, nephropathy with uremia, and osteoporosis; she succumbed to her illnesses in 1947 at age 20. The plutonium she received was tetravalent and in a citrate buffer medium. The subject, referred to in the early literature as HP-4,[1] has been assigned CHR case number 40-010. Preliminary results obtained by using both a xenon-filled proportional counter and radiochemical methods to determine plutonium in a few bones, as well as its distribution in a long bone, the tibia, were reported previously.[2,3]

The remains at the time of exhumation were in excellent condition, considering the 26 years of interment. There was no evidence of severe decay. The skin was dehydrated, shriveled, and discolored, but the flesh was moist. Examination showed that a complete autopsy had been performed and all organs as well as the brain had been removed.

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Sampling

Prior to dissection various tissue samples were taken: diaphragm, thigh muscle (quadriceps femoris) and skin, nerves (femoral), costal cartilage, trachea with larynx, scalp with hair, eyes, and teeth. At dissection the flesh was carefully removed from the bones, leaving as much as possible of the periosteum intact.

With the exception of the tibia, the samples of bone analyzed were from the left half of the skeleton. For the paired bones there were three types of samples: (1) groups of bones, such as the tarsals, (2) individual bones, such as the scapula, and (3) sections of individual bones, such as the tibia. The unpaired bones were cut in half, and there were two types of samples from these halves: (1) groups of sectioned bones, such as the processes of the cervical vertebrae and (2) individual bones, such as the sacrum. In the sampling of the skull, the paranasal and mastoid sinuses were removed, and the remainder of the skull was cut in half along the mid-saggital plane and segmented. The samples from the skull were (1) half the frontal bone and a large fraction of the left parietal bone, (2) the remainder of this parietal bone and half the occipital bone, (3) the left temporal squamous bone, and (4) half the mandible. The samples from the paranasal and mastoid sinuses were not analyzed.

Analytical Methodology

The method of analysis was alpha spectrometric-isotope dilution. The bone was ashed at 600°C, weighed, and a weight aliquot taken. This was dissolved in nitric acid, a known amount of ^{242}Pu and an excess of 9M hydrobromic acid were added and the solution was evaporated to incipient dryness. This insured isotopic exchange between the ^{239}Pu that was in the bone and the isotopic diluent, ^{242}Pu. The plutonium was separated from the other bone constituents, using the anion exchange procedure of Larsen and Oldham;[4] it was electrodeposited, using the technique reported by Kressin;[5] and the deposit was assayed in an alpha spectrometer. The plutonium recovery ranged from 75 to 90%; the resolution (FWHM) of the ^{239}Pu and ^{242}Pu peaks in

the spectra ranged from 10 to 13 fJ. The precision and accuracy of the values obtained are limited by the statistics of counting the ^{239}Pu. As a quality control procedure, one bone sample was analyzed repeatedly (10 times) during the period the determinations were made, and 25 other bone samples were analyzed in duplicate at separate times. The agreement amongst the values was, in all cases, within the statistics of counting.

For a few bones it was only possible to estimate their plutonium contents. The estimates were made in two ways: (1) from the values obtained in the analysis of a similar bone(s) and the wet weights of the bones, and (2) from the values obtained in the analysis of other sections of the same bone and the wet weights of the sections. An example of the former is the value for the sinuses where the value used was the mean of those obtained for the frontal-parietal, occipital-parietal, and temporal squamous bones. An example of the latter is the value for the diaphysis of the humerus where the mean of the values obtained for three sections was used to calculate the value for the other four sections. The amount of plutonium in those bones for which estimates were made was 6% of the skeletal burden. The assigned relative error of the estimated values was ± 25%.

Results and Discussion

The results obtained are given in detail in Table 1 and are summarized in Table 2. The relative error in the plutonium determinations was ±5% or less. Hence, the relative error for the values of relative plutonium concentrations, the last column in Table 1, is the same. The exceptions are those bones that were not analyzed in their entirety, e.g., the diaphysis of the femur (samples 1849–1855). In these cases the errors are given.

The amount of plutonium in the entire skeleton, 5.9 ± 0.2 kBq, was established by summing the amounts found in the unpaired bones and twice the amounts found in the paired bones. This is 54 ± 2% of the amount injected. The true error is probably larger than 0.2 kBq due to the fact that not all of the bones were analyzed, and lateral symmetry was assumed in the calculation of skeletal burden.

Table 1. Wet and Ash Weights and the Relative Plutonium Concentrations for the Bones of CHR 40-010.

CHR Sample No.	Bone	Weight, g		Concentration	Relative
		Wet[d]	Ash[e]	Bq/ g Ash[e,f]	Concentration[e,g]
1812	Frontal[a]-Parietal[b]	53.0	27.1	6.18	1.01
1813	Temporal squamous	16.2	8.41	5.80	0.95
1814	Occipital[a]-Parietal[b]	30.4	14.7	7.33	1.20
1815-1817	Sinuses[c]	48.8(0)	-	-	(1.09 ± .27)
1818	Mandible[a]	15.7	8.79	2.78	0.45
1819	Clavicle	18.3(0.65)	3.24	6.5 ± 0.5	1.05 ± 0.08
1820	Scapula	49.6	12.1	11.5	1.88
1821	Sternum[a]	23.8	1.69	31.7	5.19
	Ribs				
1822	1	5.5	1.06	13.2	2.16
1823	2	7.9	1.36	12.5	2.05
1824	3	9.2	1.65	12.3	2.01
1825	4	13.9	2.61	10.5	1.72
1826	5	16.1	2.76	12.7	2.08
1827	6	24.3	3.80	11.5	1.88
1828	7	22.7	4.00	11.9	1.95
1829	8	20.1	3.62	13.7	2.24
1830	9	17.4	2.78	14.5	2.37
1831	10	13.3	2.14	14.3	2.34
1832	11	6.2	1.03	12.6	2.07
1833	12	3.0	0.35	13.4	2.19
	Vertebrae, cervical[a]				
1834	Bodies	8.5(0.84)	1.11	23.5	3.85
1835	Processes	14.8(0.82)	2.78	11.0	1.80
	Vertebrae, thoracic[a]				
1836	Bodies	54.6	6.58	28.9	4.73
1837	Processes	35.2(0.90)	6.49	16.5	2.70
	Vertebrae, lumbar[a]				
1838	Bodies	39.5	4.46	26.2	4.29
1839	Processes	30.0(0.85)	4.54	12.4	2.03
	Innominate				
	Ilium				
1841	Anterior crest	13.2	1.93	29.9	4.89
1842	Posterior crest	15.2	1.84	32.8	5.37
1845	Sacroiliac area	44.2	6.30	20.9	3.42
1846	Body	76.1	15.96	15.1	2.47
1843	Ischium	34.8	5.52	21.9	3.58
1844	Pubis	21.4	3.27	24.8	4.06

Table 1 (continued)

CHR Sample No.	Bone	Weight, g Wet[d]	Weight, g Ash[e]	Concentration Bq/ g Ash[e,f]	Relative Concentration[e,g]
1840	Sacrum[a]	32.4	4.08	20.8	3.40
	Femur				
1847	Head and neck	22.5	5.46	13.1	2.14
1848	Greater trochanter	52.0	15.24	5.50	0.90
1849-1855	Diaphysis	163.2(0.37)	20.84	1.24 ± 0.18	0.20 ± 0.03
1856	Condyles	55.6	15.56	1.55	0.253
	Tibia				
1857	Proximal end	25.5	9.73	2.13	0.348
1858-1865	Diaphysis	103.5	51.74	0.65	0.106
1866	Distal end	13.0	4.88	1.22	0.200
	Fibula				
1867	Ends	13.9	3.29	1.83	0.300
1868	Diaphysis	26.4(0.21)	2.85	0.73 ± 0.16	0.12 ± 0.02
1869	Patella	13.8	2.95	1.85	0.303
	Feet				
1870	Tarsals	94.5	24.99	1.74	0.284
1871	Metatarsals	33.2	2.24	2.00	0.327
1872	Digits	17.8	2.90	1.63	0.267
	Humerus				
1873-74	Head	22.0	5.70	4.30	0.70
1875-1881	Diaphysis	55.2(0.84)	21.75	1.14	0.186
1882	Condyle	10.4	3.05	1.90	0.311
	Radius				
1883	Ends	8.7(0.70)	2.07	2.29	0.375
1884	Diaphysis	15.9(0.53)	4.17	0.73 ± 0.07	0.119 ± 0.011
	Ulna				
1885	Ends	13.0	4.43	1.34	0.219
1886	Diaphysis	17.9(0.46)	4.15	0.69 ± 0.08	0.112 ± 0.013
	Hand				
1887	Carpals	11.4(0)	-	-	(0.18)[h]
1888	Metacarpals	20.7	5.20	1.08	0.176
1889	Digits	20.5	4.81	1.30	0.212

[a] Half of these bones were analyzed.
[b] Part of the parietal was in sample 1812, the remainder in 1814.
[c] Paranasel and mastoid.
[d] When the whole of the sample was not ashed, the value in parentheses is the fraction that was.
[e] Where no error is given, the relative error in the value is less than ± 5%.
[f] 37 kBq/kg = 1 nCi/g.
[g] Relative concentration = concentration in bone/average concentration whole skeleton.
[h] Value assumed to be the same as that for metacarpals.

Table 2. Summary of Plutonium Distribution in the Skeleton of of CHR 40-010.

Bone(s)	Percent of Total Pu	Percent of Total Ash	Relative Pu Concentration
Frontal[a]-Parietal	5.7	5.6	1.0
Occipital[a]-Parietal	3.6	3.0	1.2
Temporal squamous	1.6	1.7	0.9
Sinus[a]	5.4 ± 1.3	5.0	1.1
Mandible[a]	0.8	1.8	0.4
Skull[a]	17.1 ± 1.7	17.1	1.00
Innominate	23.4	7.2	3.3
Vertebrae[a]	20.7	6.2	3.3
Ribs	11.8	5.6	2.1
Scapula	4.7	2.5	1.9
Clavicle	1.1	1.0	1.1
Sacrum[a]	2.9	0.8	3.6
Sternum[a]	1.8	0.4	4.5
Axial	66.4	23.7	2.8
Femur	8.5	20.3	0.4
Patella	0.2	0.6	0.3
Tibia	2.0	13.7	0.15
Fibula	0.5	3.4	0.15
Foot	1.8	6.2	0.3
Humerus	2.0	7.2	0.3
Radius	0.4	2.2	0.2
Ulna	0.4	2.8	0.15
Hand	0.5	2.7	0.2
Appendicular	16.3	59.1	0.27

[a] Values given are for half of this (these) bone(s).

As expected for a radionuclide that deposits on bone surfaces, the plutonium concentrations in bones that are primarily trabecular were significantly higher than those that are primarily cortical. This is evident when comparing one part of a bone with another, one bone with another, and a group of bones with another. The ratio of the concentration in the femur head and neck (1847) to that in the diaphysis (samples 1849–1855) is 10.7; the sacrum (1840) to clavicle (1819) ratio is 3.24; and the torso to limbs ratio (Table 2) is 10.4.

There are other comparisons that show the relationship between plutonium concentration and bone type. For the long bones the ratio of the concentration in the ends to that in the diaphysis ranges from 2.0 for the ulna through 2.8 for the tibia to 4.0 for the femur. Of the long bones the ulna has the lowest percentage of trabeculae and the femur the highest. For the vertebrae the ratios of the relative concentrations, bodies to processes are 2.1, 1.8, and 2.1, respectively, for the cervical, thoracic, and lumbar groups.

In considering which bone should be taken in autopsy as being representative of the concentration in the skeleton, the clavicle would appear to be superior to any other. A rib has been the bone of choice in the past as it is readily available. This could continue to be the case, but as seen in Table 2 the mean concentration is twice that of the average concentration in the skeleton. A comparison of the values for the individual ribs (1822–1833) shows that which rib is taken is not important.

The conclusions that have been drawn from these data are qualified by the facts that the subject was suffering from a disease characterized in part by osteoperosis and that as a consequence of age her skeleton may still have been metabolically active.

Two teeth were analyzed, the left lower canine and a left second lower premolar. The plutonium concentrations (in ash) were 31 and 57 Bq/kg, respectively. These values are more than two orders of magnitude lower than the mean concentration in bone ash, 6110 Bq/kg.

The results obtained for soft tissues and hair are given in Table 3. With the exception of hair, the concentrations are based on the weights of the tissues at the time the remains were dissected. A significant fraction of the

hair was cut into sections at 20 mm intervals beginning at the scalp, and the sections were analyzed for their plutonium content. Prior to analysis, the hair in each section was washed with detergent solution until microscopic examination showed the hair to be free of foreign material; it was then rinsed with water, air-dried, and weighed.

Table 3. Plutonium in Soft Tissues of CHR 40-010

Tissue	Concentration, Bq/kg[a,b]
Diaphragm	17.7
Muscle	7.8
Nerves	4.3
Eyes	46
Trachea and larynx	35
Skin	16.2
Costal cartilage	10.3
Nails	5.1
Hair section	
Proximal (20-40 mm)[c]	3.0 ± 0.7
Middle (140-160 mm)[c]	5.0 ± 0.8
Distal (260-280 mm)[c]	22 ± 3

[a] Where no error is given, the relative error is less than ± 5%.

[b] 37 Bq/kg = 1 pCi/g

[c] Distance from the scalp.

As expected, the concentrations of plutonium in the soft tissues are much lower than those in bone. The mean concentration in wet bone, 1770 Bq/kg, is about two orders of magnitude higher than the values given in Table 3.

As seen in Table 3, the concentrations of plutonium in the distal and middle sections of the hair are factors of 7 and 1.7, respectively, higher than the concentration in the proximal section. The shape of the curve obtained when the concentration in each section is plotted against the distance from the scalp, appears to reflect the concentration in blood as a function of time. Furthermore, it appears that the distal section was formed within a few months

of the time of injection. The product of the growth rate for standard man, 0.5 mm/day, and the time between injection and death, 518 days, is 260 mm. However, analysis of all the data has not been completed.

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INGESTION OF ^{241}Am SOURCES INTENDED FOR DOMESTIC SMOKE DETECTORS: REPORT OF A CASE

J. Rundo, W. D. Fairman,† M. Essling, and D. R. Huff

A woman accidentally swallowed two sources of ^{241}Am of the type used in domestic smoke detectors; the nominal activity of each source was 2.5 μCi, and they were not voided until the 16th and 24th days after intake. A little less than 1% of the total activity of the two sources was found in the fecal material after removal of the sources and ^{241}Am was detected in the urine until the second source had been voided. The systemic burden was estimated to have been very much less than 1.5% of the activity released from the sources, and this was not significant from the point of view of radiological protection.

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†Occupational Health and Safety Division.

MEASUREMENTS OF ^{241}Am IN VIVO AT LONG TIMES AFTER INHALATION*

R. E. Toohey and M. A. Essling

Introduction

The retention and distribution of ^{241}Am in the body of an industrial worker have been determined on several occasions extending over a period of ten years. The subject visited Argonne twice in 1967, and again in 1973, 1975, and 1977. Some of the results of the earlier visits have been described elsewhere.[1-3] The exact date, nature, and duration of the exposure are unknown, but the most probable exposure was inhalation of the insoluble dioxide. Our previous reports assumed the date of exposure to be sometime in 1966, but Brodsky and Horn suggested a date of September 1965 based on the recollection of the worker and on early urinary excretion data.[4] For reference purposes in comparing our in vivo counting data with the excretion data, 1965 will be taken as the date of exposure. For the period from September 1967 to December 1974, the subject underwent chelation therapy at another center. The therapy consisted of almost weekly injections of DTPA and excretion of over one-half of the initial body burden occurred during the period of therapy.

Measurements of Body Radioactivity

The body content in 1967 was determined by measurements made with a 292-mm diameter by 102-mm thick NaI(Tl) crystal suspended 350 mm above the bed on which the supine subject lay. In 1973–1977, a 292-mm diameter by 12.7-mm thick NaI(Tl) crystal was used in the 1.5-m arc geometry. The results are shown in Table 1.

The lung content in 1967 was estimated as that amount (16%) of the total activity which could not be accounted for by consideration of available bone surface area in various compartments of the body. In 1973–1977 the lung content was determined directly by measurements made over the chest

*Summary of a paper presented at the 23rd Annual Meeting of the Health Physics Society, Minneapolis, Minnesota, June 19–23, 1978.

Table 1. ^{241}Am body content (μCi) of case 30-041

Date	1967	1973	1975	1977
Whole body	1.8 ± 0.9	0.99 ± 0.13	0.85 ± 0.09	0.88 ± 0.07
Lung	0.29 ± 0.15	0.18 ± 0.02	0.18 ± 0.02	0.16 ± 0.02
Bone	1.5 ± 0.9	0.81 ± 0.13	0.67 ± 0.09	0.72 ± 0.07

with a 180-mm diameter xenon-methane filled proportional counter. The proportional counter is sensitive to the Np L x rays, centered at about 18 keV (2.9 fJ), emitted following the α decay of ^{241}Am as well as to the 60-keV (9.6 fJ) γ ray. The counter, when viewing the anterior chest, is relatively insensitive to ^{241}Am deposited in the spine and posterior portions of the ribs, and there is insufficient bone surface area (and therefore activity—see below) in the anterior ribs and sternum to account for the observed counting rates at 18 and 60 keV. The counter was calibrated by the method of calculating an "effective soft tissue thickness,"[5] and also from measurements of point sources of ^{241}Am distributed throughout a simple chest phantom containing a low-density region to simulate lungs. The estimated lung contents are shown in Table 1. It must be noted that some of the activity in the "lungs" may in fact reside in the thoracic lymph nodes.

Table 1 also gives the amount of activity contained in the skeleton, simply calculated as the difference between "whole body" and "lung." In 1967 no estimate was made of activity in the liver, and there was no evidence of activity in the liver in 1973 and 1975. However, for 1977, some activity that was observed in the liver (see below) has not been subtracted from the value for bone given in Table 1.

The patient received chelation therapy beginning soon after his second visit to Argonne in 1967, and this therapy continued until the end of 1974. The total amount of ^{241}Am excreted during therapy was 1.2 μCi (44.4 kBq),[6] indicating that the estimated total content of 1.8 μCi (66.6 kBq) in 1967 was about 20% too low.

Comparison of the results for 1975 and 1977 shows that the retention half-time of ^{241}Am in the subject's body after the cessation of chelation therapy cannot be determined from these numbers. However, some estimates can be made based on the excretion data.[4,6] Between the body radioactivity measurements in 1973 and the cessation of therapy, approximately 0.05 μCi (1.85 kBq) was excreted, and during the year between the end of therapy and our measurements in 1975, another 0.005 μCi (185 Bq) was excreted.[6] Our observation of no decrease in body content from 1975 to 1977 is explained by the fact that the expected excretion during this period was only 0.01 μCi (370 Bq), much less than the standard errors of the body radioactivity measurements.

The excretion (without chelation) of 0.005 μCi (185 Bq) per year from a body burden of 0.85 μCi (31.5 kBq) indicates a whole-body retention half-time of 110 years, in excellent agreement with the ICRP task group proposal of 100 years.[7] The retention half-time for material in the lungs (Table 1) from 1973 to 1977 is 24 years. Similar retention times have been observed elsewhere in cases of ^{241}Am inhalation.[8]

Distribution Measurements

The gross distribution of ^{241}Am in the body of the subject was determined by a series of profile scans. A lead collimator with a 25-mm wide slit was placed on a 292-mm diameter by 102-mm thick NaI(Tl) detector underneath the supine subject, with the slit perpendicular to the long axis of the body. The counting rate obtained from the 60-keV (9.6 fJ) γ ray was measured at 100-mm intervals down the body, and the interval was reduced to 50 mm when the counting rate varied rapidly from one position to the next. Similar measurements were performed in 1975 and 1977, and the three profiles are superimposed in Figure 1, along with a scale drawing of the skeleton for reference. The presence of ^{241}Am in the skeleton and its association with trabecular bone are immediately apparent.

No gross changes in the distribution of activity are evident from the three scans. A focused-slit collimator was used in 1977, and the counting rates have been scaled to reflect the greater sensitivity of that arrangement.

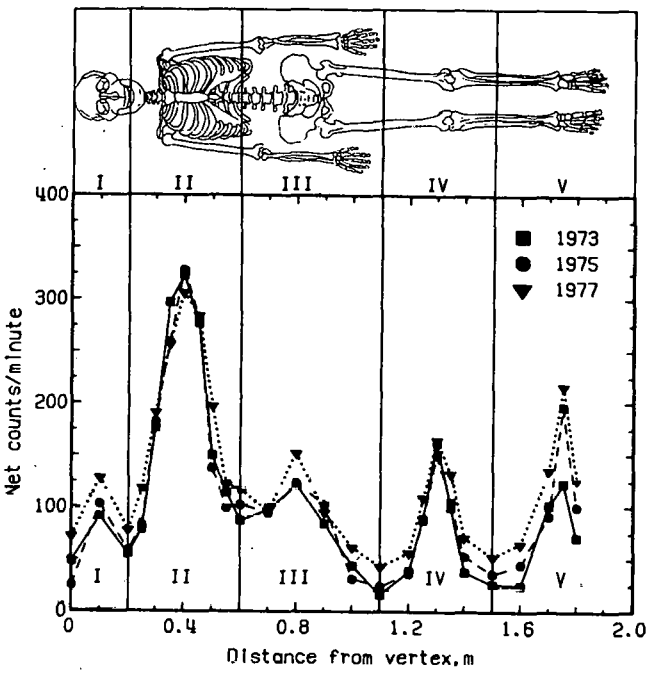

FIG. 1.--Longitudinal profile scans of case 30-040 in 1973, 1975, and 1977.

In addition, the focused-slit collimator has slightly poorer resolution than the single slit, accounting for the increased counting rates in the regions about 1.10 and 1.50 m from the vertex. The twofold increase in counting rate observed at 1.75 m from the vertex is statistically significant, but unexplained. It is possible that since there is so little soft tissue between the bones of the feet and the detector, small increases in activity are inordinately magnified.

The body was considered to be divided into five regions as shown in Figure 1 in order to analyze the distribution. Table 2 gives the percentage of the total counts found in each region for the three scans. These data indicate a slow migration of activity from the thorax (region II) to the other regions, probably due to the dissolution of material in the lungs and its deposition in bone. The deposition of ^{241}Am on bone surfaces is evident throughout the entire skeleton. Table 3 shows the percent of bone mass, bone surface area, and total counting rate (for the 1977 data) in each of the five regions. (As shown in Table 2, the percentages of total counting rate by region had not changed drastically from 1973 to 1977.) There is excellent agreement between the fraction of total surface area and the fraction of total observed counting rate in four of the five regions, but region II gives a significantly greater

Table 2. Percentage of total counts observed, by region, in case 30-041.

Region[a]	1973	1975	1977
I	11	10	13
II	42	41	37
III	21	21	20
IV	15	15	16
V	11	13	15

[a]See Figure 1.

Table 3. Comparison of counting rate and bone distribution for case 30-041 from the 1977 data.[a]

Region	% total bone mass	% total bone surface	% total counts	% non-lung counts
I	15	12	13	15
II	26	26	37	23
III	31	33	20	25
IV	7	17	16	19
V	13	12	15	18

[a]This analysis is similar to that of May,[1] but the values for bone mass and surface area are taken from Reference Man.[9]

percentage of the total counts than would be expected for the percentage of bone surface area it contains. The final column in Table 3 gives the percent of total counts in each region after correcting the counts in region II for the activity in the lungs determined as described above. The correlation coefficient of corrected counting rate and bone mass (columns 5 and 2 of Table 3) is +0.79, and the correlation coefficient of corrected couting rate and bone surface area (columns 5 and 3) is +0.96. This latter number is significant at the 99.5% level (for three degrees of freedom) and this offers striking confirmation of the deposition of ^{241}Am on bone surface.

A transverse profile scan of the thorax was made with the slit parallel to the spine and centered at 0.4 m from the vertex, the location of the highest counting rate in the longitudinal scans. This scan is shown in the uppermost portion of Figure 2. In order to remove the contribution of material in the spinal column from the scan, another scan was made 0.2 m further down the body. At this point the detector was away from the major portion of lung tissue, but still under part of the rib cage. This second scan is shown in the middle portion of Figure 2 and the difference between the two scans is plotted in the lower portion. The asymmetry between the left and right sides of the thorax confirms the presence of activity in the lung, since the right lung is larger than the left. The asymmetry also indicates that no major fraction of the lung content resides in the tracheobronchial lymph nodes, which cluster about the bifurcation of the trachea.

In order to investigate the presence of activity in the liver, the proportional counter was placed over the right side of the abdomen at a distance of 0.65 m from the vertex and tilted 45° ($\pi/4$ rad) from the vertical so as to be roughly parallel to the surface of the body. The counting rate obtained was then compared with that observed when the counter was in a similar position over the left side of the abdomen. The data are shown in Figure 3. No evidence of activity in the liver was observed in 1973, due to the removal of ^{241}Am by the chelation therapy. A higher ratio of counting rates was observed in 1975, and a still higher value in 1977. The latter data indicate that in the absence of chelation therapy, ^{241}Am is being deposited in the liver after its removal from bone or lung. Because of the severe attenuation by tissue of the photons emitted from ^{241}Am in the liver, it is extremely difficult to calibrate the counter. A crude estimate of the activity in the liver in 1977 is 25 nCi (0.925 kBq). The build-up of activity in the liver may justify the resumption of chelation therapy in order to remove this easily accessible material, but this is a medical decision.

Conclusions

1. Chelation therapy with DTPA, even though not begun until two years after exposure, removed over one-half the initial body burden.

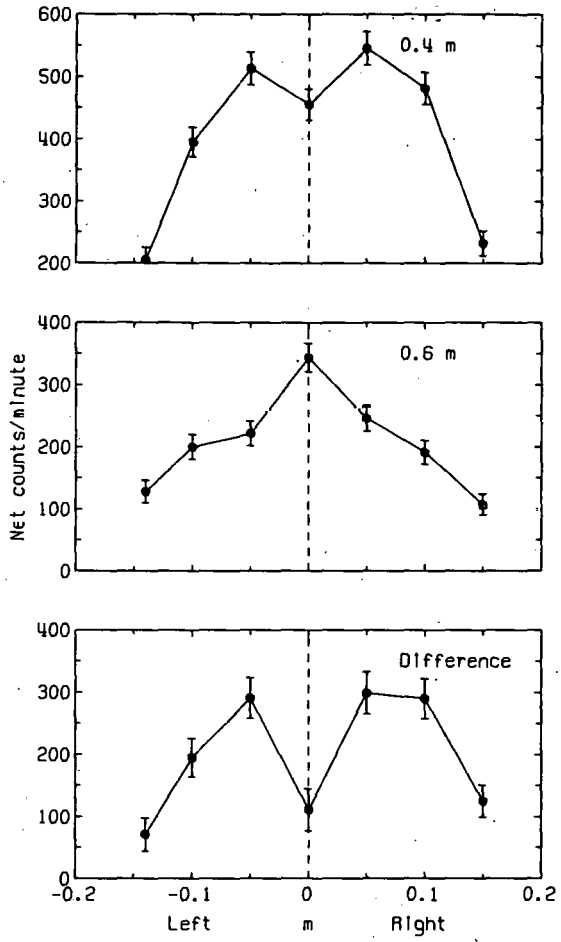

FIG. 2.--Transverse profile scans of case 30-041 in 1977.

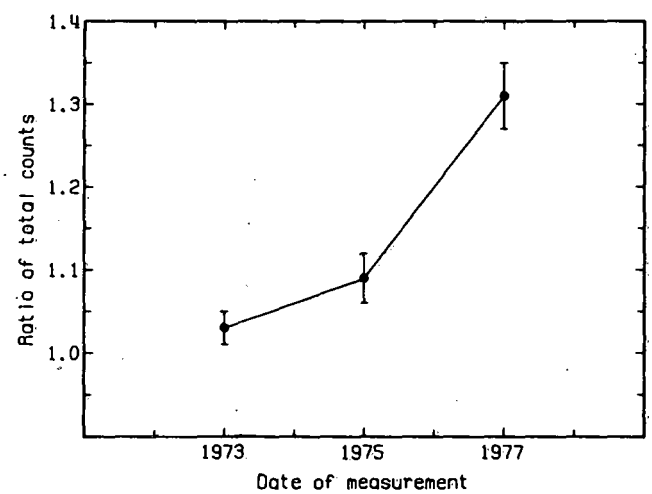

FIG. 3.--The ratio of counting rates obtained from the proportional counter over the liver and over the left side of the abdomen.

2. No extensive redistribution of activity within the body occurred over the interval of no chelation therapy.

3. Approximately one-third of the initial body burden remains in the skeleton, and its distribution indicates deposition on bone surface. The slow decrease in skeletal burden may indicate that much of the original deposition has been buried by bone remodeling.

4. Between 5 and 10% of the initial body burden remains in the lungs, including possible deposition in the thoracic lymph nodes.

5. After the cessation of chelation therapy, there was a buildup of activity in the liver, probably translocated from the lungs and also from the skeleton during bone remodeling. This buildup may justify the resumption of chelation.

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METHOD FOR THE DETERMINATION OF ^{241}Am IN URINE

R. P. Larsen, R. E. Walkup,* and R. D. Oldham

An alpha spectrometric-isotopic dilution method has been developed for and applied to the determination of ^{241}Am in urine. The limit of detection at the 95% confidence level is 200 μBq when a 24-hr counting period is used. A known amount of ^{243}Am is added to the sample, the organic constituents are destroyed by wet ashing, and the americium is separated from the other inorganic constituents. The americium is deposited on a planchet by electroplating, and the planchet is assayed in an alpha spectrometer to determine the ^{241}Am to ^{243}Am activity ratio. Americium recovery in the procedure ranges from 80 to 90%.

Introduction

We have reported previously that there was a significant amount of ^{241}Am in the skeletal remains of an individual who had received plutonium by injection in 1945.[1] It was established that this was due to the decay of ^{241}Pu which had been a minor constituent (0.04%) of the plutonium injected. It was also established that the ^{238}Pu to ^{239,240}Pu activity ratio in this plutonium was 0.005. We have also reported the plutonium urinary excretion rates for two other individuals who had received injections of this plutonium.[2] It was apparent that about 75% of the ^{241}Pu they received had decayed in vivo to ^{241}Am and that it was very probably incorporated in their skeletons. Therefore, it appeared that analyses of their urine for ^{241}Am would provide information on the late urinary excretion of americium acquired exclusively by decay of plutonium in vivo. An evaluation of the methods in the literature showed that none of them provided the requisite sensitivity and/or specificity for the analysis of these urine samples, nor could these methods be readily modified to do so. Hence, a new method was developed.

*Participant in the Undergraduate Research Program, Center for Educational Affairs.

Separation of Americium from Urine

The initial steps of the new method are similar to those in the methods developed for the determinations of ^{232}Th[3] and ^{228}Th[4] in bone. A known amount of ^{243}Am is added (about 50 mBq), the sample is wet ashed, about 2 g of calcium is added, and calcium oxalate precipitated by the addition of oxalic acid. This precipitate carries more than 95% of the americium. The calcium oxalate is dried and converted to calcium carbonate at 600°C.

Americium is separated from the added calcium and the other sample constituents that are carried by the calcium oxalate by a procedure that uses both selective precipitation and anion exchange. The calcium carbonate is dissolved in nitric acid, a small amount of iron (III) is added, and ferric hydroxide is precipitated by the addition of carbonate-free gaseous ammonia. The ferric hydroxide is dissolved in strong hydrochloric acid, the solution is passed through a Dowex-1 anion exchange column, sodium bisulfate solution is added to the eluant, the solution is evaporated to dryness, the residue is dissolved in water, and the americium is electrodeposited from this solution.

Interferences

Three radionuclides (^{238}Pu, ^{222}Rn, and ^{228}Th) have principal alpha particles of very similar energies to those from ^{241}Am. Of these, only ^{228}Th is not separated from americium by the above procedure. However, the total amount of ^{228}Th in urine, in reagents, and from the surface of glassware is usually very low (about 1 mBq), and a satisfactory correction for this amount can be made. The sources in urine are the result of dietary uptake of both ^{228}Th and its precursor, ^{228}Ra (5.7 yr).

The amount of ^{228}Th that is present on the planchet is established by initiating the alpha spectrometric assay at least 7 days after the electrodeposition of americium and measuring the amount of its daughter, ^{224}Ra, that has grown in before and during the assay. The number of counts due to ^{228}Th can then be calculated from the ^{224}Ra counts, the time from electrodeposition to initiation of assay, and the length of assay. The effect that this correction has on the accuracy of the ^{241}Am value depends on the numbers of counts in

the ^{228}Th-^{241}Am peak and their activity ratio. For a relative error in the ^{241}Am value of ±20% or less, the ^{228}Th to ^{241}Am activity ratio must be ≤4 if the number of counts due to ^{228}Th and ^{241}Am is 1000, and ≤0.7 if the number of counts is 100.

When the accuracy requirements for the ^{241}Am value cannot be met by applying the ^{224}Ra based correction for ^{228}Th, the analysis must be repeated using a modified procedure. The eluant from the anion exchange column is made 8 M in nitric acid, and this solution is passed through another anion exchange column. Thorium is absorbed and americium is not. Sodium bisulfate solution is added, the solution is evaporated to dryness, the residue is dissolved in water, and the americium is electrodeposited.

Limit of Detection

The factors that limit the sensitivity of this method are primarily the background of the detector, the counting geometry, and the time that can be devoted to the activity measurement. The background of a typical detector in the energy region of interest, 8.68 to 8.81 pJ, is one count per day; the geometry is about 35%. Blank values obtained by analyzing urine samples were not significantly different from the detector background, 1 count/day. As this corresponds to the counting rate from 70 μBq of ^{241}Am on the planchet, the limit of detection is about 200 μBq for a 24-hr observation of activity. The accuracy of the method is limited only by the ^{241}Am counting statistics.

Application

The method was tested by analyzing urine samples to which a known amount of ^{241}Am had been added. The recoveries ranged from 80 to 90%, and the resolution (FWHM) of the ^{241}Am and ^{243}Am peaks ranged from 10 to 12 fJ.

This method was used to analyze urine samples from the two individuals who had received plutonium by injection. The americium recoveries and the quality of the spectra were the same as those reported above. The decontamination factor for plutonium, derived from the counts due to $^{239,240}Pu$ in the 8.12 to 8.25 pJ region of the spectra and the amount of $^{239,240}Pu$ known to be

present in the samples, was about 300. There were no peaks due to ^{224}Ra in any of the spectra and hence no interference from ^{228}Th.

Although a complete analysis of the data remains to be made, it can be stated that the activity ratios of $^{239,240}Pu$ to ^{241}Am in the urine samples are within a factor of two of the estimated activity ratios of these nuclides in the skeletons of the individuals.

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RETENTION AND DISTRIBUTION OF ^{75}Se FOLLOWING INTRAVENOUS INJECTION OF ^{75}Se-SELENOMETHIONINE

M. A. Essling, R. E. Toohey, and D. R. Huff

The retention and distribution of ^{75}Se in two Argonne employees who received intravenous injections of ^{75}Se-selenomethione for diagnostic purposes have been followed for approximately three years post injection. The retention is consistent with Lathrop's equation, and the gross distribution of activity in the body does not change significantly with time.

Introduction

^{75}Se, injected as selenomethionine, is used as an imaging agent for diagnostic studies of pancreas function. The isotope decays by electron capture with a physical half-life of 120 days and emits photons of 136 keV and 265 keV (21.8 and 42.5 fJ, respectively) in approximately half of the disintegrations. The amount injected is usually 250 μCi (9.25 MBq).

Lathrop et al.[1] followed the retention of ^{75}Se in 24 subjects in vivo out to 923 days post injection and determined the distribution of ^{75}Se in selected tissues by analysis of samples collected at surgery or autopsy. The low background in our shielded counting rooms enables us to follow the retention for longer periods and also to determine the gross distribution in vivo by means of profile scans.

We have made a series of γ-ray measurements on two Argonne employees, each of whom received a single intravenous injection of 250 μCi (9.25 MBq) of ^{75}Se-selenomethionine. The two subjects, designated by CHR case numbers 30-053 and 30-054, have been followed for periods of 1108 and 922 days post injection, respectively.

Retention Measurements

The total body contents of ^{75}Se in the two subjects were determined by counting the 265-keV γ ray with a 152-mm diameter by 203-mm long NaI(Tl) detector (the "log" crystal) in a 1.5-m arc geometry. As the amount of ^{75}Se retained became too low to be measured in the arc, the same detector was used

in the reclining chair geometry. The detector was calibrated with a small source of ^{75}Se in solution, which was standardized by R. B. Holtzman.

The retention of ^{75}Se as percent of the injected dose in the two subjects is shown in Figure 1. Lathrop's equation is shown as a solid line, and the dashed lines represent the equation plus and minus one standard deviation. The equation is a three-component exponential, with 13.1 ± 2.4% of the injected dose exhibiting a biological half-life of 0.55 ± 0.12 days, 44.3 ± 6.9% with a half-life of 46 ± 11 days, and 41.9 ± 7.6% with a half-life of 220 ± 31 days. Both cases exhibit a retention pattern which lies within the limits of one standard deviation from the equation.

Distribution Measurements

The gross distribution of ^{75}Se in the subjects was determined in vivo by a series of longitudinal profile scans. A lead collimator with a 30-mm wide slit was placed over the face of a 292-mm diameter by 102-mm thick NaI(Tl) detector. The face of the detector was 170 mm below the bed on which the supine patient lay. The counting rate from the 265-keV γ ray was measured at 50-mm intervals along the length of the body. Three scans were made on each subject over a period of about 300 days, at which time the amount of ^{75}Se in the body had dropped to a level that would have required an excessively long counting time in each position.

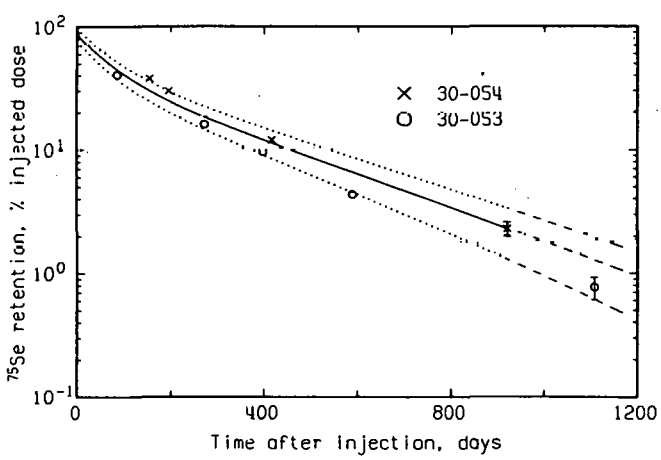

FIG. 1.--The retention of ^{75}Se by two subjects, expressed as percent of the injected dose. Lathrop's equation is shown as a solid line.

The scans on the two subjects are shown in Figures 2 and 3, along with idealized representations of the body for reference. The scans show first that there is no great difference in relative distribution in either subject over the period of the scans, and second that the distributions in the two subjects are very similar, except for a slightly higher concentration of activity in the head of case 30-054. Lathrop et al.[1] found that muscle contained the largest fraction of ^{75}Se at times ranging from 1 to 600 days post injection, with 34% of total body ^{75}Se in muscle at 1 day and 56% at 600 days. Our scans show that the observed counting rate varies in accord with the distribution of muscle along the body. For example, the peak at 80% of the height from the vertex is due to calf muscle, which is well separated from thigh muscle by the knee.

