Distribution Category:
Biology and Medicine
(UC-48)

ANL-80-115
Part II

ARGONNE NATIONAL LABORATORY
9700 South Cass Avenue
Argonne, Illinois 60439

RADIOLOGICAL AND ENVIRONMENTAL
RESEARCH DIVISION ANNUAL REPORT
Center for Human Radiobiology
July 1979-June 1980

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

- DISCLAIMER -

May 1981

Preceding Report

ANL-79-65 Part II July 1978-June 1979

DISTRIBUTION OF THIS DOCUMENT IS UNLIMITED 278

FOREWORD

Vigorous efforts to elucidate the mechanisms and dosimetry of radium-induced malignancies continued during the past year. Paper 1 marshalls evidence that, contrary to previous assumptions, ^{228}Ra may have dosimetric significance comparable to that of ^{226}Ra for the induction of carcinomas of the sinus and mastoid epithelia. This paper also raises the possibility that ^{224}Ra and actinides that emit high-energy alpha particles may be capable of inducing sinus or mastoid carcinomas. Paper 4 points out that the cells at risk for induction of bone tumors may be at some distance from bone surfaces and suggests that the entire range of alpha particles from bone-deposited radionuclides should be included in the calculation of dose, instead of the commonly used 0 to 10 microns from bone surfaces. The growth of osteosarcoma cells in culture was found to be less inhibited by alpha-particle irradiation than was the growth of normal cells (paper 5).

Paper 12 is an abstract of a report that higher than expected rates of breast cancer have been found among women radium dial painters. The differences were correlated with radium intake, but it is not yet established whether the pertinent dosimetry involves internally-deposited radium, absorbed radon, or external radiation in the work rooms. Health findings and radioactivity measurements in our study of another occupational group, former thorium workers, are summarized in paper 14.

Radioactivity studies of individuals or small groups of persons exposed to various radionuclides are reported in papers 17, 18, and 20-25. Among these it may be noted that excess fallout radioactivity was not detected in former military personnel who participated in the "Smoky" nuclear test of 1957 (paper 22). Higher than normal amounts of ^{210}Pb in urine were found for residents of houses with high levels of radon (paper 17) and for people who work at the site of a former uranium mill (paper 18). A model to estimate exposure to radon and radon daughters from ^{210}Pb excretion rates is being developed (paper 17).

Among the papers in this Annual Report are a description of our system for coding medical information from case records (paper 13) and a massive compilation of osteometric data (paper 33).

Foreword

Abstract	vi
1. Dosimetry of Paranasal Sinus and Mastoid Epithelia in Radium-Exposed Humans ROBERT A. SCHLENKER	1
2. Measurements of the Mucous Membranes in Human Paranasal Sinuses and Mastoids M. J. HARRIS	22
3. Morphometry of the Paranasal Sinuses and Mastoid Air Cells M. J. HARRIS	28
4. Radiation Dose to the Cells at Risk for the Induction of Bone Tumors by Bone-Seeking Radioisotopes E. L. LLOYD	29
5. Survival of Human Osteosarcoma Cells and Normal Human Fibroblasts Following Alpha Particle Irradiation E. L. LLOYD AND M. A. GEMMELL	33
6. Inhibition of Growth of Human Osteosarcoma Cells in Culture by Normal Human Fibroblasts E. L. LLOYD, M. A. GEMMELL, AND C. B. HENNING	41
7. Further Studies on the Suppression of Growth of Two Osteosarcoma Cell Lines by Normal Cells Using Alkaline Phosphatase M. A. GEMMELL, C. B. HENNING, AND E. L. LLOYD	42
8. Mitogenic Stimulation of Peripheral Lymphocytes from Radium Workers C. S. SERIO, C. B. HENNING, AND E. L. LLOYD	52
9. Mitogenic Responses of Normal Human Lymphocytes Incubated with Sera from Radium Workers C. S. SERIO, C. B. HENNING, R. E. TOOHEY, AND E. L. LLOYD	53
10. On the Concept of Dose, Dose-Rate, and Specific Energy JOHN H. MARSHALL	61
11. An Empirical Bayes Model for Estimating Radium Intake from Exposure Duration L. SANATHANAN AND H. F. LUCAS, JR.	63

12.	Breast Cancer in Female Radium Dial Workers First Employed Before 1930 E. E. ADAMS AND A. M. BRUES	63
13.	Coding, Storage, and Retrieval of Medical Information Experience with the Standard Nomenclature of Pathology (SNOP) A. M. BRUES	64
14.	Health Status and Body Radioactivity of Former Thorium Workers A. F. STEHNEY, A. P. POLEDNAK, J. RUNDO, A. M. BRUES, H. F. LUCAS, JR., B. C. PATTEN, AND R. E. ROWLAND	69
15.	Radioactivity in Former Workers at a Thorium Refinery J. RUNDO, D. R. BREWSTER, M. A. ESSLING, AND J. Y. SHA	72
16.	Some Determinations at Argonne National Laboratory of Radon in Houses J. RUNDO, F. MARKUN, AND N. J. PLONDKE	73
17.	Estimation of Exposure to ^{222}Rn from the Excretion Rates of ^{210}Pb R. B. HOLTZMAN AND J. RUNDO	74
18.	Excretion of ^{210}Pb and ^{210}Po by Workers in an Area with High Levels of Atmospheric Radon, and Estimates of Exposure to Short-Lived Radon Daughters R. B. HOLTZMAN, P. W. URNEZIS, AND J. RUNDO	89
19.	An Improved Analytical Procedure for the Determination of ^{210}Pb and ^{210}Po Using Alpha-Spectrometric Isotope Dilution P. W. URNEZIS AND R. B. HOLTZMAN	97
20.	Uranium Concentrations in Human Bone ROBERT A. SCHLENKER AND BILLIE G. OLTMAN	106
21.	Plutonium Microdistribution in Human Bone ROBERT A. SCHLENKER AND BILLIE G. OLTMAN	108
22.	Measurements of Radioactivity in Former Military Personnel Exposed to Weapon Debris R. E. TOOHEY, J. RUNDO, M. A. ESSLING, J. Y. SHA, R. D. OLDHAM, J. SEDLET, AND J. J. ROBINSON	109
23.	The Distribution of ^{241}Am in the Human Body as Determined by External Counting R. E. TOOHEY	119
24.	The Late Excretion of Plutonium Following Acquisition of Known Amounts J. RUNDO	120

25.	Macrodistribution of Plutonium in the Human Skeleton R. P. LARSEN, R. D. OLDHAM, AND R. E. TOOHEY	121
26.	On the Gastrointestinal Absorption of Plutonium R. P. LARSEN, R. D. OLDHAM, M. H. BHATTACHARYYA, E. S. MORETTI, AND D. J. AUSTIN	122
27.	Plutonium Retention in Mice after Gastrointestinal Absorption R. P. LARSEN, R. D. OLDHAM, M. H. BHATTACHARYYA, E. S. MORETTI, AND D. J. AUSTIN	123
28.	Continued Studies of the Gastrointestinal Absorption of Plutonium by Rodents R. P. LARSEN, R. D. OLDHAM, M. H. BHATTACHARYYA, AND E. S. MORETTI	124
29.	The Gastrointestinal Absorption of Plutonium in the Dog R. E. TOOHEY, M. H. BHATTACHARYYA, R. D. OLDHAM, R. P. LARSEN, AND E. S. MORETTI	129
30.	Energy Dependence of the Effective Soft Tissue Thickness R. E. TOOHEY	135
31.	Status and Trends in the External Counting of Inhaled Heavy Elements Deposited In Vivo K. L. SWINTH, W. J. BAIR, P. N. DEAN, J. RUNDO, AND F. K. TOMLINSON	138
32.	Identification Through Osteometric Data of Three Radium-Burdened Skeletons J. E. FARNHAM AND J. W. FORKAL	139
33.	Osteometry of the Skeletons of 101 Human Radium Cases J. E. FARNHAM AND J. W. FORKAL	149
	Appendix A. Exposure Data for Radium Patients	184
	Appendix B. Radium-Induced Malignancies	252
	Publications	258

ABSTRACT

Inquiry into the mechanisms and dosimetry for induction of malignancies by radium has continued. Evidence is presented that ^{226}Ra may have significance comparable to that of ^{223}Ra for the induction of carcinomas. Study of the radium dose to cells from bone-seeking radioisotopes suggests that the 0-10 μm range of alpha particles commonly considered in calculating the dose from bone-deposited radionuclides may be inadequate. Alpha-particle radiation inhibited the growth of osteosarcoma cells in culture less than that of normal cells. Additional studies of individuals exposed to radium and thorium, as well as to other radionuclides, are also reported; and additional exposure data have been collected for the 2223 radium cases now being investigated by the Center for Human Radiobiology.

DOSIMETRY OF PARANASAL SINUS AND MASTOID EPITHELIA IN RADIUM-EXPOSED HUMANS*

Robert A. Schlenker

Dose calculations for ^{228}Ra and ^{226}Ra are presented for the sinus and mastoid epithelia and lead to the conclusion that the isotopes are of comparable dosimetric significance for the production of carcinomas in patients exposed to comparable levels.

Introduction

Carcinomas arise in the sinus and mastoid epithelia of persons exposed to ^{226}Ra and ^{228}Ra . They have appeared sporadically over the last 40 years and have been discussed by various authors. Their importance lies in the fact that they have occurred in humans following internal exposure and, therefore, might be induced by other radioisotopes. It is hoped that by careful study of epithelial cell dosimetry, dose-response relationships developed for radium can be more widely applied.

To begin with, some background information should be considered. Table 1 gives the frequency of occurrence of the two types of cancer known to be induced by radium.¹ The data refer to 2164 persons whose body burdens have been measured by the Center for Human Radiobiology or its predecessors. As can be seen, the carcinomas far exceed the expected number and are about half as abundant as the bone sarcomas. There seems to be no doubt among scientists that the latter would be induced by other bone seekers at comparable exposure levels. The evidence from ^{224}Ra -exposed humans and from animal experiments seems overwhelming.

There is, however, no consensus about the risk of sinus and mastoid carcinomas. This is because of what might be called "the radon hypothesis." Both the ^{226}Ra and ^{228}Ra decay series include isotopes of the noble gas, radon. In

* Edited version of an invited paper with the same title presented June 3, 1980 at the 23th Annual Scientific Meeting of the Radiation Research Society, New Orleans.

Table 1. Radium-induced cancer among 2164 measured cases.

Type	Observed	Expected
Sinus, mastoid carcinomas	28	~0.8
Bone sarcomas	60	~2

the case of ^{226}Ra , most of the radon produced is not retained in the body but is excreted through the lungs. In the late 1930's, shortly after the first sinus carcinoma appeared, Martland² proposed that such cancers might be caused by radon gas entering the sinuses from the exhaled breath. Evans³ later proposed that radon could accumulate in poorly ventilated sinuses and in the mastoid air cells and act in concert with alpha particles from bone to produce these tumors. He observed that the tumor yield at high ^{228}Ra levels was low. Recently, Rowland, Stehney, and Lucas⁴ made the same observation and assumed in their dose-effect analysis of the radium data, that ^{228}Ra plays no role at all.

From this history arises the familiar conclusion that ^{226}Ra in combination with radon gas is the sole cause of these tumors. According to this point of view, the risk of sinus and mastoid carcinomas from any other bone seeker would be quite small if the body burdens were comparable to those in the radium cases. Is this really true?

Incidence Data

It should be possible to gain clues from tumor incidence data. Table 2 presents some facts about a well-defined subpopulation of radium cases, radium dial painters first exposed before 1930. Some were exposed only to ^{226}Ra and some were exposed to ^{226}Ra and ^{228}Ra in combination. Of principal interest are the numbers of carcinomas and the numbers of subjects in the high risk group; this latter comprises persons whose ^{226}Ra exposure exceeded the least intake observed to produce a tumor. These data establish one fact with certainty: that

Table 2. Carcinomas in various exposure categories for radium dial workers first exposed before 1930.

Exposure	At risk	High Risk	Observed	Expected
^{226}Ra	552	61	6	~ 0.2
$^{226}, ^{228}\text{Ra}$	247	53	11	~ 0.09
High ^{228}Ra ^a	62	10	0	~ 0.02

^aThis group is a subset of the $^{226}, ^{228}\text{Ra}$ group.

^{226}Ra alone can produce tumors well in excess of expected numbers. In addition, they suggest that when ^{226}Ra and ^{228}Ra are in combination, people in the high risk group are more likely to get a tumor than when ^{226}Ra alone is present. Before accepting this, one would have to carry out a thorough dose-response analysis of the data. While this has not been done, we have found that the dose-effect data for the combined exposure group can be fit well with equations that assume the tumors to be produced by the action of ^{226}Ra alone or by the action of ^{228}Ra alone. In light of this, what seems confounding is that no carcinomas are observed among subjects whose intake of ^{228}Ra was high compared with their ^{226}Ra intake ($\geq 5:1$). Thus, the radium data establish the importance of ^{226}Ra in tumor production but present a picture for ^{228}Ra which is difficult to interpret, yet to conclude from these data that ^{228}Ra is unimportant would be unjustified.

The absence of sinus and mastoid carcinomas among persons injected with ^{224}Ra for therapeutic purposes⁵ is sometimes offered as evidence in support of the radon hypothesis. However, this evidence is unconvincing because, as can be seen in Figure 1, the absence of tumors may simply be a reflection of the rather short period of followup compared with tumor appearance time among ^{226}Ra and ^{228}Ra cases.

FIG. 1.--Distribution of follow-up times for ^{224}Ra cases compared with distribution of appearance times for sinus and mastoid carcinomas in $^{226},^{228}\text{Ra}$ cases.

There are no other human populations which offer relevant incidence data. Among studies of animals exposed to radionuclides, the beagle dog project at the University of Utah is the most relevant. Carcinomas have been observed in the frontal sinus and tympanic bulla,^{6,7} an area comparable to the mastoid region in humans. Compared with bone cancer, the carcinoma yield has been small, but it is significantly higher than expected. Data are presented in Table 3. The control population at Utah and the combined control populations for the Utah and Davis projects are insufficiently large to establish the statistical significance of the observed carcinomas. The expected values are, therefore, based on epidemiological studies of tumor incidence among pet dogs.⁸⁻¹²

The data demonstrate that, in the beagle at least, isotopes other than ^{226}Ra are effective carcinogens. Since the incidence for ^{226}Ra is not greater than the incidence for other isotopes, it appears that radon gas in the air spaces was not

Table 3. Carcinomas of the frontal sinus and the tympanic bulla among Utah beagles.

Isotope	At risk	Observed	Expected
^{226}Ra	107	1	0.01
$^{224,228}\text{Ra}$	94	1	0.01
Actinides	558	3	0.04
All	789	5	0.06
Utah controls	145	0	0.02
Utah & Davis controls	343	0	--

a major additional carcinogenic factor.

It is clear that the incidence data support conflicting hypotheses about the importance of ^{226}Ra . It should be possible to resolve this situation by study of the target cell dose. The rest of the paper will be devoted to this with the primary objective of showing that at least one isotope besides ^{226}Ra , i.e., ^{228}Ra , is capable of producing doses in the carcinogenic range.

Dosimetry

A microradiograph of a section from the frontal sinus is shown in Figure 2; it contains a large central air cavity with cancellous bone surrounding it. The epithelial cells lie in the mucous membrane which, in life, would line the walls of this cavity and separate the airspace and bone. With ^{226}Ra present in the body, radon gas would flow into the airspace and bombard the mucous membrane from one side, while ^{226}Ra and its daughters deposited in bone would bombard it from the other side. Although the possibility is generally discounted, the same picture holds when ^{228}Ra is in the body, as will be shown. Only, in that case, the gaseous daughter product is ^{220}Rn rather than ^{222}Rn . This point is emphasized because it has been thought that significant amounts of ^{220}Rn could not accumulate in the sinuses and mastoid air cells because of the short radioactive half-life.

FIG. 2.--Cross-sectional view of frontal sinus showing large air-space (A) surrounded by a region of cancellous bone.

ANL Neg. 149-78-402

The airspace radon flows in from the surrounding bone, but it also may flow out before it can decay. The outflow is by two routes; the mucosal blood flow and by the ostium, the ventilatory duct which connects every sinus with the nasal cavity. Thus, the radon level in the airspace is a balance between the rate of inflow and the rate of outflow.

The actual target cell dose is determined by anatomical variables and by the levels of radioactivity in the bone and airspace.

Anatomical Variables

There are three such variables, two relating to the structure of the mucous membrane and one relating to the size of the airspace.

Lamina Propria. Figure 3 shows the mucous membrane, the bone and airspace. The target cells lie in the epithelial layer. In order for alpha particles to reach this layer, they must pass through the connective tissue portion of the mucosa, the so-called lamina propria. Since alpha particles have a range which is comparable to the thickness of the lamina propria, the latter serves as a shield which protects the epithelium from the alphas from bone.

Table 4 gives data on the thickness of the lamina propria. The values shown have not been corrected for tissue shrinkage nor for obliquity of the sectioning plane. It is thought that these two corrections approximately cancel one another. The data were collected from normal subjects. Keeping in mind that the maximum

FIG. 3.--A typical region of sinus mucosa showing the epithelial layer (E) and the lamina propria (L) positioned between the bone (B) and airspace (A). (ANL Neg. 149-78-404)

Table 4. Lamina propria thickness.

Number of cases	Site	Range, μm	Fraction $<75 \mu\text{m}$
2	Maxillary	29 - 541	--
3	Frontal	14 - 217	<0.25
5	Ethmoid	45 - 350	<0.25
5	Sphenoid	55 - 410	<0.25
9	Mastoid	5 - 350	>0.75

alpha particle range is about $66 \mu\text{m}$ for the ^{226}Ra decay series and about $80 \mu\text{m}$ for the ^{228}Pa series, one can see that the lamina propria can be either thinner or thicker than the maximum alpha range. A better picture of the dimensions is provided by the right-most column of Table 4 which states the fraction of the lamina propria which is less than $75 \mu\text{m}$ thick. From this statistic, it is obvious that the sinuses, as a group, have a much thicker lamina propria than the mastoid. This means that, for equal specific activities, the dose from bone in the mastoids will exceed that in the sinuses.

Figure 4 presents the dose-rate from radium in bone as a function of lamina propria thickness, in the sinuses and mastoids. Two conclusions can be drawn

FIG. 4.--Dose rate to the sinus and mastoid epithelia per unit of ^{226}Ra and ^{228}Ra activity in bone. The solid portion of each curve shows the range within which the lamina propria thickness is most likely to fall.

immediately: (1) The dose rate in the mastoid exceeds that in the sinuses, quite likely by a large amount, and (2) the dose rate from ^{228}Ra exceeds that from ^{226}Ra per unit of specific activity. This is our first piece of evidence that, in the case of combined exposure, ^{228}Ra can be as important a source of dose as ^{226}Ra .

Epithelial Cytoplasm. The second variable related to the mucous membrane structure is illustrated by the contrast between Figures 5 and 6. In Figure 5, a section is shown of mucous membrane typical of the mastoid air cells: Substantial lamina propria covered by a thin epithelial layer, usually only one cell thick. Contrasted with this is the sinus mucosa seen in Figure 6, which again has a substantial lamina propria, but a much thicker epithelial layer. Notice the structure of this layer: Cell nuclei concentrated toward the bottom with the cytoplasm from elongated cells extending to the surface of the epithelium. The cytoplasm shields the cell nuclei, which we believe to be the true targets for malignant transformation, from alpha particles entering from the airspace.

Thickness data, presented in Table 5, show that (1) the sinus epithelium

FIG. 5.--Section of mucous membrane typical of the mastoid showing an epithelial membrane one cell thick.

FIG. 6.--Section of mucous membrane typical of sinuses showing epithelial layer in which cell nuclei cluster toward the bottom with a substantial layer of cytoplasm extending above them. A, airspace; E, epithelial layer; L, lamina propria; B, bone.
(ANL Neg. 149-78-406)

Table 5. Epithelial thickness in micrometers.

	Sinus	Mastoid
Total		
Range	11 - 136	3 - 40
Typical	40 - 90	5 - 10
Over nuclei		
Range	12 - 84	0 - ~5
Typical	35	<2

is generally thicker than the mastoid epithelium, (2) the sinus nuclei lie below a thickness of cytoplasm which can exceed the range of the most energetic alpha particles in either the ^{226}Ra or the ^{228}Ra decay series, and (3) the layer of cytoplasm in the sinus far exceeds the thickness of the layer in the mastoid. Since the layer in the mastoid is so thin, it will be neglected.

The impact on the dosimetry is shown in Figure 7, which applies to the sinuses only. The upper solid and dashed lines refer to ^{220}Rn and the lower ones refer to ^{222}Rn . The solid lines indicate a constant target layer thickness, and the dashed lines indicate it was varied with the thickness of the cytoplasm layer. We see that the layer of cytoplasm has a major impact on the dosimetry. At the typical thickness of 55 μm , the dose rate is reduced by a factor of about 5 for ^{220}Rn and about 10 for ^{222}Rn . Thus, a unit concentration of radon in a sinus airspace may be an order of magnitude less effective than in the mastoid air cells. Furthermore, per unit concentration, ^{220}Rn delivers more dose than ^{222}Rn . This is the second clue we have that the ^{228}Ra decay series may be an important source of epithelial dose.

Airspace Size. The third of the anatomical variables is airspace size. The most dramatic differences in size occur between the sinuses and mastoid air cells, and within the air cell system itself. To illustrate, the distance across the airspace in the frontal sinus of Figure 2 is 1 to 1.5 cm, but the distance across the typical air cell in this mastoid section of Figure 8 is 0.2 cm, although the sizes range from less than 0.1 cm to more than 1 cm. The effect of size on dose rate per unit of ^{222}Rn concentration is seen in Figure 9. First notice that two types of behavior occur, an increase in dose rate with size for the mastoid and ethmoid and a relative constancy for the other sinuses. Note that the mastoid receives a much higher dose rate than any of the sinuses. Among the sinuses, the typical ethmoid receives the highest dose rate, but this is strongly size-dependent. The typical frontal and sphenoid sinuses receive the same dose rate, which is a little higher than that received by the typical maxillary.

In summary, the mastoid receives a greater dose from bone and a greater dose from the airspace than do the sinuses; the bone dose from the ^{228}Ra decay series exceeds that from the ^{226}Ra series in both the sinuses and mastoids; the

FIG. 7.--Dose rate to the epithelium per unit of radon concentration as a function of the thickness of cytoplasm layer. The upper solid and dashed lines represent ^{220}Rn , and the lower ones refer to ^{222}Rn .

FIG. 8.--Cross section of mastoid showing an extensive set of interconnecting air cells typified by the areas labeled "a" and bordered by a narrow region of cancellous bone. (ANL Neg. 149-78-403)

dose from ^{220}Rn exceeds that from ^{222}Rn in the sinuses but is the same in the mastoids.

Radioactivity

The next factor that must be considered is the levels of radioactivity which produce the dose.

Airspace Radon. As I mentioned earlier, the radon level in the airspace is a balance between the influx from bone and the efflux through the ventilatory duct and the circulatory system. Studies of sinus and mastoid function by introduction of ^{133}Xe into the airspaces have revealed that clearance follows a single exponential curve, the half-time of which is determined by the status of ventilation or circulation. In general, clearance through the ventilatory duct is much more rapid than clearance by the circulation. Thus, the half-time is relatively short if the duct is open and quite a bit longer if it is obstructed as is

FIG. 9.--Dose rate per unit of ^{222}Rn concentration for the mastoid, the ethmoid and the other sinuses. The typical diameters of the various airspaces are indicated as solid points; f/s designates the frontal and sphenoid which have the same typical sizes.

common in sinus inflammation. Table 6 contains data on the clearance half-times. The circulation half-times are all based on ^{133}Xe clearance experiments,¹³⁻¹⁶ and have been corrected for differences between the tissue solubilities of Xe and Rn. The ventilation half-times are based on Xe clearance,^{13,14} O_2 exchange,^{17,18} and on the frequency of eustachian tube opening.¹⁹ The numbers in parentheses show the number of patients in which the values shown were based. For the frontal and maxillary sinuses, the half-times vary considerably among subjects, but as a rule, clearance is about 10 times more rapid when the ventilatory duct is open than when it is closed. In contrast, ventilation of the healthy mastoid is very slow because the eustachian tube is closed except when swallowing. Thus, a ventilation half-time is only about 1% of the circulatory half-time. For comparison, the radioactive half-times of ^{220}Rn and ^{222}Rn are shown. It is immediately clear that ^{220}Rn which flows into an airspace will, for the most part, decay there before it can be cleared either by the ventilation or circulation. On the other hand, ^{222}Rn will, for the most part, be cleared either by the circulation or the ventilation before it can decay.

Table 6. Half-times for radon removal in minutes.

Site	Circulation ^a	Ventilation ^a
Frontal sinus	>54 (5)	0.9-6.8 (10)
Maxillary sinus	24-117 (5)	~6 (O ₂ model)
Mastoid air cells	24-105 (9)	~8000 (est.)

$$^{220}\text{Rn}: T_{\frac{1}{2}} = 0.9 \text{ min}$$

$$^{222}\text{Rn}: T_{\frac{1}{2}} = 5500 \text{ min}$$

^a () Number of patients.

This fact is expressed quantitatively in Figure 10 where the ratio of Rn activity in the airspace to the radium activity supporting it is plotted as a function of total clearance half-time. This shows that ^{220}Rn buildup is rather independent of the clearance half-time, whereas ^{222}Rn buildup is quite sensitive to it. In the region of the curve marked "ventilation," clearance is by both ventilation and circulation, but ventilation is dominant; in the region marked "circulation" the ventilatory route is blocked, and clearance is by circulation only. The uppermost curve for ^{220}Rn is based on the assumption the 100% of the unretained radon produced escapes into the airspace. Because of the isotope's short half-life, this is unlikely, so a second curve, based on breath ^{220}Rn measurements in humans, is shown in which it is assumed that 0.3% escapes. Now compare the ^{220}Rn and ^{222}Rn curves in the solid regions of the curves. First, consider the ventilation region. We see that the ^{220}Rn activity exceeds the ^{222}Rn activity, no matter which of the ^{220}Rn curves is considered. In the circulatory region, the upper ^{220}Rn curve exceeds the ^{222}Rn curve, and the lower ^{220}Rn curve is less. Apparently the ^{220}Rn level is comparable to, or will exceed, the ^{222}Rn level. These curves show that, despite the short radioactive half-life of ^{220}Rn , its level in the sinuses or mastoids can exceed the ^{222}Rn level in cases of combined ^{228}Ra and ^{226}Ra exposure. This is another indication of the importance of ^{228}Ra .

FIG. 10.--The effect of radon clearance from airspaces on radon activity. The solid portions of the curves correspond to the ranges of observed clearance half-times (see Table 6).

While Figure 10 describes the dependence of radon accumulation on clearance half-time, it does not tell how much radon actually accumulates in the airspace. An accumulation model is quite difficult to formulate with certainty because the controlling variables are not all known. The broad features of such a model are, however, clear. First, the radon which accumulates must come from a thin layer of surrounding bone. The section of mastoid in Figure 8 will help to make this clear. The thin bony septa separating the air cells contain no internal blood supply and depend totally on the blood flowing through the surface mucosa for nourishment. Likewise, clearance of the unretained radon produced in these septa can only be via the ventilation or via the mucosal circulation. Thus, the radon from these bony septa will partition between the airspace, bone, and mucosal lining, probably in proportion to the solubility in each of these regions. In order to reach the airspace, radon from the cancellous bone must cross the marrow spaces, which are well vascularized. During its passage, there is a good chance that a radon atom would be entrapped by the circulation and carried off. Thus, bone which is separated from the airspace by a vascularized region will be a less important source of radon than bone which is adjacent to the airspace. The positions of the vascular spaces lead to the conclusion that bone within a

few hundred micrometers of the airspace surface is the most important source of airspace radon. Exactly how much bone, however, is unknown.

The second major variable is radon solubility. There are no data for bone, so one can only speculate. Solubility within the bone crystals is unimportant since the diffusion times for radon through the mineral are orders of magnitude greater than the radon half-life. However, the bone crystals are very small and provide an enormous surface area which might absorb radon the same way that activated charcoal does. Thus, the solubility of radon in bone could be much higher than in tissue. On the other hand, if this were not an important mechanism for radon retention, then dissolution of radon in the fluids of bone could be the principal determinant of radon solubility and the total solubility in bone could be less than in soft tissue. A third important variable, for ^{220}Rn , is diffusion time from the site of production to the airspace. This is, however, not an important factor for ^{222}Rn . In this case, the simplest model is one which assumes rapid mixing between the bone and airspace. The predictions of such a model are shown in Figure 11 as a function of the ratio of bone solubility to tissue solubility. For comparison, we have just one in vivo measurement of the sinus radon concentration which was made in the frontal sinus. It compared well with the model predictions for a ventilated sinus.

FIG. 11.--Concentration of ^{222}Rn per unit of ^{226}Ra specific activity in bone surrounding the mastoid, the unventilated sinus (uv) and the ventilated sinus (v). Bone layers of 0.05 cm thickness (solid lines) and 0.02 cm thickness (dashed lines) are envisioned as the source of ^{222}Rn gas. The solid point in the lower right shows the one measured value of radon concentration made in vivo. The solubility corresponding to this data point is unknown, and its placement on the right-hand side of the graph is for convenience only.

^{220}Rn results are not shown on this plot, principally because a different model is used to predict the ^{220}Rn concentration. This is necessary because, as mentioned earlier, the diffusion time is such an important factor in ^{220}Rn accumulation. It is likely that diffusion times are slow enough compared with the ^{220}Rn half-life, that the assumption of rapid mixing, on which the model shown is based, would be quite unjustified. To avoid the difficulties presented by this, the assumption is made, in the ^{220}Rn model, that 0.3% of the unretained fraction escapes from the bone which supplies ^{220}Rn to the airspace. This gives mastoid concentrations similar to those for ^{222}Rn and sinus concentrations in both the ventilated and unventilated cases, which are comparable to the unventilated levels for ^{222}Rn .

In summary, ^{220}Rn decays before it can escape from the airspace, whereas ^{222}Rn escapes before it can decay. This simple difference accounts for the fact that ^{220}Rn , despite its short half-life, can contribute substantially to the dose from the airspace in mixed exposures of ^{226}Ra and ^{228}Ra . The radon concentration in the mastoid exceeds, by a large amount, that in the sinuses, and this may help explain the tendency of these cancers to appear in the mastoid.

Radium in Bone. Our early autoradiographic studies, all of which were qualitative, revealed a common pattern of uptake in the mastoid air cells. A typical example is shown in Figure 12. Notice that the intensity is low within the air cell region compared with areas of bone cortex distant from the air cells. This is probably the result of the fact that the mastoid air cells are fully formed by age 15 and only a very few of our patients were exposed younger than this. This observation led to the supposition that the specific activity adjacent to the air cell surfaces was lower than the average skeletal specific activity and, therefore, quite possibly lower than the specific activity adjacent to the sinus surfaces. This would affect the comparative dosimetry of the mastoids and sinuses.

We have begun to collect quantitative autoradiographic information. We sample, as randomly as possible, areas adjacent to and distant from the airspace surfaces. Results on five patients are shown in Table 7. The specific activity of the bone adjacent to the air cell generally lies between the diffuse and hotspot

FIG. 12.--Autoradiographs of bone sections showing less radioactivity immediately adjacent to the air cells than in bone somewhat removed from the air cell boundaries.
(ANL Neg. 149-78-345)

Table 7. ^{226}Ra specific activity in mastoid bone, picocuries/gram.

Case	Diffuse	Air cells	Average hotspot	<u>Air cells</u> <u>Uniform</u>
00-006	390	610	900	0.92
01-014	46	140	550	0.30
01-046	120	180	390	1.0
01-145	200	180	5200	0.14
03-240	190	250	2100	0.21

levels observed in the more distant bone. In addition, the air cell specific activity is generally less than the average skeletal specific activity or uniform specific activity. In any case, it does not exceed it. What is interesting is that it can be several times less than the uniform specific activity and this may signal a major difference between the dosimetry of sinuses and mastoids.

From the radon accumulation model and the specific activities, the mastoid dose rates can be computed for these 5 patients. In computing the dose rate from airspace radon, I have used the conservative assumption that bone solubility is ten times higher than tissue solubility. This produces lower dose rates

than a smaller solubility ratio would. Table 8 shows that the airspace radon is the more important source of dose, a conclusion which is relatively independent of the model assumptions. The time-integrated dose from ^{226}Ra under these assumptions would be several thousand rads for these cases.

Dose Rates

At the beginning, I said that ^{228}Ra has been assumed to be unimportant to the dosimetry of sinus and mastoid carcinomas, and I have pointed out reasons why this may not be so. From all of the data presented so far, it is now possible to predict, with some certainty, the dose rate which would be produced by 1 μCi of ^{226}Ra or ^{228}Ra in the skeleton so that the two can be compared. The comparisons are presented in Table 9 for the sinuses and for the mastoids. Notice first that the bone dose rate from ^{228}Ra exceeds the bone dose rate from ^{226}Ra in all cases. Secondly, the dose rate from ^{228}Ra in the ventilated sinuses exceeds that from ^{226}Ra . Finally, observe that the airspace dose rate from ^{228}Ra is comparable to the airspace dose rate from ^{226}Ra in the unventilated sinuses and in the mastoid air cells. Now these are dose rates for a 1 μCi body burden. In many cases of mixed ^{226}Ra and ^{228}Ra exposure, the intakes were comparable or the ^{228}Ra intake was greater. Therefore, the values in this table

Table 8. Dose rate to mastoid epithelium at time of death, rad/year.

Case	Body burden, $\mu\text{Ci } ^{226}\text{Ra}$	Bone	Airspace	Total
00-006	2.61	1.5	6.2	7.7
01-014	2.24	0.34	1.4	1.7
01-046	0.55	0.44	1.8	2.2
01-145	6.33	0.44	1.8	2.2
03-240	4.32	0.01	2.6	3.2
Dose rate ratio (Bone/airspace) = 0.24				

Table 9. Dose rates to sinus and mastoid epithelia assuming a one microcurie body burden of ^{226}Ra or ^{228}Ra , rad/year.

Cavity	Isotope	Bone	Airspace, ventilated	Airspace, unventilated
Maxillary	226	0.0046	0.0092	0.21
	228	0.075	0.12	0.15
Ethmoid	226	0.0046	0.011	0.26
	228	0.075	0.12	0.28
Frontal, sphenoid	226	0.0046	0.0095	0.22
	228	0.075	0.16	0.24
Mastoid	226	0.58	--	3.0
	228	4.4	--	2.0

lead to the conclusion that ^{228}Ra is as important a source of dose as ^{226}Ra in such cases of mixed exposure. Furthermore, the absence of tumors, when the ^{228}Ra input was much greater than the ^{226}Ra input, was not due to the fact that ^{228}Ra is dosimetrically insignificant. Indeed, in those cases, ^{228}Ra would have delivered a dose in the carcinogenic range, and we are left to puzzle over the absence of tumors among this group.

Conclusions

Would radioisotopes other than ^{226}Ra produce such tumors at comparable exposure levels? The dose calculations indicate that pure ^{228}Ra would, since errors in the assumptions would affect the ^{226}Ra and ^{228}Ra dose values to about the same extent, and the conclusions about the relative dosimetric importance of the two isotopes would remain unchanged. It is also likely that ^{224}Ra would be carcinogenic at the levels used with humans; the actinides with higher alpha particle energies may well produce a non-negligible risk in the mastoid region where the lamina propria is thin enough for particles to reach the epithelium. Because of the present results, one cannot ignore carcinomas of the sinuses and mastoids as a potential risk from alpha emitting bone seekers.

References

1. Radiological and Environmental Research Division Annual Report, Center for Human Radiobiology, June 1978-June 1979, ANL-79-65 Part II, Appendices A and B.
2. H. S. Martland, Occupational tumors, bones, Encyclopedia of Health and Hygiene, International Labor Organization, Geneva, pp. 1-23 (1939).
3. R. D. Evans, The effect of skeletally deposited alpha-ray emitters in man, Br. J. Radiol. 39, 881-895 (1966).
4. R. E. Rowland, A. F. Stehney, and H. F. Lucas, Jr., Dose-response relationships for female radium dial workers, Radiat. Res. 76, 368-383 (1978).
5. C. W. Mays, H. Spiess, and A. Gerspach, Skeletal effects following ^{224}Ra injections into humans, Health Phys. 35, 83-90 (1978).
6. G. N. Taylor, T. F. Dougherty, L. Shabestari, and J. H. Dougherty, Soft-tissue tumors in internally irradiated beagles, in Delayed Effects of Bone-Seeking Radionuclides, University of Utah Press, Salt Lake City, pp. 323-336 (1969).
7. Injection Tables, Research in Radiobiology, Annual Report of Work in Progress in the Internal Irradiation Program, Radiobiology Laboratory, University of Utah College of Medicine, Report No. COO-119-253, pp. 9-157 (1978).
8. George W. Robinson, A comparison of licensed and hospital dog populations, J. Am. Vet. Med. Assoc. 152, 1383-1389 (1968).
9. C. R. Dorn, D. O. N. Taylor, R. Schneider, H. H. Hibard, and M. R. Klauber, Survey of animal neoplasm in Alameda and Contra Costa Counties, California. II. Cancer morbidity in dogs and cats from Alameda County, J. Nat. Cancer Inst. 10, 307-318 (1968).
10. B. R. Madewell, W. A. Priester, E. L. Gillette, and S. P. Snyder, Neoplasms of the nasal passages and paranasal sinuses in domesticated animals as reported by 13 veterinary colleges, Am. J. Vet. Res. 37, 851-856 (1976).
11. Robert S. Brodey, Canine and feline neoplasia, Adv. Vet. Sci. Comp. Med. 14, 309-354 (1970).
12. William A. Priester and Nathan Mantel, Occurrence of tumors in domestic animals. Data from 12 United States and Canadian Colleges of Veterinary Medicine, J. Nat. Cancer Inst. 47, 1333-1344 (1971).
13. R. Zippel and B. Streckenbach, ^{133}Xe washout in the paranasal sinuses — a diagnostic tool for assessing ostial function, Rhinology XVII, 25-29 (1979).
14. B. Streckenbach, K. Vogt, V. Pink, and G. Kirsh, Untersuchungen zum quantitativen Gas austausch der menschlichen Starnhöle mit $^{133}\text{Xenon}$, Radiologica Diagnostica 19, 110-111 (1978).
15. R. Aust, L. Backlund, B. Drettner, B. Falck, and B. Jung, Comparative measurements of the mucosal blood flow in the human maxillary sinus by plethysmography and by xenon, Acta Otolaryng. 85, 111-115 (1978).
16. A. Elner and R. Nilsén, Preliminary studies of gas resorption from the middle ear, Acta Otolaryng. 70, 197-202 (1970).
17. K. Aust and B. Drettner, Experimental studies of the gas exchange through the ostium of the maxillary sinus, Uppsala J. Med. Sci. 79, 177-186 (1974).

18. B. Drettner and R. Aust, Pathophysiology of the paranasal sinuses, Acta Otolaryng. 83, 16-19 (1977).
19. K. Filsberg, Ventilatory studies on the eustachian tube, Acta Otolaryng., Supp. 219, 1-82 (1966).

MEASUREMENTS OF THE MUCOUS MEMBRANES IN HUMAN PARANASAL SINUSES AND MASTOIDS*

M. J. Harris

Measurements taken on eight new autopsy cases from the CHR collection expand the data base for normal epithelial and lamina propria thicknesses of paranasal sinuses and mastoid air cells. The ratios of nuclear area to epithelial cellular area calculated in this series are in close agreement with those of earlier reports.

Introduction

Determination of the radiation dose from an internally deposited alpha emitter requires a knowledge of the anatomy of the structures at risk for pathological change. Data collected on specimens of paranasal sinuses and mastoids of the temporal bone from eight new autopsy cases are reported here. These anatomical areas are the sites of carcinoma in chronically exposed radium workers.

Materials and Methods

Materials for this study were obtained from the Department of Pathology, Kansas University Medical Center and are listed in Table 1. Embedding of tissues was by a glycol methacrylate procedure modified from literature methods.^{1,2} Measurements were made of epithelial and of lamina propria thicknesses and of the percentage of epithelium occupied by cell nuclei. The specimens used in this study had no known history of radionuclide exposure and are defined as "normal." Thickness measurements were made using a Zeiss MOP-3 image analyzing system and the percentage of epithelium occupied by cell nuclei was determined on measurements of nuclear and epithelial cross-sectional area taken with this same instrument.

* Content of a talk with the same title given before the 93rd Annual Session of the American Association of Anatomists, Omaha, Nebraska, April 29, 1980.

Table 1. Description of materials taken from autopsy cases of Kansas University Medical Center Department of Pathology

Case number	Description
A217 78	71-year old Negro male, death attributed to vascular atherosclerosis; frontal, ethmoid and sphenoid paranasal sinuses and right and left mastoids
A218 78	12-year old white male, apparent cause of death was overwhelming sepsis post auto accident and amputation; ethmoid and sphenoid paranasal sinuses and right mastoid
A219 78	25-year old white male, laceration of right ventricle caused by a knife; ethmoid paranasal sinus and right and left mastoids
A223 78	27-year old Negro male, gunshot wound to the skull with multiple skull fractures and brain damage; ethmoid and sphenoid paranasal sinuses and right and left mastoids
A224 78	18-year old white female, cause of death complications from a disseminated malignant small cell neoplasm; frontal, ethmoid and sphenoid paranasal sinuses and right and left mastoids
A225 78	63-year old white female, death attributed to cardiovascular failure precipitated by emphysema; frontal and ethmoid paranasal sinuses and right and left mastoids
A226 78	81-year old white female, cause of death liver failure and complications; frontal and ethmoid paranasal sinuses and right and left mastoids
A227 79	16-year old white female, cause of death attributed to intracranial hemorrhage following failure of a cerebellar artery; frontal, ethmoid and sphenoid paranasal sinuses and right and left mastoids

Results

Representative areas of paranasal sinus and mastoid air cell epithelium are shown in Figures 1 and 2, respectively.

Table 2 shows the measurements of the epithelial and lamina propria thicknesses for the cases used in this study. Means are based on a variable number of measurements from one or two sections from a particular case. The number of measurements was never less than 7 and was usually 11 according to our protocol.

FIG. 1.--Frontal sinus, Case A225 78; b = bone; lp = lamina propria, se = sinus epithelium.

FIG. 2.--Left mastoid air cell, Case A227 78; b = bone; e = epithelium; lp = lamina propria.

Table 2. Thickness Data

Location *	Mean and standard deviation of epithelial thickness, μm	Mean and standard deviation of lamina propria thickness, μm
Frontal sinus (5)	78.32 ± 28.84	165.20 ± 47.08
Ethmoid sinus (8)	103.48 ± 37.79	328.96 ± 257.01
Sphenoid sinus (5)	72.10 ± 24.63	155.06 ± 77.12
L. mastoid air cell (7)	20.10 ± 11.53	83.10 ± 34.71
R. mastoid air cell (7)	12.44 ± 4.72	80.51 ± 51.69

* () the number of cases studied.

Table 3 summarizes features of epithelial cells which we believe are important elements for tumor risk analysis of the cranial sinuses. The percentage of epithelium occupied by nuclei in a particular area gives an estimate of the amount of

radiation sensitive material present. The more nuclear material present, the greater the risk that some of it may be induced to form neoplastic tissue.

Usually three measurements of total cell area were made from one or two sections from a particular case. Mean nuclear area was estimated by multiplying the average nuclear area of 10 nuclei by the total number of nuclei in a region studied. A variable number of measurements of nuclear area was made since each studied region had a different number of nuclei.

Table 3. Percentage of Epithelium Occupied by Nuclei

Location *	Total cell area μm^2 (a)	Total nuclear area μm^2 (b)	Ratio b/a $\times 100$
Frontal sinus (5)	18928	4300	22.72
Ethmoid sinus (8)	20390	4762	23.35
Sphenoid sinus (5)	15268	3468	22.69
L. mastoid air cell (7)	4476	1417	31.66
R. mastoid air cell (7)	3787	1513	39.94

* () the number of cases studied.

These values are similar to those reported in earlier work.^{3,4} It is noteworthy that the ratio in the mastoid air cell epithelium in this series is always larger than that in the paranasal sinuses.

Discussion

Tissue thickness affects radiation dose and risk of neoplastic change to the extent that the assumed targets (nuclei) are shielded from the radiation source. In radium exposed persons, alpha particles are the principal inducers of damage. They have a range in soft tissues of up to about 70 μm .

If one considers the source of radiation to be the bone subjacent to the mucous membrane in a sinus or mastoid cavity, he might conclude that epithelial nuclei near the lumen of a paranasal sinus, for example, might not be affected.

It would be more likely that damage could be induced in the epithelium of a mastoid air cell which is thin compared to that of paranasal sinuses and which generally lies closer to the bone, placing it potentially within the range of alpha sources from bone. There is also a likelihood that damage might be induced by the gaseous decay product of radium, radon, which may accumulate in the air spaces and bombard the epithelium of both the paranasal sinuses and the mastoid air cells.

Another aspect of the shielding question which ought to be considered but which has not yet been adequately quantified is the variable layer of cytoplasm and cell products lying above the nuclear layer, particularly in the paranasal sinuses. A cursory examination of the material used for this study indicates a wide range of thickness (from 2 to 100 μm), with a midrange of 40 μm .

The target potential of the epithelial glands was qualitatively examined for these cases. Since they are abundant only in the ethmoid paranasal sinuses or near the ostia of the other sinuses and were not prominent features in the samples taken for study in this series, it seems that there is only a small likelihood that they would receive an appreciable dose of alpha radiation. Their location is usually beyond the range of alpha sources in the subjacent bone, and they are shielded by the overlying epithelium, its superior cytoplasm, and cell products.

Acknowledgements

I want to express my gratitude to Dr. William Hunter, Department of Pathology, Autopsy Service, Kansas University Medical Center, for allowing the use of facilities for collection of specimens. Thanks are also due to Floyd M. Foltz, Ph.D., Department of Anatomy, for facilitating the collection process.

References

1. E. H. Leduc and W. Bernhard, Recent modifications of the glycol methacrylate embedding procedure, *J. Ultrastruct. Res.* 19, 196-199 (1967).
2. H. S. Bennett, A. D. Wyrick, S. W. Lee, and J. H. McNeil, Science and art in preparing tissue embedded in plastic for light microscopy, with special reference to glycol methacrylate, glass knives, and simple stains, *Stain Technol.* 51, 71-97 (1976).

3. M. J. Harris and R. A. Schlenker, Quantitative histology of human paranasal sinus and mastoid air cell epithelia, Radiological and Environmental Research Division Annual Report, July 1977-June 1978, ANL-78-65, Part II, pp. 55-69.
4. M. J. Harris and R. A. Schlenker, Quantitative histology of mastoid air cell epithelium, Radiological and Environmental Research Division Annual Report, July 1978-June 1979, Part II, pp. 70-75.

MORPHOMETRY OF THE PARANASAL SINUSES AND MASTOID AIR CELLS^{*}

M. J. Harris

The mucous membranes of human paranasal sinuses and mastoid air cells of nonpathologic specimens from thirteen autopsy cases were measured in undecalcified, plastic embedded preparations. Quantitative properties of surface and of glandular epithelium and lamina propria, position of nuclei relative to adjacent bone or air space and a sampling of the proportion of nuclear area to epithelial cell area were examined.

These data extend the sparse and incompletely documented quantitative measurements of sinus and air cell epithelia reported in early literature and provide a basis for dosimetric calculations

^{*} Abstract of a paper presented at the XI International Congress of Anatomy, Mexico City, Mexico, August 17-23, 1980.

RADIATION DOSE TO THE CELLS AT RISK FOR THE INDUCTION OF BONE TUMORS BY BONE-SEEKING RADIOISOTOPES

E. L. Lloyd

For bone-seeking radioactive isotopes, such as ^{226}Ra , ^{214}Ra , and ^{239}Pu , it has become common practice to consider a layer of cells 0 to 10 microns from bone mineral as appropriate for calculations of effective carcinogenic radiation doses. From considerations of our measurements of dimensions and positions of cells relative to bone mineral at the endosteal surface of human bone together with our in vitro studies, it would appear that limitation to less than the complete range of the emitted particles is unwarranted for calculation of the dose.

Introduction

We have previously reported¹ on the electron microscope appearance of cells at the endosteal surface of cortical bone from a radium dial painter (05-953) who died of a "well differentiated fibrosarcoma."² We have also studied the appearance of the endosteum and related cells of an age-matched unirradiated control person.³ In both cases the size and position of the cells relative to bone mineral, where radium is deposited, have been documented.^{1,3,4} In the bone from the radium patient, unlike the control, a fibrotic layer of tissue was found covering most of the endosteal surface. The effective carcinogenic dose and its relation to the cells which have the potential of giving rise to bone tumors are discussed here, with particular reference to this fibrotic layer.

Results and Discussion

For the radium patient whose femur was examined, the total average skeletal dose, based on extrapolation from measurements of the amputated leg, was estimated to be 6590 rads.² In attempts to relate these calculated doses to the cells at risk on the endosteal surface, it has been common to calculate doses to a surface layer 0 to 10 μm from the bone mineral.⁵⁻⁷ This value is estimated to be $6590 \times 0.45 = 2965$ rads, using the factor 0.45 derived by Marshall et al.⁷ to convert average skeletal dose to the surface dose within the 0 to 10 μm layer. From our in vitro studies of the survival of cells irradiated with alpha particles similar in energy to those emitted by radium and its daughter products, we

obtained a value for the mean lethal dose (D_0) of 60 rads.⁸ Since cell survival following alpha particle irradiation has been found by ourselves and others⁹ to decrease exponentially with dose, the fraction of cells capable of surviving 2965 rads would be expected to be 3.4×10^{-22} . This leads us to believe that no cells would be expected to survive over the lifetime of the patient within the 0 to 10 μm surface layer of the bone even after accounting for non-uniform distribution of radium.^{10,11} It is likely that the cells which ultimately give rise to the bone tumors are those which are separated from the mineral by fibrotic tissue^{4,12,13} and have invaded the area long after the radium was acquired. The radium retention in bone would then be reduced and the doses to these cells would be significantly less than the dose calculated for a stationary population in the 0 to 10 μm surface layer. The reduction in dose would, therefore, result from (a) the reduced radium retention, (b) the effect of the inverse square law, and (c) the limited cell residence time. This would bring the doses more in line with those shown to be effective in producing malignant transformations in vitro (typically 100 to 300 rads).¹⁴

The particular geometry of this system may go some way toward explaining the shape of the dose-response relationship for the incidence of bone tumors found in the human radium cases. In our in vitro transformation experiments, we found a very steep dose response (proportional to about the cube of the dose) for transformation frequencies when a parallel beam of 5.6 MeV alpha particles was used to irradiate flattened cells ($\sim 2 \mu\text{m}$ thick). Under these circumstances, fourteen alpha particles on average traversed each cell nucleus to give rise to the mean lethal dose (60 rads). (This corresponded to a cross-sectional area for cell killing of $23 \mu\text{m}^2$.) The corresponding number of tracks for cells with an average cross-sectional area of $168 \mu\text{m}^2$ found at the endosteal surface of the femur in the radium patient,⁴ would be 7.3 alpha particles per nucleus. The greater the distance between the cells and the mineral surface, the more the geometry resembles that of a parallel beam of radiation. Transformations of the cells in our in vitro study were only observed at doses somewhat greater (82 rads) than the mean lethal dose. If our in vitro studies are relevant and we assume that such multi-hit events are necessary for the induction of a tumor, this may

explain the steeper dose-response observed for the incidence of bone tumors in the radium patients compared with the more nearly linear dose-response relationship observed for carcinomas of the mastoid and the paranasal sinuses in the same population.¹⁵ Carcinomas arise from epithelial cells which are characteristically more spherical in appearance. Hence, a single track would be expected to traverse a much greater length of nuclear DNA and effect more damage per track in a spherical cell when compared with a flattened cell perpendicular to the direction of the radiation. Harris and Schlenker¹⁶ have recently documented the cells most likely to give rise to these other radium-related tumors.

From our previous studies,^{1,3,4} it would appear that the cells most likely to be at risk for the induction of bone tumors are the flat fibroblastic-appearing cells which are separated from bone mineral by fibrotic tissue. Although the distances between the bone mineral and these flattened cells were found to vary greatly, approximately half of those documented lay outside the 0 to 10 μm thickness commonly used for the calculation of relevant carcinogenic doses. Until more definitive studies have been completed on a larger number of radium patients in trabecular areas where bone tumors predominantly arise, it would seem more appropriate to consider the cells within the complete range of the alpha particle as potentially at risk. Meaningful predictions for the carcinogenic effects of other radioisotopes, such as ^{239}Pu , in man can only be made when both the cells at risk and the relevant doses to those cells are identified. Work now in progress to quantitate autoradiographs from the same portions of bone as those examined in our previous studies with the electron microscope should provide more definitive answers to the alpha particle fluence to which the cells were subjected.

References

1. E. L. Lloyd and C. B. Henning, Cells at risk for the production of bone tumors in man — An electron microscope study of the endosteal surface of control bone and bone from a human radium case, Radiological and Environmental Research Division Annual Report, July 1978—June 1979, ANL-79-65, Part II, pp. 39-53.
2. A. M. Brues, Recent cases of radium-induced malignancy, Radiological and Environmental Research Division Annual Report, July 1977—June 1978, ANL-78-65, Part II, pp. 1-4.

3. E. L. Lloyd, C. B. Henning, and M. A. Gemmell, 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, Radiological and Environmental Research Division Annual Report, July 1977—June 1978, ANL-78-65, Part II, pp. 9-19.
4. E. L. Lloyd and C. B. Henning, Cells at risk for the production of bone tumors in radium exposed individuals: An electron microscope study, submitted for publication.
5. F. W. Spiers, II. A review of some factors affecting the radiation damage to bone from ingested radium, Br. J. Radiol. 37, 85-88 (1964).
6. H. Spiess and C. W. Mays, Bone cancers induced by ^{224}Ra (ThX) in children and adults, Health Phys. 19, 713-729 (1970).
7. J. H. Marshall, P. G. Groer, and R. A. Schlenker, Dose to endosteal cells and relative distribution factors for radium-224 and plutonium-239 compared to radium-226, Health Phys. 35, 91-101 (1978).
8. E. L. Lloyd, M. A. Gemmell, C. B. Henning, D. S. Gemmell, and B. J. Zabransky, Cell survival following multiple-track alpha particle irradiation, Int. J. Radiat. Biol. 35, 23-31 (1979).
9. G. W. Barendsen, Impairment of the proliferative capacity of human cells in culture by α particles with differing linear-energy transfer, Int. J. Radiat. Biol. 8, 453-466 (1964).
10. R. E. Rowland and J. H. Marshall, Radium in human bone: The dose in microscopic volumes of bone, Radiat. Res. 11, 299-313 (1959).
11. E. L. Lloyd, The distribution of radium in human bone, Br. J. Radiol. 34, 521-528 (1961).
12. H. S. Martland, Occurrence of malignancy in radioactive persons, Am. J. Cancer 15, 2435-2516 (1931).
13. G. W. Casarett, Pathogenesis of radionuclide-induced tumors, Radionuclide Carcinogenesis, C. L. Sanders, R. H. Busch, J. E. Ballou, and D. E. Mahlum, Eds., USAEC Technical Information Center, Oak Ridge, pp. 1-14 (1973).
14. E. L. Lloyd, M. A. Gemmell, C. B. Henning, D. S. Gemmell, and B. J. Zabransky, Transformation of mammalian cells by alpha particles, Int. J. Radiat. Biol. 36, 467-478 (1979).
15. R. E. Rowland, A. F. Stehney, and H. F. Lucas, Jr., Dose-response relationships for female radium dial workers, Radiat. Res. 76, 368-383 (1978).
16. M. J. Harris and R. A. Schlenker, Quantitative histology of the accessory nasal sinuses and mastoid cavities, Annals of Otolaryngology, Rhinology, and Laryngology, in press.

SURVIVAL OF HUMAN OSTEOSARCOMA CELLS AND NORMAL HUMAN FIBROBLASTS FOLLOWING ALPHA PARTICLE IRRADIATION

E. L. Lloyd and M. A. Gemmell

Cell survival of human osteosarcoma cells in culture following alpha particle irradiation is reported here for the first time. The osteosarcoma cell line (TE-85) is found to be less sensitive to inactivation by 5.6 MeV alpha particles (LET 86 keV/ μ m) than normal diploid human fibroblasts (NFS). Values for the mean lethal doses were estimated to be 103 rads for the TE-85 cells compared with 68 rads for the NFS cultures irradiated under identical conditions. It is postulated that the aneuploidy of the tumor cells with increased DNA chromosomal material may confer a selective advantage for the survival of tumor cells relative to normal cells with diploid chromosomes. If this is true, most of the earlier reports on the effects of alpha particle irradiation of supposedly normal cells need to be re-evaluated, because the cells that were irradiated are now known to be of tumor origin (HeLa-carcinoma of the cervix).

Introduction

In an earlier report, Weichselbaum et al. (1977)¹ showed no significant difference between the in vitro x-ray survival curve of an exponentially dividing population of human osteosarcoma cells and normal human diploid fibroblasts. However, the osteosarcoma cells were found to be "surprisingly" sensitive to UV when compared with the normal control cells. As far as is known, the present report is the first to describe the survival of human osteosarcoma cells following alpha particle radiation. Here, in contrast to the results observed with x rays and UV, we have found an increased survival of osteosarcoma cells relative to normal human diploid fibroblasts.

Materials and Methods

Cell Cultures

The osteosarcoma cells (TE-85) were provided by Contract E-73-2001-NO1 within the Special-Virus Program, NIH, PHS, through the courtesy of Dr. W. A. Nelson-Rees. The cell line was established by Dr. R. M. McAllister's laboratory² from an osteosarcoma of the distal right femur in a 13-year-old female Caucasian. These TE-85 cells have previously been characterized both in our laboratory³ and elsewhere.⁴ They have been shown to be aneuploid with an epitheloid

morphology and to stain positively for alkaline phosphatase -- a property which is characteristic of osteosarcoma cells. The normal diploid fibroblast cells (NFS) were established from normal human foreskin and were obtained from Dr. B. Casto at Bio-Labs, Inc. (2910 MacArthur Blvd., Northbrook, Illinois 60062). The cells, TE-85 passage 68, and NFS passage 25, were plated in 60 mm Falcon plastic Petri dishes in 5 ml of Eagle's basal medium, supplemented with 10% heat-inactivated fetal bovine serum and 1% gentamicin and incubated in 5% CO₂ in a humidified incubator. The number of cells plated was varied, depending on dose. The original number was gauged from preliminary cell survival measurements to result in about 40 surviving colonies per 60 mm dish after irradiation. The plates were stained 14 days later, as described in a previous report,⁵ and the number of colonies counted. The plating efficiency for each radiation dose was determined by dividing the number of surviving colonies by the number of cells plated. Cell survival was also determined by dividing the number of surviving colonies in the irradiated plates by the number in the unirradiated control.