One particularly interesting feature of the distribution is found in the region between 40 and 60% of the height from the vertex, where the counting rate varied in sawtooth fashion from one position to the next. This region is shown in expanded form for case 30-054 in Figure 4. Note that the variations in counting rate are large compared to the statistical uncertainty of any point, and that the pattern remains very similar between 154 and 196 days post injection. It was first thought that this might reflect a greater concentration of

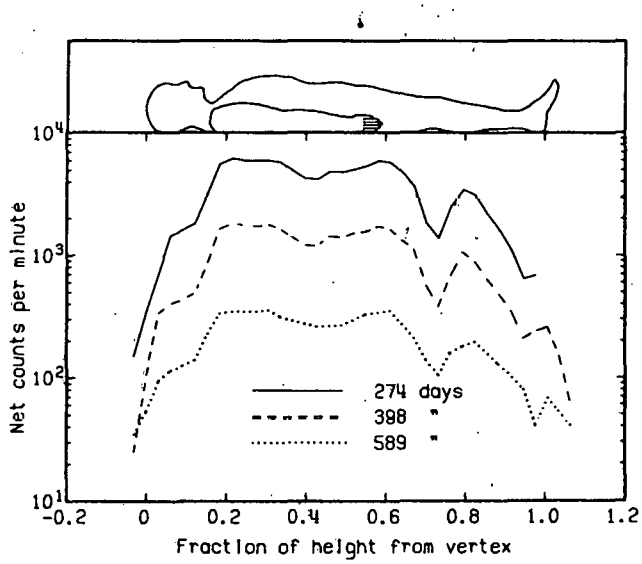

FIG. 2.--Longitudinal profile scans of case 30-053 at 274, 398, and 589 days post injection.

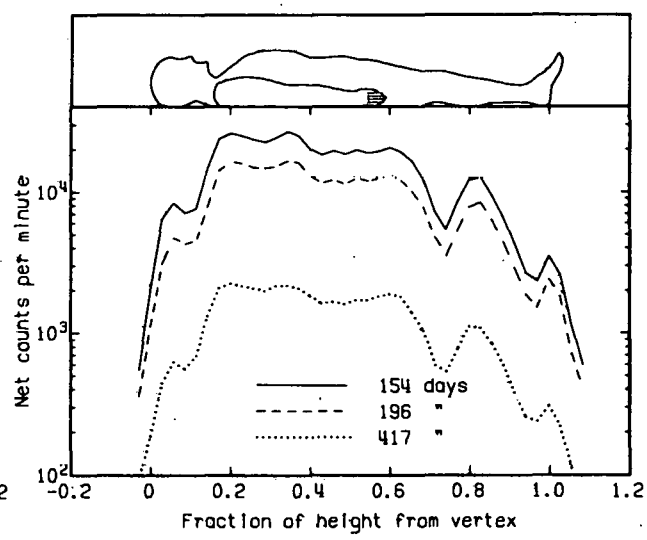

FIG. 3.--Longitudinal profile scans of case 30-054 at 154, 196, and 417 days post injection.

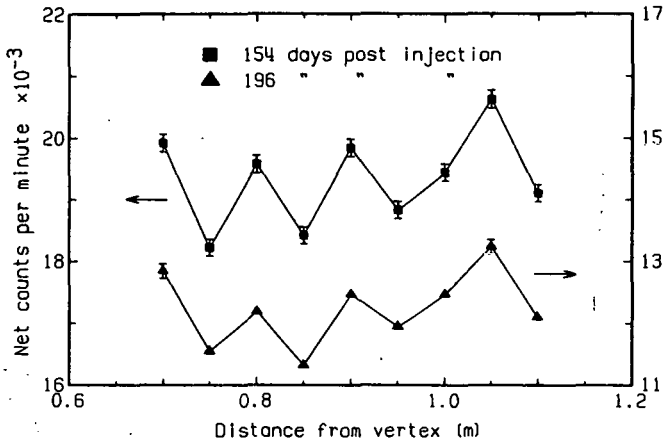

FIG. 4.--Longitudinal profile scans of case 30-054 at 154 and 196 days post injection (expanded scale).

^{75}Se in the cartilaginous discs between the lumbar vertebrae than in the vertebrae themselves. However, since the observed variations extend to 60% of the height from the vertex (about midthigh), a concentration of ^{75}Se in cartilage cannot account for the variations observed throughout the region. Since the field of view of the collimated detector is about 100 mm wide at the mid-plane of the body, future measurements of another injection case with a narrower slit and at closer intervals would be required to resolve this question.

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LATE EXCRETION OF ^{231}Pa AND ITS DECAY PRODUCTS*

R. B. Holtzman and F. H. Ilcewicz†

Excretion rates of members of the naturally-occurring actinium series (4n + 3) were determined for a 69-year-old man (case 30-021) who had acquired ^{231}Pa ($T_{\frac{1}{2}}$ = 32500 yr) about 45 years prior to this investigation. The body contents determined by whole body counting were approximately 71 nCi of ^{231}Pa, 51 nCi each of 22 yr ^{227}Ac and 18.2 day ^{227}Th, and 39 nCi of 11.4 day ^{223}Ra.[1] All urinary and fecal samples were collected, as were representative daily diets during the 10 days the subject was maintained on a metabolic ward.‡ The samples were wet ashed with nitric acid. After separation the ^{231}Pa and ^{227}Th were electrodeposited on stainless steel and alpha counted. The ^{223}Ra was coprecipitated with $BaSO_4$, mounted, and alpha counted. Actinium-227 was determined from the ^{227}Th daughter after a long growth period.

The excretion rates varied quite dramatically over the 10-day study period as shown in Figures 1 and 2 for the urinary and fecal routes. For the urinary route the ^{231}Pa and ^{227}Ac showed patterns similar to that of the calcium, peaking at about the third, fifth, and tenth days. The ^{226}Ra data were similar, but less certain. The fecal excretion patterns also exhibited large variations with similarities in their patterns for ^{231}Pa, ^{227}Ac, and calcium. Although there was an approximately 100-fold difference in the fecal excretion rates of ^{226}Ra and ^{223}Ra, the patterns were very similar for these isotopes. Except for the peak on day 8, their patterns were also similar to those of the other nuclides and of calcium (see Table 1).

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*Summary of a paper presented at the Twenty-Second Annual Meeting of the Health Physics Society, Atlanta, Georgia, July 3–8, 1977. Health Phys. 33, 673–674 (1977).

†Deceased.

‡Massachusetts Institute of Technology Clinical Research Center, Cambridge, Mass. Supported by a grant (RR-88) from the National Institutes of Health.

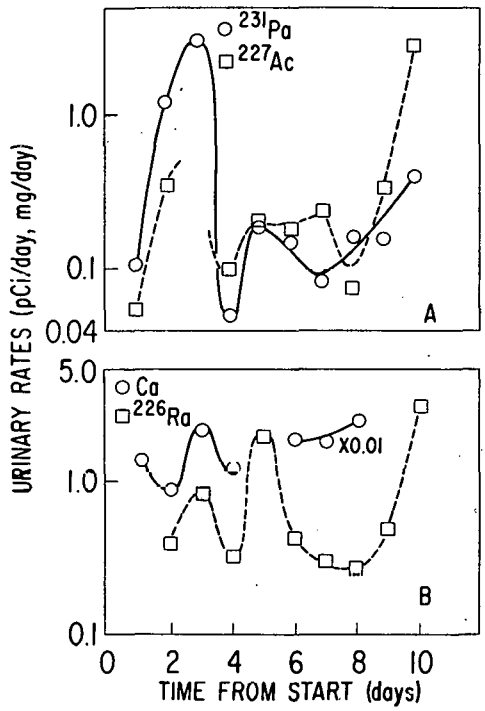

FIG. 1.--Urinary excretion rates of ^{231}Pa and ^{227}Ac (A) and calcium and ^{226}Ra (B) for case 30-021 over a 10-day study period.
(ANL Neg. 149-77-33)

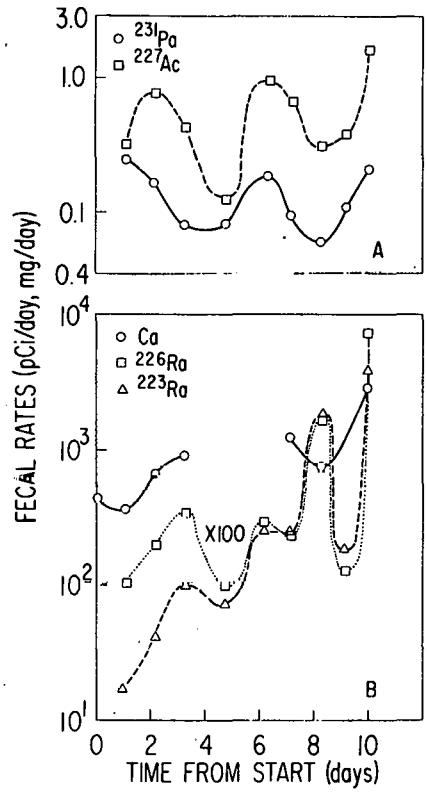

FIG. 2.--Fecal excretion rates of ^{231}Pa and ^{227}Ac (A) and of calcium, ^{226}Ra, and ^{223}Ra (B) for case 30-021 over a 10-day study period.
(ANL Neg. 149-72-32)

These results are consistent with the assumptions of ICRP Committee II[2] for the dosimetry of daughter-free ^{231}Pa that the maximum permissible body burden (MPBB) should be 100 nCi.

The activity ratios between the various nuclides in vivo, e.g., $^{227}Ac/^{231}Pa$, are consistent with their measured excretion rates, if we assume them not to have varied greatly with time. The initial intake appears to have been mainly ^{231}Pa, with less than about 10% ^{227}Ac. The low excretion rate and short half-life of the ^{227}Th support the assumption of radioactive equilibrium with the ^{227}Ac,[1] and the disequilibrium between the ^{223}Ra and the ^{227}Th is consistent with the high (2%/day) excretion rate of the former nuclide.

Table 1. Mean excretion rates and coefficients of excretion for ^{231}Pa and its decay products.

Nuclide	Body content, nCi[a]	Excretion rates, pCi/day		Coefficient of excretion, %/yr			$T_{\frac{1}{2}}$, yr
		Urine	Feces	Urine	Feces	Total	
^{231}Pa	71	0.55	0.13	0.28	0.067	0.35	200
^{227}Ac	51	0.47	0.55	0.34	0.39	0.73	95
^{227}Th[b]	51	0.16	0.08	0.11	0.06	0.17	400
^{223}Ra	39	—	760	—	700	700 (2%/day)	0.010
^{226}Ra[c]	~ 0.0	0.94	6.5	—	—	—	—

[a] 1 nCi = 37 Bq.

[b] Estimated from 1972 data.

[c] Corrected for environmental intake.

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2. International Commission on Radiological Protection, Report of Committee II on Permissible Dose for Internal Radiation, Health Phys. 3, 1–380 (1960).

NORMAL DIETARY LEVELS OF ^{226}Ra, ^{228}Ra, ^{210}Pb, AND ^{210}Po FOR MAN*

R. B. Holtzman

A review of the literature and the results of some recent measurements were presented on the levels in man's diet of the naturally-occurring radionuclides ^{226}Ra, ^{228}Ra, ^{210}Pb, and ^{210}Po. The mean intakes for "standard" U.S. diets† for these nuclides are shown in Table 1. Intakes in other countries

Table 1. Mean dietary intake of radionuclides

Nuclide	Intake, pCi/day	Range, pCi/day
^{226}Ra	1.4	0.7–2.4
^{228}Ra	1.1	1.0–1.2
^{210}Pb	1.4	1.3–1.6
^{210}Po	1.6	1.3–1.6

are similar to those in the U.S., but in localized populations the ^{226}Ra intake may be 8 or more pCi/day. The contents of ^{226}Ra in diets chosen by individuals ranged from 0.4 to 7 pCi/day. The few data on ^{228}Ra show intake of this nuclide to be about 80% that of ^{226}Ra, except in monazite areas where intakes of up to 160 pCi/day ^{228}Ra are reported, which may be 50 to 100 times that of ^{226}Ra. Drinking water contributes less than 5% of the daily intake, except in special areas. For ^{210}Pb higher levels than in the U.S. have been noted for Germany and the U.S.S.R., about 4.5 to 6 pCi/day, and the Japanese diet contains about 17 pCi/day. Persons in the Arctic who consume reindeer or

*Summary of paper presented at the Symposium on the Natural Radiation Environment III, Houston, Texas, April 24–28, 1978.

†"Standard" diet is a sampling of a typical diet, the per capita intake based on the total food consumption in the region of interest divided by the population size.

caribou meat may ingest ^{210}Pb at the rate of 10 to 40 pCi/day. Normal dietary levels of ^{210}Po are about 20 to 30% higher than those of ^{210}Pb, except in the Arctic where intakes of 60 to 100 pCi ^{210}Po/day may be 5 to 10 times those of the ^{210}Pb. The levels of these nuclides in classes of foods were compared to show that the higher levels observed in certain diets are due to the levels in particular foods.

The ^{210}Pb intakes in Japan produce skeletal dose rates estimated to be more than twice those in the U.S. A discussion was presented on the use of dietary intake of these nuclides for estimation of metabolic parameters, such as intestinal absorption.

PROGRESS REPORT ON A STUDY OF CONTAMINATION OF THE HUMAN FOOD CHAIN BY URANIUM MILL TAILINGS PILES*

R. B. Holtzman, P. W. Urnezis, A. Padova,† and C. M. Bobula, III

A study is in progress to estimate the contamination of the human food chain by uranium, ^{230}Th, ^{226}Ra, ^{210}Pb, and ^{210}Po originating from tailing piles associated with uranium ore processing mills. Rabbits, cattle, vegetables, and grass were collected on or near two uranium mill sites. For controls, similar samples were obtained from areas 20 km or more from the mining and milling operations. For the on-site rabbits the mean ^{226}Ra concentrations in muscle, lung, and kidney of 5.5, 14, and 15 pCi/kg wet, respectively, were substantially higher than those in the respective tissues of control animals (0.4, 1.5, and 0.2 pCi/kg). The levels in liver did not differ significantly between the groups. The concentrations in bone (femur and vertebra) were about 9000 and 350 pCi/kg ash for the on- and off-site animals, respectively. The levels of ^{210}Pb and ^{210}Po did not differ significantly for a given tissue between the two groups.

For cattle the results are incomplete, but the existing data indicate that the concentrations of radionuclides do not differ greatly between those grazed near the pile and the controls, except that the ^{210}Pb concentration in the liver of an exposed animal is greater than that of the control. Vegetables from a residential area on a mill site contained substantially greater concentrations of ^{226}Ra and ^{210}Pb than those reported for standard New York City diets.

Grass and cattle dung from land irrigated by water containing 60 pCi/L ^{226}Ra from uranium mines had concentrations of ^{226}Ra and ^{210}Pb 40 and 7 times, respectively, those in control samples.

It is estimated that doubling the normal concentrations in meat and vegetables of uranium and daughter products could increase the dose equivalent rates to the skeletons of persons consuming these foods by 30 mrem/yr.

This study was undertaken to obtain data on the possible contamination of the human food chain by radionuclides in tailing piles from uranium ore processing mills. These data are to be used for generic environmental impact

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statement on uranium milling operations (UMOGEIS) sponsored by the U.S. Nuclear Regulatory Commission. The nuclides of interest are the long-lived members of the ^{238}U decay series, namely ^{238}U, ^{234}U, ^{230}Th, ^{226}Ra, ^{210}Pb, and ^{210}Po shown in Figure 1. Other naturally occurring nuclides, such as in the ^{235}U and ^{232}Th series, were not considered because of their low abundance in ores from the regions considered.

The long-lived nuclides may enter the food chain through soil and inhalation of dust by animals and deposition of dust on vegetation. The ^{210}Pb and ^{210}Po are of particular interest because they are decay products of ^{222}Rn, and so may form in the atmosphere in addition to entering it from resuspension of dust particles. Moreover, for the general public the ^{210}Po deposited in the human skeleton, and supported by the long-lived ^{210}Pb, produces from 25 to 50% of the natural radiation dose to the skeleton.[1]

The usual sources of these nuclides for man are shown in Table 1. The early members of the series are acquired mainly by ingestion through food and water, with about 5 to 10% from inhalation of dust. For ^{210}Pb it has been estimated that about one half is acquired from ingestion and half from inhalation.[1] About 5% or so may be derived from internal sources, such as ^{226}Ra deposited in the body and ^{222}Rn dissolved in body fluids. The sources of ^{210}Po are more uncertain, but it has been estimated that normally about 90% originates from the ^{210}Pb deposits in the skeleton, and the remainder from ingestion and inhalation.[2]

A survey was made of contamination levels of the human food chain to see how much contamination occurred under extreme conditions, particularly with respect to the nuclides ^{226}Ra, ^{210}Pb, and ^{210}Po.

Experimental Methods

Specimens of the food chain and some related samples listed in Table 2 were collected in the Grants, New Mexico uranium mining district (shown on the map of northwestern New Mexico in Figure 2), and from the vicinity of two mills, Kerr-McGee Nuclear Corporation and the Anaconda Company.

Table 1. Nuclides of interest and sources for man

Nuclide	Sources	Fraction from source, %
^{238}U	Ingestion	
	Food	95
	Water	5
	Inhalation (dust)	-
^{230}Th	Ingestion	
	Food	~100
	Inhalation (dust)	-
^{226}Ra	Ingestion	
	Food	95
	Water	5 (95)[a]
	Inhalation (dust)	<5
^{210}Pb	Ingestion	
	Food	45
	Water	5
	Inhalation	
	^{210}Pb	45
	^{222}Rn daughters	2
	Internal	
	^{226}Ra	2
	^{222}Rn	2
^{210}Po	Ingestion	
	Food and Water	5
	Inhalation	
	^{210}Po	5
	Internal (^{210}Pb)	90

[a] In certain regions potable water containing high concentrations of radium may contribute 95% or more of the radium intake.

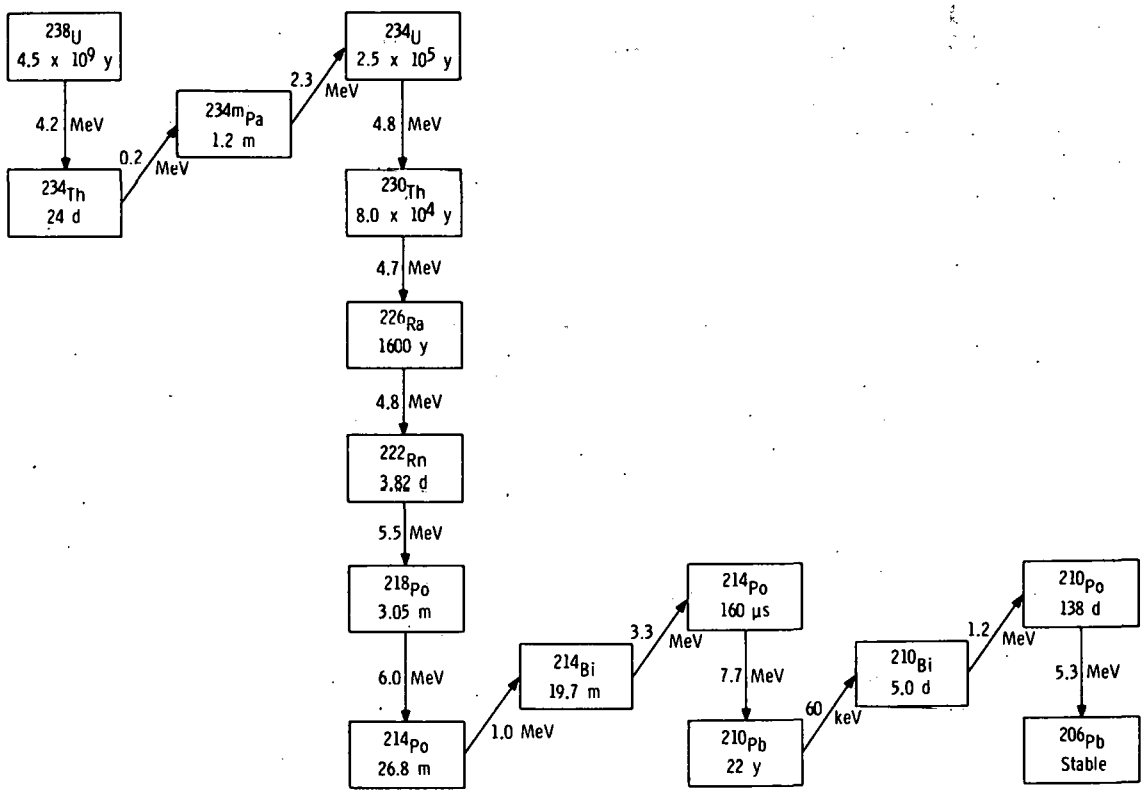

FIG. 1.--^{238}U series decay scheme. (ANL Neg. 149-78-177)

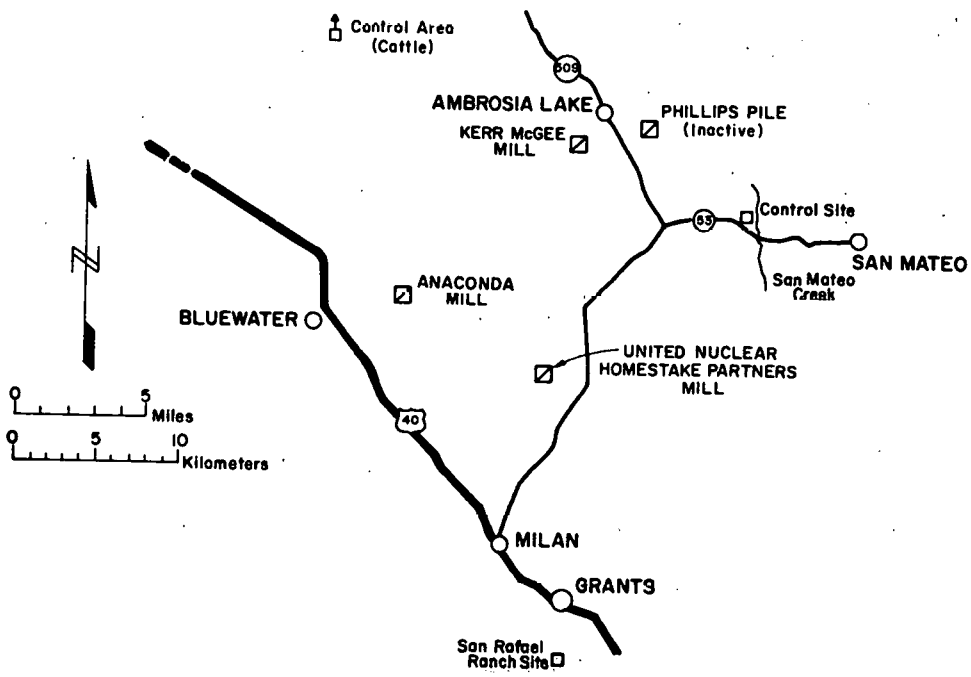

FIG. 2.--Location of sites studied in the Grants, New Mexico uranium mining district. (ANL Neg. 149-78-308)

Table 2. Specimens for analysis

Sample	Description	Number
Jack rabbit (blacktail)	Lepus californicus, Anaconda tailings pile, collected 7/77	5
"	Same as above, but from a control area, Sanchez Ranch near San Rafael, N.M., collected 7/77	3
Cottontail rabbit	Sylvilagus audoboni (?), Anaconda site, collected 7/77	1
Cattle	Grazed in open pasture adjacent to Anaconda property. North, east and west, collected 7/77	2
	1. Black cow, age ~10 yr 2. Red cow, age ~3 yr	
Cattle (controls)	Grazed about 20 miles north of Anaconda mill, collected 7/77	2
	3. Brown, age 9 4. Black, age 8	
Vegetables	From gardens on Anaconda site residential area, collected 8/77	5
Grass	From various places, collected 6/77:	
	mine-water irrigated, Kerr-McGee	2
	control, Kerr-McGee	2
	control, San Mateo Creek	2
	Anaconda mill site	2
	Phillips pile, vicinity at 75, 200 and 1000 metres	3
Cow dung	Uranium mine water irrigation at Kerr-McGee site, Ambrosia Lake, N.M., collected 6/77	5
Cow dung	Control from San Mateo Creek, collected 6/77	5

The Anaconda site is particularly useful for this study because only milling is done here, and a company village is situated within a few kilometers of the mill. The residents raised vegetables in an otherwise barren region. The Kerr-McGee site is less suitable because several mines are very close to it, thus making it difficult to separate mine contamination from that due to the tailings.

Wild animals hunted and eaten by the local residents were represented by jack and cottontail rabbits (Lepus californicus and Sylvilagus audoboni (?)) collected on the site of the Anaconda mill within a few hundred meters of the tailing pile, as shown in the circles on the map, Figure 3. Domesticated animals were represented by two head of cattle that had grazed most of their lives in a field adjacent to the Anaconda property, up to the fence on the north-eastern section as shown in Figure 3.

Where possible, control samples were collected from areas similar to the tailings area, but at least 15 km from the mining and milling areas (Figure 2). We obtained two head of cattle raised about 40 km from the Anaconda site and two rabbits from a ranch near San Rafael, N.M., about 30 km away.

Also collected were specimens of vegetables grown in the Anaconda housing area (see Figure 3) and grass and other plants from the Kerr-McGee and Anaconda sites. Some grass was also collected from the vicinity of the Phillips (now United Nuclear Corporation) tailing pile near Kerr-McGee. While this pile is inactive, it is similar to the active ones, but it provides more extreme conditions, such as a lush grass growth near the pile and cattle grazing within a kilometer or so of the leeward side of the pile.

After dissection, the samples were processed, either by freeze-drying or heat-drying at 100°C. They were then ashed in a low-temperature asher which used oxygen plasma, or wet-ashed in nitric and perchloric acids. These procedures prevent volatilization of ^{210}Po, the decay product of the ^{210}Pb. The ^{226}Ra was determined from an aliquot fraction of the dissolved samples by the radon emanation method of Lucas.[3]

The ^{210}Pb and ^{210}Po were determined by first converting an aliquot of the dissolved sample to hydrochloric acid solution from which the ^{210}Po was

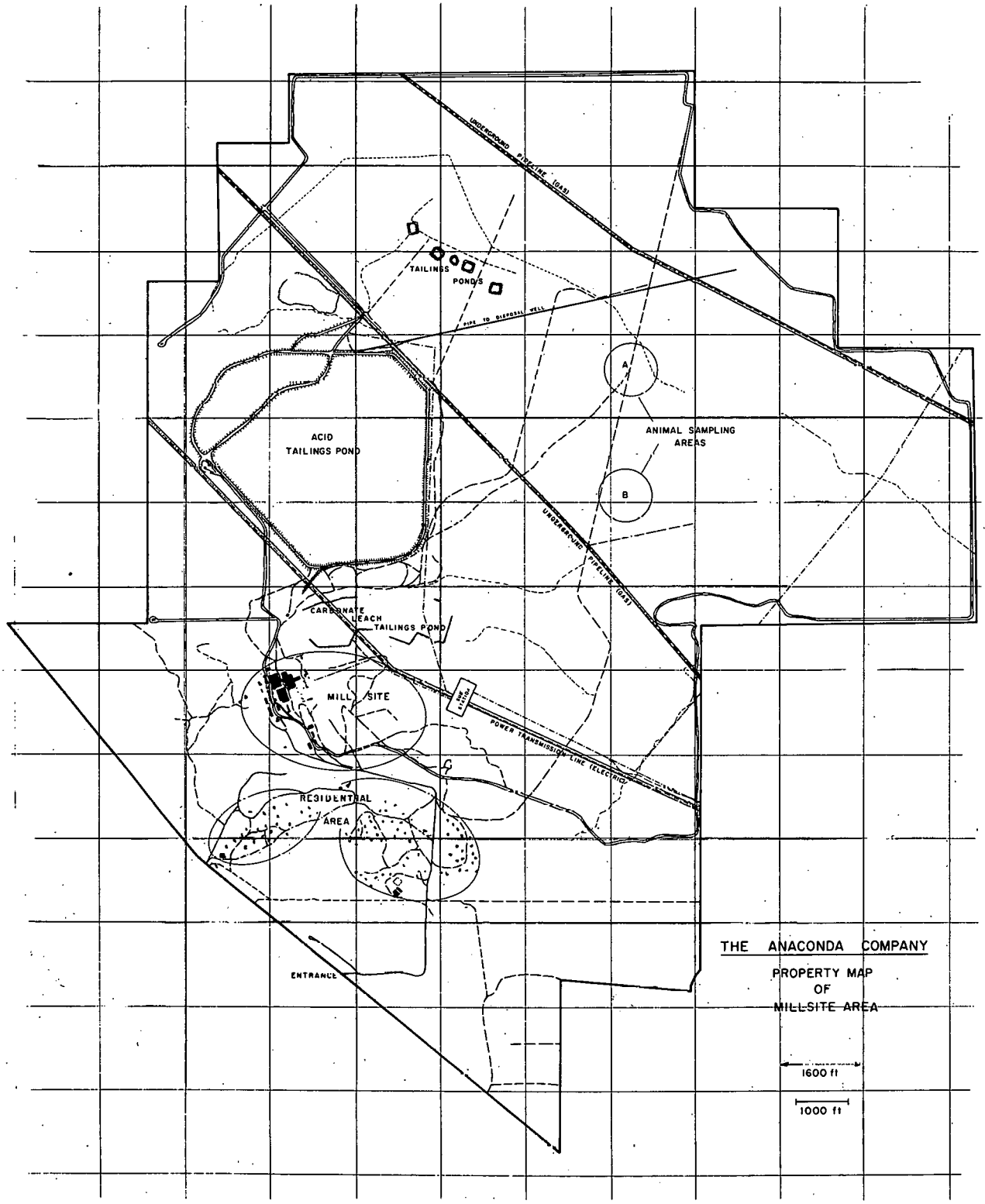

FIG. 3.--Map of Anaconda Company mill site showing cattle pasture, areas where rabbits were captured (circles), and the residential area. (ANL Neg. 149-78-293 Rev. 1)

plated onto a silver disk. The disk was then counted in an alpha counter. After several months wait to allow the 138-day ^{210}Po to grow in from the ^{210}Pb, the solution was replated and counted. The Bateman equations of radioactive decay and growth were then used to calculate the amounts of both the ^{210}Po and ^{210}Pb at the time of collection.[4]

Calcium was also determined, to estimate the mineral content of bones and as a measure of the mineral content of other biological materials, by flame atomic absorption spectrophotometry with a Perkin Elmer Model 303 instrument.

Results and Discussion

The available results of analyses of the rabbit tissues are shown in Table 3 for the individual rabbits, the cottontail (CT1A) along with the three jack rabbits (JR1A, JR2A, and JR3A) from the Anaconda mill site. The last two columns are data from the control animals (JR1C and JR2C) from the off-site ranch.

The concentrations of ^{226}Ra in muscle are higher by factors of 2 to 30 in the on-site than in the off-site animals, while in kidney they are 10 or more times greater. Differences are not apparent in liver because one control value is low and the other high, and the differences for ^{210}Pb and ^{210}Po in on-site and control animals are not significant. These differences and similarities are seen in the mean values in Table 4, where the mean concentrations of ^{226}Ra are much greater in the on-site animals, except in liver. Because of the wide variability and few samples the means are not necessarily statistically different ($P > 0.05$). However, the fact that all or most of the levels in the exposed animals are greater than those in the controls is a strong indication of significant differences.

The ^{226}Ra levels of about 9000 pCi/kg ash in the bones of the on-site rabbits are greater than in the controls by a factor of 25, while the levels of ^{210}Pb and ^{210}Po are the same in the two groups. Relative to the concentrations of ^{226}Ra, those of ^{210}Pb and ^{210}Po are much lower in the on-site compared to the control animals.

Table 3. ^{226}Ra, ^{210}Pb, and ^{210}Po (pCi/kg wet)[a] and Ca (mg/kg wet) in rabbit tissues from the mill and control sites

		Mill site[b]				Control site[b]	
Tissue	Nuclide	CT1A	JR1A	JR2A	JR3A	JR1C	JR2C
Muscle	^{226}Ra	16.5	1.26	3.18	0.95	0.32	0.48
	^{210}Pb	13.7	10.6	1.1	2.9	3.4	6.6
	^{210}Po	-	45.0	8.1	5.2	3.0	9.0
	Ca	386	199	77.0	36.7	56.3	-
Liver	^{226}Ra	42.3	22.3	8.3	3.5	0.46	28.9
	^{210}Pb	88.7	31.1	80.1	16.3	21.4	65.5
	^{210}Po	22.0	22.8	47.4	-	2.4	56.7
	Ca	319	436	-	232	276	338
Lung	^{226}Ra	27.8	9.6	19.2	0.28	0.96	2.84
	^{210}Pb	21.8	11.0	13.9	48.7	10.8	37.2
	^{210}Po	-0.36	-0.75	1.86	-	-1.33	26.2
	Ca	532	262	118	201	-	295
Kidney	^{226}Ra	29.7	15.4	10.8	4.45	0.37	-1.20
	^{210}Pb	56.1	268	79.9	55.0	21.6	46.4
	^{210}Po	-12.4	-	300	97.1	-	180
	Ca	329	303	206	185	261	345
Femur	^{226}Ra	6100	13800	10500	5650	235	460
	^{210}Pb	1820	547	527	725	656	954
	^{210}Po	820	422	483	—	238	620
Vertebra	^{226}Ra	5830	12100	10900	6130	240	453
	^{210}Pb	2670	1200	1640	496	1830	1210
	^{210}Po	796	-	-	226	29.3	494

[a] 1 pCi/kg = 37 mBq/kg.

[b] CT, cottontail rabbit; JR, jack rabbit.

Table 4. Summary of nuclide concentrations in rabbit tissues (Mean ± S.E.)

Tissue	Nuclide	Mill Site pCi/kg wet[a]	Control Site pCi/kg wet
Muscle	^{226}Ra	5.5 ± 3.7	0.40 ± 0.10
	^{210}Pb	7.1 ± 3.0	5.0 ± 1.6
	^{210}Po	19 ± 13	6 ± 3
Liver	^{226}Ra	19 ± 9	15 ± 14
	^{210}Pb	54 ± 27	43 ± 22
	^{210}Po	31 ± 8	30 ± 27
Lung[b]	^{226}Ra	14 ± 6	1.5 ± 0.5
	^{210}Pb	24 ± 9	24 ± 13
Kidney[b]	^{226}Ra	15 ± 5	0.2 ± 0.2
	^{210}Pb	115 ± 51	34 ± 12
		pCi/kg ash	pCi/kg ash
Femur	^{226}Ra	9020 ± 1900	350 ± 110
	^{210}Pb	900 ± 310	800 ± 150
	^{210}Po	1040 ± 470	430 ± 190
Vertebra	^{226}Ra	8700 ± 1600	346 ± 100
	^{210}Pb	1500 ± 450	1500 ± 300

[a] 1 pCi/kg = 37 mBq/kg.

[b] Additional analyses of ^{210}Pb are in process.

While the concentrations of ^{226}Ra in bone varied between animals, they are essentially identical for femur and vertebra in a given animal. This similarity indicates that the intake of this nuclide throughout the life of the animal was fairly constant. On the other hand, the ^{210}Pb concentrations are generally lower in the femur than in the vertebra (except for JR3A). This difference indicates a recent increase in ^{210}Pb intake, which deposits more readily on cancellous than in compact bone. The ^{210}Pb concentrations did not appear to be correlated with those of ^{226}Ra.

The calcium concentrations in the soft tissues vary considerably between animals, but they do not differ significantly between the on-site and control groups.

The data on the four head of cattle shown in Table 5 are much less complete, but some preliminary conclusions may be reached. The concentrations do not appear to differ greatly, except that the ^{210}Pb and ^{210}Po levels in the livers of the on-site animals appear to be much greater than those of the one control measured. The Ra concentrations in the control kidney are greater than in one on-site animal, but lower than in the other. The teeth of control animals have somewhat lower radium concentrations than the single bone sample from on-site, but these differences are not significant. The ^{210}Pb levels in the femurs of the exposed animals are appreciably greater than in the teeth of the controls.

The nuclide concentrations in the vegetable samples from the Anaconda residential area are shown in Table 6, and as noted, control samples are not available. However, these samples have high values compared to those reported for foods from New York,[5-7] San Francisco,[5] and other cities in the U.S.[1] Both the ^{226}Ra and ^{210}Pb are in appreciably greater concentrations in the mill site than in the New York samples. This may be the contamination from the tailings or mill, but it may also be due to the different growing conditions for different regions.

Results from grass samples collected from possible contaminated sites are shown in Table 7. The activities in the control samples from Kerr-McGee and San Mateo Creek have concentrations similar to those reported in the

Table 5. Nuclide concentrations in cattle tissues from the mill and control sites

Tissue	Nuclide	Mill Site[a] pCi/kg wet[b] Cow 1	Mill Site[a] pCi/kg wet[b] Cow 2	Control Site[a] pCi/kg wet[b] Cow 3	Control Site[a] pCi/kg wet[b] Cow 4
Muscle	^{226}Ra	3.2	1.2	-	1.5
	^{210}Pb	9.5	1.8	-	2.6
	^{210}Po	36	59	-	330
Liver	^{226}Ra	0.87	1.54	1.5	
	^{210}Pb	214	73	6	
	^{210}Po	-	340		
Kidney	^{226}Ra	220	2.6	25	
		pCi/kg ash		pCi/kg ash	
Teeth	^{226}Ra	-	-	2300	1400
	^{210}Pb				1400
Femur	^{226}Ra	-	2880		
	^{210}Pb	1620	11400		

[a] See Table 2 for description of animals.

[b] 1 pCi/kg = 37 mBq/kg.

Table 6. ^{226}Ra and ^{210}Pb concentrations in vegetables

Sample	^{226}Ra pCi/kg wet	^{210}Pb pCi/kg wet
Anaconda Mill Site		
Tomato	1.1	0.87
Peas	1.4	3.5
Green beans	5.9	11
Carrots	5.9	2.3
Beets	10.3	3.6
Mean	4.9	4.3
New York City[5]		
Fresh vegetables	0.50	1.1
Canned vegetables	0.65	0.44
Root vegetables	1.4	0.21
Dry beans	1.1	3.0

Table 7. ^{226}Ra and ^{210}Pb concentrations in grass

Sample	^{226}Ra pCi/kg dry	^{210}Pb pCi/kg dry
Exposed		
Anaconda pile	—	3100
Phillips pile – 75 m	22700	630
Phillips pile – 200 m	—	5000
Phillips pile – 1 km (dune)	—	1020
KM – Pond, Top	—	5500
Controls		
KM – Top	500	220
San Mateo Creek	200	—

midwestern United States.[8] From the Anaconda site the ^{210}Pb concentration is about 15 times that in the control. Those from the inactive Phillips pile, which is similar in structure to the active ones, but from a basic carbonate process, show high ^{226}Ra, but low ^{210}Pb, concentrations near the pile. The ^{210}Pb concentration increases in the sample at about 200 m from the pile and then drops in the 1-km sample, even though the latter sample was growing on a sand dune formed from the tailings. The low value of ^{210}Pb in the grass from the 75-m location may be due to damp soil which allows faster growth and less accumulation time for airborne ^{210}Pb.

The grass near the Kerr-McGee pond storing mine water, which contains about 60 pCi/L ^{226}Ra also has a high concentration of ^{210}Pb.

We also obtained samples of cattle dung from animals grazed on land irrigated by mine water on the Kerr-McGee site (Table 8). The mean concentration of ^{226}Ra was about 50 times, and that of the ^{210}Pb 7 times those of the control samples. Although these are not actual grass samples, the results are probably representative of the relative nuclide intake of the animals in different locations.

Discussion

These results indicate that under the somewhat extreme conditions studied (samples from close to the tailings), contamination is indeed present. On-site rabbits had higher concentrations of ^{226}Ra in most tissues than did the control animals, although the ^{210}Pb and ^{210}Po levels were not significantly greater. The most significant differences were the ^{226}Ra concentrations in bone. Bone levels represent long term intake, and if we assume a constant rate of intake, they are probably proportional to the levels in muscle and other soft tissues. Bone is probably also both a reservoir and a sink for the mineral constituents in the soft tissues, which are maintained at levels proportional to those in bone. Measurement of bone levels may be a more accurate measure of the relative nuclide content of the soft tissues of animals sampled because the activities in bone are much higher and easier to determine. The soft tissues probably represent the most important ingestion pathway for man, although if

Table 8. ^{226}Ra and ^{210}Pb concentrations in cattle dung from the area irrigated with water from uranium mines (Kerr-McGee mill site)

	Pond area			Control (San Mateo Creek)	
Sample	^{226}Ra pCi/kg dry	^{210}Pb pCi/kg dry	Sample	^{226}Ra pCi/kg dry	^{210}Pb pCi/kg dry
1	36000	20000	1	894	2740
2	45600	19000	2	853	2860
3	—	52200	3	—	5090
4	—	20100	4	—	2920
5	—	14600	mean	870	3400 ± 560
mean	41000	25000 ± 7000			

the bones are used for soup, etc., some of the nuclides may be leached from the bones and become an additional source.

While the data on cattle are less complete and definitive than those on rabbits, they indicate some level of increased concentrations of ^{210}Pb and ^{210}Po, but not of ^{226}Ra (there may be a marginal increase of ^{226}Ra in bone, as noted earlier). The differences in cattle between exposed and control groups seem to be much smaller than in rabbits, which is to be expected, since the conditions were less extreme. The closest approach of the cattle to the tailings is about 2 km, and they can graze several kilometers away from the site boundary fence. Moreover, in winter they may consume fodder not grown near the tailing pile.

The vegetables grown on site appear to contain higher concentrations of both ^{226}Ra and ^{210}Pb than do other sources, such as those from New York City.

The relative concentrations of the nuclides in cattle dung indicate that cattle grazed on land irrigated by mine water may have much higher levels of both radium and ^{210}Pb than others. While these results are not directly applicable to these studies, data on such cattle could be important for more

accurately estimating transfer coefficients. Such data are essential for determining the values of the parameters for the system models describing distribution in the environment of nuclides from the tailing piles.

Dose to Man

The effect of contamination on dose to man may be estimated from the normal nuclide intake levels and dose rates. Only the uranium (which contributes to the total dose[1]), ^{226}Ra and ^{210}Pb intakes will be considered. The relatively short half-life of the ^{210}Po (138 days) allows it to build up much less than ^{210}Pb in the body for a given constant intake. On the other hand, when considering the dose rates, the ^{210}Po, which emits alpha particles, contributes about 95% or more of the dose equivalent rate from the ^{210}Pb series.

The total dose equivalent rates to man from natural sources are listed in Table 9, which shows that internally deposited radionuclides contribute about 30% of the total dose to soft tissues (as represented by gonads) and about 70% to bone (specifically to the osteocytes).[1] It is assumed that the quality factor, QF, is 10 for alpha particles. Of the dose rates from internal emitters in Table 10, the heavy elements (mainly ^{210}Po) contribute 90% of the dose to the skeleton and about 25% of that to the gonads; ^{40}K contributes most of the remainder. These dose rates are from dietary intakes of about 0.7 pCi/day of U and 1.4 pCi/day each of ^{226}Ra and ^{210}Pb. However, only about one half the bone dose from ^{210}Pb-^{210}Po series is from dietary origin, the remainder being by inhalation from the atmosphere.

To estimate the dose from the affected foods we assume that meat consumption is 0.2 kg/day[5] and that the concentration of ^{226}Ra in meat is 0.5 pCi/kg and of ^{210}Pb 1 pCi/kg (Table 11) in the U.S. The concentrations in beef muscle are probably lower than these, but inclusion of organs, such as liver, could raise the mean intake values to the ranges assumed here. Thus, about 7% of the ^{226}Ra and 15% of the ^{210}Pb is from meat. Consequently, the doses from these dietary sources are about 1 mrem/yr from ^{226}Ra (7% of 16 mrem/yr) and 5 mrem/yr from ^{210}Pb (15% of 30 mrem/yr). Similarly, if the $^{234-238}$U pair is also included, it contributes about 2 mrem/yr.[1] The total

Table 9. Natural dose equivalent rates (NCRP 1975), QF = 10

Source	Gonads, mrer/yr	Bone (osteocytes), mrem/yr
Cosmic radiation	28	28
External terrestrial	26	26
Internally deposited radionuclides	27	115
Rounded total	80	170

Table 10. Dietary intake and dose equivalent rates[a]

Nuclide	Diet pCi/day	Concentration in human bone, pCi/kg	Dose rate, mrem/yr Osteocytes	Gonads
$^{234\text{-}238}U$	0.9	7	12	0.8
^{226}Ra	1.4	8	16	0.2
(^{228}Ra)[b]	1.1	4	19	0.3
^{210}Pb	1.4	60	1	0.1
^{210}Po	1.6	60	60	6
		Total	108	7

[a]From reference 1.
[b]A major contribution to bone dose, but not in the ^{238}U series.

Table 11. Dose rates due to consumption of meat[a] containing "normal" (New York City) concentrations of radionuclides

Nuclide	Concentration, pCi/kg	Intake rate, pCi/d	Dietary fraction, %	Skeletal dose rate, mrem/yr
$^{234\text{-}238}U$	0.7	0.14	16	2
^{226}Ra	0.5	0.1	7	1
^{210}Pb(^{210}Po)	1	0.2	15	5

[a]Assumed intake of 0.2 kg/day[5].

skeletal dose rate from the consumption of meat is thus about 8 mrem/yr. Doubling the intake from meat of these nuclides would thus increase the dose by this amount. Further, since the ^{226}Ra and ^{210}Pb levels in the control cattle appear to be several times those assumed here, doubling of these concentrations could easily approach a dose equivalent rate increase of 25 mrem/yr, the level considered undesirable by the U.S. Environmental Protection Agency.[9]

A similar effect could occur with vegetables, which appear to contribute about 25% of the dietary nuclide content, i.e., 3, 4, and 8 mrem/yr for the U, Ra, and Pb, respectively, for a total of about 15 mrem/yr. Thus a doubling of nuclide content in meat and vegetables could contribute an additional 30 mrem/yr to the skeleton. Similarly, the dose increases to soft tissues by a similar argument would be about 2 mrem/yr.

Conclusion

While these results are only preliminary they show that some contamination of the human food chain is likely. The degree is probably not large since the sampling was designed to exaggerate the contamination level. Rabbits were from near the tailings within the perimeter fence and cattle were from fields close to the tailings. Because of the incomplete results on cattle, the level of contamination is uncertain. Vegetables may reflect some influence of the mill. In any case, in the region under study, which has a small population and limited agriculture, it appears that, with an admixture of foods from outside the region, contamination of diet may be small.

We expect to finish these measurements soon to obtain more definite conclusions on the effects of tailings on cattle and, specifically, to determine uranium and ^{230}Th concentrations, and to obtain more specimens and better information on them. Acquisition of feed samples for the cattle would aid in estimating transfer coefficients. In particular, analysis of samples from cattle, grass, and soil from grazing land irrigated by mine water would greatly aid in these studies.

Acknowledgements

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APPENDIX A. Exposure Data for Radium Patients

Table 1 summarizes exposure data collected as of 31 December 1977 for 2072 radium cases under study at the Center for Human Radiobiology. It includes all persons measured for radium since the start of the Center in 1969 and all persons for whom we have analysis data from earlier work at the Radioactivity Center of the Massachusetts Institute of Technology, the New Jersey Radium Research Project of the New Jersey Department of Health, and the Argonne Radium Studies at the Argonne National Laboratory and the Argonne Cancer Research Hospital.

The corresponding table in the 1977 annual report[1] listed 1933 cases. The radium burdens of 137 persons, including 4 deceased, were measured for the first time in 1977. Three cases not previously listed have been added because reports of measurements made in prior years were located. The 140 additional cases are identified by a star following the year of measurement. One previously listed case (11-468) was transferred to a multiradionuclide exposure category. There were follow-up examinations and burden measurements in 1977 on 64 previously listed persons. Changes in basic data for several of the previously listed cases are due to review of information on exposure histories and to reassessment of old measurement data.

The cases are listed in order of identification number. In column 5, the type of exposure to radium (dial painting, medical, etc.) is indicated by code digits, which are defined in Table A1; if more than one type of exposure occurred, two non-zero digits are given. Column 7 gives the total period (in weeks) from first to last exposure. A value of 0 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 (a single exposure or longer); in these cases, zero duration was used in the calculation of the dose. For a dial painter whose first exposure was before the year 1926 but whose period of exposure extended into 1926 or beyond, the duration used in calculating the dose corresponded to the exposure terminating in 1926.

The ^{226}Ra body burdens given in the table are expressed as nanocuries (nCi) of ^{226}Ra present in the year of measurement shown in the preceding column.

TABLE A1. Type of Exposure to ^{226}Ra or ^{228}Ra or Both for TABLE 1

Code Number	Exposure to radium
1	Industrial; painted dials
2	Medical; drank Radithor nostrum
4	Medical; ingestion
5	Medical; injection
6	Laboratory; industry or research
7	Industrial; miscellaneous work or accidents
8	Offspring of a previously exposed female

TABLE A2. Principal Types of Measurement of Body Burdens of ^{226}Ra and ^{228}Ra for TABLE 1.

Code letter	Method	Subject or tissue
A	Gamma-ray	Major portions of skeletons or cremation ash
B	Whole-body gamma-ray and breath radon (thoron) with spirometer	In vivo
C	Whole-body gamma-ray	In vivo
D	Breath radon (thoron) with spirometer	In vivo
E	Whole-body gamma-ray (secondary method), alone or with a flask sample of breath radon	In vivo
F	Radiochemical or direct gamma-ray	Bone samples
G	Breath radon with flask	In vivo
Z	Ratio of ^{228}Ra to ^{226}Ra estimated from results on colleagues and/or measurements of radium materials	. . . .

TABLE A3. Error Ranges for ^{226}Ra Body Burdens and $^{228}Ra/^{226}Ra$ Ratios in TABLE 1.

Code number	Standard error[a]
1	≤ 10%
2	11–20%
3	21–50%
4	1.5 (×, ÷)
5	2 (×, ÷)
6	> 50%
7	3 (×, ÷)
8	Probably an upper limit[2]
9	Initial ratio of ^{228}Ra to ^{226}Ra probably ≤ 0.20[2]
L	90% confidence limits extend from 0.0 nCi to an upper limit between 4 and 8 nCi

[a] Either the relative standard error (given in %) or the factor (×, ÷) corresponding to one standard error in a log normal distribution. For the latter case, the upper and lower limits associated with one standard error are respectively obtained by multiplying and dividing the value in TABLE 1 by the factor; and the square of this factor is used to obtain the corresponding limits for two standard errors.

If several measurements over a period of years had been made for a given case, the result (and data) of the last measurement of highest available quality is given. Under "METHOD + ERR," the first symbol indicates the type of measurement according to the letter code of Table A2. Type A indicates that a complete skeletal measurement of bones was made, the letters B, C,..., G tend to imply increasingly uncertain types of measurement but with wide variation in size of error within each category. The digit that follows the method letter is the code symbol for an error estimated on the basis of type of measurement, amount of radium found, and examination of the data reported by the contributing laboratories. Code definitions for size of error are given in Table A3, and the errors shown include systematic errors as well as replication errors.

The letter L in place of a digit in the error column indicates that the result was taken from the New Jersey Radium Research Project records in which the measured value of ^{226}Ra was less than 4 nCi, their reported lower limit of detection. For these cases, the value 4 is shown in the ^{226}Ra column, but the letter L means that the 90% confidence limits extend from 0.0 nCi to an upper limit somewhere between 4 and 8 nCi. There are 54 of these cases which have the prefix 05 in the case number and one with case number 01-222. A "less than" indication was not used for cases measured at the other sites, even though the best measurements of small whole-body burdens have a standard deviation of 1 to 2 nCi. Instead, the measured values are given in the table when the result was zero or positive, and negative results are shown as zeros. These limitations should be kept in mind when evaluating error limits for very small body burdens.

The entries in column 11 are activity ratios of ^{228}Ra to ^{226}Ra at the time of measurement of ^{226}Ra in body content. A value of 5.7 yr for the half-life of ^{228}Ra was used in making corrections for radioactive decay. The method and error designations in column 12 are defined in Tables A2 and A3. The letter Z for method means that the ratio for the indicated person was estimated from values obtained on a group of persons with similar exposure histories or from analysis of samples of the radium material to which the person was exposed.[2] If no direct measurement of ^{228}Ra was attempted, only the letter Z and the

error designation are shown. If measurement of ^{228}Ra was attempted, the method tried is indicated by the letter after the error symbol in column 12. Ratios obtained by measurements of ^{228}Ra and ^{226}Ra are indicated by a letter other than Z. In all cases, the error designations in column 12 refer to the ratios in column 11. Errors for ratios with method codes of Z or F do not include errors in the measured values of ^{226}Ra body content.

The last four columns of Table 1 give quantities calculated from the measured body burdens and exposure data shown in the other columns. For many cases, the number of significant digits shown obviously exceeds the number justified by the accuracy of the basic data, and the errors indicated for the latter should be applied to the derived quantities. The columns under "INPUT" give the amounts of initially acquired ^{226}Ra and ^{228}Ra expressed as microcuries (μCi), calculated by applying the Norris retention function[3] to values of body burdens usually measured long after the initial intake. The cumulative rads, given in the last two columns for ^{226}Ra and ^{228}Ra separately, refer to the average ionization dose to the skeleton[4]—either up to the date of death or, for the living subjects, through 1977. Except for the fetal skeleton (case 01-579), the results in the last two columns were calculated with standard skeletal masses of 5 kg for females and 7 kg for males.

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TABLE 1 EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
00-001	M	1883	1928	06	1913	780	1967	13000	F4	0.00700	F3	1016	1298	2893	8286
00-002	F	1896	1922	01	1917	223	1966	16000	F4	0.00110	F3	996	310	2313	1369
00-003	F	1894	1927	01	1917	104	1966	7000	F4	0.01200	F1	872	3570	4074	40367
00-004	F	1900	1931	01	1917	88	1963	9000	F4	0.00080	F1	1367	264	8050	3481
00-005	F	1901	1939	01	1917	300	1963	1400	F4	0.00700	Z7	258	331	1913	4731
00-006	F	1903	1930	01	1918	128	1969	2610	A1	0.00536	A1	357	808	1859	9901
00-007	F	1903	1935	01	1919	104	1963	1000	F4	0.01000	Z7	163	302	1038	4124
00-008	M	1890	1938	06	1915	598	1972	3045	A1	0.00288	A3	525	682	2601	6775
00-009	F	1900	1928	01	1921	234	1969	2650	A1	0.00490	A2	230	283	728	2035
00-017	F	1899	1924	01	1917	156	1970	17000	A1	0.00069	Z7A	1626	580	5650	4765
00-019	F	1895	1946	01	1917	260	1976	2400	F2	0.00140	F4	525	693	4790	10252
00-020	M	1888	1925	06	1912	676	1969	920	A1	0.00228	A6	67	49	174	286
00-022	F	1889	1925	01	1917	377	1960	10000	F4	0.01000	F1	752	807	2223	5201
00-027	F	1902	1942	01	1918	130	1970	2500	A1	0.00023	F3	505	55	4187	808
00-028	F	1902	1933	01	1917	279	1969	10000	F4	0.00036	F1	1522	214	9016	2816
00-029	F	1900		01	1917	409	1969	17	G6	0.0	Z9	5	0	75	0
00-033	M	1868	1922	06	1919	156	1970	6	A6	0.00300	Z7A	0	0	0	0
01-001	F	1878	1949	05	1922	+0	1972	15400	A1	0.0	Z9A	3403	0	31456	0
01-002	F	1906	1939	01	1922	676	1936	18000	B2	0.02150	F1	2599	236	16586	3220
01-003	M	1888	1956	05	1925	304	1967	12800	A1	0.00037	A3	2882	120	19507	1273
01-004	F	1869	1953	04	1918	+0	1941	10500	E4	0.0	Z9	2134	0	23320	0
01-005	M	1877	1939	02	1927	12	1939	5000	E4	0.50000	E4	721	1530	2850	13918
01-006	F	1899	1938	01	1919	260	1970	3590	A1	0.00144	A3	612	314	4144	4361
01-007	F	1886	1949	05	1926	+0	1967	3620	A1	0.0	Z9A	736	0	6142	0
01-008	F	1900	1958	01	1917	78	1960	6000	F2	0.00067	F3	1632	186	19519	2790
01-009	F	1898	1945	01	1918	52	1960	6500	F4	0.00050	F2	1422	110	12991	1634
01-010	M	1882	1956	04	1926	+0	1967	5200	A1	0.0	Z9A	1214	0	8574	0
01-011	F	1872	1937	04	1919	156	1967	4650	A1	0.0	Z9A	794	0	5380	0
01-012	F	1867	1956	05	1922	+0	1970	5800	A1	0.0	Z9A	1445	0	15491	0
01-014	F	1901	1949	01	1916	156	1968	2240	A1	0.00036	F3	536	89	5471	1328
01-015	M	1888	1967	01	1917	780	1935	200	E4	0.0	Z9	30	0	281	0
01-016	F	1891	1966	01	1921	208	1973	1940	A1	0.00245	F2	546	578	6817	8678
01-017	F	1883	1976	02	1926	156	1977	1120	A1	0.00156	B2	336	214	4534	3221
01-018	M	1889	1958	06	1911	2340	1950	1250	B2	0.0	Z9B	185	0	1110	0
01-019	F	1903	1936	01	1922	253	1965	240	A1	0.02958	A2	35	147	193	1879

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-020	F	1905	1956	05	1923	5	1950	1500	E4	0.0	Z9	331	0	3479	0
01-021	F	1887	1973	01	1916	104	1965	1250	E4	0.0	Z9	373	0	5531	0
01-022	F	1900	1951	01	1917	110	1968	600	A2	0.0	Z9A	147	0	1544	0
01-024	F	1901	1956	01	1919	156	1943	1140	B2	0.02190	F3	229	77	2525	1149
01-025	F	1886	1952	05	1924	+0	1951	1200	B2	0.00100	F3	265	7	2509	105
01-026	F	1905	1958	01	1925	156	1950	700	B2	0.03000	D5	147	87	1531	1295
01-027	M	1889	1957	06	1912	1040	1960	500	A2	0.0	Z9F	125	0	973	0
01-028	M	1879	1965	06	1912	260	1953	250	E4	0.0	Z9	66	0	658	0
01-029	M	1876	1958	06	1902	+0	1950	300	G4	0.0	Z9	89	0	948	0
01-030	M	1882	1952	07	1936	0	1950	20	F4	0.0	Z9	3	0	15	0
01-031	F	1906	1934	01	1925	4	1975	910	A1	0.01130	A1	113	557	528	6296
01-032	F	1908	1940	01	1924	201	1968	1450	A1	0.02800	A1	236	1228	1506	16742
01-033	F	1908	1931	01	1923	42	1963	2472	A1	0.05153	A1	282	1793	1192	18509
01-034	F	1913		01	1929	18	1965	8	G6	0.01000	Z8	2	2	27	24
01-035	F	1901	1972	01	1920	19	1971	0	B6	0.01860	Z2B	0	0	0	0
01-037	F	1908		01	1928	26	1974	0	B6	0.00327	Z8B	0	0	0	0
01-038	F	1910		01	1927	111	1959	8	B2	0.02000	Z8B	2	2	26	24
01-039	F	1915		07	1934	1092	1972	1	B6	0.0	Z9B	0	0	2	0
01-040	F	1907	1929	01	1923	60	1963	4300	A1	0.05209	A1	412	2585	1422	21160
01-041	F	1909		01	1927	22	1971	0	B6	0.00470	Z8B	0	0	0	0
01-043	F	1912		01	1927	8	1958	9	B6	0.02200	Z8B	2	2	29	30
01-044	F	1904		01	1924	22	1959	4	B3	0.08000	Z2B	1	6	14	83
01-045	F	1889		01	1922	237	1959	0	B6	0.08000	Z2B	0	0	0	0
01-046	F	1903	1943	01	1920	657	1963	551	A1	0.05607	A1	104	731	793	10502
01-047	F	1896		01	1920	367	1962	80	G4	0.05700	Z2	21	136	310	2048
01-048	F	1900		01	1920	206	1957	140	B2	0.09290	F2	35	230	520	3456
01-049	F	1903	1937	01	1920	1	1960	1000	A1	0.07300	A2	174	1641	1198	22993
01-050	F	1911		01	1925	10	1976	1	B6	0.00258	Z8B	0	0	4	6
01-051	F	1904	1977	01	1923	162	1957	150	B2	0.13330	D5	36	251	519	3781
01-052	F	1910	1930	01	1924	144	1965	2000	A1	0.03500	A1	183	824	602	6301
01-054	F	1909	1937	01	1924	202	1965	2100	A1	0.03714	A1	304	1457	1692	18610
01-055	F	1907		01	1925	85	1976	4	B3	0.01024	Z2B	1	6	17	87
01-056	F	1904	1978	01	1920	364	1965	134	B1	0.03432	B2	37	206	546	3093
01-057	F	1908	1931	01	1924	81	1963	4900	A1	0.05163	A1	504	2704	1887	24482
01-059	F	1905	1967	01	1920	299	1964	180	B1	0.04277	B2	49	307	628	4608

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-060	F	1909		07	1928	20	1974	0	B6	0.00330	Z8B	0	0	0	0
01-063	F	1911		01	1927	213	1976	34	B1	0.00154	Z8B	10	5	134	69
01-066	F	1904		01	1925	0	1975	0	B6	0.00290	Z8B	0	0	0	0
01-069	F	1905		17	1922	107	1976	0	B6	0.01024	Z2B	0	0	0	0
01-070	F	1910		01	1927	63	1973	1	B6	0.00370	Z8B	0	0	4	4
01-071	F	1908	1967	01	1927	6	1958	0	B6	0.02300	Z8B	0	0	0	0
01-072	F	1899		01	1921	130	1954	100	E4	0.10000	D5	24	114	352	1709
01-073	F	1900	1969	01	1921	122	1966	87	B1	0.03563	B2	25	181	327	2722
01-074	F	1909		01	1927	47	1976	6	B3	0.00246	Z8B	2	2	25	24
01-075	F	1902		01	1922	52	1976	2	B6	0.01024	Z2B	1	4	9	65
01-078	F	1909		01	1925	40	1974	4	B6	0.00313	Z8B	1	1	17	21
01-079	F	1901	1943	01	1920	176	1960	750	F4	0.09070	F1	146	1387	1164	20106
01-080	F	1902		01	1921	204	1968	106	B1	0.02075	B3	31	150	443	2254
01-081	F	1907		01	1923	11	1959	7	B6	0.08000	Z2B	2	11	26	170
01-082	F	1902	1935	01	1919	230	1963	1030	A1	0.03786	A1	160	956	968	12727
01-084	F	1904		01	1923	712	1974	46	B2	0.01297	Z2B	14	74	198	1110
01-085	F	1913		01	1927	47	1958	6	B6	0.02200	Z8B	1	1	19	19
01-086	F	1907	1966	01	1925	4	1959	0	B6	0.08000	Z2B	0	0	0	0
01-087	F	1905		01	1921	344	1964	780	F4	0.03690	F1	213	1061	3065	15954
01-090	F	1910		01	1927	90	1977	5	B3	0.00218	Z8B	2	1	21	19
01-091	F	1907		01	1927	264	1974	0	B6	0.00327	Z8B	0	0	0	0
01-092	F	1906	1976	01	1922	24	1971	2	B6	0.01860	Z2B	1	4	9	63
01-093	F	1904		01	1926	8	1971	0	B6	0.00460	Z8B	0	0	0	0
01-094	F	1888	1966	01	1921	128	1964	11	G4	0.04400	Z4	3	21	39	322
01-095	F	1907	1977	01	1922	34	1975	6	B2	0.01163	Z2B	2	13	27	198
01-096	F	1909		01	1927	310	1960	27	D2	0.01800	Z8	6	4	83	64
01-097	F	1905		01	1921	110	1963	122	B1	0.03852	B2	33	187	491	2808
01-099	F	1905	1945	01	1924	18	1963	164	A1	0.05365	A2	32	191	248	2760
01-100	F	1905	1967	01	1924	36	1957	34	B2	0.13200	D5	8	58	103	872
01-101	F	1905		01	1924	4	1959	0	B6	0.08000	Z2B	0	0	0	0
01-105	F	1898	1945	01	1921	21	1963	460	A1	0.05217	A1	95	801	812	11743
01-106	F	1902		01	1924	155	1959	10	B2	0.08000	Z2B	2	12	35	187
01-110	F	1909		01	1925	93	1974	3	B6	0.00313	Z8B	1	1	13	15
01-111	F	1910		01	1927	16	1974	2	B6	0.00313	Z8B	1	1	8	8
01-112	F	1908	1955	01	1924	835	1960	80	F4	0.07000	F1	19	92	185	1368

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-113	F	1912		01	1928	5	1959	3	B6	0.02000	Z8B	1	1	10	9
01-115	F	1908	1944	01	1924	330	1963	472	A1	0.03093	A1	87	272	642	3883
01-116	F	1899	1965	01	1920	459	1955	290	G4	0.10000	G5	70	333	860	5000
01-118	F	1909	1971	01	1923	13	1959	0	B6	0.08000	Z2B	0	0	0	0
01-119	F	1899	1966	01	1920	14	1958	5	B6	0.09000	Z2B	1	12	17	178
01-120	F	1910		01	1925	125	1959	10	B2	0.02000	Z8B	2	3	35	44
01-122	F	1912		01	1927	49	1975	11	B2	0.00290	Z8B	3	3	45	46
01-123	F	1889		01	1923	11	1976	0	B6	0.01024	Z2B	0	0	0	0
01-124	F	1909		01	1927	64	1977	60	B1	0.00228	Z9B	18	17	248	253
01-125	F	1911		01	1927	5	1974	0	B6	0.00327	Z8B	0	0	0	0
01-126	F	1903	1969	01	1922	416	1969	150	A1	0.02667	A3	43	271	556	4074
01-127	F	1908		01	1927	9	1974	1	B6	0.00330	Z8B	0	0	4	4
01-128	F	1910		01	1927	4	1959	2	B6	0.02000	Z8B	0	0	7	7
01-129	F	1906	1934	01	1923	4	1977*	2	F6	0.00907	Z2F	0	2	2	25
01-130	F	1909		01	1926	196	1964	11	B2	0.01140	Z8B	3	3	39	39
01-132	F	1908	1944	01	1923	76	1966	1327	A1	0.03496	A1	253	1505	1946	21690
01-133	F	1910		01	1926	65	1958	13	B2	0.03000	Z8B	3	4	43	64
01-136	F	1907		01	1923	185	1976	35	B2	0.00858	B3	11	48	154	728
01-137	F	1901		01	1923	714	1977	5	B3	0.00902	Z2B	2	8	22	125
01-138	F	1883	1963	04	1919	4	1959	10	G6	0.0	Z9	3	0	34	0
01-139	M	1881	1964	02	1928	130	1962	1270	B1	0.01417	B2	310	235	2409	2509
01-140	F	1890		01	1919	78	1975	0	B6	0.0	Z9B	0	0	0	0
01-141	M	1886		02	1928	130	1974	17	B2	0.00330	Z5B	5	4	47	40
01-142	F	1899		01	1917	52	1969	0	G6	0.0	Z9	0	0	0	0
01-143	F	1904		01	1921	65	1976	7	B6	0.0	Z9B	2	0	33	0
01-144	F	1897	1973	04	1922	26	1971	694	B1	0.0	Z9B	209	0	2902	0
01-145	F	1900	1957	01	1918	60	1966	6331	A1	0.00077	A3	1681	413	19506	6195
01-146	F	1882	1967	02	1927	156	1968	100	A1	0.00870	Z5A	27	28	309	420
01-147	F	1902		01	1917	26	1965	52	G4	0.0	Z9	15	0	239	0
01-148	F	1907		06	1936	364	1958	40	G4	0.0	Z9	7	0	83	0
01-149	F	1888	1959	01	1919	26	1969	1630	A1	0.00533	A3	440	995	5226	14933
01-150	F	1881		04	1930	104	1970	3	B6	0.0	Z9B	1	0	10	0
01-151	F	1905		06	1927	52	1976	1	B6	0.0	Z9	0	0	2	0
01-152	F	1904		01	1920	17	1977	2	C6	0.00159	Z5B	1	1	10	16
01-153	M	1890	1964	06	1920	104	1963	280	B1	0.00036	B6	78	5	694	50