Irradiation

The cells were irradiated with a parallel beam of α particles which had an energy of 5.6 MeV, corresponding to an LET of 85 keV/ μ m at the cell surface.⁵ The irradiation times varied from 15 sec to 2 min. Control plates were placed in the same position as the irradiated samples with the beam switched off. Details of the experimental arrangement and its calibration have been described.^{5,6}

Results

Figure 1 shows the survival of the osteosarcoma cells and the normal human fibroblasts as a function of dose and alpha particle fluence. Each point on the graph is shown with the standard error and represents the mean of 5 to 16 replicate plates. Both curves can be described by a single exponential function within the limits of the experimental error with a D₀ value (37% survival), corresponding to 103 rads for the osteosarcoma cells and 68 rads for the normal human fibroblasts. The corresponding alpha particle fluences for D₀ are 5.0×10^6 alphas/cm² for the normal human fibroblasts and 7.6×10^6 alphas/cm² for the osteosarcoma cells. Expressed in another way, the effective cross section

FIG. 1.--The survival of human osteosarcoma cells (TE-85) and normal human fibroblasts (NFS) following alpha-particle irradiation. NFS, $D_0 = 5 \times 10^6$ α 's/cm² (68 rad); TE-85, $D_0 = 7.6 \times 10^6$ α 's/cm² (103 rad).

for cell killing is $20 \mu\text{m}^2$ for NFS and $13.2 \mu\text{m}^2$ for TE-85 cells. In order to determine what fraction of the cross-sectional area of the nuclei this represented, measurements of nuclei were made from phase contrast pictures of the cells as they were irradiated in culture. Figure 2 shows the distribution of the nuclear areas of 20 cells measured from each cell type. The TE-85 had a cross-sectional area about twice as large as the NFS cells. The mean nuclear area of the TE-85

FIG. 2.--The distribution of nuclear areas of osteosarcoma cells (TE-85) and normal fibroblasts (NFS) measured by phase contrast light microscopy.

cells was $491 \mu\text{m}^2$, compared with a mean nuclear area of $240 \mu\text{m}^2$ for the NFS cells. Figures 3 and 4 show stained preparations of the two cell types. Here, the TE-85 cells are seen to have large irregular nuclei and are often multinucleated. For the purpose of the measurements, only cells with single nuclei were included, and their area was determined as previously described⁵ by regarding them as ellipses and using the formula $\frac{\pi}{4}ab$, where a and b are the major and minor axes. From these measurements, the mean lethal dose for cell killing was calculated to be the equivalent of about 37 alpha particles through each nucleus, compared with an average value of 12 alphas for the NFS nuclei. The average thicknesses of nuclei from the two cell types were determined from electron micrographs (after flat embedding of the cells in situ as already described).⁵

FIG. 3.--Stained preparations of osteosarcoma cells (TE-85) showing the pleomorphic nature of the darkly stained nuclei ($\times 200$). (ANL Neg. 149-80-134)

FIG. 4.--Stained preparations of normal human fibroblasts (NFS). Note the regular appearance of these nuclei compared with those seen in Fig. 3 ($\times 200$). (ANL Neg. 149-80-135)

The TE-85 cells showed a larger distribution in nuclear thickness compared with the NFS cells. Average values for the two cell types, when 20 cells of each type were measured, were 1.6 μm and 1.4 μm , respectively, for the TE-85 and NFS cells. This meant that the nuclear volumes of the TE-85 cells were on average 785 μm^3 , compared with 336 μm^3 for the NFS cells.

Discussion

Most of the previous measurements on the survival of human cells showing irradiation by alpha particles or heavy ion beams have been carried out with T-1 cells.⁷⁻⁹ These cells were supposed to have originated from a male human kidney¹⁰ and, until recently, have been regarded as normal human kidney cells. A recent report,¹¹ however, has shown unequivocally that these cells, which have been widely used in this country and in Europe, have the genetic markers of HeLa cells which were originally derived from the carcinoma of the cervix.¹² Hence, the vast majority of the literature on the effect of alpha particles on human cells relates not to normal human cells as had been supposed, but to tumor cells.

Since HeLa cells are aneuploid with chromosome numbers in the ranges 54 to 70 (modal number = 65), compared with the stable number of 46 for normal human diploid cells and since we believe that cell killing and cell transformation are related to genetic damage, it would appear to be fortuitous if the cell survival should turn out to be the same for HeLa cells and normal cells. Similarly, in the experiment reported here, the aneuploid nature of the TE-85 cells, with chromosome numbers in the range 50 to 59⁴ (modal number $\sim$ 54), might be expected to give rise to a different survival when compared with the normal NFS cells. In addition, the nuclear cross-sectional area and volume of the tumor cells in the present study were found to be more than twice that of the normal cells.

In the only other published report (of which we are aware) of inactivation of normal human fibroblasts by alpha particle irradiation,¹³ a D_0 value of 32 rads was reported for alpha particles of similar LET to that used here (90 keV/ μm compared with 86 keV/ μm in our experiment). The fibroblasts used by Cox and Masson¹³ were irradiated through specially prepared Melinex plastic films, and

a feeder layer of cells was used. By contrast, our cells were irradiated directly on standard plastic tissue culture dishes without feeder layers. In our hands, cells tend to spread more and grow better on the normal culture dishes than on Melinex films; hence, the cross-sectional area of the nuclei would be expected to be larger in our experiments when the cells are exposed to a parallel beam of radiation. These differences in the conditions under which the cells were irradiated may, indeed, be important and lead to the lower value reported by Cox and Masson.¹⁶ However, although the particular geometry used would give rise to a smaller number of traversals of alpha particles through the nuclei of the more rounded cells, the total path length of the traversals would not be expected to be altered except in the case of a difference of nuclear volumes. Other differences between the experiment reported by Cox and Masson¹⁶ and that reported here involve differences in the origins of the fibroblasts. Cox and Masson¹⁶ used human diploid lung fibroblasts, designated H-19, while our cells (NFS) were established from newborn human foreskin. This difference in the origin of the cells may be significant, as well as all of the other factors known to affect cell survival, such as the passage number, the serum, medium, pH, temperature, etc.

The two cell cultures used in the experiment described here were irradiated under identical conditions using the same medium, etc.; hence, the increased survival of the osteosarcoma cells, compared with the normal fibroblasts, would appear to be a real effect reflecting basic differences in their sensitivities for cell inactivation. The cells of each culture grew well and formed good colonies; moreover, the cell doubling times of both cell types were the same within experimental error (estimated to be 24.4 hr in the logarithmic growth phase). However, before drawing any definitive conclusions about the relative sensitivities for osteosarcoma cells versus normal diploid cells, it would be necessary to determine if the culture conditions were optimal for each cell culture. In addition, different strains of normal cells and osteosarcoma cells obviously need to be tested before drawing any general conclusions.

Regardless of all these considerations, the fact that in the present experiment the osteosarcoma cells survived better than the normal fibroblasts is a result which might be expected if tumor cells are better able to propagate following

irradiation because of their generally greater reservoir of chromosomal DNA through aneuploidy. This has, indeed, been found in early experiments by others^{14,15} when cells of different ploidy were irradiated by x rays in vivo. More recent work by Cox and Masson¹⁶ also suggests an increase in radio-resistance with increased time in culture when abnormal karyotypes develop.

In conclusion, the finding of different sensitivities for inactivation of human tumor cells, when compared with normal human fibroblasts following alpha irradiation, suggests that almost all of the earlier studies with alpha particle radiation need to be re-evaluated in the light of the recent finding that T-1 cells have been mistaken for normal cells.

References

1. Ralph Weichselbaum, John B. Little, and John Nove, Response of human osteosarcoma in vitro to irradiation: Evidence of unusual cellular repair activity, *Int. J. Radiat.* 31, 295-299 (1977).
2. R. M. McAllister, M. B. Gardner, A. E. Green, C. Bradt, W. W. Nichols, and B. H. Landing, Cultivation in vitro of cells derived from a human osteosarcoma, *Cancer* 27, 397-402 (1971).
3. E. L. Lloyd, C. B. Henning, and F. Mackevicius, Differences in the characteristics of cell cultures established from seven human osteosarcomas, Radiological and Environmental Research Division Annual Report, July 1974-June 1975, ANL-75-60, Part II, pp. 43-73.
4. Helene S. Smith, Robert B. Owens, Alan J. Hiller, Walter A. Nelson-Rees, and James O. Johnston, The biology of human cells in tissue culture. I. Characterization of cells derived from osteogenic sarcomas, *Int. J. Cancer* 17, 219-234 (1976).
5. E. L. Lloyd, M. A. Gemmell, C. B. Henning, D. S. Gemmell, and B. J. Zabransky, Cell survival following multiple-track alpha particle irradiation, *Int. J. Radiat. Biol.* 35, 23-31 (1979).
6. E. L. Lloyd, M. A. Gemmell, C. B. Henning, D. S. Gemmell, and B. J. Zabransky, Transformation of mammalian cells by alpha particles, *Int. J. Radiat. Biol.* 36, 467-478 (1979).
7. G. W. Barendsen, Responses of cultured cells, tumours, and normal tissues to radiations of different linear energy transfer, Current Topics in Radiation Research, Vol. IV, Michael Ebert and Alma Howard, Eds., North-Holland Publishing Co., Amsterdam, pp. 293-355 (1968).
8. Paul Todd, Heavy-ion irradiation of human and Chinese hamster cells in vitro, *Radiat. Res.* 61, 288-297 (1975).
9. Eleanor A. Blakely, Cornelius A. Tobias, Tracy C. H. Yang, Karen C. Smith, and John T. Lyman, Inactivation of human kidney cells by high-energy monoenergetic heavy-ion beams, *Radiat. Res.* 80, 122-160 (1979).

10. J. van der Veen, L. Bots, and A. Mes, Establishment of two human cell strains from kidney and reticulosarcoma of lung, *Archiv für die Gesamte Virusforschung* 3, 230-238 (1958).
11. Walter A. Nelson-Rees, T-1 cells are HeLa and not of normal human kidney origin, *Science* 209, 719-720 (1980).
12. Susan H. Hsu, Bernice Z. Schacter, Nancy L. Delaney, Thomas B. Miller, Victor A. McKusick, R. H. Kennett, J. G. Bodmer, D. Young, and W. F. Bodmer, Genetic characteristics of the HeLa cell, *Science* 191, 392-394 (1975).
13. Roger Cox and W. K. Masson, Mutation and inactivation of cultured mammalian cells exposed to beams of accelerated heavy ions. III. Human diploid fibroblasts, *Int. J. Radiat. Biol.* 36, 149-160 (1979).
14. Roger J. Berry, Quantitative studies of relationships between tumor cell ploidy and dose response to ionizing radiation in vivo. Modification of radiation response in a previously irradiated tumor, *Radiat. Res.* 18, 236-245 (1963).
15. L. Revesz and U. Norman, Chromosome ploidy and radiosensitivity of tumours, *Nature* 137, 861-862 (1960).
16. Roger Cox and W. K. Masson, Changes in radiosensitivity during the in vitro growth of diploid human fibroblasts, *Int. J. Radiat. Biol.* 26, 193-196 (1974).

INHIBITION OF GROWTH OF HUMAN OSTEOSARCOMA CELLS IN CULTURE BY NORMAL HUMAN FIBROBLASTS*

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

Suppression of transformed cells by untransformed cells has previously been demonstrated. In the experiment described here, attempts were made to see if two well-characterized human osteosarcoma cell lines (TE-85 and SaOS-2) could be similarly inhibited by normal human fibroblasts in culture. Two hundred cells of each of the osteosarcoma cell lines were plated onto 60 mm plastic Petri dishes. Different numbers ($200, 10^3, 10^4, 10^5$) of normal fibroblasts were added to the tumor cells and mixed prior to incubation of the cells for a 4-week period, when they were stained for alkaline phosphatase. Since this stain is selective for the osteosarcoma cells, the tumor cells were clearly visible against the background of normal fibroblasts. As the number of normal cells was increased to 10^5 , the size and number of the tumor colonies were greatly reduced compared with the controls, in some cases to less than 5% of the control value. Although the mechanisms whereby the normal cells effect this reduction is not understood, the use of normal cells, or substances derived therefrom, may have potential use in the restriction of malignant tumors in man.

* Abstract of a paper presented at the 71st Annual Meeting of the American Association for Cancer Research, San Diego, California, 26-31 May 1980.

FURTHER STUDIES ON THE SUPPRESSION OF GROWTH OF TWO OSTEOSARCOMA CELL LINES BY NORMAL CELLS USING ALKALINE PHOSPHATASE

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

Two osteosarcoma cell lines (TE-85 and SaOS-2) were co-cultivated with four different normal human cell strains of both fibroblastic and epithelial origin. In all cases, the expression of the tumor cells was progressively suppressed as the number of the normal cells was increased. In addition, the tumor cells in contact with normal fibroblasts took on the spindle-shaped appearance of the fibroblasts although they stained positively for alkaline phosphatase which was used throughout as a specific enzyme marker for the osteosarcoma cells.

Introduction

In previous experiments, we have shown the inhibition of growth of malignantly transformed mouse embryo cells (C3H 10T1/2) when these cells were co-cultivated with the untransformed parental cell line.¹ Similarly, we have observed a suppression in the growth of two human osteosarcoma cell lines when grown together with a cell strain of normal human fibroblasts (NFS).² The present report extends our observations with the human osteosarcoma cell lines to determine if the effects could be reproduced with other normal human fibroblasts and also with normal human cells of epithelial origin. The effect of co-cultivation for different times was also investigated. In addition, we describe here the use of alkaline phosphatase as a marker for the osteosarcoma cells. Because of the specificity of this stain for the osteosarcoma cells, we have been able to obtain a sharper delineation between the tumor cells and the normal cells than was obtained by relying on the morphological appearance of the two cell types.²

Materials and Methods

Cell Lines

The characteristics of the osteosarcoma cell lines (TE-85 and SaOS-2) used in the earlier experiments have been described.³ In the present experiment, two more normal fibroblast cell lines, WI-38 and KD, were used. WI-38 was established by Dr. L. Hayflick and obtained from the American Type Culture

Collection Cell Repository, Rockville, Maryland 20850. This line was derived from a normal human embryonic lung and is fibroblastic. KD was initiated by Dr. R. S. Day from a skin biopsy sample taken from the lip of a normal adult female and kindly sent to us by Dr. Takeo Kakunaga, National Cancer Institute, Bethesda, Maryland 20014. An epithelial cell line, AP318, which had been established from human fetal intestine and provided by Dr. W. A. Nelson-Rees, Naval Biomedical Research Laboratory, Oakland, California, was also tested to see if epithelial cells, too, would inhibit the growth of osteosarcoma cells.

Staining Procedure

To determine the percentage of normal and osteosarcoma cells which are positive for alkaline phosphatase activity, 200 cells per dish were plated in 60 mm Petri dishes, 10 dishes per cell line, in 5 ml BME with Earle's Salts, 10% fetal bovine serum and 1% gentamicin and incubated for approximately 14 days in 5% CO₂ in a humidified incubator. Five plates from each cell line were then stained with Giemsa, which stains all cells and is the usual stain used for determining plating efficiency;¹ five plates of each line were stained for alkaline phosphatase activity, using the following method. The medium was poured off the plates which were washed twice with phosphate-buffered saline, and the cells were fixed for 30 min in 10% formalin in methanol at 4°C. The fixative was then poured off and the plates rinsed several times with distilled water. The cells were stained with a freshly prepared mixture consisting of 4 ml naphthol AS-MX phosphate substrate (Sigma) solubilized and 24 mg Fast Violet B salt in 98 ml distilled water, and left to stain for 30 min at room temperature. The dishes were then rinsed in tap water and air dried.

Co-Culturing of Normal and Tumor Cells

The method of co-cultivation has been described previously.² Briefly, 200 osteosarcoma cells (TE-85, passage 16 and SaOS-2, passage 36) were mixed with each of the following numbers of normal cells: 10², 10³, 10⁴, and 10⁵, and in some cases, the tumor cells were added to confluent monolayers of normal cells. Three plates were seeded at each dilution. The cells were then incubated in a humidified incubator in an atmosphere of 5% CO₂. The plates were fed twice weekly until confluent and then weekly until either the 4th or 12th week when

they were fixed and stained for alkaline phosphatase activity.

Results

Figure 1 shows the reduction in the extent to which tumor cells (TE-85) cover the plates as the number of normal cells (WI-38 or AP318) is increased. The cells were co-cultivated for 4 weeks. Similarly, although not shown here, each of the normal cell cultures restrained the growth of both of the osteosarcoma cell lines tested, and this inhibition increased with increasing numbers of normal cells as documented in Tables 1 and 2.

SaOS-2 had a different pattern of growth from that of TE-85 when grown alone. After 4 weeks, it did not form a confluent monolayer; instead, discrete colonies began to pile up in their centers and secondary colonies developed (Fig. 2). With increasing numbers of normal cells, this piling up and secondary colony formation did not occur (Fig. 3). These results agree with those carried

200 HUMAN OSTEOSARCOMA CELLS (TE-85, p. 18)	NUMBER OF NORMAL HUMAN FIBROBLASTS (WI-38, p. 23)	200 HUMAN OSTEOSARCOMA CELLS (TE-85, p. 19)	NUMBER OF NORMAL HUMAN EPITHELIAL CELLS (AP-318, p. 18)
0		0
100		100
1,000		1,000
10,000		10,000
100,000		100,000

FIG. 1.--TE-85 (passage 18 or passage 19) grown with normal human fibroblasts and normal human epithelial cells for 4 weeks and then stained for alkaline phosphatase. Only the tumor cells stain.
(ANL Neg. 149-80-111)

Table 1. Fraction of area of dish covered by tumor cells when 200 SaOS-2 cells were co-cultivated with different numbers of normal fibroblasts (NFS, KD, WI-38) or normal epithelial cells (AP318) for 4 weeks.

No. of cells	NFS	KD	WI-38 ^a	AP318
0	0.65	0.65	0.8	0.31
10 ²	0.06	0.2	0.8	0.34
10 ³	0.02	0.07	0.6	0.14
10 ⁴	0.01	0.07	0.15	0.024
10 ⁵	0.006	0.05	0.05	0.022
Confluent	0.0007	N.T. ^b	N.T. ^b	N.T. ^b

^a With WI-38 cells, 400 SaOS-2 cells (instead of 200) were seeded.

^b Not tested.

Table 2. Fraction of area of dish covered by tumor cells when 200 TE-85 cells were co-cultivated with different numbers of normal fibroblasts (NFS, KD, WI-38) or normal epithelial cells (AP318) for 4 weeks.

No. of cells	NFS	KD	WI-38	AP318
0	2.0	1.0	1.0	1.0
10 ²	0.9	0.9	0.98	1.0
10 ³	0.5	0.5	0.8	0.6
10 ⁴	0.04	0.3	0.4	0.08
10 ⁵	0.004	0.2	0.04	0.05
Confluent	0.004	N.T. ^a	N.T.	N.T.

^a Not tested.

FIG. 2.--SaOS-2 colony growing without normal cells, showing cells piled up in center ($\times 40$).
(ANL Neg. 149-80-123)

FIG. 3.--SaOS-2 colony growing with NFS cells ($\times 40$). Note the effect of NFS on shape of SaOS-2 colony and cells and the lack of piling up in the center. The NFS cells cannot be seen as they do not stain.
(ANL Neg. 149-80-120)

out previously² with NFS, TE-85 and SaOS-2 when Giemsa staining was used and the cells left for the same time before staining (4 weeks).

In addition to the decrease in the area occupied by the transformed cells, another effect of the untransformed cells on the transformed colonies was observed. Microscopic examination showed that normal fibroblasts affect the shape of the osteosarcoma cells. Both TE-85 and SaOS-2, which are normally epithelial-like, became fibroblastic in appearance when adjacent to, or in close contact with, the normal fibroblasts. Figure 4 shows the normal epithelial-like appearance of the SaOS cells growing alone and stained for alkaline phosphatase. In Fig. 5, the SaOS cells, growing with the NFS cells, have become fibroblastic. The NFS cells do not produce alkaline phosphatase and, therefore, are not stained. With increasing distance from the normal fibroblasts, the tumor cells retain their epithelial shape. This can be seen in Fig. 6, where the SaOS-2 cells at the bottom of the picture are in close contact with normal fibroblasts (unstained) and appear fibroblastic, whereas the cells in the center of the colony are epithelial-like.

With increased contact between the tumor and normal cells (e.g., when increasing numbers of normal cells are seeded with the tumor cells), the colony shape also changes from round to elongate (compare Figs. 2 and 3). The results shown in Fig. 1 refer to experiments where the normal and tumor cells were cultured together for 4 weeks. To determine the effect of co-cultivation for a longer time, one plate at each dilution of the KD cells mixed with either the TE-85 or SaOS-2 cells was fed weekly until the 12th week and then stained for alkaline phosphatase. As can be seen in Table 3, the growth of the tumor colonies continues to be suppressed by the normal cells and few, if any secondary tumor colonies develop.

FIG. 4.--SaOS-2 cells growing alone and stained for alkaline phosphatase ($\times 320$). Note epithelial shape.

FIG. 5.--SaOS-2 cells stained for alkaline phosphatase, growing with NFS, unstained because they do not produce alkaline phosphatase ($\times 320$). Note fibroblastic shape.

FIG. 6.--Note fibroblastic shape of SaOS-2 cells in contact with NFS cells (bottom of figure) and the progressive change to epithelial form as their distance from the normal cells increase (top of figure) ($\times 200$).
(ANL Neg. 149-80-141)

Table 3. Fraction of area of dish covered by tumor cells (SaOS-2 or TE-85) when cultivated with different numbers of KD cells for 12 weeks.

Number of KD cells	SaOS-2 (200 cells)	TE-85 (200 cells)
0	1.0	1.0
10^2	0.6	0.95
10^3	0.4	0.6
10^4	0.3	0.6
10^5	0.24	0.5

Discussion

Figure 1 and Tables 1-3 all show increased containment of both of the tumor cell lines with all of the normal cell strains studied. There was no notable difference between the extent of the suppression by epithelial cells (AP-318) when compared with the three fibroblast cultures. At the highest cell densities, the normal human foreskin cells (NFS) appeared to be superior to all the other normal cells in effecting suppression of the tumor cells. The reason for this is not clear. One can only speculate that perhaps the young age of the cell and the more spindle-like morphology may contribute to its selective advantage. Although we have been unable, so far, to identify the mechanism by which the tumor cell suppression is effected, it is our impression that cell contact plays an important role. This has also been suggested by other workers.

Eagle et al.^{4,5} studied a variety of human diploid cell strains which were self-contact inhibited. These cells were also found to inhibit each other in mixed culture. However, in agreement with our findings, the growth of some hetero-ploid cells which were not contact inhibited in pure culture, were found to be significantly inhibited when inoculated onto a formed layer of normal diploid cells. Similar suppression of the malignant state has been demonstrated by Silagi et al.,⁶ who showed that malignant mouse melanoma cells, mixed with non-malignant mouse melanoma cells and injected into immunocompetent hosts, produce only 1/47 tumors, compared with 100% tumor formation when the malignant melanoma cells are injected alone. Some cell-to-cell contact appeared to be essential for the suppressive effect since neither separate injection of the malignant and non-malignant cells at different sites or sequential inoculation within the same bleb at the same site produced tumor suppression.

In contrast to the work of Eagle et al.^{4,5} and Silagi et al.⁶ where cell contact was considered necessary for suppression of the transformed state, Lipkin et al.⁷ were able to effect suppression using a substance extracted from the medium. These workers isolated a diffusible factor (melanocyte contact inhibitory factor, MCIF) from culture medium of a contact-inhibited line of hamster melanocytes. This factor restored contact inhibition of growth to malignant human, mouse, and hamster melanocytes and was also found to inhibit

growth of a broad spectrum of malignant cell types. In a preliminary experiment carried out using Lipkin's technique with transformed 10T1/2 cells in our laboratory, we were unable to effect a similar suppression using the supernatant from confluent, contact-inhibited untransformed 10T1/2 cells. The reason for the difference between our results and those of Lipkin et al. is not clear. The different culture conditions in the two laboratories using different cells, serum, medium, etc., might give rise to different products which were unstable or similar products at different concentrations which proved to be ineffective. However, because of the great potential for therapeutic use of a substance which could suppress the growth of tumor cells, further work with many different cell lines under many different culture conditions would appear to be of paramount importance.

References

1. E. L. Lloyd, M. A. Gemmell, and C. B. Henning, Suppression of transformed foci induced by alpha radiation of C3H/10T1/2 cells, by untransformed cells, Radiological and Environmental Division Annual Report, July 1977-June 1978, ANL-78-65, Part II, pp. 20-31.
2. E. L. Lloyd and M. A. Gemmell, Suppression of growth of two human osteosarcoma cell lines by normal human fibroblasts, Radiological and Environmental Research Division Annual Report, July 1978-June 1979, ANL-79-65, Part II, pp. 55-64.
3. C. B. Henning and E. L. Lloyd, Identification of osteosarcoma cells in culture using alkaline phosphatase, Radiological and Environmental Research Division Annual Report, July 1976-June 1977, ANL-77-65, Part II, pp. 57-71.
4. Harry Eagle, Elliot M. Levine, and Hilary Koprowski, Species specificity in growth regulatory effects of cellular interactions, *Nature* 220, 266-269 (1968).
5. Harry Eagle and Elliot M. Levine, Growth regulatory effects of cellular interaction, *Nature* 213, 1102-1106 (1967).
6. S. Silagi, E. Newcomb, and J. Silverstein, Loss of tumor-forming ability by malignant melanoma cells mixed with nonmalignant BrdU-grown derivatives. Abstract of a paper presented at the Cold Spring Harbor Conf. on the Transformed Cell, May 18-22 (1977), p. 7.
7. George Lipkin, Margarete E. Knecht, and Martin Rosenberg, A potent inhibitor of normal and transformed cell growth derived from contact-inhibited cells, *Cancer Res.* 38, 635-643 (1978).

MITOGENIC STIMULATION OF PERIPHERAL LYMPHOCYTES FROM RADIUM WORKERS*

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

The immunocompetence of peripheral blood lymphocytes obtained from former radium dial painters was investigated by in vitro mitogenic stimulation assays. A reduction in lymphocyte stimulation was observed in these radium workers when compared with cells from normal age-matched controls. This reduced activity varied according to the mitogen employed (i.e., 28% with PHA, 47% with Con A, and 46% with PWM). This decreased activity could not be related to either age or ^{226}Ra body burden of the lymphocyte donor. Sera obtained from high body burden ($>0.1 \mu\text{Ci}$) radium cases was found inhibitory to normal control lymphocyte stimulation in 3/6 cases tested with PHA, 6/6 cases tested with Con A, and 1/6 cases tested with PWM. Sera from low body burden donors ($<0.1 \mu\text{Ci}$) was found inhibitory in 2/6 cases stimulated with either Con A or PHA and 0/6 cases stimulated with PWM. Normal control lymphocytes separated on discontinuous Ficoll gradients according to their buoyant densities were also examined. The resulting subpopulations were found to be stimulated to different extents upon treatment with the mitogen PHA. These subgroups are being tested with other mitogens to determine if any one subgroup is selectively responsible for the differences observed in the radium population.

* Abstract of a paper presented at the Federation of American Societies for Experimental Biology (FASEB) Meeting, Los Angeles, California, 13-17 April 1980.

MITOGENIC RESPONSES OF NORMAL HUMAN LYMPHOCYTES INCUBATED WITH SERA FROM RADIUM WORKERS

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

Sera from radium workers were incubated with normal human lymphocytes and compared with sera from normal age-matched controls for its effect on lymphocyte stimulation with different mitogens. The results obtained with sera from the radium workers with high residual body burdens ($> 0.1 \mu\text{Ci } ^{226}\text{Ra}$) were shown to inhibit stimulation following treatment with concanavalin A (Con A) but not with phytohemagglutinin (PHA) nor pokeweed mitogen (PWM).

Introduction

The increased incidence of malignancies and the pathological complications resulting from the ingestion of radium isotopes (^{226}Ra and ^{228}Ra) are well documented.¹⁻³ We have recently reported a reduction in lymphocyte stimulation by different mitogens in former radium workers when compared with age-matched controls (unexposed to radium).⁴ We were unable to relate this reduction activity in lymphocyte stimulation to the measured body burden of ^{226}Ra , but the small number of high level radium cases available for study made it impossible to preclude such an effect. In addition, we could not rule out the "healthy worker effect,"⁵ which might have been responsible for the increased stimulation of lymphocytes from the control population. We, therefore, decided to examine another index of the immune response, namely, the possible inhibitory properties of sera from high- and low-body burden radium cases. The present report is, thus, concerned with the stimulation of normal human lymphocytes by various plant mitogens in the presence of sera from radium patients and control donors.

Materials and Methods

Sera

The sera used for this study were divided into four groups: (A) Sera from four patients with residual body burdens $> 0.1 \mu\text{Ci } ^{226}\text{Ra}$ as measured with a whole body counter. Sera from this group are referred to as "high body burden sera." (B) Sera from patients with residual body burdens $< 0.1 \mu\text{Ci } ^{226}\text{Ra}$ designated "low body burden sera." (C) Sera from age-matched healthy laboratory workers

termed "control homologous sera." (D) Sera from the same subjects who donated the lymphocytes, when used with the same individual's lymphocytes called "control autologous sera." The sera from groups A, B, and C were stored at -10°C prior to use while the sera in group D were obtained fresh.

Treatment of Normal Lymphocytes with Sera from Controls and Radium Patients

The method used for measuring the stimulation of lymphocytes has already been described in detail.⁴ The basic principle of this method involved measuring the amount of tritiated thymidine (^3H) taken up by the lymphocytes as an index of their blastogenic activity. In the present study, the lymphocytes from each of six healthy laboratory workers (one in each decade of life from 20 to 80) were incubated with each test serum for one hour. The cells were then washed with phosphate buffered saline three times and resuspended at a concentration of 4×10^6 cells/ml in medium containing a 10% concentration of serum from the controls or radium patients. Aliquots (2×10^5 cells per well) were then stimulated with three different mitogens: phytohemagglutinin (PHA), concanavalin A (Con A) and pokeweed mitogen (PWM). The stimulation is expressed as the counts per minute measured for 0.5 μCi of tritiated thymidine (^3H) initially added to each well.⁴ The effects of serum from individuals were tested separately (i.e., not pooled), and duplicate measurements were made for each sample of serum.

Results

The results of the stimulation measured for lymphocytes incubated with the test sera in the four serum groups with six control subjects are given in Tables 1, 2, and 3. Each table refers to the stimulation with a different mitogen. In the high body burden group, the means of results for three of the patients are shown separately from the results for the other patient (03-404), because significantly lower stimulation was consistently obtained in the presence of serum from that patient. The mean values shown for the three serum donors were used for statistical tests of the difference between high body burden sera and sera from the other groups. A modified Student's *t*-test with two-tailed probabilities was used. Differences between the low body burden sera and the control homologous sera were similarly tested.

Table 1. Effects of serum from radium patients on normal control lymphocyte stimulation by PHA.^a The values are given in counts per minute for 0.5 μ Ci tritiated thymidine (³H) per 2×10^5 lymphocytes per well.

Serum donors	No. of serum donors	Age and sex of control donors of lymphocytes					
		25 f	32 m	42 f	52 f	65 m	72 m
A High body burden sera ($0.458 \pm 0.229 \mu\text{Ci}$)	3	78,000 ($\pm 5,700$)	98,000 ($\pm 4,800$)	59,000 ($\pm 4,400$)	78,000 ($\pm 5,200$)	81,000 ($\pm 2,800$)	64,000 ($\pm 4,500$)
Patient 03-404 (0.58 μCi)	1 ^b	12,000 3,900	22,000 26,000	9,100 10,000	33,000 37,000	48,000 42,000	10,000 13,000
B Low body burden sera ($0.044 \pm 0.017 \mu\text{Ci}$)	4	79,000 ($\pm 9,600$)	86,000 ($\pm 18,000$)	65,000 ($\pm 14,000$)	83,000 ($\pm 8,700$)	73,000 ($\pm 5,000$)	62,000 ($\pm 10,000$)
C Control homologous sera	4	91,000 ($\pm 4,800$)	97,000 ($\pm 3,900$)	71,000 ($\pm 7,500$)	84,000 ($\pm 7,000$)	76,000 ($\pm 4,400$)	76,000 ($\pm 5,200$)
D Control autologous sera	1	92,000 87,000	100,000 89,000	75,000 81,000	86,000 84,000	80,000 77,000	55,000 54,000

^aMean values for each group of sera are given $\pm$ standard deviation from the mean where more than one serum was tested.

^bWithin the high body burden group, one patient, 03-404, is listed separately since the values obtained for this case were consistently lower by more than 2 standard deviations than the values obtained for the other sera tested within this group. For a single serum donor, the entries represent values for duplicate samples.

Note: The lymphocyte stimulation by PHA was significantly lower with sera from the high body burden group (A) than with the control group (C) for control lymphocyte donors 25 and 72 ($P < 0.05$).

Table 2. Effects of serum from radium patients on normal control lymphocyte stimulation by Con A.^a The values are given in counts per minute for 0.5 μ Ci tritiated thymidine (³H) per 2×10^5 lymphocytes per well.

Serum donors	No. of serum donors	Age and sex of control donors of lymphocytes					
		25 ϕ	32 σ	42 ϕ	52 ϕ	65 σ	72 σ
A High body burden sera	3	7,600 ($\pm$ 520)	29,000 ($\pm$ 6,000)	2,400 ($\pm$ 440)	7,900 ($\pm$ 3,800)	18,000 ($\pm$ 3,300)	17,000 ($\pm$ 4,000)
Patient 03-404 (0.58 μ Ci)	1 ^b	250 210	360 360	400 570	240 280	400 400	400 400
B Low body burden sera (0.044 $\pm$ 0.017 μ Ci)	4	12,000 ($\pm$ 6,300)	27,000 ($\pm$ 8,700)	4,600 ($\pm$ 4,100)	8,300 ($\pm$ 4,200)	26,000 ($\pm$ 16,000)	21,000 ($\pm$ 12,000)
C Control homologous sera	4	22,000 ($\pm$ 6,500)	42,000 ($\pm$ 9,800)	6,200 ($\pm$ 2,900)	12,000 ($\pm$ 4,600)	27,000 ($\pm$ 8,900)	28,000 ($\pm$ 9,700)
D Control autologous sera	1	20,000 15,000	53,000 42,000	5,300 4,500	7,400 7,300	16,000 15,000	17,000 11,000

^a Mean values for each group of sera are given $\pm$ standard deviation from the mean where more than one serum was tested.

^b Within the high body burden group, one patient, 03-404, is listed separately since the values obtained for this case were consistently lower by more than 2 standard deviations than the values obtained for the other sera tested within this group. For a single serum donor, the entries represent values for duplicate samples.

Note: The lymphocyte stimulation by Con A was significantly lower with sera from the high body burden group (A) than with the control group (C) for control lymphocyte donor 25. ($P < 0.10$). Taken together all values for the high body burden group are lower than for the control group ($P < 0.02$).

Table 3. Effects of serum from radium patients on normal control lymphocyte stimulation by PWM. The values are given in counts per minute for 0.5 μ Ci tritiated thymidine (3 H) per 2×10^5 lymphocytes per well.

Serum donors	No. of serum donors	Age and sex of control donors of lymphocytes					
		25 σ	32 σ	42 σ	52 σ	65 σ	72 σ
A High body burden sera ($0.458 \pm 0.229 \mu$ Ci)	3	10,000 ($\pm 1,000$)	19,000 ($\pm 1,500$)	3,700 ($\pm 1,600$)	4,600 (± 590)	18,000 ($\pm 2,100$)	8,800 (± 880)
Patient 03-404 (0.58 μ Ci)	1 ^b	530 730	1,000 960	680 560	1,000 500	1,000 1,200	850 490
B Low body burden sera ($0.044 \pm 0.017 \mu$ Ci)	4	15,000 ($\pm 5,400$)	17,000 ($\pm 6,300$)	3,500 ($\pm 2,200$)	3,600 ($\pm 1,300$)	22,000 ($\pm 11,000$)	10,000 ($\pm 4,000$)
C Control homologous sera	4	16,000 ($\pm 5,700$)	20,000 ($\pm 7,400$)	3,000 (± 990)	3,900 ($\pm 2,800$)	13,000 ($\pm 8,500$)	9,600 ($\pm 5,400$)
D Control autologous sera	1	8,400 7,100	23,000 14,000	2,000 1,600	2,000 1,900	5,000 3,900	3,400 2,500

^a Mean values for each group of sera are given $\pm$ standard deviation from the mean where more than one serum was tested.

^b Within the high body burden group, one patient, 03-404, is listed separately since the values obtained for this case were consistently lower by more than 2 standard deviations than the values obtained for the other sera tested within this group. For a single serum donor, the entries represent values for duplicate samples.

Note: No significant difference was observed between the high body burden group (A) and either the low body group (B) or the control group (C).

PHA

From Table 1 it can be seen that for PHA, a significant difference was found between the high body burden sera and the control homologous sera for two lymphocyte donors 25 and 72 ($P < 0.05$) with the high body burden sera giving lower values. None of the other differences were found to be statistically significant for each individual's lymphocytes or when all the lymphocyte donors were considered as a group except for the individual patient, 03-404. Use of this patient's serum gave lower values with all six lymphocyte donors. These values were more than two standard deviations, both below the others in the high body burden sera group, as well as all the other serum groups. The serum from this patient gave similarly low values, both with Con A (Table 2) and with PWM (Table 3).

Con A

A significant difference ($P < 0.02$) was found between the high body burden sera and the control homologous sera for only one lymphocyte donor (25), stimulated with Con A (Table 2). Although the individual differences for these two serum groups were not statistically significant, values for the high body burden sera, analyzed as a group, were found to be significantly lower ($P < 0.02$) than the values for the control homologous group. In addition, the high body burden values were also lower than the low body burden values for all but one lymphocyte donor (32). The difference between the two groups was found not to be statistically significant ($P \sim 0.4$).

PWM

For pokeweed mitogen (PWM), no statistically significant differences were observed between the different groups of sera tested, apart from the single patient, 03-404, already mentioned.

Discussion

It has been shown that in certain cancer victims, suppressive factors are present in the serum that can inhibit lymphocyte stimulation.⁶ As far as is known, this is the first report to examine whether any similar suppressive factors could be detected in the sera of radium workers who are predisposed to certain

tumors as a result of alpha irradiation. One of the patients in the high body burden group had serum which inhibited stimulation by all the mitogens tested. A study of this patient's medical history revealed no obvious clues as to the reason for this difference. None of the patients studied, including this patient, had any clinical signs of malignancy. When sera from persons with high body burdens of radium ($> 0.1 \mu\text{Ci } ^{226}\text{Ra}$) were compared with sera from normal healthy controls for their ability to stimulate lymphocytes, from normal controls, a decreased response in stimulation was observed with the single mitogen, Con A. The difference in the effects seen with the different mitogens may reflect the selective action of the three mitogens on different cell populations. Con A and PHA primarily stimulate T-cells, while PWM stimulates B-cells. According to Janossy and Greaves,⁷ however, Con A may stimulate T-cells which have not yet reached the level of differentiation at which they can respond to PHA. Although the differences seen with Con A between the high body burden sera and the control homologous sera were found to be statistically significant, differences between the high body burden sera and the low body burden sera were not, despite a trend toward higher values for the latter. The difference found between the sera of the radium cases, when compared with the control population, is similar to that found when lymphocytes from radium workers were stimulated.⁴ The choice of appropriate controls is still a matter for concern with regard to a possible healthy worker effect, which may apply to the laboratory staff used presently as controls. This problem could only be resolved by using another control population. Future experiments, involved with the examination of larger groups of both high- and low-body burden sera, would also be necessary in order to substantiate the significance of these preliminary findings.

References

1. J. D. Aub, R. D. Evans, L. H. Hemplemann, and H. S. Martland, The late effects of internally-deposited radioactive materials in man, *Medicine* 31, 221-329 (1952).
2. R. J. Hasterlik, C. E. Miller, and A. J. Finkel, Carcinoma of the mastoid and paranasal sinuses in radium-bearing patients, *Acta Unio Int. contra Cancrum* 20, 1194-1195 (1964).

3. R. E. Rowland, A. F. Stehney, and H. F. Lucas, Jr., Dose-response relationships for female radium dial workers, *Radiat. Res.* 76, 368-383 (1978).
4. C. S. Serio, C. B. Henning, R. E. Toohey, and E. L. Lloyd, Mitogenic stimulation of peripheral lymphocytes of radium workers, *Int. J. Radiat. Biol.*, in press.
5. E. S. Gilbert and S. Marks, Analysis of the mortality of workers in a nuclear facility, *Radiat. Res.* 79, 122-148 (1979).
6. J. A. Mannick, M. Constantian, D. Pardridge, I. Saporoschetz, and A. Badger, Improvements of phytohemagglutinin responsiveness of lymphocytes from cancer patients after washing in vitro, *Cancer Res.* 37, 3066 (1977).
7. G. Janossy and M. F. Greaves, Lymphocyte activation II. Discriminating stimulation of lymphocyte subpopulations by phytomitogens and heterologous antilymphocyte sera, *Clin. Exp. Immunol.* 10, 525-536 (1971).

ON THE CONCEPT OF DOSE, DOSE-RATE, AND SPECIFIC ENERGY

John H. Marshall

In analyzing the results of experiments, it is essential to separate theory and experiment. This separation is most secure when one uses parameters that have been defined operationally. An operational parameter is based solely on numerical readings taken in the course of that measurement. Parameters which invoke, for their calculation, the results of other experiments or assumptions based on other experiments, are not operational

In an injection experiment, the operationally-defined parameters¹ (parameters defined from what was done, not from what was assumed) are $\mu\text{Ci/kg}$ body weight, and dose rate as a function of time. In an irradiation of short duration, internal or external, dose is also an operational parameter. Integrated dose, when the dose rate is continuing, begins to inject an element of theory into a "measurement" of dose. Questions arise as to the identities of the cells for which the dose is calculated, which periods of dose rate are effective, etc.

Dose at the microscopic level (10 μm to 1 mm) introduces more theory, because one must assume, or derive from data, which cells are at risk. A complete microscopic distribution of dose which covers all possibly relevant locations, is again operational, but only at the expense of an overwhelming number of doses. At the microdosimetric level (0.1 to 1 μm), dose is called specific energy.² (At all levels, dose is absorbed energy per gram of tissue.) But where dose refers to a specific biological entity such as skeleton, or marrow, or bone, or liver, it can be quite operational. When the size of the volume for which dose is defined is reduced far below the macroscopic level, the concept of dose takes on different meanings, meanings which are less operational the smaller the site. It is nonsense to define dose as the limit of $\Delta E/\Delta M$ as the mass is reduced, as is often done.

Even specific energy, which has inspired good theory³ and many measurements, is not an operational parameter, because one must assume a site size or derive a site size by comparison with different data. Site sizes derived so far have usually little correspondence to structures within the cell. Specific energy is thus a measurement on a model of radiation toxicity, albeit an interesting model.

If one had measurements for all possibly relevant site sizes, specific energy would again become operational. But there is growing doubt that specific energy is relevant to a single mutation or to a single cancer initiation which probably are events at the nanometer level. Even the recent reformulation of Dual Theory⁴ has de-emphasized specific energy by de-emphasizing the original site model for which it has meaning. Thus, while an important quantity, specific energy should be applied to specific mechanisms with full knowledge that a model, the site, is implied.

References

1. P. W. Bridgman, The Logic of Modern Physics, Macmillan, New York (1946).
2. International Commission on Radiation Units and Measurements, Radiation Quantities and Units, ICRU Report 33, Washington (1980).
3. A. M. Keller and H. H. Rossi, The theory of dual radiation action, Current Topics in Radiation Research 8, 85-158 (1972).
4. A. M. Keller and H. H. Rossi, A generalized formulation of dual radiation action, Radiat. Res. 75, 471-488 (1978).

AN EMPIRICAL BAYES MODEL FOR ESTIMATING RADIUM INTAKE FROM EXPOSURE DURATION

L. Sanathanan and H. F. Lucas, Jr.

The effects on a population exposed to various levels of internal alpha radiation have been the subject of several studies. In particular, dose-response relationships involving an incidence of bone sarcomas and head carcinomas among a group of women who entered the U.S. radium dial-painting industry before 1930 have been analyzed. However, these analyses have excluded cases for whom measurements of radium burden could not be determined, thereby causing a possible bias in the results. A method for estimating the radium intake distribution among the unmeasured cases from data on duration is provided in this paper as a means of involving growth curves and stochastic growth parameters, and is illustrated with real data.

* Abstract from The American Statistical Association, Houston, Texas, 11-12 August 1980.

BREAST CANCER IN FEMALE RADIUM DIAL WORKERS FIRST EMPLOYED BEFORE 1930*

E. E. Adams and A. M. Brues

Female radium dial workers first employed before 1930 were analyzed for breast cancer mortality and incidence using methods and rate tables described by Monson and the Mantel-Haenszel summary chi-square test for significance. Of 1180 located women, 736 were measured to estimate radium intake. This measured group was analyzed for breast cancer mortality and incidence according to four possible risk factors: radium intake dose, duration of employment, age at first exposure, and parity. The located women had a mortality ratio of 1.51 ($p < 0.05$). The measured women showed a significant excess of breast cancer incidence and mortality only among those women with a radium intake of 50 μCi or greater. Although not significant, incidence and mortality ratios were slightly higher for nulliparous women.

* Abstract of paper accepted for publication in Journal of Occupational Medicine.

CODING, STORAGE, AND RETRIEVAL OF MEDICAL INFORMATION: EXPERIENCE WITH THE STANDARD NOMENCLATURE OF PATHOLOGY (SNOP)

A. M. Blues

Pathologic conditions indicated in the records of all radium cases in CHR are coded according to the SNOP classification and filed in computer memory for future reference and inter-correlations with other individual data. An additional field has been added to characterize the source of each item and its chronology. This provides an excellent index to the records. Records of about 3700 located cases have yielded a total of about 10,000 entries. The use of this data base in epidemiologic studies on radium contamination problems related to the heterogeneous sources of data.

The Center for Human Radiobiology has registered the names of over 5000 individuals exposed to internal contamination with ^{226}Ra and/or ^{228}Ra and about 3700 of these persons have been located. Measurement of radium content is available in about 10% of the located individuals, and estimates of past intake have been obtained for many additional individuals. A sub-group of dial planters who were exposed occupationally before the hazards of ingestion were fully appreciated, the degree of personal contamination dropped off sharply between 1935 and 1945. Since the earlier group of workers varied greatly in respect to their radium "burden," some stratification of the group is possible on the basis of internal radium dose. A second wave of dial workers, employed in the 1940's, has a much lower radium content and comprise an internal control which, however, is less satisfactory for secular reasons, being a generation younger.

Aside from acute and sub-hepatic effects and the "radiogenic" malignancies, which were recognized early (and which continue to appear in the surviving group of early dial workers) no other highly characteristic effect of radium contamination on mortality has been identified,¹ although a dose-related increase in breast cancer mortality has been noted.²

A number of coding systems is available for identifying and cataloging morbid states. The most widely used is the International Classification of Diseases (ICD) and its adaptation for use in this country (ICDA). These are

subject to periodic revision, and are used in classifying causes of death on death certificates. From these, comparison data on U.S. white females are available for determining expected and standardized mortality rates. This has been done for cause-specific mortality rates in a recent paper from the Center.³ In a study of survival times,¹ the radiogenic lesions alone were sufficient to account for the decrease in survival time and increase in tumor mortality rates in the early dial patients.

In 1973, a decision was made to code all pertinent pathologic conditions in the recorded population of the Center according to the Standard Nomenclature of Pathology (SNOP). This had been done by Sharpe⁴ in a monograph detailing clinical and autopsy findings in a series of cases in the New Jersey area. SNOP is much more detailed than other codes and has been worked out with a great deal of attention to precision and consistency.⁵ We have used a modified version dictated by our special requirements.⁶ In addition to four fields identifying conditions according to topography, morphology, etiology and function, each using four digits, a fifth field has been used to characterize the source of information in each case and its chronology. (The several categories of sources are defined in the footnote to Table 1.) The computer memory has been supplied with translations of numerical codes into medical language, and the natural history of a pathologic condition can be reconstructed by reading out a chronologically sorted series of SNOP entries. As of the present time, virtually all of the original radium files have been "SNOPED," and newly received documentary information is transcribed for storage before filing the original document. The individual patient records have yielded over 150,000 items, and one diligent and seasoned assistant can search and compile this material with minimal errors of transcription and interpretation (less than one per cent error).

As is well known to those who must rely on existing clinical records for research data, significant numbers of errors and misinterpretations exist, and final evaluation of a case depends on objective study of the original sources in the light of other information. For this reason the SNOP file can best be looked upon primarily as an index to the clinical record file.⁷ In this role the SNOP data base relieves the investigator of examining a large quantity of trivial or irrelevant data.

Table 1. Located radium cases; SNOP entries by source of information and by number of entries per case.

Entries per case	Number of cases within range	Number of entries	Percent of SNOP entries derived from various sources ^a									
			CD	AN	SH	XY	O	J	L	P	F	R
1-5	1018	2 652	56.3	—	0.4	0.2	1.5	1.5	0.5	35.4	2.6	2.9
6-10	297	2 299	21.0	0.3	0.7	1.7	6.4	1.0	2.2	55.5	7.7	4.1
1-20	433	6 651	6.5	0.9	1.6	4.9	17.0	0.4	1.2	53.9	7.3	6.1
21-30	322	8 213	3.7	1.2	2.8	7.7	20.7	0.9	1.8	49.6	5.8	6.4
31-40	282	9 923	2.5	2.6	2.4	9.8	22.8	0.7	1.2	47.5	5.0	6.0
41-50	238	10 814	2.3	3.8	2.4	11.8	25.3	—	0.9	41.8	3.8	6.8
51-75	439	27 428	1.3	5.0	2.5	14.2	26.0	—	1.1	39.3	3.6	6.6
76-100	253	21 839	1.1	4.6	2.0	14.4	28.5	—	1.4	38.9	3.1	6.0
101-150	241	29 415	0.9	5.2	1.8	17.9	27.5	—	1.3	34.4	2.4	8.8
151-200	83	14 315	0.5	3.7	1.6	25.7	27.3	—	1.1	30.5	1.7	7.7
> 200	85	26 636	0.4	4.5	1.6	32.1	26.3	—	1.4	23.0	1.2	9.2
Total number of entries	(3691)	157 185	4 237	6 335	3 120	26 822	39 584	233	1 986	58 438	5 012	11 418

^aSources of information: CD = death certificate; AN = autopsy data; SH = microscopic pathology; O = direct observation; XY = x-ray plates; J = journal reference; L = letter from M.D., etc.; P = past history (by patient); F = family history (by patient); R = abstract of clinical record.

It must not be thought that the above procedures create a homogeneous file of data which permit instant intercomparison. After all, the basic records prior to 1970 were compiled by different groups of investigators and were made up from a variety of sources of different breadth and content. A hastily written preoperative note will yield a much different spectrum of data from what is found in a meticulously composed student history. Where the sources of information are numerous, a good deal of redundancy occurs.

Given the heterogeneity of the sources of the data, a serious problem exists in the bias introduced by added detail. A carefully autopsied case is comparable only with an equally carefully autopsied case with respect to a determination of occult thyroid tumor, for example. It is therefore of considerable importance, in the use of matched internal controls, to take account of the source of information.

To provide some light on this point we have sorted the located individuals in the study on the basis of their respective numbers of SNOP citations. The number of entries from each source was separately counted for each case (Table 1). The accompanying table shows the results of counting the SNOP citations according to source in those individuals with specified total numbers of entries.

The totals indicated in Table 1 illustrate the mass of information derived from each of the several sources in all of the located cases in the radium study. A first visit by a patient for examination and radium measurement yields from 25 to 100 entries. The largest number of citations is yielded by the personal history (P) and the examining physician's observations (O), and the roentgenographic studies (XY) account for most of the residue. Personal and family history and observations comprise a rather constant share of the information, and radiologic data (probably related to special interest in skeletal effects) show an increasing proportion in patients who are examined more frequently. Death certificate data predominate in those cases with minimal information.

In summary, SNOP provides a quite satisfactory vehicle for searching files for listings of particular conditions and also gives promise of providing good chronological summaries of complicated cases. Its value for control purposes is thus far untested and needs further study. A major advantage lies in the fact

that a large body of clinical information can readily be brought into juxtaposition with the other recorded materials in this studied population. This includes a number of additional medically related files; clinical laboratory data, skeletal x-ray scores, coded death certificate data, some studies of fertility, and perhaps other material not yet investigated but reposing in the records file.

References

1. A. F. Stehney, H. F. Lucas, Jr., and R. E. Rowland, Survival times of women radium dial workers first exposed before 1930, Proc. IAEA Int. Symp. on the Late Biological Effects of Ionizing Radiation, Vienna, March 13-17, 1978.
2. E. E. Adams and A. M. Brues, Breast cancer in female radium dial workers first employed before 1930, J. Occup. Med. 22, 583-587 (1980).
3. A. P. Polednak, A. F. Stehney, and R. E. Rowland, Mortality among women first employed before 1930 in the U.S. radium dial-painting industry, Am. J. Epidemiol. 107, 179-195 (1978).
4. W. D. Sharpe, Chronic radium intoxication: Clinical and autopsy findings in long-term New Jersey survivors, Environ. Res. 8, 243-383 (1974).
5. Committee on Nomenclature and Classification of Disease, Systematized Nomenclature of the Pathology, College of American Pathologists, Chicago (1965).
6. M. S. Littman, H. F. Lucas, Jr., W. D. Sharpe, and A. F. Stehney, Radiation epidemiologic surveillance using the systematized nomenclature of pathology, J. Clin. Comput. 3, 191-197 (1973).
7. A. M. Brues, Malignancies in patients contaminated with radium, The Health Effects of Plutonium and Radium, Proc. Symp. Sun Valley, Idaho, 6-9 October 1975, W.S.S. Jee, Ed., The J. W. Press, Salt Lake City, Utah, pp. 779-791 (1976).

HEALTH STATUS AND BODY RADIOACTIVITY OF FORMER THORIUM WORKERS*

A. F. Stehney, A. P. Polednak,[†] J. Rundo, A. M. Brues, H. F. Lucas, Jr.,
B. C. Patten, and R. E. Rowland

This is a progress report of a study of the health effects of industrial exposure to thorium. Included is the three-year period from inception (1976) to September, 1979. The study population comprises the former employees of a thorium mill located in West Chicago, Illinois. Thorium and rare earth chemicals were extracted and purified from monazite ores at this plant from 1932 to 1973, and thorium mantles, for gas lamps, were manufactured from 1936 to 1947.

The objectives of the study are: (1) to assess possible health effects of employment in the thorium milling industry by comparison of mortality and morbidity characteristics of former thorium workers with those of suitable general populations; (2) to examine disease outcomes by estimated exposure levels of thorium and thoron daughter products for possible radiation-related effects; and (3) to determine the body distribution of inhaled thorium (and daughters) and rare earths in humans by radioactivity measurements in vivo and by analysis of autopsy samples. The principal end points for investigation are respiratory disease and cancers of lung, liver, bone, and bone marrow.

The West Chicago plant was operated by the Lindsay Light Company and its corporate successors, which finally included the Kerr-McGee Chemical Corporation (1967). Company records dating back to 1925 identified about 4600 individuals (80% men) employed at plant sites in Chicago (up to 1936) and West Chicago (1932-1973) and at a mantle factory in Morris, Illinois (1947-1953). Since records before 1940 and for the Morris plant were incomplete, the study was limited to 3222 men and 714 women who worked at the West Chicago plant after 1939. Social Security numbers, job classifications, and work dates have been found for almost

* Executive Summary from the interim report of the same title, NUREG/CR-1420, ANL-80-37 (January 1980).

[†] Present address: Cancer Control Bureau, State Department of Health, Albany, New York 12237.

all of these former employees.

An industrial hygiene survey of the plant in 1952 showed that gamma-ray levels of 0.5 to 5 mR per hour were common in locations where thorium was processed or stored and that the levels of airborne thorium and thoron daughter products were of the order of present day maximum permissible concentrations. Estimates of radiation exposures during 1956-1973 are being compiled from inspection reports of the U.S. Atomic Energy Commission, records of personal dosimeter readings, and company records of radiation and radioactivity measurements. Filter samples that were collected while the plant was in operation are being analyzed for particle sizes and chemical composition of airborne materials.

Overall and cause-specific mortality was studied in a cohort of 3039 male thorium workers on the basis of deaths reported by the Social Security Administration and causes on death certificates. Comparisons were made with sex-, age-, time-, and cause-specific mortality rates for U.S. white males. In the total cohort, there were 511 observed deaths and 486.8 expected, and differences between observed and expected numbers were significant at the 95% confidence level only for deaths from diseases of the circulatory system (205 observed vs. 249.5 expected) and from motor vehicle accidents (38 observed vs. 23.2 expected). However, notably higher than expected numbers of deaths were observed for respiratory diseases (33 vs. 25.2) and cancers of the lung (31 vs. 21.6), pancreas (9 vs. 4.5), and rectum (6 vs. 3.2).

In general, the mortality differences were not strongly associated with job type or length of employment, but excess deaths from cancer of the pancreas were significantly greater among men employed at least one year than among shorter-term workers. Data on a small sample of the study population indicated a higher proportion of cigarette smokers than among U.S. males, and this could explain at least part of the excess mortality from lung cancer and respiratory diseases.

Medical examinations and in vivo measurements of body radioactivity are being done on a subpopulation of 592 male employees who have worked a year or more in job classifications involving probable exposure to thorium. By measurement of radioactive thoron-220 in exhaled breath, higher than background

amounts of radium-224 were found in 131 of 194 men who have been examined (range 2 to 667 pCi). By gamma-ray spectrometry, measurable amounts of bismuth-212 in the thorax were found in 55 of the men (range 0.2 to 3 nCi). Comparisons with measured amounts of thorium-232 and thorium-228 in autopsy samples are needed for interpretation of these in vivo measurements of thorium daughter products.

RADIOACTIVITY IN FORMER WORKERS AT A THORIUM REFINERY

J. Rundo, D. R. Brewster, M. A. Essling, and J. Y. Sha

As part of an epidemiological study of the possible late biological effects of thorium, measurements have been made of radioactivity attributable to thorium daughters in almost 200 men who had worked in a thorium refinery which closed in 1973. For external gamma-ray measurements statistically significant results ($> 2\sigma$) were obtained in 55 of these, with three showing more than 2 nCi ^{212}Bi in the thorax. For measurements of daughters of exhaled thoron, statistically significant results were obtained in almost every case, but for 63 subjects the values of < 2 pCi of freely emanating ^{224}Ra at the mouth could not be attributed unequivocally to thorium acquired occupationally; 131 men exceeded this lower limit and four had more than 200 pCi of ^{224}Ra . The mean ratio of emanating ^{224}Ra to retained ^{212}Bi was 101 pCi/nCi, with individual values ranging from 11 to 581. The problem of interpreting the data in terms of the actual amounts of thorium in the thorax is discussed briefly.

* Abstract of a paper presented at the Workshop on Measurements and Interpretation of Actinide Accumulation by Man, Snowbird, UT, October 15-17, 1979.

SOME DETERMINATIONS AT ARGONNE NATIONAL LABORATORY OF RADON IN HOUSES*

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

The chance observation of a radon concentration of 26 pCi per litre in the bedroom air of a frame house has led to the discovery that such levels can arise as a result of emanation of radon from bare soil in a "crawl space" under part or all of the house. They are not a consequence of "technologically enhanced" radioactivity in building materials. In a total of 23 houses investigated, the air of ten showed concentrations of radon of 5 pCi per litre or more; of these six had radon concentrations of 10 pCi per litre or more. It should also be mentioned that a concentration as low as 0.2 pCi per litre was observed in one of these houses. The presence or absence of plastic vapor barriers seems to be one important factor in determining the level, but certainly not the only one.

During July and August 1978, an Environmental Working Level Monitor that was returned to A.N.L. for repair and testing was used to determine the concentrations of radon daughters during a total of three periods, each of about 100 hours' duration in two of the houses. Mean values of 0.007 WL and 0.023 WL were observed in the first, and of 0.008 WL for the second.

On the basis of such limited data it is obviously not possible to generalize on the average concentration of radon daughters in these houses. However, it is conceivable that the average concentration might be in the region of 0.01 WL; exposure at this level for a year (say of a small child) would result in a radiation dose equivalent of 2.6 rem to the bronchial epithelium (derived from 5 mrem per WLM, for a 170-hour working month).