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-154	M	1896	1968	06	1923	+0	1959	0	G6	0.01500	Z7	0	0	0	0
01-156	F	1900	1959	01	1918	156	1959	40	G6	0.0	Z9	11	0	127	0
01-157	F	1894		02	1925	13	1975	49	B2	0.00139	Z5B	15	9	210	134
01-158	F	1901		06	1920	52	1959	1	G6	0.0	Z9	0	0	4	0
01-159	F	1915		01	1933	312	1972	2	B6	0.0	Z9B	1	0	6	0
01-160	F	1873	1965	02	1925	+0	1959	130	B1	0.02000	B3	32	40	386	607
01-161	F	1896	1973	01	1918	17	1959	1	B6	0.0	Z9B	0	0	4	0
01-162	M	1898	1966	06	1920	364	1959	95	B1	0.01000	Z7B	24	17	214	187
01-163	F	1903		01	1920	26	1972	2	B6	0.00360	Z7B	1	1	9	18
01-164	F	1900	1972	01	1918	39	1959	9	B2	0.0	Z9B	2	0	35	0
01-165	F	1904		01	1922	22	1974	13	B2	0.0	Z9C	4	0	60	0
01-166	F	1897	1969	01	1916	26	1959	0	B6	0.0	Z9B	0	0	0	0
01-168	F	1895		06	1919	468	1966	1	B6	0.0	Z9B	0	0	4	0
01-169	F	1918		01	1936	69	1975	0	B6	0.0	Z9B	0	0	0	0
01-170	M	1893	1966	05	1940	0	1959	4	G6	0.0	Z9	1	0	5	0
01-171	M	1895	1975	45	1914	6	1958	1500	B1	0.0	Z9B	427	0	4788	0
01-172	F	1898	1968	01	1916	136	1961	1960	B1	0.00112	B3	556	126	7736	1892
01-173	M	1881	1959	06	1917	1300	1959	70	G4	0.0	Z9	16	0	110	0
01-175	F	1900	1966	02	1927	13	1965	1710	B1	0.00760	B2	451	343	5269	5139
01-176	F	1893	1969	01	1917	104	1969	0	G6	0.0	Z9	0	0	0	0
01-177	M	1915		06	1936	312	1969	61	B1	0.0	Z9B	14	0	117	0
01-178	M	1939		07	1958	0	1973	2	B6	0.0	Z9C	0	0	1	0
01-179	F	1890	1966	45	1924	58	1959	2000	B1	0.0	Z9B	502	0	6115	0
01-180	F	1900		01	1918	26	1971	3	B3	0.0	Z9B	1	0	14	0
01-181	M	1913	1963	06	1940	130	1959	220	B1	0.0	Z9B	39	0	225	0
01-182	M	1902	1959	02	1936	+0	1959	7	D3	0.02600	Z5D	1	1	8	6
01-183	F	1901	1969	01	1915	78	1969	203	A1	0.0	Z9A	64	0	917	0
01-184	M	1887	1969	05	1922	10	1968	48	B2	0.0	Z9B	14	0	132	0
01-185	M	1881	1962	06	1912	+0	1959	40	G6	0.0	Z9	12	0	116	0
01-186	M	1925		06	1943	416	1976	19	B2	0.0	Z9B	4	0	31	0
01-187	M	1917		06	1943	78	1959	42	B2	0.0	Z9B	7	0	52	0
01-188	F	1886		04	1933	3	1959	4	G6	0.0	Z9	1	0	11	0
01-189	M	1921		07	1958	0	1973	0	B6	0.0	Z9C	0	0	0	0
01-190	F	1927		07	1958	0	1973	0	B6	0.0	Z9C	0	0	0	0
01-191	M	1897	1966	06	1913	78	1959	4	B6	0.0	Z9B	1	0	12	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-192	F	1902	1962	01	1925	52	1959	34	B2	0.0	Z9B	8	0	94	0
01-193	F	1886	1960	06	1917	156	1974	31	A2	0.0	Z9	9	0	105	0
01-194	M	1898		01	1916	676	1972	7	B6	0.0	Z9B	2	0	23	0
01-195	F	1893	1958	06	1912	520	1959	1	A6	0.0	Z9	0	0	3	0
01-196	M	1907		02	1930	20	1972	69	B1	0.00540	Z5B	19	17	180	179
01-197	F	1883	1965	04	1916	+0	1958	16	G6	0.0	Z9	4	0	61	0
01-200	F	1910		01	1925	220	1977	3	B3	0.00914	Z2B	1	4	13	67
01-201	F	1911		01	1925	55	1959	26	B2	0.02100	Z8B	6	8	90	119
01-203	F	1908		01	1923	1	1973	0	B6	0.01470	Z2B	0	0	0	0
01-204	F	1901		01	1917	22	1959	5	B3	0.0	Z9B	1	0	21	0
01-205	M	1921	1974	06	1951	52	1972	7	B3	0.0	Z9C	1	0	8	0
01-206	M	1896		06	1918	17	1975	9	B2	0.0	Z9B	3	0	32	0
01-207	F	1909	1957	01	1927	9	1959	4	B3	0.02000	Z8B	1	1	11	14
01-208	M	1901	1972	06	1939	1144	1974	818	A1	0.0	Z9	157	0	900	0
01-209	F	1908	1975	01	1926	16	1959	6	B6	0.02700	Z8B	1	2	20	32
01-210	M	1878	1971	06	1918	2028	1959	12	B2	0.0	Z9B	2	0	15	0
01-214	M	1891	1954	06	1915	1248	1959	82	B1	0.00700	Z7B	19	4	156	47
01-216	F	1903	1963	01	1924	4	1959	0	B6	0.08000	Z2B	0	0	0	0
01-217	M	1894	1971	01	1914	208	1959	5	B3	0.0	Z9B	1	0	15	0
01-218	M	1924		06	1950	780	1974	0	B6	0.0	Z9B	0	0	0	0
01-219	F	1910		01	1927	10	1976	0	B6	0.00246	Z8B	0	0	0	0
01-220	F	1907		01	1924	26	1959	2	B6	0.07100	Z2B	1	2	7	37
01-221	M	1892	1970	06	1916	520	1967	10	B2	0.00320	Z7B	3	2	28	25
01-222	F	1910		01	1925	17	1964	4	CL	0.04400	Z2C	1	5	15	79
01-223	F	1912		01	1927	7	1963	0	G6	0.01200	Z8	0	0	0	0
01-225	F	1906		01	1931	35	1959	0	D6	0.0	Z9D	0	0	0	0
01-226	F	1911		01	1927	22	1976	0	B6	0.00258	Z8B	0	0	0	0
01-227	F	1908		07	1933	2184	1975	0	B6	0.0	Z9B	0	0	0	0
01-228	F	1906		01	1926	61	1972	6	B6	0.00420	Z8B	2	2	24	27
01-229	F	1903		01	1923	2	1959	8	B2	0.08000	Z2B	2	13	30	196
01-230	F	1913		01	1927	19	1973	1	B6	0.00370	Z8B	0	0	4	4
01-231	F	1910	1969	01	1930	84	1959	0	B6	0.0	Z9B	0	0	0	0
01-232	F	1909	1961	04	1926	43	1959	0	B6	0.0	Z9B	0	0	0	0
01-233	F	1912	1973	01	1927	145	1959	2	B6	0.02000	Z8B	0	0	6	6
01-234	F	1913	1966	01	1927	1	1959	0	B6	0.02000	Z8B	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-235	F	1908		01	1925	8	1959	1	B6	0.08000	Z2B	0	1	4	19
01-236	F	1910	1976	01	1927	9	1965	1	G6	0.01000	Z8	0	0	4	4
01-237	F	1908		01	1927	8	1975	0	B6	0.00290	Z8B	0	0	0	0
01-238	F	1896	1967	01	1920	2	1959	1	B6	0.08000	Z2B	0	2	4	37
01-239	F	1901	1958	01	1917	78	1957	830	F4	0.00157	F3	223	41	2665	620
01-240	F	1910		01	1927	13	1971	7	D6	0.00450	Z8D	2	2	27	28
01-243	M	1873	1959	06	1905	520	1958	15	G6	0.0	Z9	4	0	43	0
01-244	F	1901		01	1927	18	1975	1	B6	0.00307	Z8	0	0	4	5
01-245	F	1920		01	1957	30	1969	0	G6	0.0	Z9	0	0	0	0
01-246	F	1885	1970	06	1915	39	1967	3	B6	0.0	Z9B	1	0	14	0
01-247	M	1901		06	1923	689	1976	5	B3	0.00195	Z7B	1	1	14	8
01-248	F	1903		01	1917	208	1976	21	B2	0.0	Z9B	7	0	104	0
01-249	M	1928		08	1928	39	1967	2	G6	0.02700	Z2	1	2	5	17
01-250	M	1894		06	1916	520	1975	0	B6	0.0	Z9B	0	0	0	0
01-251	M	1890	1965	06	1912	156	1974	11	A2	0.0	Z9	3	0	34	0
01-252	F	1898		01	1917	104	1976	22	B1	0.0	Z9B	7	0	111	0
01-253	F	1898	1964	01	1916	104	1959	40	G6	0.0	Z9	11	0	147	0
01-254	F	1910		01	1927	2	1971	1	B6	0.00460	Z8B	0	0	4	4
01-255	F	1920		01	1942	52	1975	0	B6	0.0	Z9B	0	0	0	0
01-256	M	1920		06	1949	208	1959	14	G6	0.0	Z9	2	0	11	0
01-257	M	1885	1962	06	1941	624	1959	0	G6	0.0	Z9	0	0	0	0
01-258	M	1903		06	1923	1092	1969	17	G6	0.0	Z9	4	0	38	0
01-259	F	1910		06	1927	416	1977	0	B6	0.0	Z9	0	0	0	0
01-260	F	1891	1960	04	1918	50	1959	15	G6	0.0	Z9	4	0	50	0
01-261	F	1909	1969	01	1927	2	1959	0	B6	0.02000	Z8B	0	0	0	0
01-262	F	1895		06	1918	0	1969	22	G4	0.0	Z9	7	0	104	0
01-263	F	1897	1976	01	1917	17	1976	9	B6	0.0	Z9B	3	0	46	0
01-264	M	1906	1967	01	1944	770	1964	90	G4	0.0	Z9	13	0	59	0
01-265	F	1902		01	1919	2	1959	3	B6	0.08000	Z2B	1	8	12	126
01-266	F	1904	1961	01	1923	3	1959	1	B6	0.08000	Z2B	0	2	3	24
01-267	F	1904		01	1926	104	1966	45	G4	0.0	Z9	12	0	166	0
01-268	F	1901	1968	01	1920	48	1967	100	B2	0.01000	B2	29	84	391	1264
01-269	M	1911		06	1932	52	1974	0	B6	0.0	Z9B	0	0	0	0
01-270	F	1901		01	1943	32	1976	4	B3	0.0	Z9	1	0	10	0
01-271	F	1900		01	1917	86	1974	1	B6	0.0	Z9B	0	0	5	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-272	M	1888		06	1956	130	1959	78	G6	0.0	Z9	4	0	21	0
01-273	F	1907		01	1924	1	1959	2	B6	0.08400	Z2B	1	3	7	45
01-274	F	1906		01	1922	5	1961	5	B3	0.06200	Z2B	1	9	20	142
01-275	M	1930		06	1959	+0	1959	23	G6	0.0	Z9	0	0	0	0
01-276	M	1930	1962	06	1945	208	1959	60	G6	0.0	Z9	9	0	39	0
01-277	F	1909		01	1925	6	1976	0	G6	0.01054	Z2	0	0	0	0
01-278	F	1904	1976	06	1925	0	1969	10	G6	0.0	Z9	3	0	40	0
01-279	M	1901	1969	06	1928	1404	1966	0	G6	0.0	Z9	0	0	0	0
01-280	F	1905		01	1926	7	1971	0	B6	0.00460	Z8B	0	0	0	0
01-282	M	1893	1973	06	1916	156	1972	42	B2	0.0	Z9B	13	0	141	0
01-283	F	1895	1971	07	1918	52	1959	3	B6	0.0	Z9B	1	0	12	0
01-284	M	1892	1970	06	1943	780	1959	5	B3	0.0	Z9B	1	0	3	0
01-285	F	1900		01	1923	1	1960	4	B6	0.07100	Z2B	1	7	15	100
01-287	F	1908		01	1927	674	1977	2	B6	0.00232	Z8C	1	0	7	3
01-288	F	1894	1970	01	1926	2	1960	2	C6	0.02400	Z8C	1	1	6	11
01-289	F	1899	1975	01	1919	80	1971	4	B3	0.01860	Z2B	1	12	18	175
01-291	F	1910	1969	01	1928	17	1960	5	B6	0.01800	Z8B	1	1	15	16
01-293	F	1911		01	1924	11	1973	0	B6	0.01470	Z2B	0	0	0	0
01-294	F	1912		01	1927	52	1971	3	B3	0.00450	Z8B	1	1	12	11
01-295	F	1910		01	1927	14	1976	0	B6	0.00258	Z8B	0	0	0	0
01-296	F	1908		01	1927	5	1960	0	B6	0.01800	Z8B	0	0	0	0
01-297	F	1901		01	1921	122	1960	16	B2	0.09375	B3	4	39	62	588
01-299	F	1896		01	1917	104	1968	3	G6	0.0	Z9	1	0	14	0
01-301	F	1904		05	1926	5	1969	17	G4	0.0	Z9	5	0	67	0
01-302	F	1899	1966	05	1927	10	1968	2850	A1	0.0	Z9A	761	0	8910	0
01-303	M	1919		01	1940	104	1974	0	B6	0.0	Z9B	0	0	0	0
01-305	M	1925	1968	06	1946	1040	1966	160	E4	0.0	Z9C	15	0	56	0
01-306	M	1928		06	1955	364	1976	26	B1	0.0	Z9B	5	0	24	0
01-307	M	1930		06	1957	104	1975	4	B6	0.0	Z9B	1	0	4	0
01-308	M	1918	1957	06	1943	728	1958	1200	F4	0.0	Z9F	90	0	247	0
01-309	F	1908	1973	01	1923	2	1961	2	B6	0.06200	Z2B	1	3	7	50
01-310	F	1928		08	1928	39	1975	0	B6	0.01148	Z2	0	0	0	0
01-311	F	1911		01	1927	2	1961	1	B6	0.01500	Z8B	0	0	3	4
01-312	F	1907		01	1925	13	1976	0	B6	0.0	Z9B	0	0	0	0
01-313	M	1892		06	1911	624	1961	3	B3	0.0	Z9B	1	0	9	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-314	F	1909		01	1924	0	1961	1	B6	0.06200	Z2B	0	1	4	22
01-324	F	1907		01	1923	15	1962	1	G6	0.05700	Z2	0	2	4	26
01-326	F	1896	1972	02	1925	156	1966	100	G4	0.01100	Z5	27	36	349	539
01-327	F	1908		01	1927	1	1965	0	G6	0.01000	Z8	0	0	0	0
01-330	M	1915		06	1942	364	1976	66	B2	0.0	Z9B	16	0	113	0
01-331	M	1901		02	1927	+0	1966	80	G4	0.01100	Z5	21	27	211	290
01-332	F	1912	1971	01	1927	52	1965	0	G6	0.01000	Z8	0	0	0	0
01-333	F	1905		01	1924	10	1976	0	B6	0.01075	Z2	0	0	0	0
01-335	F	1899		16	1917	78	1975	3	B3	0.0	Z9B	1	0	15	0
01-336	M	1899		06	1945	1092	1976	39	B1	0.0	Z9B	7	0	41	0
01-341	M	1883		06	1943	176	1961	5	B3	0.0	Z9B	1	0	6	0
01-342	M	1897		06	1944	56	1961	1	B6	0.0	Z9B	0	0	1	0
01-343	F	1873	1954	04	1927	+0	1963	0	F6	0.0	Z9	0	0	0	0
01-344	F	1904	1976	01	1922	19	1962	7	G6	0.05700	Z2	2	14	27	206
01-345	F	1910	1977	01	1924	1	1962	4	G6	0.05700	Z2	1	6	15	92
01-346	F	1911		01	1927	17	1962	44	G6	0.01700	Z8	11	13	153	196
01-347	M	1896	1968	06	1926	1872	1962	14	B2	0.0	Z9B	2	0	10	0
01-348	F	1902	1973	01	1924	19	1966	112	B1	0.03482	B2	31	175	422	2628
01-349	F	1907	1967	01	1924	10	1966	93	B1	0.03225	B2	26	136	322	2043
01-350	F	1898	1973	01	1923	108	1962	0	G6	0.05700	Z2	0	0	0	0
01-351	F	1906		01	1923	3	1962	0	G6	0.05700	Z2	0	0	0	0
01-352	M	1922		06	1940	338	1962	191	B1	0.0	Z9B	35	0	265	0
01-356	M	1912	1973	06	1937	572	1969	23	B2	0.0	Z9B	5	0	36	0
01-357	F	1907	1970	07	1927	408	1962	0	G6	0.01400	Z8	0	0	0	0
01-358	F	1906		07	1923	168	1962	0	G6	0.05700	Z2	0	0	0	0
01-359	F	1908		01	1925	55	1962	25	B2	0.05600	Z2B	6	31	91	460
01-360	F	1911		01	1928	34	1962	0	G6	0.01400	Z8	0	0	0	0
01-361	F	1907	1976	01	1924	20	1974	1	B6	0.01323	Z2B	0	2	4	26
01-362	F	1906		01	1923	5	1962	0	G6	0.05700	Z2	0	0	0	0
01-363	F	1888		01	1918	260	1962	7	G6	0.05700	Z2	2	17	29	253
01-364	F	1911		07	1927	440	1964	6	G6	0.01140	Z8	1	1	19	13
01-365	F	1901		01	1924	40	1962	10	G6	0.05700	Z2	3	15	37	218
01-367	F	1899		01	1920	221	1976	4	B6	0.01024	Z2B	1	9	19	135
01-368	M	1925		06	1947	65	1976	28	B2	0.0	Z9B	6	0	44	0
01-369	F	1906		01	1923	33	1975	0	B6	0.01043	Z2	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-370	F	1904		01	1927	21	1962	0	G6	0.01500	Z8	0	0	0	0
01-371	F	1912		07	1928	36	1962	0	G6	0.01400	Z8	0	0	0	0
01-372	F	1911	1975	01	1927	1	1962	7	G6	0.01470	Z8	2	2	24	28
01-373	F	1910		01	1927	84	1962	2	G6	0.01400	Z8	1	0	7	7
01-374	F	1910		01	1927	+0	1962	12	G6	0.01470	Z8	3	3	42	47
01-376	F	1907	1973	01	1927	33	1963	2	G6	0.01300	Z8	1	1	7	8
01-377	F	1915		17	1929	208	1975	0	B6	0.0	Z9B	0	0	0	0
01-378	F	1907		01	1925	94	1976	0	B6	0.00258	Z8B	0	0	0	0
01-379	F	1909		01	1926	7	1975	18	B2	0.00281	Z8B	5	6	76	88
01-380	F	1910		01	1927	3	1972	0	B6	0.00420	Z8B	0	0	0	0
01-381	M	1887		02	1927	1	1964	5	G6	0.01400	Z5	1	2	13	18
01-382	F	1900		01	1920	320	1963	43	G4	0.01000	Z2	12	15	169	221
01-383	F	1907		01	1923	2	1976	0	B6	0.01006	Z2B	0	0	0	0
01-384	F	1905		01	1923	1	1975	0	B6	0.01177	Z2	0	0	0	0
01-385	F	1906	1971	01	1924	11	1963	5	G6	0.05000	Z2	1	8	18	114
01-386	F	1904		01	1927	15	1963	9	G4	0.01300	Z8	2	2	32	35
01-388	F	1873	1944	02	1928	+0	1965	2580	A1	0.01027	A1	434	401	2886	5555
01-389	F	1910	1930	01	1923	26	1963	1029	A1	0.06812	A1	111	946	435	9072
01-390	F	1887	1931	02	1925	260	1965	7400	A1	0.02527	A1	519	1180	1358	6351
01-391	F	1914	1969	07	1950	520	1964	1	B6	0.0	Z9B	0	0	1	0
01-392	M	1913	1972	07	1950	520	1964	1	B6	0.0	Z9B	0	0	1	0
01-393	M	1937		07	1950	520	1972	2	B6	0.0	Z9B	0	0	2	0
01-394	F	1944		07	1950	520	1972	4	B3	0.0	Z9B	1	0	6	0
01-395	F	1945		07	1950	520	1972	5	B3	0.0	Z9B	1	0	7	0
01-396	M	1947		07	1950	520	1972	1	B6	0.0	Z9B	0	0	1	0
01-397	F	1950		07	1950	498	1973	4	B3	0.0	Z9B	1	0	6	0
01-398	M	1951		07	1951	429	1972	0	B6	0.0	Z9B	0	0	0	0
01-399	F	1953		07	1953	350	1972	1	B6	0.0	Z9B	0	0	1	0
01-400	M	1903		07	1961	156	1964	2	B6	0.0	Z9B	0	0	0	0
01-401	F	1910		07	1961	156	1964	3	B6	0.0	Z9B	0	0	1	0
01-402	F	1898		01	1920	18	1963	0	G6	0.05000	Z2	0	0	0	0
01-403	F	1912		02	1926	+0	1971	27	B2	0.01838	C3	8	34	109	516
01-404	M	1875	1945	67	1912	1716	1965	2800	A1	0.0	Z9A	330	0	1523	0
01-405	F	1885	1957	67	1912	1716	1965	52	A1	0.0	Z9A	11	0	106	0
01-406	M	1902	1969	67	1916	250	1963	18	B2	0.0	Z9B	5	0	51	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
C1-407	M	1912	1977	67	1930	416	1963	38	B2	0.0	Z9B	9	0	78	0
01-408	F	1918		06	1934	416	1975	13	B2	0.0	Z9B	3	0	40	0
01-409	F	1914		06	1930	13	1975	34	B3	0.0	Z9B	10	0	129	0
01-410	F	1920		06	1940	156	1975	33	B1	0.0	Z9B	8	0	90	0
01-411	M	1915		06	1935	200	1973	8	B2	0.0	Z9C	2	0	18	0
01-412	M	1915	1970	02	1929	+0	1963	1	D6	0.01600	Z5D	0	0	2	3
01-413	F	1901	1965	01	1924	229	1964	11	G4	0.04400	Z2	3	15	35	222
01-414	F	1897		06	1931	572	1974	1	B6	0.0	Z9B	0	0	3	0
01-415	M	1898		06	1921	520	1964	0	B6	0.0	Z9B	0	0	0	0
01-416	F	1908		01	1924	2	1963	9	G6	0.04900	Z2	2	14	34	203
01-417	F	1907		01	1923	1	1963	0	G6	0.05000	Z2	0	0	0	0
01-418	M	1900	1972	06	1919	104	1963	6	G6	0.0	Z9	2	0	17	0
01-419	M	1895	1965	06	1916	260	1963	9	G6	0.0	Z9	3	0	24	0
01-420	F	1903	1967	06	1920	65	1963	2	G6	0.0	Z9	1	0	7	0
01-421	F	1887	1976	06	1915	312	1963	8	G6	0.0	Z9	2	0	35	0
01-423	M	1897		06	1919	260	1973	22	B2	0.0	Z9B	7	0	72	0
01-424	F	1882		05	1924	+0	1964	280	G4	0.0	Z9	76	0	1087	0
01-425	M	1933		07	1961	104	1964	0	B6	0.0	Z9B	0	0	0	0
01-426	F	1930		07	1961	104	1964	5	B3	0.0	Z9B	0	0	2	0
01-427	F	1960		07	1961	104	1964	5	E4	0.0	Z9	0	0	2	0
01-428	F	1957		07	1961	104	1964	2	E6	0.0	Z9	0	0	1	0
01-429	F	1897		06	1922	208	1974	0	B6	0.0	Z9B	0	0	0	0
01-430	M	1880	1969	02	1930	+0	1966	41	B2	0.02195	B3	11	18	88	197
01-431	F	1901	1975	05	1922	52	1971	765	B1	0.0	Z9B	229	0	3262	0
01-432	M	1895	1973	06	1915	520	1964	17	B2	0.0	Z9B	5	0	49	0
01-434	M	1880	1932	02	1927	156	1965	6126	A1	0.02189	A1	456	828	865	3250
01-435	F	1907		01	1925	5	1977	0	B6	0.00228	Z9B	0	0	0	0
01-436	F	1895		01	1927	180	1964	8	G6	0.01140	Z8	2	2	27	25
01-437	F	1910	1971	06	1931	104	1965	1	B6	0.0	Z9B	0	0	3	0
01-438	M	1867	1940	02	1925	208	1965	1850	A1	0.01372	A1	279	382	1163	3571
01-439	F	1880	1953	04	1922	8	1968	406	A2	0.0	Z9F	96	0	971	0
01-440	F	1908		01	1924	204	1965	0	G6	0.03900	Z2	0	0	0	0
01-446	F	1907		01	1925	0	1964	0	G6	0.04400	Z2	0	0	0	0
01-447	F	1909		17	1925	110	1965	3	G6	0.01000	Z8	1	1	11	14
01-448	F	1907		01	1925	5	1964	25	G4	0.01140	Z8	7	9	94	131

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-449	F	1899		01	1922	2	1965	7	G6	0.03900	Z2	2	14	29	215
01-450	M	1877	1936	06	1912	364	1966	0	A6	0.0	Z9A	0	0	0	0
01-451	F	1908		01	1924	4	1977*	14	G4	0.00907	Z2	4	25	63	375
01-454	F	1880	1970	01	1920	884	1974	1990	A1	0.0		586	0	7760	0
01-456	M	1878	1948	02	1928	26	1965	74	A1	0.03648	A3	14	44	75	454
01-457	F	1904		06	1920	78	1964	8	G4	0.0	Z9	2	0	34	0
01-459	M	1886	1971	06	1921	52	1964	10	G6	0.0	Z9	3	0	27	0
01-460	M	1882	1966	06	1912	104	1964	0	G6	0.0	Z9	0	0	0	0
01-461	M	1914	1970	06	1930	26	1964	9	G4	0.0	Z9	2	0	19	0
01-464	F	1914		01	1927	4	1970	4	G6	0.00540	Z8	1	1	16	17
01-466	F	1902	1946	01	1920	52	1965	0	A6	0.03800	Z2A	0	0	0	0
01-468	F	1910		01	1927	0	1965	0	G6	0.01000	Z8	0	0	0	0
01-469	M	1894		06	1918	52	1965	4	G6	0.0	Z9	1	0	13	0
01-470	F	1912		01	1927	70	1965	0	G6	0.01000	Z8	0	0	0	0
01-472	F	1896	1969	06	1919	156	1965	7	G6	0.0	Z9	2	0	27	0
01-474	F	1904		07	1921	100	1974	0	B6	0.01163	Z2B	0	0	0	0
01-475	F	1901		01	1928	4	1974	0	B6	0.00330	Z8B	0	0	0	0
01-476	F	1909		07	1927	71	1972	4	B3	0.00420	Z8B	1	1	16	16
01-477	F	1897		02	1925	+0	1965	1240	B1	0.00475	B2	336	207	4754	3111
01-478	F	1914		01	1935	24	1965	0	G6	0.0	Z9	0	0	0	0
01-479	F	1912		01	1927	1	1965	3	G6	0.01000	Z8	1	1	11	12
01-480	F	1915		01	1927	1	1965	38	G6	0.01000	Z8	10	10	138	153
01-481	F	1909		01	1927	14	1965	0	G6	0.01000	Z8	0	0	0	0
01-482	F	1912		01	1927	4	1974	1	B6	0.00330	Z8B	0	0	4	4
01-483	M	1907		17	1922	104	1975	0	B6	0.01184	Z2B	0	0	0	0
01-484	F	1908	1974	01	1926	0	1965	0	G6	0.01000	Z8	0	0	0	0
01-485	M	1870	1951	05	1911	1300	1965	340	A1	0.0	Z9A	74	0	488	0
01-486	F	1907		01	1923	6	1974	0	B6	0.01318	Z2B	0	0	0	0
01-487	F	1911		07	1927	565	1976	0	B6	0.00257	Z8B	0	0	0	0
01-489	F	1910		01	1926	348	1965	225	G6	0.01000	Z8	57	42	766	637
01-490	F	1908		01	1924	17	1974	2	B6	0.01318	Z2B	1	3	9	52
01-491	F	1922	1966	01	1943	728	1963	7	G6	0.0	Z9	1	0	7	0
01-492	F	1900		06	1921	260	1973	1	B6	0.0	Z9B	0	0	4	0
01-493	M	1893	1975	06	1927	1820	1973	4	B3	0.0	Z9C	1	0	6	0
01-494	M	1906	1966	06	1926	999	1966	0	G6	0.0	Z9	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS IO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-495	F	1908		01	1924	4	1965	0	G6	0.03900	Z2	0	0	0	0
01-496	F	1918		07	1934	106	1966	3	G6	0.0	Z9	1	0	9	0
01-497	F	1902		01	1921	8	1966	13	G6	0.03400	Z2	4	30	56	451
01-498	F	1897		06	1920	104	1976	1	B6	0.0	Z9C	0	0	5	0
01-501	M	1867	1937	02	1926	156	1966	2500	A1	0.00760	A1	320	260	1102	2149
01-503	M	1936		08	1936	39	1966	0	B6	0.0	Z9B	0	0	0	0
01-504	F	1913		01	1927	2	1975	0	B6	0.0	Z9B	0	0	0	0
01-505	F	1902		01	1927	1	1966	9	G4	0.00880	Z8	2	2	33	37
01-506	F	1897		04	1923	4	1966	7	B3	0.0	Z9C	2	0	29	0
01-507	F	1909		01	1927	22	1974	10	B2	0.00313	Z8B	3	3	40	41
01-508	F	1906	1968	01	1944	52	1966	30	G6	0.0	Z9	6	0	50	0
01-509	F	1943		08	1943	39	1967	0	B6	0.0	Z9B	0	0	0	0
01-510	F	1897		01	1927	12	1966	38	G6	0.00880	Z8	10	10	140	152
01-511	F	1908		07	1927	9	1974	0	B6	0.00330	Z8B	0	0	0	0
01-512	F	1895	1976	04	1912	13	1973	0	B6	0.0	Z9B	0	0	0	0
01-514	F	1904		07	1924	2184	1975	0	B6	0.00200	Z5B	0	0	0	0
01-515	F	1886		05	1940	0	1966	4	G6	0.0	Z9	1	0	10	0
01-516	F	1907	1976	01	1927	2	1967	7	G6	0.00780	Z8	2	2	26	29
01-518	M	1912		05	1949	+0	1977	0	B6	0.0	Z9B	0	0	0	0
01-519	M	1919		06	1937	260	1967	13	G6	0.0	Z9	3	0	24	0
01-520	F	1882	1969	02	1930	+0	1967	670	B1	0.00492	B2	174	77	2044	1158
01-521	M	1910		06	1942	520	1976	26	B1	0.0	Z9B	6	0	42	0
01-522	M	1905		06	1923	1924	1969	240	B1	0.0	Z9B	42	0	299	0
01-523	M	1917		06	1942	312	1968	30	G4	0.0	Z9	6	0	45	0
01-525	M	1923		06	1943	104	1968	17	G6	0.0	Z9	4	0	27	0
01-526	M	1921		06	1945	38	1976	35	B1	0.0	Z9B	8	0	60	0
01-529	M	1920		06	1943	260	1975	14	B2	0.0	Z9B	3	0	24	0
01-530	M	1920	1971	06	1943	104	1968	52	B1	0.0	Z9B	11	0	71	0
01-531	M	1918		06	1941	354	1974	13	B2	0.0	Z9B	3	0	22	0
01-532	M	1914	1973	06	1945	138	1968	1	G6	0.0	Z9	0	0	1	0
01-533	F	1903		04	1911	+0	1969	4	G6	0.0	Z9	1	0	22	0
01-534	M	1920		06	1944	154	1976	1	B6	0.0	Z9B	0	0	2	0
01-536	M	1916		06	1943	286	1968	17	G6	0.0	Z9	3	0	25	0
01-537	M	1917	1971	06	1944	208	1968	59	B1	0.0	Z9B	12	0	74	0
01-540	M	1890		07	1940	260	1968	0	G6	0.0	Z9	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD - ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-543	M	1920	1976	06	1943	167	1975	19	B2	0.0	Z9B	4	0	32	0
01-544	F	1879	1953	02	1930	+0	1968	93	A1	0.00430	A3	19	8	158	121
01-546	F	1897		01	1914	52	1967	0	G6	0.0	Z9	0	0	0	0
01-547	F	1897		06	1920	104	1975	4	B3	0.0	Z9B	1	0	19	0
01-548	M	1917		02	1930	+0	1972	5	B3	0.00200	Z5B	1	0	13	5
01-552	M	1907		06	1936	104	1967	20	G4	0.0	Z9	5	0	40	0
01-553	F	1910		01	1948	988	1967	0	G6	0.0	Z9	0	0	0	0
01-554	F	1928		01	1952	780	1967	490	G4	0.0	Z9	38	0	267	0
01-555	F	1894		01	1921	2	1975	0	B6	0.01155	Z2B	0	0	0	0
01-556	F	1910		01	1927	0	1967	0	G6	0.00780	Z8	0	0	0	0
01-557	F	1908		01	1925	35	1975	2	B6	0.00293	Z8B	1	1	9	11
01-558	M	1913		02	1927	130	1976	255	B1	0.00077	B2	76	19	734	206
01-562	F	1901	1931	01	1920	52	1970	10300	A1	0.0	Z9A	1392	0	7143	0
01-565	F	1892	1957	05	1925	26	1970	1600	A2	0.0	Z9A	385	0	3946	0
01-567	M	1885	1949	02	1925	+0	1970	1100	A2	0.00400	A2	229	218	1400	2282
01-568	M	1907	1928	05	1927	+0	1969	4900	A1	0.0	Z9A	195	0	183	0
01-569	F	1896		76	1922	282	1977*	6	G6	0.00907	Z2	2	10	27	144
01-570	F	1908		01	1926	260	1968	10	G4	0.0	Z9	3	0	36	0
01-571	F	1911		01	1928	44	1968	1	B6	0.00680	Z8B	0	0	4	3
01-573	F	1892	1945	01	1916	312	1970	670	A1	0.00195	F3	145	135	1307	2000
01-574	F	1885	1937	05	1924	77	1968	2730	A1	0.0	Z9A	400	0	2255	0
01-575	M	1910	1977	01	1950	1196	1973	2	B6	0.0	Z9B	0	0	1	0
01-576	F	1930		01	1946	780	1968	160	B1	0.0	Z9B	25	0	207	0
01-578	F	1904	1930	05	1926	17	1968	2000	A2	0.0	Z9A	160	0	452	0
01-579	F	1928	1928	08	1928	26	1973	2	A1	0.00289	Z2A	0	0	1	0
01-580	F	1894		01	1918	52	1972	1	B6	0.0	Z9B	0	0	5	0
01-581	M	1918		06	1946	52	1968	10	G4	0.0	Z9	2	0	14	0
01-582	F	1893		06	1917	24	1974	0	B6	0.0	Z9B	0	0	0	0
01-583	M	1890	1969	06	1918	104	1968	0	G6	0.00250	Z7	0	0	0	0
01-584	F	1908	1975	01	1926	260	1968	10	B2	0.0	Z9B	3	0	35	0
01-585	F	1906	1969	01	1925	26	1968	0	B6	0.00450	Z5B	0	0	0	0
01-586	F	1879	1973	05	1924	+0	1968	130	G6	0.0	Z9	37	0	504	0
01-588	F	1908		01	1929	104	1968	5	G6	0.0	Z9	1	0	17	0
01-589	M	1907		06	1927	78	1964	2	G6	0.0	Z9	1	0	5	0
01-590	M	1929		08	1929	39	1976	0	B6	0.01062	Z2C	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-591	F	1891	1975	01	1918	52	1973	0	G6	0.00016	Z7	0	0	0	0
01-592	F	1903	1971	01	1917	6	1968	0	G6	0.0	Z9	0	0	0	0
01-594	M	1926		01	1962	34	1975	2	B6	0.0	Z9B	0	0	1	0
01-595	F	1897		01	1917	130	1969	5	G6	0.0	Z9	2	0	24	0
01-597	F	1923		01	1940	364	1973	1	B6	0.0	Z9C	0	0	2	0
01-598	M	1879	1953	06	1941	+0	1952	400	G6	0.0	Z9	55	0	220	0
01-599	F	1909		01	1927	7	1973	2	B6	0.00370	Z8B	1	1	8	9
01-601	F	1902		01	1918	6	1969	0	G6	0.00020	Z7	0	0	0	0
01-603	F	1894		01	1915	676	1968	7	G6	0.00450	Z5	2	3	31	41
01-604	F	1896		01	1914	52	1971	1	B6	0.0	Z9B	0	0	5	0
01-608	F	1906	1976	01	1927	11	1974	0	G6	0.00330	Z8B	0	0	0	0
01-609	F	1906		01	1926	366	1973	3	B6	0.0	Z9B	1	0	11	0
01-610	M	1904	1969	06	1919	208	1968	10	G6	0.00450	Z7	3	4	28	43
01-612	F	1859	1936	17	1923	255	1972	18	A1	0.00680	Z4A	3	6	14	71
01-613	F	1906	1936	17	1923	265	1972	658	A1	0.00680	F2	88	165	450	1987
01-614	M	1882	1922	06	1920	+0	1974	24	A2	0.0	Z9	1	0	2	0
01-617	M	1922		08	1922	39	1973	4	B3	0.00020	Z3B	1	0	13	1
01-619	F	1909		01	1927	52	1969	0	G6	0.0	Z9	0	0	0	0
01-621	F	1908		01	1924	2	1975	7	B2	0.01135	Z2B	2	12	31	181
01-625	F	1911		01	1927	468	1968	6	G6	0.0	Z9	2	0	20	0
01-626	F	1932		08	1932	39	1971	0	B6	0.0	Z9B	0	0	0	0
01-627	F	1897		01	1917	52	1970	0	G6	0.0	Z9	0	0	0	0
01-628	F	1908		01	1925	312	1975	0	B6	0.00200	Z5B	0	0	0	0
01-629	F	1892	1977	01	1926	260	1969	12	G6	0.0	Z9	3	0	44	0
01-633	F	1878	1926	05	1925	4	1970	2600	A2	0.0	Z9A	101	0	130	0
01-635	M	1880	1937	06	1918	312	1973	1900	A1	0.0	Z9A	318	0	1509	0
01-640	F	1908		01	1924	21	1969	34	G6	0.00420	Z5	10	10	140	143
01-653	F	1910		01	1925	78	1969	7	G6	0.00420	Z5	2	2	28	25
01-659	F	1912		01	1928	26	1969	11	G6	0.0	Z9	3	0	41	0
01-660	F	1881	1957	04	1932	+0	1970	15	A6	0.0	Z9A	3	0	28	0
01-661	M	1874	1934	06	1914	572	1974	2	A6	0.0	Z9	0	0	1	0
01-663	M	1927		08	1927	39	1969	11	G4	0.0	Z9	3	0	30	0
01-665	M	1923		08	1923	39	1969	0	G6	0.0	Z9	0	0	0	0
01-667	F	1918		01	1941	234	1972	0	B6	0.0	Z9B	0	0	0	0
01-668	M	1933		07	1964	+0	1974	1	B6	0.0	Z9	0	0	1	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
01-669	F	1917		01	1934	104	1969	0	G6	0.0	Z9	0	0	0	0
01-670	M	1897		04	1928	+0	1969	0	G6	0.0	Z9	0	0	0	0
01-671	F	1923		01	1941	260	1972	2	B6	0.0	Z9B	0	0	5	0
01-674	M	1908		01	1931	1716	1973	0	B6	0.0	Z9B	0	0	0	0
01-681	F	1904		07	1920	4	1972	0	G6	0.00320	Z7	0	0	0	0
01-684	F	1894	1974	01	1917	1	1973	0	G6	0.0	Z9	0	0	0	0
01-688	M	1868	1948	07	1920	+0	1972	0	A6	0.00320	Z7A	0	0	0	0
01-690	M	1878	1940	04	1918	+0	1970	21	A1	0.0	Z9A	4	0	24	0
01-691	F	1913	1974	04	1935	0	1971	0	B6	0.0	Z9B	0	0	0	0
01-692	M	1885	1974	02	1925	+0	1970	30	G6	0.00680	Z5	9	14	84	150
01-694	M	1886	1953	54	1928	+0	1971	10000	F4	0.0	Z9	2123	0	13346	0
01-701	M	1892	1974	06	1916	312	1970	0	G6	0.0	Z9	0	0	0	0
01-706	F	1908		07	1923	100	1975	0	B6	0.01149	Z2	0	0	0	0
01-707	F	1908	1974	01	1927	1	1971	0	G6	0.00470	Z8	0	0	0	0
01-710	F	1901		01	1925	289	1970	0	G6	0.00370	Z5	0	0	0	0
01-711	F	1905		01	1925	312	1970	0	G6	0.00370	Z5	0	0	0	0
01-715	F	1907		01	1927	5	1976	0	B6	0.00258	Z8B	0	0	0	0
01-717	M	1910		27	1927	13	1974	4	B6	0.00420	Z5	1	1	12	16
01-728	F	1912		01	1927	6	1973	3	B3	0.00370	Z8B	1	1	12	13
01-736	F	1907	1931	01	1923	52	1977*	1	F6	0.00170	Z7F	0	0	0	1
01-739	F	1856	1928	05	1926	7	1972	11500	A1	0.0	Z9A	645	0	1226	0
03-005	M	1917		07	1948	+0	1973	0	B6	0.0	Z9C	0	0	0	0
03-008	F	1934		08	1934	39	1971	0	B6	0.0	Z9C	0	0	0	0
03-009	F	1918		01	1941	104	1972	1	B6	0.0	Z9C	0	0	2	0
03-101	F	1908	1971	05	1931	15	1963	1580	C2	0.0	Z9	380	0	4523	0
03-102	M	1908	1976	05	1931	15	1973	628	B1	0.0	Z9C	174	0	1598	0
03-103	F	1868	1952	05	1931	15	1951	420	E4	0.0	Z9	79	0	621	0
03-104	F	1880	1945	05	1931	15	1931	13900	E4	0.0	Z9	449	0	2727	0
03-105	M	1903	1957	05	1931	16	1951	2600	E4	0.0	Z9	490	0	3143	0
03-106	F	1876	1959	05	1931	16	1931	4600	B2	0.0	Z9	147	0	1388	0
03-107	F	1884	1957	05	1931	16	1931	3600	B2	0.0	Z9	115	0	1036	0
03-108	F	1875	1953	05	1931	16	1932	1900	E4	0.0	Z9	69	0	558	0
03-109	F	1904	1957	05	1931	18	1953	630	B2	0.0	Z9	125	0	1120	0
03-110	F	1899	1957	05	1931	20	1964	584	B1	0.0	Z9	143	0	1583	0
03-111	F	1909		05	1931	20	1973	879	B2	0.0	Z9C	244	0	3174	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
03-112	F	1899	1968	05	1931	26	1960	5310	B1	0.0	Z9	1212	0	13669	0
03-113	F	1914	1946	05	1931	38	1932	1300	E4	0.0	Z9	39	0	244	0
03-114	F	1901	1968	05	1931	36	1964	949	B1	0.0	Z9	231	0	2606	0
03-115	F	1911		05	1931	26	1973	745	B1	0.0	Z9C	206	0	2686	0
03-116	F	1907		05	1931	25	1973	1411	B1	0.0	Z9C	391	0	5088	0
03-117	M	1898	1957	05	1931	45	1953	1540	B2	0.0	Z9	303	0	1931	0
03-118	F	1898	1955	05	1931	41	1953	3090	B2	0.0	Z9	608	0	5159	0
03-119	F	1880	1960	05	1931	7	1959	1038	C2	0.0	Z9	233	0	2256	0
03-120	F	1879	1937	05	1931	11	1932	5300	E4	0.0	Z9	199	0	721	0
03-121	F	1911	1972	05	1931	9	1964	371	B1	0.0	Z9	91	0	1099	0
03-122	M	1908		05	1931	10	1931	6500	E4	0.0	Z9	92	0	856	0
03-123	M	1914	1937	05	1931	9	1931	9700	B2	0.0	Z9	139	0	361	0
03-124	M	1910		05	1931	9	1977	196	B2	0.0	Z9C	57	0	533	0
03-125	F	1913	1976	05	1931	11	1973	556	B1	0.0	Z9C	154	0	1983	0
03-126	F	1910	1965	05	1931	20	1965	1300	C2	0.0	Z9	323	0	3449	0
03-127	F	1908		05	1931	26	1962	565	C2	0.0	Z9	134	0	1737	0
03-135	M	1905		05	1931	+0	1973	1431	B1	0.0	Z9C	398	0	3709	0
03-139	M	1908		05	1933	11	1973	373	C2	0.0	Z9C	101	0	913	0
03-140	M	1905	1937	05	1933	11	1961	500	F4	0.0	Z9	40	0	82	0
03-141	M	1906	1963	05	1933	11	1962	961	C2	0.0	Z9	220	0	1550	0
03-201	F	1909	1963	04	1922	+0	1962	2968	C2	0.0	Z9	805	0	9741	0
03-202	M	1895		05	1925	+0	1960	1800	G4	0.0	Z9	455	0	4599	0
03-203	F	1903	1973	05	1933	+0	1959	84	C2	0.0	Z9	18	0	217	0
03-204	F	1896	1970	04	1922	+0	1960	21	C2	0.0	Z9	6	0	74	0
03-205	F	1900		05	1929	15	1968	291	C2	0.0	Z9	78	0	1041	0
03-206	M	1914	1975	05	1936	4	1973	3297	B1	0.0	Z9C	858	0	7176	0
03-207	F	1879	1969	04	1922	416	1960	755	C2	0.0	Z9	188	0	2344	0
03-209	M	1894	1960	05	1925	572	1973	1105	A1	0.0	Z9A	254	0	1776	0
03-210	M	1906	1958	05	1926	+0	1957	1350	C2	0.00089	F2	321	12	2360	132
03-211	M	1890		05	1923	20	1960	10	C3	0.0	Z9	3	0	27	0
03-212	F	1902	1951	04	1927	+0	1951	1300	B2	0.00130	F1	270	7	2317	95
03-213	F	1892	1955	05	1925	+0	1952	6570	B2	0.0	Z9	1452	0	14358	0
03-214	F	1895	1966	05	1925	+0	1964	1382	C2	0.0	Z9F	370	0	4477	0
03-215	M	1896	1971	05	1925	+0	1961	3630	C2	0.0	Z9	932	0	8685	0
03-216	F	1907	1961	05	1922	+0	1961	530	C2	0.0	Z9F	142	0	1662	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP TYPE	YEAR FIRST EXP	EXP DUR WKS	YEAR OF MEAS	RA226 NCI	RA226 METHOD + ERR	RA228 TO RA226 RATIO	RA228 METHOD + ERR	INPUT RA226 UCI	INPUT RA228 UCI	CUM RADS RA226	CUM RADS RA228
03-217	M	1912	1974	05	1921	+0	1963	460	C2	0.0	Z9	128	0	1308	0
03-218	M	1908		05	1924	+0	1972	3	B3	0.0	Z9C	1	0	10	0
03-219	F	1888	1961	04	1919	+0	1951	60	B2	0.0	Z9	14	0	178	0
03-220	M	1920		04	1928	208	1976	130	B1	0.0	Z9C	38	0	357	0
03-221	M	1908	1963	05	1924	+0	1957	620	C2	0.0	Z9	152	0	1273	0
03-222	M	1872	1954	05	1922	+0	1951	1600	B2	0.0	Z9	367	0	2702	0
03-223	F	1886	1968	05	1929	156	1951	4200	B2	0.0	Z9	804	0	9181	0
03-224	M	1869	1960	54	1922	364	1951	5400	B2	0.0	Z9	1155	0	8929	0
03-225	M	1922		04	1929	+0	1977	31	B1	0.0	Z9C	9	0	89	0
03-226	M	1874	1953	05	1934	39	1951	10700	B2	0.0	Z9	1837	0	9588	0
03-227	F	1878	1952	05	1930	+0	1952	1000	B2	0.0	Z9	199	0	1612	0
03-228	M	1900	1955	05	1927	+0	1951	5600	B2	0.0	Z9	1164	0	7866	0
03-230	F	1899		05	1927	+0	1976	438	B1	0.0	Z9C	132	0	1817	0
03-231	F	1879	1973	05	1939	+0	1952	60	E4	0.0	Z9	9	0	97	0
03-232	F	1898	1957	05	1917	+0	1956	4700	D2	0.0	Z9	1257	0	14981	0
03-233	F	1879	1947	05	1922	+0	1947	4000	C4	0.0	Z9	849	0	7473	0
03-234	F	1890	1965	05	1915	+0	1965	920	C2	0.0	Z9	280	0	3861	0
03-235	F	1900	1968	05	1928	+0	1965	1290	C2	0.0	Z9	336	0	4001	0
03-236	F	1880	1961	05	1927	+0	1951	500	B2	0.0	Z9	104	0	1114	0
03-237	F	1890		04	1923	156	1961	3	C6	0.0	Z9	1	0	11	0
03-238	M	1883	1954	05	1926	+0	1951	13900	B2	0.0	Z9	2951	0	19944	0
03-239	F	1883	1953	05	1925	+0	1970	10000	A1	0.0	Z9A	2252	0	21306	0
03-240	F	1916	1955	05	1930	+0	1973	4320	A1	0.0	Z9A	917	0	8071	0
03-401	F	1900	1963	01	1923	95	1960	2287	C2	0.0	Z9	588	0	6896	0
03-402	F	1905		01	1923	260	1974	1223	B1	0.00010	F2	370	15	5271	220
03-403	F	1915	1964	01	1935	572	1957	8	C3	0.0	Z9	1	0	11	0
03-404	F	1897		01	1923	195	1975	577	B1	0.0	Z9C	177	0	2513	0
03-405	F	1904		16	1924	273	1962	625	C2	0.0	Z9	159	0	2200	0
03-406	F	1914		01	1935	481	1972	7	B3	0.0	Z9C	2	0	19	0
03-407	F	1905	1961	01	1923	1196	1958	1545	B1	0.00022	F2	382	5	4286	73
03-408	F	1908	1959	01	1924	676	1957	160	C2	0.0	Z9	39	0	414	0
03-409	F	1923		01	1942	78	1972	8	B2	0.0	Z9C	2	0	21	0
03-410	F	1895	1974	01	1923	104	1957	60	C2	0.0	Z9	15	0	203	0
03-411	F	1908		01	1931	572	1976	1	B3	0.0	Z9C	0	0	5	0
03-412	F	1894		01	1922	134	1977	227	B2	0.0	Z9C	72	0	1037	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
03-413	F	1917		01	1939	169	1972	1	B6	0.0	Z9C	0	0	2	0
03-414	F	1921		01	1946	557	1972	3	B6	0.0	Z9C	1	0	5	0
03-415	F	1911	1973	01	1930	780	1957	15	C3	0.0	Z9	3	0	30	0
03-416	F	1907		01	1923	65	1977	917	B1	0.0	Z9C	289	0	4155	0
03-417	F	1909	1966	01	1924	60	1964	617	C2	0.0	Z9	166	0	2023	0
03-418	F	1896		61	1926	602	1972	4	B3	0.0	Z9C	1	0	14	0
03-419	F	1906		01	1924	208	1962	679	C2	0.0	Z9	177	0	2500	0
03-420	F	1906	1960	01	1922	212	1957	18	C2	0.0	Z9	4	0	49	0
03-421	F	1908		01	1924	117	1974	5	B3	0.0	Z9C	2	0	21	0
03-422	F	1907		06	1925	104	1975	11	B2	0.0	Z9C	3	0	47	0
03-423	F	1907	1972	01	1923	641	1962	591	C2	0.0	Z9	155	0	2064	0
03-424	F	1905		01	1923	186	1976	277	B2	0.0	Z9C	86	0	1219	0
03-425	F	1916		01	1935	260	1973	2	B6	0.0	Z9C	1	0	6	0
03-426	F	1906		01	1924	2184	1972	139	B1	0.0	Z9C	41	0	580	0
03-427	F	1906		01	1925	823	1973	12	B2	0.0	Z9C	4	0	52	0
03-428	F	1908		01	1925	164	1974	493	B1	0.0	Z9C	148	0	2074	0
03-429	F	1908	1976	01	1923	208	1974	1169	B1	0.0	Z9C	354	0	4975	0
03-430	F	1922		01	1941	468	1971	4	B3	0.0	Z9C	1	0	10	0
03-431	F	1901		01	1922	156	1963	1297	C2	0.0	Z9	349	0	5032	0
03-432	F	1902		01	1923	112	1977	24	C2	0.0	Z9C	7	0	106	0
03-433	F	1904		01	1924	117	1964	1052	C2	0.0	Z9	281	0	3980	0
03-434	F	1920		01	1941	125	1975	5	B2	0.0	Z9C	1	0	13	0
03-435	F	1912		01	1935	104	1971	3	B6	0.0	Z9C	1	0	8	0
03-436	F	1910		01	1926	619	1975	8	B3	0.0	Z9C	2	0	30	0
03-437	F	1906		01	1926	52	1957	55	C2	0.0	Z9	13	0	180	0
03-438	F	1908		01	1925	8	1957	0	C6	0.0	Z9	0	0	0	0
03-439	F	1906		01	1925	56	1957	0	C6	0.0	Z9	0	0	0	0
03-440	F	1908		01	1925	3	1974	1	B6	0.0	Z9C	0	0	4	0
03-441	F	1905		01	1925	528	1957	56	C2	0.0	Z9	13	0	188	0
03-442	F	1904		01	1924	13	1976	4	B2	0.0	Z9	1	0	17	0
03-443	F	1914		01	1935	316	1971	0	B6	0.0	Z9C	0	0	0	0
03-444	F	1907		01	1925	56	1977	11	C3	0.0	Z9C	3	0	49	0
03-445	F	1905	1974	01	1922	260	1966	1367	C2	0.0	Z9	380	0	5237	0
03-446	F	1903		01	1921	260	1977	65	B1	0.0	Z9C	20	0	293	0
03-447	F	1906		01	1924	4	1958	2	C6	0.0	Z9	1	0	7	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
03-448	F	1903	1963	01	1924	19	1958	25	C2	0.0	Z9	6	0	73	0
03-449	F	1905	1974	01	1922	1456	1964	1135	B1	0.0	Z9	308	0	4239	0
03-450	F	1910		01	1924	697	1974	8	B2	0.0	Z9C	2	0	33	0
03-451	F	1922		01	1940	524	1972	1	B6	0.0	Z9C	0	0	2	0
03-452	F	1909		16	1925	728	1977	13	B2	0.0	Z9C	4	0	49	0
03-453	F	1907		01	1924	8	1976	3	B2	0.0	Z9C	1	0	13	0
03-454	F	1914		06	1934	572	1958	48	C2	0.0	Z9	9	0	98	0
03-455	F	1906		01	1922	56	1975	491	B1	0.00054	F1	153	49	2233	737
03-456	F	1921	1965	01	1943	416	1958	33	C2	0.0	Z9	4	0	32	0
03-457	F	1915		01	1939	520	1972	1	B6	0.0	Z9C	0	0	2	0
03-458	F	1925		01	1946	1560	1976	33	B2	0.0	Z9C	4	0	24	0
03-459	F	1906		01	1924	43	1976	774	B1	0.0	Z9C	239	0	3410	0
03-460	F	1905		01	1923	19	1977	4	C6	0.0	Z9C	1	0	17	0
03-461	F	1896		01	1922	6	1958	6	C3	0.0	Z9	2	0	23	0
03-462	F	1906		01	1922	2756	1975	240	B1	0.0	Z9C	74	0	1057	0
03-463	F	1918	1966	01	1942	832	1958	33	C2	0.0	Z9	3	0	18	0
03-464	F	1907		01	1923	104	1974	0	C6	0.0	Z9C	0	0	2	0
03-465	F	1908		01	1925	8	1976	5	B2	0.0	Z9	2	0	22	0
03-466	F	1904		01	1924	10	1976	2	B3	0.0	Z9C	1	0	8	0
03-467	F	1911		01	1926	416	1976	8	B2	0.0	Z9C	2	0	30	0
03-468	F	1908		01	1926	121	1958	29	C2	0.0	Z9	7	0	94	0
03-469	F	1903	1960	01	1925	30	1958	10	C3	0.0	Z9	2	0	27	0
03-470	F	1926		01	1943	247	1971	3	B3	0.0	Z9C	1	0	7	0
03-471	F	1908		01	1926	91	1958	13	C3	0.0	Z9	3	0	43	0
03-472	F	1922		01	1941	247	1972	5	B3	0.0	Z9C	1	0	13	0
03-473	F	1904	1965	01	1922	156	1962	1170	C2	0.0	Z9	311	0	3793	0
03-474	F	1909		01	1925	21	1958	19	C2	0.0	Z9	5	0	66	0
03-475	F	1903	1962	01	1921	65	1958	0	C6	0.0	Z9	0	0	0	0
03-476	F	1895	1970	01	1927	6	1958	0	C6	0.0	Z9	0	0	0	0
03-477	F	1911		01	1925	11	1972	3	B3	0.0	Z9C	1	0	12	0
03-478	F	1907		01	1924	8	1958	5	C6	0.0	Z9	1	0	18	0
03-479	F	1908		01	1924	52	1975	27	B1	0.00017	F2	8	1	117	10
03-480	F	1909		01	1924	10	1975	2	B3	0.0	Z9	1	0	9	0
03-481	F	1922		01	1942	481	1972	9	B2	0.0	Z9C	2	0	18	0
03-482	F	1927		01	1944	130	1972	3	B6	0.0	Z9C	1	0	6	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) ECRN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
03-483	F	1901		01	1922	177	1975	1	B6	0.0	Z9C	0	0	4	0
03-484	F	1888	1966	01	1919	156	1962	1622	C2	0.0	Z9	448	0	5807	0
03-485	F	1909	1977	01	1929	364	1958	0	C6	0.0	Z9	0	0	0	0
03-486	F	1909		01	1925	156	1977	208	B1	0.0	Z9	64	0	905	0
03-487	F	1907	1964	61	1924	676	1958	367	C2	0.0	Z9	90	0	1055	0
03-488	F	1907	1975	01	1922	26	1958	170	C2	0.0	Z9	43	0	621	0
03-489	F	1911	1964	01	1926	73	1958	120	C2	0.0	Z9	29	0	326	0
03-490	M	1904		07	1925	177	1973	5	B3	0.0	Z9C	1	0	13	0
03-491	F	1908		01	1924	2	1976	34	B1	0.0	Z9C	10	0	150	0
03-492	F	1928		01	1946	325	1973	5	B3	0.0	Z9C	1	0	9	0
03-493	F	1893		01	1920	199	1975	6	B3	0.0	Z9C	2	0	25	0
03-494	F	1902		01	1924	177	1959	4	C3	0.0	Z9	1	0	14	0
03-495	F	1910		01	1923	7	1976	0	B6	0.0	Z9C	0	0	2	0
03-496	F	1907		01	1923	8	1976	1	B6	0.0	Z9C	0	0	3	0
03-497	F	1903	1970	01	1923	260	1959	16	C2	0.0	Z9	4	0	52	0
03-498	F	1905		67	1923	1040	1976	2	B3	0.0	Z9C	1	0	6	0
03-499	F	1905		01	1924	56	1976	219	B1	0.00223	C3	68	79	962	1182
03-500	F	1901	1959	01	1922	8	1959	0	C6	0.0	Z9	0	0	0	0
03-501	F	1912		01	1928	8	1959	7	C3	0.0	Z9	2	0	23	0
03-502	F	1887	1964	01	1918	156	1959	170	C2	0.0	Z9	46	0	585	0
03-503	F	1894	1960	01	1922	112	1959	125	C2	0.0	Z9	32	0	362	0
03-504	F	1905		01	1922	30	1974	9	B2	0.0	Z9C	3	0	41	0
03-505	F	1907	1976	01	1923	1300	1975	169	B2	0.0	Z9C	52	0	725	0
03-506	F	1917		01	1935	1872	1975	9	B2	0.0	Z9C	2	0	14	0
03-507	F	1907	1962	01	1923	6	1959	12	C3	0.0	Z9	3	0	36	0
03-508	F	1905	1963	01	1923	8	1959	10	C3	0.0	Z9	3	0	31	0
03-509	F	1907		01	1924	2548	1973	28	B1	0.0	Z9C	8	0	117	0
03-510	F	1907	1977	01	1923	2028	1962	729	C2	0.0	Z9	191	0	2719	0
03-511	F	1910		01	1946	673	1959	10	C3	0.0	Z9	1	0	7	0
03-512	F	1906		01	1925	26	1959	11	C3	0.0	Z9	3	0	38	0
03-513	F	1908		01	1925	48	1974	73	B1	0.0	Z9	22	0	309	0
03-514	F	1909		01	1925	208	1959	26	C2	0.0	Z9	6	0	90	0
03-515	F	1908		01	1925	156	1959	11	C3	0.0	Z9	3	0	38	0
03-516	F	1911		01	1925	624	1976	7	B2	0.0	Z9C	2	0	32	0
03-517	F	1922		01	1943	260	1972	1	B6	0.0	Z9C	0	0	1	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
03-518	F	1921		01	1940	464	1972	8	B3	0.0	Z9C	2	0	17	0
03-519	F	1903		01	1924	8	1959	98	C2	0.0	Z9	25	0	354	0
03-520	F	1907		01	1925	780	1974	112	C2	0.0	Z9	33	0	470	0
03-521	F	1907	1961	01	1925	39	1959	10	C3	0.0	Z9	2	0	27	0
03-522	F	1898		01	1921	52	1977	88	B1	0.0	Z9C	28	0	416	0
03-523	F	1900		01	1923	30	1977	9	B2	0.0	Z9C	3	0	41	0
03-524	F	1903		01	1925	260	1972	48	B2	0.0	Z9C	14	0	196	0
03-525	F	1911	1976	01	1931	2132	1959	19	C2	0.0	Z9	3	0	25	0
03-526	F	1896		01	1925	52	1959	0	C6	0.0	Z9	0	0	0	0
03-527	F	1909		01	1925	130	1959	5	C3	0.0	Z9	1	0	17	0
03-528	F	1904		01	1922	524	1959	1630	C2	0.0	Z9	412	0	5901	0
03-529	F	1902		01	1921	104	1977	74	C2	0.0	Z9C	24	0	349	0
03-530	F	1907	1965	01	1923	91	1963	474	C2	0.0	Z9	127	0	1541	0
03-531	F	1906		01	1925	403	1959	41	C2	0.0	Z9	10	0	142	0
03-532	F	1910		01	1926	190	1977	43	C2	0.0	Z9C	13	0	176	0
03-533	F	1908		01	1925	260	1974	15	B1	0.0	Z9C	4	0	61	0
03-534	F	1910		01	1925	104	1976	3	B3	0.0	Z9	1	0	14	0
03-535	F	1907		01	1922	21	1964	227	C2	0.0	Z9	63	0	922	0
03-536	F	1910		01	1925	7	1959	35	C2	0.0	Z9	9	0	123	0
03-537	F	1900		07	1916	52	1977	1	C6	0.0	Z9C	0	0	6	0
03-538	F	1909	1976	01	1927	13	1959	61	C2	0.0	Z9	15	0	200	0
03-539	F	1900		01	1922	20	1974	5	B2	0.0	Z9C	2	0	25	0
03-540	F	1904		01	1923	364	1973	1605	B1	0.0	Z9C	481	0	6844	0
03-541	F	1913		01	1935	156	1973	1	B6	0.0	Z9C	0	0	2	0
03-542	F	1904		01	1924	13	1974	24	B1	0.0	Z9C	7	0	105	0
03-543	F	1918		01	1947	100	1972	1	B6	0.0	Z9C	0	0	2	0
03-544	F	1906	1975	01	1922	26	1959	5	C3	0.0	Z9	1	0	19	0
03-545	F	1898		01	1920	208	1959	0	C6	0.0	Z9	0	0	0	0
03-546	F	1903		01	1925	52	1959	95	C2	0.0	Z9	23	0	330	0
03-547	F	1907	1962	01	1923	108	1959	19	C2	0.00370	F2	5	1	55	19
03-548	F	1906		01	1922	17	1971	80	B1	0.0	Z9C	24	0	352	0
03-549	F	1910		01	1925	936	1977	43	C2	0.0	Z9C	13	0	189	0
03-550	F	1900		01	1917	104	1977	9	B3	0.0	Z9C	3	0	44	0
03-551	F	1903		01	1922	338	1973	1077	C2	0.0	Z9C	324	0	4645	0
03-552	F	1904		01	1924	108	1972	123	B1	0.0	Z9C	36	0	513	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
03-553	F	1904		01	1924	13	1974	5	B2	0.0	Z9C	2	0	23	0
03-554	F	1899	1977	01	1924	433	1961	2000	G4	0.0	Z9	513	0	7258	0
03-555	F	1913		71	1930	260	1972	2	B6	0.0	Z9C	1	0	8	0
03-556	F	1911		01	1928	100	1976	2	B3	0.0	Z9C	1	0	9	0
03-557	F	1910	1978	01	1925	3	1959	0	C6	0.0	Z9	0	0	0	0
03-558	F	1904	1971	01	1923	13	1959	115	C2	0.02173	C6	29	50	395	755
03-559	F	1907	1975	01	1922	21	1959	17	C2	0.0	Z9	4	0	63	0
03-561	F	1909		61	1924	416	1959	67	C2	0.0	Z9	17	0	236	0
03-562	F	1908		01	1927	520	1972	4	B3	0.0	Z9C	1	0	13	0
03-563	F	1909		01	1924	10	1975	2	B3	0.0	Z9C	1	0	11	0
03-564	F	1906		01	1923	3	1976	3	B2	0.0	Z9C	1	0	15	0
03-565	F	1913		01	1930	676	1972	5	B3	0.0	Z9C	1	0	16	0
03-566	F	1910		01	1930	624	1972	1	B6	0.0	Z9C	0	0	2	0
03-567	F	1900		01	1922	104	1972	26	B2	0.0	Z9C	8	0	112	0
03-568	F	1905	1977	01	1922	260	1959	120	C2	0.0	Z9	30	0	434	0
03-569	F	1901	1973	01	1922	312	1959	144	C2	0.0	Z9	36	0	495	0
03-570	F	1908		01	1925	43	1976	8	B2	0.0	Z9C	3	0	36	0
03-571	F	1909		01	1925	52	1976	636	B1	0.0	Z9C	195	0	2736	0
03-572	F	1906		01	1924	56	1977	62	C2	0.0	Z9C	19	0	277	0
03-573	F	1900		01	1925	52	1977	16	C6	0.0	Z9C	5	0	67	0
03-574	F	1904		71	1920	624	1976	1	B6	0.0	Z9C	0	0	3	0
03-575	F	1913		01	1931	52	1973	0	B6	0.0	Z9C	0	0	0	0
03-576	F	1909		01	1925	156	1976	4	B2	0.0	Z9C	1	0	16	0
03-577	F	1901	1961	01	1921	104	1959	81	C2	0.0	Z9	21	0	247	0
03-578	F	1909		01	1924	30	1976	8	B2	0.0	Z9	2	0	35	0
03-579	F	1905		01	1922	13	1959	30	C2	0.0	Z9	8	0	114	0
03-580	F	1904		01	1923	4	1959	2	C6	0.0	Z9	1	0	7	0
03-581	F	1904		01	1922	10	1959	13	C3	0.0	Z9	3	0	50	0
03-583	M	1893	1962	07	1930	+0	1959	50	C2	0.0	Z9	11	0	84	0
03-584	F	1905	1959	01	1923	+0	1959	6000	A4	0.0	Z9	1540	0	17131	0
03-585	F	1894		01	1918	260	1966	74	C2	0.0	Z9	21	0	320	0
03-586	F	1908	1968	01	1926	82	1967	900	C2	0.0	Z9	245	0	2972	0
03-587	F	1906		01	1925	34	1959	13	C3	0.0	Z9	3	0	45	0
03-588	F	1901	1967	01	1922	229	1962	316	C2	0.0	Z9	83	0	1041	0
03-589	F	1906	1969	01	1924	21	1959	77	C2	0.0	Z9	19	0	249	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
03-590	F	1900		01	1922	26	1965	104	C2	0.0	Z9	29	0	427	0
03-591	F	1907		17	1926	2340	1976	5	B2	0.0	Z9C	1	0	10	0
03-592	F	1905		01	1922	78	1966	123	C2	0.0	Z9	35	0	505	0
03-593	F	1905		01	1922	10	1977	10	C3	0.0	Z9C	3	0	45	0
03-594	F	1905	1968	01	1922	52	1959	41	C2	0.0	Z9	11	0	137	0
03-595	F	1902		01	1927	52	1975	1	B6	0.0	Z9C	0	0	4	0
03-596	F	1904		01	1922	8	1975	12	B2	0.0	Z9C	4	0	53	0
03-597	F	1903		16	1925	1300	1972	74	B1	0.0	Z9C	18	0	216	0
03-598	M	1890		07	1933	4	1971	1	B6	0.0	Z9C	0	0	2	0
03-599	F	1906	1975	01	1922	26	1959	9	C3	0.0	Z9	2	0	33	0
03-600	F	1902		07	1926	988	1972	0	B6	0.0	Z9C	0	0	0	0
03-601	F	1893	1969	01	1925	260	1960	6	C3	0.0	Z9	2	0	19	0
03-602	F	1899		01	1925	104	1960	3	C6	0.0	Z9	1	0	11	0
03-603	F	1888		01	1924	520	1960	0	C6	0.0	Z9	0	0	0	0
03-604	F	1899		01	1916	624	1976	2	B3	0.0	Z9C	1	0	9	0
03-605	F	1900		01	1921	364	1972	1	B6	0.0	Z9C	0	0	3	0
03-606	F	1903		01	1924	6	1971	2	B6	0.0	Z9C	1	0	8	0
03-607	F	1906		01	1922	26	1977	77	B2	0.0	Z9C	24	0	357	0
03-608	F	1895	1976	01	1919	104	1960	19	C2	0.0	Z9	5	0	76	0
03-609	F	1896	1974	01	1923	4	1960	0	C6	0.0	Z9	0	0	0	0
03-610	F	1917		01	1935	104	1973	1	B6	0.0	Z9C	0	0	4	0
03-611	F	1893	1969	01	1916	208	1960	3	C6	0.0	Z9	1	0	12	0
03-612	F	1892	1968	01	1918	234	1960	500	C2	0.0	Z9	135	0	1806	0
03-613	F	1905		01	1925	95	1972	2	B6	0.0	Z9C	0	0	7	0
03-614	F	1909		01	1924	56	1975	94	B2	0.0	Z9	29	0	409	0
03-615	F	1905		01	1923	107	1975	14	B1	0.0	Z9	4	0	63	0
03-617	F	1902	1951	01	1921	312	1963	7000	F4	0.0	Z9	1560	0	14586	0
03-618	F	1893	1969	01	1920	43	1960	10	C3	0.0	Z9	3	0	36	0
03-619	F	1903	1962	01	1922	34	1962	1576	C3	0.00144	F1	425	76	5041	1143
03-620	F	1923		01	1942	208	1971	5	B3	0.0	Z9C	1	0	11	0
03-621	F	1916		01	1944	208	1971	4	B3	0.0	Z9C	1	0	9	0
03-622	F	1910		01	1926	104	1960	0	G6	0.0	Z9	0	0	0	0
03-623	F	1902		01	1924	+0	1960	4	G6	0.0	Z9	1	0	15	0
03-624	F	1905	1959	01	1923	156	1959	1000	A4	0.0	Z9	251	0	2716	0
03-625	F	1901		01	1923	13	1976	1	B6	0.0	Z9C	0	0	2	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
03-626	F	1906		01	1924	208	1960	200	G4	0.0	Z9	51	0	715	0
03-627	F	1905	1966	01	1924	208	1960	50	G4	0.0	Z9	13	0	153	0
03-628	F	1905	1974	01	1921	34	1962	0	C6	0.0	Z9	0	0	0	0
03-629	F	1903	1969	01	1922	+0	1960	0	G6	0.0	Z9	0	0	0	0
03-630	F	1908		01	1924	17	1974	19	B1	0.0	Z9C	6	0	80	0
03-632	F	1905	1975	01	1922	0	1960	0	G6	0.0	Z9	0	0	0	0
03-633	F	1902		01	1922	780	1960	20	G6	0.0	Z9	5	0	73	0
03-634	F	1909	1961	01	1924	+0	1960	3	G6	0.0	Z9	1	0	9	0
03-635	F	1907		01	1925	+0	1960	47	G6	0.0	Z9	12	0	168	0
03-636	F	1904		01	1924	192	1976	5	B2	0.0	Z9C	2	0	23	0
03-637	F	1906		01	1924	6	1976	40	B1	0.0	Z9C	12	0	177	0
03-638	F	1902	1972	01	1924	+0	1960	7	G6	0.0	Z9	2	0	24	0
03-639	F	1912		01	1925	156	1960	67	G4	0.0	Z9	17	0	236	0
03-640	F	1902		01	1924	60	1960	5	C3	0.0	Z9	1	0	18	0
03-641	F	1904		01	1922	26	1976	8	B2	0.0	Z9C	3	0	38	0
03-642	F	1905		01	1922	52	1976	31	B2	0.0	Z9C	10	0	144	0
03-643	F	1909		01	1926	156	1975	10	B2	0.0	Z9C	3	0	39	0
03-645	F	1906		01	1924	312	1959	56	C2	0.0	Z9	14	0	197	0
03-646	F	1888		01	1926	+0	1960	0	G6	0.0	Z9	0	0	0	0
03-647	F	1901		01	1925	5	1960	35	G6	0.0	Z9	9	0	125	0
03-648	F	1903	1956	01	1922	155	1956	5000	B2	0.00430	F2	1216	271	12670	4043
03-649	F	1906	1954	01	1924	1352	1951	1300	B2	0.0	Z9F	282	0	2725	0
03-671	F	1906	1953	01	1922	8	1952	3820	B2	0.00500	F1	890	169	8980	2525
03-672	F	1899		01	1924	+0	1960	3	G6	0.0	Z9	1	0	11	0
03-673	F	1909		71	1926	8	1960	35	G6	0.0	Z9	9	0	121	0
03-674	F	1908		01	1925	43	1976	2	B3	0.0	Z9C	1	0	9	0
03-676	F	1897	1977	01	1924	+0	1963	1700	C2	0.0	Z9	455	0	6514	0
03-677	M	1899	1965	06	1924	+0	1961	232	G4	0.0	Z9	60	0	522	0
03-678	M	1919		71	1953	988	1972	6	B3	0.0	Z9C	1	0	2	0
03-679	F	1910		01	1930	10	1977	1	B3	0.0	Z9C	0	0	5	0
03-681	F	1906		01	1922	6	1962	1	G6	0.0	Z9	0	0	2	0
03-682	F	1907		01	1925	60	1973	1	B6	0.0	Z9C	0	0	5	0
03-683	F	1906		01	1923	0	1961	0	C6	0.0	Z9	0	0	0	0
03-684	F	1907		01	1927	17	1977	1	B6	0.0	Z9C	0	0	5	0
03-685	F	1902		01	1921	65	1976	70	B1	0.0	Z9C	22	0	327	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD ± ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD ± ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
03-686	F	1904		01	1923	1040	1975	20	B2	0.0	Z9C	6	0	85	0
03-687	F	1900	1974	01	1925	43	1961	51	C2	0.0	Z9	13	0	176	0
03-688	F	1918		01	1936	260	1972	3	B6	0.0	Z9C	1	0	7	0
03-689	F	1903		01	1923	208	1977	84	B1	0.0	Z9	26	0	371	0
03-690	F	1909	1967	01	1924	290	1958	320	C2	0.0	Z9	78	0	965	0
03-692	M	1887	1976	07	1920	+0	1961	6	C3	0.0	Z9	2	0	17	0
03-693	F	1920		01	1942	520	1952	14	G6	0.0	Z9	1	0	9	0
03-695	F	1920		01	1942	34	1972	7	B3	0.0	Z9C	2	0	18	0
03-696	F	1932		01	1950	52	1963	0	C6	0.0	Z9	0	0	0	0
03-697	F	1902		01	1924	34	1967	181	C2	0.0	Z9	51	0	724	0
03-701	F	1907		01	1924	9	1977*	0	M6	0.0	Z9	0	0	0	0
03-703	F	1921		01	1946	416	1974	0	B6	0.0	Z9C	0	0	1	0
03-710	F	1907		01	1924	728	1977*	3	C6	0.0	Z9C	1	0	15	0
03-712	F	1922		01	1943	52	1977*	7	C3	0.0	Z9C	2	0	18	0
03-713	F	1921		01	1941	1456	1971	2	B6	0.0	Z9C	0	0	2	0
03-714	F	1923		01	1942	364	1971	3	B3	0.0	Z9C	1	0	7	0
03-716	F	1920	1976	01	1941	104	1971	0	B6	0.0	Z9C	0	0	0	0
03-717	F	1906	1977	01	1922	156	1977*	150	P6	0.0	Z9	47	0	682	0
03-720	F	1910		01	1926	52	1976	6	B2	0.0	Z9C	2	0	23	0
03-722	F	1905		01	1924	4	1977	3	B2	0.0	Z9C	1	0	12	0
03-726	F	1905	1972	01	1922	186	1968	574	C2	0.0	Z9	164	0	2206	0
03-727	F	1906	1977	01	1923	988	1972	165	B1	0.0	Z9B	49	0	696	0
03-729	F	1926		01	1943	208	1973	1	B6	0.0	Z9C	0	0	3	0
03-730	M	1894	1963	06	1923	+0	1961	7	C3	0.0	Z9	2	0	16	0
03-732	F	1924		01	1942	78	1973	2	B6	0.0	Z9C	0	0	4	0
03-736	F	1895		16	1919	22	1975	1	B6	0.0	Z9C	0	0	2	0
03-741	F	1908		01	1925	260	1975	4	B3	0.0	Z9C	1	0	15	0
03-748	F	1910		01	1927	+0	1977*	5	B2	0.0	Z9C	1	0	20	0
03-752	F	1904		01	1922	15	1977	10	B2	0.0	Z9C	3	0	48	0
03-753	F	1906		01	1922	+0	1977	12	B1	0.0	Z9C	4	0	57	0
03-757	F	1902		01	1923	91	1974	14	B2	0.0	Z9C	4	0	59	0
03-761	F	1901		01	1927	1144	1977	26	B2	0.0	Z9C	7	0	81	0
03-763	F	1901		01	1931	52	1976	0	C6	0.0	Z9C	0	0	0	0
03-764	F	1908		01	1926	364	1976	2	B3	0.0	Z9C	1	0	8	0
03-771	F	1900		01	1923	13	1977*	96	B1	0.0	Z9C	31	0	442	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
03-774	F	1909		01	1924	3	1977	1	B6	0.0	Z9C	0	0	3	0
03-775	F	1922		01	1942	52	1974	4	B3	0.0	Z9C	1	0	10	0
03-778	F	1904		01	1923	104	1973	54	B1	0.0	Z9C	16	0	235	0
03-782	F	1908		01	1923	5	1976	2	B3	0.0	Z9C	1	0	11	0
03-784	F	1905		01	1923	178	1954	750	C4	0.0	Z9	173	0	2469	0
03-788	F	1905		01	1926	104	1976	1	B6	0.0	Z9C	0	0	3	0
03-795	F	1897	1944	01	1926	78	1944	8	G6	0.0	Z9	1	0	10	0
03-796	F	1907		01	1925	2	1972	0	B6	0.0	Z9C	0	0	1	0
03-801	F	1906		01	1924	13	1976	2	B3	0.0	Z9C	1	0	10	0
03-807	F	1923		01	1954	780	1973	0	B6	0.0	Z9C	0	0	0	0
03-810	F	1919		01	1934	312	1972	2	B6	0.0	Z9C	0	0	5	0
03-817	F	1907		01	1926	13	1973	0	B6	0.0	Z9C	0	0	2	0
03-818	F	1902		01	1927	62	1975	4	B3	0.0	Z9C	1	0	17	0
03-825	F	1906		01	1922	4	1976	1	B3	0.0	Z9C	0	0	5	0
03-828	M	1915		17	1950	936	1972	0	B6	0.0	Z9C	0	0	0	0
03-834	F	1907		01	1925	+0	1976	1	B3	0.0	Z9C	0	0	6	0
03-836	F	1908		01	1924	23	1967	0	C6	0.0	Z9	0	0	0	0
03-838	F	1928		01	1947	130	1975	2	B3	0.0	Z9C	1	0	5	0
03-842	F	1910		01	1926	416	1976	3	B2	0.0	Z9C	1	0	12	0
03-845	F	1908		01	1925	104	1974	0	B6	0.0	Z9C	0	0	1	0
03-850	F	1923		01	1942	78	1976	13	B1	0.0	Z9C	3	0	34	0
05-001	F	1900		01	1919	52	1975	45	B1	0.00056	Z7B	14	7	219	103
05-002	F	1903	1973	01	1917	104	1971	1	B6	0.0	Z9B	0	0	5	0
05-003	F	1900	1959	01	1917	8	1958	0	G6	0.0	Z9	0	0	0	0
05-004	F	1904		01	1920	104	1959	12	G6	0.01600	Z7	3	5	47	77
05-005	F	1901		01	1916	13	1960	0	G6	0.0	Z9	0	0	0	0
05-007	F	1896		01	1920	95	1967	23	B2	0.00600	Z7B	7	11	100	164
05-008	M	1894	1964	07	1916	104	1963	4	CL	0.0	Z9C	1	0	11	0
05-010	F	1901	1974	01	1921	34	1961	4	CL	0.01200	Z7C	1	2	15	24
05-011	F	1902		01	1917	52	1959	12	G6	0.0	Z9	3	0	51	0
05-012	F	1901	1959	01	1917	52	1970	16	A1	0.0	Z9A	4	0	54	0
05-014	F	1900		01	1916	208	1977	106	B1	0.00083	B6	35	38	542	570
05-015	F	1891		01	1916	67	1970	1	B6	0.0	Z9B	0	0	5	0
05-016	M	1891	1965	06	1916	100	1958	15	G4	0.0	Z9	4	0	40	0
05-017	F	1894		01	1919	+0	1968	5	G6	0.00520	Z7	2	3	23	46

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
05-018	M	1886		06	1918	156	1971	4	B3	0.00180	Z7B	1	1	13	12
05-019	F	1885	1958	01	1921	2	1960	0	G6	0.01400	Z7	0	0	0	0
05-020	F	1898		01	1917	52	1959	3	G6	0.0	Z9	1	0	13	0
05-022	F	1900	1969	07	1916	32	1964	4	CL	0.0	Z9C	1	0	17	0
05-023	F	1899	1960	01	1918	104	1960	38	C2	0.00320	Z7C	10	5	126	73
05-024	M	1890	1965	06	1916	208	1961	4	CL	0.01200	Z7C	1	2	11	27
05-025	F	1893		01	1917	78	1971	86	B1	0.00020	Z7B	27	4	417	53
05-037	F	1898	1977	01	1916	260	1971	2	B6	0.0	Z9B	1	0	10	0
05-038	F	1901		07	1916	156	1972	99	G4	0.0	Z9	32	0	487	0
05-039	F	1899		01	1917	156	1977	20	B1	0.00062	Z7B	7	5	101	75
05-040	F	1899		01	1917	54	1971	10	B2	0.0	Z9B	3	0	49	0
05-042	F	1918		01	1940	130	1972	1	B6	0.0	Z9B	0	0	3	0
05-043	M	1888	1960	06	1919	208	1965	0	F6	0.00430	Z7F	0	0	0	0
05-044	M	1895	1975	06	1915	468	1971	2	B6	0.0	Z9B	1	0	7	0
05-045	F	1899	1960	01	1917	60	1965	5	F4	0.0	Z9F	1	0	17	0
05-049	F	1905		01	1923	13	1965	6	C3	0.0	Z9C	2	0	24	0
05-072	M	1893	1950	07	1919	13	1976	0	A6	0.00100	Z7	0	0	0	0
05-088	F	1886		01	1917	4	1959	4	G6	0.0	Z9	1	0	17	0
05-089	F	1900		01	1916	78	1971	13	B2	0.0	Z9B	4	0	64	0
05-092	F	1901		01	1916	104	1959	6	G6	0.0	Z9	2	0	26	0
05-093	F	1897	1974	71	1915	78	1961	6	C6	0.0	Z9C	2	0	26	0
05-094	F	1927		01	1946	39	1973	6	B3	0.0	Z9B	1	0	13	0
05-096	F	1901	1971	01	1918	26	1962	234	C2	0.00050	Z7C	66	7	949	102
05-097	M	1892	1976	06	1918	26	1961	4	CL	0.00050	Z7C	1	0	12	1
05-100	F	1907		01	1919	156	1968	4	G6	0.00520	Z7	1	2	18	30
05-101	F	1902		01	1924	6	1964	4	CL	0.00850	Z7C	1	1	16	18
05-102	F	1900		01	1915	364	1960	6	C6	0.00350	Z7C	2	1	25	13
05-103	F	1906		01	1923	4	1959	1	G6	0.01600	Z7	0	0	4	5
05-104	F	1900		01	1918	13	1964	4	CL	0.00040	Z7C	1	0	18	2
05-105	M	1903	1959	07	1918	30	1959	0	G6	0.00070	Z7	0	0	0	0
05-111	M	1895	1977	07	1920	312	1970	5	G6	0.00660	Z7	1	3	15	31
05-116	F	1898	1959	01	1917	52	1972	19	A1	0.0	Z9A	5	0	64	0
05-117	M	1887	1968	06	1915	208	1964	4	CL	0.0	Z9C	1	0	12	0
05-118	F	1901		01	1917	65	1977	2	B3	0.0	Z9B	1	0	10	0
05-119	F	1905		01	1924	212	1977	10	B2	0.00175	Z7	3	3	44	46