* Published in Radon in Buildings, Proc. Roundtable Discussion, National Bureau of Standards, June 15, 1979, NBS Special Publication 581, June 1980

ESTIMATION OF EXPOSURE TO ^{222}Rn FROM THE EXCRETION RATES OF ^{210}Pb

R. B. Holtzman and J. Rundo

A model is proposed with which estimates of exposure to ^{222}Rn and its daughter products may be made from urinary excretion rates of ^{210}Pb . It is assumed that 20% of all the ^{210}Pb inhaled (as short-lived precursors or as ^{210}Pb itself) reaches the blood and that 50% of the endogenous excretion is through the urine. For an exposure of one Working Level (WL), the model predicts a urinary excretion rate (in excess of normal) in the range 1.8 to 5.4 pCi day⁻¹, if intake and excretion are in equilibrium. The estimates from the model are compared with the results of measurements on a subject residing in a house with high levels of radon. Whole body radioactivity and excretion data were consistent with the model, but the estimates of exposure (WL) were higher than those measured with an Environmental Working Level Monitor.

A major problem in studying exposure of man to ^{222}Rn and to its short-lived decay products is estimation of the integrated exposure to an individual over a long period of time. Such studies are important because exposure to very high levels of ^{222}Rn and its short-lived decay products is known to cause lung cancer in uranium miners,¹ and consequently it is desirable to estimate the exposure to large population groups exposed to much lower but greater than average levels of radon.

The long-lived decay product of the ^{222}Rn series, ^{210}Pb (22 yr), has been proposed and studied as a retrospective indicator of such exposures,²⁻⁴ but the results are uncertain because of the complexities of ^{210}Pb metabolism and the uncertainties in the exposures. The concentrations of this nuclide in the bones of deceased uranium miners have been correlated with dose experienced by the miners,²⁻⁴ expressed in units of Working Level Months (WLM).^{*} Since it is difficult to determine the ^{210}Pb content of bone from external measurements,

* The unit of Working Level (WL) is defined as any combination of the concentrations of short-lived ^{222}Rn daughter products that on decay would release 1.3×10^5 MeV of alpha energy in 1 liter. This is equivalent to that released by the daughter products initially in radioactive equilibrium with 100 pCi/L of ^{222}Rn . The Working Level Month is then the exposure to one WL for a working month of 170 hr.

especially for low exposures, the concentrations in bone have been correlated with those in blood³ and with the urinary excretion rates of ^{210}Pb ² in order to estimate exposures in vivo.

Presented here is a somewhat different approach in which a metabolic model is proposed from which estimates of exposure to radon and its daughter products may be made from urinary excretion rates of ^{210}Pb . The predictions of this model are then compared with data from a subject exposed to higher levels of radon than are usually thought to be normal.

The Model

The relevant parameters are set out in Table 1. The only entry that calls for comment is the concentration of daughter atoms in air for 1 WL. In Table 2 are set out the results of calculations of this quantity for 1 WL in air of different effective ages. In making these calculations, we used the equations in Appendix A and Ref. 4 that give the radon daughter concentrations relative to those of radon. It was assumed that the system is in a steady state, that the various nuclides are removed from the air by some mechanism, such as attachment to surfaces, and that the rates of removal are the same for all nuclides.

It is seen that except for very young air, the concentration of daughter atoms per WL does not vary much with age; in adopting a rounded value of $15,500 \text{ atoms L}^{-1} \text{ WL}^{-1}$ we have chosen a concentration that corresponds closely to a value for F of 0.5, which is commonly thought to be a typical value.⁵ Note that even for the absurd situation of RaA in equilibrium with radon and no RaB or RaC, the concentration of atoms for 1 WL would still be 9500 L^{-1} .

We now use the data in Table 1 to calculate the daily intake of ^{210}Pb , of which three sources are considered: (a) production in vivo from inhaled short-lived radon daughters, (b) production in vivo from short-lived daughters supported by radon dissolved in body fats and fluids, and (c) inhalation of ^{210}Pb produced in the air. The first of these is essentially constant (per unit WL), the second increases with decreasing age of the air, while the third decreases with decreasing age.

(a) The intake of ^{210}Pb from short-lived Rn daughters inhaled at a constant

Table 1. Parameters used in calculations for the model using ^{210}Pb to estimate exposure to ^{222}Rn and its daughters.

<u>Metabolic parameters</u>	
Body mass	70 kg ^a
Breathing rate	10 m ³ /day, 7 L/min ^a
Effective retention of aerosols in lung (0.50 deposited in respiratory tract; 1/3 of this is absorbed into the blood and 2/3 are removed by ciliary action to the gut where 0.08 enters the blood).	0.20 ^b
Urinary-to-fecal excretion ratio	1.0 ^c
Concentration of Rn in body relative to that in air	2.0 [L(air)/kg(body)] ^d
<u>Physical parameters</u>	
Approximate number of atoms of short-lived radon daughters in air	15500 (L-WL) ^{-1e}
Half lives:	
^{218}Po (RaA)	3.05 min
^{214}Pb (RaB)	26.8 min
^{214}Bi (RaC)	19.7 min
^{210}Pb (RaD)	22 yr (1.16×10^7 min)

^a Ref. 6.

^b Refs. 7 and 8.

^c Unpublished data of R. B. Holtzman.

^d Derived from data in Ref. 9.

^e See Table 2 and text.

Table 2. Concentrations of short-lived radon daughters and of radon for air of different ages at one WL, and the corresponding equilibrium factors.

Effective age of air, min	Activity ratios				Concentration of daughter atoms, L ⁻¹	Concentration of radon, pCi L ⁻¹	Equilibrium Factor, ^a F
	Rn	RaA	RaB	RaC			
∞	1	1	1	1	16150	100	1.0
60	1	0.95	0.66	0.50	15760	160	0.61
30	1	0.91	0.48	0.29	15370	225	0.44
15	1	0.83	0.30	0.13	14710	350	0.28
5	1	0.62	0.098	0.020	13010	830	0.12

$$^a F = \frac{100 \text{ WL}}{\text{pCi Rn L}^{-1}} .$$

concentration of 1 WL is then

$$10 \times 10^3 \text{ L/day} \times 15,500 \text{ atoms/L} \times 0.20 = 3.1 \times 10^7 \text{ atoms/day} = 0.84 \text{ pCi/day} .$$

(b) For the ²¹⁰Pb formed in the body from ²²²Rn dissolved in body fluids, and with its daughters in secular equilibrium, a 70-kg man (Reference Man)⁶ contains 14,000 pCi ²²²Rn, and 1.21 pCi ²¹⁰Pb are produced daily from radon inhaled at a constant concentration of 100 pCi/L.

(c) Estimation of the magnitude of the contribution from ²¹⁰Pb in the atmosphere is a more complex problem than were those for the other sources. As derived in Appendix A, the ²¹⁰Pb concentration in air, A_D, as a function of Working Level, W, is

$$A_D = \frac{0.450 \lambda_A \lambda_B \lambda_C \lambda_D W}{E_A P + E_{BC} Q} \text{ pCi/L} , \quad (1)$$

where

$$P = (\lambda_B + \lambda_{BR})(\lambda_C + \lambda_{CR})(\lambda_D + \lambda_{DR}) ,$$

$$Q = \lambda_A(\lambda_C + \lambda_{CR})(\lambda_D + \lambda_{DR}) + \lambda_A \lambda_B(\lambda_D + \lambda_{DR}) ,$$

λ_A , λ_B , λ_C , and λ_D are the physical decay constants of the respective nuclides ^{218}Po , ^{214}Pb , ^{214}Bi , and ^{210}Pb ,

λ_{BR} , λ_{CR} , and λ_{DR} are the inverses of the mean residence times of these nuclides,

$E_A = 13.68 \text{ MeV}$, the alpha decay energy to ^{210}Pb per atom of ^{218}Po (RaA), and

$E_{BC} = 7.68 \text{ MeV}$, the alpha decay energy to ^{210}Pb per atom of ^{214}Pb (RaB) or of ^{214}Bi (RaC).

If it is assumed that the residence times of the various nuclides are the same, ($\lambda_{BR} = \lambda_{CR} = \lambda_{DR} = \lambda_R$), then for a residence half-time of 1 hr and $W = 1 \text{ WL}$,

$$A_D = 4.15 \cdot 10^{-4} \text{ pCi L}^{-1} \quad (2)$$

This concentration is about 20 to 30 times the normal levels of ^{210}Pb in air. With a breathing rate of $10 \text{ m}^3/\text{day}$, and with 20% entering the blood (from Table 1), the uptake by blood ranges from 0.014 to 0.84 pCi/day for atmospheric residence (half) times of 5 to 60 min (Table 3). Thus, unless the atmospheric residence time of the ^{210}Pb is long, the contribution of this nuclide to the intake is small, amounting to less than 25% of the total for a residence time of 60 min.

The predictions of the model for the daily intake and excretion of ^{210}Pb are summarized in Table 3 for three values of the equilibrium factor, F^* . The value used for the retention of the daughters in the lung is not critical at the lower values of F , since the intake depends mainly on the concentration of ^{222}Rn . Thus, for values for F of 0.61, 0.44, and 0.12, if the fraction of inhaled radioactive aerosol that reaches the blood is 0.4 (instead of 0.2), the total ^{210}Pb intake would be increased by 46, 30, and 8%, respectively.

The last column shows that a total of 11 pCi/day per WL could be available for excretion. While some fraction of the daily intake of ^{210}Pb is stored in bone and other compartments, a portion of the ^{210}Pb stored previously is also excreted.

$$^* F = \frac{WL \cdot 100}{^{222}\text{Rn conc. (pCi/L)}} \quad .$$

Table 3. Summary of data on intake and elimination of ^{210}Pb for exposure to 1 WL.

<u>Conditions</u>			
Effective age of air, min	60	30	5
Equilibrium factor, F^a	0.61	0.44	0.12
Corresponding concentration of radon in air, pCi L^{-1}	160	225	830
<u>Intake of ^{210}Pb</u>			
From short-lived Rn daughters (retention=0.2), pCi d^{-1}	0.84	0.84	0.84
From ^{222}Rn in body fluids, pCi d^{-1}	1.94	2.70	10.04
From ^{210}Pb in air, pCi d^{-1}	0.84	0.34	0.014
Totals	3.62	3.88	10.89
<u>Excretion of ^{210}Pb</u>			
Total excretion, pCi d^{-1}	3.62	3.88	10.89
Urinary excretion, pCi d^{-1} (U/F=1.0)	1.81	1.94	5.44
Normal urinary excretion, pCi d^{-1b}	0.2	0.2	0.2
Total urinary excretion, pCi d^{-1}	2.0	2.1	5.6

^a See Table 2.

^b May range from 0.1 to 0.4 pCi d^{-1} .

The net fraction stored will be disregarded (see Appendix B). The amount excreted in the urine is then 5.4 pCi/day in excess of normal environmental levels of about 0.2 pCi/day, since endogenous excretion of ^{210}Pb is assumed to be divided equally between urine and feces (Table 1).

Discussion

The excretion rates of ^{210}Pb estimated for the model in Table 3 could vary appreciably, depending on the validity of the assumptions in Table 1. A reduction in the ratio of radon concentrations, body:air, to 1.0 L/kg would decrease the available ^{210}Pb from 10.9 to 5.9 pCi/day. On the other hand, older air, e.g., with a 2-hr residence time, would increase the ^{210}Pb by 1 pCi/day over the value for a 1-hr residence time.

As shown in Table 2, changes in the RaA:RaB:RaC ratios have little effect on the concentrations of atoms of short-lived daughter products per WL, except for very young air. In some experiments in a large isolated room with a high filtration rate, we have observed a value for F of about 0.1, a condition similar to that in the last lines in Table 2.

The predicted excretion rates may be compared to those of an extensively studied case, 50-026, who lives in a house with elevated levels of radon (3 to 30 pCi/L).^{9,10} The mean urinary excretion rate from two 24-hr samples from this subject was 0.75 pCi ^{210}Pb /day (0.5 and 1.0), and the mean excess above normal environmental levels was thus about 0.55 pCi d⁻¹, but with a possible range of 0.35 to 0.65 pCi d⁻¹. From the range of estimates of the model of 1.8 to 5.4 pCi d⁻¹ excess excreted in the urine, the exposure appears to have been in the range 0.06 to 0.36 WL. Because we have some data on conditions in the house, we can be more specific.

Measurements at various times of the concentrations of both radon and its short-lived daughters in the house suggested the low value of about 0.1 for F.¹⁰ This indicates that the exposure was at the lower end of the range, i.e., 0.06 WL. This is higher than the values determined with an Environmental Working Level Monitor (EWLM),¹¹ which ranged from 0.0078 to 0.024 WL (mean values of three sets of measurements),¹⁰ by a factor of two to eight.

At 94.5 kg, subject 50-026 weighed substantially more than the average for her height (1.58 m), and the excess was adipose tissue. Radon is at least an order of magnitude more soluble in fat than in water (i.e., aqueous tissues) so the value of 2 L kg⁻¹ for the concentration of radon in the body relative to that in air may be too low. A value of 5 L kg⁻¹ might be entirely reasonable

for this subject, and this would yield a calculated urinary excretion rate for ^{210}Pb of $13 \text{ pCi d}^{-1} \text{ WL}^{-1}$ in excess of normal. If this were the case, the observed excess of 0.55 pCi d^{-1} would correspond to an exposure (with $F = 0.1$) of 0.042 WL , very close to the upper limit of the results obtained with the EWLM. However, the calculated exposure must be adjusted by the occupancy factor, which the subject estimated to be 0.50. This has the effect of increasing the calculated exposure by a factor of 2.0. Perhaps even the value of 5 L kg^{-1} suggested above is too low.

On the other hand, there was reasonable agreement between the production rate of ^{210}Pb in vivo and the observed excretion rate. It was estimated that the subject maintained a total of about 18 nCi RaC while in the house, of which 12.7 nCi were in the lung (unsupported) and 5.1 nCi were supported by radon dissolved in body fats and fluids.⁹ The production rate of ^{210}Pb from 18 nCi RaC is 1.56 pCi d^{-1} , so up to 0.78 pCi could be excreted daily in the urine, but reduced by the occupancy factor of 0.50. We then have a predicted urinary excretion rate of 0.39 pCi d^{-1} , essentially the value of 0.35 pCi d^{-1} noted above for the lower end of the range of the excess ^{210}Pb in the urine. It should be noted that there is some uncertainty in the value for the equilibrium body content of 18 nCi RaC . This is because some of the unsupported daughters may have been distributed throughout the body as a legacy of radon associated with pools that cleared rapidly via the exhaled breath after the subject left her house. This RaC would have been detected with a lower efficiency (by a factor of 1.6) than RaC in the lung. If none of the unsupported RaC were in the lung, the equilibrium content would have been $(12.7 \times 1.6) + 5.1 = 25.4 \text{ nCi}$, and the predicted urinary excretion rate of ^{210}Pb would be increased from 0.39 pCi d^{-1} to 0.55 pCi d^{-1} , but this was clearly not the case. In any event, the uncertainty in the body content is small and it does not have a major effect on the calculated excretion rate of ^{210}Pb .

It should be noted that the calculation above of the urinary excretion rate of ^{210}Pb was made with the implicit assumption that all the RaC in the body or the ^{210}Pb produced from it was available to the blood. In the model (Table 1) only about 0.4 of the lung deposit was assumed to reach the blood. The

reasonable agreement between calculated and observed urinary excretion rates suggests that the factor of 0.4 is too low.

It must be remembered that the test of the predictions of the model is based on very limited data (one subject, two 24-hr urine samples analyzed). Nevertheless, we believe that the model takes into account most (if not all) of the factors involved in the use of ^{210}Pb as an indicator of exposure to radon and its short-lived daughters. The agreement between model and data was not good, even with the use of what appeared to be the correct value for F and an appropriate value for the concentration ratio, body:air, although the excretion rate calculated from the results of body radioactivity measurement agreed with that observed, this was not dependent on any assumption in, or predictions of, the model. What the model does show is that ^{210}Pb produced from the radon dissolved in body fats and fluids is the major contributor to the excretion rate, regardless of the age of the air. It is also clear that one should use parameters that apply in an individual case, when they are known, rather than average values. More data are needed on the ^{210}Pb excretion rate for both this and other subjects, on the accuracy of the EWLM, and on the deposition and metabolism of radon and its short-lived daughters inhaled in houses or in other locations where persons are exposed to elevated levels.

APPENDIX A: Calculation of Radon Daughter Concentrations in the Atmosphere as a Function of Residence Time of the Particles

The radon daughter concentrations present in the atmosphere can be derived from data on the radon concentrations and the residence times of the nuclides in the atmosphere (which determine the value of F). Given these parameters, the radon concentrations as well as those of ^{210}Pb may be calculated from Working Level values.

In this derivation the system is assumed to be in a steady state, i.e., the concentration of each nuclide, A_i , is constant with time, and A_i is greater than or equal to that for its immediate successor, i.e.,

$$A_{i+1} \leq A_i . \quad (\text{A1})$$

Then,

$$A_{i+1} = \frac{\lambda_{i+1}}{\lambda_{i+1} + \lambda_{(i+1)R}} A_i \quad (A2)$$

where A_i is the concentration (pCi L^{-1}) of the precursor with the subscripts Rn, A, B, C, and D referring to the respective nuclides, ^{222}Rn , ^{218}Po , ^{214}Pb , ^{214}Bi , and ^{210}Pb , λ_i is the respective decay constant, and $\lambda_{(i+1)R}$ is the removal constant or inverse mean residence time of the respective nuclide in the air.

The latter value may be determined from the ratio of the measured concentrations A_i and A_{i+1} .

The activities of the daughter products are then,

$$A_A = \frac{\lambda_A}{\lambda_A + \lambda_{AR}} A_{\text{Rn}} \text{ pCi L}^{-1} \quad (A3)$$

$$A_B = \frac{\lambda_B}{\lambda_B + \lambda_{BR}} A_A = \frac{\lambda_A \lambda_B}{(\lambda_A + \lambda_{AR})(\lambda_B + \lambda_{BR})} A_{\text{Rn}} \text{ pCi L}^{-1} \quad (A3a)$$

$$A_C = \frac{\lambda_C}{\lambda_C + \lambda_{CR}} A_B = \frac{\lambda_A \lambda_B \lambda_C}{(\lambda_A + \lambda_{AR})(\lambda_B + \lambda_{BR})(\lambda_C + \lambda_{CR})} A_{\text{Rn}} \text{ pCi L}^{-1} \quad (A3b)$$

and

$$A_D = \frac{\lambda_A \lambda_B \lambda_C \lambda_D A_{\text{Rn}}}{(\lambda_A + \lambda_{AR})(\lambda_B + \lambda_{BR})(\lambda_C + \lambda_{CR})(\lambda_D + \lambda_{DR})} \text{ pCi L}^{-1} \quad (A4)$$

The Working Level, W, is calculated from the concentrations of the various nuclides

$$\begin{aligned} W &= n_A E_A + n_B E_{BC} + n_C E_{BC} \\ &= 2.22 A_{\text{Rn}} \left[\frac{E_A}{\lambda_A + \lambda_{AR}} + \frac{\lambda_A E_{BC}}{(\lambda_A + \lambda_{AR})(\lambda_B + \lambda_{BR})} \right. \\ &\quad \left. + \frac{\lambda_A \lambda_B E_{BC}}{(\lambda_A + \lambda_{AR})(\lambda_B + \lambda_{BR})(\lambda_C + \lambda_{CR})} \right] \quad (A5) \end{aligned}$$

where n_i is the concentration of atoms of nuclide i , E_A and E_{BC} are the alpha decay energies of the respective nuclides, namely $E_A = 13.68 \text{ MeV}$ for $^{218}\text{Po}(\text{RaA})$

and $E_{BC} = 7.68 \text{ MeV}$ for $^{214}\text{Pb}(\text{RaB})$ and $^{214}\text{Bi}(\text{RaC})$.

Finally, by combining Eqs. (4) and (5), one may calculate the amount of ^{210}Pb present in the atmosphere from the known value of the Working Level,

$$A_D = \frac{0.450 \cdot \lambda_A \lambda_B \lambda_C \lambda_D^W}{E_A P + E_{BC} Q} \text{ pCi/L} , \quad (\text{A6})$$

where

$$P = (\lambda_B + \lambda_{BR})(\lambda_C + \lambda_{CR})(\lambda_D + \lambda_{DR}) ,$$

$$Q = \lambda_A (\lambda_C + \lambda_{CR})(\lambda_D + \lambda_{DR}) + \lambda_A \lambda_B (\lambda_D + \lambda_{DR}) .$$

The value of the removal constant for ^{210}Pb , λ_{DR} , is not known, but with little error it can probably be set equal to λ_{CR} . To simplify the calculations further, we set $\lambda_{iR} = \lambda_R$, since all of the nuclides are formed from decay of a solid radionuclide, probably attached to a particle, except for the ^{218}Po formed by decay of the radon gas. The latter decay product is formed unattached to an aerosol and consequently it has a higher probability of reaching surfaces than do ^{214}Pb and ^{214}Bi . However, because λ_{AR} does not appear in Eq. (6), this is not significant in estimating the amount of ^{210}Pb formed.

APPENDIX B: Retention and Excretion of ^{210}Pb during and after Chronic Exposure

Let the retention at any time t after a single intake of q units of ^{210}Pb be R , where

$$R(t) = qf(t) . \quad (\text{B1})$$

Then the retention at the end of an exposure to q units day^{-1} for T days is

$$R_T = q \int_0^T f(\tau) d\tau \quad (\text{B2})$$

and the retention at t days after the end of the exposure is

$$R_q(t, T) = q \int_0^T R(t' - \tau) d\tau = q \int_0^T R(t + T - \tau) d\tau , \quad (\text{B3})$$

where t' is the time from the beginning of the exposure and $t = t' - T$.

The excretion rate at time t is then

$$-\frac{dR_q(t, T)}{dt} = q \frac{d}{dt} \left[\int_0^T R(t + T - \tau) d\tau \right] . \quad (B4)$$

For the modified power function of Norris et al.,¹²

$$R(t) = q \left(\frac{t + \epsilon}{T + \epsilon} \right)^{-b} , \quad (B5)$$

integration gives the retention after continuous intake of q units day^{-1} ,

$$R_q = q \int_0^T \left(\frac{t + \epsilon}{T + \epsilon} \right)^{-b} dt = q \frac{\epsilon}{1-b} \left[(T + \epsilon)^{1-b} - \epsilon^{1-b} \right] , \quad (B6)$$

and the excretion rate is

$$-\frac{dR_q(t, T)}{dt} = q \frac{b}{1-b} \left[(t + \epsilon)^{-b} - (t + T + \epsilon)^{-b} \right] . \quad (B7)$$

Similar arguments apply to a retention function expressed as a sum of n exponential terms in which λ_i is the decay constant of the i -th compartment. Integration of the retention equation for a single intake of q units,

$$R(t) = q \sum_{i=1}^n A_i e^{-\lambda_i t} , \quad (B8)$$

where $\sum A_i = 1$, gives the retention at the end of an exposure to q units day^{-1} for T days:

$$R_q(T) = q \left[\sum_{i=1}^n \frac{A_i}{\lambda_i} (1 - e^{-\lambda_i T}) \right] ,$$

and the elimination rate at t days after the end of the exposure is

$$-\frac{dR_q(t, T)}{dt} = q \left[\sum_i A_i (1 - e^{-\lambda_i T}) e^{-\lambda_i t} \right] . \quad (B9)$$

The problem reduces to one of identification of a retention function for ^{210}Pb in man which is reliable for long times. Models which are based on experimental observations have been proposed for animals,¹³⁻¹⁶ but the necessary data

Table B1. Predicted retentions of ^{210}Pb after continuous exposures for 1000 and 3600 days, for various models.

Species	Retention function	Retention in units of the daily intake after exposure for	
		1000 days	3600 days
Man ¹⁷	Sum of 5 exponentials	305	670
Dog ¹⁴	Sum of 4 exponentials	228	500
	Power function ^a	219	540
Baboon ¹³	Power function ^a	135	340

^aIt is assumed that $\epsilon = 2$ (based on the data from Ref. 14), and that $b = 0.3$ for dogs¹⁴ and $b = 0.4$ for baboons.¹³

Table B2. Predicted excretion rates of ^{210}Pb after a continuous exposure for 3600 days.

Species	Retention function	Daily excretion rate relative to daily intake		Ratio of rates, E_3/E_0
		At end of exposure [$E_0(t=0)$]	3 days after end of exposure [$E_3(t=3)$]	
Man ¹⁷	Sum of 5 exponentials	0.84	0.72	0.86
Dog ¹⁴	Sum of 4 exponentials	0.85	0.80	0.84
	Power function ^a	0.89	0.65	0.73
Baboon ¹³	Power function ^a	0.95	0.62	0.65
Means		0.91 ± 0.05	0.70 ± 0.08	0.77 ± 0.10

^aIt is assumed that $\epsilon = 2$ (based on the data from Ref. 14), and that $b = 0.3$ for dogs¹⁴ and $b = 0.4$ for baboons.¹³

are not available for man. Bernard¹⁷ has proposed a multi-exponential model, the predictions of which are compared to those obtained for animals in Tables B1 and B2. In the first of these, the retentions at the end of chronic exposures for 1000 days and 3600 days are shown, in units of the daily intake. The choice of these times was dictated (a) by the time that subject 50-026 had lived in her present house (1000 days at the time the urine samples were collected), and (b) by the exposure periods (average 9 years, range 5 to 14 years) of a group of subjects to whom the model is applied, as described in the next report. In this case, urine collections were made 3 days after the end of the exposure, and in Table B2, the excretion rates at the end of a 3600-day exposure and 3 days later are presented for the various models.

References

1. V. E. Archer, J. D. Gillam, and J. K. Waggoner, Respiratory disease mortality among uranium workers, Occupational Carcinogenesis, New York Academy of Sciences, pp. 280-293 (1976).
2. S. C. Black, V. E. Archer, W. C. Dixon, and G. Saccomanno, Correlation of radiation exposure and lead-210 in uranium miners, Health Phys. 14, 81-93 (1968).
3. R. L. Blanchard, V. E. Archer, and G. Saccomanno, Blood and skeletal levels of ^{210}Pb - ^{210}Po as a measure of exposure to inhaled radon daughter products, Health Phys. 16, 585-596 (1969).
4. R. B. Holtzman, Sources of ^{210}Pb in uranium miners, Health Phys. 18, 105-112 (1970).
5. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), Sources and Effects of Ionizing Radiation, United Nations, New York, p. 76, para. 197 (1977).
6. International Commission on Radiological Protection (ICRP), Report of the Task Group on Reference Man, ICRP No. 23, Pergamon Press, Oxford (1975).
7. International Commission on Radiological Protection (ICRP), Report of Committee II in Permissible Dose for Internal Radiation (1959), Health Phys. 3, 1-360 (1960).
8. National Council on Radiation Protection and Measurements (NCRP), Natural Background Radiation in the United States, NCRP Report 45, Washington, D.C. (1975).
9. J. Rundo, J. Y. Sha, and R. E. Toohey, Radon daughters in the occupants of a house with an unusually high concentration of airborne radon, Radiological and Environmental Research Division Annual Report, July 1978-June 1979, ANL-79-65, Part II, pp. 13-20.

10. J. Rundo, F. Markun, H. A. May, N. J. Plondke, and D. J. Keefe, Some measurements of radon and its daughters in houses, Radiological and Environmental Research Division Annual Report, July 1978-June 1979, ANL-79-65, Part II, pp. 1-12.
11. D. J. Keefe, M. P. McDowell, and P. G. Groer, Use of a microprocessor in a remote working level monitor, IEEE Trans. Nucl. Sci., NS-23(1), 705-708 (1976).
12. W. P. Norris, S. A. Tyler, and A. M. Brues, Retention of radioactive bone seekers, Science 128, 456-462 (1958).
13. N. Cohen, The retention of lead-210 in the adult baboon, Institute of Environmental Medicine, Progress Report NYO-3086 -10, Vol. 1 (1970).
14. J. B. Hursh, Retention of ^{210}Pb in beagle dogs, Health Phys. 25, 29-35 (1973).
15. W. Bolonowska, J. Piotrowski, and B. Trojanowska, The kinetics of distribution and excretion of lead (Pb-210) in rats, Proc. 14th Int. Congr. Occupational Health, Madrid, II: 420-422 (1963).
16. H. F. Lucas, Jr. and J. E. Stanford, Excretion and retention of lead-210 in rats, Radiological Physics Division Annual Report, July 1966-June 1967, ANL-7360, pp. 105-110.
17. S. R. Bernard, Dosimetric data and metabolic model for lead, Health Phys. 32, 44-46 (1977).

EXCRETION OF ^{210}Pb AND ^{210}Po BY WORKERS IN AN AREA WITH HIGH LEVELS OF ATMOSPHERIC RADON, AND ESTIMATES OF EXPOSURE TO SHORT-LIVED RADON DAUGHTERS

R. B. Holtzman, P. W. Urnezis, and J. Rundo

The urinary excretion rates of ^{210}Pb were used to estimate the exposure to ^{222}Rn and its short-lived daughter-products for 12 persons who worked at an industrial site formerly used for the processing of uranium ores. The geometric mean excretion rates of ^{210}Pb and ^{210}Po of 0.86 and 1.93 pCi/day were significantly above normal environmental levels; the geometric standard deviations (σ_g) were 2.0 and 5.7, respectively. The mean value of the "available" ^{210}Pb (that in the soft tissue) was estimated from the excretion rates of ^{210}Po to be about 2.6 nCi. The geometric mean lung exposure derived from the ^{210}Pb excretion rates was estimated at 0.41 WL based on a conversion factor of 1.49 pCi day $^{-1}$ WL $^{-1}$. This mean did not agree with the mean value of 0.14 WL that had been estimated from radon decay products in the atmosphere of the building.

Introduction

A survey of an industrial park in Canonsburg, PA, at which uranium ores had been processed and residues dumped, showed high levels of radon in the air in some buildings (up to 200 pCi/L), and the presence of high levels of uranium and ^{226}Ra in the dust and buildings.¹ Consequently, a group of people who worked at the site were measured at Argonne for possible internal contamination. Measurements of their body radioactivity in a low-background counting facility did not show levels in excess of those found in the general population.² Urine specimens were collected for bioassay for possible contaminants from the uranium decay series, uranium, ^{226}Ra , ^{210}Pb , and ^{210}Po . The results of the analyses for ^{210}Pb and ^{210}Po are discussed here.

Experimental Methods

Urine specimens were obtained from the subjects when they came to Argonne for the studies starting on a Monday morning. Each subject was asked to collect all urine over a known period of time (approximately 1 day) in clean plastic bottles supplied by us. The urine samples were then wet ashed in hydrogen peroxide and perchloric acid, and the samples were aliquoted for the various analyses.

The ^{226}Ra was determined by the de-emanation method of Lucas.³ The ^{210}Pb and ^{210}Po were determined by removal of the ^{210}Po from the solution by the spontaneous deposition of ^{210}Po , the decay product of ^{210}Pb , onto a silver disk which was then alpha counted.^{3,4} This plating process was repeated after about 3 months, and the activities of both the ^{210}Pb and ^{210}Po were determined from the counting data and application of the Bateman equations for radioactive growth and decay.

Results

Data on the subjects are presented in Table 1, including their age at the time of measurement, the numbers of the buildings in which they worked, and the approximate length of time they had worked in the building. The radon concentrations and Working Levels (WL)^{*} measured in 1977 in buildings in which the subjects worked are presented in Table 2.¹ It should be noted that the values reported for the WL were not independent measurements made simultaneously with those of the radon concentrations, but were derived from the latter by application of an average value of about 0.4 for the equilibrium factor. This is a source of uncertainty in determining the exposure to individuals in our study.

The results of the analyses of the urine from 12 subjects (10 males, 2 females) are presented in columns 2 and 3 of Table 3, and they are plotted by case number in Figure 1, which includes the results for ^{226}Ra for comparison. It is seen that there is little correlation between the values for the three nuclides. The exceptionally high value for ^{210}Pb for case 30-170 was confirmed on re-analysis. The geometric mean excretion rates of ^{210}Pb and ^{210}Po were about four and seven times normal, respectively, and the geometric standard deviation for the latter was much higher than that for the former, as expected from the very large range of values.

Five subjects (30-159, -163, -167, -170, and -171) gave values for the urinary excretion rate of ^{226}Ra that were more than five times normal

* "Working Level" is defined as any combination of concentrations of radon daughters (^{218}Po , ^{214}Bi , and ^{214}Pb) in one liter of air that results in 1.3×10^5 MeV of potential alpha energy.

Table 1. Data on the subjects.

Subject, ^a CHR case number	Age at time of measurement, yr	Building number	Time worked in building, yr	Comment
30-159	43	18	5	
160	37	10 and 18	1 and 6	
161	46	10, 9	12	
162 F	45	16, 9	12	
163	32	10A	7	Exposed to dust.
164	65	10A	7	
165	39	7	7	
166	40	15	9	10 days since last exposure, spends much time in a concrete pit.
167 F	46	3	9	
170		10A	7	Works in various places.
171	68	10	12	One month since last exposure.
172	54	16	14	

^aMales, except as noted by F.

(0.2 pCi d⁻¹). This suggested the possibility of contamination from inhaled dust, which might also have contained significant amounts of ²¹⁰Pb and ²¹⁰Po, in addition to that produced from radioactive decay from the ²²⁶Ra content. Many of the subjects commented that their working environments were quite dusty.

Table 2. Average daytime concentrations of radon and its short-lived daughter products in buildings at the Canonsburg site, March 14-April 1, 1977.^a

Building number	Concentration of ^{222}Rn , pCi L ⁻¹	Working Levels
3	106	0.43
7	34	0.14
9	51	0.21
10	38	0.15
15	15	0.06
16	38	0.16
18	31 (2.6 ^b)	0.12 (0.01 ^b)
19	19	0.08

^a From Ref. 1.

^b In office.

FIG. 1.--Urinary excretion rates of Canonsburg cases plotted against case number. x, ^{210}Pb ; $\square$, ^{210}Po ; Δ , ^{226}Ra . The horizontal lines represent the normal urinary excretion rates for the respective nuclides.

Table 3. Excretion rates and estimated exposure.

Subject, CHR case number ^a	^{210}Pb , pCi d ⁻¹	^{210}Po , pCi d ⁻¹	Estimated exposure, ^b WL	Daytime levels, ^c WL
30-159 ^d	0.89 ± 0.17	0.38 ± 0.09	0.46	0.1
160	0.73 ± 0.08	3.17 ± 0.17	0.36	0.12
161	0.47 ± 0.11	0.79 ± 0.14	0.18	0.15
162 (F)	0.49 ± 0.17	0.85 ± 0.19	0.19	0.16
163 ^d	1.95 ± 0.30	2.73 ± 0.36	1.17	0.15
164	0.49 ± 0.13	2.09 ± 0.30	0.19	0.15
165	0.61 ± 0.22	6.67 ± 0.29	0.28	0.14
166	1.09 ± 0.22	11.59 ± 0.62	0.60	0.06
167 (F) ^d	0.68 ± 0.10	0.094 ± 0.098	0.32	0.43
170 ^d	5.0 ± 0.5	1.20 ± 0.46	3.22	0.15
171 ^d	1.00 ± 0.15	0.54 ± 0.15	0.54	0.15
172	0.52 ± 0.19	2.00 ± 0.24	0.21	0.16
Geometric Mean	0.86	1.37	0.41	0.14
σ_g	2.0	3.7	2.4	1.6
Normal excretion rates (range): 0.2(0.1-0.4) 0.2(0.1-0.5)				

^a Males, except as denoted by F.

^b Calculated with the assumptions that an exposure of 1 WL (equilibrium factor 0.4) will produce sufficient ^{210}Pb to cause an excretion rate of 1.49 pCi day⁻¹ 3 days after the end of chronic exposure for 10 years, and that the normal excretion rate of ^{210}Pb is 0.2 pCi d⁻¹.

^c Average daytime levels in building taken from Table 2.

^d Subjects showing high urinary excretion rates of ^{226}Ra .

Discussion

It has been shown that the mean excretion rates of ^{210}Pb and ^{210}Po from natural sources are similar at about 0.2 pCi d^{-1} and exhibit similar ranges.⁵ Under such conditions, the principal site of deposition of the ^{210}Pb is bone and the rate-controlling factor for excretion of both nuclides is the loss from bone. By contrast, exposure of the subjects in the present study was relatively short-term and the deposition in bone may supply a much smaller fraction of the total excreted. While the ^{210}Pb may be moderately tightly bound in its soft tissue pools (with a biological half-life of the order of 1 year)⁶ the plutonium may be much less so, and thus more readily available for excretion. If we assume that the higher excretion rates of the polonium are not due to much greater intakes of this nuclide, we may estimate the amount of ^{210}Pb in the soft tissue pools. Thus, $200 \text{ pCi } ^{210}\text{Pb}$ produce $1 \text{ pCi of } ^{210}\text{Po/day}$ by radioactive decay. With a ratio of endogenous fecal-to-urinary excretion rates of ^{210}Po of 10,^{7,8} a net urinary rate of 1.2 pCi d^{-1} (1.4 pCi d^{-1} observed, less 0.2 pCi d^{-1} environmental, Table 3) represents about $2.6 \text{ nCi } ^{210}\text{Pb}$ that is "available" to produce the readily eliminated ^{210}Po . It does not necessarily represent the total ^{210}Pb .

We may use the excretion rates of ^{210}Pb (in excess of the normal level of 0.2 pCi d^{-1}) to calculate the exposure to short-lived radon daughters using the model developed in the previous report.⁹ We use the conversion factor of $1.94 \text{ pCi d}^{-1} \text{ WL}^{-1}$ appropriate to the average equilibrium factor of 0.4 reported¹ for the buildings in the Canon Industrial Park, and modify it by a factor of 0.77 to allow for the decrease in the excretion rate in the 2 or 3 days between the end of the exposure and the collection of urine (as discussed in Appendix B of Ref. 8). This results in a value of $1.49 \text{ pCi d}^{-1} \text{ WL}^{-1}$; the normal level of 0.2 pCi d^{-1} was subtracted from each value in column 2 of Table 3 and the result was divided by 1.49 to give the estimated WL in column 4 of Table 3. In column 5 the appropriate WL reported in Ref. 1 and shown in Table 2 is given for each subject. The geometric mean of the values estimated from the model is three times that for the values in column 5, but it should be noted that the ratio of the estimate from the model to the value for the appropriate building is less than the geometric standard deviation for each of six (i.e., half) of the subjects and that only one

of these (30-167) showed a high excretion rate of ^{226}Ra . Furthermore, the geometric mean for the seven subjects who did not show high levels of ^{226}Ra in the urine, was 0.27 WL (with a geometric standard deviation of 1.5), little different from the arithmetic mean of 0.30 (standard deviation ± 0.16). If we accept that the larger differences in the subjects who showed high excretion rates of ^{226}Ra were indeed due to their having inhaled contaminated dust, we are left with only two subjects (30-160 and 30-166) where the discrepancy between estimated and "observed" WL is unreasonably high. The difference (a factor of 10) for subject 30-166 (a mechanic) might well be explained by his having spent much time in a concrete pit where the concentration of radon would be expected to be higher than in the building proper. No such explanation is apparent for subject 30-160.

The extent of the agreement between the values in columns 4 and 5 of Table 3 leaves little room for complacency, because we have not taken into account the fact that the subjects were only exposed to high levels of radon and daughters during the working day of about 8 hr, for 5 days a week. Thus, the "occupancy factor" was only about 0.25, and the estimates in column 4 should probably be multiplied by four, eliminating any agreement between the pairs of values in columns 4 and 5.

Two possible reasons for this suggest themselves. First, the model may be in error, and possible ranges of the conversion factor deduced from the model are discussed in Ref. 9. Second, the value of 0.2 pCi d^{-1} for the normal excretion rate of ^{210}Pb may not apply to the individuals of this study. Examination of the values in column 2 of Table 3 shows that five of them do not differ significantly from the upper limit (0.4 pCi d^{-1}) of the normal range. If that value is used for the seven subjects who did not show high ^{226}Ra excretion rates, the geometric mean of the estimated exposures is 0.12 WL ($\sigma_g 2.3$) but application of the "occupancy factor" again results in no agreement with the mean "observed" value for the WL.

It is clear that the model has not yet been sufficiently refined to give accurate results for the exposure, but it is also clear that subjects who have been exposed to high levels of radon and its short-lived daughter products do

excrete significant amounts of ^{210}Pb . The problem of interpretation of those amounts must await further developments of the model.

References

1. Oak Ridge National Laboratory, Formerly Utilized MED/AEC Sites Remedial Action Program: Radiological Survey of the former Vitro Rare Metals Plant, Canonsburg, Pennsylvania, DOE/EV-0005/3 (1978).
2. R. E. Toohey, Radiocativity measurements of former military personnel exposed to weapon debris, Paper P/16 presented at the 25th Annual Meeting of the Health Physics Society, Seattle, Washington, July 20-25, 1980.
3. R. B. Holtzman, P. W. Urnezis, A. Padova, and C. M. Bobula III, Contamination of the human food chain by uranium mill tailings piles, U.S. Nuclear Regulatory Commission and Argonne National Laboratory Report, NUREG/CR-0758 and ANL/ES-69, pp. 29 ff. (1979).
4. P. W. Urnezis and R. B. Holtzman, An improved analytical procedure for the determination of ^{210}Pb and ^{210}Po using alpha-spectrometric isotope dilution, this report.
5. H. Spencer, R. B. Holtzman, L. Kramer, and F. W. Ilcewicz, Metabolic balances of ^{210}Pb and ^{210}Po at natural levels, Radiat. Res. 69, 166-184 (1977).
6. International Commission on Radiation Protection (ICRP), Report of Committee II on Permissible Dose for Internal Radiation, Health Phys. 3, 217-218 (1960).
7. J. N. Stannard and F. A. Smith, Distribution and excretion of polonium-210. X. Species comparison, Radiat. Res., Supplement 5, 166-174 (1964).
8. R. B. Holtzman, ^{210}Pb and ^{210}Po metabolism in radium-dial painters, Radiological Physics Division Annual Report, July 1969-June 1970 ANL-7760, p. 35.
9. R. B. Holtzman and J. Rundo, Estimation of exposure to ^{222}Rn from the excretion rates of ^{210}Pb , this report.

AN IMPROVED ANALYTICAL PROCEDURE FOR THE DETERMINATION OF ^{210}Pb AND ^{210}Po USING ALPHA-SPECTROMETRIC ISOTOPE DILUTION

P. W. Urnezis and R. B. Holtzman

An isotope dilution method has been incorporated into the ^{210}Pb - ^{210}Po analysis. A known amount of ^{209}Po is added to the sample before analysis. Then both ^{209}Po and ^{210}Po are deposited on a silver planchet which is assayed in an alpha spectrometer to determine the activities of each isotope. The recoveries generally range from 70% to 90%.

Introduction

Several methods are available for the determination of ^{210}Pb .¹ The 47 keV gamma ray can be measured directly. This allows for measurements in vivo, but this method is practical only when sufficient amounts of ^{210}Pb are present, since the gamma ray occurs in only about 5% of the disintegrations. For low-level activity, other methods are necessary.

The activity of ^{210}Pb can be determined by measurements of its daughters, ^{210}Bi or ^{210}Po . Because of the short half-life of ^{210}Bi (5 days) radioactive equilibrium is rapidly established between it and ^{210}Pb , so that only a single analysis of ^{210}Bi is necessary to determine the amount of ^{210}Pb . If ^{210}Po is used to determine the activity of ^{210}Pb , two analyses are required, but the resulting data may then be used to determine the amount initially present of both ^{210}Pb and ^{210}Po . Since ^{210}Po is an alpha emitter, it can be measured with alpha-particle counters which have significantly lower backgrounds than do the beta-particle counters used to measure ^{210}Bi . If the activities of ^{210}Pb are low and time is not critical, the indirect method of measuring ^{210}Po is the method of choice. We have improved our routine method^{2,3} by modifying it to use alpha-spectrometric isotope dilution.

Experimental Methods

The sample is ashed at a low temperature to destroy any organic matter present while at the same time preventing volatilization of polonium and lead. The methods used depend on the type of sample, as previously described.^{2,3}

All samples, whether bone, metabolic (e.g., food and excreta), or soft tissue samples, may be ashed by repeated additions of nitric acid while being heated. Alternatively, metabolic and soft tissue samples may be ashed with hydrogen peroxide acidified with nitric acid while being heated. Soft tissue may also be oxidized in a low temperature asher that uses an oxygen plasma. After ashing is complete, or nearly so, the sample is fumed with perchloric acid to complete the oxidation and to remove residual nitric acid or hydrogen peroxide, since both of these attack silver and would disrupt the analysis.

The sample holder (see Fig. 1) is assembled with a silver planchet in the Teflon insert and the sample is poured in, after the pH has been adjusted to 0.3. The ^{209}Po spike is added, the sample holder is placed in the heating mantle, and the stirrer is lowered into place. A watch glass with a slot for the stirrer covers the sample to reduce the evaporation rate. The sample is heated to 95°C and stirred for 6 hr. The stirrers are then raised and the sample holder is removed from the heating block. The sample is saved for a repeat analysis several months later. The silver planchet is rinsed with distilled water and 1.0 M HCl, and dried, and the activity on it is assayed in an alpha spectrometer to determine the contributions of ^{209}Po and ^{210}Po . The procedure is described in detail in Appendix A and the calculation in Appendix B.

The heating mantle is a cylindrical aluminum block into which 4 stainless steel tubes are inserted. These tubes are 6.4 cm i.d. by 15 cm deep and contain water. The stirrer assembly is supported above the mantle by a vertical rod anchored to the center of the heating block. The solutions are stirred by four Nalge polyethylene propeller-type stirring rods linked to a variable speed motor by a slip-proof belt.

The activity of the ^{209}Po standard solution was determined by stippling a small known amount onto a silver planchet which was gently heated from below with a hot plate and from above with a heat lamp. The planchet was then counted in an internal gas proportional counter of known counting efficiency and then in an alpha spectrometer. The proportional counter was used to determine the activity and the alpha spectrometer to check the purity. Another aliquot of the ^{209}Po solution was diluted to 200 mL with distilled water, and the pH was adjusted

FIG. 1.--Sample container for plating polonium onto one side of a 3/4-in (19.0 mm) disk).

to 0.3. This was then deposited on a 38.1-mm diameter ($1\frac{1}{2}$ -in) silver planchet during a ^{210}Pb - ^{210}Po analysis. Use of this size planchet assured a minimum recovery of 98%. The planchet was also then counted in the proportional counter to check the stippling procedure. The detectors are Princeton Gamma-Tech solid state silicon barrier detectors with 400-mm² active surface area connected to standard charge-coupled pre-amplifiers, amplifiers and bias supplies (Kicksort, Vern Roberts Associates, Albuquerque, New Mexico). The amplifiers provide the input signals to an Elscint Promeda multichannel analyzer that handles 16 detectors with 256 channels for each. The detector holders are based on the design of Larsen and Selman,⁴ but modified to accept planchets of up to 38.1 mm diameter.

Discussion

The choice of the polonium isotope for use in this procedure was between ^{208}Po and ^{209}Po . The other isotopes of polonium had half-lives too short to make their use feasible. ^{209}Po was chosen for several reasons. Its half-life is 102 yr while that of ^{208}Po is 2.93 yr so that a solution of ^{209}Po can be used for an extended period of time without appreciable decay corrections which would have to be made with ^{208}Po . The energies of the alpha particles are 4.88 MeV for ^{209}Po , 5.11 MeV for ^{208}Po , and 5.31 MeV for ^{210}Po . The greater difference in the energies of the alpha particles between ^{209}Po and ^{210}Po than between ^{208}Po and ^{210}Po makes it easier to resolve the two resulting peaks in an alpha spectrum. Finally, the ^{209}Po is readily available with good purity. (The ^{210}Po content was < 0.1% that of the ^{209}Po .)

As a result of the decision to include isotopic dilution in the analysis, alterations to the previously established procedure were necessary.² The first changes were the addition of a known amount of ^{209}Po and the use of the alpha spectrometer to assay the sample. This altered procedure worked, but the counting efficiency was low (16%) because the diameter of the planchet (38 mm) was considerably greater than that of the active surface of the detector (22.6 mm). Consequently, the planchet size was reduced to 19 mm diameter, thereby increasing the counting efficiency to 30%.

However, reducing the surface area of the planchet resulted in a marked slowing of the rate of deposition. This effect could not be compensated for by an increased deposition time which was already 6 hr. Investigations showed that more efficient mixing of the sample was needed. The old stirrers were glass rods with their ends flattened. These were easily produced and inexpensive, but they did not mix the sample well. Several different stirrers were evaluated to determine a replacement for the glass rod. A polyethylene propeller-type stirrer was chosen for its good mixing abilities and its mechanical durability.

The effects of the two different stirrers and the two planchet sizes on the rates of deposition are shown in Figure 2. A comparison of the two stirring systems shows a large increase in the rate of deposition for the polyethylene stirrers with the larger planchet. The time required to obtain 95% recovery for the glass stirrer was twice that required for the polyethylene stirrer. The reduction in surface area of the planchet drastically reduced the rate of deposition, but this was partially offset by the improved mixing efficiency of the propeller stirrer which resulted in good recovery in the 6 hr available. Nevertheless, for about a 90% recovery, the small planchet needed about four times the time required for the large planchet (the ratio of the surface areas of the planchet).

Finally, an overall comparison between the old and the new systems may be summarized as follows. The recovery for the new system is from 70% to 90% while the recovery for the old system was 90% to 98%. The old system used a proportional counter with a counting efficiency of 50%, whereas the solid state

FIG. 2.--Yield of ^{210}Po versus plating time. The squares represent the glass stirrer and the 38 mm planchet. The triangles represent the polyethylene stirrers and the 38 mm planchet. The circles represent the polyethylene stirrer and the 19 mm planchet (the new system).

detector of the new system has about a 30% counting efficiency. The decreased efficiency is more than compensated for by the reduction in background counting rate, which is one-fourth to one-fifth that of the proportional counters. While the background counting rate for the new system (0.001–0.004 cpm) is substantially lower than that of the old one (0.020 cpm), the sensitivity of the new system, based on the criterion of S^2/B ((signal)²/background), is essentially identical to that of the old one because of the reduced counting geometry (30%) and reduced yields (to about 80%).^{*} However, the new system has the substantial advantage that the overall yield may be determined reliably.

Acknowledgement

We thank Dr. J. Sedlet, Occupational Health and Safety Division, Argonne National Laboratory, for supplying the stock of ²⁰⁹Po for this work.

APPENDIX A: Analytical Procedures

1. Adjust the volume of the sample to about 150 μ L and the pH to 0.3 (0.5 N) by adding concentrated HCl dropwise.
2. Place a small quantity of Apiezon H grease[†] on the inside lip of the Teflon insert. Place the silver disk (19-mm dia $\times$ 0.13-mm thick, i.e., 3/4-in dia $\times$ 0.005-in) in the Teflon insert. Put the rubber plug behind the silver disk. Make sure that the silver disk is resting against the inside lip of the insert and the grease forms a complete seal around the disk.
3. Place the Teflon insert inside the cap of the sample holder and screw the cap tightly onto the sample holder (250 mL linear polyethylene bottle with the bottom cut out). Check the seal by adding 50 mL of water to the bottle and allow to stand about 15 min. If the seal is poor, water will be seen in the cap.

^{*} If $S_{\text{new}} = (\text{ratio of counting geometries}) \cdot (\text{ratio of yields}) \cdot S_{\text{old}} = (0.3/0.5) \cdot (0.80/0.96) \cdot S_{\text{old}} = 0.50 S_{\text{old}}$, and $B_{\text{new}} = 0.2 B_{\text{old}}$, then $(S_{\text{new}}^2/B_{\text{new}})/(S_{\text{old}}^2/B_{\text{old}}) = (0.50)^2 \cdot 5 = 1.25$, only a 25% improvement.

[†] This grease maintains its viscosity at high temperatures and is easily removed from the disk by petroleum ether.

If this happens, check for the presence of foreign matter between the Teflon insert and sample holder and to see that the silver disk is seated next to the lip on the insert.

4. After obtaining a good seal, wrap the edge of the cap and the neck of the bottle with 0.75-in wide vinyl electrical tape to prevent the heating water from entering the cap.

5. Pour the sample into the holder and add 200 to 300 mg of ascorbic acid. This is to reduce Fe^{+3} to Fe^{+2} ions in solution and prevent interference with the plating of Po onto the silver.

6. Add a known amount of ^{209}Po to sample.

7. Place the sample holder in a water bath at about 95 C, cover with a watch glass with a slot or hole for the shaft of the stirrer.

8. Stir as rapidly as possible for about 6 hr at a rate just below that at which the solution splashes out of the container or the vortex rises above the walls of the container.

9. Add water as necessary every few hours to maintain a constant volume in the bottle.

10. After completion of the plating, remove the watch glass and stirrer, wash the convex surface of the watch glass with water, and collect the wash water in the sample. Rinse the stirrer and collect the washings in the sample.

11. Pour the sample into a storage bottle and save it. Rinse sample holder several times with a few mL of 1 M HCl and add the rinse to the sample.

12. Remove the tape from the sample holder and remove silver disk. Record time of removal. Rinse the disk with a few drops of 1 M HCl (from a dropping bottle) and dry it on a watch glass heated by the water bath. Remove the grease on the planchet by swirling it in a beaker filled with petroleum ether and rinse the planchet with water.

13. Count the silver disk in an alpha spectrometer. This laboratory uses an Elscint Promeda multichannel analyzer with Kicksort preamplifiers and amplifiers and Princeton Gamma-Tech detectors. The alpha counting efficiency is about 30%, and the background counting rate is about 0.0005 to 0.003 cpm in the regions of interest. The disk is usually counted for about 24 hr. Print out the

spectra and time channel. Type in the start time, silver disk number, and the counter number.

14. Store the sample for about four months (one half-life of the ^{210}Po) to allow ^{210}Po to grow in from the ^{210}Pb . The exact storage time is not important, as long as it is known to ± 1 day.

15. Repeat steps 1 to 13 for the second plating. The sample may be dark and contain a black or gray precipitate of organic material from the original ascorbic acid. This does not appear to affect the efficiency of the second plating.

APPENDIX B: Calculation of Results

The results of the two depositions are used to calculate the activities of both the ^{210}Pb and ^{210}Po from the Bateman equations for radioactive decay and growth.^{*}

The equation^{*} for the ^{210}Pb activity at the time of collection is:

$$A_1 = \frac{A_2(2) - A_2(1) e^{-\lambda_2 t_2}}{e^{-\lambda_1(t_2-t_1)} - e^{-\lambda_2(t_2-t_1)}} \cdot \frac{2^{-t_1/t_{1/2}}}{\lambda_2}$$

and similarly for the ^{210}Po :

$$A_2^0 = \left[A_2(1) - \frac{2}{2^{-t_1/t_{1/2}} - 1} A_1^0 \left(e^{-\lambda_1 t_1} - e^{-\lambda_2 t_1} \right) \right] e^{-\lambda_2 t_1}$$

where

$A_2(1)$ is the activity of the ^{210}Po from the first separation,

$A_2(2)$ is the activity of the ^{210}Po from the second separation,

λ_1 is the decay constant of the ^{210}Pb ,

λ_2 is the decay constant of the ^{210}Po .

* The equations shown do not account for the presence of ^{210}Bi , the ^{210}Pb daughter product, which introduces an error of about 1% in the calculations for ^{210}Pb . For completeness the computer program does account for the presence of ^{210}Bi . However, because of its short half-life (5.0 days) and because the minimum time between collection and analysis is several weeks, the initial amounts of ^{210}Bi are not determined. An initial activity of ^{210}Bi equal to that of the ^{210}Po increases the ^{210}Po activity by 3.6%.

t_1 is the time between collection and first separation,

$t_2 - t_1$ is the time between first and second separation, and

Y is the recovery as determined either from the ^{209}Po values or from other experiments.

$A_2(1)$ and $A_2(2)$ are corrected for the recovery (Y) and the detector efficiencies.

This calculation is usually done by a computer program (POLOSP) from raw counting data, dates, times, and counts. The activities A_1^0 and A_2^0 are calculated along with estimates of errors based on counting statistics.

References

1. R. B. Holtzman, Application of radiolead to metabolic studies, The Biogeochemistry of Lead in the Environment, Vol. 1B, J. Nriagu, Ed., Elsevier/North Holland Biomedical Press, pp. 37-96 (1978).
2. R. B. Holtzman, P. W. Urnezis, A. Padova, and C. M. Bobula, III, Contamination of the human food chain by uranium mill tailings piles, U.S. Nuclear Regulatory Commission and Argonne National Laboratory Report, NUREG/CR-0758, ANL/ES-69, p. 29 ff. (1979).
3. R. B. Holtzman, Measurement of the natural contents of RaD (Pb^{210}) and RaF (Po^{210}) in human bone-estimates of whole-body burdens, *Health Phys.* 9(4), 385-400 (1963).
4. R. P. Larsen and R. F. Selman, An alpha counting chamber for use with surface barrier detectors, *Radiochem. Radioanal. Letters* 30(4), 247-252 (1977).

URANIUM CONCENTRATIONS IN HUMAN BONE^{*}

Robert A. Schlenker and Billie G. Oltman

The natural uranium content in the bone of one person has been determined by a new method. It is hoped these data will shed light on the large discrepancy between the recent and the earlier data reported by Welford and his collaborators,^{1,2} a discrepancy they attribute (see Ref. 1) to methodological errors in the earlier work.

The results, obtained by quantitative analysis of fission track autoradiographs of bone from a person injected with ²³⁹Pu (case HP4 in Ref. 3, also known as case 40-010 in our records) are given in Table 1.

Table 1. Uranium Concentrations in the Bone Volume of Case 40-010

Location	²³⁵ U, pg/g ^a
Long-bone midshafts ^b	21 (8 - 31)
Proximal femur metaphysis ^b	102 (35 - 180)
Pelvis ^b	246 (68 - 576)
Vertebrae	104 (83 - 147)

^aThese units are picogram ²³⁵U/gram fresh wet bone. The range of observed concentrations is given in parentheses following the average value.

^bRight side of skeleton.

Welford and Baird² reported an average natural uranium concentration of 20,000 pg/g bone ash, and Hamilton⁴ found 24,000 pg/g bone ash. These are equivalent to 81 and 97 pg ²³⁵U/g fresh wet bone (assuming 0.56 g ash/g fresh wet bone). Our data, collected using much less bone and excluding possible

^{*}Extended abstract of a paper presented at the Workshop on Measurement and Interpretation of Actinide Accumulation by Man, Snowbird, Utah, October 14-18, 1979.

bone surface deposition of uranium, range on both sides of these values and are consistent with them, considering the smallness of our sample size. However, they are not consistent with the values, about an order of magnitude lower, reported more recently.¹

References

1. G. A. Welford, R. Baird, and I. M. Fisenne, "Concentration of Natural Uranium in the Human Body," 10th Midyear Topical Symposium of the Health Physics Society, October 11-13, 1976, Saratoga Springs, New York.
2. G. A. Welford and R. Baird, Uranium levels in human diet and biological materials, *Health Phys.* 13, 1321 (1967).
3. P. W. Durbin, Plutonium in man: A new look at the old data, in Radio-biology of Plutonium, B. J. Stover and W. S. S. Jee, Eds., J. W. Press, Salt Lake City, Utah, p. 469 (1972).
4. E. I. Hamilton, The concentration and distribution of uranium in human skeletal tissues, *Calc. Tiss. Res.* 7, 150 (1971).

PLUTONIUM MICRODISTRIBUTION IN HUMAN BONE^{*}

Robert A. Schlenker and Billie G. Oltman

The amount and location of plutonium in bone from three humans injected during the mid-1940's have been studied by autoradiography and alpha-particle spectrometry. Concentrations are similar on endosteal surfaces, Haversian canal surfaces, and on the periosteal surfaces at the midshafts of long bones 17 months after injection. Endosteal surface concentrations are higher in the axial skeleton than in the appendicular skeleton 15 and 17 months post injection. For dosimetric purposes, volume deposits may be considered to be "infinitely thick," whereas surface deposits may be considered to have zero thickness. Secondary surface deposits are dosimetrically important, even when the plutonium is almost completely deposited in bone volume.

^{*}Abstract of a paper presented at the Workshop on Measurements and Interpretation of Actinide Accumulation by Man, Snowbird, Utah, October 15-17, 1979.