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
05-120	F	1890		07	1919	6	1959	5	G6	0.00770	Z7	1	1	21	20
05-121	F	1906		01	1921	26	1970	9	B2	0.00390	Z7B	3	4	40	60
05-122	M	1879	1962	07	1922	208	1959	11	G6	0.01600	Z7	3	3	23	33
05-123	F	1897	1972	01	1918	1	1960	4	G6	0.00060	Z7	1	0	16	2
05-125	F	1902		07	1916	104	1959	26	G4	0.0	Z9	7	0	112	0
05-126	M	1889	1970	01	1921	52	1970	0	B6	0.0	Z9B	0	0	0	0
05-127	M	1893		06	1918	999	1967	20	B2	0.0	Z9B	5	0	52	0
05-129	F	1900	1969	07	1917	104	1960	4	CL	0.0	Z9C	1	0	16	0
05-130	F	1920		01	1940	78	1972	0	B6	0.0	Z9B	0	0	0	0
05-132	F	1898		07	1918	52	1969	0	G6	0.00020	Z7	0	0	0	0
05-133	M	1903	1967	07	1918	13	1959	0	G6	0.00070	Z7	0	0	0	0
05-134	F	1900		01	1917	6	1959	9	G6	0.0	Z9	3	0	39	0
05-135	F	1919		01	1941	106	1976	0	B6	0.0	Z9B	0	0	0	0
05-136	M	1896	1966	06	1917	78	1959	94	G4	0.0	Z9	26	0	249	0
05-138	F	1917		01	1941	104	1968	5	B3	0.0	Z9B	1	0	12	0
05-139	F	1891	1966	01	1919	70	1962	4	CL	0.00540	Z7C	1	1	15	16
05-140	F	1897	1960	01	1916	+0	1965	490	F4	0.0	Z9F	140	0	1770	0
05-142	F	1904		01	1919	39	1960	11	G6	0.00680	Z7	3	3	45	43
05-143	F	1899	1962	07	1918	+0	1961	4	CL	0.00050	Z7C	1	0	14	2
05-145	M	1883	1961	07	1916	572	1961	4	CL	0.00150	Z7C	1	0	9	2
05-146	M	1897		06	1920	286	1968	2	G6	0.00490	Z7	1	1	6	7
05-150	F	1899	1969	07	1917	6	1960	45	G6	0.0	Z9	13	0	179	0
05-151	F	1897		01	1924	95	1963	7	C3	0.00960	Z7C	2	2	26	27
05-154	F	1900		01	1916	11	1970	0	G6	0.0	Z9	0	0	0	0
05-155	F	1898	1965	07	1916	28	1963	4	CL	0.0	Z9C	1	0	16	0
05-160	F	1917		01	1942	156	1969	0	G6	0.0	Z9	0	0	0	0
05-161	M	1901		06	1918	9	1971	0	B6	0.00016	Z7B	0	0	0	0
05-162	F	1914		07	1942	+0	1960	29	G6	0.0	Z9	5	0	57	0
05-163	M	1912	1970	07	1941	104	1960	35	G6	0.0	Z9	6	0	42	0
05-165	F	1899	1964	01	1919	13	1972	1	A6	0.0	Z9A	0	0	3	0
05-172	F	1907	1960	01	1934	999	1960	24	G4	0.0	Z9	4	0	26	0
05-174	F	1902		01	1919	130	1977	0	M6	0.00126	Z7	0	0	0	0
05-179	F	1921		01	1940	182	1974	0	B6	0.0	Z9B	0	0	0	0
05-181	F	1901		01	1918	4	1970	0	B6	0.00018	Z7B	0	0	0	0
05-184	M	1901	1974	41	1922	156	1964	5	C6	0.0	Z9C	1	0	14	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
05-185	F	1912		01	1941	208	1972	2	B6	0.0	Z9B	0	0	5	0
05-186	F	1922		01	1941	156	1972	1	B6	0.0	Z9B	0	0	3	0
05-188	M	1889	1964	07	1917	104	1961	4	CL	0.0	Z9C	1	0	10	0
05-189	M	1890	1972	07	1921	104	1964	4	CL	0.00850	Z7C	1	2	11	17
05-197	M	1898		07	1919	7	1973	0	B6	0.00140	Z7B	0	0	0	0
05-199	F	1901		16	1917	2	1967	0	B6	0.0	Z9B	0	0	0	0
05-201	F	1919		01	1941	221	1976	6	B3	0.0	Z9B	1	0	16	0
05-203	F	1899		01	1919	52	1960	0	G6	0.00680	Z7	0	0	0	0
05-204	M	1880	1961	07	1918	78	1960	0	G6	0.00320	Z7	0	0	0	0
05-205	F	1907		01	1924	208	1961	4	CL	0.0	Z9C	1	0	15	0
05-206	F	1894		01	1922	52	1971	2	B6	0.00360	Z7B	1	1	9	12
05-207	M	1893		06	1917	+0	1962	6	G6	0.0	Z9	2	0	19	0
05-210	F	1899	1971	01	1916	158	1977	1060	A1	0.0	Z9A	334	0	4814	0
05-212	F	1903		07	1918	8	1965	4	CL	0.00030	Z7C	1	0	18	2
05-215	F	1886	1968	01	1920	52	1969	1410	A1	0.00198	A3	418	301	5571	4528
05-237	M	1896	1969	06	1920	364	1961	4	CL	0.0	Z9C	1	0	10	0
05-246	F	1884	1969	06	1911	728	1962	4	CL	0.0	Z9C	1	0	16	0
05-251	F	1896		01	1917	34	1965	13	G4	0.0	Z9	4	0	60	0
05-252	F	1890	1976	01	1917	52	1964	4	CL	0.0	Z9C	1	0	18	0
05-255	M	1886	1966	07	1920	104	1964	5	C6	0.00850	Z7C	1	2	13	24
05-257	F	1895	1975	01	1932	1248	1972	3	G6	0.0	Z9	1	0	7	0
05-258	F	1901		01	1917	1	1970	0	G6	0.0	Z9	0	0	0	0
05-259	F	1900		07	1917	52	1960	6	G6	0.0	Z9	2	0	26	0
05-260	F	1898		07	1917	32	1960	0	G6	0.0	Z9	0	0	0	0
05-261	F	1892	1977	01	1943	104	1960	4	CL	0.0	Z9C	1	0	7	0
05-262	F	1917		01	1942	260	1972	3	B3	0.0	Z9C	1	0	7	0
05-263	M	1883	1967	07	1919	104	1962	4	CL	0.00800	Z7C	1	1	11	16
05-264	M	1903		07	1917	5	1961	4	CL	0.0	Z9C	1	0	13	0
05-265	M	1884	1963	07	1916	104	1962	4	CL	0.0	Z9C	1	0	11	0
05-266	M	1881	1970	07	1918	130	1964	4	CL	0.00200	Z7C	1	1	11	6
05-268	F	1893		01	1918	39	1960	4	CL	0.00060	Z7C	1	0	17	2
05-269	M	1887	1971	07	1918	52	1964	4	CL	0.00040	Z7C	1	0	12	1
05-270	M	1901		07	1916	52	1961	8	C3	0.0	Z9C	2	0	26	0
05-272	M	1895		06	1918	65	1972	0	B6	0.00014	Z7B	0	0	0	0
05-273	F	1889	1968	01	1918	104	1960	4	CL	0.01400	Z7C	1	2	15	34

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
05-274	F	1903		07	1920	4	1970	0	G6	0.0	Z9	0	0	0	0
05-276	F	1906		01	1921	75	1961	4	CL	0.01200	Z7C	1	2	16	23
05-277	M	1894	1973	06	1918	104	1960	4	CL	0.00320	Z7C	1	1	11	6
05-278	F	1893	1965	01	1917	52	1964	37	C2	0.0	Z9F	11	0	145	0
05-279	F	1896		01	1917	1820	1969	0	G6	0.0	Z9	0	0	0	0
05-281	F	1898	1964	01	1916	148	1963	660	B2	0.00216	F1	191	105	2519	1580
05-282	F	1898		01	1917	34	1964	8	C6	0.0	Z9C	2	0	36	0
05-284	F	1899	1973	01	1919	156	1969	218	B1	0.00080	Z7B	65	19	930	284
05-286	M	1901	1963	06	1916	104	1965	1	F4	0.0	Z9F	0	0	1	0
05-287	M	1889	1970	07	1917	390	1965	4	CL	0.00420	Z7C	1	1	11	11
05-288	F	1897		01	1918	10	1960	4	CL	0.00060	Z7C	1	0	17	2
05-290	F	1898	1967	01	1918	52	1960	8	C3	0.00060	Z7C	2	0	30	3
05-291	F	1902	1974	01	1920	8	1968	4	G6	0.00540	Z7	1	2	17	33
05-292	M	1904	1974	07	1918	+0	1965	4	CL	0.00033	Z7C	1	0	13	1
05-303	F	1894		01	1917	2184	1977*	1	M6	0.0	Z9	0	0	7	0
05-304	F	1897		01	1921	26	1962	4	CL	0.01100	Z7C	1	2	16	26
05-306	F	1903		01	1921	156	1976	3	B3	0.00195	Z7B	1	1	14	18
05-307	F	1920		01	1944	74	1972	0	B6	0.0	Z9B	0	0	0	0
05-308	M	1893	1964	07	1916	208	1962	4	CL	0.00130	Z7C	1	0	11	3
05-310	F	1894	1965	01	1916	78	1964	5	C6	0.0	Z9C	1	0	20	0
05-311	M	1887	1961	06	1920	156	1960	4	CL	0.01400	Z7C	1	2	9	17
05-312	M	1886	1961	01	1919	34	1961	2	F6	0.00610	Z7F	1	1	5	6
05-318	M	1901	1961	07	1918	+0	1965	4	F4	0.00030	Z7F	1	0	10	1
05-321	F	1899		01	1916	208	1966	16	G6	0.00330	Z7	5	5	73	80
05-322	M	1900	1975	07	1917	312	1973	4	B3	0.0	Z7B	1	0	13	0
05-323	F	1899	1961	01	1915	26	1961	2	A5	0.0	Z9	1	0	7	0
05-351	F	1891		01	1917	30	1968	23	G6	0.0	Z9	7	0	109	0
05-352	M	1900	1963	07	1917	40	1964	1	F6	0.0	Z9F	0	0	3	0
05-353	M	1900		07	1915	13	1973	1	B6	0.0	Z9B	0	0	4	0
05-357	F	1890		07	1917	104	1972	3	G6	0.0	Z9	1	0	15	0
05-360	M	1892	1968	01	1914	+0	1963	4	CL	0.0	Z9C	1	0	12	0
05-363	F	1899		07	1917	9	1964	4	CL	0.0	Z9C	1	0	18	0
05-368	F	1901		07	1917	104	1977	0	B6	0.0	Z9C	0	0	0	0
05-369	F	1901		07	1919	26	1973	1	B6	0.00140	Z7B	0	0	5	5
05-370	F	1895		01	1920	26	1965	4	CL	0.00760	Z7C	1	2	17	30

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
05-372	F	1888	1970	01	1916	104	1968	14	G4	0.0	Z9	4	0	62	0
05-374	F	1905		01	1923	8	1964	4	CL	0.00850	Z7C	1	1	16	20
05-377	F	1895	1974	01	1916	15	1969	0	G6	0.0	Z9	0	0	0	0
05-380	F	1904	1970	07	1925	104	1962	4	CL	0.01100	Z7C	1	1	13	13
05-383	F	1901		06	1917	165	1973	73	B1	0.00060	Z7B	23	10	354	156
05-387	M	1902		06	1918	9	1975	0	B6	0.00010	Z7B	0	0	0	0
05-395	F	1911		01	1928	728	1977	0	M6	0.0	Z9	0	0	0	0
05-397	F	1900	1976	07	1918	13	1962	4	CL	0.0	Z9C	1	0	17	0
05-399	M	1892		07	1916	104	1961	4	CL	0.0	Z9C	1	0	13	0
05-401	M	1898		76	1917	169	1971	5	B3	0.00170	Z7B	2	2	17	16
05-407	F	1898		01	1916	9	1973	1	B6	0.0	Z9B	0	0	5	0
05-409	F	1900		07	1918	61	1974	0	B6	0.00011	Z7B	0	0	0	0
05-410	F	1899		01	1916	26	1971	2	B6	0.0	Z9B	1	0	10	0
05-413	F	1900	1971	01	1916	39	1969	18	B2	0.0	Z9B	6	0	82	0
05-420	F	1889	1935	01	1917	104	1970	50	A1	0.0	Z9A	9	0	60	0
05-437	F	1888		07	1923	26	1971	3	B3	0.00350	Z7B	1	1	13	16
05-438	F	1907		01	1926	13	1961	4	CL	0.0	Z9C	1	0	14	0
05-439	F	1898	1970	01	1916	104	1967	200	G6	0.0	Z9	61	0	872	0
05-440	F	1896	1975	01	1922	1	1971	0	B6	0.00360	Z7B	0	0	0	0
05-442	F	1888		07	1917	6	1962	8	G6	0.0	Z9	2	0	36	0
05-443	F	1922		07	1941	52	1972	3	B6	0.0	Z9B	1	0	8	0
05-444	M	1899	1963	06	1917	43	1961	4	CL	0.0	Z9C	1	0	11	0
05-446	M	1888	1971	45	1925	+0	1964	4	CL	0.0	Z9C	1	0	10	0
05-447	F	1902		01	1916	9	1970	2	B6	0.0	Z9B	1	0	10	0
05-448	F	1903		01	1916	1	1961	4	CL	0.0	Z9C	1	0	18	0
05-449	F	1892	1961	01	1919	52	1961	4	CL	0.00610	Z7C	1	1	13	16
05-450	F	1903		07	1918	117	1971	1	B6	0.00090	Z7B	0	0	5	2
05-459	F	1917		01	1933	208	1961	8	C6	0.0	Z9C	2	0	21	0
05-460	F	1898		07	1916	182	1961	4	CL	0.0	Z9C	1	0	17	0
05-464	F	1895	1969	01	1917	+0	1968	5	G6	0.0	Z9	2	0	22	0
05-473	M	1899	1970	06	1921	26	1962	4	CL	0.01100	Z7C	1	2	11	18
05-528	F	1892		01	1917	52	1967	0	G6	0.0	Z9	0	0	0	0
05-541	F	1913		01	1937	884	1972	0	B6	0.0	Z9B	0	0	0	0
05-546	F	1902		01	1918	52	1973	1	B6	0.00012	Z7B	0	0	5	0
05-551	F	1895		01	1918	9	1970	15	G6	0.00018	Z7	5	0	71	7