MEASUREMENTS OF RADIOACTIVITY IN FORMER MILITARY PERSONNEL EXPOSED TO WEAPON DEBRIS

R. E. Toohey, J. Rundo, M. A. Essling, J. Y. Sha, R. D. Oldham,
J. Sedlet,* and J. J. Robinson*

Sixteen former military personnel who were present at the "Smoky" atmospheric nuclear weapons test have been investigated for internal deposits of radioactivity. Whole-body and thorax γ -ray measurements, thorax and skeletal actinide measurements, and urinalyses for ^{239}Pu and ^{90}Sr were performed. No evidence of radioactivity in excess of that found in the general population was observed.

Introduction

During the 1950's, an estimated 250,000 military personnel were present during nuclear weapons tests at the Nevada Test Site, either as permanent party support troops, or as participants in maneuvers designed to determine their ability to accomplish assigned missions on a nuclear battlefield. On 31 August 1957, a 44-kiloton device code-named "Smoky" was exploded. Eight cases of leukemia have been reported in 1900 of the 3200 troops present at that test. Film badge readings were available for seven of the eight cases, and ranged from 0 to 2,997 mrem with a mean of 1,178 mrem. The mean 1957 cumulative dose for the entire 3200-man cohort was 493.4 mrem.¹

Because the film badges recorded only the external gamma dose, the Center for Human Radiobiology was asked to determine the levels of internal radioactivity in a group of men present at the "Smoky" test. Nineteen men were selected by the Center for Disease Control on the basis of high film-badge readings and/or their opportunity for inhalation or ingestion of weapon debris. None of them exhibited any clinical signs of malignancy or other radiation-induced pathology. The nineteen were assigned CHR case numbers 30-140 to 30-158. Sixteen of them visited ANL in 1979 for body radioactivity measurements; three chose not to participate.

* Occupational Health and Safety Division.

The men were briefly interviewed during their visits, and several reported being present at up to three tests. Their locations at the time of detonation ranged from a trench at 2.5 km from ground zero to a hillside 16 km away. After the tests, some were trucked or marched to within an estimated 200 m of ground zero where they were timed on their performance of routine tasks such as weapon cleaning. One man reported that protective masks were worn, while another reported that they were not. One man was a wireman whose job was to re-install telephone lines that were destroyed by each test, and he reported that packed lunches were eaten frequently while in the vicinity of ground zero. All the men reported that the test site was an extremely dusty environment. Thus it appears that the potential for internal deposition of radioactivity did exist.

Measurement Techniques

The whole-body contents of γ -ray emitters were measured with large NaI(Tl) detectors in both the reclining chair and flat bed geometries. This latter geometry maximized the efficiency of detection of γ -ray emitters in the thorax. The detectors were calibrated for ^{40}K and ^{137}Cs by counting water-filled phantoms made of plastic bottles arranged to simulate the shape of a human body and containing known amounts of these radionuclides. Different arrangements of bottles were used to calibrate for different body sizes. The γ -ray spectra from the subjects were analyzed by a computer method of least squares.

Measurements of actinide (^{239}Pu and ^{241}Am) content were made with a 180-mm diameter xenon-filled proportional counter. A mean subject background was subtracted from the counting rate observed over the chest in the energy range 16 to 24 keV; the remainder was assumed to be due to pure ^{239}Pu , since no evidence of the 60-keV line from ^{241}Am was observed. It must be noted, however, that this method overestimates the plutonium alpha activity, since ^{238}Pu , ^{240}Pu , and ^{242}Pu all emit more x rays per alpha than does ^{239}Pu . The counting rate was converted to a lung content by applying a calibration factor obtained from a realistic thorax phantom containing lungs loaded with a known amount of ^{239}Pu and having a variable chest wall thickness.² The appropriate

phantom calibration factor was determined for each subject by measuring the subject's average chest wall thickness with an ultrasonic probe. The skeletal contents of ^{239}Pu were determined from the measurements over the skulls. (The skull has been shown to be a representative bone for the determination of skeletal burdens of bone-surface seeking radionuclides in humans.)^{3,4} A mean background was determined by counting over the skulls of several controls. The net counting rate from each subject was converted to skeletal ^{239}Pu by applying a calibration factor of $23 \pm 1 \text{ nCi } ^{239}\text{Pu per cpm}$.⁵ This factor was derived from measurements made over the skull of a case from a different group of subjects⁶ whose skeletal burdens of plutonium were determined radiochemically.³ Note that because the thickness of soft tissue over the skull does not vary appreciably, the calibration factor is constant despite the range of body sizes.⁴

While they were at ANL, the subjects in this study supplied 24-hr urine specimens. Aliquots were analyzed for ^{239}Pu by isotopic dilution alpha spectrometry, and for ^{90}Sr by chemical separation and beta-counting. Estimates of an upper limit for the body content of ^{239}Pu were made (a) with the aid of Langham's power function equation,⁷ which yields a value for the initial systemic content, and (b) with the application of the retention function proposed by a Task Group of the ICRP.⁸ The second method required an assumption about the amount excreted in feces. We multiplied the urinary excretion rate by 1.47 to derive a value for the total excretion rate.⁹ It should be mentioned that it is known that use of the Langham equation results in an over-estimate of the initial systemic content of plutonium when it is applied at late times after intake.⁹

The possible body contents of ^{90}Sr were calculated in the following manner. The urinary excretion rates of ^{90}Sr were multiplied by 1.3 to allow for fecal excretion, and the total excretion rates were converted to current body contents with the aid of the retention equation suggested by a Task Group of the ICRP.¹⁰ The ^{90}Sr levels of control subjects were determined in like manner.

Results

The results are presented in tabular form. Table 1 gives biometric data on the subjects, and Table 2 presents the results of the analyses of the gamma-ray

Table 1. Biometric data on the 16 subjects.

CHR case No.	Date of birth	Height, m	Weight, kg	Chest wall thickness, mm
30-140	Feb. 22, 1931	1.78	108	29
-141	Mar. 20, 1935	1.78	68.6	33
-143	Jan. 31, 1936	1.66	64.8	13
-145	Jul. 14, 1937	1.64	62.2	17
-146	Sep. 24, 1938	1.75	79.2	20
-147	Dec. 1, 1930	1.71	58.8	15
-148	Apr. 22, 1926	1.70	83.5	28
-149	Aug. 24, 1936	1.75	75.4	36
-150	Feb. 14, 1938	1.83	93.5	36
-151	Nov. 21, 1930	1.85	76.2	20
-153	May 30, 1939	1.85	79.9	28
-154	Mar. 2, 1923	1.75	83.9	33
-155	Feb. 11, 1933	1.73	66.0	13
-156	Apr. 11, 1927	1.85	81.3	29
-157	Jul. 29, 1919	1.83	76.7	35
-158	Aug. 23, 1936	1.80	101	30

spectra, which were made with the assumption that the whole of the response was attributable to naturally occurring ^{40}K and fallout ^{137}Cs . Below the individual entries are shown the mean potassium content (as % of body mass) and the mean cesium to potassium ratio ($\text{pCi } ^{137}\text{Cs/g K}$), with their standard deviations and variance ratios (i.e., ratio of observed to predicted variances). The standard deviations are given rather than the standard errors of the means because the former are more indicative of the spread of the individual values. The fact that the variance ratios are greater than unity is indicative of biological variation in the potassium and ^{137}Cs contents, and is to be expected. This biological

Table 2. Potassium and ^{137}Cs contents.

Case No.	Potassium		^{137}Cs	
	g	% body wt	nCi	pCi/g K
30-140	185 $\pm$ 4	0.171 $\pm$ 0.004	2.1 $\pm$ 0.3	11.1 $\pm$ 1.5
-141	158 $\pm$ 4	0.231 $\pm$ 0.006	1.8 $\pm$ 0.2	11.6 $\pm$ 1.6
-143	133 $\pm$ 4	0.206 $\pm$ 0.006	0.8 $\pm$ 0.2	6.2 $\pm$ 1.6
-145	123 $\pm$ 4	0.197 $\pm$ 0.006	0.8 $\pm$ 0.2	6.4 $\pm$ 1.8
-146	151 $\pm$ 3	0.191 $\pm$ 0.005	0.7 $\pm$ 0.2	4.5 $\pm$ 1.3
-147	112 $\pm$ 4	0.191 $\pm$ 0.006	not significant	
-148	124 $\pm$ 3	0.149 $\pm$ 0.004	1.4 $\pm$ 0.2	10.9 $\pm$ 1.9
-149	185 $\pm$ 4	0.245 $\pm$ 0.006	1.9 $\pm$ 0.3	10.2 $\pm$ 1.4
-150	175 $\pm$ 3	0.188 $\pm$ 0.004	1.7 $\pm$ 0.2	9.6 $\pm$ 1.3
-151	144 $\pm$ 3	0.189 $\pm$ 0.005	0.9 $\pm$ 0.2	6.1 $\pm$ 1.4
-153	194 $\pm$ 4	0.242 $\pm$ 0.005	1.9 $\pm$ 0.3	9.9 $\pm$ 1.3
-154	144 $\pm$ 4	0.172 $\pm$ 0.005	0.9 $\pm$ 0.2	6.6 $\pm$ 1.7
-155	170 $\pm$ 4	0.258 $\pm$ 0.007	not significant	
-156	140 $\pm$ 4	0.172 $\pm$ 0.005	1.1 $\pm$ 0.2	8.1 $\pm$ 1.6
-157	174 $\pm$ 4	0.227 $\pm$ 0.005	0.9 $\pm$ 0.2	5.3 $\pm$ 1.2
-158	176 $\pm$ 4	0.174 $\pm$ 0.004	not significant	
Mean $\pm$ S.D.		0.20 $\pm$ 0.03	1.3 $\pm$ 0.5	8.2 $\pm$ 2.5
Variance ratio		36	5.5	2.6
<u>12 Control subjects</u>				
Mean $\pm$ S.D.		0.19 $\pm$ 0.03	1.4 $\pm$ 0.4	8.9 $\pm$ 2.4
Variance ratio		38	2.7	2.0

variation was confirmed by the corresponding values given in Table 2 for 12 other men who were investigated during the same period and whose spectra were analyzed in the same way. The results for the mean potassium concentrations and the ^{137}Cs /potassium ratio of the 12 controls are similar to those for the 16 subjects, and the variance ratios were again high. It should be noted that the mean potassium content is exactly that given for Reference Man,¹¹ indicating that the calibration is correct. The ^{137}Cs contents and the $^{137}\text{Cs}/\text{K}$ ratios agree quite well with the results of others for the general population. The mean $^{137}\text{Cs}/\text{K}$ ratio for a group of 16 controls at the Atomic Energy Research Establishment, Harwell, UK, was 8.5 pCi/g in 1976,¹² and for a group of 40 workers at Los Alamos Scientific Laboratory, also in 1976, the ratio was 10.3 pCi/g, with a mean ^{137}Cs content of 1.3 nCi.¹³ Thus it is evident that these subjects do not contain an excess of this fission product. All other gamma-ray emitters in the 16 subjects were below the limit of detection of ~ 0.2 to 0.5 nCi (depending on the gamma-ray energy), with the exception of short-lived radon daughters. There was some evidence for the presence of ^{214}Pb and ^{214}Bi in most members of the group, as well as in the control group. However, statistically significant amounts were present in only three subjects. The results of the measurements of ^{239}Pu made with the proportional counter are set out in Table 3. Not one result is greater than zero at even the 90% (1.64 σ) confidence level, and the mean value for each set of measurements is close to zero. The fact that the variance ratio is close to unity indicates that biological variation is not playing a large role in these measurements, and that the results vary randomly about zero.

The plutonium (^{239}Pu) content of each urine sample was below the limit of detection (4.5 fCi) of our standard method. From Langham's equation relating daily urinary excretion to systemic intake, we deduce that the latter was less than 1.7 nCi in August 1957 (7900 days before the urine collections). Since use of the equation at times much longer than 5 years is known to over-estimate the systemic intake,⁹ the value of 1.7 nCi must be regarded as an extreme upper limit. A limiting value for the current body content of < 200 pCi results from use of the retention equation suggested in ICRP Report 19.⁸ These low values

Table 3. External counting of low energy photon emitters.

Case No.	Measurements over chest			Measurements over skull	
	Net cpm ^a	Calibration factor, nCi/cpm	²³⁹ Pu in chest, nCi ^b	Net cpm ^c	²³⁹ Pu in skeleton, nCi ^b
30-140	0.45 ± 0.54	119	54 ± 64	0.62 ± 0.46	14 ± 10
-141	0.25 ± 0.53	154	38 ± 82	0.02 ± 0.41	0.5 ± 10
-143	-0.15 ± 0.51	22	-3 ± 11	-0.52 ± 0.37	-12 ± 9
-145	-1.08 ± 0.44	34	-37 ± 15	0.15 ± 0.42	3.5 ± 10
-146	0.12 ± 0.52	50	6 ± 26	-0.78 ± 0.34	-18 ± 8
-147	-0.48 ± 0.48	29	-14 ± 14	0.42 ± 0.45	10 ± 11
-148	-0.81 ± 0.46	110	-89 ± 51	0.55 ± 0.46	13 ± 11
-149	0.12 ± 0.52	192	23 ± 26	0.62 ± 0.46	14 ± 10
-150	-0.01 ± 0.51	192	-2 ± 98	-1.05 ± 0.37	-24 ± 9
-151	0.12 ± 0.52	50	6 ± 26	0.15 ± 0.42	3.5 ± 10
-153	0.25 ± 0.53	110	27 ± 58	-0.12 ± 0.40	-3 ± 9
-154	-0.41 ± 0.49	154	-63 ± 75	0.28 ± 0.43	6 ± 10
-155	1.05 ± 0.58	22	23 ± 13	0.55 ± 0.46	13 ± 11
-156	-0.01 ± 0.51	132	-1 ± 67	0.14 ± 0.42	3 ± 10
-157	-0.28 ± 0.50	182	-51 ± 91	-0.25 ± 0.39	-6 ± 9
-158	0.72 ± 0.56	127	91 ± 71	-0.52 ± 0.41	-12 ± 9
Mean ± S.D.	-0.01 ± 0.54		0.5 ± 45	0.02 ± 0.52	0.3 ± 12
Variance ratio	1.1		0.6	1.5	1.5

^aAfter subtraction of control background of 3.68 ± 0.13 cpm (± 1 S.E.)

^bCalculated with the assumption that the whole of the net response was due to pure ²³⁹Pu.

^cAfter subtraction of control background of 2.05 ± 0.18 cpm,

for plutonium content based on excretion rates confirm the negative results of the external measurements of plutonium content made with the proportional counter.

The results for the daily urinary excretion of ^{90}Sr are given in Table 4. Also included are the values for seven control samples. Three of the control values were obtained from 24-hr urine collections by individuals, and the remaining four were obtained from pooled urine samples obtained from a number of individuals during working hours. The values from the pooled samples (pCi/L) were converted to daily excretion values by multiplying them by the average daily urinary output of 1.4 L given in ICRP Report 23.¹¹ None of the control subjects was ever exposed to ^{90}Sr other than that from global fallout.

The mean excretion rate and its standard deviation from the 16 test subjects are essentially identical to those from the controls. We thus infer that both groups are drawn from the same population, i.e., the ^{90}Sr excreted by the test subjects arises from global fallout rather than from their presence at weapon tests.

Summary and Conclusions

We tested 16 subjects exposed to weapon debris for internal deposition of radioactivity by a combination of external counting and urinalysis. In none of the 16 subjects were we able to detect radioactivity in excess of normal levels carried by all members of the population, and we conclude that long-lived isotopes have not contributed any internal component to the radiation doses received by these men due to their participation in the "Smoky" test.

Table 4. Urinary excretion rates and calculated current body contents of ^{90}Sr .

Case No.	Urinary excretion, pCi/d	Systemic burden, nCi ^a
30-140	0.47 ± 0.06	1.8 ± 0.2
* 141	0.92 ± 0.08	3.4 ± 0.3
143	0.40 ± 0.05	1.5 ± 0.2
-145	0.30 ± 0.05	1.1 ± 0.2
-146	1.39 ± 0.13	5.2 ± 0.5
-147	0.19 ± 0.05	0.7 ± 0.2
-148	0.29 ± 0.07	1.1 ± 0.3
-149	0.69 ± 0.06	2.6 ± 0.2
-150	0.74 ± 0.11	2.8 ± 0.4
-151	0.66 ± 0.08	2.5 ± 0.3
-153	0.44 ± 0.05	1.6 ± 0.2
-154	0.72 ± 0.06	2.7 ± 0.2
-155	0.42 ± 0.07	1.6 ± 0.3
-156	0.50 ± 0.10	1.9 ± 0.4
-157	1.23 ± 0.09	4.6 ± 0.3
-158	0.70 ± 0.09	2.6 ± 0.3
Mean $\pm$ S.D.	0.63 ± 0.33	2.4 ± 1.2
Variance ratio	17.6	17.8
Controls		
1	1.13 ± 0.09	4.2 ± 0.3
2	0.95 ± 0.10	3.6 ± 0.4
3	0.58 ± 0.09	2.2 ± 0.3
4 ^b	0.50 ± 0.07	1.9 ± 0.3
5 ^b	0.21 ± 0.07	0.8 ± 0.3
6 ^b	0.57 ± 0.06	2.1 ± 0.2
7 ^b	0.67 ± 0.09	2.5 ± 0.3
Mean $\pm$ S.D.	0.66 ± 0.30	2.5 ± 1.1
Variance ratio	13.4	13.6

^a Calculated from the retention equation suggested in ICRP Report 20.^b Pooled sample, $\text{pCi/L} \times 1.4 = \text{pCi/d}$.

References

1. Leukemia among persons present at an atmospheric nuclear test (Smoky), Center for Disease Control, Morbidity and Mortality Weekly Report 28, 361-362 (1979).
2. R. V. Ciffith, P. N. Dean, A. L. Anderson, and J. C. Fisher, Fabrication of a tissue-equivalent torso phantom for intercalibration of in vivo trans-uranic nuclide counting facilities, Proc. Symp. on Advances in Radiation Protection Monitoring, Stockholm, June 26-30, 1978, Int. Atomic Energy Agency, Vienna (1979).
3. R. P. Larsen, R. D. Oldham, C. G. Cacic, J. E. Farnham, and J. R. Schneider, Distribution of injected plutonium in the skeleton and certain soft tissues, Radiological and Environmental Research Division Annual Report July 1977-June 1978, ANL-78-65 Part II, pp. 145-153.
4. N. Cohen, H. B. Spitz, and McD. E. Wrenn, Estimation of skeletal burden of "bone-seeking" radionuclides in man from in vivo scintillation measurements of the head, Health Phys. 33, 431 (1977).
5. R. E. Toohey, The determination of skeletal ^{239}Pu by external counting at the skull, Radiological and Environmental Research Division Annual Report July 1976-June 1977, ANL-77-65 Part II, pp. 102-106.
6. R. E. Rowland and P. W. Durbin, Survival, causes of death, and estimated tissue doses in a group of human beings injected with plutonium, The Health Effects of Plutonium and Radium, W. Jee, Ed., J. W. Press, Salt Lake City, pp. 329-341 (1976).
7. W. H. Langham, S. H. Basset, P. S. Harris, and R. E. Carter, Distribution and Excretion of Plutonium Administered Intravenously to Man, Los Alamos Scientific Laboratory Report LA-1151 (1950).
8. The Metabolism of Compounds of Plutonium and Other Actinides, ICRP publication 19, (1972).
9. J. Rundo, P. M. Starzyk, J. Sedlet, R. P. Larsen, R. D. Oldham, and J. J. Robinson, The excretion rate and retention of plutonium 10,000 days after acquisition, Diagnosis and Treatment of Incorporated Radionuclides, Proc. seminar, Vienna, 8-12 December 1975, Int. Atomic Energy Agency, Vienna, pp. 15-23 (1976).
10. Alkaline earth metabolism in adult man, ICRP Publication 20 (1972); also Health Phys. 24, 125 (1973).
11. Reference Man, ICRP Publication 23 (1974).
12. D. Newton, M. C. Eagle, and J. B. Vena, The cesium-137 content of man related to fallout in rain, 1957-76, Int. J. Environ. Studies 11, 83 (1977).
13. R. G. Thomas, E. C. Anderson, and C. R. Richmond, Cesium-137 whole-body content in a normal New Mexico population, Biomedical and Environmental Research Program, January-December 1976, Los Alamos Scientific Laboratory Report LA-6898-PR, pp. 5-7 (1977).

THE DISTRIBUTION OF ^{241}Am IN THE HUMAN BODY AS DETERMINED BY EXTERNAL COUNTING*

R. E. Toohey

Methods for determining the distribution of ^{241}Am within the body of a contaminated subject and their application to several cases under study at the Center for Human Radiobiology are described. In general, ^{241}Am is found in the lungs long after inhalation, and systemic ^{241}Am is observed to be deposited in the liver and in the skeleton; similar findings have been reported in animal studies.¹ Analysis of the skeletal distribution of ^{241}Am indicates deposition on bone surfaces. In contrast, the distribution of injected ^{239}Pu in an abnormal skeleton was found to be rather non-uniform when compared to that of ^{241}Am .²

References

1. R. D. Lloyd, W. S. S. Jee, D. R. Atherton, G. N. Taylor, and C. W. Mays, Americium-241 in beagles: Biological effects and skeletal distribution, *Radiobiology of Plutonium*, B. J. Stover and W. S. S. Jee, Eds., J. W. Press, Salt Lake City, pp. 141-148 (1972).
2. R. P. Larsen, R. D. Oldham, C. G. Cacic, J. E. Farnham, and J. R. Schneider, Distribution of injected plutonium in the skeleton and certain soft tissues, Radiological and Environmental Research Division Annual Report, July 1977-June 1978, ANL-78-65, Part II, pp. 145-153.

* Abstract of paper presented at the Workshop on Measurements and Interpretation of Actinide Accumulation by Man, Snowbird, Utah, 15-17 October 1979.

THE LATE EXCRETION OF PLUTONIUM FOLLOWING ACQUISITION OF KNOWN AMOUNTS*

J. Rundo

The urinary and fecal excretion rates of plutonium 10 000 days after intravenous injection of known amounts are compared with the predictions of various models. Both Langham's and Durbin's equations underestimated the urinary excretion by about an order of magnitude; the observed fecal excretion rates were also higher than the predictions. The total excretion rate predicted by the ICRP model was in quite good agreement with the observed rate, but it overestimated the observed rate at 1500 days and grossly underestimated it at early times (< 20 days). These differences are discussed.

The increase in the excretion rate between 1500 days and 10 000 days is real, as shown by the increase in apparent body content of ^{239}Pu of former Manhattan Project plutonium workers, as calculated from the urinary excretion and application of Langham's equation. In one of these subjects the urinary excretion rate started to increase at about 6000 days, reached a maximum at about 9500 days, and declined for the next 2700 days.

* Abstract of a paper presented at the Workshop on Measurements and Interpretation of Actinide Accumulation by Man, Snowbird, Utah, October 15-17, 1979.

MACRODISTRIBUTION OF PLUTONIUM IN THE HUMAN SKELETON*

R. P. Larsen, R. D. Oldham, and R. E. Toohey

The skeletal remains of two individuals who received plutonium by intravenous injection have been analyzed to establish the skeletal burden and its macrodistribution both among and within individual bones. The concentrations in most axial bones were factors of 2 to 4 higher than the average in the entire skeleton, the concentrations in the skull bones were about the same as the average, and the concentrations in the appendicular bones were factors of 2.5 to 8 lower than the average. The results obtained when the trabecular and cortical portions of bones were analyzed separately show that (1) within a particular bone the concentration in the trabecular portion is always higher than that in the cortical portion, and (2) among individual bones plutonium concentration is correlated with metabolic activity, not degree of trabecularity. The bone that could be readily taken at autopsy and whose plutonium concentration closely approximates the skeletal average is the clavicle.

* Abstract of a paper presented at the Workshop on Measurements and Interpretation of Actinide Accumulation by Man, Snowbird, Utah, 15-17 October 1979.

ON THE GASTROINTESTINAL ABSORPTION OF PLUTONIUM^{*}

R. P. Larsen, R. D. Oldham, M. H. Bhattacharyya,[†] E. S. Moretti,[†]
and D. J. Austin[§]

An investigation has been made of the effect of the oxidation state of plutonium on its absorption from the gastrointestinal tract. For mice and rats that have been starved prior to gastrointestinal administration, there is no significant difference between the absorption factors for Pu(IV) and Pu(VI). The value obtained for Pu(VI) is an order of magnitude lower than that reported by Weeks et al.¹ The value obtained for Pu(IV) is two orders of magnitude higher than those reported previously for nitrate solutions and the same as those reported for citrate solutions.¹⁻³

References

1. M. H. Weeks, J. Katz, W. D. Oakley, J. E. Ballou, L. A. George, R. C. Thompson, and H. A. Kornberg, Further studies on the gastrointestinal absorption of plutonium, *Radiat. Res.* 4, 339-347 (1956).
2. D. W. Baxter and M. F. Sullivan, Gastrointestinal absorption and retention of plutonium chelates, *Health Phys.* 22, 785-786 (1972).
3. J. Carritt, R. Fryxell, J. Kleinschmidt, R. Kleinschmidt, W. H. Langham, A. San Pietro, R. Schaffer, and B. Schnap, The distribution and excretion of plutonium administered intravenously to the rat, *J. Biol. Chem.* 171, 273-283 (1947).

^{*} Abstract of a paper presented at the Workshop on Measurements and Interpretation of Actinide Accumulation by Man, Snowbird, Utah, 15-17 October 1979.

[†] Biological and Medical Research Division.

[§] Participant in the Undergraduate Research Program, Center for Educational Affairs.

PLUTONIUM RETENTION IN MICE AFTER GASTROINTESTINAL ABSORPTION*

R. P. Larsen, R. D. Oldham, M. H. Bhattacharyya,[†] E. S. Moretti,[†]
and D. J. Austin[§]

The gastrointestinal absorption of plutonium was studied using mice. The concentration of plutonium in the solutions administered (1×10^{-10} g/mL = 5 pCi/mL) was that for ^{239}Pu at its maximum permissible concentration in drinking water. The administered solutions contained hexavalent and tetravalent plutonium in 0.01 M bicarbonate, to simulate the compositions of treated (chlorinated) and untreated drinking water, and tetravalent plutonium in 0.01 M nitric acid and 0.17 M citrate, to provide absorption data that could be compared with those obtained in earlier investigations. The absorption of plutonium was found to be essentially independent of its oxidation state and the medium in which it was administered. The mean of the values obtained was 0.2%. This value is two orders of magnitude higher than those obtained in earlier investigations when rats were administered 0.01 M nitric acid solutions of tetravalent plutonium. The particular significance of this difference in results is that the data obtained from the earlier experiments were basic to the establishment of a gastrointestinal absorption factor of plutonium in man, and this factor was in turn used to set the MPC for plutonium in drinking water.

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

[†] Biological and Medical Research Division.

[§] Participant in the Undergraduate Research Program, Center for Educational Affairs.

CONTINUED STUDIES OF THE GASTROINTESTINAL ABSORPTION OF PLUTONIUM BY RODENTS

R. P. Larsen, R. D. Oldham, M. H. Bhattacharyya,* and E. S. Moretti*

An investigation is being made of the absorption of plutonium from the gastrointestinal tract of rodents. In the mouse it has been found to be essentially independent of the oxidation state of plutonium and the administration medium. In the rat the absorption was higher than it was in the mouse, but not appreciably so. The values obtained for both mice and rats are about two orders of magnitude higher than the value adopted for the gastrointestinal absorption factor for plutonium in man.

Introduction

The maximum permissible concentration (MPC) of plutonium in drinking water is to a major degree based on the results of experiments by Katz et al.¹ and Weeks et al.² in which 0.01 M nitric acid solutions of Pu(IV) ranging in concentration from 10^{-12} to 10^{-6} g/mL were administered to rats for a protracted period. The values obtained for G.I. absorption were 0.002 to 0.003% of the amounts administered. Weeks et al. also reported a value of 2.3% when Pu(VI) rather than Pu(IV) was administered. Since Larsen and Oldham³ had found that the form of plutonium in chlorinated drinking water is Pu(VI), we re-investigated the effect of plutonium oxidation state on gastrointestinal absorption. The results that we obtained, which were reported in last year's annual report,⁴ were significantly different from those of the earlier investigators. The value obtained for Pu(VI) was significantly lower than the value of Weeks et al. (0.17 vs. 2.3%) and the value for Pu(IV) was much higher than the values of Katz et al. and Weeks et al. (0.20 vs. 0.002 to 0.003%). The value obtained for Pu(IV) in citrate was not significantly different from those of earlier workers (0.25 vs. 0.15 to 0.3%).^{2,5,6}

There were differences between the experimental conditions of our studies and those of the earlier investigations.^{1,2} These were in the administration medium (0.01 M HCO_3^- to simulate Lake Michigan water vs. 0.01 M HNO_3), the

* Biological and Medical Research Division.

experimental animal (mouse vs. rat), and the feeding regimen (food-deprived vs. fed). We therefore carried out additional experiments in an attempt to resolve the discrepancies.

Experimental

In these experiments a solution of Pu(IV) in 0.01 M nitric acid was administered to fasted mice, a solution of Pu(VI) in 0.01 M bicarbonate was administered to fasted rats, and a solution of Pu(IV) in 0.17 M citrate was administered to fasted rats. There were 12 animals in each experiment. The 0.01 M nitric acid solution was prepared by evaporating a portion of the stock solution (in 8 M nitric acid) to incipient dryness, adding 0.01 mL of 1 M nitric acid, and adding 10 mL of water just prior to the administration. All the other experimental conditions, including the plutonium concentration, 10^{-10} g/mL, were the same as those previously reported.⁴

Results and Discussion

The results of these experiments, as well as the relevant results from our earlier experiments and from those of Weeks et al.² are presented in Table 1. From the data we have obtained it appears that (1) in the mouse, the gastrointestinal absorption of plutonium is essentially independent of its oxidation state and the medium in which it is administered, and (2) in the rat the absorption is higher than it is in the mouse, but not appreciably so.

Although the discrepancy has not been resolved, the explanation which we proposed in our previous report for the difference between our value for Pu(IV) in 0.01 M bicarbonate and their value for Pu(IV) in 0.01 M nitric acid still seems to be the most likely one: The plutonium in their solutions was polymeric. In the mouse, our values for Pu(IV) in 0.01 M nitric acid and 0.01 M bicarbonate were 0.20 and 0.17%, respectively; in the rat their value for Pu(IV) in 0.01 M nitric acid was 0.0028%. The difference in animals cannot be the reason for the difference in values. In the rat, our values for Pu(VI) in bicarbonate and Pu(IV) in citrate agree, each of these values is quite comparable with the corresponding value in the mouse, and in the mouse our values for Pu(VI) in bicarbonate, Pu(IV) in bicarbonate and Pu(IV) in citrate agree.

Table 1. Gastrointestinal Absorption of Plutonium in Fasted Mice and Rats.^a

Animal	Oxidation state	Medium	pH	Percent absorption ^b	Feeding regimen	Reference
Rat	IV	HNO ₃	2.0	0.0028 ± 0.0008	Fed	Weeks et al. ²
Mouse	IV	HCO ₃ ⁻	8.3	0.20 ± 0.02	Fasted	Larsen et al. ⁴
Mouse	IV	HNO ₃	2.0	0.17 ± 0.03	Fasted	This report
Mouse	VI	HCO ₃ ⁻	8.3	0.15 ± 0.03	Fasted	Larsen et al. ⁴
Rat	VI	HCO ₃ ⁻	8.3	0.32 ± 0.05	Fasted	This report
Rat	IV	Citrate	2.0	0.3	Fasted	Weeks et al. ²
Mouse	IV	Citrate	6.2	0.24 ± 0.05	Fasted	Larsen et al. ⁴
Rat	IV	Citrate	6.2	0.39 ± 0.06	Fasted	This report
Mouse	IV	HCO ₃ ⁻	8.3	0.20 ± 0.02	Fasted	Larsen et al. ⁴

^a There were 12 animals in each experiment.^b Errors are the standard deviations of the means.

The suggestion made in the previous report that the gastrointestinal absorption factor for man should be increased by two orders of magnitude appears to be warranted. This is substantiated by recent data from another laboratory. Sullivan et al.⁷ obtained values of 0.51% and 0.026% when a 0.01 *M* nitric acid solution of Pu(VI), 5×10^{-4} g/mL, was administered to starved and fed rats, respectively. (An explanation for the difference in these values may be that Pu(VI) was reduced to Pu(IV) in the G.I. tracts of the fed rats and Pu(OH)₄ · x H₂O⁸ precipitated in the small intestine because of the high concentration.) Sullivan⁸ obtained a value of 0.048% when a 0.01 *M* nitric acid solution of Pu(IV), 7×10^{-11} g/mL, was administered to fed rats. In this experiment there were five animals. Our experience has shown that this number is not sufficient.

The tissues of two mice given Pu(IV) in 0.01 M nitric acid were analyzed by another method to validate the results obtained in this and the earlier investigation. The primary method has been comparison of the amounts of ^{237}Pu found in the tissues with the amounts administered. This is done by placing a portion of the solution administered or the tissues (eviscerated bodies and livers) on a sodium iodide crystal and measuring the neptunium K x rays emitted in the decay of ^{237}Pu . Since the ^{237}Pu contained ^{236}Pu as a contaminant, and its concentration in the solution administered was known, determinations of the ^{236}Pu concentrations in the tissues could be made by the alpha-spectrometric isotopic dilution technique. After addition of a known amount of ^{242}Pu , the tissues were ashed, and the ash was dissolved in nitric acid. The plutonium was separated from the other sample constituents by anion exchange and it was then electrodeposited; the deposition was assayed in an alpha spectrometer.

For the two mouse livers that were analyzed by both methods, the fractional absorptions based on the ^{237}Pu analyses were factors of 1.02 and 1.03 times the absorptions based on the ^{236}Pu analyses. For the eviscerated bodies, the fractional absorptions based on the ^{237}Pu analyses were factors of 0.84 and 0.86 times those based on the ^{236}Pu analyses. These differences were undoubtedly the result of our failure to establish and use corrections for geometry and mass absorption when assaying the eviscerated bodies of the mice. Considering the size of the mouse, it was apparent that each of these corrections would be comparatively small. In assays of the eviscerated bodies of rats, the geometry-mass absorption correction was established and used.

References

1. J. Katz, H. A. Kornberg, and H. M. Parker, Absorption of plutonium fed chronically to rats, *Am. J. Roentgenol.* 73, 303-308 (1955).
2. M. H. Weeks, J. Katz, W. D. Oakley, J. E. Ballou, L. A. George, L. K. Bustad, R. C. Thompson, and H. A. Kornberg, Further studies on the gastrointestinal absorption of plutonium, *Radiat. Res.* 4, 339-347 (1956).
3. R. P. Larsen and R. D. Oldham, Plutonium in drinking water: effects of chlorination on its maximum permissible concentration, *Science* 201, 1008 (1978).
4. R. P. Larsen, R. D. Oldham, M. H. Bhattacharyya, E. S. Moretti, D. J. Austin, and C. G. Cacic, Progress report on the gastrointestinal absorption

- of plutonium in mice, Radiological and Environmental Research Division Annual Report, July 1978-June 1979, ANL-79-65, Part II. pp. 124-129.
5. J. Carritt, R. Fryxwell, J. Kleinschmidt, R. Kleinschmidt, W. Langham, A. San Pietro, R. Schaffer, and B. Schnap, The distribution and excretion of plutonium administered intravenously to the rat, J. Biol. Chem. 171, 273-283 (1947).
 6. D. W. Baxter and M. F. Sullivan, Gastrointestinal absorption and retention of plutonium chelates, Health Phys. 22, 785-786 (1972).
 7. M. F. Sullivan, J. L. Ryan, L. S. Gorham, and K. M. McFadden, The influence of oxidation state on the absorption of plutonium from the gastrointestinal tract, Radiat. Res. 80, 116 (1979).
 8. M. F. Sullivan, Absorption of actinide elements from the gastrointestinal tract of rats, guinea pigs, and dogs, Health Phys. 38, 150-171 (1980).

THE GASTROINTESTINAL ABSORPTION OF PLUTONIUM IN THE DOG

R. E. Toohey, M. H. Bhattacharyya,^{*} R. D. Oldham, R. P. Larsen,
and E. S. Moretti^{*}

The gastrointestinal absorption of plutonium in the beagle has been determined to be $0.066 \pm 0.014\%$ of the amount administered. This result is quite comparable with the results reported for the dog by other workers, and a factor of 3 smaller than that observed by us for mice. On the average, the retained plutonium was found to be almost equally divided between the liver and the skeleton.

Introduction

Several experiments have been carried out in this laboratory to determine the appropriateness of the maximum permissible concentration of plutonium in drinking water.^{1,2} In general, the gastrointestinal absorption of plutonium by rodents has been found to be independent of the oxidation state of the plutonium, and of the medium in which it was administered. The mean value for mice under all experimental conditions was found to be $0.2 \pm 0.1\%$ of the amount administered, while that for rats was $0.3 \pm 0.1\%$. Because of obvious species differences in the length of the gastrointestinal tract, it was decided to determine the G.I. absorption value in a larger mammal, the dog.

Materials and Methods

The plutonium used was a mixture of ^{237}Pu and ^{239}Pu . The ^{237}Pu can be easily assayed by counting with an NaI(Tl) crystal the Np K x rays which are emitted following electron capture. A 0.01 M bicarbonate solution of plutonium in the +6 oxidation state was prepared as described for the previous experiments,¹ with the exception that the solution contained 1 ppm Cl_2 to simulate chlorinated drinking water.

A gelatin capsule containing 1.0 mL of the plutonium solution was administered to each of six adult male beagles following a 21-hr fast. Food was returned to the dogs three hours later. Feces were collected from each dog

^{*} Biological and Medical Research Division.

until no detectable ^{237}Pu could be observed by photon counting of a bulk sample. The feces from each dog were then combined, ashed at 450°C in a muffle furnace, and dissolved in concentrated HNO_3 . The solution was brought to 200 mL and adjusted to 8 N HNO_3 , and placed in a 200-ml polyethylene bottle. The activity in the sample was determined by placing the bottle directly on the window of an inverted 199-mm diameter "phoswich" detector which consisted of a 3-mm-thick NaI(Tl) crystal optically coupled to a 50-mm-thick CsI(Tl) crystal and viewed by a 180-mm diameter phototube. (The CsI(Tl) crystal is employed as an anti-coincidence shield, resulting in high sensitivity and relatively low background for low-energy ($< 150\text{ keV}$) photon counting.) After correction for radioactive decay since the time of administration, the net counting rate in the energy band 80 to 130 keV was taken as a direct measure of the amount of plutonium administered. The average counting rate from the feces was $93,000 \pm 300\text{ cpm}$. The background counting rate from the feces of a control dog was $10.1 \pm 0.3\text{ cpm}$, not significantly different from a blank solution.

The dogs were sacrificed four weeks after plutonium administration, skinned and eviscerated. The livers and gall bladders were retained, and the skeletal muscles were then removed. The skeletons were divided into six portions as follows: skull; cervical and thoracic vertebrae; lumbar and sacral vertebrae and pelvis; ribs and scapulae; femora and humeri; and remaining skeleton, consisting of the tibiae, fibulae, radii, ulnae, feet, tail, sternum, trachea, larynx, and os penis. The liver, gall bladder and each portion of the skeleton were dry ashed, dissolved in HNO_3 and solutions prepared as for the feces. The skull and skeletal remains were divided into two portions, since not all the ash could be dissolved in 200 mL. Each sample was counted on the phoswich detector for at least 100 min.

The background was determined by counting samples prepared in identical fashion from a control dog. The counting rates from these ranged from 10.2 ± 0.1 to $11.8 \pm 0.2\text{ cpm}$, with a mean of 11.3 ± 0.3 . Because of this small range, the mean value was used as the background and subtracted from the counting rate from each sample. The net counting rates were again corrected for decay, and each was divided by the counting rate obtained from the feces of that dog

in order to determine the absorbed fraction of the administered dose.

In order to check the accuracy of this technique, the uptake in the lumbar and sacral vertebrae and the pelvis of dog 3657 was determined by measuring the ^{236}Pu content of the ashed and dissolved sample via isotope dilution alpha spectrometry. (The ^{236}Pu was an impurity in the ^{237}Pu .) The result was $0.061 \pm 0.003\%$ of the administered dose, which compared quite well with the value of $0.067 \pm 0.002\%$ determined by photon counting of the ^{237}Pu in the sample.

Results and Discussion

The results are presented in Table 1. The extraordinarily high uptake by dog 3657 is unexplained, and the values for this animal have been excluded from the means. The mean value of 1.1 ± 0.6 for the ratio of liver to skeleton is consistent with that of 1.0 suggested in ICRP Report 19,³ but the large standard error is indicative of the biological variability encountered in this experiment.

There are two other reported values for the gastrointestinal absorption of plutonium by beagles. In the experiments of Buldakow et al.,⁴ adult dogs were administered Pu(IV) citrate and the retained amount was determined 10 days post-administration. The amount retained was 0.064 ± 0.014 , and the ratio of the amount of plutonium in the liver to that in the skeleton was 1.07 ± 0.03 .

The other experiment was that of Bulou et al.,⁶ in which a single female beagle was administered 580 μCi of $^{239}\text{Pu(IV)}$ citrate in gelatin capsules, and the absorption was measured three days later. The total absorbed was 0.083% of which 0.04% was in the skeleton and 0.02% in the liver. These results are essentially identical to those observed for dog 3657 in our experiment.

The distribution of plutonium within the skeleton is presented in Table 2. The observed distribution is quite similar to that reported by Stover et al.⁵ following intravenous injection of Pu(IV) citrate. The distribution is also comparable to that observed in the human skeleton, in which plutonium is found primarily in the axial skeleton, with very little in the appendicular skeleton.^{7,8} One exception, however, is the concentration found in the skull of the dog. In this experiment, negligible amounts of plutonium were found in the skull, while in the human, the concentration of plutonium in the skull was found to be the

Table 1. Gastrointestinal absorption of plutonium in the dog, expressed as fraction of the administered dose. All entries have been multiplied by 10^5 . Values for dog 3657 have been omitted from the means.

Dog	L & GB ^a	Skeleton	Total	L & GB/Skel.
3640	19.3 ± 1.0	24.7 ± 2.3	44.1 ± 2.5	0.78 ± 0.08
3652	27.2 ± 1.1	40.1 ± 2.8	67.5 ± 3.0	0.67 ± 0.05
3657	103.0 ± 1.4	271.2 ± 3.6	374.2 ± 3.9	0.38 ± 0.01
3658	39.5 ± 1.7	26.7 ± 3.2	66.2 ± 3.5	1.48 ± 0.19
3661	44.1 ± 1.5	22.0 ± 3.1	66.2 ± 3.4	2.00 ± 0.29
3663	35.7 ± 0.9	47.9 ± 3.3	83.6 ± 3.5	0.75 ± 0.06
Mean	33.3 ± 9.9	32.3 ± 11.2	65.5 ± 14.1	1.14 ± 0.58

^a L & GB = Liver plus gall bladder

same as the mean concentration for the entire skeleton.

Summary and Conclusions

Although the value for the gastrointestinal absorption of plutonium by the dog is only one-third of that observed for the mouse, our value of $0.066 \pm 0.014\%$ is more than twenty times the value of 0.003% adopted for man by the ICRP.³ On the average, the absorbed plutonium has been found to be almost equally partitioned between the liver and the skeleton, and the distribution within the skeleton is that to be expected if the plutonium is deposited on bone surfaces.^{8,9} This distribution of plutonium in the dog has also been observed following both injection and inhalation,^{5,6} and thus the metabolism of Pu(VI) following oral administration to the fasting dog does not differ from that of Pu(IV) following other routes of administration.

Table 2. Fraction of total skeletal burden found in each sample.^a

Sample ^b	Dog number					
	3640	3652	3658	3661	3663	Mean
Skull A	-0.06 ± 0.03	0.002 ± 0.022	-0.07 ± 0.04	-0.05 ± 0.05	-0.05 ± 0.03	-0.05 ± 0.03
Skull B	-0.01 ± 0.02	0.001 ± 0.023	-0.06 ± 0.03	-0.005 ± 0.05	0.01 ± 0.03	-0.01 ± 0.03
CTV	0.35 ± 0.05	0.32 ± 0.03	0.27 ± 0.06	0.38 ± 0.08	0.37 ± 0.04	0.34 ± 0.04
LSV + P	0.31 ± 0.05	0.26 ± 0.03	0.18 ± 0.05	0.31 ± 0.06	0.30 ± 0.03	0.27 ± 0.06
R + S	0.42 ± 0.05	0.32 ± 0.03	0.43 ± 0.07	0.46 ± 0.07	0.32 ± 0.04	0.39 ± 0.07
F + H	0.10 ± 0.03	0.09 ± 0.03	0.004 ± 0.04	-0.001 ± 0.05	0.10 ± 0.02	0.06 ± 0.05
Sk Rm A	-0.08 ± 0.03	-0.02 ± 0.02	0.15 ± 0.05	-0.11 ± 0.04	-0.02 ± 0.03	-0.02 ± 0.10
Sk Pm B	-0.03 ± 0.03	0.02 ± 0.03	0.10 ± 0.05	0.005 ± 0.04	-0.03 ± 0.02	0.01 ± 0.05

^aNegative values in the table result from subtraction of an average background.

^bCTV = cervical and thoracic vertebrae; LSV + P = lumbar and sacral vertebrae plus pelvis; R + S = ribs and scapulae; F + H = femora and humeri; Sk Rm = remainder of skeleton.

References

1. R. P. Larsen, R. D. Oldham, M. H. Bhattacharyya, E. S. Moretti, D. J. Austin, and C. G. Cacic, Progress report on the gastrointestinal absorption of plutonium in mice, Argonne National Laboratory Radiological and Environmental Research Division Annual Report, July 1978-June 1979, ANL-79-65, Part II, pp. 124-129 (1979).
2. R. P. Larsen, R. D. Oldham, M. H. Bhattacharyya, and E. S. Moretti, Continued studies of the gastrointestinal absorption of plutonium by rodents, this report.
3. International Commission on Radiation Protection, Publication 19, The metabolism of compounds of plutonium and other actinides, Pergamon Press, London, p. 49 (1972).
4. L. A. Buldakov, E. R. Lyubchanskii, Y. I. Moskalev, and A. P. Nifatov, Problems of plutonium toxicology, Atom Publication Moscow, 1969, LF-tr-41, prepared for the U.S. Atomic Energy Commission (1970).
5. B. J. Stover, D. R. Atherton, and D. S. Buster, Retention of $^{239}\text{Pu}(\text{IV})$ in the beagle, in Radiobiology of Plutonium, B. J. Stover and W.S.S. Jee, Eds., J. W. Press, Salt Lake City, pp. 149-169 (1972).
6. J. E. Ballou, J. F. Park, and W. G. Morrow, On the metabolic equivalence of ingested, injected, and inhaled ^{239}Pu citrate, Health Phys. **22**, 857 (1971).
7. R. P. Larsen, R. D. Oldham, C. G. Cacic, J. E. Farnham, and J. R. Schneider, Distribution of injected plutonium in the skeleton and certain soft tissues, Argonne National Laboratory Radiological and Environmental Research Division Annual Report, July 1977-June 1978, ANL-78-65, Part II, pp. 145-153 (1978).
8. R. P. Larsen, R. D. Oldham, and S. M. Martin, Distribution of plutonium among and within selected bones from an injection case, Argonne National Laboratory Radiological and Environmental Research Division Annual Report, July 1978-June 1979, ANL-79-65, Part II, pp. 115-119 (1979).
9. W.S.S. Jee, ^{239}Pu in bones as visualized by photographic and neutron-induced autoradiography, Radiobiology of Plutonium, B. J. Stover and W.S.S. Jee, Eds., J. W. Press, Salt Lake City, pp. 171-193 (1972).

ENERGY DEPENDENCE OF THE EFFECTIVE SOFT TISSUE THICKNESS *

R. E. Toohey

The concept of "effective soft tissue thickness" (ESTT) was proposed by Rundo et al.¹ as a method for the calibration of lung counting systems for the detection of plutonium in vivo. ESTT is defined as that thickness of tissue-equivalent absorber which reduces the counting rate from a bare point source at 100 mm from a detector to that observed from the same amount of activity in the lungs, as measured with the same detector in vivo. It is important to note, however, that when the source in question is a low-energy photon emitter, such as plutonium, the additional absorption in vivo by the rib cage must be taken into account. The values of ESTT were determined in Ref. 1 for seven subjects who had inhaled an aerosol labelled with ^{103}Pd and ^{51}Cr ("mock plutonium"). It was found that ESTT could be related to the "mean tissue thickness" (MTT) of Ramsden et al.² by the equation

$$\text{ESTT} = 30 \text{ mm} + 0.9 \text{ MTT} \quad (1)$$

MTT was determined by ultrasonic measurements of the soft tissue overlying the rib cage and was related to the weight (W), height (H), and chest circumference (CC) as follows

$$\text{MTT (mm)} = 1.53 W(\text{kg}) / H(\text{m}) - 10 \text{ CC}(\text{m}) - 35.5 \quad (2)$$

Thus, a lung counting system can be calibrated with a point source and absorber, once the weight, height, and chest circumference of the subject are known. This method was successfully used to calibrate our 180-mm diameter xenon-filled proportional counter for ^{103}Pd during the 1972 IAEA "mock plutonium" inter-calibration experiment.^{3,4}

Since the definition of ESTT was based on experiments following the inhalation of ^{103}Pd , which emits 20.2- and 22-keV x rays, there is a question about the applicability of ESTT to plutonium, whose principal x-ray emission is at 17 keV. Thus, the energy dependence of ESTT, if any, needs to be investigated.

* Summary of a paper presented at the LASL/DOE Instrumentation Workshop for Low-Level Transuranic Measurements Applied to in Vivo and Environmental Monitoring, Los Alamos, NM, 4-6 March 1980.

The only way to establish the behavior of ESTT with photon energy is to determine the actual values of ESTT in vivo in the same subject who, at different times, has inhaled radioactive aerosols with different photon energies. This opportunity arose for one of the participants in the 1972 intercalibration experiment, subject DN, who inhaled an aerosol labelled with ^{92m}Nb in November 1979. The inhalation took place at another laboratory, and the subject subsequently visited ANL, where the photon emission from his chest was measured with the proportional counter. ^{92m}Nb emits zirconium K x rays at 15.8 keV. The calculated ESTT value for this subject was 40 mm in 1972.

The experimental ESTT can be derived as follows

$$\text{ESTT} = -\frac{1}{\mu} \ln \{ \epsilon_{\text{in vivo}} / \epsilon_{\text{pt source}} \} , \quad (3)$$

where μ is the linear attenuation coefficient (energy-dependent) of the tissue-equivalent material in mm^{-1} and ϵ is the observed counting efficiency, in counts per photon. For subject DN with ^{103}Pd in 1972,

$$\text{ESTT} = -\frac{1}{0.066} \ln \{ 1.07 \times 10^{-3} / 1.72 \times 10^{-2} \} = 42 \text{ mm at } 20.2 \text{ keV}.$$

The agreement between this value and the calculated value of 40 mm is not surprising, since DN was also one of the volunteers in the experiment which originally determined the ESTT formula (Eq. 1).

For subject DN with ^{92m}Nb in 1979, the calculated value of ESTT was 41 mm, while the experimental value was

$$\text{ESTT} = -\frac{1}{0.11} \ln \{ 2.27 \times 10^{-4} / 2.22 \times 10^{-2} \} = 42 \text{ mm at } 15.8 \text{ keV}.$$

Thus the value of ESTT does not change with photon energy.

It must be mentioned, however, that ^{92m}Nb also emits 934-keV γ rays. These photons enable the subject's true content of ^{92m}Nb to be determined with standard whole-body counting techniques. They also result in a sizeable scattering background under the x-ray peak. The value of $\epsilon_{\text{in vivo}}$ depends on how this scattering contribution is corrected for, and therefore, the value of ESTT is also affected. A value of ESTT of as much as 46 mm can be obtained,

depending on the method of determining the scatter contribution. Since the estimated error on the calculated value of ESTT (Eq. 1) is $\pm 14\%$,¹ however, 46 mm is not significantly different from 42 mm.

References

1. J. Rundo, K. Rudran, and B. T. Taylor, Effective tissue thicknesses for external counting of low energy emitters in lung, *Health Phys.* 17, 155 (1969).
2. D. Ramsden, C. O. Peabody, and R. G. Speight, The use of ultrasonics to investigate soft tissue thicknesses on the human chest, United Kingdom Atomic Energy Authority Report AEEW-R493 (1967).
3. J. Rundo, K. F. Eckerman, R. E. Tookey, and M. A. Essling, The measurement of "mock plutonium" in vivo, Argonne National Laboratory Radiological and Environmental Research Division Annual Report, July 1972-June 1973, ANL-8060, Part II, pp. 228-241 (1974).
4. D. Newton, F. A. Fry, B. T. Taylor, M. C. Eagle, and R. C. Sharma, Interlaboratory comparison of techniques for measuring lung burdens of low-energy photon emitters, *Health Phys.* 35, 751 (1978).

STATUS AND TRENDS IN THE EXTERNAL COUNTING OF INHALED HEAVY ELEMENTS DEPOSITED IN VIVO*

K. L. Swinth,[†] W. J. Bair,[†] P. N. Dean,[‡] J. Rundo, and
F. K. Tomlinson[§]

External counting has been routinely used for estimation of plutonium in vivo for approximately 10 years. However, this method is fraught with inherent uncertainties resulting from the few radiations and the severe attenuation of the radiations. Present counting capability allows detection of from 1/2 to 1 Maximum Permissible Lung Burden (16 nCi). Current efforts in the development of counters and in intercalibration may lead to small improvements in detection limits and accuracy, but substantial improvements are not expected. Two troublesome areas in the in vivo counting area are the non-uniform distribution of material within the lungs and the influence of material translocated from the primary deposition site. New concepts such as induced nuclear fission of the deposited material can possibly lead to improvements in accuracy and in the detection limit.

* Abstract of a paper published in Health Phys. 37, 641-657 (1979).

[†] Battelle Pacific Northwest Laboratories, Richland, WA 99352.

[‡] Lawrence Livermore Laboratory, Livermore, CA 94550.

[§] Monsanto Research Corporation, Mound Laboratory, Miamisburg, OH 45342.

IDENTIFICATION THROUGH OSTEOMETRIC DATA OF THREE RADIUM-BURDENED SKELETONS

J. E. Farnham and J. W. Forkal

The skeletal remains of four persons of uncertain identity were disinterred from a family grave. The weight and descriptive morphological traits of each bone were recorded, as were the lengths of the long bones. Analyses of these data, combined with information obtained from medical records and disinterment reports, led to the specific identification of each skeleton.

Introduction

One of the Center for Human Radiobiology's responsibilities is the long-range study of persons who have been exposed to bone-seeking radionuclides. For some years the Center has had a program to exhume the remains of persons exposed to radium and mesothorium, and many exhumations have been carried out. Occasionally there is difficulty identifying a set of remains. In the present case, four skeletons were removed from a common family grave known to be the burial site of two radium dial painters of interest to our studies. The individual identities of the skeletons were unknown. This report describes the use of osteometric and descriptive morphological data to solve the identity problem. Gamma-ray analyses for ^{226}Ra present in a set of bones were all below 1 nCi, which is generally considered to be below the lower limit of detectability. Thus, the radioactivity measurements were of no help in determining the identity of each case.

Materials and Methods

Two radium-exposed females were discovered to have been buried with three other family members in a single, three-grave cemetery plot. The available records indicate the burials occurred in the years 1886, 1912, 1924, 1930, and 1943. The records do not specify the location of each person's remains within the grave plot, but they do indicate the persons buried in the years 1886 and 1924 to be males. The two radium-exposed females painted luminous watch dials around the year 1918.

After obtaining disinterment permissions from all living next-of-kin and a permit from the State Board of Health, a team from the Center exhumed the skeletonized remains of four persons buried in the plot. A fifth person was identified by remaining clothing as being male and was left undisturbed. The exhumed skeletal remains were taken to the Center's research laboratory for identification, osteometry, roentgenographic studies, and gamma-ray measurements to determine the ^{226}Ra and ^{228}Ra content of the bones.

Determination of age, sex, race, etc., from skeletal remains has been the subject of numerous investigations by physical anthropologists for many years.^{1,2} Sex determinations are based on descriptive morphology and/or morphometry of various bones. Our study used a combination of morphological traits and osteometric data patterned after the methods of several authors. The morphological traits of the bones studied or observed for this report are given in Table 1, together with references to descriptive sources. Our experience with over 100 other disinterred skeletal remains also played an important role in the identification process.¹¹⁻¹³

Osteometric data were obtained on all bones recovered from the grave site. The weights were determined on either a Mettler PS15 or PR-1200 top-loading balance, reading to an accuracy of ± 1.0 or ± 0.01 g, respectively. Maximum lengths and other measurements were obtained using a standard osteometric board which read to an accuracy of 0.1 cm. The uncertainty of our measurements is 1% or less. Radiographs of the bones were obtained on Kodak NS medical x-ray film. The radiographic exposures were made with a Portable Industrial X-Ray Unit, Picker #6191. The kVp and mAs were varied to yield the appropriate exposure for each bone. Measurements of bone structures as visualized on the radiographs were made using a Carl Zeiss Mop-3 Digital Image Analyzer System.

The bones from each skeleton were air-dried and cleaned by rubbing with a soft bristle brush before osteometric data and roentgenograms were taken. Five control skeletons (two female and three male) were examined at the same time as the study group. One control skeleton was from a sister of the three females under study. Table 2 indicates the percentage of each skeleton recovered at disinterment and the total dry, fat-free weight of all bones present.

Table 1. Morphological traits of the bones studied

Bone observed or measured	Trait	Reference
Long bones		
Humerus	Maximum length	Trotter ³
	Diameter of head	Dwight ⁴
Ulna	Maximum length	Trotter ⁵
Radius	Maximum length	Trotter ⁵
Femur	Maximum length	Trotter ⁵
	Diameter of head	Pearson ⁶
Tibia	Maximum length	Trotter ⁵
Sacrum	Curvature	Bass ²
	Promontory width versus ala width	Anderson ⁷
Pelvic	Subpubic angle	Bass ²
	Ventral arc	Bass ² ; Phenice ⁸
	Subpubic concavity	Bass ² ; Phenice ⁸
	Ischiopubic ramus, medial	Schultz ⁹ ; Montagu ¹⁰
	Sciatic notch	Bass ² ; Krogman ¹
	Sacroiliac joint	Bass ²
	Preauricular sulcus	Bass ²
	Obturator foramen	Bass ²
Skull	Supraorbital ridges	Bass ² ; Krogman ¹
	Occipital ridges	
	Zygomatic processes	
	Mastoid processes	
	Frontal sinuses	
Mandible	Chin shape	Bass ²
Vertebra	Osteoarthritic lipping	Bass ² (pp. 213-214)

Table 2. Skeleton identification data

Skeleton	Sex	Estimated percent of entire skeleton recovered	Total weight of bones recovered, (g)	Estimated weight of total skeleton, (g)	Estimated weight normalized to dry, fat-free, ^a (g)
A	-	99	3640	3673	3673
B	-	20	568	2869	2869
C	-	89	2606	2928	2928
M	-	71	1807	2559	2559
D (05-349)	F	99	2982	3012	3012
E (03-779)	F	100	4277	4277	4277
F (01-208)	M	100	7700 ^b	7700 ^b	4697
G (10-831)	M	100	7791 ^b	7791 ^b	4753
H (03-238)	M	99	6377 ^b	6441 ^b	3929

^a See Reference 13.^b Weight is with moisture and fat.

Results

Osteometric and descriptive morphological data are given in Table 3 for all bones that were available for measurement. Most investigators agree that no single bone or morphological trait is always accurate for sex determination, and usually no complete skeleton will have only those traits associated with one sex. Any given skeleton has a mixture of sexual traits, but the traits of one sex predominate.

Pelvic Criteria

The innominate bone in the female features a wide subpubic angle; this angle in the male is narrow. Skeletons A and the control males have a narrow (about 34°) subpubic angle, and all four lack a ventral arc or subpubic concavity. The latter traits are present in cases C, M, D, and E. Other traits, such as the sciatic notch, the medial aspect of the ischiopubic ramus, and the

Table 3. Osteometric and descriptive data of bones measured.

Bone measured or observed	Skeletons of unknown sex				Sister of unknowns	Controls	
						Female	Male
Long bones:	A	B	C	M	D	E	F
Length of humerus, cm	33.6	--	32.7	32.2	31.5	30.6	33.0
Diameter of head, mm	47.0	--	43.6	40.0	42.5	42.6	46.6
Length of ulnae, cm	26.3	--	--	23.8	24.8	23.8	26.9
Length of radii, cm	24.8	--	--	21.5	23.1	22.1	25.1
Length of femora, cm	46.1	--	45.0	46.1	43.8	43.1	44.9
Diameter of head, mm	48.1	--	46.1	43.1	44.9	44.0	50.4
CCT ^a , mm	17.8	--	16.5	9.3	12.5	18.5	18.0
CI ^b	0.64	--	0.67	0.37	0.43	0.66	0.56
Sacrum: AP curvature	Sharp	--	--	--	Blunt	Blunt	Sharp
Pelvis:							
Subpubic angle	34°	--	52°	57°	65°	65°	33°
Ventral arc	No	--	Yes	Yes	Yes	Yes	No
Subpubic concavity	No	--	Yes	Yes	Yes	Yes	No
Medial aspect IP ramus	Broad	--	Narrow	--	Narrow	Narrow	Broad
Sciatic notch	Narrow	Broad	Broad	Broad	Broad	Broad	Narrow
Sacroiliac joint	--	Raised	Flat	Raised	Flat	Raised	Flat
Preauricular sulcus	Slight	Prom.	Prom.	Prom.	Prom.	Prom.	Slight
Skull:							
Eye orbits	Blunt	Sharp	Sharp	--	Blunt	Blunt	Blunt
Supraorbital ridges	Bump	Flat	Flat	Flat	Flat	Flat	Bump
Occipital muscle ridge	Large	Large	Small	Small	Large	Medium	Medium
Post-zygomatic processes	Post.	Ant.	Ant.	Ant.	Ant.	Post.	--
Mastoid processes	Large	Small	Small	Small	Large	Small	Large
Frontal sinuses ^c	0.93	1.13	1.25	1.01	1.08	--	0.62
Mandible	Flat	--	Flat	Pointed	Flat	Pointed	Flat

^a Combined cortical thickness.

^b Cortical index, CI = cct/total diameter at midshaft.

^c Width of largest sinus divided by maximum height.

preauricular sulcus, appear quite different between the known male and female skeletons. A prominent preauricular sulcus is seen in skeletons C, M, and the control female. In contrast, only a slight depression is seen in the known male and case A innominates. A narrow sciatic notch is present in skeletons A and F, but is broad in the other cases. The medial aspect of the ischiopubic ramus is broad in cases F and A, and narrow in C and the two known female innominates D and E. Two other traits observed, a ventral arc and a subpubic concavity,

are present in the skeletons of cases C, M, D, and E, but lacking in cases A and F.

Sacrum

Two morphological features of the sacrum are distinctively different between the sexes, anterior-posterior curvature and body width versus ala width. As noted in Table 3, the sacrum from cases F and A has a sharp curvature, whereas a blunt curvature is indicated for the two known females. The sacrum bone from unknown cases B, C, and M is too eroded for an accurate assessment.

Skull

Bass² indicates the supraorbital ridges are more prominent in males and the upper edges of the eye orbits of males are blunt. The skulls from case A and control case F both have a bump at the supraorbital ridge, while the other skulls appear flat in this region. The eye orbits are blunt in both male and female controls used in this study. Large mastoid processes are present in skulls A, D, and F, while the other skulls have small processes. The two other features observed for this study (occipital muscle ridges and position of the posterior end of the zygomatic processes) do not correlate with the known sexes of the controls.

The frontal sinuses of all skulls in this series were measured using a specialized technique. A Caldwell's projection radiogram was produced for each skull. Several parameters of the projected frontal sinus images were measured with a Carl Zeiss MOP-3 Modular System for Quantitative Digital Image Analysis. These parameters and the values are presented in Table 4. The maximum height and width parameters are the same as those described by Schüller.¹⁴ Other measurements include the area and maximum diameter of each sinus.

The total frontal sinus area (left plus right) of each control male skull is smaller than the area of either the control female or the unknown females. The data suggest females have a larger (though more scalloped) frontal sinus area than males, a conclusion opposite the usual textbook statement. Various indices were calculated using the data shown in Table 4. An attempt was made to correlate the index values with known sex. We report only the index value of the width of the larger of the two frontal sinuses divided by the maximum

Table 4. Osteometry of frontal sinuses

Case No.	Projected area, mm ² ± S.D. ^a			Maximum diameter mm ± S.D.	Largest sinus <u>Width</u> Height
	Left	Right	Both		
A	1123 ± 84	720 ± 79	1843	38.6 ± 5.9	0.928
B	1051 ± 65	958 ± 244	2009	43.8 ± 4.0	1.126
C	532 ± 48	938 ± 122	1470	30.8 ± 3.3	1.249
M	469 ± 88	641 ± 41	1110	34.0 ± 4.8	1.006
D	664 ± 82	1211 ± 79	1875	47.2 ± 7.5	1.083
F	563 ± 79	381 ± 58	944	31.3 ± 8.4	0.621
G	426 ± 46	410 ± 28	836	31.9 ± 6.6	0.845
H	593 ± 47	621 ± 9.0	1214	35.4 ± 2.0	0.719

^a At least three measurements were taken on each radiograph.

height. The three known male skulls, plus skull A, have a frontal sinus index of less than 1, whereas the other skulls in the series, including the known female, have an index greater than 1. This small sampling of frontal sinus area in males and females does not justify a conclusion of the frontal sinuses of females having a larger area than males.

Mandible

The shape of the mental protuberance (chin) was observed and recorded for each skull in the series. Bass² indicates "the chin is more square in males and rounded with a point in the midline in females." The mandible of one of the two known females, plus one other (M), have pointed chins.

Long Bones

Definite skeletal sexing is not possible through the measurement of long bones alone, but a high degree of accuracy is obtained when combined with measurements of other bones and morphological descriptions. Krogman gives the percentage of accuracy for adult material (Ref. 1, p. 149). Most authors agree that the male bones are generally larger and more massive than female bones.

Some measurement data on long bones, especially the femur and humerus, is available in the literature (see Table 1). In this study, the lengths of the long bones are useful for stature estimations, which in turn can be compared with the medical records of these cases when available. The estimated stature and the estimated total skeletal weight of each subject used in this study is given in Table 5. Estimates of the stature were calculated using the formula given by Trotter and Gleser.⁵ The values reported are arithmetic means based on calculations derived using the lengths of several long bones. The total skeletal weights reported are calculated according to the fractional weight of various bones as reported by Farnham and Forkal.¹³ The three known males and unknown subject A all have a tall estimated stature, which suggests that of the four unknown subjects, skeleton A is most probably a male.

Table 5. Estimated stature and skeletal weight of subjects.

Case	CHR No.	Medical record		Estimated stature, ^a cm $\pm$ S.D.	Estimated skeletal weight, ^a kg
		Height, cm	Body weight, lbs		
A	-	-	-	172.4 $\pm$ 2.7	3.673
B	-	-	-	-	2.869
C	01-636	-	-	166.6 $\pm$ 1.3	2.928
M	00-034	-	-	164.3 $\pm$ 6.0	2.559
D	05-349	-	-	164.4 $\pm$ 2.2	3.012
E	03-779	163	140	160.9 $\pm$ 1.8	4.277
F	01-208	170	182	171.9 $\pm$ 4.1	4.697
G	10-851	183	180	180.0 $\pm$ 2.1	4.753
H	03-238	183	200	181.0 $\pm$ 1.5	3.929
Std. male ^b					4.40
Std. female ^b					3.20

^a See Ref. 13.

^b See Ref. 15.

During disinterment, photographs and records were taken to show the grave site, vegetation, horizontal profile of earth, soil compaction, position of skeletal remains on floor of the grave, etc. Analyses of the disinterment, osteometric and observation (descriptive morphological traits) data lead to the following conclusions: (1) Case A of the four unknowns is a male, most probably the brother of the three sisters, who was buried in 1924. The ^{226}Ra body burden was 0.2 ± 0.6 nCi; (2) Since cases B and M were buried one over the other, and the sex of both skeletons was female, the uppermost remains were a more recent burial. Furthermore, a nameplate was found near these two skeletons which identified the uppermost as CHR patient 00-034. This patient was buried in 1943 and had a terminal body burden of 0.6 ± 0.9 nCi ^{226}Ra ; (3) Because of the condition of the skeleton and its burial depth, case B is most probably the sister who was buried in the year 1912. The body burden measurement, extrapolated to a complete skeleton, was 0.0 ± 1.0 nCi ^{226}Ra ; (4) The remaining case C was identified as the female (CHR patient 01-636) buried in 1930. The measured body burden was 0.7 ± 0.7 nCi ^{226}Ra .

References

1. W. M. Krogman, The Human Skeleton in Forensic Medicine, Charles C. Thomas, Springfield, IL (1962).
2. W. M. Bass, Human Osteology: A Laboratory and Field Manual of the Human Skeleton, David R. Evans, Ed., Missouri Archaeological Society, Columbia, Missouri (1971).
3. Mildred Trotter and G. C. Gleser, A re-evaluation of estimates of stature based on measurements of stature taken during life and of long bones after death, *Am. J. Physiol. Anthropol.* 16, 79-123 (1958).
4. Thomas Dwight, The size of the articular surfaces of the long bones as characteristic of sex: An anthropological study, *Am. J. Anat.* 4, 19-31 (1905).
5. Mildred Trotter and G. C. Gleser, Estimation of stature from long bones of American whites and Negroes, *Am. J. Physiol. Anthropol.* 10, 463-514 (1952).
6. K. Pearson, A study of the long bones of the English skeleton. I. The femur, Draper's Company Research Memoirs, U. of London, Biom. Ser. X, Ch. 1-4 (1917-1919).
7. J. E. Anderson, The Human Skeleton: A Manual for Archaeologists, National Museum of Canada, p. 142 (1962).
8. T. W. Phenice, A newly developed visual method of sexing the os pubis, *Am. J. Physiol. Anthropol.* 30, 85-88 (1967).

9. A. H. Schultz, The skeleton of the trunk and limbs of higher primates, *Human Biology* 2, 303-438 (1930).
10. Ashley Montagu, An Introduction to Physical Anthropology, Charles C. Thomas, Springfield, IL (1960).
11. J. E. Farnham, K. M. Rybicki, J. Sonza-Novera, and W. VonSeggen, A preliminary report on the microradiographic appearance of exhumed bone, Radiological and Environmental Research Division Annual Report, July 1972-June 1973, ANL-8060, Part II, pp. 255-267.
12. J. E. Farnham, K. Martens Rybicki, and M. L. Johnson, Anthropometry on a portion of the skeletal collection in the Center for Human Radiobiology, Radiological and Environmental Research Division Annual Report, July 1970-June 1971, ANL-7860, Part II, pp. 138-151.
13. J. E. Farnham and J. W. Forkal, Osteometry of the skeletal remains of 101 radium cases, this report.
14. A. Schüller, A note on the identification of skulls by x-ray pictures of the frontal sinuses, *Med. J. Australia* 1, 554-556 (1943).
15. International Commission on Radiological Protection, Report of the Task Group on Reference Man, Pergamon Press, New York (1975).

OSTEOMETRY OF THE SKELETONS OF 101 HUMAN RADIUM CASES

J. E. Farnham and J. W. Forkal

Osteometry has been performed on the skeletal remains of 101 persons in the radium studies population. The measured values for lengths and weights of various bones are reported, and values for the estimated total skeletal weight and height are calculated. Further, the data have been normalized to a dry, fat-free skeleton and the estimated total skeletal weight recalculated. The mean values reported did not differ significantly between this study group and normal values reported in the literature. These findings indicate no detectable effect of large skeletal doses of radiation on the stature or total skeletal weight of these individuals.

Introduction

The Bone Microdosimetry Group of the Argonne Center for Human Radiobiology (CHR) continues, as an ongoing project, to compare anthropometrically the adult body size of our radium study population with the size reported in normal populations.¹⁻⁵ Our growing skeletal collection is being derived from the CHR exhumation and willed-body programs. The collection also includes bones from autopsies and surgeries. The exhumed and willed remains provide the bulk of our most complete osteometric data. The CHR collection thus consists of bones (in various states of dryness) from persons who had internally acquired significant amounts of radium during their lifetimes. Most of these skeletons are from white females who ingested radium while working in radium dial painting factories during the 1920's or earlier and whose first exposure to radium generally occurred while young.⁶ The median and standard deviation of age at first exposure were 19.7 ± 8.2 for the 67 dial-painter cases in the present study. Other skeletons are from persons who drank commercially available radium "health water," or who obtained their radium for medical purposes, and a few skeletons are from chemists or physicists who worked in the radium industry. The estimated body burdens of the subjects considered in this study ranged from less than 1 nCi to 24,800 nCi ^{226}Ra .

Keane et al.⁷ provided a "summarized and preliminary presentation" of osteometric data from 18 dry, female, exhumed skeletons at MIT. A more extensive

study was done by Farnham et al.⁸ at Argonne on 40 skeletons from exhumed radium patients. The authors estimated the total skeletal weights according to sex and age groups and compared their data to the normal populations reported in the literature. They also calculated an estimation of stature for each of the 40 cases and compared individual mean lengths, male and female, in a similar manner to the normal populations. They reported no evidence of an appreciable effect on stature or skeletal weight for persons carrying a radium body burden, a finding that has been substantiated by two subsequent analyses by A. P. Polednak⁹ and Polednak and Farnham.¹⁰

The purpose of this study is twofold: 1) to continue to compare anthropometrically this ²²⁶Ra burdened skeletal collection with statistics previously established for a normal population, and 2) to create a data source for a "best estimate" of living stature and total skeletal weights of radium patients for whom data were not obtained while living. The size of the skeletal collection has more than tripled since the 1970 preliminary report, and the data become more reliable as the number of samples increases. These data are provided to aid in future calculations and extrapolations in determining radium dose distributions and total body burdens.

Materials and Methods

The CHR skeletal material procurement program has yielded 145 cases, most of which are in a condition to elicit accurate osteometric data. If we eliminate the 15 cremains cases, 6 non-radium cases, 5 incomplete autopsy cases, and 18 cases which are unmeasurable for a variety of other reasons, we find that 101 cases can be treated anthropometrically.

These specimens included in the present study are distributed according to age at death and sex as shown in Figure 1. The age groups are comparable to those chosen by Merz, et al.¹¹

The weights of the bones were taken on either a Mettler P1200N top-loading balance or a Mettler PR1200 top-loading balance (both balances reading to an accuracy of ± 0.01 g), or, in a very few cases, from previously recorded data. The maximum lengths were measured using a standard osteometric board or

FIG. 1.--Age and sex distribution of skeletons used in this study.

Age and sex distribution

<u>Age, yr</u>	<u>Females</u>	<u>Males</u>
20-29	13	1
30-39	12	2
40-49	11	4
50-59	6	8
60-69	9	6
70-79	12	9
80 and above	<u>4</u>	<u>4</u>
Total	67	34

directly off roentgenograms using a metric ruler, all to a precision of ± 0.1 cm. No correction for roentgenographic magnification was made. This will be discussed later.

Some measurements of the long bones had to be done by roentgenograms. Owing to the nature of the research at CHR, portions of these bones have been consumed in destructive tests by the various research groups within the Center and can never again be measured directly for anthropometric purposes. This is one reason that the Bone Microdosimetry Group will continue to place a high priority on the complete roentgenological survey of all skeletal materials.

Results

The raw data, consisting of the weights and lengths of the available long bones from each case, are shown in Table 1 (females) and in Table 2 (males). Included in the tables are the case number and age at death of each subject. Table 3 lists the mean values of measured weights and lengths ($\bar{X}_t$) and the standard deviations (S.D.)_t calculated for the groups of specific long bones. In addition, females are treated as a separate group, yielding values reported as $\bar{X}_F$ and (S.D.)_F and males as a group reported as $\bar{X}_M$ and (S.D.)_M. The mean and standard deviations for weights and lengths for sample groups classified according to both sex and age are given in Table 4.

No attempt has been made to standardize the condition of these bones for this study; the purpose of obtaining the skeletons was other than anthropometric studies. Most of the bones were exhumed, devoid of flesh and dry. However, some bones, especially those obtained from amputation, autopsy, or willied remains, are wet and include marrow and fatty oils and are represented by the largest total skeletal weights. Only bones that were complete and without obvious gross pathology (i.e., fractures, etc.) were used for length and weight measurements.

Discussion

Weights

In order to compare these radium burdened skeletons with those of the normal population, estimations were made of the total skeletal weight (in grams) for each case based on the weights of various long bones. These estimations are shown in Table 5, as is the average of all the individual estimates. In the same manner, the estimated total skeletal weights for various age groups of each sex calculated from the measured weight of a specific bone are shown in Table 6, along with the average age for the groups.

These weights were calculated according to the fractional weights of long bones (shown in Table 7) reported by Ingalls¹² for a United States male population, by Latimer and Lowrance¹³ for an Asian population, and by Baker¹⁴ for a normal U.S. male and female population. The skeletons used in both the Ingalls and

TABLE 1. Weight and Maximum Length Measurements on Long Bones - Females

Specimen	Age, yr	Humerus		Radius		Ulna		Femur		Tibia		Fibula	
		Weight, g	Length, cm	Weight, g	Length, cm	Weight, g	Length, cm	Weight, g	Length, cm	Weight, g	Length, cm	Weight, g	Length, cm
00-006	27	--	28.7 (B)	--	19.1 (B)	--	20.8 (B)	--	40.7 (L)	183.3 (R)	32.7 (B)	--	31.8 (R)
00-009	29	149.3 (R) ^a	34.0 (B)	40.9 (R)	23.2 (B)	50.4 (R)	25.0 (B)	421.0 (R)	48.0 (B)	267.8 (R)	38.5 (B)	58.8 (R)	36.2 (B)
00-017	30	71.4 (R)	28.1 (R)	23.2 (R)	20.3 (L)	--	21.3 (L)	217.7 (R)	41.8 (B)	141.7 (R)	32.6 (R)	--	--
00-019	51	--	--	--	--	--	--	--	--	154.3 (L)	29.4 (L)	--	--
00-023	29	--	--	29.3 (B)	21.7 (B)	34.6 (B)	22.8 (B)	304.8 (L)	41.3 (L)	140.2 (B)	33.2 (B)	--	--
00-027	40	143.3 (B)	30.3 (B)	41.6 (R)	24.0 (B)	48.6 (R)	24.8 (R)	417.5 (B)	41.8 (B)	208.9 (L)	35.8 (B)	35.4 (L)	34.5 (B)
01-001	71	133.1 (B)	32.7 (B)	38.5 (B)	23.9 (B)	47.1 (B)	25.8 (B)	455.5 (B)	46.0 (B)	287.9 (B)	38.2 (B)	56.4 (B)	37.2 (B)
01-006	39	143.1 (B)	31.0 (B)	38.9 (B)	21.5 (B)	44.9 (B)	23.7 (B)	--	42.8 (R)	--	34.4 (B)	56.4 (R)	34.5 (B)
01-007	63	--	28.9 (R)	--	20.3 (R)	29.8 (R)	22.2 (R)	--	41.1 (B)	--	34.4 (B)	30.1 (L)	33.7 (B)
01-011	65	105.3 (R)	31.2 (B)	30.0 (R)	23.5 (B)	35.7 (R)	24.7 (B)	--	45.2 (L)	--	35.0 (L)	--	--
01-012	89	58.6 (L)	29.7 (B)	--	21.7 (R)	19.4 (L)	22.5 (B)	247.1 (B)	41.2 (B)	147.5 (L)	34.8 (B)	23.1 (L)	33.4 (B)
01-014	48	--	30.1 (R)	--	22.2 (B)	--	23.8 (B)	--	44.0 (R)	--	36.2 (B)	--	33.8 (R)
01-016	75	146.1 (L)	31.4 (L)	--	--	--	--	--	--	--	--	--	--
01-017	93	151.5 (B)	30.6 (B)	38.8 (B)	23.3 (B)	44.3 (B)	24.5 (B)	676.3 (B)	41.7 (B)	259.3 (B)	34.7 (B)	41.6 (B)	33.9 (B)
01-019	33	91.7 (B)	29.1 (B)	--	--	--	--	343.5 (B)	41.9 (B)	--	--	--	--
01-022	51	102.7 (L)	27.9 (B)	27.6 (L)	20.8 (B)	32.2 (L)	22.5 (B)	258.4 (L)	37.7 (L)	155.0 (L)	32.3 (B)	29.5 (L)	33.7 (B)
01-031	28	104.0 (B)	27.5 (B)	31.1 (R)	21.3 (B)	35.3 (L)	23.2 (B)	300.6 (R)	42.7 (R)	164.7 (L)	34.3 (B)	35.9 (L)	32.8 (B)
01-032	32	--	29.8 (B)	--	21.4 (B)	--	22.6 (B)	--	42.8 (B)	--	34.6 (B)	--	32.0 (B)
01-033	23	--	30.8 (B)	--	21.9 (B)	--	23.1 (B)	281.6 (R)	40.1 (R)	--	--	--	--
01-040	22	--	29.0 (B)	--	20.5 (B)	--	22.0 (B)	~265.2 (R)	41.2 (B)	156.1 (L)	33.7 (B)	--	32.2 (B)
01-046	40	88.4 (B)	27.8 (B)	27.3 (R)	21.5 (B)	35.4 (L)	23.2 (B)	236.1 (B)	39.8 (B)	--	33.2 (R)	--	31.4 (R)
01-049	34	--	29.8 (B)	--	21.9 (B)	--	23.5 (B)	437.0 (R)	42.0 (R)	294.1 (R)	36.7 (B)	--	33.8 (B)
01-052	20	--	30.0 (B)	--	21.5 (B)	--	--	232.6 (B)	41.6 (B)	--	35.5 (B)	--	--
01-054	28	75.3 (L)	28.5 (B)	24.8 (L)	20.5 (B)	30.2 (L)	22.0 (B)	221.7 (B)	38.4 (B)	~116.1 (R)	31.2 (B)	~28.5 (R)	29.7 (R)
01-057	23	--	31.6 (B)	--	21.6 (B)	35.1 (R)	23.2 (B)	332.7 (R)	42.2 (R)	198.2 (L)	36.5 (L)	--	--

^a(R) = Right; (L) = Left; (B) = Average of R and L.

TABLE 1. (Cont.)

Specimen	Age, yr	Humerus		Radius		Ulna		Femur		Tibia		Fibula	
		Weight, g	Length, cm	Weight, g	Length, cm	Weight, g	Length, cm	Weight, g	Length, cm	Weight, g	Length, cm	Weight, g	Length, cm
01-082	33	75.2 (R)	28.4 (B)	--	21.9 (L)	27.6 (B)	24.7 (B)	249.1 (R)	42.6 (R)	--	35.7 (B)	--	--
01-099	40	--	30.6 (B)	--	22.0 (B)	26.4 (R)	23.6 (B)	--	42.3 (B)	--	33.8 (B)	--	33.1 (B)
01-103	43	--	--	41.4 (R)	23.3 (B)	40.3 (R)	24.7 (R)	109.3 (R)	44.1 (R)	258.7 (R)	36.5 (B)	50.5 (B)	35.4 (B)
01-105	47	79.3 (R)	24.7 (B)	22.1 (R)	20.6 (R)	27.1 (R)	22.5 (R)	--	39.6 (R)	132.3 (L)	33.6 (B)	23.7 (L)	31.5 (B)
01-115	36	102.3 (R)	26.9 (B)	33.7 (B)	20.2 (B)	37.6 (R)	21.6 (B)	327.9 (R)	43.2 (B)	177.3 (L)	33.6 (R)	40.6 (L)	33.2 (B)
01-132	36	141.9 (L)	33.0 (B)	45.4 (R)	23.7 (R)	27.1 (L)	25.5 (R)	474.7 (B)	43.3 (B)	--	35.3 (P)	--	36.5 (B)
01-144	76	136.1 (B)	29.4 (B)	34.4 (B)	23.2 (R)	30.7 (R)	21.7 (B)	341.7 (R)	46.0 (P)	249.5 (B)	36.5 (B)	33.6 (B)	32.7 (B)
01-145	57	131.7 (B)	31.7 (B)	33.6 (R)	22.7 (R)	41.1 (R)	24.4 (R)	151.3 (L)	43.7 (L)	--	37.2 (L)	--	35.4 (L)
01-146	85	117.7 (R)	34.7 (B)	33.6 (R)	23.3 (B)	31.7 (R)	25.7 (R)	111.5 (R)	47.5 (B)	243.7 (B)	36.0 (B)	53.0 (B)	35.6 (B)
01-149	71	--	31.1 (B)	--	23.1 (L)	--	21.7 (L)	--	45.6 (L)	--	36.2 (B)	--	35.1 (B)
01-175	66	--	--	44.7 (L)	25.4 (R)	31.0 (R)	27.7 (B)	--	PF ^a	--	--	44.9 (L)	36.8 (L)
01-183	68	--	30.8 (B)	--	20.4 (B)	--	23.1 (R)	--	46.4 (B)	--	37.2 (B)	--	35.0 (B)
01-302	67	--	30.1 (B)	--	22.6 (B)	--	23.7 (L)	--	PF	--	36.3 (L)	--	34.3 (L)
01-388	71	107.1 (L)	32.4 (L)	27.2 (L)	23.4 (R)	33.5 (L)	25.2 (B)	326.7 (L)	45.3 (L)	146.1 (L)	34.9 (B)	--	37.9 (B)
01-389	20	95.7 (R)	32.2 (B)	32.3 (R)	23.0 (B)	32.9 (R)	24.5 (R)	363.2 (L)	47.1 (B)	177.7 (R)	35.4 (R)	35.4 (B)	34.8 (R)
01-390	44	117.7 (B)	32.3 (B)	36.7 (L)	24.5 (B)	41.1 (B)	25.6 (B)	353.4 (B)	44.5 (B)	149.3 (B)	37.5 (B)	41.1 (L)	35.8 (L)
01-405	72	--	31.0 (B)	--	21.9 (B)	--	23.6 (R)	393.3 (B)	42.4 (B)	155.9 (L)	36.2 (B)	--	32.3 (L)
01-439	73	--	32.5 (L)	--	23.4 (L)	--	25.4 (L)	--	--	--	41.0 (L)	--	38.9 (L)
01-466	44	79.1 (R)	29.2 (B)	--	21.4 (B)	26.3 (L)	23.1 (R)	260.6 (L)	42.6 (B)	--	35.2 (B)	--	33.2 (B)
01-520	87	98.4 (B)	30.8 (B)	24.3 (L)	21.9 (B)	30.4 (L)	23.1 (B)	304.1 (B)	42.0 (R)	154.6 (L)	34.8 (B)	28.4 (L)	34.7 (B)
01-562	30	97.3 (L)	29.6 (B)	--	22.4 (B)	--	23.7 (R)	333.9 (L)	43.2 (B)	--	35.7 (B)	44.5 (L)	33.8 (B)
01-565	65	--	30.5 (R)	--	21.4 (L)	--	23.6 (L)	367.9 (R)	40.5 (B)	193.3 (R)	32.7 (B)	--	--
01-573	53	140.6 (L)	31.2 (B)	--	23.8 (R)	49.1 (L)	25.4 (B)	484.6 (L)	44.4 (B)	261.9 (L)	37.4 (B)	--	36.3 (B)
01-574	52	--	32.3 (B)	--	22.0 (B)	--	22.4 (R)	398.4 (R)	43.4 (B)	--	34.9 (B)	--	33.2 (L)
01-578	26	--	32.0 (R)	--	--	--	--	--	--	--	--	--	--

^a (R) = Right; (L) = Left; (B) = Average of R and L.

^b pathological fracture.

TABL 1. (Cont.)

Specimen	Age, yr.	Humerus		Radius		Ulna		Femur		Tibia		Fibula	
		Weight, g	Length, cm	Weight, g	Length, cm	Weight, g	Length, cm	Weight, g	Length, cm	Weight, g	Length, cm	Weight, g	Length, cm
01-612	77	129.6 (B)	29.6 (B)	33.4 (B)	22.0 (B)	40.1 (B)	23.5 (B)	448.3 (B)	42.9 (B)	250.7 (B)	34.3 (B)	44.8 (B)	32.7 (B)
01-613	30	101.8 (B)	29.6 (B)	30.2 (B)	21.2 (B)	37.6 (B)	22.8 (B)	347.4 (B)	40.6 (B)	224.0 (B)	33.4 (B)	38.0 (B)	32.7 (B)
01-633	48	103.2 (R)	31.1 (B)	--	21.7 (B)	37.1 (R)	23.1 (B)	320.1 (R)	46.7 (B)	162.7 (R)	37.7 (B)	35.3 (R)	35.2 (B)
01-660	76	163.0 (L)	34.2 (L)	41.9 (L)	24.5 (B)	44.1 (L)	26.0 (B)	543.0 (R)	48.9 (R)	--	--	--	--
01-739	72	76.8 (B)	32.5 (B)	24.7 (B)	22.1 (B)	25.7 (R)	23.9 (R)	225.8 (B)	43.7 (B)	112.7 (B)	35.5 (B)	--	--
03-240	39	108.3 (B)	30.5 (B)	35.3 (B)	23.1 (B)	36.9 (B)	24.7 (B)	294.9 (B)	43.7 (B)	187.0 (B)	36.6 (B)	34.3 (B)	35.5 (B)
03-666	23	110.2 (B)	32.0 (B)	28.4 (B)	21.8 (B)	35.6 (B)	23.5 (B)	363.9 (B)	44.0 (B)	230.6 (B)	35.5 (B)	33.0 (B)	34.4 (B)
03-779	36	125.7 (B)	30.7 (B)	39.7 (B)	21.9 (B)	46.7 (B)	23.9 (B)	379.9 (B)	43.0 (B)	204.0 (B)	35.3 (B)	50.5 (B)	34.7 (B)
05-116	61	--	--	24.5 (R)	22.7 (R)	--	--	250.2 (B)	44.4 (B)	152.9 (B)	37.8 (B)	--	--
05-165	65	105.5 (B)	30.2 (B)	27.4 (B)	20.6 (B)	34.4 (B)	22.3 (B)	312.8 (B)	41.3 (B)	174.7 (B)	34.2 (B)	41.6 (B)	33.0 (B)
05-210	72	99.1 (B)	29.4 (B)	30.3 (B)	21.3 (B)	34.7 (B)	22.4 (B)	PATH. (B) ^c	PATH. (B)	162.9 (B)	36.0 (B)	27.9 (B)	34.5 (B)
05-349	72	73.2 (B)	31.5 (B)	24.5 (B)	23.2 (B)	31.9 (R)	24.8 (B)	284.0 (B)	43.8 (B)	162.0 (B)	36.8 (B)	--	--
05-420	47	--	29.4 (L)	--	21.2 (B)	--	23.1 (B)	363.9 (R)	42.9 (B)	--	34.0 (R)	--	--
05-555	67	68.3 (B)	28.6 (B)	19.7 (B)	20.4 (B)	26.2 (B)	22.3 (B)	302.9 (B)	40.4 (B)	122.6 (R)	34.8 (B)	25.6 (B)	33.4 (B)
05-751	32	100.9 (R)	30.1 (B)	30.7 (R)	21.8 (B)	32.3 (R)	23.1 (B)	--	44.8 (B)	155.3 (L)	35.6 (B)	--	--
09-044	49	--	29.5 (B)	--	21.1 (B)	--	22.4 (B)	--	43.7 (B)	--	35.7 (B)	--	33.7 (B)
10-883	52	60.2 (B)	28.4 (B)	26.6 (B)	21.3 (B)	31.0 (B)	22.2 (B)	176.6 (R)	40.0 (B)	111.9 (B)	33.1 (B)	30.4 (L)	32.5 (L)

^a (R) = Right; (L) = Left; (B) = Average of R and L.

^b Pathological fracture.

^c Pathological.

TABLE 2. Weight and Maximum Length Measurements of 100+ Specimens

Specimen ^a	Age	Measure		Length		Weight		Length		Weight		Length		Weight		Length	
		Weight	Length	Weight	Length	Weight	Length	Weight	Length	Weight	Length	Weight	Length	Weight	Length	Weight	Length
00-000	45	152.0 (L)	31.1 (R)	41.0 (R)	27.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
00-020	57	132.3 (L)	30.2 (R)	35.0 (L)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
00-033	54	--	32.1 (R)	--	22.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-003	60	110.0 (L)	33.3 (L)	42.1 (L)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-010	74	81.0 (L)	28.7 (R)	64.0 (L)	22.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-139	40	109.3 (R)	31.0 (R)	40.0 (R)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-141	72	109.0 (R)	31.0 (R)	41.0 (R)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-218	71	277.2 (R)	31.1 (R)	71.0 (R)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-251	75	130.0 (L)	32.0 (R)	40.0 (L)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-305	43	--	--	--	--	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-404	70	--	33.0 (R)	41.0 (L)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-434	52	160.0 (L)	32.4 (R)	--	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-438	73	100.0 (L)	32.0 (R)	40.0 (L)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-450	53	130.0 (L)	31.0 (R)	40.0 (L)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-456	70	--	32.1 (R)	--	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-485	61	100.0 (L)	32.0 (R)	40.0 (L)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-501	71	134.0 (R)	34.4 (R)	41.0 (R)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-567	64	160.0 (R)	34.0 (R)	--	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-568	21	120.1 (L)	33.2 (R)	34.0 (L)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-635	57	--	--	41.0 (L)	25.2 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-661	60	100.0 (L)	34.0 (R)	40.0 (R)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
01-690	62	164.0 (R)	34.2 (R)	--	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
03-209	60	145.0 (R)	34.2 (R)	44.0 (R)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
03-236	71	190.0 (R)	35.7 (R)	44.0 (R)	27.3 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
05-044	60	259.7 (R)	35.0 (R)	74.7 (R)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
05-072	57	155.0 (R)	35.7 (R)	51.0 (L)	--	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
05-912	74	--	35.0 (R)	--	25.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
09-041	63	--	34.1 (R)	42.0 (R)	--	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
09-084	39	162.0 (L)	34.2 (R)	53.1 (L)	24.0 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
09-105	42	160.9 (R)	33.0 (R)	51.1 (R)	24.7 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
09-120	56	114.4 (R)	32.0 (R)	31.0 (R)	25.4 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
10-644	57	--	--	--	--	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
10-831	47	239.5 (R)	34.7 (R)	64.3 (R)	27.3 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)
10-840	57	141.0 (R)	35.0 (R)	43.4 (R)	24.2 (R)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)	1.0 (L)

^a (R) = Right; (L) = Left; (B) = Body; (P) = P; (M) = M.

TABLE 3. Mean Value of Measured Weights and Lengths, and Standard Deviation about the Mean of Long Bones

	Humeru				Radius				Ulna				Femur				Tibia				Fibula			
	Weight		Length		Weight		Length		Weight		Length		Weight		Length		Weight		Length		Weight		Length	
	N ^a	g	N	cm	N	g	N	cm	N	g	N	cm	N	g	N	cm	N	g	N	cm	N	g	N	cm
$\bar{X}_t^b$	68	130.1	93	31.5	64	39.5	94	23.0	69	45.2	92	24.6	77	401.0	92	44.3	64	226.9	93	36.5	52	45.4	78	35.2
(S.D.) _t		44.6		2.08		12.8		1.74		15.1		1.96		131.2		2.97		84.11		2.54		15.98		2.47
$\bar{X}_F$	42	108.6	62	30.4	34	32.3	63	22.1	44	37.5	61	23.6	46	347.6	60	43.0	41	166.8	61	35.4	30	36.4	49	34.1
(S.D.) _F		28.8		1.62		6.93		1.25		4.34		1.26		191.0		2.30		52.2		2.01		10.10		1.84
$\bar{X}_M$	26	165.0	31	33.4	26	50.0	31	24.7	25	47.7	31	24.1	29	449.6	32	46.8	25	300.6	32	38.6	22	54.8	29	37.1
(S.D.) _M		43.9		1.42		12.0		1.17		14.6		1.56		126.7		2.44		83.3		2.09		17.79		2.20

^a N = Number of measurements.

^b t = total; F = female; M = male.

TABLE 4. Means of Weights and Lengths of Bone According to Age Groups

Sex	Age, yr	Humerus				Radius				Ulna				Femur				Tibia				Fibula			
		Weight		Length		Weight		Length		Weight		Length		Weight		Length		Weight		Length		Weight		Length	
		N ^a	g	N	cm	N	g	N	cm	N	g	N	cm	N	g	N	cm	N	g	N	cm	N	g	N	cm
F	20-29	$\bar{X}$	6 101.0	12 30.4		7 30.0	12 21.4		7 36.3	11 22.9		11 300.5	12 42.4		1 177.7	11 34.5		5 38.3	7 33.1						
		S.D.	28.3	2.03		5.80	1.15		6.40	1.27		65.4	1.76		15.4	2.07		11.8	2.17						
	30-39	$\bar{X}$	10 108.9	12 30.0		7 36.3	11 21.9		5 39.1	11 23.1		9 352.1	11 42.1		2 77.0	1 37.7		6 44.1	9 34.1						
		S.D.	21.7	1.20		5.58	0.97		8.11	1.17		70.1	1.03		47.7	6.71		8.22	1.40						
	40-49	$\bar{X}$	6 101.8	10 29.9		5 33.8	11 22.1		5 37.4	11 23.6		7 351.6	11 42.9		1 180.4	11 35.4		5 37.2	10 33.8						
		S.D.	25.2	1.31		8.75	1.20		4.25	1.01		93.1	2.06		47.5	1.56		9.76	1.52						
	50-59	$\bar{X}$	4 108.8	5 30.3		3 29.3	5 22.1		4 36.1	5 23.1		5 346.7	5 41.4		4 175.8	6 34.1		2 29.5	5 33.6						
		S.D.	36.2	2.03		3.79	1.17		5.37	1.35		125.6	1.17		73.6	3.08		630.4	2.25						
F	60-69	$\bar{X}$	3 93.0	7 30.0		5 29.3	9 22.0		5 36.1	9 23.6		4 306.5	7 42.1		4 162.1	8 35.3		4 35.6	6 34.4						
		S.D.	21.4	0.96		9.38	1.73		10.7	1.12		44.2	2.21		32.2	1.65		9.18	1.38						
	70-79	$\bar{X}$	9 118.2	12 31.5		8 31.9	11 22.9		4 37.2	11 24.6		5 381.5	1 45.6		1 190.0	10 37.6		4 40.7	8 35.2						
		S.D.	31.0	1.44		6.38	0.99		6.95	1.02		114.2	2.10		60.0	1.90		12.6	2.57						
	≥ 80	$\bar{X}$	4 119.1	4 31.5		3 34.7	4 22.6		4 36.4	4 23.4		4 432.3	4 43.1		4 212.6	4 35.6		4 36.5	4 34.4						
		S.D.	50.6	2.27		9.07	0.87		14.9	1.17		195.8	2.95		72.2	1.62		13.5	0.96						
	≥ 80	$\bar{X}$	4 119.1	4 31.5		3 34.7	4 22.6		4 36.4	4 23.4		4 432.3	4 43.1		4 212.6	4 35.6		4 36.5	4 34.4						
		S.D.	50.6	2.27		9.07	0.87		14.9	1.17		195.8	2.95		72.2	1.62		13.5	0.96						
M	20-29	$\bar{X}$	1 120.1	1 33.2		1 38.3	1 22.7		1 44.1	1 24.4		1 403.3	1 42.1		--	--		--	--						
		S.D.	--	--		--	--		--	--		--	--		--	--		--	--						
	30-39	$\bar{X}$	2 142.3	2 32.2		2 55.0	2 23.9		1 45.4	2 25.1		2 467.0	2 47.1		2 295.1	2 37.5		1 42.3	2 35.7						
		S.D.	6162.6	634.2		554.1	524.0		--	525.3		5495.9	547.5		5343.7	638.6		--	637.0						
	40-49	$\bar{X}$	3 185.4	3 34.5		3 54.7	3 26.2		2 77.1	3 26.1		2 642.5	3 50.1		3 351.7	4 40.7		4 68.1	4 39.4						
		S.D.	46.9	0.67		8.35	1.33		500.3	1.11		5466.0	0.601		76.4	1.65		6.01	1.67						
	50-59	$\bar{X}$	5 141.8	6 32.4		5 36.8	6 24.1		6 47.5	6 24.7		6 396.7	7 46.1		5 221.4	7 37.2		5 35.7	5 35.9						
		S.D.	20.5	1.42		7.29	0.92		10.3	1.23		59.5	2.37		47.3	1.77		5.37	1.78						
M	60-69	$\bar{X}$	5 182.9	6 34.0		4 54.1	6 24.1		7 61.0	6 26.0		6 491.7	6 47.8		5 247.6	6 36.9		4 54.0	6 37.4						
		S.D.	22.0	0.45		10.5	0.40		11.7	0.76		76.9	1.11		49.0	1.54		9.70	1.25						
	70-79	$\bar{X}$	6 149.8	9 33.5		7 51.8	9 25.1		6 64.7	9 26.1		5 507.2	9 46.1		4 352.7	9 39.1		4 63.8	8 37.3						
		S.D.	64.9	1.93		14.3	1.21		17.8	2.10		184.2	2.00		126.1	2.59		25.9	3.13						
	≥ 80	$\bar{X}$	4 196.6	4 33.1		4 54.2	4 24.4		4 65.7	4 26.1		4 594.6	4 46.0		4 315.4	4 37.6		4 60.5	4 36.1						
		S.D.	43.5	1.22		15.0	0.65		14.4	0.52		105.7	1.64		83.5	0.99		18.1	1.02						

^a N = Number of measurements.

When N = 2, both measured values are shown.

Table 5. Estimation of Total Skeletal Weight Based on Weight of Long Bones in Glass

Case number	Age	FEMALES						Average of all estimates $\pm$ S.D.	Normalization factor	Estimated weight normalized to dry, fat-free
		Humerus	Radius	Ulna	Femur	Tibia	Fibula			
00-006	27	-- ^a	--	--	--	3385 3449	--	3417	1.00	3417
00-009	29	4165 4680 4212	3752 3752	3720 3789	4539 4765 4415	4946 5039	5069 4761	4374 $\pm$ 507	0.95	4155
00-017	25	1992 2238 2014	2128 2128	--	2347 2464 2283	2617 2668	--	2288 $\pm$ 236	1.00	2288
00-019	51	--	--	--	--	2849 2904	--	2876	0.70	2013
00-023	29	--	2688 2688	2554 2602	3286 3450 3197	2589 2638	--	2828 $\pm$ 341	1.00	2828
00-027	40	1994 4489 4039	3817 3817	3587 3654	4501 4726 4379	3858 3930	3052 2866	3867 $\pm$ 504	0.61	2359
01-001	71	3713 4172 3755	3532 3532	3476 3541	4911 5156 4777	5317 5417	4862 4567	4321 $\pm$ 739	0.61	2636
01-006	39	1992 4486 4037	3569 3569	3314 3376	--	--	4862 4567	3917 $\pm$ 561	0.61	2389
01-007	63	--	--	2199 2241	--	--	2595 2437	2368 $\pm$ 183	0.98	2321
01-011	65	2937 3301 2970	2752 2752	2635 2694	--	--	--	2836 $\pm$ 225	0.61	1730
01-012	89	1635 1837 1653	--	1432 1459	2664 2797 2592	2724 2775	2991 1870	2144 $\pm$ 532	1.00	2144

^a Estimations are calculated according to data reported by Ingalls,¹² first number per bone; Latimer,¹³ second number; and Baker,¹⁴ third number.

Table 5. (Cont.)

Case number	Age	Humerus a	Radius	Ulna	Femur	Tibia	Fibula	Average of all elements, %	Normalization factor	Estimated weight normalized to dry, fat-free
01-014	48	4075	4580	4121					1.00	--
01-016	75	4075						4254	0.61	2598
01-017	93	4226	3560	3269	3792	4789	3586	4581	0.50	2291
		4749	3560	3131	3655	4879	3168			
		4374		1364	3093		4117			
01-019	33	2558			3704			3203	1.00	3203
		2875			3888					
		2587			1603					
01-022	51	2865	2532	2376	2786	2862	2543		0.61	1641
		3219	2532	2421	2925	2916	2389	2660		
		2897		2421	2710		2647			
01-031	28	2901	2853	2605	3241	4042	3095	2791	1.00	2793
		3260	2853	2654	3402	3099	2997			
		2934		2688	3151		4778			
01-032	32	--	--	--	--	--	--	--	1.00	--
01-033	23	--	--	--	3046			3059	1.00	3059
		--	--	--	3147			3059		
		--	--	--	2913					
01-040	22	--	--	--	2874	2983		2852	0.96	2776
		--	--	--	3002	2917				
		--	--	--	2781					
01-046	40	2466	2505	2613	2510			2568	1.00	2568
		2771	2505	2662	2672					
		2494		2538	2476					
01-049	34	--	--	--	4712	5411		5041	1.00	5041
		--	--	--	4916	5531				
		--	--	--	4583					

Estimates are calculated according to data reported by Ingalls.¹² first number per bone, tatter, second number and Baker, third number

Table 5. (Cont.)

Case number	Age	Humerus	Radius	Ulna	Femur	Tibia	Fibula	Average of all estimates $\pm$ S.D.	Normalization factor	Estimated weight normalized to dry, fat-free
01-520	87	2745 ^a 3085 2776	2229 2229 2215	2244 2286	3279 3442 3189	2855 2404	2448 2300 2626	2679 $\pm$ 419	0.61	1634
01-562	30	2714 3050 2745	-- -- --	-- -- --	3594 3778 3501	-- --	3836 3603	3353 $\pm$ 452	1.00	3353
01-565	65	-- -- --	-- -- --	-- -- --	3967 4164 3859	3662 3731	-- --	3876 $\pm$ 194	0.70	2713
01-573	53	3922 4408 3966	-- -- --	3624 3692	5225 5485 5082	5206 5304	-- --	4591 $\pm$ 741	0.61	2801
01-574	52	-- -- --	-- -- --	-- -- --	4295 4509 4178	-- --	-- --	4327 $\pm$ 168	0.95	4111
01-578	26	-- -- --	-- -- --	-- -- --	-- -- --	-- --	-- --	-- --	1.00	--
01-612	77	3615 4063 3656	3101 3101	2959 3015	4833 5074 4702	4630 4717	3862 3628 4240	3887 $\pm$ 740	0.61	2371
01-613	30	2840 3191 2872	2771 2771	2775 2827	3746 3932 3643	4137 4214	3276 3077 3759	3286 $\pm$ 534	0.64	2103
01-633	48	2879 3235 2911	-- -- --	2738 2789	3451 3623 3357	3005 3061	3043 2858 2841	3061 $\pm$ 276	0.80	2469
01-660	76	4547 5110 4598	3844 3844	3255 3316	5854 6146 5695	-- --	-- --	4517 $\pm$ 1058	0.73	3297
01-739	72	2142 2408 2166	2266 2266	1897 1932	2435 2556 2368	2081 2120	-- --	2206 $\pm$ 199	1.00	2206

^a Estimates are calculated according to data reported by Ingalls,¹² first number per bone, Latimer,¹³ second number, and Baker,¹⁴ third number

Table 5. (Cont.)

Case number	Age	Humerus	Radius	Ulna	Femur	Tibia	Fibula	Average of all estimates $\pm$ S.D.	Normalization factor	Estimated weight normalized to dry, fat-free
03-240	39	3021 ^a 3395 3055	3239 3239	2723 2774	3180 3336 3093	3453 3518	2957 2777 3175	3116 $\pm$ 245	0.98	3054
03-666	23	3074 3455 3109	2606 2606	2627 2677	3923 4119 3816	4262 4342	2845 2672 3785	3082 $\pm$ 663	0.98	3216
03-779	36	3506 3940 3546	3642 3642	3446 3511	4096 4300 3984	3767 3838	4353 4089 3651	3801 $\pm$ 295	1.00	3801
05-116	61	-- -- --	2248 2248	-- --	2698 2832 2624	2824 2877	-- -- --	2622 $\pm$ 269	1.00	2622
05-165	65	2943 3307 2976	2550 2550	2568 2617	3373 3540 3281	3226 3287	3586 3368 3103	3051 $\pm$ 378	0.64	1953
05-210	72	2764 3107 2795	2780 2780	2561 2609	-- -- --	3008 3065	2405 2259 2737	2731 $\pm$ 245	0.64	1748
05-349	72	2042 2295 2065	2248 2248	2354 2398	3062 3214 2979	2992 3048	-- -- --	2556 $\pm$ 429	1.00	2556
05-420	47	-- -- --	-- --	-- --	3923 4119 3816	-- --	-- -- --	3953 $\pm$ 154	1.00	3953
05-555	67	1905 2141 1927	1807 1807	1934 1970	3266 3428 3177	2264 2307	2207 2073 2126	2262 $\pm$ 535	1.00	2262
05-751	32	2815 3163 2846	2817 2817	2384 2429	-- -- --	2868 2922	-- -- --	2761 $\pm$ 238	1.00	2761
09-044	49	-- -- --	-- --	-- --	-- -- --	-- --	-- -- --	--	0.40	--

^a Estimates are calculated according to data reported by Ingalls,¹² first number per bone, Latimer,¹³ second number, and Baker,¹⁴ third number.

Table 5. (Cont.)

Case number	Age	Humerus	Radius	Ulna	Femur	Tibia	Fibula	Average of all estimates $\pm$ S.D.	Normalization factor	Estimated weight normalized to dry, fat-free
10-883	52	1679 ^a 1887 1698	2440 2440	2268 2331	1904 1999 1852	2066 2105	2621 2462	2134 $\pm$ 289	1.00	2134
MALES										
00-008	48	4346 4884 4395	4550 4550	4450 4534	5024 5274 4887	4922 5014	5957 5595	4853 $\pm$ 454	1.00	4853
00-020	37	3969 4461 4014	5046 5046	3387 3451	5035 5286 4898	5450 5552	3647 3425	4475 $\pm$ 761	1.00	4475
00-033	54	-- -- --	-- -- --	-- -- --	-- -- --	-- -- --	2991 2810	2901	1.00	2901
01-003	68	6050 6799 6118	5697 5697	5373 5474	6201 6509 6031	5645 5752	5216 4899	5761 $\pm$ 500	0.64	3687
01-010	74	2538 2853 2567	3119 3119	3041 3098	3375 3543 3283	-- --	-- --	3053 $\pm$ 308	1.00	3053
01-139	83	4722 5307 4776	4294 4294	4450 4534	5418 5688 5270	5110 5206	4302 4040	4777 $\pm$ 494	0.64	3057
01-141	92	5325 5984 5385	5046 5046	4745 4835	6518 6842 6340	6259 6376	5940 5579	5681 $\pm$ 675	0.50	2841
01-208	71	7174 8063 7255	6468 6468	6465 6586	8604 9032 8369	9075 9246	7638 7174	7646 $\pm$ 1034	0.61	4664
01-251	75	3782 4251 3825	3606 3606	3830 3902	4152 4359 4039	-- --	2371 2227	3665 $\pm$ 650	0.61	2236

^aEstimates are calculated according to data reported by Ingalls,¹² first number per bone, Latimer,¹³ second number, and Baier,¹⁴ third number.

Table 5. (Cont.)

Case number	Age	Humerus	Radius	Ulna	Femur	Tibia	Fibula	Average of all estimates $\pm$ S.D.	Normalization factor	Estimated weight normalized to dry, fat-free
01-305	43	-- ^a -- --	-- -- --	-- -- --	-- -- --	7645 7789	6353 5968	6950 $\pm$ 792	0.61	4240
							6997			
01-404	70	-- -- --	5991 5991	5594 5699	6454 6775 6278	6443 6564	-- --	6150 $\pm$ 410	0.61	3751
				5713			--			
01-434	52	4650 5226 4702	-- --	4937 5030	4836 5076 4704	5315 5415	-- --	4989 $\pm$ 270	0.98	4889
							--			
01-438	73	2471 2777 2499	4193 4193	4066 4143	3935 4131 3828	3452 3516	-- --	3637 $\pm$ 650	1.00	3637
				4081			--			
01-450	59	3643 4094 3684	4275 4275	3476 3541	4215 4424 4100	-- --	3862 3628	3924 $\pm$ 321	1.00	3924
				3794			--			
01-456	70	-- -- --	-- --	-- --	7320 7684 7120	-- --	-- --	7375 $\pm$ 286	0.61	4499
				--			--			
01-485	81	4644 5219 4697	3679 3679	3860 3932	5857 6148 5697	4192 4271	3586 3368	4402 $\pm$ 885	0.70	3081
				3741			3854			
01-501	70	3721 4182 3763	4064 4064	-- --	3474 3647 3379	-- --	-- --	3787 $\pm$ 792	1.00	3787
				--			--			
01-567	64	4519 5078 4570	-- --	-- --	5503 5777 5353	6198 6314	-- --	5414 $\pm$ 676	0.96	5197
				--			--			
01-568	21	3350 3765 3388	3514 3514	3306 3368	4348 4565 4230	-- --	-- --	3701 $\pm$ 461	1.00	3701
				3364			--			
01-635	57	-- -- --	3844 3844	3114 3173	-- -- --	-- --	-- --	3476 $\pm$ 353	1.00	3476
				3405			--			

^a Estimates calculated according to data reported by Ingalls,¹² first number per bone, Latimer,¹³ second number, and Baker,¹⁴ third number.

Table 5. (Cont.)

Case number	Age	Humerus	Radius	Ulna	Femur	Tibia	Fibula	Average of all estimates $\pm$ S.D.	Normalization factor	Estimated weight normalized to dry, fat-free
01-661	60	5169 ^a	4459	4598	5149	4578	4138	4746 $\pm$ 507	0.98	4653
		5809	4459	4684	5406	4664	3887			
		5227			5009		4245			
01-690	62	4597	--	3351	4836	4140	3759	4252 $\pm$ 632	0.95	4039
		5166	--	3414	5076	4218	3530			
		4649	--		4704		3842			
03-209	66	5169	5826	5247	6116	5994	5500	5670 $\pm$ 390	0.61	3459
		5809	5826	5346	6421	6107	5166			
		5227			5950		5572			
03-238	71	5378	5798	5631	6438	7082	5733	6050 $\pm$ 590	0.61	3691
		6044	5798	5737	6758	7215	5385			
		5439			6262		6456			
05-044	80	7244	6853	6347	8024	7765	7026	7281 $\pm$ 661	0.50	3641
		8141	6853	6466	8423	7912	6599			
		7326			7805		7202			
05-072	57	4349	2798	2768	4875	4554	3060	3843 $\pm$ 947	0.95	3651
		4887	2798	2820	5118	4640	2874			
		4398			4743		4047			
05-912	74	--	--	--	--	--	6250	6060	0.98	5939
		--	--	--	--	--	5870			
		--	--	--	--	--	--			
09-041	63	--	3853	3926	4002	--	--	3948 $\pm$ 120	1.00	3948
		--	3853	4000	4201	--	--			
		--			3893		--			
09-084	39	4536	4963	--	5347	6347	--	5312 $\pm$ 661	0.64	3400
		5097	4963	--	5613	6467	--			
		4581			5201		--			
09-105	42	4488	4596	--	--	--	5138	4747 $\pm$ 259	0.66	3133
		5044	4596	--	--	--	4826			
		4539			--	--	--			
09-120	56	3202	2899	2915	3543	3605	2776	3202 $\pm$ 361	1.00	3202
		3599	2899	2970	3719	3673	2607			
		3238			3446		3263			

^a Estimates calculated according to data reported by Ingalls,¹² first number per bone, Latimer,¹³ second number, and Baker,¹⁴ third number.

Table 5. (Cont.)

Case number	Age	Humerus	Radius	Ulna	Femur	Tibia	Fibula	Average of all estimates $\pm$ S.D.	Normalization factor	Estimated weight normalized to dry, fat-free
10-644	57	-- ^a	--	--	4225	3919	--	4136 $\pm$ 204	1.00	4136
		--	--	--	4436	3992	--			
		--	--	--	4110		--			
10-831	47	6681	5899	5690	6927	6920	6034	6434 $\pm$ 619	0.61	3925
		7508	5899	5797	7272	7050	5668			
		6756			6738		6382			
10-840	57	3933	3982	3801	3319	3053	2698	3504 $\pm$ 552	1.00	3504
		4420	3982	3872	3484	3110	2534			
		3977		3842	3228		2821			

^aEstimates calculated according to data reported by Ingalls,¹² first number per bone, Latimer,¹³ second number, and Baker,¹⁴ third number.

Table 6. Total Estimated Skeletal Weights According to Age Group.

Age, yr.	N ^a	Average age of group, yr	Mean value of estimated skeletal weights of group, g	Estimated weight normalized to dry, fat-free, g
<u>Females</u>				
20-29	12	24.8	3022	2980
30-39	11	34.4	3459	3099
40-49	9	43.7	3230	2570
50-59	6	52.7	3069	2488
60-69	7	64.6	2901	2288
70-79	10	73.4	3404	2349
≥ 80	4	88.5	3553	2230
<u>Males</u>				
20-29	1	21	3701	3701
30-39	2	38.0	4632	3938
40-49	4	45.0	5629	4038
50-59	8	56.1	3788	3710
60-69	6	63.8	5022	4164
70-79	9	72.0	5205	3917
≥ 80	4	84.0	5536	3155

^aN = number of cases in group.

Latimer studies were macerated, cleaned, and dried. Latimer's collections were degreased, whereas the bones used by Ingalls were not. The skeletons used for the Baker studies were macerated by natural processes and were fat-free. Table 7 includes fractional weights of various bone groupings as determined at CHR. Our use of these data is discussed later.

The bones in the CHR skeletal collection are in various states of dryness. The research requirements for which the skeletal material is obtained prohibits treatment of the bones in any manner in order to standardize the dryness. Therefore, our data on skeletal weights are normalized to dry, fat-free skeletal weights.

Each skeleton, after being weighed in its existing condition, is coded in terms of four criteria: (1) wet or dry, (2) with or without marrow, (3) with or without cartilage, and (4) fatty or fat-free. Using the data reported for reference man¹⁵ a normalization factor is determined for each individual skeleton. Table 5 lists the measured weights and the normalized weights, as well as the calculated total skeletal weights in terms of a normalized, dry, fat-free skeleton.

The estimated total normalized skeletal weights for each group vs. the average age for that group are plotted in Figure 2 (females) and Figure 3 (males). The solid lines indicated on these two figures represent the total skeletal weight of a normal population as described by Merz, Trotter, and Peterson.¹¹ The line represents our computer least squares fit through the scattering points of their data. The dashed line represents a linear regression line which best fits our grouped and normalized data points. Thus, these two figures compare the total skeletal weights of the normal population and the normalized weights for our collection. Weight loss with age occurs at the same rate in our population and in the population studied by Merz et al. Thus, our population appears to be normal in that regard, despite the presence of radium.

Lengths

The lengths of the long bones in our group were compared with those of the normal population. Various studies were found in the literature which list the average lengths of long bones in specific skeletal collections. Brief descriptions of these collections and the average bone lengths reported are given in

FIG. 2.--Mean values of estimated skeletal weights (normalized to dry, fat-free) plotted against the average age for each female age group: —, linear regression fit to data in Ref. 11; ---, linear regression fit to CHR data points (o).

FIG. 3.--Mean values of estimated skeletal weights (normalized to dry, fat-free) plotted against the average age for each male age group: —, linear regression fit to data in Ref. 11; ---, linear regression fit to CHR data points (o).

Table 8, as are the mean lengths of the samples used for this study.