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS IO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
05-555	F	1898	1965	07	1917	27	1975	1	A6	0.0	Z9	0	0	4	0
05-560	M	1894	1965	07	1921	260	1962	4	CL	0.01100	Z7C	1	1	9	13
05-574	F	1903		01	1918	1	1977*	0	M6	0.00008	Z7	0	0	0	0
05-580	M	1904	1975	07	1919	6	1968	4	G6	0.00260	Z7	1	1	13	13
05-602	M	1899		06	1925	1300	1975	0	B6	0.0	Z9B	0	0	0	0
05-611	F	1900	1938	01	1914	156	1974	0	A6	0.0	Z9A	0	0	0	0
05-631	F	1897	1976	01	1917	17	1970	0	G6	0.0	Z9	0	0	0	0
05-639	M	1906	1962	06	1922	39	1964	1	F6	0.00850	Z7F	0	0	2	4
05-674	M	1922		06	1946	156	1965	4	CL	0.0	Z9C	1	0	5	0
05-688	F	1921	1976	01	1939	130	1965	5	C6	0.0	Z9C	1	0	12	0
05-736	F	1898	1954	06	1918	156	1972	150	F4	0.00410	F1	38	91	407	1359
05-737	M	1895	1957	06	1918	156	1971	10	F4	0.00462	Z4F	3	6	21	68
05-742	F	1898	1975	01	1916	30	1969	0	G6	0.0	Z9	0	0	0	0
05-751	F	1901	1933	01	1920	+0	1969	0	A6	0.00500	Z7A	0	0	0	0
05-765	F	1900		07	1916	117	1964	4	CL	0.0	Z9C	1	0	18	0
05-802	F	1893		01	1918	+0	1972	1	B6	0.00014	Z7B	0	0	2	0
05-818	F	1901	1969	01	1918	52	1967	25	B2	0.00026	Z7B	7	1	104	11
05-873	F	1894		07	1917	286	1962	39	C2	0.00350	Z7C	11	6	164	95
05-880	F	1921		01	1939	520	1974	2	B6	0.0	Z9B	0	0	5	0
05-882	F	1917	1965	01	1935	468	1964	13	G6	0.0	Z9	3	0	24	0
05-885	F	1917		01	1939	572	1969	0	G6	0.0	Z9	0	0	0	0
05-892	F	1904		01	1917	4	1968	70	G6	0.0	Z9	22	0	334	0
05-897	F	1899	1968	01	1917	69	1968	1310	G4	0.0	Z9	400	0	5541	0
05-898	F	1919		01	1936	468	1972	0	B6	0.0	Z9B	0	0	0	0
05-900	F	1919	1973	01	1936	312	1972	3	B3	0.0	Z9C	1	0	8	0
05-901	F	1918		01	1934	468	1972	2	B6	0.0	Z9B	0	0	6	0
05-902	F	1919		01	1936	988	1962	6	C6	0.0	Z9C	1	0	10	0
05-905	F	1916		76	1937	156	1972	0	B6	0.0	Z9B	0	0	0	0
05-906	F	1913		01	1935	624	1972	2	B6	0.0	Z9B	0	0	5	0
05-907	F	1915		01	1935	260	1972	3	B6	0.0	Z9C	1	0	9	0
05-911	M	1886		07	1923	6	1972	0	G6	0.00310	Z7	0	0	0	0
05-912	M	1877	1951	07	1918	26	1969	0	A6	0.00020	Z7A	0	0	0	0
05-917	F	1902		01	1918	39	1966	83	B1	0.00030	Z7C	25	2	377	36
05-920	M	1895	1963	06	1917	43	1962	4	CL	0.0	Z9C	1	0	11	0
05-921	F	1896		01	1916	30	1969	67	G4	0.0	Z9	21	0	328	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD - ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
05-942	M	1901		06	1918	9	1975	0	B6	0.00010	Z7B	0	0	0	0
05-949	M	1899	1974	06	1921	422	1968	0	G6	0.0	Z9	0	0	0	0
05-953	F	1902		01	1918	65	1977*	1200	F4	0.00008	Z7F	396	36	6042	547
05-962	F	1894	1977	01	1918	84	1964	47	C2	0.00200	Z7C	14	7	207	99
05-974	F	1900		07	1918	104	1970	0	G6	0.00100	Z7	0	0	0	0
05-979	F	1897		01	1917	4	1969	194	G4	0.0	Z9	60	0	936	0
05-993	M	1902	1972	07	1917	6	1971	7	B3	0.0	Z9B	2	0	23	0
05-994	F	1886		01	1922	26	1967	9	G4	0.00570	Z7	3	3	38	51
05-998	F	1902		01	1918	3	1974	0	B6	0.00011	Z7B	0	0	0	0
09-001	F	1901		01	1917	39	1971	4	B3	0.0	Z9B	1	0	20	0
09-002	F	1902	1970	01	1917	17	1959	10	B3	0.0	Z9B	3	0	40	0
09-003	M	1892	1963	06	1914	572	1959	410	B1	0.0	Z9B	110	0	989	0
09-004	F	1890	1961	01	1912	416	1960	550	C2	0.0	Z9C	156	0	2013	0
09-006	F	1898	1971	61	1917	65	1963	1	B6	0.0	Z9B	0	0	4	0
09-007	F	1901	1965	01	1917	104	1960	33	C2	0.0	Z9C	9	0	121	0
09-008	F	1900		01	1917	8	1960	20	C6	0.0	Z9C	6	0	87	0
09-009	F	1893	1969	01	1915	78	1960	2	B6	0.0	Z9B	1	0	8	0
09-010	F	1897	1964	01	1914	+0	1960	10	C6	0.0	Z9C	3	0	40	0
09-013	F	1900	1976	01	1917	13	1971	4	B3	0.0	Z9B	1	0	19	0
09-015	M	1890	1972	04	1914	52	1960	0	G6	0.0	Z9	0	0	0	0
09-019	F	1903		01	1917	18	1975	0	B6	0.0	Z9B	0	0	0	0
09-020	F	1897	1968	01	1917	156	1963	1	B6	0.0	Z9B	0	0	4	0
09-024	M	1873	1960	06	1915	+0	1960	0	F6	0.0	Z9	0	0	0	0
09-026	F	1902		01	1917	48	1975	19	B2	0.0	Z9B	6	0	96	0
09-028	F	1897	1976	01	1916	78	1975	60	B2	0.0	Z9B	20	0	305	0
09-029	F	1901	1962	01	1917	13	1960	16	C2	0.0	Z9C	5	0	58	0
09-031	F	1897		07	1913	364	1960	286	C2	0.0	Z9	81	0	1264	0
09-032	F	1902	1969	01	1917	52	1969	97	B1	0.0	Z9B	30	0	421	0
09-038	F	1903		01	1919	1	1960	0	B6	0.0	Z9B	0	0	0	0
09-041	M	1889	1952	06	1914	260	1965	114	A1	0.0	Z9A	29	0	229	0
09-043	F	1898	1976	01	1917	26	1971	11	B6	0.0	Z9B	3	0	53	0
09-044	F	1906	1955	01	1917	13	1975	17	A2	0.0	Z9	4	0	52	0
09-046	F	1902	1965	01	1917	104	1960	10	C3	0.0	Z9C	3	0	37	0
09-049	F	1902		01	1915	+0	1969	14	G6	0.0	Z9	4	0	70	0
09-051	F	1900	1971	01	1917	104	1960	50	C6	0.0	Z9C	14	0	199	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
09-052	F	1900	1971	01	1916	52	1960	20	C6	0.0	Z9C	6	0	83	0
09-053	M	1874	1966	04	1919	+0	1960	81	B1	0.0	Z9B	22	0	210	0
09-057	F	1890	1973	01	1917	52	1960	0	B6	0.0	Z9B	0	0	0	0
09-058	F	1899		01	1917	39	1960	4	B6	0.0	Z9B	1	0	17	0
09-059	F	1903	1972	01	1917	1	1971	2	B6	0.0	Z9B	1	0	9	0
09-060	F	1899	1975	01	1917	65	1969	43	B2	0.0	Z9B	13	0	200	0
09-061	F	1892		01	1914	208	1970	0	G6	0.0	Z9	0	0	0	0
09-062	F	1901		01	1918	52	1972	4	B3	0.0	Z9B	1	0	19	0
09-064	F	1891		01	1916	9	1973	1	B6	0.0	Z9B	0	0	5	0
09-065	F	1887		06	1914	78	1960	1	B6	0.0	Z9B	0	0	5	0
09-066	F	1899		01	1917	8	1972	2	B6	0.0	Z9B	1	0	10	0
09-070	M	1875	1967	06	1913	208	1960	3	B6	0.0	Z9B	1	0	9	0
09-071	F	1897		01	1917	104	1975	2	B6	0.0	Z9B	1	0	10	0
09-072	F	1893	1974	01	1917	39	1972	2	B6	0.0	Z9C	1	0	10	0
09-073	M	1886	1963	06	1916	468	1962	0	B6	0.0	Z9B	0	0	0	0
09-074	F	1892	1976	01	1920	104	1962	13	G6	0.0	Z9	4	0	52	0
09-075	M	1893	1967	06	1913	884	1963	1	B6	0.0	Z9B	0	0	3	0
09-076	M	1882	1966	06	1913	1872	1964	14	D3	0.0	Z9D	3	0	25	0
09-077	M	1894		06	1914	520	1972	2	B6	0.0	Z9B	1	0	7	0
09-078	M	1883	1966	06	1911	832	1963	3	B6	0.0	Z9B	1	0	8	0
09-079	M	1891		06	1916	570	1962	0	G6	0.0	Z9	0	0	0	0
09-080	M	1886		06	1919	312	1962	5	G6	0.0	Z9	1	0	14	0
09-082	M	1892		06	1916	312	1974	4	B3	0.0	Z9B	1	0	14	0
09-083	M	1889	1964	06	1915	17	1962	5	G6	0.0	Z9	1	0	14	0
09-084	M	1888	1927	06	1912	676	1965	382	A1	0.0	Z9A	42	0	131	0
09-086	M	1895		06	1921	78	1974	1	B6	0.0	Z9B	0	0	3	0
09-088	M	1900		06	1922	338	1971	18	B2	0.0	Z9B	5	0	53	0
09-089	M	1890	1973	06	1915	78	1959	64	C2	0.0	Z9C	18	0	194	0
09-090	M	1888	1971	06	1913	78	1963	0	G6	0.0	Z9	0	0	0	0
09-095	M	1894	1975	06	1918	416	1975	0	B6	0.0	Z9B	0	0	0	0
09-096	M	1892		06	1919	17	1963	9	G6	0.0	Z9	3	0	28	0
09-097	M	1896		07	1916	988	1974	1	B6	0.0	Z9B	0	0	3	0
09-098	M	1902	1971	06	1921	104	1963	14	G6	0.0	Z9	4	0	37	0
09-099	M	1898	1971	06	1913	208	1963	1	G6	0.0	Z9	0	0	3	0
09-100	M	1888		06	1918	364	1963	9	G6	0.0	Z9	2	0	26	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
09-101	M	1884	1964	06	1920	39	1963	6	G6	0.0	Z9	2	0	15	0
09-102	M	1882	1951	46	1915	1	1964	150	A1	0.0	Z9A	38	0	306	0
09-103	M	1895	1971	06	1918	416	1965	1	G6	0.0	Z9	0	0	3	0
09-104	M	1880	1967	06	1906	364	1965	42	B2	0.0	Z9B	13	0	146	0
09-105	M	1886	1928	06	1912	728	1966	1390	A1	0.00093	A6	156	34	507	252
09-106	M	1901		06	1919	156	1974	0	B6	0.0	Z9B	0	0	0	0
09-107	M	1897	1974	06	1913	104	1965	1	G6	0.0	Z9	0	0	3	0
09-108	M	1891		06	1915	104	1965	4	G6	0.0	Z9	1	0	14	0
09-109	M	1895		06	1914	104	1965	4	G6	0.0	Z9	1	0	14	0
09-110	M	1900		06	1914	52	1965	7	G6	0.0	Z9	2	0	24	0
09-111	M	1874	1944	06	1913	520	1967	0	A6	0.0	Z9A	0	0	0	0
09-112	M	1898		06	1940	416	1966	84	G4	0.0	Z9	17	0	125	0
09-115	M	1893		06	1920	52	1969	3	G6	0.0	Z9	1	0	10	0
09-117	F	1899		01	1917	24	1971	4	B3	0.0	Z9B	1	0	20	0
09-118	F	1901		07	1921	+0	1970	50	G4	0.0	Z9	15	0	224	0
09-120	M	1889	1945	06	1918	104	1974	1	A6	0.0	Z9	0	0	2	0
09-123	M	1890		06	1917	156	1974	0	B6	0.0	Z9B	0	0	0	0
10-007	F	1916		01	1934	1144	1971	0	B6	0.0	Z9B	0	0	0	0
10-008	F	1904		01	1918	13	1976	0	B6	0.00009	Z7B	0	0	0	0
10-010	F	1895	1975	05	1930	+0	1971	8600	B1	0.0	Z9C	2361	0	30382	0
10-012	M	1886	1941	05	1925	+0	1972	0	A6	0.0	Z9	0	0	0	0
10-018	F	1920		01	1952	416	1975	1	B6	0.0	Z9B	0	0	1	0
10-024	M	1914		06	1936	1612	1971	50	G4	0.0	Z9	8	0	52	0
10-025	M	1937		07	1963	416	1971	7	B3	0.0	Z9C	0	0	1	0
10-026	M	1948		07	1968	200	1971	2	B6	0.0	Z9C	0	0	0	0
10-027	F	1928		01	1946	156	1972	0	B6	0.0	Z9C	0	0	0	0
10-028	M	1886	1976	06	1918	156	1976	0	B6	0.0	Z9B	0	0	0	0
10-031	F	1928		01	1946	52	1972	8	B2	0.0	Z9C	2	0	18	0
10-032	M	1937		07	1961	156	1972	0	B6	0.0	Z9C	0	0	0	0
10-033	F	1927		01	1946	264	1974	3	B3	0.0	Z9C	1	0	7	0
10-034	F	1919		01	1943	202	1973	9	B2	0.0	Z9C	2	0	20	0
10-035	F	1922		01	1942	689	1974	10	B2	0.0	Z9C	2	0	21	0
10-036	F	1920		76	1945	208	1972	0	B6	0.0	Z9C	0	0	0	0
10-037	F	1927		01	1951	52	1976	3	B6	0.0	Z9C	1	0	6	0
10-038	F	1929		01	1947	78	1974	1	B6	0.0	Z9C	0	0	1	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-039	F	1922		07	1942	260	1972	4	B3	0.0	Z9C	1	0	9	0
10-040	F	1917		01	1946	+0	1972	0	B6	0.0	Z9C	0	0	0	0
10-041	F	1924		01	1943	13	1972	1	B6	0.0	Z9C	0	0	2	0
10-042	F	1927		01	1947	130	1972	0	B6	0.0	Z9C	0	0	0	0
10-043	F	1919		05	1941	8	1975	0	B6	0.0	Z9B	0	0	0	0
10-044	F	1925		01	1948	13	1972	19	B2	0.0	Z9C	4	0	39	0
10-045	F	1923		01	1946	13	1972	1	B6	0.0	Z9C	0	0	2	0
10-046	F	1927		17	1947	208	1975	0	B6	0.0	Z9C	0	0	0	0
10-047	F	1924		01	1942	52	1974	10	B2	0.0	Z9C	2	0	25	0
10-048	F	1894		06	1917	156	1977	0	B6	0.0	Z9B	0	0	0	0
10-049	F	1926		01	1946	104	1972	0	B6	0.0	Z9C	0	0	0	0
10-050	F	1920		01	1943	104	1974	11	B2	0.0	Z9C	2	0	26	0
10-051	M	1914		06	1931	468	1972	0	B6	0.0	Z9C	0	0	0	0
10-053	F	1926		17	1946	260	1972	2	B6	0.0	Z9C	0	0	3	0
10-054	F	1926		71	1943	364	1972	1	B6	0.0	Z9C	0	0	3	0
10-055	M	1922		08	1922	39	1972	0	B6	0.00040	Z7B	0	0	0	0
10-056	M	1924		08	1924	39	1972	2	B6	0.00040	Z7B	1	0	6	1
10-057	F	1929		01	1946	52	1972	1	B6	0.0	Z9C	0	0	3	0
10-058	F	1923		01	1941	208	1972	6	B3	0.0	Z9C	1	0	15	0
10-059	F	1915		01	1944	104	1972	0	B6	0.0	Z9C	0	0	0	0
10-060	F	1919		01	1943	104	1972	0	B6	0.0	Z9C	0	0	0	0
10-061	F	1923		07	1939	260	1972	6	B3	0.0	Z9C	1	0	15	0
10-062	F	1920		01	1939	182	1972	1	B6	0.0	Z9C	0	0	3	0
10-063	F	1911		01	1928	624	1976	2	B3	0.0	Z9C	1	0	7	0
10-064	F	1921		07	1943	156	1972	0	B6	0.0	Z9C	0	0	0	0
10-065	F	1920		01	1941	260	1972	0	B6	0.0	Z9C	0	0	1	0
10-066	F	1924		01	1942	104	1972	12	B2	0.0	Z9C	3	0	29	0
10-067	F	1923		01	1942	468	1972	8	B2	0.0	Z9C	2	0	17	0
10-068	F	1918		71	1942	78	1972	0	B6	0.0	Z9C	0	0	0	0
10-069	F	1923		01	1947	1300	1972	8	B3	0.0	Z9C	1	0	5	0
10-070	F	1921		01	1945	1352	1974	14	B2	0.0	Z9C	2	0	15	0
10-071	F	1924		01	1943	1508	1972	13	B2	0.0	Z9C	1	0	10	0
10-072	F	1924		01	1947	1300	1972	12	B2	0.0	Z9C	1	0	8	0
10-073	M	1919		07	1953	208	1972	0	B6	0.0	Z9C	0	0	0	0
10-074	M	1921		06	1950	1248	1976	34	B2	0.0	Z9C	5	0	21	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-075	F	1929		01	1949	260	1972	5	B3	0.0	Z9C	1	0	9	0
10-076	F	1923		01	1951	52	1972	0	B6	0.0	Z9C	0	0	0	0
10-077	F	1920		01	1951	17	1972	1	B6	0.0	Z9C	0	0	1	0
10-078	F	1923		01	1941	676	1977	11	B2	0.0	Z9C	3	0	25	0
10-079	F	1920		01	1940	624	1974	13	B3	0.0	Z9C	3	0	29	0
10-080	F	1913		76	1943	1508	1972	5	B3	0.0	Z9C	1	0	4	0
10-081	F	1916		01	1946	104	1972	5	B3	0.0	Z9C	1	0	11	0
10-082	F	1915		01	1951	758	1972	5	B3	0.0	Z9C	1	0	5	0
10-083	F	1924		01	1943	104	1972	5	B3	0.0	Z9C	1	0	12	0
10-084	F	1928		71	1946	82	1972	0	B6	0.0	Z9C	0	0	0	0
10-085	M	1946		71	1964	17	1972	0	B6	0.0	Z9C	0	0	0	0
10-086	F	1915		01	1943	156	1972	3	B6	0.0	Z9C	1	0	6	0
10-087	F	1920		01	1942	1560	1972	19	B2	0.0	Z9C	2	0	16	0
10-088	F	1923		17	1946	260	1972	3	B6	0.0	Z9C	1	0	5	0
10-089	F	1921		01	1942	13	1972	0	B6	0.0	Z9C	0	0	1	0
10-090	F	1922		01	1941	78	1972	1	B6	0.0	Z9C	0	0	3	0
10-091	M	1883	1952	05	1930	+0	1974	423	A1	0.0	Z9A	84	0	487	0
10-094	M	1905	1974	07	1919	104	1972	0	B6	0.00240	Z7C	0	0	0	0
10-095	F	1927		01	1946	260	1972	5	B3	0.0	Z9C	1	0	10	0
10-096	F	1930		01	1951	832	1972	0	B6	0.0	Z9C	0	0	0	0
10-097	F	1919		01	1943	364	1972	4	B3	0.0	Z9C	1	0	8	0
10-098	F	1917		01	1935	208	1972	4	B3	0.0	Z9C	1	0	12	0
10-099	F	1924		01	1942	104	1977	17	C3	0.0	Z9C	4	0	46	0
10-100	F	1924		76	1942	78	1972	7	B3	0.0	Z9C	2	0	18	0
10-101	F	1925		01	1943	208	1972	0	B6	0.0	Z9C	0	0	0	0
10-102	F	1926		01	1944	60	1972	1	B6	0.0	Z9C	0	0	2	0
10-103	F	1912		01	1946	104	1972	2	B6	0.0	Z9C	0	0	4	0
10-104	F	1929		01	1948	208	1972	2	B6	0.0	Z9C	0	0	4	0
10-105	F	1927		01	1946	260	1972	0	C6	0.0	Z9C	0	0	0	0
10-106	F	1926		01	1946	104	1972	1	B6	0.0	Z9C	0	0	2	0
10-107	F	1909		01	1926	9	1972	0	B6	0.0	Z9C	0	0	0	0
10-108	F	1916		04	1950	+0	1972	3	B6	0.0	Z9C	1	0	5	0
10-109	F	1951		07	1969	78	1972	0	B6	0.0	Z9C	0	0	0	0
10-110	F	1917		01	1946	520	1972	0	B6	0.0	Z9C	0	0	0	0
10-111	F	1906		01	1923	2	1976	7	B2	0.0	Z9C	2	0	31	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BCRN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-112	M	1902		01	1923	+0	1976	3	B3	0.0	Z9C	1	0	10	0
10-113	F	1924		01	1942	52	1972	0	B6	0.0	Z9C	0	0	0	0
10-114	F	1937		01	1970	104	1972	1	B6	0.0	Z9C	0	0	0	0
10-115	F	1921		07	1970	130	1972	1	B6	0.0	Z9C	0	0	0	0
10-116	F	1924		01	1969	312	1976	5	B2	0.0	Z9C	0	0	1	0
10-117	F	1924		01	1967	208	1972	2	B6	0.0	Z9C	0	0	1	0
10-118	F	1924		01	1945	1352	1972	23	B2	0.0	Z9C	3	0	21	0
10-119	F	1952		71	1971	82	1972	2	B6	0.0	Z9C	0	0	0	0
10-120	F	1950		01	1971	98	1974	4	C3	0.0	Z9C	0	0	1	0
10-121	F	1926		01	1945	17	1972	1	B6	0.0	Z9C	0	0	1	0
10-122	F	1921		07	1921	+0	1972	0	B6	0.0	Z9C	0	0	0	0
10-125	F	1903		01	1917	8	1975	1	B6	0.0	Z9B	0	0	5	0
10-126	F	1927		01	1946	13	1972	0	B6	0.0	Z9C	0	0	0	0
10-128	F	1923		01	1942	364	1972	6	B3	0.0	Z9C	1	0	14	0
10-129	F	1923		01	1942	269	1975	9	B2	0.0	Z9C	2	0	22	0
10-130	F	1922		01	1942	147	1975	14	B2	0.0	Z9C	3	0	35	0
10-131	F	1917		07	1941	260	1972	1	B6	0.0	Z9C	0	0	2	0
10-132	F	1929		07	1970	130	1972	0	B6	0.0	Z9C	0	0	0	0
10-133	F	1910		01	1941	1248	1976	5	B2	0.0	Z9C	1	0	8	0
10-134	F	1913		01	1932	1872	1972	1	B6	0.0	Z9C	0	0	1	0
10-135	F	1922		01	1939	130	1972	6	B3	0.0	Z9C	1	0	17	0
10-136	F	1920		01	1941	26	1972	0	B6	0.0	Z9C	0	0	0	0
10-137	F	1918		01	1935	117	1972	1	B6	0.0	Z9C	0	0	2	0
10-139	F	1922		01	1942	130	1972	3	B6	0.0	Z9C	1	0	7	0
10-140	F	1935		07	1956	17	1972	2	B6	0.0	Z9C	0	0	2	0
10-141	F	1918		01	1965	104	1972	0	B6	0.0	Z9C	0	0	0	0
10-142	F	1922		01	1942	156	1972	2	B6	0.0	Z9C	1	0	5	0
10-144	F	1926		01	1945	156	1972	0	B6	0.0	Z9C	0	0	0	0
10-145	F	1928		07	1946	130	1976	6	C3	0.0	Z9C	1	0	13	0
10-146	F	1921		01	1940	364	1972	4	B3	0.0	Z9C	1	0	9	0
10-147	F	1927		01	1946	156	1972	2	B6	0.0	Z9C	0	0	4	0
10-148	F	1913		01	1935	13	1972	0	B6	0.0	Z9C	0	0	0	0
10-149	F	1924		01	1945	104	1972	4	B3	0.0	Z9C	1	0	10	0
10-150	F	1889	1976	01	1919	13	1972	0	G6	0.0	Z9	0	0	0	0
10-151	M	1887		06	1915	520	1974	0	G8	0.0	Z9	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-152	F	1923		01	1941	52	1972	2	B6	0.0	Z9B	0	0	5	0
10-153	F	1921		01	1941	234	1972	1	B6	0.0	Z9B	0	0	2	0
10-160	F	1921		01	1941	208	1976	20	B1	0.0	Z9C	5	0	53	0
10-162	F	1931		01	1951	13	1974	3	B2	0.0	Z9C	1	0	5	0
10-164	F	1915		01	1937	156	1974	0	B6	0.0	Z9C	0	0	0	0
10-165	F	1919		01	1942	416	1972	2	B6	0.0	Z9C	0	0	5	0
10-171	F	1924		01	1942	156	1974	3	B3	0.0	Z9C	1	0	7	0
10-172	F	1930	1977	07	1948	60	1974	3	B3	0.0	Z9C	1	0	7	0
10-173	F	1915	1977	01	1948	123	1973	0	B6	0.0	Z9C	0	0	0	0
10-180	F	1919		01	1941	728	1974	9	B2	0.0	Z9C	2	0	18	0
10-181	F	1912		01	1931	287	1973	5	B3	0.0	Z9C	1	0	15	0
10-190	F	1921		01	1946	156	1972	3	B6	0.0	Z9C	1	0	6	0
10-191	F	1940		71	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-192	F	1924		01	1942	78	1974	3	B3	0.0	Z9C	1	0	7	0
10-193	F	1921		01	1941	104	1972	3	B6	0.0	Z9C	1	0	7	0
10-195	F	1920		01	1937	1560	1973	11	C3	0.0	Z9C	2	0	17	0
10-198	F	1920		01	1946	378	1977	8	B3	0.0	Z9C	2	0	17	0
10-201	F	1918		71	1946	1352	1972	9	B2	0.0	Z9C	1	0	6	0
10-202	F	1925		01	1947	49	1974	2	B6	0.0	Z9C	0	0	4	0
10-203	F	1926		01	1946	0	1974	2	B6	0.0	Z9C	0	0	4	0
10-204	F	1950		07	1971	43	1972	6	B3	0.0	Z9C	0	0	1	0
10-205	F	1923		01	1942	39	1972	1	B6	0.0	Z9C	0	0	3	0
10-206	F	1924		01	1943	230	1972	6	B3	0.0	Z9C	1	0	14	0
10-207	F	1923		61	1942	208	1972	12	B2	0.0	Z9C	3	0	28	0
10-208	F	1922		01	1941	2	1972	1	B6	0.0	Z9C	0	0	2	0
10-209	F	1920		01	1942	69	1972	6	B3	0.0	Z9C	1	0	15	0
10-210	F	1909		01	1926	1040	1972	17	B2	0.0	Z9C	4	0	51	0
10-212	M	1950		07	1971	55	1973	1	B6	0.0	Z9C	0	0	0	0
10-213	M	1951		07	1971	45	1973	1	B6	0.0	Z9C	0	0	0	0
10-214	F	1942		07	1972	30	1974	0	B6	0.0	Z9C	0	0	0	0
10-215	F	1921		01	1943	208	1972	1	B6	0.0	Z9C	0	0	2	0
10-216	F	1916		01	1946	1456	1973	2	B6	0.0	Z9C	0	0	1	0
10-218	F	1915		01	1934	492	1973	0	B6	0.0	Z9C	0	0	0	0
10-219	F	1916		16	1937	364	1976	13	B2	0.0	Z9B	3	0	37	0
10-221	F	1917		01	1941	676	1973	1	B6	0.0	Z9B	0	0	2	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-222	F	1919		01	1941	234	1972	C	G6	0.0	Z9	0	0	0	0
10-225	F	1911		01	1933	1872	1976	4	B2	0.0	Z9C	1	0	6	0
10-226	F	1923		01	1941	1612	1972	3	B6	0.0	Z9C	0	0	2	0
10-227	M	1912		71	1928	2548	1977	6	B2	0.0	Z9C	1	0	5	0
10-228	F	1912		01	1940	1508	1975	0	B6	0.0	Z9C	0	0	0	0
10-229	F	1920		01	1942	260	1972	1	B6	0.0	Z9C	0	0	3	0
10-230	F	1929		01	1948	13	1973	0	C6	0.0	Z9C	0	0	0	0
10-231	M	1968		08	1968	39	1972*	1	C6	0.0	Z9C	0	0	0	0
10-232	M	1969		08	1969	39	1972*	0	C6	0.0	Z9C	0	0	0	0
10-233	F	1919		01	1941	52	1976	2	B3	0.0	Z9C	1	0	6	0
10-234	F	1928	1972	07	1959	676	1972	0	B6	0.0	Z9C	0	0	0	0
10-236	F	1919		01	1949	156	1974	0	B6	0.0	Z9C	0	0	0	0
10-237	F	1910		01	1940	156	1977	2	C6	0.0	Z9C	1	0	7	0
10-239	M	1908		06	1934	1300	1976	0	B6	0.0	Z9B	0	0	0	0
10-240	M	1906		06	1931	884	1976	3	B6	0.0	Z9B	1	0	5	0
10-241	F	1904		01	1922	17	1972	0	C6	0.0	Z9C	0	0	0	0
10-242	F	1947		07	1966	156	1974	1	B6	0.0	Z9C	0	0	0	0
10-244	F	1916		01	1942	1	1972	0	B6	0.0	Z9C	0	0	1	0
10-245	M	1914		67	1941	104	1972	0	B6	0.0	Z9C	0	0	0	0
10-247	M	1915	1976	07	1948	364	1972	1	B6	0.0	Z9C	0	0	1	0
10-249	M	1943		07	1963	130	1973	1	B6	0.0	Z9C	0	0	0	0
10-250	F	1938		07	1956	30	1972	0	B6	0.0	Z9C	0	0	0	0
10-251	F	1923		01	1941	65	1974	2	B3	0.0	Z9C	0	0	5	0
10-252	F	1919		01	1935	416	1972	4	B3	0.0	Z9C	1	0	11	0
10-254	F	1905		07	1953	832	1976	0	B6	0.0	Z9C	0	0	0	0
10-256	F	1917		01	1940	78	1972	1	B6	0.0	Z9B	0	0	3	0
10-257	F	1932		07	1951	104	1972	0	B6	0.0	Z9C	0	0	0	0
10-258	F	1923		01	1943	26	1972	3	B6	0.0	Z9C	1	0	7	0
10-261	F	1922		01	1940	104	1972	3	B6	0.0	Z9C	1	0	7	0
10-262	F	1919		01	1941	104	1973	2	B6	0.0	Z9C	0	0	4	0
10-263	F	1921		01	1941	130	1972	2	B6	0.0	Z9B	0	0	5	0
10-266	F	1905		01	1926	2236	1973	2	B6	0.0	Z9C	0	0	4	0
10-269	F	1925		01	1945	17	1972	0	B6	0.0	Z9C	0	0	0	0
10-270	F	1926		71	1946	104	1972	1	B6	0.0	Z9C	0	0	1	0
10-272	F	1915		01	1935	52	1972	2	B6	0.0	Z9C	0	0	5	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-273	F	1929		01	1948	22	1973	2	B6	0.0	Z9C	0	0	4	0
10-274	F	1924		01	1946	62	1973	3	B3	0.0	Z9C	1	0	7	0
10-276	F	1932		01	1951	6	1973	1	B6	0.0	Z9C	0	0	1	0
10-277	F	1915		71	1946	154	1973	1	B6	0.0	Z9C	0	0	1	0
10-278	F	1908		71	1929	1872	1976	2	B6	0.0	Z9C	0	0	3	0
10-279	F	1937		01	1955	728	1973	2	B6	0.0	Z9C	0	0	1	0
10-280	F	1904		07	1921	2132	1976	1	B6	0.0	Z9C	0	0	2	0
10-281	F	1931		01	1950	416	1973	1	B6	0.0	Z9C	0	0	1	0
10-282	F	1921	1974	01	1941	22	1974	2	C6	0.0	Z9C	0	0	5	0
10-283	F	1918		01	1937	208	1974	0	B6	0.0	Z9C	0	0	1	0
10-284	F	1918		71	1936	1456	1974	3	B3	0.0	Z9C	1	0	5	0
10-285	M	1917		07	1935	81	1973	0	G6	0.0	Z9	0	0	0	0
10-286	F	1937		07	1968	104	1973	0	B6	0.0	Z9C	0	0	0	0
10-287	F	1923		01	1944	2	1973	1	B6	0.0	Z9C	0	0	3	0
10-291	F	1916		01	1934	156	1973	4	B3	0.0	Z9C	1	0	14	0
10-292	F	1913	1975	01	1934	102	1973	6	B3	0.0	Z9C	2	0	20	0
10-293	F	1938		07	1970	24	1973	0	B6	0.0	Z9C	0	0	0	0
10-294	F	1916		01	1934	415	1974	2	B6	0.0	Z9C	0	0	5	0
10-295	M	1923		07	1946	282	1973	2	B6	0.0	Z9C	0	0	2	0
10-296	F	1930		01	1948	50	1973	0	B6	0.0	Z9C	0	0	0	0
10-297	F	1929	1973	07	1969	65	1973	0	B6	0.0	Z9C	0	0	0	0
10-299	F	1923		01	1942	43	1973	6	B3	0.0	Z9C	2	0	16	0
10-300	F	1911		01	1940	1612	1977	0	B6	0.0	Z9C	0	0	1	0
10-301	M	1930		07	1949	69	1973	0	B6	0.0	Z9C	0	0	0	0
10-302	F	1917		07	1933	312	1973	0	B6	0.0	Z9C	0	0	0	0
10-304	F	1926		01	1950	364	1973	2	B6	0.0	Z9C	0	0	4	0
10-306	F	1907		01	1923	4	1976	5	B2	0.0	Z9C	1	0	21	0
10-307	F	1893	1948	05	1930	+0	1974	85	A2	0.0	Z9A	15	0	109	0
10-309	F	1925		01	1943	28	1973	2	B6	0.0	Z9C	0	0	4	0
10-310	F	1916		01	1936	53	1973	2	B6	0.0	Z9C	0	0	6	0
10-311	F	1919		01	1943	15	1973	0	B6	0.0	Z9C	0	0	0	0
10-312	F	1923		01	1943	16	1973	2	B6	0.0	Z9C	0	0	4	0
10-313	F	1924		01	1942	110	1973	9	B3	0.0	Z9C	2	0	23	0
10-314	F	1918		01	1943	119	1973	4	B3	0.0	Z9C	1	0	9	0
10-316	M	1946		07	1965	167	1973	2	B6	0.0	Z9C	0	0	1	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-318	M	1908		07	1968	364	1977*	0	C6	0.0	Z9C	0	0	0	0
10-319	F	1912		07	1934	832	1973	6	B3	0.0	Z9C	1	0	15	0
10-320	M	1918		07	1939	1352	1973	1	B6	0.0	Z9C	0	0	1	0
10-321	F	1910		01	1942	1456	1976	1	B6	0.0	Z9C	0	0	1	0
10-322	F	1904		07	1936	1092	1976	5	B2	0.0	Z9C	1	0	11	0
10-324	F	1912		01	1926	13	1973	0	B6	0.0	Z9C	0	0	0	0
10-325	M	1952		07	1970	22	1974	1	B6	0.0	Z9	0	0	0	0
10-326	F	1954		07	1973	39	1974	0	B6	0.0	Z9C	0	0	0	0
10-327	M	1953		71	1973	52	1977*	1	C6	0.0	Z9C	0	0	0	0
10-329	F	1914		07	1938	884	1973	1	B6	0.0	Z9C	0	0	1	0
10-330	F	1921		07	1945	520	1973	0	B6	0.0	Z9C	0	0	0	0
10-331	F	1911		07	1934	162	1976	1	B6	0.0	Z9B	0	0	3	0
10-332	F	1901		01	1927	0	1977*	0	G6	0.00230	Z8	0	0	0	0
10-333	F	1915		01	1941	208	1973	1	B6	0.0	Z9B	0	0	3	0
10-334	F	1921		01	1943	26	1973	0	B6	0.0	Z9B	0	0	0	0
10-335	F	1939		07	1969	24	1973	0	B6	0.0	Z9C	0	0	0	0
10-336	F	1923		07	1943	1092	1973	0	B6	0.0	Z9C	0	0	0	0
10-337	M	1892	1971	06	1913	260	1974	1	A6	0.0	Z9A	0	0	2	0
10-339	F	1902		01	1925	1	1976	0	B6	0.00260	Z8	0	0	0	0
10-340	F	1920		67	1942	104	1974	6	B3	0.0	Z9B	1	0	15	0
10-341	F	1919		01	1939	312	1973	1	B6	0.0	Z9B	0	0	3	0
10-347	M	1947		08	1947	39	1973	1	B6	0.0	Z9B	0	0	2	0
10-348	F	1921		01	1941	104	1974	0	B6	0.0	Z9C	0	0	0	0
10-350	F	1924		01	1941	27	1973	1	B6	0.0	Z9C	0	0	2	0
10-351	M	1931		07	1964	14	1973	1	B6	0.0	Z9C	0	0	1	0
10-352	F	1926		07	1947	104	1974	1	B6	0.0	Z9C	0	0	2	0
10-353	F	1922		01	1942	21	1973	1	B6	0.0	Z9C	0	0	1	0
10-356	F	1915		07	1947	46	1973	1	B6	0.0	Z9C	0	0	2	0
10-357	F	1923		01	1942	68	1973	3	B3	0.0	Z9C	1	0	8	0
10-358	F	1920		01	1946	16	1973	3	B3	0.0	Z9C	1	0	6	0
10-359	M	1950		07	1971	32	1973	3	B3	0.0	Z9C	0	0	0	0
10-360	F	1919		01	1941	46	1975	0	B6	0.0	Z9B	0	0	0	0
10-362	F	1922		01	1941	364	1973	4	B3	0.0	Z9C	1	0	10	0
10-365	F	1920		01	1939	260	1973	0	B6	0.0	Z9C	0	0	1	0
10-367	F	1919		01	1940	260	1973	1	B6	0.0	Z9C	0	0	2	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 JCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-375	F	1924		01	1943	20	1973	1	B6	0.0	Z9C	0	0	3	0
10-377	F	1898		07	1923	1976	1976	3	B2	0.0	Z9C	1	0	8	0
10-378	F	1906		07	1946	520	1976	0	B6	0.0	Z9C	0	0	0	0
10-379	F	1917		01	1941	89	1977	32	B2	0.0	Z9C	8	0	88	0
10-381	F	1927		01	1946	27	1973	6	B3	0.0	Z9C	1	0	12	0
10-382	F	1923		01	1942	119	1973	5	B3	0.0	Z9C	1	0	13	0
10-384	F	1919		71	1943	884	1973	1	B6	0.0	Z9C	0	0	2	0
10-385	F	1921		07	1964	16	1973	0	B6	0.0	Z9C	0	0	0	0
10-386	F	1933		01	1953	52	1973	1	B6	0.0	Z9C	0	0	2	0
10-387	F	1928		01	1947	15	1973	0	B6	0.0	Z9C	0	0	0	0
10-389	F	1919		01	1943	24	1973	0	B6	0.0	Z9C	0	0	0	0
10-390	F	1923		01	1942	38	1973	3	B3	0.0	Z9C	1	0	8	0
10-392	F	1903		71	1932	520	1973	0	B6	0.0	Z9C	0	0	0	0
10-393	F	1907		01	1925	208	1976	5	B2	0.0	Z9C	2	0	23	0
10-394	F	1907	1975	01	1923	728	1974	1	B6	0.0	Z9C	0	0	2	0
10-395	F	1908		01	1925	260	1976	2	B3	0.0	Z9C	1	0	10	0
10-397	F	1927		01	1946	16	1973	1	B6	0.0	Z9C	0	0	2	0
10-398	F	1918		71	1951	624	1973	1	B6	0.0	Z9C	0	0	1	0
10-409	F	1921		01	1943	118	1973	0	B6	0.0	Z9C	0	0	0	0
10-410	F	1926		01	1943	52	1973	0	B6	0.0	Z9C	0	0	0	0
10-411	F	1920		01	1942	14	1973	3	B3	0.0	Z9C	1	0	7	0
10-412	F	1908		01	1925	13	1976	1	B6	0.0	Z9C	0	0	3	0
10-414	F	1926		01	1948	104	1973	1	B6	0.0	Z9C	0	0	2	0
10-415	F	1943		07	1973	8	1974	0	B6	0.0	Z9C	0	0	0	0
10-419	M	1913		06	1936	1924	1973	6	B3	0.0	Z9C	1	0	4	0
10-432	F	1920		01	1940	104	1975	0	B6	0.0	Z9C	0	0	1	0
10-438	F	1907		01	1925	17	1977*	14	M6	0.0	Z9	4	0	59	0
10-439	F	1925		01	1943	20	1973	2	B6	0.0	Z9C	0	0	5	0
10-440	F	1920		01	1948	1	1973	0	B6	0.0	Z9C	0	0	0	0
10-442	F	1932		01	1951	8	1973	0	B6	0.0	Z9C	0	0	0	0
10-444	F	1927		01	1949	4	1973	1	B6	0.0	Z9C	0	0	1	0
10-445	F	1924		01	1943	2	1973	2	B6	0.0	Z9C	0	0	5	0
10-446	F	1920		01	1939	3	1973	1	B6	0.0	Z9C	0	0	2	0
10-447	F	1929		01	1947	5	1973	6	B3	0.0	Z9C	1	0	13	0
10-449	F	1923		01	1942	52	1976	4	B2	0.0	Z9C	1	0	10	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-451	F	1921		01	1945	3	1973	0	B6	0.0	Z9C	0	0	1	0
10-453	F	1927		01	1943	1	1973	0	B6	0.0	Z9C	0	0	1	0
10-454	F	1926		01	1942	5	1973	0	B6	0.0	Z9C	0	0	1	0
10-455	F	1909		01	1928	104	1977	0	B6	0.0	Z9C	0	0	1	0
10-457	F	1921		01	1941	65	1973	1	B6	0.0	Z9C	0	0	3	0
10-458	M	1927		01	1954	1040	1973	24	B2	0.0	Z9C	2	0	9	0
10-459	F	1923		01	1956	832	1973	0	B6	0.0	Z9C	0	0	0	0
10-460	F	1936		01	1959	676	1973	0	B6	0.0	Z9C	0	0	0	0
10-461	M	1925		06	1948	1300	1973	10	B2	0.0	Z9C	1	0	5	0
10-462	M	1927		06	1951	1144	1973	8	B3	0.0	Z9C	1	0	4	0
10-464	M	1940		07	1961	12	1973	0	B6	0.0	Z9C	0	0	0	0
10-465	F	1924		01	1942	8	1973	0	B6	0.0	Z9C	0	0	0	0
10-470	F	1924		01	1943	176	1973	0	B6	0.0	Z9C	0	0	0	0
10-471	F	1924		01	1943	34	1973	3	B3	0.0	Z9C	1	0	7	0
10-472	F	1928		01	1947	12	1973	0	B6	0.0	Z9C	0	0	0	0
10-473	F	1926		01	1945	18	1973	0	B6	0.0	Z9C	0	0	1	0
10-474	F	1921		01	1946	77	1974	2	B6	0.0	Z9C	0	0	5	0
10-475	F	1927		07	1946	90	1973	0	B6	0.0	Z9C	0	0	0	0
10-476	F	1928		01	1945	11	1973	1	B6	0.0	Z9C	0	0	1	0
10-477	F	1924		01	1944	42	1975	2	B3	0.0	Z9C	1	0	6	0
10-478	F	1922		01	1940	10	1973	0	B6	0.0	Z9C	0	0	0	0
10-479	F	1926		01	1946	11	1973	0	B6	0.0	Z9C	0	0	0	0
10-480	F	1924		01	1943	4	1973	0	B6	0.0	Z9C	0	0	1	0
10-481	F	1925		01	1940	5	1973	1	B6	0.0	Z9C	0	0	3	0
10-482	F	1925		01	1943	28	1973	4	B3	0.0	Z9C	1	0	11	0
10-483	M	1934		07	1950	5	1973	2	B6	0.0	Z9C	0	0	2	0
10-485	F	1918		01	1948	4	1973	0	B6	0.0	Z9C	0	0	1	0
10-486	F	1919		01	1942	32	1973	0	B6	0.0	Z9C	0	0	1	0
10-487	F	1924		01	1943	220	1973	0	B6	0.0	Z9C	0	0	0	0
10-488	F	1921		01	1942	20	1973	0	B6	0.0	Z9C	0	0	0	0
10-490	F	1922		01	1943	20	1974	8	B2	0.0	Z9C	2	0	19	0
10-492	F	1925		01	1945	326	1973	2	B6	0.0	Z9C	0	0	3	0
10-494	F	1913		01	1939	312	1973	1	B6	0.0	Z9C	0	0	2	0
10-495	F	1924		01	1942	312	1973	0	B6	0.0	Z9B	0	0	0	0
10-496	F	1922		01	1940	108	1975	0	B6	0.0	Z9C	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-501	F	1928		01	1946	15	1973	2	B6	0.0	Z9C	0	0	4	0
10-502	F	1928		01	1946	13	1973	2	B6	0.0	Z9C	0	0	4	0
10-505	F	1933		01	1951	3	1973	2	B6	0.0	Z9C	0	0	4	0
10-506	F	1920		07	1946	4	1973	0	B6	0.0	Z9C	0	0	1	0
10-510	F	1924		07	1942	26	1973	1	B6	0.0	Z9C	0	0	3	0
10-511	F	1923		01	1943	12	1973	5	B3	0.0	Z9C	1	0	12	0
10-512	F	1936		01	1965	1	1973	0	B6	0.0	Z9C	0	0	0	0
10-518	F	1905		06	1928	1196	1973	2	B6	0.0	Z9B	0	0	4	0
10-520	F	1924		01	1942	5	1973	1	B6	0.0	Z9C	0	0	3	0
10-521	F	1923		01	1955	416	1973	1	B6	0.0	Z9C	0	0	2	0
10-523	F	1922		01	1942	17	1973	0	B6	0.0	Z9C	0	0	0	0
10-525	F	1928		01	1947	1	1973	1	B6	0.0	Z9C	0	0	2	0
10-530	F	1952		07	1971	52	1973	3	B6	0.0	Z9C	0	0	0	0
10-531	F	1924		01	1946	1	1973	2	B6	0.0	Z9C	0	0	4	0
10-532	F	1916		01	1942	2	1973	1	B6	0.0	Z9C	0	0	3	0
10-533	F	1925		01	1943	5	1973	2	B6	0.0	Z9C	0	0	5	0
10-534	F	1925		01	1946	54	1973	2	C6	0.0	Z9C	0	0	4	0
10-535	F	1927		01	1946	16	1973	1	C6	0.0	Z9C	0	0	2	0
10-536	F	1927		01	1942	1	1973	1	B6	0.0	Z9C	0	0	3	0
10-538	M	1896		07	1938	2028	1977	1	B2	0.0	Z9C	0	0	1	0
10-540	M	1917		01	1939	1768	1973	2	B6	0.0	Z9C	0	0	1	0
10-543	M	1891		06	1916	26	1973	3	B3	0.0	Z9B	1	0	11	0
10-546	F	1906		07	1929	208	1973	5	B3	0.0	Z9C	1	0	17	0
10-549	F	1919		01	1941	62	1973	4	B3	0.0	Z9C	1	0	11	0
10-550	F	1914		17	1965	230	1973	1	B6	0.0	Z9C	0	0	0	0
10-557	F	1921		01	1942	43	1974	4	B3	0.0	Z9C	1	0	10	0
10-558	M	1927		07	1951	+0	1973	5	B3	0.0	Z9C	1	0	6	0
10-559	F	1919		01	1941	69	1973	2	B6	0.0	Z9C	0	0	4	0
10-560	F	1923		01	1942	96	1973	4	B3	0.0	Z9C	1	0	9	0
10-561	M	1906		06	1927	52	1973	6	B6	0.0	Z9B	2	0	17	0
10-566	M	1914	1977	02	1930	13	1976	5	B2	0.00334	Z5B	1	1	14	14
10-569	F	1925		01	1946	1	1975	0	B6	0.0	Z9C	0	0	0	0
10-570	M	1907		06	1934	780	1977	2	B3	0.0	Z9C	0	0	3	0
10-573	F	1922		01	1944	14	1973	3	B3	0.0	Z9C	1	0	6	0
10-574	M	1908		71	1930	2236	1973	7	B2	0.0	Z9C	1	0	6	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-575	F	1930		01	1948	1040	1973	4	B3	0.0	Z9C	1	0	4	0
10-579	M	1926		07	1948	1248	1973	0	B6	0.0	Z9C	0	0	0	0
10-580	F	1930		01	1948	52	1973	3	B3	0.0	Z9C	1	0	5	0
10-582	F	1938		01	1965	416	1973	1	B6	0.0	Z9C	0	0	0	0
10-583	M	1918		06	1939	1352	1973	0	B6	0.0	Z9C	0	0	0	0
10-584	F	1925		01	1942	3	1973	1	B6	0.0	Z9C	0	0	2	0
10-585	M	1908		06	1930	52	1973	2	B6	0.0	Z9C	1	0	5	0
10-587	M	1946		07	1966	416	1973	1	B6	0.0	Z9C	0	0	0	0
10-588	F	1910		01	1927	2	1974	0	G6	0.00330	Z8	0	0	0	0
10-589	M	1938		07	1971	78	1973	2	B3	0.0	Z9C	0	0	0	0
10-590	M	1912		06	1948	728	1974	0	B6	0.0	Z9B	0	0	0	0
10-592	M	1899		06	1923	1300	1973	0	B6	0.0	Z9B	0	0	0	0
10-594	F	1917		01	1943	5	1973	5	B3	0.0	Z9C	1	0	13	0
10-595	F	1908		01	1928	104	1977*	6	M6	0.0	Z9	2	0	24	0
10-596	F	1909		01	1927	6	1973	6	B3	0.0	Z9C	2	0	22	0
10-597	F	1911		01	1928	17	1976	2	B3	0.0	Z9C	1	0	8	0
10-598	F	1914		01	1934	156	1973	1	B6	0.0	Z9C	0	0	3	0
10-601	M	1920		07	1951	0	1975	0	B6	0.0	Z9B	0	0	0	0
10-606	F	1910		07	1928	468	1975	0	B6	0.0	Z9B	0	0	0	0
10-608	F	1917		01	1936	4	1975	1	B6	0.0	Z9C	0	0	2	0
10-609	F	1925		01	1943	42	1973	2	B6	0.0	Z9C	0	0	4	0
10-610	F	1920		01	1941	22	1975	2	B3	0.0	Z9C	0	0	5	0
10-611	F	1924		01	1942	13	1973	2	B6	0.0	Z9C	0	0	5	0
10-613	F	1919		01	1945	12	1973	0	B6	0.0	Z9C	0	0	0	0
10-614	F	1915		01	1942	30	1975	1	B6	0.0	Z9C	0	0	2	0
10-616	F	1929		01	1948	15	1973	2	B6	0.0	Z9C	0	0	3	0
10-617	F	1922		01	1942	182	1974	10	B2	0.0	Z9C	2	0	25	0
10-618	F	1923		01	1942	130	1975	0	B6	0.0	Z9C	0	0	1	0
10-621	M	1905		06	1925	1716	1973	1	B6	0.0	Z9B	0	0	1	0
10-623	M	1917		06	1938	1144	1973	1	B6	0.0	Z9B	0	0	1	0
10-627	M	1911		07	1928	208	1974	4	G6	0.00420	Z5	1	1	11	11
10-628	M	1906		06	1927	156	1976	0	B6	0.0	Z9B	0	0	0	0
10-630	F	1915		01	1937	13	1973	0	B6	0.0	Z9C	0	0	1	0
10-631	F	1929		01	1946	26	1974	0	B6	0.0	Z9C	0	0	0	0
10-635	F	1922		01	1943	156	1973	3	B6	0.0	Z9C	1	0	6	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-644	M	1870	1927	05	1927	0	1975	5300	A1	0.0	Z9	29	0	4	0
10-645	F	1930		76	1948	90	1973	0	B6	0.0	Z9C	0	0	0	0
10-648	F	1923		01	1942	30	1974	2	B6	0.0	Z9C	0	0	4	0
10-649	F	1921		01	1942	15	1973	2	B6	0.0	Z9C	0	0	4	0
10-650	F	1926		01	1946	59	1973	8	B2	0.0	Z9C	2	0	17	0
10-651	F	1923		01	1942	260	1974	0	B6	0.0	Z9C	0	0	0	0
10-653	F	1926		01	1946	16	1973	0	B6	0.0	Z9C	0	0	0	0
10-656	F	1923		01	1942	20	1973	1	B6	0.0	Z9C	0	0	2	0
10-657	F	1922	1976	01	1943	13	1973	1	B6	0.0	Z9C	0	0	3	0
10-658	F	1906		01	1927	208	1974	6	B2	0.0	Z9C	2	0	23	0
10-659	F	1904		01	1927	52	1974	0	B6	0.0	Z9C	0	0	2	0
10-660	F	1924		01	1942	172	1973	18	B2	0.0	Z9C	4	0	44	0
10-662	F	1909		01	1930	13	1977	2	B3	0.0	Z9C	1	0	9	0
10-664	F	1925		01	1940	1	1973	3	B3	0.0	Z9C	1	0	8	0
10-665	F	1927		01	1946	104	1973	1	B6	0.0	Z9C	0	0	3	0
10-666	F	1924		01	1943	13	1974	1	B6	0.0	Z9C	0	0	2	0
10-667	F	1908	1974	01	1925	52	1973	7	B2	0.0	Z9C	2	0	26	0
10-668	F	1925		01	1943	19	1973	1	B6	0.0	Z9C	0	0	2	0
10-670	M	1932		06	1955	780	1974	2	B3	0.0	Z9C	0	0	1	0
10-672	M	1916		06	1936	1040	1974	0	B6	0.0	Z9B	0	0	0	0
10-673	M	1911	1976	06	1932	364	1973	0	B6	0.0	Z9B	0	0	0	0
10-683	F	1924		01	1942	14	1973	0	B6	0.0	Z9C	0	0	0	0
10-684	M	1927		07	1950	104	1974	1	B6	0.0	Z9C	0	0	2	0
10-688	F	1923	1976	01	1942	12	1974	4	B2	0.0	Z9C	1	0	11	0
10-689	F	1919		01	1943	26	1974	3	B3	0.0	Z9C	1	0	6	0
10-696	F	1911		01	1929	15	1977=	75	G6	0.0	Z9	22	0	299	0
10-714	F	1908		01	1925	57	1974	0	B6	0.00230	Z4B	0	0	0	0
10-723	F	1911		01	1929	15	1977=	1	C6	0.0	Z9C	0	0	4	0
10-725	M	1927		07	1952	1	1973	5	B2	0.0	Z9C	1	0	6	0
10-728	F	1923		01	1946	2	1974	0	B6	0.0	Z9C	0	0	0	0
10-729	F	1902		06	1920	832	1973	1	B6	0.0	Z9B	0	0	4	0
10-730	F	1907		01	1928	260	1974	4	B6	0.0	Z9C	1	0	13	0
10-731	M	1921		07	1951	1196	1974	2	B3	0.0	Z9C	0	0	1	0
10-732	M	1924		07	1950	1300	1974	0	B6	0.0	Z9C	0	0	0	0
10-736	F	1929		01	1948	9	1974	0	B6	0.0	Z9C	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-738	M	1923		07	1965	6	1974	3	B3	0.0	Z9C	0	0	1	0
10-739	F	1931		01	1951	7	1974	1	B6	0.0	Z9C	0	0	1	0
10-741	F	1927		01	1945	60	1977*	1	B6	0.0	Z9C	0	0	2	0
10-742	F	1929		07	1946	1	1974	2	B3	0.0	Z9C	0	0	4	0
10-744	F	1890		05	1925	0	1975	120	G4	0.0	Z9	37	0	517	0
10-754	F	1881	1977	05	1925	0	1975	12	G4	0.0	Z9	4	0	52	0
10-786	F	1866	1928	05	1927	0	1976	1360	A4	0.0	Z9	54	0	71	0
10-807	M	1894	1976	05	1925	1	1976	388	B1	0.0	Z9B	119	0	1190	0
10-825	M	1904		05	1927	0	1977	1173	B1	0.0	Z9B	357	0	3516	0
10-831	M	1879	1926	05	1925	+0	1977*	786	A1	0.0	Z9	31	0	29	0
10-840	M	1869	1926	05	1925	0	1976	390	A1	0.0	Z9	16	0	15	0
10-850	F	1925		01	1943	0	1974	1	B6	0.0	Z9C	0	0	3	0
10-851	F	1921		01	1951	139	1974	0	B6	0.0	Z9B	0	0	0	0
10-852	F	1905		01	1923	13	1974	0	B6	0.01300	Z2B	0	0	0	0
10-853	F	1919		17	1947	1300	1974	1	B6	0.0	Z9B	0	0	1	0
10-854	M	1909		06	1928	104	1974	0	B6	0.0	Z9B	0	0	0	0
10-855	F	1928		01	1946	28	1976	7	B2	0.0	Z9C	2	0	15	0
10-856	F	1952		01	1973	6	1974	1	B6	0.0	Z9C	0	0	0	0
10-859	F	1951		07	1973	0	1974	0	B6	0.0	Z9C	0	0	0	0
10-860	F	1925		07	1962	7	1974	7	B2	0.0	Z9C	1	0	7	0
10-861	F	1954		01	1973	22	1974	1	B6	0.0	Z9C	0	0	0	0
10-862	F	1928		01	1946	10	1974	0	B6	0.0	Z9C	0	0	0	0
10-864	M	1906		01	1949	1300	1974	1	B6	0.0	Z9C	0	0	0	0
10-867	F	1915		07	1929	209	1974	0	B6	0.0	Z9B	0	0	0	0
10-869	F	1902		01	1927	132	1974	0	B6	0.00330	Z8B	0	0	0	0
10-870	F	1911		07	1944	650	1974	0	B6	0.0	Z9B	0	0	0	0
10-874	F	1924		01	1942	728	1974	4	B3	0.0	Z9B	1	0	8	0
10-880	M	1912		06	1935	156	1974	0	B6	0.0	Z9B	0	0	0	0
10-883	F	1883	1935	02	1930	+0	1975	27	A1	0.0	Z9	2	0	8	0
10-890	F	1912		01	1927	2	1974	0	B6	0.00330	Z8B	0	0	0	0
10-893	F	1926		01	1943	78	1977	5	B2	0.0	Z9C	1	0	14	0
10-894	F	1924		01	1942	38	1974	1	B6	0.0	Z9C	0	0	2	0
10-895	F	1925		01	1943	9	1974	2	B3	0.0	Z9C	0	0	4	0
10-896	F	1923		01	1941	8	1974	0	B6	0.0	Z9C	0	0	1	0
10-897	F	1930		07	1951	208	1975	3	B6	0.0	Z9C	1	0	5	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-901	F	1910		01	1924	3	1975	0	B6	0.01160	Z2B	0	0	0	0
10-903	F	1909		01	1943	4	1976	0	B6	0.0	Z9C	0	0	1	0
10-905	F	1928		01	1946	10	1974	0	B6	0.0	Z9C	0	0	0	0
10-906	F	1921		07	1958	52	1976	1	B6	0.0	Z9C	0	0	1	0
10-907	F	1910		01	1946	5	1974	0	B6	0.0	Z9C	0	0	0	0
10-908	F	1928		01	1946	4	1974	1	B6	0.0	Z9C	0	0	2	0
10-909	F	1919		01	1941	4	1974	2	B3	0.0	Z9C	1	0	6	0
10-911	F	1928		01	1947	2	1974	2	B6	0.0	Z9C	0	0	4	0
10-915	F	1931		01	1953	0	1974	1	B6	0.0	Z9C	0	0	1	0
10-916	F	1915		01	1946	2	1974	0	B6	0.0	Z9C	0	0	0	0
10-918	F	1907		01	1923	0	1976	0	B6	0.01000	Z2B	0	0	0	0
10-919	F	1924		01	1943	8	1974	2	B6	0.0	Z9C	0	0	4	0
10-920	F	1929		01	1947	4	1977*	0	C6	0.0	Z9C	0	0	0	0
10-921	F	1905		01	1923	1	1977*	0	G6	0.00907	Z2	0	0	0	0
10-928	M	1918		07	1948	0	1958	1	G6	0.0	Z9	0	0	1	0
10-931	M	1911		01	1946	1040	1974	5	B2	0.0	Z9C	1	0	5	0
10-932	M	1903		76	1919	208	1977	15	B1	0.0	Z9B	5	0	51	0
10-933	F	1924		01	1943	3	1974	2	B6	0.0	Z9C	0	0	5	0
10-934	F	1924		01	1948	1196	1974	0	B6	0.0	Z9C	0	0	0	0
10-935	M	1925		07	1959	780	1974	0	B6	0.0	Z9C	0	0	0	0
10-938	F	1952		01	1971	8	1974	0	B6	0.0	Z9C	0	0	0	0
10-940	F	1939		07	1958	4	1974	1	B6	0.0	Z9C	0	0	1	0
10-941	F	1928		01	1948	13	1974	1	B6	0.0	Z9C	0	0	1	0
10-944	F	1922		01	1951	6	1974	0	B6	0.0	Z9C	0	0	0	0
10-945	F	1915		01	1943	12	1974	9	B2	0.0	Z9C	2	0	24	0
10-948	F	1923		01	1943	3	1974	0	B6	0.0	Z9C	0	0	1	0
10-949	F	1925		01	1943	0	1974	2	B3	0.0	Z9C	0	0	5	0
10-950	F	1922		01	1943	1	1974	5	B2	0.0	Z9C	1	0	12	0
10-951	F	1916		01	1943	8	1974	1	B6	0.0	Z9C	0	0	2	0
10-952	F	1911		01	1927	10	1974	1	B6	0.00329	Z8B	0	0	4	4
10-955	F	1922		01	1942	104	1974	1	B6	0.0	Z9B	0	0	3	0
10-957	F	1922		01	1941	130	1974	1	B6	0.0	Z9B	0	0	3	0
10-958	F	1931		01	1951	13	1975	3	B3	0.0	Z9C	1	0	6	0
10-959	F	1929		01	1946	2	1974	4	B3	0.0	Z9C	1	0	8	0
10-963	F	1901		01	1919	10	1975	647	B1	0.00170	B3B	209	318	3168	4784