Estimates of stature in centimeters for each subject in the CHR collection, based upon the length of the long bones, are reported in Table 9. These estimates were calculated from formulas given by Trotter and Gleser¹⁶ for American white females and males. The general form of the equation is:

$$\text{Bone length (cm)} \times K + A = \text{estimated stature} \pm \text{S.E.},$$

where K, A, and S.E. (Standard Error) are reported, specific constants for each long bone. These values are given in Table 10. When estimating the stature of individuals over 30 years of age, $0.06 \times (\text{age} - 30)$ cm has been subtracted.

According to Trotter and Gleser,¹⁶ these equations are applicable to maximum lengths of long bones which are dry and without articular surface cartilage cover. It is appropriate to use either a single left or right bone or to use the average of a pair of bones in the calculations. In addition, it has been noted that estimations of stature utilizing bones of the lower limb result in a smaller standard error than estimations from lengths of the bones of the upper

Table 7. Percentage of Total Skeletal Weight

Axial skeleton	CHR percent skeletal weight	Appendicular skeleton	CHR percent skeletal weight	Percent skeletal weight		
				Ingalls ¹² (U.S.)	Latimer ¹³ (Asian)	Baker ¹⁴ (U.S.)
Skull	13.13	Clavicles	0.99			
Mandible	1.52	Scapulae	2.87			
Vertebrae, cervical	1.44	Humeri	6.89	7.17	6.38	7.09
Vertebrae, thoracic	4.24	Radius	2.16	2.18	2.18	4.94
Vertebra, lumbar	3.64	Ulnae	2.60	2.71	2.66	
Sacrum & coccyx	2.12	Innomates	9.83			
Sternum & xiphoid	0.65	Patellae	0.80			
Hyoid	0.02	Femora	19.11	18.55	17.67	19.07
Ribs	6.41	Tibiae	11.48	10.83	10.63	13.94
		Fibulae	2.43	2.37	2.47	
		Wrist & hands	2.66			
		Ankles & feet	6.52			

Table 8. Average Bone Lengths Reported in Various Studies in Centimeters.

Long bone	Todd ¹ (U.S.) F&M	Telkka ² (Finland) F&M	This study (U.S.) F&M	Trotter ⁵ (U.S.) M	Trotter & Gleser ¹⁶ (U.S.) M	This study (U.S.) M	This study (U.S.) F	Trotter & Gleser ¹⁶ (U.S.) F	Mean of reported values
Humerus	32.9	32.9	31.5	33.6	33.0	33.4	30.4	30.4	32.3
Radius	24.4	--	23.0	25.2	24.4	24.7	22.1	22.2	23.7
Ulna	--	--	24.6	27.0	26.2	26.4	23.6	24.0	25.3
Femur	45.3	45.5	44.3	47.3	45.7	46.8	43.0	43.0	45.1
Tibia	36.8	36.2	36.5	37.8	36.4	38.6	35.4	34.0	36.5
Fibula	--	36.1	35.2	38.1	36.9	37.1	34.1	34.3	36.0

Table 9. Estimation of Stature from Length of Long Bones in Centimeters.

Case no.	Age, yr	Humerus	Radius	Ulna	Femur	Tibia	Fibula	Femur and tibia	Lower extremity, ^a mean $\pm$ S.D.	Weighted mean $\pm$ S.E., humerus through fibula
<u>FEMALES</u>										
00-006	27	154.40	145.46	146.58	154.63	156.36	152.78	155.23	154.59 $\pm$ 1.79	152.11 $\pm$ 1.61
00-009	29	172.21	164.90	164.51	172.66	173.18	165.68	173.44	170.51 $\pm$ 4.19	169.02 $\pm$ 1.61
00-017	25	152.39	151.15	148.71	157.35	156.07	-	156.62	156.71 -	153.58 $\pm$ 1.80
00-019	51	-	-	-	-	145.53	-	-	145.53 -	145.53 $\pm$ 3.66
00-023	29	-	157.79	155.12	156.11	157.81	-	156.76	156.96 -	156.76 $\pm$ 1.97
00-027	40	159.18	168.09	163.06	156.75	164.75	160.10	156.16	160.53 $\pm$ 4.02	161.82 $\pm$ 1.61
01-001	71	165.38	165.76	165.47	165.26	169.85	166.15	158.60	167.09 $\pm$ 2.43	166.45 $\pm$ 1.61
01-006	39	161.59	155.30	158.42	159.28	160.75	160.16	159.97	160.06 $\pm$ 0.74	159.50 $\pm$ 1.61
01-007	63	153.09	149.17	150.57	153.64	153.31	156.37	156.58	156.44 $\pm$ 2.84	154.15 $\pm$ 1.61
01-011	65	160.70	164.22	161.13	163.64	160.93	-	162.58	162.29 -	162.16 $\pm$ 1.80
01-012	89	154.22	154.25	150.30	152.32	158.91	153.93	155.30	155.05 $\pm$ 3.44	154.17 $\pm$ 1.61
01-014	48	158.03	159.08	158.31	161.70	165.43	157.56	163.60	161.56 $\pm$ 3.94	160.25 $\pm$ 1.61
01-016	75	160.77	-	-	-	-	-	-	- -	160.77 $\pm$ 4.45
01-017	93	157.01	161.59	158.60	153.32	158.38	155.16	155.62	155.62 $\pm$ 2.56	157.09 $\pm$ 1.61
01-019	33	155.57	-	-	157.41	-	-	-	157.41 -	156.65 $\pm$ 2.85
01-022	51	150.45	152.26	143.30	145.96	153.94	148.30	149.24	149.40 $\pm$ 4.10	149.10 $\pm$ 1.61
01-031	28	150.37	155.89	150.06	159.57	161.00	155.71	160.23	158.76 $\pm$ 2.74	155.99 $\pm$ 1.61
01-032	32	157.98	156.25	147.38	159.70	161.75	153.25	160.67	158.23 $\pm$ 4.44	156.33 $\pm$ 1.61
01-033	23	161.46	158.74	149.64	153.15	-	-	-	153.15 -	155.48 $\pm$ 2.07
01-040	22	155.41	152.10	144.94	155.86	159.26	153.96	157.19	156.36 $\pm$ 2.69	154.00 $\pm$ 1.61
01-046	40	150.78	156.24	149.46	151.81	157.21	151.01	154.07	153.34 $\pm$ 3.37	152.87 $\pm$ 1.61
01-049	34	157.86	158.50	151.11	157.60	167.72	158.40	162.35	161.24 $\pm$ 5.63	158.98 $\pm$ 1.61
01-052	20	158.77	156.84	-	156.85	164.48	-	160.37	160.67 -	159.48 $\pm$ 1.99
01-054	28	153.73	152.10	144.94	148.95	152.01	146.63	149.94	149.20 $\pm$ 2.70	149.59 $\pm$ 1.61
01-057	23	164.15	157.31	150.06	158.33	167.38	-	162.59	162.86 -	157.82 $\pm$ 1.80
01-082	33	153.21	158.56	153.30	159.14	164.88	-	161.36	162.01 -	158.43 $\pm$ 1.80
01-099	40	160.19	158.61	151.17	157.98	158.95	155.99	158.38	157.64 $\pm$ 1.51	157.19 $\pm$ 1.61
01-103	43	-	164.59	155.69	162.25	166.60	162.55	164.45	163.80 $\pm$ 2.43	162.62 $\pm$ 1.73
01-105	47	153.38	151.55	146.06	150.89	157.95	150.89	153.93	153.24 $\pm$ 4.08	152.00 $\pm$ 1.61
01-115	36	154.71	150.32	142.87	160.44	158.61	156.53	159.59	158.53 $\pm$ 1.96	154.62 $\pm$ 1.61

^aMean of estimates for femur, tibia, and fibula.

Table 9 (cont.)

Case no.	Age, yr	Humerus	Radius	Ulna	Femur	Tibia	Fibula	Femur and tibia	Lower extremity, ^a mean $\pm$ S.D.	Weighted mean $\pm$ S.E., humerus through fibula
FEMALES (cont.)										
01-132	36	168.49	167.86	159.53	160.69	172.24	166.20	166.26	166.38 $\pm$ 5.78	165.94 $\pm$ 1.61
01-144	76	155.34	162.14	152.86	164.96	164.62	152.66	165.12	160.75 $\pm$ 7.01	159.03 $\pm$ 1.61
01-145	57	163.20	160.43	153.57	160.42	167.79	161.71	164.03	163.31 $\pm$ 3.94	161.51 $\pm$ 1.61
01-146	85	171.60	162.07	154.45	168.13	168.43	160.62	168.75	165.72 $\pm$ 4.43	164.32 $\pm$ 1.61
01-149	71	160.01	161.96	154.01	164.27	165.79	159.99	165.28	163.35 $\pm$ 3.01	161.36 $\pm$ 1.61
01-175	66	-	173.17	164.13	PF ^b	-	165.27	-	165.27	167.28 $\pm$ 2.31
01-183	68	159.18	151.24	147.36	166.43	167.13	159.88	167.12	164.48 $\pm$ 4.00	159.42 $\pm$ 1.61
01-302	67	156.89	159.83	150.41	PF	164.58	157.89	-	161.24	158.38 $\pm$ 1.79
01-388	71	164.37	165.28	156.14	163.53	171.88	168.20	167.78	167.87 $\pm$ 4.18	165.42 $\pm$ 1.61
01-389	20	166.16	163.95	155.62	170.44	164.19	161.57	167.88	165.40 $\pm$ 4.56	163.85 $\pm$ 1.61
01-390	44	165.66	170.22	159.47	163.18	169.44	163.66	166.34	165.43 $\pm$ 3.48	165.31 $\pm$ 1.61
01-405	72	159.61	156.22	149.25	156.31	163.99	151.73	159.93	157.34 $\pm$ 6.19	156.29 $\pm$ 1.61
01-439	73	164.59	163.27	156.88	-	177.85	171.01	-	174.43	167.79 $\pm$ 1.79
01-466	44	155.24	155.53	148.80	158.48	162.77	156.05	160.50	159.10 $\pm$ 3.40	156.61 $\pm$ 1.61
01-520	87	158.04	155.32	146.22	154.42	159.93	157.86	156.53	157.10 $\pm$ 2.40	155.47 $\pm$ 1.61
01-562	30	157.43	161.11	152.63	160.80	165.06	158.64	162.87	161.50 $\pm$ 3.27	159.64 $\pm$ 1.61
01-565	65	158.35	154.27	149.67	152.04	154.26	-	152.85	153.15	153.60 $\pm$ 1.80
01-573	53	161.42	166.36	158.08	162.39	168.61	164.59	165.52	165.20 $\pm$ 3.15	163.88 $\pm$ 1.61
01-574	52	165.18	157.89	147.04	159.98	161.42	155.57	160.72	158.99 $\pm$ 3.05	157.93 $\pm$ 1.61
01-578	26	165.49	-	-	-	-	-	-	-	165.49 $\pm$ 4.45
01-612	77	154.61	156.39	148.53	157.24	159.19	152.60	157.69	156.01 $\pm$ 2.99	154.79 $\pm$ 1.61
01-613	30	157.43	155.42	148.36	154.38	158.39	154.42	156.36	156.06 $\pm$ 2.08	155.07 $\pm$ 1.61
01-633	48	162.39	156.71	148.56	148.37	169.78	161.67	169.44	166.61 $\pm$ 4.33	162.04 $\pm$ 1.61
01-660	76	170.12	168.30	159.26	172.12	-	-	-	172.12	167.78 $\pm$ 2.07
01-739	72	164.65	157.16	150.53	159.52	161.96	-	160.77	160.74	158.95 $\pm$ 1.80
03-240	39	159.91	163.88	155.93	161.50	167.11	163.79	164.28	163.91 $\pm$ 2.90	162.27 $\pm$ 1.61
03-666	23	165.49	158.26	151.35	162.78	164.48	160.40	163.71	162.55 $\pm$ 2.05	160.72 $\pm$ 1.61
03-779	36	160.76	158.38	152.69	159.95	163.54	160.92	161.66	161.47 $\pm$ 1.86	159.70 $\pm$ 1.61
05-116	61	-	160.67	-	161.91	169.29	-	165.60	165.60	164.29 $\pm$ 2.22
05-165	65	157.34	150.47	144.12	154.01	158.61	154.20	156.05	155.61 $\pm$ 2.60	153.48 $\pm$ 1.61

^aMean estimates for femur, tibia, and fibula.^bPathological fracture.

Table 9 (cont.)

Case no.	Age, yr	Humerus	Radius	Ulna	Femur ^a	Tibia	Fibula	Pathology ^b	Lower limb length ^c	Adjusted mean ± S.E. Humerus (Ulna)
FEMALES										
05-210	72	154.23	153.37	144.13	PATH ^b	153.41	154.18		153.41	155.51 ± 1.79
05-349	72	161.29	162.38	154.38	159.77	163.75		162	162.75	160.99 ± 1.80
05-420	47	155.73	154.40	148.62	159.04	157.11		159.11	157.11	155.84 ± 1.80
05-555	67	151.85	149.41	144.09	151.67	151.23	155.2		151.23	152.68 ± 1.61
05-751	32	158.99	158.14	149.52	161.64	164.0		164.44	164.05	159.87 ± 1.80
09-044	49	155.95	153.80	145.51	160.90	153.92	151.21	161.43	153.92 ± 1.46	154.90 ± 1.61
10-883	52	152.07	154.57	144.47	151.58	157.2	153.52	153.41	157.2 ± 1.51	152.37 ± 1.61
MALES										
00-008	48	177.48	178.10	177.68	179.57	177.88	180.31	177.1	177.88	179.69 ± 1.50
00-020	37	169.21	168.93	166.50	172.85	171.7	177.04	171.71	170.81 ± 1.41	169.86 ± 1.50
00-033	54	167.88	162.24	153.19	169.21	168.15	167.07	168.4	168.81 ± 1.28	165.47 ± 1.50
01-003	68	173.81	169.72	167.97	174.73	172.14	172.22	172.11	171.69 ± 2.11	173.76 ± 1.50
01-010	74	159.29	165.96	153.18	156.59	164.11	161.22	164.58	157.66 ± 1.05	156.28 ± 1.50
01-139	83	174.45	170.33	169.29	173.33	173.22	168.03	171.75	171.53 ± 2.00	170.87 ± 1.50
01-141	92	164.98	165.63	165.05	167.46	166.03	162.40	167.5	164.0 ± 2.75	164.11 ± 1.50
01-208	71	169.94	171.43	171.86	168.05	177.46	174.11	171.04	172.54 ± 1.80	171.90 ± 1.50
01-251	75	166.31	165.14	163.85	164.38	171.08	167.70	167.1	167.91 ± 3.65	166.82 ± 1.50
01-305	43	-	-	-	-	174.1	171.23		171.67	172.63 ± 2.35
01-404	70	171.54	169.22	180.43	166.82	171.22	164.94	167.2	167.99 ± 1.93	170.11 ± 1.50
01-434	52	168.92	168.41	166.56	169.81	168.52	167.06	167.49	167.80 ± 2.46	168.12 ± 1.50
01-438	73	169.20	171.69	171.00	166.88	171.19	168.63	168.35	168.50 ± 1.71	169.33 ± 1.50
01-450	59	166.65	168.37	167.40	164.34	171.1	167.59	167.37	167.71 ± 3.37	167.56 ± 1.50
01-456	70	179.24	176.78	174.88	173.49	177.62	174.17	173.65	174.06 ± 2.16	175.80 ± 1.50
01-485	81	168.41	165.54	166.08	169.02	169.81	163.06	169.3	167.30 ± 3.69	167.07 ± 1.50
01-501	70	174.00	171.49	171.55	171.82	180.30		176.2	176.96	174.24 ± 1.69
01-567	64	172.82	170.34	170.43	174.56	176.62	169.70	175.76	173.62 ± 3.55	172.70 ± 1.50
01-568	21	172.71	164.82	164.33	161.61				161.61	165.41 ± 1.96
01-635	57	-	172.65	171.22	-					171.94 ± 3.05

^aMeans of estimates for femur, tibia, and fibula.^bPathology present.

Table 9 (cont.)

Case no.	Age, yr	Humerus	Radius	Ulna	Femur	Tibia	Fibula	Femur and tibia	Lower extremity, ^a mean $\pm$ S.D.	Weighted mean $\pm$ S.E., humerus through fibula
MALES (cont.)										
01-661	60	174.91	169.44	169.56	166.47	170.31	165.66	168.09	167.48 $\pm$ 2.48	168.96 $\pm$ 1.50
01-690	62	170.79	168.94	170.55	178.01	179.52	172.77	179.15	176.77 $\pm$ 3.54	174.22 $\pm$ 1.50
03-209	66	173.63	176.64	175.86	172.30	174.49	171.73	173.45	172.84 $\pm$ 1.40	173.76 $\pm$ 1.50
03-238	71	177.95	179.74	179.26	178.66	186.54	181.61	183.16	182.27 $\pm$ 3.96	180.95 $\pm$ 1.50
05-044	80	168.78	169.75	169.10	170.03	170.62	167.40	170.27	169.35 $\pm$ 1.71	169.28 $\pm$ 1.50
05-072	57	163.39	-	-	163.08	165.45	-	163.72	164.27 -	164.02 $\pm$ 2.03
05-912	74	172.22	172.38	170.57	172.77	173.25	168.03	173.19	171.35 $\pm$ 2.89	171.49 $\pm$ 1.50
09-041	63	173.50	169.26	169.01	170.81	170.13	166.55	170.36	169.16 $\pm$ 2.29	169.75 $\pm$ 1.50
09-084	39	175.25	169.19	167.49	174.63	175.86	170.40	175.33	173.63 $\pm$ 2.86	172.55 $\pm$ 1.50
09-105	42	173.83	171.66	174.34	178.50	182.23	174.51	180.74	178.41 $\pm$ 3.86	176.43 $\pm$ 1.50
09-120	56	168.99	173.46	171.65	177.90	177.36	174.29	177.95	176.49 $\pm$ 2.00	174.49 $\pm$ 1.50
10-644	57	-	-	-	176.17	176.04	-	176.33	176.11 -	176.10 $\pm$ 2.35
10-831	47	176.31	181.18	179.96	181.06	181.42	179.57	181.74	180.68 $\pm$ 0.98	180.05 $\pm$ 1.50
10-840	57	176.63	168.87	167.15	173.79	168.22	162.62	171.00	168.21 $\pm$ 5.59	169.25 $\pm$ 1.50

^aMeans of estimates for femur, tibia, and fibula.

Table 10. Constants for Estimation of Stature by the Formula of Trotter and Gleser¹⁶

Bone	K	A	S.E.
Female			
Humerus	3.36	57.97	4.46
Radius	4.74	54.94	4.24
Ulna	4.27	52.76	4.30
Femur	2.47	54.10	3.72
Tibia	2.90	51.53	3.66
Fibula	2.93	59.61	3.57
Femur + tibia	1.39	53.20	3.55
Male			
Humerus	3.68	70.45	4.05
Radius	4.78	70.01	4.32
Ulna	3.70	74.65	4.32
Femur	2.38	67.47	3.27
Tibia	2.52	78.62	3.47
Fibula	2.68	71.78	3.29
Femur + tibia	1.30	63.20	2.99

limb, because the latter would not necessarily have a direct relationship on the height of the person.

Table 9 also includes the weighted mean stature (M) and the standard error of the weighted mean (SE_M) for N long bones according to the following formulas:

$$M = \frac{\sum \left(\frac{1}{SE_n} \right)^2 M_n}{\sum \left(\frac{1}{SE_n} \right)^2} \quad \pm \quad SE_M = \frac{1}{\sqrt{\sum \left(\frac{1}{SE_n} \right)^2}},$$

where M_n and SE_n are the calculated stature and standard error values for each long bone.

The Use of X-rays for Measurement

Trotter and Peterson¹⁷ determined a correction factor which is needed because of the distortion due to the projection of the bone onto the film. In order to evaluate our average error due to projection for various long bones, more than 25 of the long bones were measured on the osteometric board and on the roentgenogram.

The percent error introduced into the calculations owing to the projection of a long bone onto the film was calculated. The results were as expected, i.e.,

the bones which lie flattest against the film have the smallest error due to projection. In decreasing order of size, we found the increases in length on the films of the six long bones to be: femur (2.87%), tibia (2.32%), humerus (1.94%), ulna (1.49%), radius (1.41%), and fibula (1.24%). These values are smaller than those reported by Trotter and Peterson: femur (3.2%), tibia (3.3%), humerus (2.2%), and radius (1.3%). The difference probably lies in the physical factors of the roentgenographic equipment. As these values are insignificant in a study of this scope, they have not been entered into our calculations. In this particular study, the error introduced by distortion is further diminished since only about 25 percent of the measurements were taken from the films. However, under certain conditions, one must be aware that these errors do exist.

Summary and Conclusions

A summary of our "best estimates" of living stature and raw data and normalized total skeletal weights for all 101 cases is shown in Table 11. In order to correlate these estimates more easily with other data pertaining to radium exposure, the following categories have also been included in the table: age at death, type of exposure, how many weeks exposed, how many years from the time exposed to death, and the ^{226}Ra body burden.

Also presented in Table 11 is the actual weight measured at Argonne, the estimated percent skeleton present (by weight) and the estimated total skeletal weight. Table 7 included the data that have been used at CHR for many years to calculate the total skeletal weight for incomplete skeletons. Missing parts represent a certain percentage of the total skeletal weight. Therefore, the total weight of the available skeleton is corrected to represent the complete skeletal weight. Included in Table 11 is a total skeletal weight estimate derived using CHR normalized bone weights. Compare this estimate with the mean estimate given in Table 5 and shown again in Table 11. The second from the last column in Table 11 gives a normalized estimate value for these latter data. It is noted that the CHR estimate of total skeletal weight is higher in most cases than the estimates based on the long bones. Nevertheless, we believe that the CHR estimate is the preferred one since it takes under consideration the whole of what is available to us rather than the available long bones alone.

Table 11. Data summary - females and males

[illegible]^aExposure type: see Appendix A this report^b Calculated for complete skeleton.^cFrom Table 9.

Table 11 (cont.)

Case number	Age	Exposure type ^a	Weeks of exposure	Time, first exposure to death, yr	Body burden at death, nCi ^b	Actual weight, present ^c	Percent skeleton present	Estimated skeleton weight (CHB)	Estimated skeleton weight (Table 5)	Estimated skeleton weight (Table 5)	Estimated skeleton weight (Table 5)	Estimated skeleton weight (Table 5)	Estimated skeleton weight (Table 5)	Estimated skeleton weight (Table 5)
FEMALES (cont.)														
01-132	36	1	76	21	1327	2704	56.3	1800	1800	1800	1800	1800	1800	1800
01-144	76	4	26	51	694	6143	100.0	6143	6143	6143	6143	6143	6143	6143
01-145	57	1	60	39	6331	3848	43.8	4100	4100	4100	4100	4100	4100	4100
01-146	85	2	156	40	100	4179	96.1	4179	4179	4179	4179	4179	4179	4179
01-149	71	1	26	40	1630	-	-	-	-	-	-	-	-	-
01-175	66	2	13	39	1710	1312	70.4	1312	1312	1312	1312	1312	1312	1312
01-183	68	1	78	54	203	-	-	-	-	-	-	-	-	-
01-302	67	5	10	39	2850	1555	42.5	1555	1555	1555	1555	1555	1555	1555
01-388	71	2	+0	16	2580	1948	57.2	1948	1948	1948	1948	1948	1948	1948
01-389	20	1	26	7	1029	9.8	30.0	9.8	9.8	9.8	9.8	9.8	9.8	9.8
01-390	44	2	260	6	7400	3646	96.3	3646	3646	3646	3646	3646	3646	3646
01-405	72	6 - 7	1716	45	52	1904	54.0	1904	1904	1904	1904	1904	1904	1904
01-439	73	4	8	31	406	1485	22.6	1485	1485	1485	1485	1485	1485	1485
01-466	44	1	52	26	0	1529	51.6	1529	1529	1529	1529	1529	1529	1529
01-520	87	2	+0	39	670	3500	98.0	3500	3500	3500	3500	3500	3500	3500
01-562	30	1	52	11	10300	1784	45.3	1784	1784	1784	1784	1784	1784	1784
01-565	65	5	26	32	16000	1347	35.0	1347	1347	1347	1347	1347	1347	1347
01-573	53	1	312	29	670	2021	39.0	2021	2021	2021	2021	2021	2021	2021
01-574	52	5	77	13	2730	1786	49.9	1786	1786	1786	1786	1786	1786	1786
01-578	26	5	17	4	2000	1558	12.6	1558	1558	1558	1558	1558	1558	1558
01-612	77	1 - 7	255	13	18	4295	98.5	4295	4295	4295	4295	4295	4295	4295
01-613	30	1 - 7	265	13	656	3619	99.9	3619	3619	3619	3619	3619	3619	3619
01-633	48	5	4	44	2600	1557	38.6	1557	1557	1557	1557	1557	1557	1557
01-660	76	4	+0	25	15	1004	20.3	1004	1004	1004	1004	1004	1004	1004
01-739	72	5	7	2	11500	2283	83.0	2283	2283	2283	2283	2283	2283	2283
03-240	39	5	+0	25	4320	3129	96.3	3129	3129	3129	3129	3129	3129	3129
03-666	23	1	347	6	24812	3501	95.6	3501	3501	3501	3501	3501	3501	3501
03-779	36	1	+0	20	1835	4277	100.0	4277	4277	4277	4277	4277	4277	4277
05-116	61	1	52	42	19	2601	95.4	2601	2601	2601	2601	2601	2601	2601
05-165	65	1	13	45	1	3535	100.0	3535	3535	3535	3535	3535	3535	3535

^a Exposure type: see Appendix A this report.

^b Calculated from complete skeleton.

^c From Table 9.

Table 11 (cont.)

Case number	Age	Exposure type ^a	Weeks of exposure	Time, first exposure to death, yr	Body burden at death, nCi ^b	Actual weight present, g	Percent skeleton present	Normalization factor ^c	Total estimated skeletal weights, g				Estimation of stature, ^c cm ± S.E.	
									Un-normalized (Table 4)	Normalized to dry, fat-free (Table 5)	Normalized to dry, fat-free (Table 5)	Normalized to dry, fat-free (Table 5)		
FEMALES (cont.)														
05-210	72	1	158	55	1060	3521	100.0	64	6521	2273	2751 ± 248	1748	155.51 ± 1.79	
05-349	72	1	156	17	7.3	2982	99.0	1.0	3012	3012	4281 ± 429	2556	160.99 ± 1.80	
05-420	47	1	194	18	50	1656	100.0	1.0	1661	1661	413 ± 154	3953	155.84 ± 1.80	
05-555	67	7	27	48	1	2651	100.0	1.0	2678	2678	1262 ± 535	2262	152.68 ± 1.61	
05-751	32	1	+0	13	0	1692	100.0	1.0	1639	1639	176 ± 138	1761	159.87 ± 1.80	
09-044	49	1	13	38	17	8570	100.0	1.4	857	347	-	-	156.90 ± 1.61	
10-883	52	2	+0	5	27	2794	100.0	1.0	307	307	114 ± 289	2134	152.37 ± 1.61	
MALES														
00-008	48	6	598	23	3045	4736	100.0	1.0	4736	4736	4651 ± 454	4653	179.69 ± 1.50	
00-020	37	6	676	13	920	2667	100.0	1.0	4674	4674	4475 ± 761	4475	169.86 ± 1.50	
00-033	54	6	156	3	6	1747	100.0	1.0	4150	4150	2901 ± 503	2901	165.47 ± 1.50	
01-003	68	5	304	31	12800	3292	100.0	64	6040	6040	4701 ± 503	3637	173.76 ± 1.50	
01-010	74	4	+0	30	5200	1488	100.0	3	3534	3534	3053 ± 308	3053	158.28 ± 1.50	
01-139	83	2	130	36	1070	1365	100.0	64	4611	2434	4777 ± 494	3057	170.87 ± 1.50	
01-141	92	2	130	50	17	6451	100.0	1.0	6453	3127	5681 ± 675	2841	164.11 ± 1.50	
01-208	71	6	1144	33	918	7700	100.0	1.0	7700	4637	7646 ± 1034	4564	171.90 ± 1.50	
01-251	75	6	156	53	11	1587	100.0	1.0	1611	244	3665 ± 650	2236	166.82 ± 1.50	
01-305	43	6	1040	22	160	488	100.0	64	1585	1585	6960 ± 792	4240	172.63 ± 2.35	
01-404	70	6 - 7	1716	33	2800	1397	100.0	1.0	6411	1517	6150 ± 410	3751	170.11 ± 1.50	
01-434	52	2	156	5	6126	2327	100.0	1.0	4438	4438	4489 ± 270	4869	168.12 ± 1.50	
01-438	73	2	208	15	1850	2606	100.0	1.0	1749	1749	3637 ± 650	3637	169.33 ± 1.50	
01-450	59	6	364	24	0	1583	100.0	1.0	4178	4178	3924 ± 321	3924	167.56 ± 1.50	
01-456	70	2	26	20	74	1656	100.0	1.0	41	3147	7175 ± 286	4499	175.80 ± 1.50	
01-485	81	5	1300	40	340	2433	100.0	1.0	1593	1593	4412 ± 885	3051	167.07 ± 1.50	
01-501	70	2	156	11	3500	1048	100.0	1.0	1071	1071	3787 ± 192	3787	174.24 ± 1.69	
01-567	64	2	+0	24	1100	1338	100.0	1.0	184	184	5414 ± 676	5197	172.70 ± 1.50	
01-568	21	5	+0	1	4900	2774	100.0	1.0	3963	3963	1711 ± 461	3701	165.41 ± 1.96	
01-635	57	6	312	19	1900	2454	100.0	1.0	4562	4562	476 ± 353	3476	171.94 ± 3.05	

^a Exposure type: see Appendix A this report.^b Calculated from complete skeleton.^c From Table 9.

Table 11 (cont.)

Case number	Age	Exposure type ^a	Weeks of exposure	Time, first exposure to death, yr	Body burden at death, nCi L	Actual weight present, g	Percent skeleton present	Estimated skeleton weight, g	Normalized (CHR)	Total estimated skeleton weights, g	Normalized to dry, fat-free (Table 5)	Estimation of stature, ^c cm $\pm$ S.E.
MALES												
01-661	60	6	572	20	2	5225	98.2	5198	5131	4719 $\pm$ 507	4653	168.96 $\pm$ 1.50
01-690	62	4	+0	32	21	1699	36.0	4747	466	4252 $\pm$ 632	4039	174.22 $\pm$ 1.50
03-209	66	5	572	35	1105	5401	91	4921	440	4672 $\pm$ 190	4359	173.76 $\pm$ 1.50
03-238	71	5	+0	28	1390	6377	94	6141	544	4551 $\pm$ 590	3691	180.95 $\pm$ 1.50
05-044	80	6	468	60	2	8151	99.5	8146	8176	7281 $\pm$ 661	3641	169.28 $\pm$ 1.50
05-072	57	7	13	31	0	3721	43.3	85	4184	3643 $\pm$ 947	3651	164.02 $\pm$ 2.03
05-912	74	7	26	33	0	1253	14.9	184	5332	4971	5939	171.49 $\pm$ 1.50
09-041	63	6	260	38	114	2025	56.7	116	3616	3648 $\pm$ 120	3948	169.75 $\pm$ 1.50
09-084	39	6	676	15	382	2286	33.3	74	6927	44	3480	172.55 $\pm$ 1.50
09-105	42	6	728	16	1390	2032	36.3	606	5644	4747 $\pm$ 219	3133	176.43 $\pm$ 1.50
09-120	56	6	104	27	1	2949	43.3	110	3552	3202 $\pm$ 361	3202	174.49 $\pm$ 1.50
10-644	57	5	0	0	5300	2796	77.3	2791	3631	4136 $\pm$ 204	4136	176.10 $\pm$ 2.35
10-831	47	5	+0	1	786	7741	100.3	7741	7791	6434 $\pm$ 619	3925	180.05 $\pm$ 1.50
10-840	57	5	0	1	390	7131	91.1	7131	4477	3514 $\pm$ 551	3504	169.25 $\pm$ 1.50

^a Exposure type: see Appendix A this report.^b Calculated for complete skeleton.^c From Table 9.

All of the results of this study have been presented in the form of tables and figures. These all illustrate data having no significant difference from the normal. From the data on these 101 skeletons, there does not seem to be an effect upon skeletal weight or living stature in radium burdened persons. Furthermore, an up-to-date data source has been established to aid in future determinations of radium dose distributions and radium body burdens in humans with varying amounts of radium or other internal emitters.

References

1. T. W. Todd and A. Lindala, Dimensions of the body, white and American Negroes of both sexes, *Am. J. Anthropometry* 12, 35-119 (1928).
2. A. Telkkä, On the prediction of human stature from long bones, *Acta Anat.* 9, 103-117 (1950).
3. R. J. Terry, The American Negro, *Science* 69, 337-341 (1929).
4. Mildred Trotter and G. C. Gleser, The effect of aging on stature, *Am. J. Phys. Anthropol.* 9, 311-324 (1951).
5. Mildred Trotter and G. C. Gleser, Estimation of stature from long bones of American whites and Negroes, *Am. J. Phys. Anthropol.* 10, 463-514 (1952).
6. R. E. Rowland, A. F. Stehney, and H. F. Lucas, Jr., Dose response relationships for female radium dial workers, *Radiat. Res.* 76, 368-383 (1978).
7. A. T. Keane, M. R. Lewis, and D. L. Gunner, Osteometric data, Massachusetts Institute of Technology Annual Progress Report of Radioactivity Center, MIT-952-5, Part II, pp. 517-520 (May 1968).
8. J. E. Farnham, K. Martens Rybicki, and M. L. Johnson, Anthropometry on a portion of the skeletal collection in the center for human radiobiology, Radiological and Environmental Research Division Annual Report, July 1970-June 1971, ANL-7860, Part II, pp. 138-151.
9. Anthony P. Polednak, Adult height and weight of female radium dial painters with analysis of radiation effect on growth, *Hum. Biol.* 49(No. 1), 1-10 (1977).
10. Anthony P. Polednak and J. E. Farnham, Osteometry and autoradiography of the long bones of skeletons of women exposed to internal radiation at 13-19 years of age, *Am. J. Phys. Anthropol.* 52, 335-340 (1980).
11. Ann L. Merz, Mildred Trotter, and Roy R. Peterson, Estimation of skeletal weights in the living, *Am. J. Phys. Anthropol.* 14, 589-609 (1956).
12. N. W. Ingalls, Observations on bone weights, *Am. J. Anat.* 48, 45-98 (1931).
13. Homer B. Latimer and E. W. Lowrance, Relative weights and lengths of the bones for 105 human skeletons from Asia, *Am. J. Anat.* 101, 445-449 (1957).
14. P. T. Baker and R. W. Newman, The use of bone weight for human identification, *Am. J. Phys. Anthropol.* 15, 601-618 (1957).

15. International Commission on Radiological Protection, Report of the Task Group on Reference Man, Pergamon Press, New York (1975).
16. Mildred Trotter and G. C. Gleser, A re-evaluation of estimates of stature based on measurements of stature taken during life and of long bones after death, *Am. J. Phys. Anthropol.* 16, 79-123 (1958).
17. Mildred Trotter and Roy R. Peterson, Weight of the skeleton during post-natal development, *Am. J. Phys. Anthropol.* 33, 313-324 (1970).

APPENDIX A. Exposure Data for Radium Patients

Table 1 summarizes exposure data collected as of 31 December 1979 for 2223 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 analytic 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 1979 annual report¹ listed 2164 cases. The radium burdens of 59 persons, including 5 deceased, were measured for the first time in 1979. The 59 additional cases are identified by a star following the year of measurement. There were follow-up examinations and burden measurements in 1979 on 92 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 corresponds 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. If several measurements over a period of years had been made for a given case, the result (and date) 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}Pa 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 body content. A value of 5.7 y 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

TABLE A1. Type of Exposure to ^{225}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 $^{228}\text{Ra}/^{226}\text{Ra}$ Ratios in TABLE 1.

Code number	Standard error ^a
1	$\leq 10\%$
2	11-20%
3	21-50%
4	1.5 ($\times, \div$)
5	2 ($\times, \div$)
6	$> 50\%$
7	3 ($\times, \div$)
8	Probably an upper limit ^b
9	Initial ratio of ^{228}Ra to ^{226}Ra probably ≤ 0.20 ^b
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 ($\times, \div$) 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.

^b Ref. 2

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. Error 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 function³ 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 skeleton⁴ — either up to the date of death or, for the living subjects, through 1979. 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.

References

1. Exposure data for radium patients, Radiological and Environmental Research Division Annual Report, July 1978-June 1979, ANL-79-65, Part II, pp. 147-212.
2. A. T. Keane, A survey of MsTh/Ra ratios, Annual Progress Report, Radioactivity Center, Massachusetts Institute of Technology Report, MIT-952-3, pp. 13-22 (1966).
3. W. P. Norris, T. W. Speckman, and P. F. Gustafson, Studies of the metabolism of radium in man, *Am. J. Roentgen.* 73, 785-802 (1955).
4. W. R. Neal, Dose variables and their calculation. Annual Progress Report, MIT-952-3, pp. 94-141 (May 1966).

TABLE 1
FACDSUFF DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BOEN	DIED	TYPE	EXF	YR	YR	RA226	RA226	RA226	RA226	RA226	RA226	RA226	RA226
00-001	F	1893	1924	00	1913	783	1967	13000	F4	0.00700	F3	996	310	2313	1369
00-002	F	1896	1922	01	1917	223	1966	7000	F4	0.01100	F3	972	3570	4074	40367
00-003	F	1894	1927	01	1917	104	1964	9000	F4	0.01200	F1	1367	264	8050	3481
00-004	F	1900	1931	01	1917	88	1963	1400	F4	0.00600	F7	258	331	1913	4731
00-005	F	1901	1930	01	1917	300	1963								
00-006	F	1903	1930	01	1918	128	1969	2610	A1	0.00536	A1	357	808	1859	9901
00-007	F	1903	1930	01	1919	104	1963	1000	F4	0.01000	F7	163	302	1038	4124
00-008	F	1900	1930	01	1915	534	1972	3045	A1	0.00288	A3	525	682	2601	6775
00-009	F	1900	1929	01	1918	266	1969	2650	A1	0.00490	A2	295	504	1224	5064
00-010	F	1900	1929	01	1917	156	1970	17000	A1	0.00069	F7A	1626	580	5650	4765
00-011	F	1905	1946	01	1917	260	1976	2400	F2	0.00140	F4	525	693	4790	10252
00-012	F	1900	1925	01	1912	676	1969	920	A1	0.00228	A6	67	49	174	286
00-013	F	1900	1925	01	1917	377	1960	10000	F4	0.01000	F1	752	807	2223	5201
00-014	F	1900	1929	01	1917	65	1978	7214	A1	0.00007	F2A	1016	116	5475	1453
00-015	F	1902	1940	01	1918	130	1970	2500	A1	0.00023	F3	505	55	4187	808
00-016	F	1902	1940	01	1917	279	1969	10000	F4	0.00036	F1	1522	214	5016	2816
00-017	F	1900	1922	01	1917	409	1969	17	G6	0.0	Z9	5	0	77	0
00-018	F	1900	1922	01	1917	156	1970	6	A6	0.00300	Z7A	0	0	0	0
00-019	F	1902	1943	06	1917	232	1979*	1	A6	0.00060	Z7	0	0	1	2
00-020	F	1902	1943	06	1917	232	1979*	15400	A1	0.0	Z9A	3403	0	31456	0
00-021	F	1906	1936	01	1922	676	1935	18000	P2	0.02150	F1	2599	236	16586	3220
00-022	F	1906	1936	01	1925	304	1967	12800	A1	0.00037	A3	2882	120	19507	1273
00-023	F	1906	1936	01	1918	40	1941	10500	E4	0.0	Z9	2134	0	23320	0
00-024	F	1906	1936	01	1927	12	1939	5000	E4	0.50000	E4	721	1530	2850	13918
00-025	F	1906	1936	01	1919	260	1970	3500	A1	0.00144	A3	612	314	4144	4361
00-026	F	1906	1936	01	1926	40	1967	3620	A1	0.0	Z9A	736	0	6142	0
00-027	F	1906	1936	01	1917	78	1960	6000	P2	0.00067	F3	1632	186	19519	2790
00-028	F	1906	1936	01	1918	52	1960	6500	F4	0.00050	F2	1422	110	12991	1634
00-029	F	1906	1936	01	1926	40	1967	5200	A1	0.0	Z9A	1214	0	8574	0
00-030	F	1906	1936	01	1919	156	1975	6000	A1	0.0	Z9A	1025	0	6942	0
00-031	F	1906	1936	01	1922	40	1970	5800	A1	0.0	Z9A	1445	0	15491	0
00-032	F	1906	1936	01	1916	156	1968	2240	A1	0.00036	F3	536	89	5471	1328
00-033	F	1906	1936	01	1917	780	1935	200	E4	0.0	Z9	30	0	281	0
00-034	F	1906	1936	01	1921	208	1973	1940	A1	0.00245	F2	546	578	6817	8678
00-035	F	1906	1936	01	1926	155	1977	1120	A1	0.00156	P2	336	214	4534	3221

TABLE 1 (CONT.) EXPECTED DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SPX	SUBJ	DESC	EXP	TYPE	YEAR	EXP	YFAR	PAZ26	RAZ26	RAZ26	INPUT	INPUT	CUD	CUD
									RAZ26	MEHOD	TO	RAZ26	RAZ26	RAZ26	RAZ26
						FIRST	WKS	MPAS	NCI	RAZ26	RAZ26	RAZ26	RAZ26	RAZ26	RAZ26
01-018	M	1899	1958	06	1911	2340	1950	1250	B2	0.0	0.02958	238	185	1110	1879
01-019	F	1933	1936	01	1922	253	1965	240	A1	0.0	0.02958	A2	35	193	1879
01-020	F	1935	1956	05	1923	5	1950	1500	B4	0.0	0.0	79	331	3479	0
01-021	F	1897	1973	01	1916	104	1965	1250	B4	0.0	0.0	79	373	5531	0
01-022	F	1930	1957	01	1917	110	1968	600	A2	0.0	0.0	294	147	1544	0
01-024	F	1931	1956	01	1916	308	1943	1140	B2	0.02190	0.0	79	94	2682	1493
01-025	F	1896	1952	05	1924	40	1954	1200	E2	0.01100	0.0	79	7	2539	105
01-026	F	1935	1959	01	1925	156	1950	700	B2	0.03000	0.0	79	87	1531	1295
01-027	F	1889	1957	06	1912	1040	1960	500	A2	0.0	0.0	298	0	973	0
01-028	F	1879	1965	16	1912	260	1953	250	B4	0.0	0.0	29	0	658	0
01-029	M	1876	1958	06	1902	40	1950	300	B4	0.0	0.0	79	0	948	0
01-030	F	1882	1952	07	1936	0	1950	20	P4	0.0	0.0	29	0	15	0
01-031	F	1906	1974	01	1925	4	1975	910	A1	0.01130	0.0	113	557	528	6296
01-032	F	1908	1940	01	1924	201	1969	1450	A1	0.02800	0.0	236	1228	1536	16742
01-033	F	1908	1931	01	1923	42	1963	2472	A1	0.05153	0.0	282	1793	1192	18509
01-034	F	1913		01	1929	19	1965	8	G6	0.01000	0.0	2	2	28	24
01-035	F	1901	1972	01	1920	19	1971	0	B6	0.01860	0.0	0	0	0	0
01-037	F	1908		01	1928	26	1974	0	B6	0.00327	0.0	0	0	0	0
01-038	F	1910		01	1927	111	1959	8	B2	0.02000	0.0	2	2	26	24
01-039	F	1915		07	1934	1092	1972	1	B6	0.0	0.0	0	0	2	0
01-040	F	1907	1920	01	1923	60	1963	4300	A1	0.05209	0.0	412	2585	1422	21160
01-041	F	1909		01	1927	22	1971	0	B6	0.04470	0.0	0	0	0	0
01-043	F	1912		01	1927	3	1958	9	B6	0.02200	0.0	2	2	30	30
01-044	F	1904		01	1924	22	1959	4	B3	0.08000	0.0	1	6	15	83
01-045	F	1899		01	1922	237	1955	0	E6	0.08000	0.0	0	0	0	0
01-046	F	1903	1943	01	1920	657	1963	551	A1	0.05607	0.0	104	731	793	10502
01-047	F	1896		01	1920	367	1962	80	B4	0.05700	0.0	22	136	318	2048
01-048	F	1900	1970	01	1920	206	1957	140	B2	0.09290	0.0	35	230	532	2048
01-049	F	1933	1937	01	1920	1	1960	1000	A1	0.07300	0.0	174	1641	1198	22993
01-050	F	1911		01	1925	10	1976	1	B6	0.00258	0.0	0	0	4	6
01-051	F	1904	1977	01	1923	162	1957	150	B2	0.13330	0.0	36	251	519	3781
01-052	F	1910	1930	01	1924	144	1965	2000	A1	0.03500	0.0	183	824	602	6301
01-054	F	1909	1937	01	1924	202	1965	2100	A1	0.03714	0.0	304	1457	1692	18610
01-055	F	1907		01	1925	85	1976	4	B3	0.01024	0.0	1	6	18	87
01-056	F	1904	1978	01	1920	364	1965	134	B1	0.03432	0.0	37	206	546	3093

TABLE 1 (CONT'D.) SUCCESSFUL DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SPX	DOB	DIED	TYPE	YR	AGE	WGT	OP	RA226	RA226	METHOD	INUT	INPUT	CUM	CUM
01-057	F	1908	1931	01	1924	81	1363		RA226	RA226	RA226	RA226	RA226	RA226	RA226
01-059	F	1905	1967	01	1928	29	1364	140	B1	C.04777	B2	49	307	628	4678
01-060	F	1909		07	1928	20	1974	0	E4	C.06330	72B	0	0	0	0
01-063	F	1911	1970	01	1927	21	1976	34	R1	C.00154	29B	10	5	138	69
01-066	F	1904		01	1925	2	1675	1	B6	C.02292	28B	0	0	0	0
01-069	F	1905		07	1924	17	1970	0	B6	C.01024	22F	0	0	0	0
01-070	F	1910		01	1927	63	1973	1	B6	C.00370	23B	0	0	4	4
01-071	F	1928	1967	01	1927	6	1958	0	B6	C.02300	23B	0	0	0	0
01-072	F	1939		01	1921	130	1954	100	B4	C.03000	05	24	114	360	1709
01-073	F	1900	1960	01	1921	122	1966	37	B1	C.03563	92	25	191	327	2722
01-074	F	1909		01	1927	47	1973	4	B3	C.00172	28B	1	1	17	17
01-075	F	1902		01	1922	52	1979	4	B6	C.00713	29B	1	9	19	134
01-078	F	1909		01	1925	50	1979	3	B6	C.00193	28B	1	1	14	16
01-079	F	1931	1943	01	1920	176	1966	750	B4	C.09070	P1	146	1387	1164	20106
01-080	F	1932		01	1921	204	1963	106	B1	C.02075	51	31	150	454	2255
01-081	F	1907		01	1923	11	1959	7	B6	C.05120	223	2	11	27	170
01-082	F	1902	1935	01	1919	230	1963	1030	A1	C.03786	A1	150	956	968	12727
01-084	F	1934		01	1923	712	1974	46	E2	C.01297	22B	14	74	203	1110
01-085	F	1913		01	1927	47	1968	6	R6	C.02207	23F	1	1	20	19
01-086	F	1907	1966	01	1925	4	1950	0	B6	C.08000	22B	0	0	0	0
01-087	F	1905	1970	01	1921	344	1964	750	F4	C.03690	F1	213	1061	3140	15955
01-090	F	1910		01	1927	90	1977	5	B3	C.00218	23B	2	1	21	19
01-091	F	1907		01	1927	264	1979	0	R6	C.02179	28B	0	0	0	0
01-092	F	1906	1976	01	1922	24	1971	2	B6	C.01860	22B	1	4	9	63
01-093	F	1904		01	1926	8	1971	0	B6	C.00460	28B	0	0	0	0
01-094	F	1888	1966	01	1921	128	1964	11	G4	C.04400	22	3	21	39	322
01-095	F	1907	1977	01	1922	54	1975	6	P2	C.01163	72B	2	13	27	198
01-096	F	1909		01	1927	310	1960	27	D2	C.01809	28	6	4	86	64
01-097	F	1905		01	1921	110	1963	122	B1	C.03852	B2	33	187	502	2809
01-099	F	1905	1945	01	1924	18	1963	164	A1	C.05365	A2	32	191	248	2760
01-100	F	1905	1967	01	1924	36	1957	34	P2	C.03200	D5	8	58	103	872
01-101	F	1905		01	1924	4	1959	0	B6	C.08000	22B	0	0	0	0
01-103	F	1903	1946	17	1922	172	1978	374	A1	C.03800	22A	75	440	613	6412
01-105	F	1898	1945	01	1921	21	1963	460	A1	C.05217	A1	95	801	812	11743
01-106	F	1902	1977	01	1924	155	1954	10	B2	C.08000	22B	2	12	35	187

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

[illegible]

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	POB	DIED	EXP YR	YR FST	EXP DUR	OF	FA226 NCI	FA226 METHO + BR	FA226 TC FA226 RATIO	FA226 METHO + BR	INPUT UCI	INPUT UCI	LOM RADS	LOM RADS
01-151	F	1935	1975	06	1935	53	1974	1	B6	0.0	79	0	0	3	0
01-152	F	1934	1974	01	1934	17	1977	2	C6	0.03150	258	1	1	10	16
01-153	M	1890	1964	06	1920	104	1963	280	B1	0.00036	86	78	5	694	50
01-154	M	1846	1969	06	1923	40	1959	0	B6	0.01500	27	1	0	0	0
01-156	F	1900	1950	01	1918	150	1950	40	G6	0.0	20	11	0	127	0
01-157	F	1894	1972	02	1925	13	1975	40	B2	0.00139	258	15	9	216	134
01-158	F	1901	1977	06	1920	52	1959	1	G6	0.0	29	0	0	4	0
01-159	F	1915	1975	01	1935	220	1972	2	B6	0.0	298	1	0	6	0
01-160	F	1873	1965	02	1925	40	1959	130	B1	0.02000	83	32	40	386	607
01-161	F	1896	1973	01	1918	17	1959	1	B6	0.0	298	0	0	4	0
01-162	M	1898	1968	06	1920	364	1959	95	B1	0.01000	778	24	17	214	187
01-163	F	1903	1972	01	1920	26	1972	2	B6	0.00360	278	1	1	9	18
01-164	F	1900	1972	01	1918	39	1959	9	B2	0.0	298	2	0	35	0
01-165	F	1904	1972	01	1922	22	1972	14	C3	0.0	298	4	0	55	0
01-166	F	1897	1969	01	1916	26	1959	0	B6	0.0	298	0	0	0	0
01-168	F	1895	1995	06	1919	468	1968	1	B6	0.0	298	0	0	1	0
01-169	F	1918	1968	01	1936	69	1975	0	B6	0.0	298	0	0	0	0
01-170	M	1891	1965	05	1940	0	1959	4	G6	0.0	29	1	0	5	0
01-171	M	1895	1972	45	1914	6	1958	1500	B1	0.0	298	427	0	4788	0
01-172	F	1898	1969	01	1916	136	1961	1960	B1	0.00112	83	555	126	7736	1892
01-173	F	1891	1959	06	1917	1300	1959	70	B4	0.0	79	16	0	110	0
01-175	F	1900	1968	02	1927	13	1965	1710	B1	0.00760	82	451	343	5269	5139
01-176	F	1893	1960	01	1917	104	1969	0	B6	0.0	29	0	0	0	0
01-177	M	1915	1972	06	1936	312	1969	61	B1	0.0	298	14	0	121	0
01-178	M	1919	1970	07	1958	0	1973	2	B6	0.0	298	0	0	2	0
01-179	F	1890	1965	45	1924	58	1959	2000	B1	0.0	298	502	0	6115	0
01-180	F	1900	1967	01	1918	26	1971	3	B3	0.0	298	1	0	15	0
01-181	M	1913	1967	06	1940	130	1959	220	B1	0.0	298	39	0	225	0
01-182	M	1902	1959	02	1936	40	1959	7	D3	0.02600	250	1	1	8	6
01-183	F	1901	1960	01	1915	73	1969	203	A1	0.0	298	64	0	917	0
01-184	M	1897	1969	05	1922	10	1968	48	B2	0.0	298	14	0	132	0
01-185	M	1881	1962	06	1912	40	1959	40	B6	0.0	29	12	0	116	0
01-186	M	1925	1967	06	1943	416	1976	19	B2	0.0	298	4	0	32	0
01-187	M	1917	1970	06	1943	78	1959	42	B2	0.0	298	7	0	54	0
01-188	F	1886	1979	04	1933	3	1959	4	B6	0.0	29	1	0	11	0

TABLE 1 (CONT.) EVROUSEE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP	TYPE	Y2R	FY1	FY2	FA226 METHOD + ERR	FA228 METHOD + ERR	FA226 METHOD + ERR	INPUT RA226 UCI	INPUT RA228 UCI	CUM RA226 RADS	CUM RA228 RADS
01-139	M	1921		07	07	1958	0	1973	06	0.0	79C	0	0	0	0
01-140	F	1927		07	07	1954	0	1973	06	0.0	79C	0	0	0	0
01-141	F	1897	1965	76	76	1913	78	1959	4	0.0	79B	1	0	12	0
01-192	F	1902	1952	01	01	1925	52	1959	34	0.0	79B	8	0	94	0
01-193	F	1906	1960	06	06	1917	156	1974	31	0.0	79	0	0	105	0
01-194	M	1899		01	01	1916	676	1972	7	0.0	79B	2	0	23	0
01-195	F	1893	1958	06	06	1912	520	1959	1	0.0	79	0	0	3	0
01-196	M	1907		22	22	1930	20	1972	69	0.0540	75B	19	17	185	179
01-197	F	1893	1965	01	01	1916	40	1959	16	0.0	79	4	0	61	0
01-198	F	1888	1972	45	45	1943	40	1959	0	0.0	76	0	0	0	0
01-200	F	1910		01	01	1925	225	1977	3	0.09914	72B	1	4	13	67
01-201	F	1911		01	01	1925	55	1959	26	0.02100	78B	6	8	93	119
01-203	F	1909		01	01	1923	1	1973	0	0.01470	72B	0	0	0	0
01-204	F	1901		01	01	1917	22	1959	5	0.0	79B	1	0	22	0
01-205	M	1921	1974	06	06	1951	52	1972	7	0.0	79C	1	0	8	0
01-206	M	1896		06	06	1918	17	1959	9	0.0	79B	3	0	33	0
01-207	F	1909	1967	01	01	1927	9	1959	4	0.02000	78B	1	1	11	14
01-208	M	1901	1972	01	01	1939	1144	1974	818	0.0	79	157	0	900	0
01-209	F	1908	1975	01	01	1926	16	1959	6	0.02700	78B	1	2	20	32
01-210	M	1878	1971	06	06	1918	208	1959	12	0.0	79B	2	0	15	0
01-214	M	1891	1954	06	06	1915	1248	1959	42	0.00700	77B	19	4	156	47
01-216	F	1903	1963	01	01	1924	4	1959	0	0.08000	72B	0	0	0	0
01-217	M	1894	1971	01	01	1914	208	1959	5	0.0	79B	1	0	15	0
01-218	M	1924		06	06	1959	741	1974	0	0.0	79B	0	0	0	0
01-219	F	1910		01	01	1927	10	1976	0	0.00246	79B	0	0	0	0
01-220	F	1906		01	01	1924	26	1959	2	0.07100	72B	1	2	7	37
01-221	M	1892	1970	06	06	1915	520	1967	10	0.00320	77B	3	2	28	25
01-222	F	1910		01	01	1925	17	1964	4	0.04400	72C	1	5	15	79
01-223	F	1912		01	01	1927	7	1963	0	0.01200	78	0	0	0	0
01-225	F	1906		01	01	1931	35	1959	0	0.0	79D	0	0	0	0
01-226	F	1911		01	01	1927	22	1976	0	0.00258	78B	0	0	0	0
01-227	F	1908		07	07	1933	2184	1975	0	0.0	79B	0	0	0	0
01-228	F	1906		01	01	1926	61	1972	6	0.00420	78B	2	2	25	27
01-229	F	1903		01	01	1923	2	1959	8	0.08000	72B	2	13	30	196
01-230	F	1913		01	01	1927	19	1978	0	0.00203	78B	0	0	0	0

TABLE 1 (CONT.)
EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SPY	BORN	DIED	REF	TYPE	REF	YEAR	FA226	METHOD	TC	FA226	INPUT	INPUT	CUM	FADS
								NCI	+ERR	ESTIO	+ERR	NCI	NCI	RA226	RA228
01-231	P	1910	1960	01	1930	84	1959	0	B6	0.0	Z9B	0	0	0	0
01-232	P	1909	1961	04	1926	43	1959	0	B6	0.0	Z9B	0	0	0	0
01-233	P	1912	1973	04	1927	105	1959	2	B6	0.02000	Z8B	0	0	6	6
01-234	P	1913	1966	04	1927	1	1959	0	B6	0.02000	Z8B	0	0	0	0
01-235	P	1908		01	1926	5	1959	1	B6	0.08000	Z2B	0	1	4	19
01-236	P	1910	1976	01	1927	0	1965	1	B6	0.01000	Z9	0	0	4	4
01-237	P	1903		01	1927	8	1979	0	B6	0.00179	Z8B	0	0	0	0
01-238	P	1896	1967	01	1920	2	1959	1	B6	0.08000	Z2B	0	2	4	37
01-239	P	1901	1958	04	1917	78	1957	830	F4	0.00157	P3	223	41	2665	620
01-240	P	1910		04	1927	13	1971	7	D6	0.00450	Z8D	2	2	28	28
01-243	M	1873	1959	06	1905	520	1958	15	B6	0.0	Z9	4	0	43	0
01-244	P	1901	1979	01	1927	18	1975	1	B6	0.00307	Z9	0	0	4	5
01-245	P	1920		04	1957	30	1969	0	B6	0.0	Z9	0	0	0	0
01-246	P	1885	1970	06	1916	30	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	P	1903	1959	04	1917	208	1976	21	B2	0.0	Z9B	7	0	106	0
01-249	M	1928		08	1928	39	1967	2	B6	0.02700	Z2	1	2	5	17
01-250	M	1834		06	1916	520	1975	0	B6	0.0	Z9B	0	0	0	0
01-251	M	1899	1955	06	1912	156	1974	11	A2	0.0	Z9	3	0	34	0
01-252	P	1898		04	1917	104	1976	22	B1	0.0	Z9E	7	0	114	0
01-253	P	1898	1964	04	1916	104	1959	40	B6	0.0	Z9	11	0	147	0
01-254	P	1910		04	1927	2	1971	1	B6	0.00460	Z8B	0	0	4	4
01-255	P	1920		04	1942	52	1975	0	B6	0.0	Z9B	0	0	0	0
01-256	M	1919		06	1949	208	1959	14	B6	0.0	Z9	2	0	11	0
01-257	M	1885	1962	06	1941	624	1959	0	B6	0.0	Z9	0	0	0	0
01-258	M	1903		06	1923	1092	1969	17	B6	0.0	Z9	4	0	40	0
01-259	P	1910		06	1927	416	1977	0	B6	0.0	Z9	0	0	0	0
01-260	P	1891	1960	04	1918	50	1959	15	B6	0.0	Z9	4	0	50	0
01-261	P	1909	1969	04	1927	2	1959	0	B6	0.02000	Z8B	0	0	0	0
01-262	P	1895		06	1918	0	1969	22	B4	0.0	Z9	7	0	106	0
01-263	P	1897	1976	04	1917	17	1976	9	B6	0.0	Z9B	3	0	46	0
01-264	M	1906	1967	04	1944	770	1964	90	B4	0.0	Z9	13	0	59	0
01-265	P	1902		04	1919	3	1959	3	B6	0.08000	Z2B	1	8	13	126
01-266	P	1904	1961	04	1923	3	1959	1	B6	0.08000	Z2B	0	2	3	24
01-267	P	1904		04	1926	104	1966	45	B4	0.0	Z9	12	0	170	0

1 3 1344 (U.S.N.)
EXCELSECAVE 304 3740 304 3740
MIGRAPH 304 3740 304 3740
di SENTIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SER	BORN	DIED	FKP	TYPE	EXP	YRSE	TYPE	OP	MEAS	RA226 METHOD + EEE	RA226 INPUT UCI	RA226 FADS	RA226 FADS	RA226 FADS
01-268	F	1904	1968	01	1924	48	1967	100	E2	0.01000	R2	29	84	391	1264
01-269	M	1911	1932	06	1932	624	1970	2	B6	0.0	Z9B	1	0	5	0
01-270	P	1901	1943	01	1943	32	1976	4	B3	0.0	Z9	1	0	11	0
01-271	P	1909	1917	01	1917	86	1979	2	B6	0.0	Z9B	1	0	11	0
01-272	M	1888	1956	06	1956	130	1959	78	G6	0.0	Z9	4	0	22	0
01-273	P	1917	1924	01	1924	1	1959	2	B6	0.08400	Z2B	1	3	7	45
01-274	P	1906	1922	01	1922	5	1978	0	B6	0.00799	Z2B	0	0	0	0
01-275	M	1930	1959	06	1959	20	1959	23	G6	0.0	Z9	0	0	0	0
01-276	M	1934	1962	06	1945	208	1959	60	G6	0.0	Z9	9	0	39	0
01-277	P	1909	1925	01	1925	6	1978	5	C6	0.00828	W2	1	7	21	112
01-278	P	1904	1975	06	1925	3	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	P	1905	1926	01	1926	7	1971	0	B6	0.00460	Z9B	0	0	0	0
01-282	M	1893	1973	06	1916	156	1972	42	B2	0.0	Z9B	13	0	141	0
01-283	P	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	1923	01	1923	1	1960	4	B6	0.07100	Z9B	1	7	15	100
01-287	P	1908	1927	01	1927	674	1977	2	B6	0.00232	Z9B	1	0	7	3
01-288	P	1894	1972	01	1926	2	1960	2	C6	0.02400	Z9B	1	1	6	11
01-289	P	1899	1975	01	1919	80	1971	4	B3	0.01860	Z2B	1	12	18	175
01-291	P	1910	1969	01	1928	17	1960	5	F6	0.01800	Z9B	1	1	15	16
01-293	P	1911	1924	01	1924	11	1978	0	B6	0.00804	Z2B	0	0	0	0
01-294	P	1912	1927	01	1927	52	1971	3	B3	0.00450	Z2B	1	1	12	11
01-295	P	1917	1927	01	1927	14	1976	0	B6	0.00258	Z9B	0	0	0	0
01-296	P	1908	1927	01	1927	5	1960	0	B6	0.01800	Z9B	0	0	0	0
01-297	P	1901	1921	01	1921	122	1960	16	R2	0.09375	B3	4	39	63	589
01-299	P	1906	1917	01	1917	104	1968	3	G6	0.0	Z9	1	0	14	0
01-301	P	1904	1926	05	1926	5	1969	17	G4	0.0	Z9	5	0	68	0
01-302	P	1909	1966	05	1927	10	1968	450	A1	0.0	Z9A	7	0	6910	0
01-303	M	1919	1940	01	1940	104	1974	0	B6	0.0	Z9B	0	0	0	0
01-305	M	1925	1969	06	1946	140	1966	160	B4	0.0	Z9C	15	0	56	0
01-306	M	1928	1955	06	1955	364	1979	22	B1	0.0	Z9B	4	0	23	0
01-307	M	1930	1957	06	1957	104	1975	4	B6	0.0	Z9B	1	0	4	0
01-308	M	1918	1957	06	1943	728	1958	1200	P4	0.0	Z9F	90	0	247	0
01-309	P	1908	1972	01	1923	2	1961	2	B6	0.06200	Z2B	1	3	7	50

TABLE 1 (CONT.) SUMMARY DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP	YEAR	EXP	YEAR	FA225 NCI	FA226 METHOD	FA228 RATIO	FA229 METHOD + P35	INPUT UCI	INPUT UCI	CUM FADS FA226	CUM FADS FA228
01-310	F	1928	1928	06	1928	39	1975	0	B6	0.01148	Z2	0	0	0	0
01-311	F	1911	1927	01	1927	2	1961	1	B6	0.01500	Z9B	0	0	4	4
01-312	F	1907	1925	01	1925	13	1976	0	B6	0.0	Z9B	0	0	0	0
01-313	M	1892	1911	06	1911	624	1961	3	B3	0.0	Z9B	1	0	0	0
01-314	F	1909	1924	01	1924	0	1961	1	B6	0.06200	Z2B	0	1	4	22
01-324	F	1927	1923	01	1923	15	1962	1	B6	0.05700	Z2	0	2	4	26
01-326	F	1896	1977	02	1925	156	1966	100	B4	0.01100	Z5	27	36	349	539
01-327	F	1908	1927	01	1927	1	1965	0	B6	0.01000	Z8	0	0	0	0
01-330	M	1915	1942	06	1942	364	1976	66	B2	0.0	Z9B	16	0	118	0
01-331	M	1901	1927	02	1927	70	1966	80	B4	0.01100	Z5	21	27	216	290
01-332	F	1912	1971	01	1927	52	1965	0	B6	0.01000	Z8	0	0	0	0
01-333	F	1905	1924	01	1924	10	1976	0	B6	0.01075	Z2	0	0	0	0
01-335	F	1909	1917	16	1917	78	1975	3	B3	0.0	Z9B	1	0	15	0
01-336	M	1899	1945	06	1945	1092	1979	41	B1	0.0	Z9B	8	0	49	0
01-341	M	1883	1943	06	1943	176	1961	5	B3	0.0	Z9B	1	0	7	0
01-342	M	1897	1947	06	1947	56	1961	1	B6	0.0	Z9B	0	0	1	0
01-343	F	1873	1954	04	1927	70	1963	0	F6	0.0	Z9	0	0	0	0
01-344	F	1904	1976	01	1922	19	1962	7	B6	0.05700	Z2	2	14	27	206
01-345	F	1910	1977	01	1924	1	1962	4	B6	0.05700	Z2	1	6	15	92
01-346	F	1911	1977	01	1927	17	1962	44	B6	0.01700	Z8	11	13	157	196
01-347	M	1896	1969	06	1926	1672	1962	10	B2	0.0	Z9B	2	0	10	0
01-348	F	1902	1973	01	1924	19	1966	112	B1	0.03492	B4	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	B6	0.05700	Z2	0	0	0	0
01-351	F	1906	1973	01	1923	3	1962	0	B6	0.05700	Z2	0	0	0	0
01-352	M	1922	1973	06	1940	338	1962	191	B1	0.0	Z9B	35	0	275	0
01-356	M	1912	1973	06	1937	572	1969	23	B2	0.0	Z9B	5	0	36	0
01-357	F	1907	1972	07	1927	408	1962	0	B6	0.01400	Z8	0	0	0	0
01-358	F	1906	1978	07	1923	168	1962	0	B6	0.05700	Z2	0	0	0	0
01-359	F	1908	1978	01	1925	55	1962	25	B2	0.05600	Z2B	6	31	93	460
01-360	F	1911	1977	01	1928	34	1967	0	B6	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	1988	01	1923	5	1962	0	B6	0.05700	Z2	0	0	0	0
01-363	F	1911	1978	01	1918	260	1962	7	B6	0.05700	Z2	2	17	29	253
01-364	F	1908	1977	07	1927	440	1964	6	B6	0.01140	Z8	1	1	20	13

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

[illegible]

TABLE 1 (CONT.) EXCISE DATA FOR ALIUM PATIENTS TO END OF 1973

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SPX	BOUN	UICD	FXP	YPR	EXE	YPR	FA226	FA226	FA226	FA226	FA226	FA226	FA226	FA226
01-403	P	1912	1912	02	1912	1912	1912	27	27	0.0183R	C3	330	34	112	516
01-404	M	1915	1915	06	1915	1915	1915	2800	A1	0.0	29A	330	0	1523	0
01-405	M	1915	1915	06	1915	1915	1915	52	A1	0.0	29A	11	0	100	0
01-406	M	1912	1912	06	1912	1912	1912	18	B2	0.0	29B	0	0	51	0
01-407	M	1912	1912	06	1912	1912	1912	38	B2	0.0	29B	9	0	78	0
01-408	P	1918	1918	06	1918	1918	1918	14	A1	0.0	29B	4	0	46	0
01-409	P	1914	1914	06	1914	1914	1914	34	B3	0.0	29B	10	0	133	0
01-410	P	1910	1910	06	1910	1910	1910	33	B1	0.0	29B	9	0	97	0
01-411	M	1915	1915	06	1915	1915	1915	6	B2	0.0	29C	2	0	18	0
01-412	M	1915	1915	02	1915	1915	1915	1	B6	0.01600	25D	0	0	2	3
01-413	P	1901	1901	01	1901	1901	1901	11	G4	0.04400	22	0	15	35	222
01-414	P	1907	1907	06	1907	1907	1907	2	C6	0.0	23B	1	0	9	0
01-415	M	1908	1908	06	1908	1908	1908	0	B6	0.0	29B	0	0	0	0
01-416	P	1908	1908	01	1908	1908	1908	9	G6	0.04900	22	2	14	35	203
01-417	P	1907	1907	01	1907	1907	1907	0	G6	0.05000	22	0	0	0	0
01-418	M	1900	1900	06	1900	1900	1900	6	B6	0.0	29	2	0	17	0
01-419	M	1895	1895	06	1895	1895	1895	9	G6	0.0	29	3	0	24	0
01-420	M	1902	1902	06	1902	1902	1902	2	G6	0.0	29	1	0	7	0
01-421	P	1887	1887	06	1887	1887	1887	8	G6	0.0	29	2	0	35	0
01-422	M	1897	1897	06	1897	1897	1897	22	B2	0.0	29P	7	0	73	0
01-423	P	1882	1882	05	1882	1882	1882	20	G4	0.0	29	76	0	1114	0
01-424	P	1882	1882	05	1882	1882	1882	0	B6	0.0	29B	0	0	0	0
01-425	M	1933	1933	07	1933	1933	1933	5	B6	0.0	29B	0	0	0	0
01-426	P	1930	1930	07	1930	1930	1930	5	B6	0.0	29B	0	0	2	0
01-427	P	1960	1960	07	1960	1960	1960	5	B6	0.0	29	0	0	2	0
01-428	P	1957	1957	07	1957	1957	1957	2	B6	0.0	29	0	0	1	0
01-429	P	1897	1897	06	1897	1897	1897	1	B6	0.0	29B	0	0	5	0
01-430	M	1903	1903	02	1903	1903	1903	41	B2	0.02105	B3	11	18	38	197
01-431	P	1901	1901	05	1901	1901	1901	765	B1	0.0	29B	229	0	3262	0
01-432	M	1935	1935	06	1935	1935	1935	17	B2	0.0	29B	5	0	49	0
01-433	M	1880	1880	02	1880	1880	1880	6126	A1	0.02189	A1	456	029	865	3250
01-434	P	1907	1907	01	1907	1907	1907	0	B6	0.00228	29B	0	0	0	0
01-435	P	1895	1895	01	1895	1895	1895	8	G6	0.01140	29	2	2	27	25
01-436	P	1910	1910	06	1910	1910	1910	1	B6	0.0	29B	0	0	3	0
01-437	P	1910	1910	06	1910	1910	1910	1850	A1	0.01372	A1	279	382	1163	3571
01-438	M	1967	1967	02	1967	1967	1967	406	A2	0.0	29F	96	0	971	0
01-439	P	1880	1880	04	1880	1880	1880	0	A2	0.0	29F	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
	SPX-ROPN	DIED	TYPE	PKG	AGE	SEX	DOB	FAZ26	FAZ26	FAZ26	FAZ26	FAZ26	FAZ26	FAZ26	FAZ26
01-440	F	1909		01	1924	204	1965	0	66	0.03000	22	0	0	0	0
01-441	F	1911		01	1927	74	1975	8	36	0.02000	28	2	2	34	33
01-442	F	1903		01	1925	110	1965	1	36	0.01000	28	1	1	11	14
01-447	F	1907		01	1925	10	1974	45	34	0.01140	28	7	9	97	131
01-448	F	1907		01	1925	10	1974	45	34	0.01140	28	7	9	97	131
01-449	F	1909		01	1922	2	1955	7	76	0.03900	22	2	14	30	215
01-450	M	1977	1978	05	1912	364	1966	0	56	0.0	22A	0	0	0	0
01-451	P	1909	1978	01	1924	4	1977	14	64	0.00907	22	4	25	64	375
01-453	P	1939	1963	01	1920	20	1970*	4	84	0.00780	22	1	11	14	168
01-454	P	1980	1970	01	1920	284	1974	1990	81	0.0	586	0	0	7760	0
01-456	M	1978	1943	02	1928	26	1985	74	81	0.03648	83	14	44	75	454
01-457	L	1906	1906	01	1924	10	1965	10	36	0.0	28	2	0	34	0
01-458	M	1906	1906	01	1924	10	1965	10	36	0.0	28	2	0	27	0
01-460	M	1942	1966	06	1912	104	1964	0	66	0.0	29	0	0	0	0
01-461	M	1914	1970	06	1930	26	1964	9	34	0.0	29	2	0	19	0
01-464	P	1903		01	1927	4	1970	4	36	0.00540	28	1	1	16	17
01-466	F	1902	1946	01	1920	52	1965	0	86	0.03800	22A	0	0	0	0
01-468	F	1910		01	1927	0	1978	0	66	0.00209	28	0	0	0	0
01-469	M	1894		06	1914	52	1965	4	76	0.0	29	1	0	13	0
01-470	P	1912		01	1927	70	1965	0	66	0.01000	28	0	0	0	0
01-472	P	1896	1963	06	1916	155	1965	7	36	0.0	28	2	0	27	0
01-474	P	1904		07	1921	100	1979	0	26	0.00537	22B	0	0	0	0
01-475	P	1901		01	1926	4	1974	0	66	0.00330	28B	0	0	0	0
01-476	P	1909		07	1927	71	1972	4	83	0.00420	28B	1	1	16	16
01-477	P	1897	1979	02	1925	40	1965	1240	81	0.00475	82	336	207	4814	3111
01-478	F	1914		01	1935	24	1965	0	36	0.0	20	0	0	0	0
01-479	F	1914		01	1927	1	1978	2	66	0.00209	28	1	1	10	11
01-480	P	1915		01	1927	1	1965	38	66	0.01000	28	10	10	142	153
01-481	P	1909		01	1927	14	1965	0	76	0.01000	28	0	0	0	0
01-482	P	1912		01	1927	6	1979	1	86	0.00141	28B	0	0	4	5
01-483	M	1917		07	1922	104	1975	0	86	0.01194	22B	0	0	0	0
01-484	P	1908	1974	01	1926	0	1965	0	66	0.01000	28	0	0	0	0
01-485	M	1870	1951	05	1911	1307	1965	340	41	0.0	28A	74	0	488	0
01-486	P	1907		01	1923	6	1974	0	86	0.01319	22B	0	0	0	0
01-487	P	1911		07	1927	565	1976	0	86	0.00257	28B	0	0	0	0
01-489	P	1910		01	1926	348	1965	225	66	0.01000	28	57	42	787	637

АЛЕ, И ГРЕО СИНТАВО ПО-МЪС (4 ВЪЛ ИЛИ-ОСТА (200)) И ИЛИ

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE#	SEX	BORN	DIED	EXP	YEAR	EXP	YEAR	FA226	FA226	FA226	FA226	FA226	FA226	FA226	FA226
01-490	F	1904	1966	01	1924	17	1974	2	36	0.01318	22B	1	1	9	52
01-491	F	1922	1966	01	1943	74	1963	7	36	0.0	73	1	0	7	0
01-492	F	1922	1966	06	1941	260	1973	1	86	0.0	29C	0	0	4	0
01-493	M	1943	1975	06	1927	1920	1973	4	83	0.0	29C	1	0	6	0
01-494	M	1906	1966	06	1926	499	1965	0	66	0.0	75	0	0	0	0
01-495	F	1908		01	1924	4	1965	0	66	0.03900	24	0	0	0	0
01-496	F	1918		07	1934	106	1966	3	66	0.0	29	1	0	9	0
01-497	P	1922	1978	01	1921	8	1966	13	66	0.03400	72	4	30	56	451
01-498	F	1897		06	1920	104	1976	1	86	0.0	29C	0	0	5	0
01-501	M	1967	1937	02	1926	156	1965	2500	81	0.0760	81	320	2	1102	2149
01-503	M	1936		08	1936	39	1966	0	86	0.0	29B	0	0	0	0
01-504	F	1913		01	1927	2	1975	0	86	0.0	29B	0	0	0	0
01-505	F	1902		01	1927	1	1966	9	84	0.00880	28	2	2	34	37
01-506	F	1897		04	1923	4	1966	7	83	0.0	29C	2	0	29	0
01-507	F	1909		01	1927	22	1974	10	82	0.00313	78B	3	3	41	41
01-508	P	1906	1969	01	1944	52	1966	30	86	0.0	29	6	0	50	0
01-509	P	1943		06	1943	39	1967	0	86	0.0	29B	0	0	0	0
01-510	F	1897		01	1927	12	1966	38	86	0.00880	28	10	10	143	152
01-511	F	1908		07	1927	9	1979	0	86	0.00181	28B	0	0	0	0
01-512	F	1895	1976	04	1912	13	1973	0	86	0.0	29B	0	0	0	0
01-514	P	1904		07	1924	2184	1975	0	86	0.02200	25B	0	0	0	0
01-515	F	1886		05	1940	0	1966	4	86	0.0	29	1	0	10	0
01-516	P	1907	1976	01	1927	2	1967	7	86	0.00780	28	2	2	26	29
01-518	M	1912		05	1949	40	1977	0	86	0.0	29B	1	0	0	0
01-519	M	1919		06	1937	260	1967	13	86	0.0	29	3	0	24	0
01-520	F	1882	1969	02	1930	40	1967	670	81	0.00492	P2	174	77	2044	1158
01-521	M	1910		06	1942	520	1979	21	B2	0.0	29B	5	0	37	0
01-522	M	1905		06	1928	7288	1979	169	C2	0.0	29B	35	0	237	0
01-523	M	1917		06	1942	312	1968	30	G4	0.0	25	6	0	47	0
01-525	M	1923		06	1943	104	1968	17	06	0.0	79	4	0	28	0
01-526	M	1921		06	1945	38	1979	29	B1	0.0	29B	7	0	55	0
01-529	M	1920		06	1943	260	1975	14	92	0.0	29B	3	0	25	0
01-530	M	1918	1971	06	1943	104	1966	52	B1	0.0	29B	11	0	71	0
01-531	M	1918		06	1941	354	1974	13	B2	0.0	29B	3	0	23	0
01-532	M	1914	1979	06	1945	138	1968	1	G6	0.0	29	0	0	1	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SPX	BORN	DIED	EXP	TYPE	AGE	WKS	DUR	YFAE	RA22b	RA22b	RA22b	RA22b	RA22b	RA22b
01-533	F	1913	1978	04	07	1911	15	10	06	0.0	0.0	0.0	0	22	0
01-534	M	1910		06	06	1944	154	1076	1	06	0.0	0.0	0	2	0
01-536	M	1916		06	06	1942	245	1554	17	34	0.0	0.0	0	20	0
01-537	M	1917	1971	06	06	1944	204	1545	34	31	0.0	12	0	74	0
01-540	M	1942		07	07	1940	260	1548	0	06	0.0	0	0	0	0
01-543	M	1920	1978	06	06	1943	147	1075	10	02	0.0	4	0	32	0
01-544	F	1979	1953	02	02	1930	42	1369	03	01	0.00430	19	0	158	12
01-546	F	1907		01	01	1914	52	1967	01	06	0.0	0	0	0	0
01-547	P	1907		06	06	1920	104	1570	4	03	0.0	1	0	20	0
01-548	M	1917		02	02	1931	42	1572	5	03	0.00200	1	0	14	5
01-552	M	1937		06	06	1936	134	1507	27	34	0.0	5	0	41	0
01-553	F	1913		01	01	1948	988	1967	0	06	0.0	0	0	0	0
01-554	F	1928		01	01	1952	780	1967	490	04	0.0	38	0	286	0
01-555	F	1934		01	01	1921	2	1975	0	06	0.01155	0	0	0	0
01-556	F	1913		01	01	1927	0	1967	0	06	0.00780	0	0	0	0
01-557	P	1903		01	01	1925	35	1975	2	06	0.00293	1	1	9	11
01-558	M	1913		02	02	1927	137	1979	313	01	0.00053	06	24	955	262
01-562	F	1901	1931	01	01	1920	52	1970	10300	01	0.0	102	0	7143	0
01-565	F	1942	1957	05	05	1925	24	1970	1600	02	0.0	385	0	5946	0
01-567	M	1945	1940	02	02	1925	40	1970	1100	02	0.00470	229	218	1400	2282
01-569	M	1907	1928	05	05	1927	42	1960	4900	01	0.0	237	0	270	0
01-569	F	1906		07	07	1922	282	1978	4	06	0.00004	1	6	18	97
01-570	F	1909		01	01	1926	260	1968	10	04	0.0	3	0	37	0
01-571	P	1911		01	01	1928	44	1979	0	06	0.00161	0	0	0	0
01-573	F	1942	1944	01	01	1916	312	1970	670	01	0.00195	145	135	1307	2000
01-574	P	1985	1937	05	05	1924	77	1968	2730	01	0.0	400	0	2255	0
01-575	M	1910	1977	01	01	1950	1196	1973	2	06	0.0	0	0	1	0
01-576	F	1930		01	01	1946	780	1964	160	01	0.0	25	0	219	0
01-578	F	1904	1930	05	05	1926	17	1964	3700	02	0.0	296	0	636	0
01-579	F	1928	1928	06	06	1928	26	1973	2	01	0.00269	0	0	1	0
01-580	P	1934		01	01	1918	52	1972	1	06	0.0	0	0	5	0
01-581	M	1918		06	06	1946	52	1968	10	04	0.0	2	0	15	0
01-582	P	1903		06	06	1917	24	1979	0	06	0.0	0	0	5	0
01-583	M	1898	1949	06	06	1918	104	1968	1	06	0.00250	0	0	0	0
01-584	P	1938	1975	01	01	1926	257	1968	10	02	0.0	3	0	35	0

TABLE 1 (CONT.) POSSIBLE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP	YR	EXP	YR	FA225	RA226	RA228	RA226	INPUT	INPUT	COM	CUM
								NCI	METHOD	TO RA226	METHOD	UCI	UCI	RA226	RA228
01-585	F	1906	1969	31	1975	26	1968	3	B6	0.0045	75B	37	0	0	0
01-586	F	1924	1973	35	1924	40	1968	133	B6	0.0	29	37	0	504	3
01-588	F	1909	1973	31	1929	104	1968	5	B6	0.0	29	1	0	18	0
01-589	M	1907	1973	36	1927	78	1978	1	B6	0.0	29	0	0	3	0
01-590	M	1929	1973	39	1929	39	1976	0	B6	0.01062	22C	0	0	0	0
01-591	F	1918	1975	41	1918	52	1973	0	B6	0.00016	27	0	0	0	0
01-592	F	1923	1974	41	1917	6	1968	0	B6	0.0	29	0	0	0	0
01-594	M	1927	1974	41	1962	34	1975	2	B6	0.0	29B	0	0	1	0
01-595	F	1927	1974	41	1917	130	1969	5	B6	0.0	29	2	0	24	0
01-597	F	1923	1974	41	1943	364	1973	1	B6	0.0	29C	0	0	3	0
01-598	M	1979	1953	36	1941	572	1952	403	B6	0.0	29	27	0	71	0
01-599	F	1909	1974	41	1927	7	1978	0	B6	0.00203	29B	0	0	0	0
01-601	F	1902	1974	41	1918	6	1969	0	B6	0.00020	27	0	0	0	0
01-603	F	1954	1974	41	1915	676	1968	7	B6	0.00450	25	2	3	32	41
01-604	F	1966	1974	41	1914	52	1971	1	B6	0.0	29B	0	0	5	0
01-607	F	1937	1974	41	1927	40	1978	0	B6	0.00203	28	0	0	0	0
01-608	F	1936	1974	41	1927	11	1974	0	B6	0.00330	28B	0	0	0	0
01-609	F	1936	1974	41	1926	366	1978	1	B6	0.0	29B	0	0	4	0
01-610	M	1904	1969	36	1919	208	1968	10	B6	0.00450	27	3	4	28	43
01-612	F	1959	1936	17	1923	255	1972	18	A1	0.00680	24A	2	5	13	57
01-613	F	1936	1936	17	1923	265	1972	658	A1	0.00680	P2	88	165	450	1987
01-614	M	1982	1922	36	1920	40	1974	24	A2	0.0	29	1	0	2	0
01-617	M	1922	1974	41	1922	39	1973	4	P3	0.00020	23B	1	0	13	1
01-619	F	1959	1974	41	1927	52	1969	0	B6	0.0	29	0	0	0	0
01-621	F	1908	1974	41	1924	2	1978	8	B2	0.00731	29C	3	14	37	214
01-625	F	1911	1974	41	1927	468	1968	6	B6	0.0	29	2	0	21	0
01-626	F	1932	1974	41	1932	39	1971	0	B6	0.0	29B	0	0	0	0
01-627	F	1937	1974	41	1937	52	1970	0	B6	0.0	29	0	0	0	0
01-628	F	1908	1974	41	1925	312	1975	0	B6	0.00200	25B	3	0	0	0
01-629	F	1992	1977	31	1926	260	1969	12	B6	0.0	29	3	0	44	0
01-633	F	1978	1926	35	1925	4	1970	2600	A2	0.0	29A	131	0	130	0
01-635	M	1983	1937	46	1918	312	1973	1900	A1	0.0	29A	319	0	1509	0
01-636	F	1979	1930	41	1919	1	1974*	1	A6	0.00075	27	0	0	1	1
01-640	F	1908	1974	41	1924	21	1969	34	B6	0.00420	25	13	13	143	143
01-653	F	1910	1974	41	1925	73	1969	7	B6	0.00420	25	2	2	29	25

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIIUM PATIENTS TO END OF 1975

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	DOB	DATE	TYPE	YR	EXP	OF	FA226	FA226	FA226	FA226	INPUT	INPUT	CUH	CUH
					1928	1928	1928	1928	1928	1928	1928	1928	1928	1928	1928
					1928	1928	1928	1928	1928	1928	1928	1928	1928	1928	1928
01-559	F	1912	1957	01	1928	24	1063	11	36	0.0	79	3	0	42	0
01-560	F	1911	1957	04	1932	40	1070	15	46	0.0	79A	3	0	29	0
01-561	F	1874	1934	06	1914	572	1074	2	44	0.0	79	0	0	1	0
01-563	F	1927		08	1927	35	1563	11	54	0.0	79	3	0	31	0
01-565	F	1923		08	1923	39	1565	3	56	0.0	79	0	0	0	0
01-567	F	1918		01	1941	234	1572	0	56	0.0	79B	0	0	0	0
01-568	F	1933		07	1964	40	1974	1	56	0.0	79	0	0	1	0
01-569	F	1917		01	1934	104	1969	0	56	0.0	79	0	0	0	0
01-570	F	1927		04	1928	40	1969	0	56	0.0	79	0	0	0	0
01-571	F	1923		01	1941	260	1972	2	56	0.0	79B	0	0	5	0
01-574	F	1928		01	1931	1716	1973	0	56	0.0	79B	0	0	0	0
01-581	F	1904	1978	07	1920	4	1972	0	56	0.00320	77	0	0	0	0
01-584	F	1924	1974	01	1917	1	1973	0	56	0.0	79	0	0	0	0
01-588	F	1868	1948	07	1920	40	1972	0	56	0.00320	77A	0	0	0	0
01-590	F	1978	1940	04	1919	40	1970	21	51	0.0	79A	4	0	24	0
01-591	F	1913	1974	04	1935	0	1971	0	56	0.0	79B	0	0	0	0
01-592	F	1905	1974	02	1925	40	1970	30	56	0.03680	75	0	0	84	150
01-594	F	1986	1952	04	1928	40	1971	10000	54	0.0	74	2123	0	13346	0
01-701	F	1892	1974	06	1916	312	1970	0	56	0.0	74	0	0	0	0
01-706	F	1908		07	1922	100	1975	0	56	0.01149	72	0	0	0	0
01-707	F	1908	1974	01	1927	1	1971	0	56	0.00470	78	0	0	0	0
01-710	F	1921		01	1925	289	1978	0	56	0.00141	75	0	0	0	0
01-711	F	1905		01	1925	312	1970	0	56	0.00370	75	0	0	0	0
01-715	F	1907		01	1927	5	1976	0	56	0.00258	78B	0	0	0	0
01-717	F	1917		27	1927	13	1979	3	53	0.00230	75	1	1	9	13
01-728	F	1912		01	1927	6	1978	0	56	0.00203	78B	0	0	0	0
01-731	F	1925		01	1926	4	1970*	3	56	0.00200	78	1	1	13	18
01-733	F	1911		01	1927	61	1978	45	56	0.00200	78	14	13	193	190
01-736	F	1927	1931	01	1923	52	1977	1	56	0.00170	27F	0	0	0	1
01-739	F	1856	1929	05	1926	7	1972	11500	51	0.0	79A	645	0	1226	0
03-005	F	1917	1978	07	1948	40	1973	0	56	0.0	79C	0	0	0	0
03-008	F	1934		08	1934	39	1971	0	56	0.0	79C	0	0	0	0
03-029	F	1918		01	1941	104	1972	1	56	0.0	79C	0	0	2	0
03-101	F	1908	1971	05	1931	15	1963	1580	51	0.0	79	390	0	4523	0
03-102	F	1908	1976	05	1931	15	1973	628	51	0.0	79C	174	0	1598	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADON PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP. TYPE	YAP	EXP. DUE	YAP	RA226 NCI	RA226 METHOD + RRF	RA226 TO RA226 RATIO	RA226 METHOD + RRF	INPUT RA226 UCI	INPUT RA226 UCI	CUM. PADS RA226	CUM. RADS RA226
03-103	F	1868	1952	05	1931	15	1931	420	B4	0.0	29	79	0	621	0
03-104	F	1887	1945	05	1931	15	1931	13900	B4	0.0	29	419	0	2727	0
03-105	F	1903	1957	05	1931	16	1931	2600	B4	0.0	29	437	0	3143	0
03-106	F	1876	1950	05	1931	16	1931	4600	B2	0.0	29	147	0	1388	0
03-107	F	1894	1957	05	1931	16	1931	3600	B2	0.0	29	115	0	1036	0
03-108	F	1875	1953	05	1931	16	1932	1900	B4	0.0	29	82	0	660	0
03-109	F	1904	1957	05	1931	18	1953	630	B2	0.0	29	125	0	1120	0
03-110	F	1899	1967	05	1931	20	1964	584	B1	0.0	29	143	0	1583	0
03-111	F	1909		05	1931	20	1973	879	B2	0.0	29C	244	0	3264	0
03-112	F	1899	1968	05	1931	26	1960	5310	B1	0.0	29	1212	0	13669	0
03-113	F	1914	1946	05	1931	38	1932	7800	B4	0.0	29	356	0	2115	0
03-114	F	1901	1968	05	1931	36	1964	949	B1	0.0	29	231	0	2606	0
03-115	F	1911		05	1931	26	1973	745	B1	0.0	29C	206	0	2762	0
03-116	F	1907		05	1931	25	1973	1411	B1	0.0	29C	391	0	5232	0
03-117	M	1898	1957	05	1931	45	1953	1540	B2	0.0	29	303	0	1931	0
03-118	F	1898	1955	05	1931	41	1953	3090	B2	0.0	29	608	0	5159	0
03-119	F	1880	1960	05	1931	7	1959	1038	C2	0.0	29	233	0	2256	0
03-120	F	1879	1937	05	1931	11	1931	5300	B4	0.0	29	171	0	622	0
03-121	F	1911	1972	05	1931	9	1964	371	B1	0.0	29	91	0	1099	0
03-122	M	1908		05	1931	10	1931	6500	B4	0.0	29	92	0	880	0
03-123	M	1914	1937	05	1931	9	1931	9700	B2	0.0	29	139	0	361	0
03-124	M	1910		05	1931	9	1979	207	C2	0.0	29C	62	0	590	0
03-125	F	1913	1976	05	1931	11	1973	556	B1	0.0	29C	154	0	1983	0
03-126	F	1910	1965	05	1931	20	1965	1300	C2	0.0	29	323	0	3449	0
03-127	F	1908		05	1931	26	1962	565	C2	0.0	29	134	0	1787	0
03-135	M	1905		05	1931	10	1973	1431	B1	0.0	29C	398	0	3814	0
03-139	M	1908		05	1933	11	1973	373	C2	0.0	29C	101	0	940	0
03-140	M	1905	1937	05	1933	11	1961	500	F4	0.0	29	40	0	82	0
03-141	M	1906	1963	05	1933	11	1962	961	C2	0.0	29	220	0	1550	0
03-201	F	1909	1963	04	1922	10	1962	2968	C2	0.0	29	875	0	9741	0
03-202	M	1895		05	1925	10	1960	1800	B4	0.0	29	455	0	4714	0
03-203	F	1903	1973	05	1933	10	1959	84	C2	0.0	29	18	0	217	0
03-204	F	1896	1970	04	1922	10	1960	21	C2	0.0	29	6	0	74	0
03-205	F	1900	1979	05	1929	15	1968	291	C2	0.0	29	78	0	1969	0
03-206	M	1914	1975	05	1936	4	1973	3297	B1	0.0	29C	858	0	7176	0

TABLE 1 (CONT.) SUCCESS DATA FOR BARIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP	YEAR	FIRST	EXP	NCI	METHOD	TO RATIO	METHOD	INPUT	INPUT	CUM	CUM
				TYPE	EXP	DOF	YEAS	NCI	+	+	+	RA226	RA226	RA226	RA226
03-207	P	1879	1969	04	1922	416	1969	755	C2	C.C	Z9	188	0	2344	0
03-209	F	1894	1960	05	1925	572	1973	1105	A1	C.C	Z9A	254	0	1776	0
03-210	F	1906	1959	05	1926	40	1957	1350	C2	C.C	F2	321	12	2360	132
03-211	M	1890	1950	05	1923	30	1950	10	C3	C.C	Z9	3	0	27	0
03-212	F	1922	1951	04	1927	40	1951	1300	B2	C.C	F1	270	7	2317	95
03-213	P	1922	1955	05	1925	40	1952	6570	B2	C.C	Z9	1452	0	14358	0
03-214	F	1895	1961	05	1925	40	1964	1382	C2	C.C	Z9F	370	0	4477	0
03-215	M	1896	1971	05	1925	40	1961	3630	C2	C.C	Z9	932	0	8685	0
03-216	P	1907	1961	05	1922	40	1961	530	C2	C.C	Z9F	142	0	1662	0
03-217	M	1912	1974	05	1921	40	1963	460	C2	C.C	Z9	128	0	1308	0
03-218	M	1908	1968	05	1924	40	1972	3	B3	C.C	Z9C	1	0	10	0
03-219	F	1888	1961	04	1919	208	1951	60	B2	C.C	Z9	14	0	178	0
03-220	M	1920	1928	04	1928	208	1976	130	B1	C.C	Z9C	38	0	367	0
03-221	M	1908	1963	05	1924	40	1957	620	C2	C.C	Z9	152	0	1273	0
03-222	M	1872	1954	05	1922	40	1951	1600	B2	C.C	Z9	167	0	2702	0
03-223	F	1886	1968	05	1929	156	1951	4200	B2	C.C	Z9	804	0	9181	0
03-224	M	1869	1960	04	1922	364	1951	5400	B2	C.C	Z9	1155	0	8929	0
03-225	M	1922	1953	04	1929	40	1977	31	B1	C.C	Z9C	9	0	92	0
03-226	M	1874	1953	05	1934	39	1951	10700	B2	C.C	Z9	1837	0	9588	0
03-227	P	1878	1952	05	1930	40	1951	1000	B2	C.C	Z9	193	0	1612	0
03-228	M	1900	1955	05	1927	40	1951	5600	B2	C.C	Z9	1164	0	7866	0
03-230	P	1899	1965	05	1927	40	1976	438	B1	C.C	Z9C	132	0	1865	0
03-231	F	1879	1972	05	1939	40	1952	60	B4	C.C	Z9	9	0	97	0
03-232	P	1898	1957	05	1917	40	1956	4700	D2	C.C	Z9	1257	0	14981	0
03-233	F	1979	1947	05	1922	40	1947	4000	C4	C.C	Z9	949	0	7473	0
03-234	F	1990	1965	05	1915	40	1965	920	C2	C.C	Z9	280	0	3861	0
03-235	P	1930	1968	05	1928	40	1965	1290	C2	C.C	Z9	336	0	4001	0
03-236	P	1880	1961	05	1927	40	1951	500	B2	C.C	Z9	104	0	1114	0
03-237	P	1890	1954	04	1923	156	1961	3	C6	C.C	Z9	1	0	11	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	DOB	DIED	EXP	YEAR	EXP	YEAR	FA226 NFI	FA226 METHOD	FA226 TO	FA226 METHOD	INPUT	INPUT	CUM	CUM
				TYPE	EXP	WKS	OF		+ ERE	FA226	+ ERE	RA226	RA226	RA226	RA226
												UCI	UCI	FA226	FA226
03-4304	F	1897		01	1923	195	1975	577	B1	0.0	29C	177	0	2575	0
03-4305	F	1924		16	1924	273	1962	625	C2	0.0	29	159	0	2257	0
03-4306	F	1914		01	1935	481	1972	7	B3	0.0	29C	2	0	20	0
03-4307	F	1905	1961	01	1923	1196	1958	1545	B1	0.00022	F	362	5	4285	73
03-4308	F	1908	1950	01	1924	676	1957	160	C2	0.0	29	39	0	414	0
03-4309	F	1923		01	1942	78	1972	8	B2	0.0	29C	2	0	21	0
03-4310	F	1895	1974	01	1923	104	1957	60	C2	0.0	29	15	0	203	0
03-4311	F	1928		01	1931	572	1976	1	B3	0.0	29C	0	0	5	0
03-4312	F	1894		01	1922	124	1977	227	B2	0.0	29C	72	0	1962	0
03-4313	F	1917	1978	01	1939	169	1972	1	B6	0.0	29C	0	0	2	0
03-4314	F	1921		01	1946	557	1972	3	B6	0.0	29C	1	0	5	0
03-4315	F	1911	1973	01	1930	780	1957	15	C3	0.0	29	3	0	30	0
03-4316	F	1907		01	1923	65	1970	1075	C2	0.0	29C	345	0	5085	0
03-4317	F	1909	1967	01	1924	60	1964	617	C2	0.0	29	166	0	2023	0
03-4318	F	1896		61	1926	602	1972	4	B3	0.0	29C	1	0	14	0
03-4319	F	1906		01	1924	208	1962	679	C2	0.0	29	177	0	2562	0
03-4320	F	1906	1950	01	1922	212	1957	18	C2	0.0	29	4	0	49	0
03-4321	F	1908		71	1924	117	1979	3	C3	0.0	29C	1	0	14	0
03-4322	F	1907		06	1925	104	1978	10	C1	0.0	29C	3	0	45	0
03-4323	F	1907	1972	01	1923	641	1962	591	C2	0.0	29	155	0	2064	0
03-4324	F	1905		01	1923	186	1978	245	C2	0.0	29C	77	0	1126	0
03-4325	F	1916		01	1935	260	1973	2	B6	0.0	29C	1	0	6	0
03-4326	F	1906		01	1924	2184	1979	131	C2	0.0	29C	41	0	691	0
03-4327	F	1906		01	1925	823	1973	12	B2	0.0	29C	4	0	53	0
03-4328	F	1908		01	1925	164	1974	493	B1	0.0	29C	148	0	2127	0
03-4329	F	1908	1976	01	1923	208	1974	1169	B1	0.0	29C	354	0	4975	0
03-4330	F	1922		01	1941	468	1971	4	B3	0.0	29C	1	0	10	0
03-4331	F	1931		01	1922	156	1963	1297	C2	0.0	29	349	0	5155	0
03-4332	F	1902		01	1923	112	1977	24	C2	0.0	29C	7	0	108	0
03-4333	F	1904		01	1924	117	1964	1052	C2	0.0	29	281	0	4080	0
03-4334	F	1920		01	1941	125	1975	5	B2	0.0	29C	1	0	13	0
03-4335	F	1912		01	1924	104	1971	3	B6	0.0	29C	1	0	8	0
03-4336	F	1910		01	1926	619	1975	8	B3	0.0	29C	2	0	31	0
03-4337	F	1906		01	1926	52	1957	55	C2	0.0	29	13	0	184	0
03-4338	F	1908		01	1925	8	1957	0	C6	0.0	29	0	0	0	0

TABLE 1 (CONT.) BYOSUPE DATA FOR RADIUM PATIENTS TO END OF 1975

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DATE	EXP	YEAR	EXP	OF	FA226	METHOD	CO	FA226	INPUT	INPUT	RADS	RADS
				TYPE	EXP	WKS	MEAS	MCI	+ERR	RATIO	FA226	UCI	UCI	HA226	HA226
03-439	F	1906		01	1925	56	1957	3	C6	0.0	79	0	0	0	0
03-440	F	1908		01	1925	3	1970	1	C6	0.0	29C	0	0	3	0
03-441	F	1905		01	1925	528	1957	56	C2	0.0	29	13	0	193	0
03-442	F	1914		01	1924	13	1976	4	B2	0.0	29	1	0	14	0
03-443	F	1914		01	1935	316	1971	0	B6	0.0	29C	0	0	0	0
03-444	F	1907		01	1925	56	1977	11	C3	0.0	29C	3	0	50	0
03-445	F	1905	1974	01	1922	260	1966	1367	C2	0.0	29	380	0	5237	0
03-446	F	1903		01	1921	260	1977	65	B1	0.0	29C	20	0	300	0
03-447	F	1906		01	1924	4	1958	2	C6	0.0	29	1	0	7	0
03-448	F	1903	1963	01	1924	19	1958	25	C2	0.0	79	6	0	73	0
03-449	F	1905	1974	01	1922	1456	1964	1135	B1	0.0	29	308	0	4239	0
03-450	F	1910		01	1924	697	1979	8	C3	0.0	29C	2	0	34	0
03-451	F	1922		01	1940	524	1972	1	B6	0.0	29C	0	0	2	0
03-452	F	1909		16	1925	728	1977	13	B2	0.0	29C	4	0	51	0
03-453	F	1907		01	1924	8	1976	3	B2	0.0	29C	1	0	14	0
03-454	F	1914		06	1934	572	1958	48	C2	0.0	29	9	0	102	0
03-455	F	1906		01	1922	56	1975	491	B1	0.00054	P1	153	49	2287	738
03-456	F	1921	1965	01	1942	470	1958	33	C2	0.0	29	5	0	33	0
03-457	F	1915		01	1939	520	1972	1	B6	0.0	29C	0	0	2	0
03-458	F	1925		01	1946	1560	1976	33	B2	0.0	29C	4	0	26	0
03-459	F	1906		01	1924	43	1976	774	B1	0.0	29C	239	0	3495	0
03-460	F	1905		01	1923	19	1977	4	C6	0.0	29C	1	0	18	0
03-461	F	1906		01	1922	6	1958	6	C3	0.0	29	2	0	23	0
03-462	F	1906		01	1922	2912	1979	217	C2	0.0	29C	69	0	1019	0
03-463	F	1918	1966	01	1942	832	1958	33	C2	0.0	29	3	0	18	0
03-464	F	1907		01	1923	104	1974	0	C6	0.0	29C	0	0	2	0
03-465	F	1904		01	1925	8	1976	5	B2	0.0	29	2	0	22	0
03-466	F	1914		01	1924	10	1976	2	B3	0.0	29C	1	0	8	0
03-467	F	1911		01	1926	416	1976	8	B2	0.0	29C	2	0	30	0
03-468	F	1908		01	1926	121	1958	29	C2	0.0	29	7	0	97	0
03-469	F	1903	1965	01	1925	30	1958	10	C3	0.0	29	2	0	27	0
03-470	F	1926		01	1943	247	1971	1	B3	0.0	29C	1	0	8	0
03-471	F	1908		01	1926	91	1958	13	C3	0.0	29	3	0	44	0
03-472	F	1922		01	1941	247	1972	5	B3	0.0	29C	1	0	13	0
03-473	F	1904	1965	01	1922	156	1962	1170	C2	0.0	29	311	0	3743	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	YR	YR	YR	YR	FA226 METHOD + RA226 RATIO	FA226 METHOD + RA226 RATIO	FA226 METHOD + RA226 RATIO	FA226 METHOD + RA226 RATIO	FA226 METHOD + RA226 RATIO	FA226 METHOD + RA226 RATIO	FA226 METHOD + RA226 RATIO
03-474	F	1909	1960	01	1925	21	1958	19	C2	0.0	79	5	0	67	0
03-475	F	1903	1960	01	1921	05	1958	0	C6	0.0	79	0	0	0	0
03-476	F	1905	1970	01	1927	6	1958	0	C6	0.0	79	0	0	0	0
03-477	F	1911	1971	01	1925	11	1972	3	B3	0.0	79C	1	0	13	0
03-478	F	1907	1967	01	1924	8	1959	5	C6	0.0	79	1	0	18	0
03-479	F	1908	1968	01	1924	52	1978	28	C2	0.0012	79	0	1	127	11
03-480	F	1909	1977	01	1924	10	1975	2	B3	0.0	79	1	0	9	0
03-481	F	1922	1942	01	1942	481	1972	9	B2	0.0	79C	2	0	19	0
03-482	F	1927	1945	01	1945	83	1972	3	B6	0.0	79C	1	0	6	0
03-483	F	1901	1922	01	1922	177	1975	1	B6	0.0	79C	1	0	4	0
03-484	F	1908	1966	01	1919	156	1962	1622	C2	0.0	79	448	0	5837	0
03-485	F	1919	1977	01	1923	354	1958	0	C6	0.0	79	0	0	0	0
03-486	F	1909	1977	01	1925	156	1977	208	B1	0.0	79	64	0	928	0
03-487	F	1907	1964	01	1924	676	1958	367	C2	0.0	79	90	0	1055	0
03-488	F	1907	1975	01	1922	26	1959	170	C2	0.0	79	43	0	621	0
03-489	F	1911	1964	01	1926	73	1958	123	C2	0.0	79	29	0	326	0
03-490	M	1914	1977	07	1925	177	1973	5	B3	0.0	79C	1	0	14	0
03-491	F	1908	1977	01	1924	2	1979	19	C2	0.0	79C	6	0	98	0
03-492	F	1928	1946	01	1946	325	1973	5	B3	0.0	79C	1	0	9	0
03-493	F	1893	1923	01	1923	199	1975	8	B3	0.0	79C	2	0	26	0
03-494	F	1902	1964	01	1924	177	1959	4	C3	0.0	79	1	0	14	0
03-495	F	1910	1977	01	1923	7	1976	0	B6	0.0	79C	0	0	2	0
03-496	F	1907	1977	01	1923	6	1976	1	B6	0.0	79C	0	0	3	0
03-497	F	1903	1970	01	1923	260	1959	15	C2	0.0	79	4	0	52	0
03-498	F	1925	1923	67	1923	1040	1976	2	B3	0.0	79C	1	0	7	0
03-499	F	1905	1959	31	1924	56	1976	185	C2	0.00175	79	58	0	848	1019
03-500	F	1901	1959	01	1922	8	1959	0	C6	0.0	79	0	0	0	0
03-501	F	1912	1977	01	1928	8	1959	7	C3	0.0	79	2	0	25	0
03-502	F	1907	1964	01	1918	156	1959	170	C2	0.0	79	46	0	585	0
03-503	F	1904	1960	01	1922	112	1959	125	C2	0.0	79	32	0	362	0
03-504	F	1905	1977	01	1922	3	1978	11	C3	0.0	79C	3	0	52	0
03-505	F	1917	1976	01	1923	1302	1975	169	B2	0.0	79C	52	0	725	0
03-506	F	1917	1976	01	1923	1972	1975	9	B2	0.0	79C	2	0	14	0
03-507	F	1907	1962	01	1923	6	1959	17	C3	0.0	79	3	0	36	0
03-508	F	1905	1963	01	1923	8	1959	13	C3	0.0	79	3	0	31	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SPX	BORN	DIED	TYPE	EXP	YR	YR	YR	RA226 MKS	RA226 MKS	RA226 MKS	RA226 MKS	RA226 MKS	RA226 MKS	CUM RA226 RADS
03-509	F	1907		01	1924	2548	1973	28	B1	0.0	Z9C	131	0	120	0
03-510	F	1907	1977	01	1923	2028	1952	72	C2	0.0	Z9C	1	0	7	0
03-511	F	1910	1979	01	1946	673	1956	15	C3	0.0	Z9C	3	0	39	0
03-512	F	1906		01	1925	26	1959	11	C3	0.0	Z9C	22	0	317	0
03-513	F	1908		01	1925	34	1974	73	B1	0.0	Z9C	0	0	0	0
03-514	F	1903		01	1925	214	1959	26	C2	0.0	Z9C	5	0	93	0
03-515	F	1908		01	1925	156	1959	11	C3	0.0	Z9C	3	0	39	0
03-516	F	1911		01	1925	624	1974	7	B2	0.0	Z9C	2	0	33	0
03-517	F	1922		01	1943	260	1972	1	B6	0.0	Z9C	0	0	1	0
03-518	F	1921		01	1944	464	1972	8	B3	0.0	Z9C	2	0	18	0
03-519	F	1903		01	1924	8	1950	94	C2	0.0	Z9C	25	0	363	0
03-520	F	1907		01	1925	780	1974	112	C2	0.0	Z9C	33	0	481	0
03-521	F	1907	1961	01	1925	39	1959	10	C3	0.0	Z9C	2	0	27	0
03-522	F	1898		01	1921	5	1978	88	C2	0.0	Z9C	29	0	433	0
03-523	F	1900		01	1923	30	1977	9	B2	0.0	Z9C	3	0	42	0
03-524	F	1903		01	1925	260	1972	48	B2	0.0	Z9C	14	0	201	0
03-525	F	1911	1976	01	1931	2132	1959	19	C2	0.0	Z9C	3	0	25	0
03-526	F	1896		01	1925	52	1959	0	C6	0.0	Z9C	0	0	0	0
03-527	F	1925		01	1925	130	1959	5	C3	0.0	Z9C	1	0	18	0
03-528	F	1904		01	1922	524	1959	1630	C2	0.0	Z9C	412	0	6046	0
03-529	F	1902		01	1921	104	1977	74	C2	0.0	Z9C	24	0	257	0
03-530	F	1907	1965	01	1923	91	1963	474	C2	0.0	Z9C	127	0	1541	0
03-531	F	1906		01	1925	403	1959	41	C2	0.0	Z9C	10	0	146	0
03-532	F	1910		01	1926	190	1977	43	C2	0.0	Z9C	13	0	180	0
03-533	F	1909		01	1925	260	1970	12	C3	0.0	Z9C	4	0	54	0
03-534	F	1910		01	1925	104	1976	3	B3	0.0	Z9C	1	0	15	0
03-535	F	1907		01	1922	21	1954	127	C2	0.0	Z9C	63	0	944	0
03-536	F	1910		01	1925	7	1959	35	C2	0.0	Z9C	9	0	126	0
03-537	F	1903		01	1916	52	1977	1	C6	0.0	Z9C	0	0	6	0
03-538	F	1903	1974	01	1927	13	1959	61	C2	0.0	Z9C	15	0	200	0
03-539	F	1900		01	1922	20	1974	5	C3	0.0	Z9C	2	0	23	0
03-540	F	1904		01	1923	264	1973	1605	C1	0.0	Z9C	481	0	7014	0
03-541	F	1913		01	1935	178	1978	0	C6	0.0	Z9C	0	0	0	0
03-542	F	1904		01	1922	13	1973	23	C2	0.0	Z9C	7	0	109	0
03-543	F	1918		01	1947	100	1972	1	B6	0.0	Z9C	0	0	3	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP	YEAR	YR	YEAR	FA226	FA226	FA226	FA226	INPUT	INPUT	CUM	CUM
				TYPE	EXP	WKS	OF	NCI	METHOD	TO FA226	+ ERR	RA226	RA226	RA226	RA226
03-544	F	1916	1975	01	1922	26	1959	5	C3	0.0	79	1	0	19	0
03-545	F	1894		01	1922	298	1959	3	CF	0.0	79	0	0	0	0
03-546	F	1903		01	1925	52	1959	25	C2	0.0	79	23	0	338	0
03-547	F	1907	1962	01	1924	108	1959	14	C2	0.0	79	5	1	55	19
03-548	F	1906		01	1922	17	1971	80	B1	0.0	29C	24	0	361	0
03-549	F	1910		01	1925	936	1977	43	C2	0.0	29C	13	0	193	0
03-550	F	1900		01	1917	104	1977	9	B3	0.0	79C	3	0	45	0
03-551	F	1903		01	1922	338	1973	1077	C2	0.0	29C	324	0	4760	0
03-552	F	1904		01	1924	108	1978	114	C2	0.0	79C	36	0	520	0
03-553	F	1904		01	1924	13	1979	6	CF	0.0	29C	2	0	30	0
03-554	F	1899	1977	01	1924	433	1961	2000	B4	0.0	29	513	0	7258	0
03-555	F	1913	1978	01	1930	260	1972	2	B6	0.0	29C	1	0	8	0
03-556	F	1911		01	1928	100	1976	2	B3	0.0	29C	1	0	9	0
03-557	F	1910	1978	01	1925	3	1959	0	CF	0.0	29	0	0	0	0
03-558	F	1904	1971	01	1923	13	1959	115	C2	0.0	29C	29	50	395	755
03-559	F	1907	1975	01	1922	21	1959	17	C2	0.0	29	4	0	63	0
03-561	F	1909		01	1924	416	1959	67	C2	0.0	29	17	0	242	0
03-562	F	1908		01	1927	520	1972	4	B3	0.0	29C	1	0	13	0
03-563	F	1909		01	1924	10	1975	2	B3	0.0	29C	1	0	11	0
03-564	F	1906		01	1923	3	1976	3	B2	0.0	29C	1	0	16	0
03-565	F	1910	1979	01	1930	676	1978	7	C3	0.0	29C	2	0	25	0
03-566	F	1910		01	1930	624	1978	2	CF	0.0	29C	1	0	7	0
03-567	F	1900		01	1922	104	1972	26	B2	0.0	29C	3	0	115	0
03-568	F	1905	1977	01	1922	260	1969	120	C2	0.0	29	30	0	434	0
03-569	F	1901	1970	01	1922	112	1959	144	C2	0.0	29	36	0	495	0
03-570	F	1908		01	1925	43	1976	9	B2	0.0	29C	3	0	37	0
03-571	F	1909		01	1925	52	1979	110	C2	0.0	29C	224	0	3224	0
03-572	F	1905		01	1924	56	1971	62	C2	0.0	29C	19	0	284	0
03-573	F	1900	1970	01	1925	52	1971	10	C6	0.0	29C	5	0	69	0
03-574	F	1901		01	1920	624	1976	1	B6	0.0	29C	0	0	3	0
03-575	F	1913		01	1931	52	1973	0	B6	0.0	29C	0	0	0	0
03-576	F	1909		01	1925	156	1976	4	B2	0.0	29C	1	0	17	0
03-577	F	1901	1961	01	1921	104	1959	81	C2	0.0	29	21	0	247	0
03-578	F	1909		01	1924	30	1976	8	B2	0.0	29	2	0	30	0
03-579	F	1905		01	1922	11	1959	30	C2	0.0	29	8	0	117	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADTUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP	YEAF	EXP	YEAF	RA226	RA226	PA228	RA228	INPUT	INPUT	CUM	CUM
				TYPE	EXP	WKS	MEAS	NCI	METHOD	RATIO	ERR	RA226	RA228	RA226	RA228
												UCI	UCI	FA226	FA228
03-580	F	1904		01	1923	4	1959	2	C6	0.0	29	1	0	8	0
03-581	F	1904		01	1922	10	1959	13	C3	0.0	29	3	0	51	0
03-583	M	1803	1962	07	1930	40	1959	50	C2	0.0	29	11	0	84	0
03-584	F	1905	1959	01	1923	40	1959	6000	A4	0.0	20	1540	0	17131	0
03-585	F	1804		01	1919	260	1968	74	C2	0.0	29	21	0	328	0
03-586	F	1908	1968	01	1926	82	1967	902	C2	0.0	29	245	0	2972	0
03-587	F	1906		01	1925	34	1959	13	C3	0.0	29	3	0	47	0
03-588	F	1901	1967	01	1922	229	1962	316	C2	0.0	29	83	0	1041	0
03-589	F	1905	1960	01	1924	21	1959	77	C2	0.0	29	19	0	249	0
03-590	F	1903		01	1922	26	1965	194	C2	0.0	29	29	0	437	0
03-591	F	1907		17	1926	2340	1976	5	B2	0.0	29C	1	0	10	0
03-592	F	1905		01	1922	78	1979	70	C3	0.0	29	23	0	337	0
03-593	F	1905		01	1922	10	1977	10	C3	0.0	29C	3	0	47	0
03-594	F	1905	1969	01	1922	52	1959	41	C2	0.0	29	11	0	137	0
03-595	F	1902		01	1923	52	1975	1	B6	0.0	29C	0	0	5	0
03-596	F	1904		01	1922	8	1979	10	C3	0.0	29C	3	0	50	0
03-597	F	1903		16	1925	1300	1972	74	B1	0.0	29C	18	0	223	0
03-598	M	1890		07	1933	4	1971	1	B6	0.0	29C	0	0	2	0
03-599	F	1906	1975	01	1922	26	1959	9	C3	0.0	29	2	0	33	0
03-600	F	1902		07	1926	938	1974	0	B6	0.0	29C	0	0	0	0
03-601	F	1893	1969	01	1925	260	1960	6	C3	0.0	29	2	0	19	0
03-602	F	1899	1979	01	1925	104	1960	3	C6	0.0	29	1	0	11	0
03-603	F	1886	1979	01	1924	520	1960	0	C6	0.0	29	0	0	0	0
03-604	F	1899		01	1916	624	1976	2	C3	0.0	29C	1	0	10	0
03-605	F	1900		01	1921	364	1972	1	B4	0.0	29C	0	0	3	0
03-606	F	1903		01	1924	6	1971	2	B6	0.0	29C	1	0	8	0
03-607	F	1906		01	1922	26	1979	81	C2	0.0	29C	26	0	395	0
03-608	F	1895	1976	01	1917	104	1960	19	C2	0.0	29	5	0	80	0
03-609	F	1896	1974	01	1923	4	1960	0	C6	0.0	29	0	0	0	0
03-610	F	1917		01	1935	104	1973	1	B6	0.0	29C	0	0	4	0
03-611	F	1893	1969	01	1916	208	1960	3	C6	0.0	29	1	0	12	0
03-612	F	1892	1968	01	1918	234	1960	500	C2	0.0	29	135	0	1806	0
03-613	F	1900		01	1920	37	1972	0	B6	0.0	29C	0	0	410	0
03-614	F	1901		01	1924	54	1975	94	C2	0.0	29	0	0	64	0
03-615	F	1905		01	1923	107	1975	14	B1	0.0	29	4	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR FALLOUT PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SPY	BOBY	DIED	EXF	VEZF	EXP	YEAR	FAZ26 NCL	FAZ26 MTHD	FAZ26 RATIO	FAZ26 MTHD	FAZ26 RATIO	FAZ26 RATIO	CUM FADS	CUM FADS
03-617	P	1902	1951	01	1921	112	1963	7000	FA226 MTHD	0.0	FA226 MTHD	1500	0	14500	FA226
03-618	P	1903	1963	01	1920	43	1963	10	FA226 MTHD	0.0	FA226 MTHD	3	0	36	0
03-619	P	1903	1963	01	1920	43	1963	1570	FA226 MTHD	0.0	FA226 MTHD	43	75	5041	1143
03-620	P	1923	1963	01	1942	204	1971	5	FA226 MTHD	0.0	FA226 MTHD	1	0	12	0
03-621	P	1916	1963	01	1944	204	1971	4	FA226 MTHD	0.0	FA226 MTHD	1	0	9	0
03-622	P	1910	1963	01	1926	104	1960	1	FA226 MTHD	0.0	FA226 MTHD	0	0	0	0
03-623	P	1902	1971	01	1924	40	1963	4	FA226 MTHD	0.0	FA226 MTHD	1	0	15	0
03-624	P	1905	1950	01	1923	156	1950	1000	FA226 MTHD	0.0	FA226 MTHD	251	0	2716	0
03-625	P	1901	1963	01	1923	13	1976	1	FA226 MTHD	0.0	FA226 MTHD	0	0	2	0
03-626	P	1904	1963	01	1924	204	1960	200	FA226 MTHD	0.0	FA226 MTHD	11	0	733	0
03-627	P	1905	1966	01	1924	408	1960	50	FA226 MTHD	0.0	FA226 MTHD	13	0	152	0
03-628	P	1905	1974	01	1921	34	1962	0	FA226 MTHD	0.0	FA226 MTHD	0	0	0	0
03-629	P	1903	1964	01	1922	40	1960	0	FA226 MTHD	0.0	FA226 MTHD	0	0	0	0
03-630	P	1903	1964	01	1924	17	1974	19	FA226 MTHD	0.0	FA226 MTHD	5	0	82	0
03-632	P	1905	1975	01	1922	40	1960	0	FA226 MTHD	0.0	FA226 MTHD	0	0	0	0
03-633	P	1902	1963	01	1922	760	1960	20	FA226 MTHD	0.0	FA226 MTHD	0	0	75	0
03-634	P	1909	1961	01	1924	40	1960	3	FA226 MTHD	0.0	FA226 MTHD	1	0	9	0
03-635	P	1907	1963	01	1925	40	1960	47	FA226 MTHD	0.0	FA226 MTHD	12	0	172	0
03-636	P	1904	1963	01	1924	142	1976	5	FA226 MTHD	0.0	FA226 MTHD	2	0	24	0
03-637	P	1906	1963	01	1924	6	1970	33	FA226 MTHD	0.0	FA226 MTHD	13	0	184	0
03-638	P	1902	1970	01	1924	40	1960	7	FA226 MTHD	0.0	FA226 MTHD	2	0	24	0
03-639	P	1912	1963	01	1925	156	1960	57	FA226 MTHD	0.0	FA226 MTHD	17	0	242	0
03-640	P	1902	1963	01	1924	60	1960	5	FA226 MTHD	0.0	FA226 MTHD	1	0	13	0
03-641	P	1904	1963	01	1922	26	1970	3	FA226 MTHD	0.0	FA226 MTHD	10	0	43	0
03-642	P	1905	1974	01	1922	52	1974	11	FA226 MTHD	0.0	FA226 MTHD	0	0	146	0
03-643	P	1903	1970	01	1924	156	1974	10	FA226 MTHD	0.0	FA226 MTHD	3	0	40	0
03-645	P	1906	1963	01	1924	112	1960	56	FA226 MTHD	0.0	FA226 MTHD	14	0	202	0
03-646	P	1908	1963	01	1926	40	1960	0	FA226 MTHD	0.0	FA226 MTHD	0	0	0	0
03-647	P	1901	1963	01	1925	5	1960	35	FA226 MTHD	0.0	FA226 MTHD	9	0	128	0
03-648	P	1903	1966	01	1922	155	1960	5000	FA226 MTHD	0.00430	FA226 MTHD	216	-71	12670	4043
03-649	P	1906	1954	01	1924	156	1951	1300	FA226 MTHD	0.0	FA226 MTHD	290	0	2725	0
03-650	P	1905	1924	01	1924	147	1974	24810	FA226 MTHD	0.00324	FA226 MTHD	2127	332	6560	2306
03-671	P	1906	1955	01	1923	4	1952	3820	FA226 MTHD	0.00500	FA226 MTHD	130	130	5940	2525
03-672	P	1900	1963	01	1924	40	1960	3	FA226 MTHD	0.0	FA226 MTHD	1	0	11	0
03-73	P	1903	1903	01	1924	4	1903	35	FA226 MTHD	0.0	FA226 MTHD	5	0	125	0