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
10-966	F	1908		01	1929	4	1974	0	B6	0.0	Z9B	0	0	0	0
10-967	F	1924		01	1943	2	1974	0	B6	0.0	Z9C	0	0	0	0
10-969	M	1920		07	1969	52	1976	0	B6	0.0	Z9C	0	0	0	0
10-970	F	1955		07	1973	22	1974	2	B3	0.0	Z9C	0	0	0	0
10-971	F	1952		17	1973	22	1975	1	B6	0.0	Z9C	0	0	0	0
10-972	F	1926		01	1947	5	1974	0	B6	0.0	Z9C	0	0	1	0
10-974	F	1924		01	1941	48	1974	0	B6	0.0	Z9B	0	0	0	0
10-975	F	1929		01	1947	13	1974	0	B6	0.0	Z9C	0	0	0	0
10-977	F	1923		01	1943	38	1974	6	B2	0.0	Z9C	1	0	15	0
10-978	M	1927		07	1943	1612	1974	4	B3	0.0	Z9C	0	0	2	0
10-979	F	1925		01	1943	13	1974	1	B6	0.0	Z9C	0	0	2	0
10-980	F	1926		07	1945	1	1974	1	B6	0.0	Z9C	0	0	2	0
10-981	F	1928		07	1946	0	1974	0	B6	0.0	Z9C	0	0	0	0
10-987	F	1926		01	1946	26	1974	1	B6	0.0	Z9C	0	0	3	0
10-988	M	1952	1974	07	1973	22	1974	0	B6	0.0	Z9C	0	0	0	0
10-989	F	1927		07	1945	52	1975	1	B6	0.0	Z9C	0	0	1	0
10-990	F	1920		07	1943	20	1974	0	B6	0.0	Z9C	0	0	0	0
10-991	M	1901		07	1941	1716	1974	1	B6	0.0	Z9C	0	0	1	0
10-992	F	1919		01	1942	39	1974	0	B6	0.0	Z9C	0	0	0	0
10-993	F	1904		07	1942	4	1974	0	B6	0.0	Z9C	0	0	0	0
10-996	F	1900		07	1943	806	1974	7	G3	0.0	Z9	1	0	13	0
10-997	F	1926		07	1945	572	1974	8	G3	0.0	Z9	2	0	15	0
10-998	F	1909		07	1942	988	1974	7	G6	0.0	Z9	1	0	12	0
11-002	F	1919		01	1951	130	1976	0	G6	0.0	Z9	0	0	0	0
11-003	F	1919		07	1942	+0	1974	3	G6	0.0	Z9	1	0	8	0
11-004	M	1924		01	1946	702	1974	0	G6	0.0	Z9	0	0	0	0
11-005	M	1926		17	1948	520	1974	2	G6	0.0	Z9	0	0	2	0
11-009	F	1913		07	1942	884	1974	0	B6	0.0	Z9B	0	0	0	0
11-010	F	1922		07	1942	572	1974	0	G6	0.0	Z9	0	0	0	0
11-015	F	1907		01	1925	2	1976	0	G6	0.01000	Z2	0	0	0	0
11-017	F	1906		01	1923	1	1977*	0	G6	0.00907	Z2	0	0	0	0
11-018	F	1908		01	1925	5	1974	0	B6	0.00330	Z8B	0	0	0	0
11-023	F	1911		17	1927	2	1975	0	B6	0.00290	Z8B	0	0	0	0
11-026	F	1916		01	1941	52	1976	0	B6	0.0	Z9C	0	0	0	0
11-027	F	1910		71	1948	312	1974	0	G6	0.0	Z9	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
11-028	F	1925		01	1944	78	1974	0	B6	0.0	Z9B	0	0	0	0
11-030	F	1928		07	1951	112	1975	4	B3	0.0	Z9B	1	0	7	0
11-032	M	1931		06	1956	936	1974	3	B3	0.0	Z9C	0	0	1	0
11-033	M	1951		06	1973	104	1975	0	B6	0.0	Z9C	0	0	0	0
11-034	M	1915		06	1934	2184	1977	51	B2	0.0	Z9C	8	0	45	0
11-035	M	1949		07	1973	60	1977*	0	C6	0.0	Z9C	0	0	0	0
11-036	M	1914		07	1946	1456	1974	7	B2	0.0	Z9C	1	0	4	0
11-038	M	1914		06	1940	1456	1974	18	B2	0.0	Z9C	3	0	19	0
11-040	M	1915		67	1939	1560	1974	6	B3	0.0	Z9C	1	0	6	0
11-042	M	1923		07	1946	1456	1974	5	B3	0.0	Z9C	1	0	3	0
11-045	M	1915	1976	06	1943	1560	1974	27	B2	0.0	Z9C	4	0	18	0
11-049	F	1908		01	1923	13	1975	0	B6	0.01160	Z2B	0	0	0	0
11-053	F	1905		01	1923	0	1977*	0	G6	0.00907	Z2	0	0	0	0
11-056	F	1908		01	1927	40	1974	2	B6	0.00330	Z8B	1	1	8	8
11-059	F	1925		01	1943	13	1974	0	B6	0.0	Z9B	0	0	0	0
11-065	F	1928		07	1943	13	1974	0	B6	0.0	Z9B	0	0	0	0
11-070	F	1924		01	1945	26	1974	1	B6	0.0	Z9	0	0	1	0
11-071	F	1935		07	1967	2	1974	2	B3	0.0	Z9C	0	0	1	0
11-086	F	1919		01	1941	208	1977*	2	C6	0.0	Z9C	0	0	5	0
11-087	M	1923		07	1941	52	1977*	3	C6	0.0	Z9C	1	0	6	0
11-092	F	1911		01	1943	52	1977*	0	C6	0.0	Z9C	0	0	0	0
11-107	F	1916		01	1942	52	1977*	0	B6	0.0	Z9C	0	0	1	0
11-108	F	1923		07	1943	208	1977*	1	B6	0.0	Z9C	0	0	2	0
11-112	F	1916		01	1943	52	1977*	1	B6	0.0	Z9C	0	0	2	0
11-119	F	1918		01	1941	117	1976	0	B6	0.0	Z9B	0	0	0	0
11-121	F	1909		01	1950	520	1977*	0	C6	0.0	Z9C	0	0	0	0
11-143	F	1923		01	1940	104	1977*	0	C6	0.0	Z9C	0	0	1	0
11-161	F	1921		01	1940	130	1976	0	B6	0.0	Z9B	0	0	0	0
11-176	F	1915		01	1942	208	1977*	2	C6	0.0	Z9C	1	0	6	0
11-192	F	1924		07	1943	104	1977*	1	B1	0.0	Z9C	0	0	2	0
11-196	F	1916		06	1941	312	1977*	1	B6	0.0	Z9C	0	0	2	0
11-207	M	1917		01	1939	208	1974	0	B6	0.0	Z9B	0	0	0	0
11-230	F	1904		07	1942	104	1976	4	B6	0.0	Z9B	1	0	11	0
11-246	F	1916		07	1942	78	1977*	1	B6	0.0	Z9C	0	0	2	0
11-262	F	1913		01	1933	208	1975	2	B3	0.0	Z9C	1	0	7	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BCRN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
11-264	F	1915		01	1934	130	1976	0	B6	0.0	Z9C	0	0	0	0
11-285	F	1915		07	1946	208	1974	0	B6	0.0	Z9C	0	0	0	0
11-291	F	1919		17	1951	164	1974	3	B3	0.0	Z9C	1	0	5	0
11-294	M	1943		07	1968	6	1974	0	B6	0.0	Z9C	0	0	0	0
11-296	M	1923		07	1961	156	1976	45	G3	0.0	Z9	7	0	31	0
11-297	M	1914		67	1934	1872	1976	9	B2	0.0	Z9C	2	0	10	0
11-302	F	1901		01	1924	0	1976	0	B6	0.01000	Z2B	0	0	0	0
11-361	F	1910		01	1925	23	1977*	1	B6	0.00230	Z8B	0	0	4	6
11-368	F	1910		01	1927	1	1977*	0	G6	0.00230	Z8	0	0	0	0
11-389	F	1908		01	1924	7	1976	3	B3	0.01150	Z2B	1	6	13	89
11-411	F	1905		17	1922	345	1977*	35	B1	0.00907	Z2B	11	51	153	768
11-453	F	1923		01	1942	13	1976	0	B6	0.0	Z9B	0	0	0	0
11-521	F	1910		01	1927	4	1974	0	B6	0.00330	Z8C	0	0	0	0
11-531	F	1894		01	1918	54	1977	7	G4	0.00134	Z5	2	4	35	57
11-561	F	1910		01	1925	2	1976	0	G6	0.00260	Z8	0	0	0	0
11-565	F	1911		01	1927	76	1974	2	B6	0.00330	Z8B	1	1	8	8
11-584	F	1904		01	1922	15	1977*	4	B6	0.0	Z9B	1	0	19	0
11-637	M	1902		06	1934	52	1975	0	B6	0.0	Z9B	0	0	0	0
11-655	M	1922		06	1953	156	1976	1	B3	0.0	Z9C	0	0	1	0
11-660	F	1928		01	1947	416	1976	5	B2	0.0	Z9C	1	0	9	0
11-661	M	1926		07	1948	1456	1976	6	B2	0.0	Z9C	1	0	3	0
11-803	F	1905		06	1942	13	1976	0	G6	0.0	Z9	0	0	0	0
11-861	F	1922		01	1941	364	1977*	0	B6	0.0	Z9B	0	0	0	0
11-863	F	1916		01	1942	52	1977	0	B6	0.0	Z9	0	0	0	0
11-866	F	1907		17	1942	156	1977*	0	B6	0.0	Z9B	0	0	0	0
11-871	F	1925		01	1940	276	1977*	5	B3	0.0	Z9B	1	0	13	0
11-875	F	1923		01	1941	364	1977*	1	B6	0.0	Z9B	0	0	3	0
11-916	F	1918		01	1941	108	1975	1	B6	0.0	Z9B	0	0	3	0
11-923	F	1924		01	1942	208	1976	1	B1	0.0	Z9C	0	0	2	0
11-925	F	1920		01	1941	78	1975	0	B6	0.0	Z9B	0	0	0	0
11-938	F	1931		01	1951	56	1975	0	B6	0.0	Z9B	0	0	0	0
11-947	F	1925		01	1947	260	1975	4	B3	0.0	Z9B	1	0	8	0
11-959	F	1912		01	1941	208	1977*	0	B6	0.0	Z9B	0	0	0	0
11-960	F	1924		01	1942	31	1975	0	B6	0.0	Z9B	0	0	0	0
11-973	F	1919		01	1950	108	1975	1	B6	0.0	Z9B	0	0	2	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
11-974	F	1917		01	1944	40	1977*	0	B6	0.0	Z9B	0	0	0	0
11-982	F	1922		01	1942	208	1976	0	B6	0.0	Z9B	0	0	0	0
11-989	F	1921		01	1943	35	1977*	0	C6	0.0	Z9C	0	0	0	0
11-991	F	1924		01	1942	6	1976	2	B6	0.0	Z9B	1	0	5	0
12-002	F	1918		01	1941	52	1976	0	B6	0.0	Z9B	0	0	0	0
12-016	F	1919		01	1941	111	1977*	0	B6	0.0	Z9B	0	0	0	0
12-025	F	1924		01	1951	182	1975	1	B6	0.0	Z9C	0	0	2	0
12-026	F	1914		01	1942	166	1976	0	B6	0.0	Z9B	0	0	0	0
12-033	F	1925		07	1950	52	1975	3	B3	0.0	Z9B	1	0	6	0
12-040	F	1921		01	1942	156	1976	3	G6	0.0	Z9	1	0	8	0
12-045	F	1925		01	1942	160	1977*	0	B6	0.0	Z9B	0	0	0	0
12-061	F	1920		01	1942	182	1975	1	B6	0.0	Z9B	0	0	3	0
12-074	F	1923		01	1943	104	1977*	1	B6	0.0	Z9B	0	0	3	0
12-075	F	1923		01	1941	208	1977*	1	B6	0.0	Z9B	0	0	3	0
12-086	F	1925		07	1942	156	1977*	2	B6	0.0	Z9B	0	0	5	0
12-089	F	1928		01	1943	52	1974	0	B6	0.0	Z9B	0	0	0	0
12-094	F	1929		01	1946	4	1975	3	B6	0.0	Z9C	1	0	6	0
12-095	F	1927		01	1947	1	1974	0	B6	0.0	Z9C	0	0	1	0
12-098	F	1930		01	1951	52	1974	1	B6	0.0	Z9C	0	0	1	0
12-099	F	1929		07	1950	156	1976	0	B6	0.0	Z9C	0	0	0	0
12-108	F	1915		01	1943	26	1974	0	B6	0.0	Z9C	0	0	0	0
12-110	F	1927		01	1945	13	1976	0	B6	0.0	Z9C	0	0	0	0
12-111	F	1929		01	1947	19	1974	4	B3	0.0	Z9C	1	0	9	0
12-113	F	1915		01	1940	22	1975	0	B6	0.0	Z9C	0	0	1	0
12-115	F	1953		07	1972	52	1975	0	B6	0.0	Z9C	0	0	0	0
12-117	F	1914		01	1943	3	1974	3	B3	0.0	Z9C	1	0	8	0
12-118	F	1932		16	1950	104	1977*	1	B3	0.0	Z9C	0	0	3	0
12-119	F	1938		17	1968	52	1975	1	B6	0.0	Z9C	0	0	0	0
12-123	F	1924		01	1945	17	1976	1	B3	0.0	Z9C	0	0	3	0
12-127	F	1917		01	1941	17	1975	0	B6	0.0	Z9C	0	0	0	0
12-129	F	1927		01	1946	4	1976	0	B6	0.0	Z9C	0	0	1	0
12-130	F	1924		01	1947	2	1976	5	B2	0.0	Z9C	1	0	10	0
12-133	F	1926		01	1944	104	1976	1	B3	0.0	Z9C	0	0	3	0
12-134	F	1927		01	1944	4	1975	0	B6	0.0	Z9C	0	0	0	0
12-136	F	1928		07	1965	30	1975	1	B6	0.0	Z9C	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
12-142	F	1922		01	1942	8	1976	0	B6	0.0	Z9C	0	0	0	0
12-143	F	1924		01	1941	52	1975	1	B6	0.0	Z9C	0	0	2	0
12-145	F	1921		01	1941	35	1976	0	B6	0.0	Z9C	0	0	0	0
12-146	F	1920		01	1942	208	1977*	0	B6	0.0	Z9C	0	0	1	0
12-148	F	1925		01	1946	4	1975	0	B6	0.0	Z9C	0	0	0	0
12-150	F	1919		01	1943	104	1976	6	B3	0.0	Z9C	1	0	15	0
12-155	F	1929		01	1955	52	1976	0	B6	0.0	Z9C	0	0	1	0
12-163	F	1920		01	1942	78	1974	4	B3	0.0	Z9C	1	0	10	0
12-164	F	1920		01	1943	13	1976	0	B6	0.0	Z9C	0	0	1	0
12-165	F	1917		01	1947	78	1974	3	B3	0.0	Z9C	1	0	7	0
12-168	F	1926		01	1946	13	1975	1	B6	0.0	Z9C	0	0	2	0
12-171	F	1921		01	1940	4	1976	2	C6	0.0	Z9C	0	0	5	0
12-173	F	1930		01	1949	1	1974	2	B3	0.0	Z9C	0	0	4	0
12-174	F	1924		01	1947	21	1976	0	B6	0.0	Z9C	0	0	0	0
12-175	F	1927		01	1946	39	1975	1	B6	0.0	Z9C	0	0	1	0
12-178	F	1925		01	1943	8	1976	0	B6	0.0	Z9C	0	0	1	0
12-179	F	1924		01	1943	9	1976	1	B6	0.0	Z9C	0	0	2	0
12-182	F	1922		01	1942	26	1977*	0	B6	0.0	Z9C	0	0	1	0
12-185	F	1920		01	1943	52	1975	0	B6	0.0	Z9C	0	0	0	0
12-186	F	1927		01	1945	4	1974	8	B2	0.0	Z9C	2	0	18	0
12-188	F	1936		07	1965	1	1976	1	B6	0.0	Z9C	0	0	0	0
12-190	F	1927		01	1948	8	1975	0	B6	0.0	Z9C	0	0	0	0
12-192	F	1921		01	1946	52	1976	1	B6	0.0	Z9C	0	0	2	0
12-193	F	1925		01	1942	1	1974	1	B6	0.0	Z9C	0	0	3	0
12-194	F	1924		01	1946	5	1977*	1	C6	0.0	Z9C	0	0	3	0
12-195	F	1925		01	1945	2	1976	1	B3	0.0	Z9C	0	0	3	0
12-197	F	1906		01	1922	26	1974	1	B6	0.0	Z9C	0	0	3	0
12-198	M	1909		17	1929	520	1976	0	B6	0.0	Z9B	0	0	0	0
12-204	M	1918		06	1941	104	1977*	0	C6	0.0	Z9C	0	0	0	0
12-206	F	1914		01	1942	130	1977*	1	B6	0.0	Z9C	0	0	2	0
12-212	M	1930		17	1958	988	1977*	2	C6	0.0	Z9C	0	0	1	0
12-214	F	1937		01	1967	26	1977*	0	C6	0.0	Z9C	0	0	0	0
12-215	F	1936		01	1958	936	1977*	0	B6	0.0	Z9C	0	0	0	0
12-216	F	1931		01	1957	104	1977*	0	B6	0.0	Z9C	0	0	0	0
12-218	M	1937		16	1955	17	1977*	0	B6	0.0	Z9C	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
12-221	F	1914		07	1954	572	1977*	1	B6	0.0	Z9C	0	0	1	0
12-223	F	1923		67	1963	728	1977*	0	B6	0.0	Z9C	0	0	0	0
12-224	F	1927		01	1963	738	1977*	0	B6	0.0	Z9C	0	0	0	0
12-226	F	1926		17	1961	520	1977*	0	B6	0.0	Z9C	0	0	0	0
12-228	F	1935		01	1959	22	1977*	0	B6	0.0	Z9C	0	0	0	0
12-229	F	1921		01	1955	676	1977*	1	B6	0.0	Z9C	0	0	1	0
12-236	F	1928		01	1960	130	1977*	1	B6	0.0	Z9C	0	0	1	0
12-237	F	1936		01	1954	52	1977*	0	B6	0.0	Z9C	0	0	1	0
12-239	F	1922		16	1956	104	1977*	2	C6	0.0	Z9C	0	0	3	0
12-262	F	1921		01	1942	52	1975	0	B6	0.0	Z9C	0	0	1	0
12-270	F	1919		01	1943	18	1975	0	B6	0.0	Z9C	0	0	1	0
12-304	F	1923		01	1943	52	1975	0	B6	0.0	Z9C	0	0	0	0
12-308	F	1900		01	1942	52	1975	2	B3	0.0	Z9C	1	0	6	0
12-330	M	1928		07	1944	63	1974	1	B6	0.0	Z9B	0	0	2	0
12-331	M	1930		07	1944	65	1974	0	B6	0.0	Z9B	0	0	0	0
12-333	M	1932		06	1955	728	1974	3	B3	0.0	Z9C	0	0	2	0
12-334	F	1908		01	1924	17	1975	4	B3	0.0	Z9C	1	0	18	0
12-343	F	1900	1976	07	1918	208	1974	0	G6	0.00530	Z4	0	0	0	0
12-344	F	1908		07	1930	104	1974	0	B6	0.0	Z9B	0	0	0	0
12-346	F	1908		01	1926	3	1975	3	B3	0.0	Z9C	1	0	13	0
12-349	F	1940		07	1961	156	1974	1	B6	0.0	Z9C	0	0	1	0
12-350	F	1906		01	1923	39	1974	1	B6	0.0	Z9C	0	0	5	0
12-352	F	1906		06	1928	416	1975	1	B6	0.0	Z9C	0	0	5	0
12-358	F	1913		01	1940	520	1976	7	B2	0.0	Z9C	2	0	17	0
12-364	F	1927		01	1968	364	1975	1	B6	0.0	Z9C	0	0	0	0
12-365	F	1931		01	1952	520	1975	1	B6	0.0	Z9	0	0	1	0
12-368	F	1923		01	1958	884	1975	2	C6	0.0	Z9C	0	0	1	0
12-370	F	1908		07	1924	104	1974	0	B6	0.01300	Z2B	0	0	0	0
12-375	F	1917		01	1958	312	1975	0	B6	0.0	Z9C	0	0	0	0
12-376	M	1945		07	1964	520	1977*	0	B6	0.0	Z9C	0	0	0	0
12-377	F	1920		01	1961	676	1975	0	B6	0.0	Z9C	0	0	0	0
12-383	F	1909		01	1923	988	1977*	0	G6	0.00159	Z5	0	0	0	0
12-397	M	1916		06	1947	520	1975	28	B2	0.0	Z9B	6	0	36	0
12-422	F	1907		01	1937	39	1975	0	B6	0.0	Z9B	0	0	0	0
12-425	M	1938		07	1960	6	1975	0	B6	0.0	Z9B	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
12-426	M	1923		07	1946	18	1975	1	B6	0.0	Z9B	0	0	2	0
12-428	F	1907		01	1922	13	1977	167	B1	0.0	Z9C	53	0	782	0
12-429	F	1922		01	1945	13	1975	0	B6	0.0	Z9C	0	0	0	0
12-430	F	1927		01	1941	26	1975	1	B6	0.0	Z9C	0	0	2	0
12-432	M	1937		06	1959	572	1977*	1	B6	0.0	Z9C	0	0	0	0
12-436	F	1896		01	1918	26	1975	1	B6	0.0	Z9C	0	0	3	0
12-437	F	1926		01	1943	104	1975	1	B6	0.0	Z9C	0	0	3	0
12-438	M	1942		06	1964	122	1977*	1	C6	0.0	Z9C	0	0	1	0
12-443	M	1919		06	1945	13	1976	1	B6	0.0	Z9C	0	0	2	0
12-447	M	1918		06	1940	260	1976	6	B2	0.0	Z9C	2	0	12	0
12-450	M	1911		07	1946	20	1977*	0	B6	0.0	Z9B	0	0	0	0
12-451	M	1949		06	1969	13	1977*	0	C6	0.0	Z9C	0	0	0	0
12-452	M	1948		06	1970	52	1977*	1	B3	0.0	Z9C	0	0	0	0
12-456	M	1918		06	1938	364	1976	249	B1	0.0	Z9C	62	0	491	0
12-460	M	1923		17	1945	1092	1975	0	B6	0.0	Z9B	0	0	0	0
12-499	F	1908		01	1925	8	1975	2	C6	0.0	Z9C	1	0	8	0
12-502	F	1924		01	1945	13	1975	0	B6	0.0	Z9C	0	0	0	0
12-508	F	1937		17	1957	884	1975	0	B6	0.0	Z9C	0	0	0	0
12-509	F	1918		01	1941	160	1977*	0	B6	0.0	Z9C	0	0	0	0
12-510	F	1923		01	1941	364	1977*	1	C6	0.0	Z9C	0	0	3	0
12-522	F	1921		01	1941	30	1977*	0	B6	0.0	Z9C	0	0	1	0
12-523	F	1923		01	1941	104	1977*	0	C6	0.0	Z9C	0	0	1	0
12-529	F	1920		01	1941	104	1977*	0	C6	0.0	Z9C	0	0	1	0
12-530	M	1920		07	1958	364	1976	3	B2	0.0	Z9C	1	0	2	0
12-532	M	1905		17	1929	2132	1975	1	B6	0.0	Z9C	0	0	1	0
12-533	F	1952		07	1970	260	1975	2	B6	0.0	Z9C	0	0	0	0
12-544	F	1921		01	1941	534	1975	4	B3	0.0	Z9B	1	0	9	0
12-545	F	1920		01	1937	902	1975	11	B2	0.0	Z9B	3	0	25	0
12-547	F	1918		01	1942	1508	1975	3	B3	0.0	Z9B	0	0	4	0
12-548	F	1919		17	1939	832	1975	1	B6	0.0	Z9B	0	0	2	0
12-549	F	1917		01	1943	604	1975	2	B6	0.0	Z9B	0	0	4	0
12-552	F	1922		01	1940	338	1975	7	B3	0.0	Z9B	2	0	18	0
12-553	F	1922		01	1950	260	1976	0	B6	0.0	Z9C	0	0	0	0
12-556	F	1922		01	1942	213	1975	3	B3	0.0	Z9B	1	0	7	0
12-557	F	1919		01	1936	676	1976	2	B3	0.0	Z9C	1	0	6	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
12-559	F	1919		0	1939	104	1976	1	B6	0.0	Z9C	0	0	2	0
12-561	F	1917		16	1942	243	1975	0	B6	0.0	Z9B	0	0	0	0
12-563	F	1913		01	1940	289	1976	11	B2	0.0	Z9B	3	0	29	0
12-579	F	1921		01	1941	208	1977*	0	C6	0.0	Z9C	0	0	0	0
12-582	F	1914		01	1941	26	1977*	0	C6	0.0	Z9C	0	0	0	0
12-583	M	1923		08	1923	39	1976	0	B6	0.0	Z9B	0	0	0	0
12-623	F	1934		01	1957	102	1977*	0	C6	0.0	Z9C	0	0	0	0
12-624	F	1939		01	1965	312	1976	0	B6	0.0	Z9C	0	0	0	0
12-640	F	1946		07	1964	9	1977*	0	B6	0.0	Z9C	0	0	0	0
12-643	F	1933		01	1957	126	1977*	0	C6	0.0	Z9C	0	0	0	0
12-644	F	1934		01	1972	52	1977*	1	B6	0.0	Z9C	0	0	0	0
12-645	F	1944		01	1963	156	1977*	1	B6	0.0	Z9C	0	0	1	0
12-646	F	1946		01	1965	260	1977*	0	B6	0.0	Z9C	0	0	0	0
12-650	F	1931		01	1949	1456	1977*	2	B3	0.0	Z9C	0	0	1	0
12-652	F	1931		01	1953	56	1977*	2	B3	0.0	Z9C	0	0	3	0
12-654	M	1942		07	1962	43	1977*	3	B3	0.0	Z9C	0	0	2	0
12-656	M	1944		01	1962	104	1976	2	B2	0.0	Z9C	0	0	1	0
12-657	M	1924		06	1950	520	1977*	6	B2	0.0	Z9C	1	0	7	0
12-660	M	1926		16	1955	39	1977*	2	B3	0.0	Z9C	0	0	2	0
12-661	F	1946		01	1965	13	1977*	0	B6	0.0	Z9C	0	0	0	0
12-665	F	1925		07	1971	260	1977*	1	B6	0.0	Z9C	0	0	0	0
12-669	M	1957		07	1974	22	1977*	0	B6	0.0	Z9C	0	0	0	0
12-670	M	1929		01	1951	52	1977*	1	B3	0.0	Z9C	0	0	2	0
12-688	F	1917		01	1944	17	1977*	1	B6	0.0	Z9C	0	0	2	0
12-694	F	1931		01	1949	13	1976	0	B6	0.0	Z9B	0	0	0	0
12-702	F	1918		61	1942	160	1977*	1	B6	0.0	Z9C	0	0	3	0
12-709	F	1925		01	1952	121	1976	0	B6	0.0	Z9C	0	0	0	0
12-710	F	1911		01	1952	104	1976	0	B6	0.0	Z9C	0	0	1	0
12-746	F	1913		01	1942	124	1976	0	B6	0.0	Z9B	0	0	0	0
12-757	F	1922		01	1941	104	1976	1	B3	0.0	Z9C	0	0	3	0
12-764	F	1924		01	1952	104	1977*	1	B3	0.0	Z9C	0	0	2	0
12-765	F	1921		71	1949	1352	1976	0	B6	0.0	Z9C	0	0	0	0
12-771	F	1930		01	1949	936	1976	0	B6	0.0	Z9C	0	0	0	0
12-779	F	1929		01	1952	52	1976	0	B6	0.0	Z9C	0	0	0	0
12-784	F	1930		01	1953	17	1977*	5	C6	0.0	Z9C	1	0	9	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1977

(1) CASE	(2) SEX	(3) BORN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT FA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
12-795	F	1918		01	1949	17	1977*	1	B6	0.0	Z9C	0	0	1	0
12-810	F	1910		01	1943	104	1977*	0	B6	0.0	Z9C	0	0	0	0
12-826	F	1906		01	1943	8	1977*	2	C6	0.0	Z9C	1	0	5	0
12-829	F	1922		01	1949	18	1977*	0	C6	0.0	Z9C	0	0	0	0
12-841	F	1922		01	1952	1	1977*	0	C6	0.0	Z9C	0	0	0	0
12-849	F	1916		01	1941	208	1977*	3	C6	0.0	Z9C	1	0	7	0
12-850	F	1917		01	1951	26	1977*	0	B6	0.0	Z9C	0	0	0	0
12-857	F	1926		17	1951	208	1977*	0	B6	0.0	Z9C	0	0	0	0
12-878	F	1920		01	1949	237	1976	1	B6	0.0	Z9C	0	0	2	0
12-880	F	1917		01	1950	52	1977*	1	B3	0.0	Z9C	0	0	3	0
12-887	F	1925		01	1942	78	1977*	0	C6	0.0	Z9C	0	0	0	0
12-889	F	1924		01	1947	260	1976	1	B3	0.0	Z9C	0	0	2	0
12-901	F	1915		01	1951	13	1977*	0	C6	0.0	Z9C	0	0	0	0
12-905	F	1914		01	1949	312	1976	2	B3	0.0	Z9C	0	0	3	0
12-908	F	1923		01	1952	87	1976	0	B6	0.0	Z9B	0	0	0	0
12-916	F	1921		17	1942	676	1977*	2	C6	0.0	Z9C	0	0	4	0
12-918	F	1918		01	1940	208	1977*	1	B6	0.0	Z9C	0	0	2	0
12-924	F	1905		01	1950	17	1977*	0	B6	0.0	Z9C	0	0	0	0
12-927	F	1919		01	1942	13	1977*	2	B6	0.0	Z9C	0	0	5	0
12-929	F	1911		01	1942	4	1977*	0	C6	0.0	Z9C	0	0	0	0
12-942	F	1898		01	1944	+0	1977*	2	C6	0.0	Z9C	0	0	5	0
12-943	F	1917		01	1952	52	1976	1	B6	0.0	Z9C	0	0	2	0
12-965	F	1924		01	1945	52	1977*	2	B3	0.0	Z9C	0	0	5	0
12-978	F	1919		08	1919	39	1976	0	B6	0.0	Z9B	0	0	0	0
12-981	F	1907		01	1923	19	1977*	0	B6	0.00907	Z9B	0	0	0	0
12-983	F	1921		01	1940	1040	1976	6	B2	0.0	Z9C	1	0	12	0
12-985	M	1934		08	1934	39	1976	1	B6	0.0	Z9B	0	0	2	0
12-986	M	1932	1976	08	1932	39	1976	6	B3	0.0	Z9B	2	0	15	0
13-002	F	1901		61	1923	468	1977*	0	B6	0.0	Z9C	0	0	0	0
13-007	M	1911		67	1951	676	1976	1	B6	0.0	Z9B	0	0	1	0
13-010	F	1923		01	1942	26	1977*	2	B3	0.0	Z9C	0	0	5	0
13-011	F	1924		01	1942	39	1976	0	G6	0.0	Z9	0	0	0	0
13-015	F	1910		01	1954	884	1976	1	B6	0.0	Z9C	0	0	1	0
13-019	F	1915		01	1942	104	1977*	0	B6	0.0	Z9B	0	0	0	0
13-025	F	1914		01	1940	32	1977*	0	C6	0.0	Z9C	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS IC END OF 1977

(1) CASE	(2) SEX	(3) BCEN	(4) DIED	(5) EXP TYPE	(6) YEAR FIRST EXP	(7) EXP DUR WKS	(8) YEAR OF MEAS	(9) RA226 NCI	(10) RA226 METHOD + ERR	(11) RA228 TO RA226 RATIO	(12) RA228 METHOD + ERR	(13) INPUT RA226 UCI	(14) INPUT RA228 UCI	(15) CUM RADS RA226	(16) CUM RADS RA228
13-026	F	1921		01	1941	26	1977*	0	C6	0.0	Z9C	0	0	0	0
13-027	F	1922		01	1942	156	1977*	1	C6	0.0	Z9C	0	0	4	0
13-044	F	1954		07	1977	+0	1977*	0	B6	0.0	Z9C	0	0	0	0
13-050	M	1932		07	1977	+0	1977*	1	B6	0.0	Z9C	0	0	0	0
13-051	F	1878	1962	04	1925	+0	1949*	700	G4	0.0	Z9	145	0	1648	0
13-056	M	1958		06	1976	52	1977*	3	C6	0.0	Z9C	0	0	0	0
13-058	M	1956		16	1976	62	1977*	0	C6	0.0	Z9C	0	0	0	0

APPENDIX B. Radium-Induced Malignancies

Measured Persons

Tables 1 and 2 summarize measured radium cases considered to have radium-induced bone sarcomas and paranasal sinus or mastoid carcinomas, respectively. The cases are listed in order of skeletal dose, from both ^{226}Ra and ^{228}Ra, accumulated to the date of diagnosis of the tumor or to the date of death if there was no diagnosis before death. Detailed exposure and dosimetric data for these cases can be found in Table 1 of Appendix A of this report.

There are 58 bone sarcoma cases and 29 sinus or mastoid carcinoma cases among the 2072 persons whose body burdens of radium have been measured. Five persons had both types of tumor (cases 01-179, 03-110, 03-402, 03-429, and 03-648) so that there are 82 measured persons considered to have radium-induced malignancies. There are two more cases (03-106 and 05-953) in Table 1 than appeared in the corresponding table in the 1977 annual report.[1] A copy of a 1959 death certificate for case 03-106, listing a sarcoma of bone, left leg (2-yr duration) as the underlying cause of death, was obtained by CHR in 1977.[2] A fibrosarcoma of the right femur of case 05-953 was diagnosed in 1977.[2]

Positive evidence is lacking that two of the cases (03-110 and 03-417) listed in Table 2 were bona fide cases of malignant tumor of the mastoid or paranasal sinuses. Case 03-110 had a possible carcinoma of the mastoid and a possible sarcoma of the left first metacarpal diagnosed radiographically in 1963; biopsy was refused. She died in 1967 of a myocardial infarction; autopsy was refused. Case 03-417 had an epidermoid carcinoma, which apparently arose in the right gingiva and invaded the right maxilla, diagnosed in 1962. She died with widespread metastases in 1966.

Table 1. Bone Sarcomas in Persons with Known Radium Body Content as of 31 December 1977

CASE	SEX	BORN	DIED	EXPOSED	CUM.RADS	DIAGNOSED
00-003	F	1894	1927	1917	44441	1927
01-079	F	1901	1943	1920	21115	1942
01-032	F	1908	1940	1924	18248	1940
01-033	F	1908	1931	1923	18023	1930
03-584	F	1905	1959	1923	16821	1958
03-648	F	1903	1956	1922	16713	1956
00-019	F	1895	1946	1917	15042	1946
01-009	F	1898	1945	1918	14306	1944
03-213	F	1892	1955	1925	14049	1954
01-105	F	1898	1945	1921	12555	1945
00-006	F	1903	1930	1918	11760	1930
03-671	F	1906	1953	1922	11314	1952
01-046	F	1903	1943	1920	11190	1942
00-004	F	1900	1931	1917	11063	1930
00-028	F	1902	1933	1917	10265	1930
01-172	F	1898	1968	1916	9628	1968
03-201	F	1909	1963	1922	9586	1962
01-389	F	1910	1930	1923	9507	1930
05-215	F	1886	1968	1920	9458	1960
01-562	F	1901	1931	1920	7143	1931
03-215	M	1896	1971	1925	6860	1957
01-031	F	1906	1934	1925	6824	1934
03-401	F	1900	1963	1923	6781	1962
00-005	F	1901	1939	1917	6643	1939
05-953	F	1902	L	1918	6589	1977
03-619	F	1903	1962	1922	6184	1962
01-007	F	1886	1949	1926	5972	1948
01-059	F	1905	1967	1920	5182	1962
01-011	F	1872	1937	1919	5175	1936
03-118	F	1898	1955	1931	5159	1955
00-007	F	1903	1935	1919	5046	1934
00-027	F	1902	1942	1918	4995	1942
03-429	F	1908	1976	1923	4387	1967
01-051	F	1904	1977	1923	4265	1972
03-234	F	1890	1965	1915	3810	1964
05-281	F	1898	1964	1916	3804	1956
03-402	F	1905	L	1923	3761	1953
01-024	F	1901	1956	1919	3674	1956
01-179	F	1890	1966	1924	3642	1943
01-239	F	1901	1958	1917	3153	1955
01-520	F	1882	1969	1930	3132	1967
01-073	F	1900	1969	1921	3048	1969
01-099	F	1905	1945	1924	2923	1942
01-026	F	1905	1958	1925	2729	1955
03-649	F	1906	1954	1924	2664	1953

Table 1. (contd.)

CASE	SEX	BORN	DIED	EXPOSED	CUM.RADS	DIAGNOSED
01-025	F	1886	1952	1924	2497	1950
01-613	F	1906	1936	1923	2436	1936
03-212	F	1902	1951	1927	2412	1951
03-210	M	1906	1958	1926	2396	1956
03-209	M	1894	1960	1925	1698	1958
03-216	F	1907	1961	1922	1606	1959
01-268	F	1901	1968	1920	1602	1959
01-112	F	1908	1955	1924	1547	1954
03-227	F	1878	1952	1930	1470	1949
03-110	F	1899	1967	1931	1467	1963
03-455	F	1906	L	1922	1445	1934
03-106	F	1876	1959	1931	1323	1957
01-439	F	1880	1953	1922	888	1949

Table 2. Carcinomas of the Paranasal Sinuses and Mastoid Air Cells in Persons with Known Radium Body Content as of 31 December 1977

CASE	SEX	BORN	DIED	EXPOSED	CUM.RADS	DIAGNOSED
01-145	F	1900	1957	1918	25701	1957
01-008	F	1900	1958	1917	22309	1958
01-149	F	1888	1959	1919	20067	1958
01-087	F	1905	L	1921	18114	1957
03-648	F	1903	1956	1922	16455	1955
03-232	F	1898	1957	1917	14736	1956
01-006	F	1899	1938	1919	8505	1938
03-240	F	1916	1955	1930	7655	1953
03-206	M	1914	1975	1936	7056	1974
01-014	F	1901	1949	1916	6799	1949
03-676	F	1897	1977	1924	6433	1976
01-179	F	1890	1966	1924	6019	1965
03-429	F	1908	1976	1923	4783	1973
03-402	F	1905	L	1923	4596	1964
03-101	F	1908	1971	1931	4448	1970
01-171	M	1895	1975	1914	4311	1966
03-407	F	1905	1961	1923	4206	1959
03-214	F	1895	1966	1925	3964	1959
03-235	F	1900	1968	1928	3803	1965
03-126	F	1910	1965	1931	3449	1965
01-573	F	1892	1945	1916	3307	1945
03-105	M	1903	1957	1931	3143	1957
03-423	F	1907	1972	1923	2036	1971
03-417[a]	F	1909	1966	1924	1894	1962
03-141	M	1906	1963	1933	1550	1963
01-022	F	1900	1951	1917	1544	1951
03-110	F	1899	1967	1931	1467	1963
05-284	F	1899	1973	1919	1179	1970
03-488	F	1907	1975	1922	605	1973

[a] Carcinoma of case 03-417 apparently arose in R. gingiva (posterior maxilla).

Table 3. Probable or Confirmed Bone Sarcomas in Exposed Persons with Unknown or Uncertain Radium Body Content[a]

Case	Sex	Born	Died	Exposed	Diagnosed
00-011	F	1896	1936	1917	1935
00-013	F	1899	1933	1917	1933
00-023	F	1900	1929	1917	1929
00-030	F	1903	1924	1918	1923
00-031	F	1903	1940	1921	1938
00-035	F	1900	1941	1917	1941
01-088	F	1906	1931	1923	1931
01-103	F	1903	1946	1922	1946
01-107	F	1909	1935	1923	1935
01-108	F	1908	1947	1924	1947
01-117	F	1907	1931	1922	1931
01-387	F	1895	1943	1918	1943
01-465	M	1881	1943	1925	1943
01-695	F	1908	1935	1923	1935
03-658	F	1903	1938	1922	1938
03-660	F	1907	1936	1923	1935
03-661	F	1906	1934	1922	1934
03-665	F	1909	1930	1924	1929
03-680	F	1906	1946	1924	1943
03-759	F	1904	1930	1924	1930
03-779	F	1905	1942	1922	1942
03-800	F	1908	1945	1924	1944
03-806	F	1896	1956	1922	1956
03-848	F	1903	1958	1922	1958
05-987	F	1901	1962	1918	1962
09-087	M	1891	1934	1912	1933

[a]All were dial painters except cases 01-387 (iatrogenic, i.v. and oral), 01-465 (drank Radithor), and 09-087 (chemist).

Table 4. Probable or Confirmed Malignant Tumors of the Paranasal Sinuses and Mastoid Air Cells in Exposed Persons with Unknown or Uncertain Radium Body Content[a]

Case	Sex	Born	Died	Exposed	Diagnosed
01-587	F	1894	1943	1919	1943
03-675[b]	F	1896	1960	1922	1959
03-760	F	1907	1946	1924	1946
03-772	F	1904	1953	1922	1953
03-785	F	1903	1955	1925	1953

[a]All were dial painters.

[b]Death certificate lists paranasal sinus carcinoma as cause of death; Histologic diagnosis from biopsy tissue was rhabdomyosarcoma of the maxillary antrum.

Unmeasured Cases

Tables 3 and 4 list exposed persons with unknown or uncertain radium body content who had probable or confirmed bone sarcomas and probable or confirmed paranasal sinus or mastoid carcinomas, respectively. There are 26 probable or confirmed bone sarcoma cases and 5 probable or confirmed sinus or mastoid carcinoma cases among the approximately 1300 radium cases with unmeasured body burdens for whom medical data are available. We have evidence that 10 of these unmeasured persons had early radioactivity measurements which were interpreted to show a positive indication of radium in the body; work is in progress to estimate lower limits of radium content for these cases.

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