TABLE 1 (CONT.) EXP. SURV. DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
	SPX	DOB	DIAG	TYPE	YR	AGE	SEX	FA226	FA226	FA226	FA226	FA226	FA226	CUM	FA226
CASE								NCI	FA226	FA226	FA226	FA226	FA226	FA226	FA226
03-736	P	1928		1	1928	22	M	1976	1	B6	0.0	0	0	2	0
04-741	F	1904		1	1904	66	F	1975	4	B3	0.2	1	0	16	0
03-739	F	1910		1	1927	40	F	1977	5	P2	0.0	1	0	21	0
01-752	F	1924		1	1922	15	F	1977	10	P2	0.0	3	0	49	0
01-753	F	1926		1	1922	40	F	1977	12	B1	0.0	4	0	58	0
03-757	F	1922		1	1923	51	F	1978	10	B6	0.0	1	0	48	0
03-761	F	1931		1	1927	144	F	1977	26	B2	0.0	7	0	91	0
03-763	F	1931		1	1931	52	F	1974	0	C6	0.0	0	0	0	0
03-764	F	1908		1	1926	364	F	1976	2	P3	0.0	1	0	8	0
03-771	F	1920		1	1923	13	F	1978	112	C2	0.0	36	0	514	0
03-774	P	1909		1	1924	5	P	1977	1	P6	0.0	0	0	3	0
03-775	F	1922		1	1942	52	F	1974	4	B3	0.0	1	0	10	0
03-778	P	1904		1	1923	104	P	1973	54	B1	0.0	16	0	240	0
03-779	P	1905	1942	1	1922	40	P	1979*	1935	A1	0.0	147	0	2651	0
03-782	F	1928		1	1923	5	F	1975	2	B3	0.0	1	0	11	0
03-734	F	1925		1	1923	178	F	1954	750	C4	0.0	173	0	2530	0
03-738	F	1925		1	1925	194	F	1976	1	B6	0.0	0	0	3	0
03-785	F	1927	1944	1	1926	73	F	1944	8	G6	0.0	1	0	19	0
03-736	F	1927		1	1925	2	F	1972	0	B6	0.0	0	0	1	0
03-738	F	1915		1	1935	281	F	1978	2	C6	0.0	0	0	5	0
03-831	P	1926		1	1924	13	P	1975	1	B3	0.0	1	0	10	0
03-837	P	1923		1	1954	780	P	1973	0	B6	0.0	0	0	0	0
03-810	F	1919		1	1934	117	F	1972	2	P1	0.0	0	0	6	0
03-817	F	1927		1	1926	13	F	1972	0	C6	0.0	0	0	0	0
03-818	F	1922		1	1927	62	F	1975	4	B3	0.0	1	0	17	0
03-825	F	1926		1	1922	4	F	1976	1	B3	0.0	0	0	5	0
03-828	F	1915		1	1955	935	F	1972	0	B6	0.0	0	0	0	0
03-834	F	1927		1	1925	40	F	1976	0	B3	0.0	0	0	6	0
03-836	F	1928		1	1924	20	F	1977	0	C6	0.0	0	0	0	0
03-838	F	1928		1	1927	130	F	1975	2	B3	0.0	1	0	5	0
03-842	F	1910		1	1926	416	F	1975	1	B2	0.0	1	0	13	0
03-845	F	1908		1	1927	124	F	1976	7	C6	0.0	0	0	31	0
03-850	F	1923		1	1942	73	F	1976	41	B1	0.0	12	7	219	102
05-011	F	1900		1	1919	50	F	1978	1	B6	0.0	0	0	5	0
05-012	F	1903	1972	1	1917	174	F	1971	1	B6	0.0	0	0	5	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BOLV	DIED	EXP	YEAR	FX2	OF	RA226	RA226	RA226	RA226	RA226	RA226	CUM	RA226
			DATE	TYPE	FIRST	PKS	YEAS	NCI	METHOD	RATIO	METHOD	UCI	UCI	RADS	RADS
					EXP	PKS	YEAS								
05-003	F	1900	1950	01	1917	8	1958	0	66	0.0	71	0	0	0	0
05-004	F	1904		01	1920	104	1959	12	36	0.01630	27	3	5	48	77
05-005	F	1901		01	1913	13	1960	0	36	0.0	74	0	0	0	0
05-006	F	1906		01	1920	35	1967	23	B2	0.00500	778	7	11	102	164
05-008	F	1904	1954	07	1916	104	1963	4	C1	0.0	795	1	0	11	0
05-010	F	1901	1974	01	1921	34	1961	4	C1	0.01200	77C	1	2	15	24
05-011	F	1902		01	1917	50	1959	12	G6	0.0	29	3	0	52	0
05-012	F	1907	1955	01	1917	52	1970	16	A1	0.0	29A	4	0	54	0
05-014	F	1900		01	1916	208	1978	116	B1	0.00074	B6	39	42	610	628
05-015	F	1901		01	1916	67	1978	4	C1	0.0	298	1	0	19	0
05-016	F	1901	1965	06	1916	100	1958	15	G4	0.0	29	4	0	40	0
05-017	F	1894		01	1919	40	1969	5	G6	0.03520	27	2	3	23	46
05-018	F	1836	1979	06	1918	156	1971	4	B3	0.00180	278	1	1	14	12
05-019	F	1985	1988	01	1921	2	1960	0	G6	0.01400	27	0	0	0	0
05-020	F	1898		01	1917	52	1959	3	G5	0.0	29	1	0	13	0
05-022	F	1900	1969	07	1916	32	1964	4	C1	0.0	29C	1	0	17	0
05-023	F	1895	1960	01	1918	104	1960	38	C2	0.00320	77C	10	5	126	73
05-024	F	1890	1965	06	1916	208	1961	4	C1	0.01200	27C	1	2	11	27
05-025	F	1893		01	1917	78	1971	86	B1	0.00020	77B	27	4	426	53
05-037	F	1898	1977	01	1916	260	1971	2	B6	0.0	29B	1	0	10	0
05-038	F	1901		07	1916	156	1972	99	G4	0.0	29	32	0	498	0
05-039	F	1900		07	1917	156	1977	20	A1	0.00002	27B	7	5	103	75
05-040	F	1859		01	1917	54	1971	10	B4	0.0	29B	3	0	50	0
05-042	F	1918		01	1940	120	1972	1	B6	0.0	29B	0	0	3	0
05-043	F	1958	1965	06	1915	208	1965	0	F6	0.00430	27E	0	0	0	0
05-044	F	1995	1975	06	1915	468	1971	2	R6	0.0	79B	1	0	7	0
05-045	F	1909	1960	01	1917	60	1965	5	F4	0.0	29F	1	0	17	0
05-049	F	1905		01	1923	13	1965	6	C3	0.0	29C	2	0	25	0
05-072	F	1893	1950	07	1919	13	1976	0	A6	0.00100	27	0	0	0	0
05-088	F	1886		01	1917	4	1959	4	G6	0.0	79	1	0	18	0
05-089	F	1900		01	1915	74	1971	13	R2	0.0	29B	0	0	66	0
05-092	F	1911		01	1916	104	1959	0	G6	0.0	29	2	0	26	0
05-093	F	1907	1974	01	1915	30	1961	6	C6	0.0	29C	2	0	26	0
05-094	F	1927		01	1940	30	1973	6	B3	0.0	29B	1	0	14	0
05-096	F	1901	1971	01	1918	20	1962	234	C2	0.00350	27C	56	7	949	102

TABLE 1 (CONT.) ENCLOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	DOB	DEPT	TYPE	YR	EXP	OF	RA226	RA226	RA226	RA226	INPUT	INPUT	CUM	CUM
								NCI	RA226	FA226	RA226	FA226	RA226	RA226	RA226
									FA226	FA226	FA226	FA226	FA226	FA226	FA226
05-097	M	1892	1976	01	1914	28	1961	4	CL	0.00050	27	1	0	12	1
05-100	F	1907		01	1916	15	1964	4	CL	0.00520	27	1	0	18	30
05-111	F	1902		01	1924	0	1964	4	CL	0.00650	27	1	0	18	18
05-112	F	1900		01	1917	14	1960	0	CL	0.00350	27	1	0	26	13
05-113	F	1906		01	1923	4	1959	1	CL	0.01600	27	0	0	4	5
05-114	F	1900		01	1918	13	1964	4	CL	0.00040	27	1	0	18	2
05-105	M	1903	1950	07	1918	30	1950	0	CL	0.00070	27	0	0	0	0
05-111	M	1905	1977	07	1920	12	1970	5	CL	0.00660	27	1	0	15	31
05-116	F	1908	1950	01	1917	5	1972	19	CL	0.0	25A	1	0	04	0
05-117	F	1907	1969	06	1915	208	1964	4	CL	0.0	29C	1	0	12	0
05-118	F	1901		01	1917	65	1977	2	CL	0.0	29B	1	0	10	0
05-119	F	1925		01	1924	212	1977	10	CL	0.00175	27	3	0	45	46
05-120	F	1890		07	1919	6	1959	5	CL	0.00770	27	1	0	21	20
05-121	F	1906		01	1921	26	1970	9	CL	0.00350	27B	5	0	41	60
05-122	M	1879	1962	07	1922	208	1959	11	CL	0.01600	27	3	0	23	33
05-123	F	1897	1972	01	1918	1	1960	4	CL	0.00060	27	1	0	16	2
05-125	F	1902	1976	07	1916	104	1959	26	CL	0.0	29	7	0	111	0
05-126	M	1909	1970	01	1921	52	1970	0	CL	0.0	29B	0	0	0	0
05-127	M	1893		06	1918	39	1907	20	CL	0.0	255	5	0	53	0
05-129	F	1900	1960	07	1917	104	1960	4	CL	0.0	29C	1	0	16	0
05-130	F	1920		01	1940	78	1972	0	CL	0.0	25B	0	0	0	0
05-132	F	1899		07	1918	52	1960	0	CL	0.00120	27	0	0	0	0
05-133	M	1903	1967	07	1918	13	1960	0	CL	0.00070	27	0	0	0	0
05-134	F	1900		01	1917	1	1960	0	CL	0.0	29	3	0	40	0
05-135	F	1919		01	1941	115	1975	0	CL	0.0	29B	0	0	0	0
05-136	M	1906	1966	06	1917	78	1960	54	CL	0.0	24	26	0	249	0
05-138	F	1917		01	1941	104	1968	5	CL	0.0	29B	1	0	12	0
05-139	F	1891	1966	01	1918	70	1962	4	CL	0.00540	27	1	0	15	16
05-140	F	1907	1960	01	1918	40	1975	670	CL	0.00182	27	184	197	2227	2957
05-142	F	1904		01	1919	30	1960	11	CL	0.00680	27	3	0	47	43
05-143	F	1890	1960	07	1918	40	1961	4	CL	0.00050	27	1	0	14	2
05-145	M	1903	1961	07	1916	57	1961	4	CL	0.00150	27	1	0	9	7
05-146	M	1897		05	1920	246	1968	0	CL	0.00400	27	1	0	6	0
05-150	F	1909	1960	07	1917	0	1960	45	CL	0.0	29	13	0	175	0
05-151	F	1907		01	1924	93	1963	7	CL	0.00360	27	2	0	27	27

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIATION PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP TYPE	VFAP	FIFST DUR	VFAP	EXP	FA226 METHOD	FA226 IC FA226b	FA226 METHOD + FA226	NCI	INPUT	INPUT	CUM
05-154	F	1902	1978	01	1916	11	1970	0	56	0.0	29	0	0	0	0
05-155	F	1898	1965	07	1916	2	1963	4	0	0.0	79C	1	0	16	0
05-160	F	1917		01	1942	15	1965	0	36	0.0	79	0	0	0	0
05-161	F	1901		06	1918	2	1971	0	36	0.00316	773	0	0	0	0
05-162	F	1914		07	1942	40	1960	29	36	0.0	29	5	0	59	0
05-163	M	1912	1970	07	1941	104	1960	35	66	0.0	29	6	0	42	0
05-165	F	1899	1964	01	1919	13	1972	1	86	0.0	29A	0	0	3	0
05-172	F	1907	1960	01	1934	99	1960	24	34	0.0	29	4	0	26	0
05-174	F	1902		01	1919	130	1977	0	0	0.00126	27	0	0	0	0
05-179	F	1921		01	1940	182	1974	0	86	0.0	25B	0	0	0	0
05-181	F	1901		01	1918	4	1970	0	86	0.00018	27B	0	0	0	0
05-184	M	1901	1974	01	1922	156	1964	5	0	0.0	29C	1	0	14	0
05-185	F	1912		01	1941	238	1972	2	0	0.0	29B	0	0	5	0
05-186	F	1922		01	1941	156	1972	1	86	0.0	29B	0	0	3	0
05-188	M	1889	1964	07	1917	104	1961	4	0	0.0	29C	1	0	10	0
05-189	M	1890	1972	07	1921	104	1964	4	0	0.00850	27C	1	2	11	17
05-194	F	1902	1965	01	1926	5	1975	31	0	0.0	29	8	0	97	0
05-197	M	1898		07	1919	7	1973	0	86	0.00140	27F	0	0	0	0
05-199	F	1901		16	1917	2	1967	0	86	0.0	29B	0	0	0	0
05-201	F	1919		01	1941	221	1976	6	83	0.0	29B	1	0	16	0
05-203	F	1899		01	1919	52	1960	0	86	0.00680	27	0	0	0	0
05-204	M	1890	1961	07	1918	78	1960	0	36	0.00320	27	0	0	0	0
05-205	F	1907		01	1924	208	1961	1	0	0.0	29C	1	0	15	0
05-206	F	1894		01	1922	52	1971	2	86	0.00360	27B	1	1	9	12
05-207	M	1893		06	1917	40	1962	6	36	0.0	29	2	0	20	0
05-210	F	1899	1971	01	1916	158	1977	106	81	0.0	29A	334	0	4814	0
05-212	F	1903		07	1918	78	1965	4	0	0.00030	27C	1	0	18	2
05-215	F	1886	1968	01	1920	78	1965	1410	81	0.00198	A3	417	291	5536	4376
05-237	F	1896	1960	06	1920	164	1961	4	0	0.0	29C	1	0	10	0
05-246	F	1884	1969	06	1911	728	1962	4	0	0.0	29C	1	0	16	0
05-251	F	1896		01	1917	34	1965	13	84	0.0	29	4	0	61	0
05-252	F	1890	1974	01	1917	52	1964	4	0	0.0	29C	1	0	18	0
05-255	M	1886	1965	07	1920	124	1964	5	0	0.00850	29C	1	2	13	24
05-257	F	1895	1975	01	1932	1248	1972	3	86	0.0	29	1	0	7	0
05-258	F	1901		01	1917	1	1970	0	86	0.0	29	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	TYPE	EXP	WKS	CF	FA226	METHOD	FA226	METHOD	FA226	UCI	CUM	CUM
05-259	F	1900		07	1917	52	1960	6	G6	0.0	29	27	0	0	0
05-260	F	1898		07	1917	32	1960	0	G6	0.0	70	0	0	0	0
05-261	F	1932	1977	01	1943	154	1960	4	CL	0.0	29C	1	0	7	0
05-262	F	1917		01	1942	26	1972	3	D3	0.0	79C	1	0	7	0
05-263	M	1883	1967	07	1918	104	1962	4	CL	0.00900	77C	1	1	11	16
05-264	M	1903		07	1917	5	1961	4	CL	0.0	29C	1	0	13	0
05-265	M	1924	1963	07	1916	104	1962	4	CL	0.0	29C	1	0	11	0
05-266	M	1881	1979	07	1918	130	1964	4	CL	0.00200	27C	1	1	11	6
05-269	F	1993		01	1918	35	1960	4	CL	0.00060	27C	1	0	17	2
05-269	M	1887	1971	07	1918	52	1964	4	CL	0.00040	27C	1	0	12	1
05-270	M	1901		07	1916	52	1961	8	C3	0.0	29C	2	0	26	0
05-272	M	1895		06	1918	65	1972	0	B6	0.00714	77B	0	0	0	0
05-273	F	1849	1968	01	1918	194	1960	4	CL	0.01400	27C	1	2	15	34
05-274	F	1903		07	1920	4	1970	0	G6	0.0	29	0	0	0	0
05-276	F	1906		01	1921	75	1961	4	CL	0.01200	27C	1	2	16	23
05-277	M	1994	1973	06	1918	104	1960	4	CL	0.00320	27C	1	1	11	6
05-278	F	1893	1965	01	1917	52	1964	37	C2	0.0	29F	11	0	145	0
05-279	F	1996	1979	01	1917	1820	1969	0	G6	0.0	29	0	0	0	0
05-281	F	1998	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	29C	2	0	37	0
05-284	F	1899	1973	01	1919	156	1969	219	B1	0.00090	77B	65	15	930	284
05-286	M	1921	1963	06	1916	104	1965	1	F4	0.0	29F	0	0	1	0
05-287	M	1889	1970	07	1917	300	1965	4	C1	0.00420	27C	1	1	11	11
05-288	F	1997		01	1918	10	1960	4	CL	0.00060	27C	1	0	17	2
05-290	F	1898	1967	01	1918	52	1960	8	C3	0.00060	27C	2	0	30	3
05-291	F	1902	1974	01	1920	8	1968	4	G6	0.00540	77	1	2	17	33
05-292	M	1904	1974	07	1918	47	1965	4	CL	0.00133	27C	1	0	13	1
05-303	F	1894		01	1917	2184	1977	1	CL	0.0	29	0	0	7	0
05-304	F	1907		01	1921	24	1962	4	CL	0.01100	27C	1	2	17	26
05-306	F	1903		01	1921	150	1976	3	F3	0.00195	27b	1	1	14	18
05-307	F	1920		01	1944	74	1972	0	P6	0.0	29F	0	0	0	0
05-308	M	1893	1964	07	1916	208	1962	4	CL	0.00130	27C	1	0	11	3
05-310	F	1894	1965	01	1916	78	1964	5	C6	0.0	29C	1	0	20	0
05-311	M	1887	1961	06	1920	156	1960	4	CL	0.01400	27C	1	2	9	17
05-312	M	1886	1961	01	1919	34	1961	2	F4	0.00610	27F	1	1	5	6

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP TYPE	YEAR	EXP DUE	OF	FA226 METHOD	FA226 METHOD	FA226 METHOD	FA226 METHOD	INPUT	INPUT	CUM	CUM
								NCI	FA226 METHOD	FA226 METHOD	FA226 METHOD	FA226 METHOD	FA226 METHOD	FA226 METHOD	FA226 METHOD
05-318	M	1901	1961	07	1918	40	1965	4	F4	0.00030	27F	1	0	10	1
05-321	F	1899		01	1916	20	1966	16	G6	0.00330	27	5	5	75	80
05-322	M	1900	1976	07	1917	312	1973	4	B3	0.0	27B	1	0	13	0
05-323	F	1899	1961	01	1915	26	1961	2	A5	0.0	29	1	0	7	0
05-349	F	1884	1956	01	1919	40	1975*	7	A2	0.00075	27	2	2	22	31
05-351	F	1891		01	1917	30	1968	23	G6	0.0	29	7	0	112	0
05-352	M	1903	1963	07	1917	40	1964	1	P6	0.0	29F	0	0	3	0
05-353	M	1900		07	1915	13	1978	0	C6	0.0	29B	0	0	0	0
05-357	F	1890	1978	07	1917	104	1972	3	G6	0.0	25	1	0	15	0
05-360	M	1892	1968	01	1914	40	1963	4	CI	0.0	29C	1	0	12	0
05-363	F	1899		07	1917	0	1964	4	CI	0.0	29C	1	0	19	0
05-368	F	1901		07	1917	104	1977	0	B6	0.0	29C	0	0	0	0
05-369	F	1901		07	1916	26	1978	1	R6	0.00077	27B	0	0	5	5
05-370	F	1895		01	1920	26	1965	4	CI	0.00760	27C	1	2	18	30
05-372	F	1888	1970	01	1916	104	1968	14	G4	0.0	29	4	0	62	0
05-374	F	1905		01	1923	8	1964	4	CI	0.00850	27C	1	1	16	20
05-377	F	1895	1974	01	1916	15	1969	0	G6	0.0	29	0	0	0	0
05-380	F	1904	1970	07	1925	104	1962	4	CI	0.01100	27C	1	1	13	13
05-383	F	1901		06	1917	165	1973	73	R1	0.00360	27B	23	10	362	156
05-387	M	1902		06	1918	9	1975	0	R6	0.00010	27B	0	0	0	0
05-395	F	1911		01	1928	728	1977	0	CF	0.0	25	0	0	0	0
05-397	F	1900	1976	07	1919	13	1962	4	CI	0.0	29C	1	0	17	0
05-399	M	1892		07	1916	104	1961	4	CI	0.0	29C	1	0	13	0
05-401	M	1893		76	1917	169	1971	5	A3	0.00170	27B	2	2	17	16
05-407	F	1898		01	1916	9	1978	0	H6	0.0	29B	0	0	0	0
05-409	F	1900		07	1916	61	1974	0	R6	0.00011	27B	0	0	0	0
05-410	F	1899		01	1916	26	1971	2	R6	0.0	29B	1	0	10	0
05-413	F	1900	1971	01	1916	39	1960	18	R2	0.0	29B	6	0	92	0
05-420	F	1889	1935	01	1917	104	1970	50	A1	0.0	29A	0	0	60	0
05-437	F	1888		07	1923	26	1971	3	B3	0.00350	27B	1	1	13	16
05-438	F	1907		01	1926	13	1961	4	CI	0.0	29C	1	0	14	0
05-439	F	1899	1970	01	1916	104	1967	20	F6	0.0	29	61	0	872	0
05-440	F	1896	1975	01	1922	1	1971	0	B6	0.00360	27B	0	0	0	0
05-442	F	1888		07	1917	6	1962	8	36	0.0	23	2	0	37	0
05-443	F	1922		07	1941	52	1972	3	B6	0.0	29B	1	0	8	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	DOBN	DIED	EXP	YEAR	EXP	YEAR	RA226	RA226	RA226	RA226	RA226	RA226	CUM	RA226
05-444	M	1890	1963	06	1917	43	1961	4	CL	0.0	Z9C	1	0	11	0
05-446	M	1898	1971	45	1925	40	1964	4	CL	0.0	Z9C	1	0	10	0
05-447	F	1902	01	01	1916	9	1970	2	B6	0.0	Z9B	1	0	10	0
05-448	F	1903	01	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	07	1918	117	1971	1	B6	0.00090	Z7B	0	0	5	2
05-459	F	1917	01	01	1933	208	1961	8	C6	0.0	Z9C	2	0	22	0
05-460	F	1898	1979	07	1916	182	1961	4	CL	0.0	Z9C	1	0	18	0
05-464	F	1895	1969	01	1917	40	1968	5	36	0.0	Z9	2	0	22	0
05-473	M	1899	1970	06	1921	26	1952	4	CL	0.01100	Z7C	1	2	11	18
05-528	F	1892	01	01	1917	52	1967	0	G6	0.0	Z9	0	0	0	0
05-541	F	1913	01	01	1937	984	1972	0	B6	0.0	Z9B	0	0	0	0
05-546	F	1902	07	07	1918	52	1973	1	B6	0.00012	Z7B	0	0	5	0
05-551	F	1895	01	01	1918	9	1970	15	G6	0.00018	Z7	5	0	73	7
05-555	F	1898	1965	07	1917	27	1975	1	A6	0.0	Z9	0	0	4	0
05-560	F	1894	1965	07	1921	260	1962	4	CL	0.01190	Z7C	1	1	9	13
05-574	F	1903	01	01	1918	1	1977	0	C6	0.00008	Z7	0	0	0	0
05-580	M	1914	1975	07	1919	6	1968	4	36	0.00260	Z7	1	1	13	13
05-602	M	1899	06	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
05-621	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	P6	0.00950	Z7P	0	0	2	4
05-674	M	1922	06	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	P4	0.00410	P1	38	91	437	1359
05-737	M	1895	1957	06	1918	156	1971	10	P4	0.00462	Z4P	3	0	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	40	1969	0	A6	0.00500	Z7A	0	0	0	0
05-765	F	1900	07	07	1916	117	1964	4	CL	0.0	Z9C	1	0	19	0
05-812	F	1893	01	01	1918	40	1972	1	B6	0.00014	Z7B	0	0	2	0
05-819	F	1901	1969	01	1918	52	1967	25	B2	0.00026	Z7B	7	1	104	11
05-873	F	1894	07	07	1917	286	1962	39	C2	0.00350	Z7C	11	6	168	95
05-880	F	1921	01	01	1939	520	1974	0	B6	0.0	Z9B	0	0	5	0
05-892	F	1917	1965	01	1935	468	1964	13	G6	0.0	Z9	3	0	24	0
05-895	F	1917	01	01	1939	572	1969	0	G6	0.0	Z9	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DEATH	EXP	YEAR	WKS	MEAS	RA226 NCI	RA226 METHOD + REP	RA226 TO RA226 RATIO	RA226 METHOD + REP	INPUT RA226 UCI	INPUT RA226 UCI	COM RA226 RADS	CU4 RADS
05-892	F	1934	1968	01	1917	4	1968	70	66	0.0	29	22	0	342	0
05-897	F	1899	1968	01	1917	69	1968	1310	34	0.0	29	400	0	5541	0
05-898	F	1919		01	1936	463	1972	0	86	0.0	29B	0	0	0	0
05-900	F	1919	1973	01	1936	112	1972	3	83	0.0	29C	1	0	8	0
05-901	F	1918		01	1934	468	1972	2	86	0.0	29B	0	0	6	0
05-902	F	1919		01	1936	982	1962	5	66	0.0	29C	1	0	10	0
05-905	F	1916		76	1937	156	1972	0	86	0.0	29B	0	0	0	0
05-906	F	1913		01	1935	624	1972	2	86	0.0	29B	0	0	5	0
05-907	F	1915		01	1935	260	1972	3	56	0.0	29C	1	0	9	0
05-911	M	1885		07	1923	6	1972	0	66	0.00310	27	0	0	0	0
05-912	M	1877	1951	07	1918	26	1969	0	46	0.00020	27A	0	0	0	0
05-917	F	1902		01	1918	39	1966	83	B1	0.00030	27C	25	2	385	36
05-920	M	1895	1963	06	1917	43	1962	4	CL	0.0	29C	1	0	11	0
05-921	F	1896		01	1916	30	1969	67	34	0.0	29	21	0	335	0
05-942	M	1901		06	1918	9	1975	0	86	0.00010	27B	0	0	0	0
05-949	M	1999	1974	06	1921	422	1968	0	G6	0.0	23	0	0	0	0
05-953	F	1902	1976	01	1918	65	1977	1200	P4	0.00008	27F	396	36	6110	547
05-962	F	1894	1977	01	1919	84	1969	47	C2	0.00200	27C	14	7	207	99
05-974	F	1903		07	1919	104	1970	0	G6	0.00100	27	0	0	0	0
05-979	F	1807		01	1917	4	1969	194	G4	0.0	29	60	0	956	0
05-993	M	1902	1972	07	1917	6	1971	7	B3	0.0	258	2	0	23	0
05-994	F	1886		01	1922	26	1967	6	34	0.00570	27	3	3	39	51
05-998	F	1902		01	1918	3	1974	0	B6	0.00011	27B	0	0	0	0
09-001	F	1901		01	1917	39	1971	1	P3	0.0	29B	1	0	20	0
09-002	F	1902	1970	01	1917	17	1950	1	B3	0.0	29B	1	0	40	0
09-003	M	1892	1962	06	1914	572	1959	410	B1	0.0	29B	110	0	989	0
09-004	F	1893	1961	01	1912	416	1960	550	C2	0.0	29C	156	0	2013	0
09-006	F	1898	1971	51	1917	65	1963	1	B6	0.0	29B	0	0	4	0
09-007	F	1901	1965	01	1917	104	1962	33	C2	0.0	29C	9	0	121	0
09-008	F	1903		01	1917	8	1962	20	C6	0.0	29C	6	0	89	0
09-009	F	1893	1969	01	1915	78	1960	2	86	0.0	29B	1	0	8	0
09-010	F	1897	1964	01	1914	40	1960	4	C6	0.0	29C	3	0	40	0
09-013	F	1900	1976	01	1917	13	1971	4	B3	0.0	29B	1	0	19	0
09-015	M	1890	1970	04	1914	52	1960	0	56	0.0	23	0	0	0	0
09-019	F	1903		01	1917	13	1975	0	36	0.0	29B	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	LIPD	EXP	YR	EXP	YR	FA226	RA226	RA226	RA226	RA226	RA226	RA226	RA226
								NCI	FA226	RA226	RA226	RA226	RA226	RA226	RA226
09-020	F	1897	1968	01	1917	155	1963	1	B6	0.0	29B	0	0	4	0
09-024	F	1873	1960	06	1915	43	1960	0	F6	0.0	29B	0	0	0	0
09-026	F	1902	1960	01	1917	43	1972	16	B1	0.0	29B	5	0	85	0
09-028	F	1897	1976	01	1916	78	1975	63	B2	0.0	29B	22	0	305	0
09-029	F	1901	1962	11	1917	13	1969	16	C2	0.0	29C	5	0	58	0
09-031	F	1897	1967	07	1913	364	1969	286	C2	0.0	29B	81	0	1491	0
09-032	F	1902	1969	01	1917	52	1969	97	B1	0.0	29B	30	0	421	0
09-038	F	1903	1969	01	1919	1	1960	0	F6	0.0	29B	0	0	0	0
09-041	M	1883	1952	06	1914	260	1965	114	A1	0.0	29A	29	0	229	0
09-043	F	1898	1976	01	1917	26	1971	11	B6	0.0	29B	3	0	53	0
09-044	F	1900	1955	01	1917	13	1975	17	A2	0.0	29	4	0	52	0
09-046	F	1902	1965	01	1917	104	1960	10	C3	0.0	29C	3	0	37	0
09-049	F	1902	1965	01	1915	40	1969	14	G6	0.0	29	4	0	72	0
09-051	F	1900	1971	01	1917	104	1960	50	C6	0.0	29C	14	0	199	0
09-052	F	1903	1971	01	1916	52	1960	20	C6	0.0	29C	6	0	83	0
09-053	M	1874	1966	04	1919	40	1960	81	B1	0.0	29B	22	0	210	0
09-057	F	1890	1973	01	1917	52	1960	0	B6	0.0	29B	0	0	0	0
09-058	F	1899	1973	01	1917	39	1960	4	B6	0.0	29B	1	0	18	0
09-059	F	1903	1972	01	1917	1	1971	2	B6	0.0	29B	1	0	9	0
09-060	F	1899	1975	01	1917	65	1969	43	B2	0.0	29B	13	0	200	0
09-061	F	1992		01	1914	208	1970	0	G6	0.0	29	0	0	0	0
09-062	F	1901		01	1918	52	1972	4	B3	0.0	29B	1	0	20	0
09-064	F	1891		01	1916	9	1973	1	B4	0.0	29B	0	0	5	0
09-065	F	1887	1975	06	1914	78	1960	1	B6	0.0	29B	0	0	5	0
09-066	F	1899		01	1917	8	1972	2	B6	0.0	29B	1	0	10	0
09-070	M	1875	1967	06	1913	209	1960	3	B6	0.0	29B	1	0	9	0
09-071	F	1897	1977	01	1917	104	1975	2	B6	0.0	29B	1	0	10	0
09-072	F	1893	1974	01	1917	29	1972	2	B6	0.0	29C	1	0	10	0
09-073	M	1886	1963	06	1916	468	1962	0	B6	0.0	29E	0	0	0	0
09-074	F	1892	1976	01	1920	104	1962	13	G6	0.0	29	4	0	52	0
09-075	M	1893	1967	06	1913	884	1963	1	F6	0.0	29F	0	0	3	0
09-076	M	1882	1966	06	1913	1672	1964	14	D3	0.0	29D	3	0	25	0
09-077	M	1894		06	1914	520	1972	2	F6	0.0	29B	1	0	7	0
09-078	M	1883	1966	06	1911	832	1963	3	F6	0.0	29B	1	0	8	0
09-079	M	1831		06	1916	570	1962	0	G6	0.0	29	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	DOB	DIED	EXP	YEAR	EXP	YEAR	FA226 NCI	FA226 METHOD	FA226 TO E226 RATIO	FA226 METHOD + E226 + E226	FA226 UCI	INPUT RA228	CUM RADS	CUM FADS
09-080	M	1896		06	1910	312	1962	5	36	0.0	29	1	0	15	0
09-082	M	1892		06	1916	1.2	1979	5	B3	0.0	29B	2	0	22	0
09-083	M	1889	1964	06	1915	17	1962	5	G6	0.0	73	1	0	14	0
09-084	M	1888	1927	06	1912	676	1965	362	A1	0.0	29A	42	0	131	0
09-086	M	1895	1970	06	1921	78	1974	1	E6	0.0	29E	0	0	3	0
09-088	M	1900		06	1922	336	1971	18	B2	0.0	29B	5	0	54	0
09-089	M	1890	1973	06	1915	78	1959	64	C2	0.0	29C	18	0	194	0
09-090	M	1888	1971	06	1913	78	1963	0	G6	0.0	29	0	0	0	0
09-095	M	1894	1975	06	1918	116	1975	0	B6	0.0	29B	0	0	0	0
09-096	M	1892	1978	06	1919	17	1963	9	36	0.0	29	3	0	28	0
09-097	M	1896		07	1916	988	1974	1	B6	0.0	29B	0	0	3	0
09-098	M	1902	1971	06	1921	104	1963	14	G6	0.0	29	4	0	37	0
09-099	M	1898	1971	06	1913	208	1963	1	G6	0.0	29	0	0	3	0
09-100	M	1889		06	1918	364	1963	9	G6	0.0	29	2	0	27	0
09-101	M	1884	1964	06	1920	39	1963	6	G6	0.0	29	2	0	15	0
09-102	M	1892	1951	46	1915	1	1964	150	A1	0.0	29A	38	0	306	0
09-103	M	1895	1971	06	1918	416	1965	1	56	0.0	29	0	0	3	0
09-104	M	1880	1967	06	1906	364	1965	42	B2	0.0	29B	13	0	146	0
09-105	M	1886	1978	06	1912	832	1965	130	A1	0.00003	Ac	112	17	333	114
09-106	M	1901		06	1919	156	1979	0	B6	0.0	29B	0	0	0	0
09-107	M	1897	1974	06	1913	104	1965	1	G6	0.0	29	0	0	3	0
09-108	M	1891		06	1915	104	1965	4	36	0.0	29	1	0	14	0
09-109	M	1895		06	1914	104	1965	4	36	0.0	29	1	0	14	0
09-110	M	1900		06	1914	52	1965	7	G6	0.0	29	2	0	25	0
09-111	M	1874	1900	06	1913	520	1967	0	36	0.0	29A	0	0	0	0
09-112	M	1898		06	1940	416	1966	04	34	0.0	29	17	0	130	0
09-115	M	1893		06	1920	52	1966	3	36	0.0	29	1	0	10	0
09-117	F	1899		01	1917	24	1971	4	B3	0.0	29B	1	0	20	0
09-118	F	1901		07	1921	40	1970	50	34	0.0	73	15	0	225	0
09-120	M	1869	1945	06	1915	104	1974	1	46	0.0	29	0	0	2	0
09-123	M	1890		06	1917	156	1979	0	36	0.0	29B	0	0	0	0
10-007	F	1916		01	1934	1144	1971	0	B6	0.0	29B	0	0	0	0
10-008	F	1904		01	1918	13	1975	0	E6	0.00009	27E	0	0	0	0
10-010	F	1895	1975	05	1930	40	1971	600	A1	0.0	29C	2361	0	30382	0
10-012	M	1886	1941	05	1925	40	1972	0	A6	0.0	29	0	0	0	0

TABLE 1 (CONT.) XPCSUITE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP	YEAR	PPX	YR	RA226	RA226	RA226	RA226	RA226	RA226	RA226	RA226
				TYPE	FIRST	EXP	OF	NCI	RA226	RA226	RA226	RA226	RA226	RA226	RA226
10-018	F	1920		01	1952	116	1975	1	B6	0.0	Z9B	0	0	2	0
10-024	M	1914		06	1936	142	1977	50	B4	0.0	Z9	8	0	55	0
10-025	M	1937		07	1963	416	1971		B3	0.0	Z9C	0	0	2	0
10-026	M	1948		07	1969	203	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	1979	3	C6	0.0	Z9C	1	0	8	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	232	1973	9	B2	0.0	Z9C	2	0	21	0
10-035	F	1922		01	1942	689	1974	10	B2	0.0	Z9C	2	0	22	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	73	1974	1	B6	0.0	Z9C	0	0	1	0
10-039	F	1922		07	1942	260	1972	4	B3	0.0	Z9C	1	0	9	0
10-040	F	1917		01	1946	40	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	40	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	26	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	27	0
10-051	M	1914		06	1931	468	1979	1	C6	0.0	Z9C	0	0	3	0
10-053	F	1926		17	1946	260	1972	2	B6	0.0	Z9C	0	0	3	0
10-054	F	1926		71	1946	104	1972	1	B6	0.0	Z9C	0	0	3	0
10-055	M	1922		08	1922	34	1972	0	B6	0.00340	Z9B	0	0	0	0
10-056	M	1924		08	1924	39	1972	2	B6	0.0040	Z9B	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	16	0
10-059	F	1915		01	1954	143	1972	0	B6	0.0	Z9C	0	0	0	0
10-060	F	1919		01	1943	134	1972	0	B6	0.0	Z9C	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	FXP TYPE	YFAB	EXP OF	YFAB	RA226 NCI	RA226 METHOD + ERR	RA226 METHOD + ERR	RA226 INPUT UCI	RA226 INPUT UCI	RA226 INPUT UCI	CUM RADS	CUM RADS
10-061	F	1923		17	1942	164	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	155	1972	0	B6	0.0	Z9C	0	0	0	0
10-065	F	1920		01	1941	240	1972	0	B6	0.0	Z9C	0	0	1	0
10-066	F	1924	1978	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	18	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	6	0
10-070	F	1921		01	1945	1352	1974	14	B2	0.0	Z9C	2	0	16	0
10-071	F	1924		01	1943	1508	1972	13	B2	0.0	Z9C	1	0	11	0
10-072	F	1924		01	1947	1300	1972	12	B2	0.0	Z9C	1	0	9	0
10-073	M	1919		07	1953	208	1972	0	B6	0.0	Z9C	0	0	0	0
10-074	M	1921		06	1950	1508	1979	21	C3	0.0	Z9C	2	0	10	0
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	26	0
10-079	F	1920		01	1940	624	1978	8	C3	0.0	Z9C	2	0	20	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	6	0
10-083	F	1924		01	1943	104	1972	5	B3	0.0	Z9C	1	0	13	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	1979	5	C6	0.0	Z9C	1	0	7	0
10-087	F	1920	1978	01	1942	1500	1972	19	B2	0.0	Z9C	2	0	17	0
10-088	F	1923		17	1946	260	1972	3	B6	0.0	Z9C	1	0	6	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	70	1974	423	A1	0.0	Z9A	14	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	11	0
10-096	F	1930		01	1951	832	1972	0	B6	0.0	Z9C	0	0	0	0
10-097	F	1914		01	1943	364	1972	4	B3	0.0	Z9C	1	0	8	0

TABLE 1 (CONT.) EXPOSURE DATA OF RADIUM PATIENTS TO END OF 1973

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	PCN	DIED	EXP	YR	EXP	YR	PA226	PA226	PA226	PA226	PA226	PA226	CUM	CUM
				TYPE	EXP	WKS	MEAS	NCI	METHOD	PA226	PA226	UCI	RA226	RA226	RA226
10-136	F	1920		01	1941	26	1972	0	B6	0.0	Z9C	0	0	0	0
10-137	F	1919		01	1935	117	1972	1	B6	0.0	Z9C	0	0	0	0
10-138	F	1922		01	1942	130	1972	3	B6	0.0	Z9C	0	0	0	0
10-139	F	1922		01	1942	130	1972	3	B6	0.0	Z9C	0	0	0	0
10-140	F	1922		01	1942	130	1972	3	B6	0.0	Z9C	0	0	0	0
10-141	F	1919		01	1942	130	1972	3	B6	0.0	Z9C	0	0	0	0
10-142	F	1922		01	1942	130	1972	3	B6	0.0	Z9C	0	0	0	0
10-143	F	1922		01	1942	130	1972	3	B6	0.0	Z9C	0	0	0	0
10-144	F	1926		01	1945	156	1972	2	B6	0.0	Z9C	0	0	0	0
10-145	F	1928		01	1946	130	1976	6	C3	0.0	Z9C	0	0	0	0
10-146	F	1921		01	1940	364	1972	4	B3	0.0	Z9C	0	0	0	0
10-147	F	1927		01	1946	156	1972	2	B6	0.0	Z9C	0	0	0	0
10-148	F	1913		01	1935	13	1978	2	C6	0.0	Z9C	0	0	0	0
10-149	F	1924		01	1943	114	1972	4	B3	0.0	Z9C	0	0	0	0
10-150	F	1919	1976	01	1919	13	1972	0	G6	0.0	Z9	0	0	0	0
10-151	F	1897	1976	06	1915	520	1974	0	G8	0.0	Z9	0	0	0	0
10-152	F	1923		01	1941	52	1972	2	B6	0.0	Z9F	0	0	0	0
10-153	F	1921		01	1941	234	1972	1	B6	0.0	Z9B	0	0	0	0
10-154	F	1921		01	1941	208	1976	20	B1	0.0	Z9C	0	0	0	0
10-155	F	1921		01	1951	13	1974	3	B2	0.0	Z9C	0	0	0	0
10-156	F	1915		01	1937	156	1974	0	B6	0.0	Z9C	0	0	0	0
10-157	F	1919		01	1942	416	1972	2	B6	0.0	Z9C	0	0	0	0
10-158	F	1924		01	1942	156	1974	3	B3	0.0	Z9C	0	0	0	0
10-159	F	1930	1977	07	1945	123	1974	3	B3	0.0	Z9C	0	0	0	0
10-160	F	1915	1977	01	1945	123	1973	0	B6	0.0	Z9C	0	0	0	0
10-161	F	1919		01	1941	728	1974	0	R2	0.0	Z9C	0	0	0	0
10-162	F	1912		01	1931	287	1978	2	C6	0.0	Z9C	0	0	0	0
10-163	F	1921		01	1951	117	1972	2	B6	0.0	Z9C	0	0	0	0
10-164	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-165	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-166	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-167	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-168	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-169	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-170	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-171	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-172	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-173	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-174	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-175	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-176	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-177	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-178	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-179	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-180	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-181	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-182	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-183	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-184	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-185	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-186	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-187	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-188	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-189	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-190	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-191	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-192	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-193	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-194	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-195	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-196	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-197	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-198	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-199	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-200	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-201	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-202	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-203	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0
10-204	F	1940		01	1971	17	1972	2	B6	0.0	Z9C	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADON PATIENTS TO END OF 1973

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DATE	TYPE	AGE	WKS	YEARS	FA226	FA226	FA226	FA226	FA226	FA226	FA226	FA226
								AC	AC	AC	AC	AC	AC	AC	AC
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	Z9C	0	0	3	0
10-257	F	1932		07	1951	104	1972	0	B6	0.0	Z9C	0	0	1	0
10-258	F	1923		01	1943	26	1972	3	B6	0.0	Z9C	1	0	7	0
10-260	F	1913		01	1928	52	1978	2	C6	0.0	Z9C	1	0	7	0
10-261	F	1922		01	1941	28	1972	3	B6	0.0	Z9C	1	0	8	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	1978	1	C6	0.0	Z9C	0	0	3	0
10-269	F	1925		01	1945	17	1972	0	B6	0.0	Z9C	0	0	0	0
10-270	F	1926		01	1946	104	1972	1	B6	0.0	Z9C	0	0	1	0
10-272	F	1915		01	1935	60	1976	5	C3	0.0	Z9C	2	0	19	0
10-273	F	1929		01	1948	22	1973	2	B6	0.0	Z9C	0	0	4	0
10-274	F	1924		01	1947	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		01	1946	154	1973	1	B6	0.0	Z9C	0	0	1	0
10-278	F	1908		01	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	2	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		01	1936	156	1974	3	B2	0.0	Z9C	1	0	6	0
10-285	F	1917		07	1935	81	1973	0	B6	0.0	Z9C	0	0	0	0
10-286	F	1937		07	1956	124	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	416	1974	2	B6	0.0	Z9C	0	0	5	0
10-295	F	1923		07	1946	282	1973	2	B6	0.0	Z9C	0	0	2	0
10-296	F	1950		01	1948	50	1973	0	B6	0.0	Z9C	0	0	0	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXF	YEAR	EXP	YFAR	RA226	RA226	RA226	RA226	INPUT	INPUT	CUM	CUM
				TYPE	FIRST	DUR	OF	METHOD	METHOD	TO	METHOD	RA226	RA226	RA226	RA226
					EXP	WKS	MEAS	NCI	+ EFF	RATIO	+ EFF	UCI	UCI	RA226	RA226
10-297	F	1929	1973	07	1969	66	1973	0	B6	0.0	Z9C	0	0	0	0
10-298	F	1923		01	1942	43	1973	6	B3	0.0	Z9C	2	3	17	0
10-300	F	1911		01	1940	1612	1977	0	B6	0.0	Z9C	0	0	1	0
10-301	F	1930		07	1948	74	1973	0	B6	0.0	Z9C	0	0	0	0
10-302	F	1917		07	1933	312	1975	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	22	0
10-307	F	1893	1948	05	1920	40	1974	85	A2	0.0	Z9A	15	0	109	0
10-309	F	1925		01	1943	28	1973	2	E6	0.0	Z9C	0	0	4	0
10-310	F	1916		01	1935	53	1973	2	B6	0.0	Z9C	0	0	6	0
10-311	F	1919		01	1942	16	1973	0	B6	0.0	Z9C	0	0	1	0
10-312	F	1923		01	1942	16	1973	2	B6	0.0	Z9C	0	0	4	0
10-313	F	1924		01	1942	202	1973	9	B3	0.0	Z9C	2	0	23	0
10-314	F	1918		01	1943	119	1973	4	R3	0.0	Z9C	1	0	10	0
10-316	M	1946		07	1965	167	1973	2	B6	0.0	Z9C	0	0	1	0
10-318	M	1908		07	1970	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
10-323	F	1951		07	1973	57	1979*	2	B3	0.0	Z9C	0	0	1	0
10-324	F	1912		01	1926	13	1978	0	C6	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	864	1979	0	C6	0.0	Z9C	0	0	0	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	1978	0	G6	0.00204	Z9	0	0	0	0
10-333	F	1915		01	1941	258	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	E6	0.0	Z9C	0	0	0	0
10-336	F	1923		07	1941	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

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIO* PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BOB	DIED	TYPE	EXP	WKS	MEAS	FA226 NCI	FA226 METHOD	FA226 RATIO	RA226 METHOD	INPUT UCI	INPUT RA226	COM FADS	COM FADS
10-340	F	1920		07	1942	104	1974	6	B3	0.0	Z9C	1	3	16	0
10-341	F	1919		01	1939	312	1973	1	B6	0.0	Z9A	3	0	3	0
10-347	F	1947		09	1947	39	1973	1	B6	0.0	Z9A	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	F	1931		07	1944	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	1948	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	F	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
10-369	F	1921		01	1941	104	1978	1	C6	0.0	Z9C	0	0	2	0
10-375	F	1924		01	1943	20	1973	1	B6	0.0	Z9C	0	0	3	0
10-377	F	1958		07	1921	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	91	0
10-381	F	1927		01	1946	27	1973	6	B3	0.0	Z9C	1	0	13	0
10-382	F	1923		01	1942	119	1973	5	B3	0.0	Z9C	1	0	14	0
10-384	F	1919		01	1943	884	1973	1	B6	0.0	Z9C	0	0	3	0
10-385	F	1921		07	1964	16	1973	0	B6	0.0	Z9C	0	0	0	0
10-386	F	1933		01	1954	56	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	1923		01	1922	520	1973	0	B6	0.0	Z9C	0	0	0	0
10-393	F	1907		01	1925	208	1974	5	B2	0.0	Z9C	2	0	24	0
10-394	F	1907	1974	01	1923	729	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		01	1951	224	1973	1	B6	0.0	Z9C	0	0	1	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIOM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP	YEAR	EXP	YEAR	FA226	FA226	FA226	FA226	INPUT	INPUT	CUM	CUM
				TYPE	FIRST	DUR	OF	NCI	METHOD	TO	METHOD	RA226	RA226	RA226	RA226
					EXP	WKS	MEAS		+ ERR	PAIO	+ ERR	UCL	UCL		
10-474	F	1921		01	1946	77	1974	2	B6	0.0	Z9C		0	5	0
10-475	F	1927		07	1946	90	1973	0	B6	0.0	Z9C		0	0	0
10-476	F	1928		01	1946	12	1972	1	B6	0.0	Z9C		0	1	0
10-477	F	1924		01	1944	42	1975	2	B3	0.0	Z9C		0	6	0
10-478	F	1922		01	1942	11	1973	0	B6	0.0	Z9C		0	0	0
10-479	F	1926		01	1946	11	1973	0	B6	0.0	Z9C		0	0	0
10-480	F	1924		01	1943	4	1973	0	B6	0.0	Z9C		0	1	0
10-481	F	1925		01	1942	5	1973	1	B6	0.0	Z9C		0	3	0
10-482	F	1925		01	1943	28	1973	4	B3	0.0	Z9C		0	11	0
10-483	F	1934		07	1951	5	1973	2	B6	0.0	Z9C		0	2	0
10-485	F	1918		01	1948	4	1973	0	B6	0.0	Z9C		0	1	0
10-486	F	1919		01	1942	32	1973	0	B6	0.0	Z9C		0	1	0
10-487	F	1924		01	1943	220	1973	0	B6	0.0	Z9C		0	0	0
10-488	F	1921		01	1942	20	1973	0	B6	0.0	Z9C		0	0	0
10-490	F	1922		01	1943	20	1974	8	B2	0.0	Z9C		0	20	0
10-492	F	1925		01	1951	326	1973	2	B6	0.0	Z9C		0	3	0
10-494	F	1913		01	1939	312	1973	1	B6	0.0	Z9C		0	2	0
10-495	F	1924		01	1942	312	1973	0	B6	0.0	Z9C		0	0	0
10-496	F	1922		01	1940	108	1975	0	B6	0.0	Z9C		0	0	0
10-501	F	1928		01	1946	15	1973	2	B6	0.0	Z9C		0	4	0
10-502	F	1928		01	1945	13	1973	2	B6	0.0	Z9C		0	4	0
10-505	F	1933		01	1951	3	1973	2	B6	0.0	Z9C		0	4	0
10-506	F	1920		07	1946	4	1973	0	B6	0.0	Z9C		0	1	0
10-510	F	1924		07	1942	26	1973	1	B6	0.0	Z9C		0	3	0
10-511	F	1923		01	1943	12	1973	5	B3	0.0	Z9C		0	13	0
10-512	F	1936		01	1965	1	1973	0	B6	0.0	Z9C		0	0	0
10-518	F	1905		06	1928	1196	1978	1	B6	0.0	Z9C		0	3	0
10-520	F	1924		01	1942	5	1973	1	B6	0.0	Z9C		0	3	0
10-521	F	1923		01	1955	416	1973	1	B6	0.0	Z9C		0	2	0
10-523	F	1922		01	1942	17	1973	0	B6	0.0	Z9C		0	0	0
10-525	F	1928		01	1947	1	1973	1	B6	0.0	Z9C		0	2	0
10-530	F	1952		07	1971	52	1973	1	B6	0.0	Z9C		0	1	0
10-531	F	1924		01	1946	1	1973	2	B6	0.0	Z9C		0	4	0
10-532	F	1916		01	1942	2	1973	1	B6	0.0	Z9C		0	3	0
10-533	F	1925		01	1943	5	1973	2	B6	0.0	Z9C		0	5	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	TYPE	EXP	YEAR	EXP	YEAR	FA226	METHOD	FA226	INPUT	RA226	CUM	RA226
									NCI	+ EFF	RA226	RA226	RA226	RA226	RA226
10-534	F	1925		01	1946	54	1973		2	C6	0.0	0	0	5	0
10-535	F	1927		01	1946	16	1973		1	C6	0.0	0	0	2	0
10-536	F	1927		01	1942	1	1973		1	B6	0.0	0	0	3	0
10-538	M	1896	1978	07	1938	232	1977		1	B2	0.0	0	0	1	0
10-540	M	1917	1978	01	1939	176	1973		2	B6	0.0	0	0	1	0
10-543	M	1891		06	1916	26	1973		3	B3	0.0	1	0	11	0
10-546	F	1906		07	1929	208	1979		6	C3	0.0	2	0	23	0
10-549	F	1914		01	1941	62	1973		4	B3	0.0	1	0	12	0
10-550	F	1921		01	1942	43	1974		1	C6	0.0	0	0	1	0
10-557	F	1921		01	1942	43	1974		4	B3	0.0	1	0	11	0
10-558	M	1927		07	1951	40	1973		5	B3	0.0	1	0	7	0
10-559	F	1919		01	1941	69	1973		2	B6	0.0	0	0	5	0
10-560	F	1921		01	1942	96	1973		4	B3	0.0	1	0	9	0
10-561	M	1906		06	1927	52	1978		2	B6	0.0	1	0	6	0
10-566	M	1914	1977	02	1930	13	1976		5	B2	0.00334	1	1	14	14
10-569	F	1925		01	1946	1	1975		0	B6	0.0	0	0	0	0
10-570	M	1907		06	1934	780	1977		2	B3	0.0	0	0	4	0
10-573	F	1922		01	1944	14	1973		3	B3	0.0	1	0	7	0
10-574	M	1908		01	1930	2236	1973		7	B2	0.0	1	0	6	0
10-575	F	1930		01	1948	1040	1973		4	B3	0.0	1	0	5	0
10-579	M	1926	1979	07	1948	1248	1973		0	B6	0.0	0	0	0	0
10-580	F	1930		01	1946	52	1973		3	B3	0.0	1	0	6	0
10-582	F	1938		01	1965	416	1973		1	B6	0.0	0	0	0	0
10-583	M	1918		06	1939	1352	1973		0	B6	0.0	0	0	0	0
10-584	F	1925		01	1942	3	1973		1	B6	0.0	0	0	2	0
10-585	M	1908		06	1930	52	1978		1	C6	0.0	0	0	4	0
10-587	M	1946		07	1966	416	1973		0	B6	0.00330	0	0	0	0
10-588	F	1910		01	1927	2	1974		0	B6	0.0	0	0	0	0
10-589	M	1938		07	1971	78	1973		2	B3	0.0	0	0	0	0
10-590	M	1912		06	1948	728	1979		0	B6	0.0	0	0	0	0
10-592	M	1899		06	1923	1300	1978		1	B6	0.0	0	0	2	0
10-594	F	1917		01	1943	5	1973		5	B3	0.0	1	0	13	0
10-595	F	1908		01	1928	194	1977		6	C6	0.0	2	0	24	0
10-596	F	1909		01	1927	6	1973		6	B3	0.0	2	0	23	0
10-597	F	1911		01	1928	17	1976		2	B3	0.0	1	0	8	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP	YR	EXP	YR	FA226	RA226	RA226	RA226	INPUT	INPUT	CUM	CUM
								NCI	FA226	RA226	RA226	FA226	RA226	FA226	FA226
								NCI	FA226	RA226	RA226	FA226	RA226	FA226	FA226
10-851	F	1921		01	1951	139	1974	0	B6	0.0	Z9B	0	0	0	0
10-852	F	1905		01	1921	13	1974	0	B6	0.01300	Z25	0	0	0	0
10-853	F	1919		01	1947	100	1974	1	B6	0.0	Z9B	0	0	1	0
10-854	M	1909		01	1928	104	1974	1	B6	0.0	Z9B	0	0	3	0
10-855	F	1928		01	1905	26	1974	7	B2	0.0	Z9C	2	0	16	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	1975		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	1925		01	1946	10	1974	0	B6	0.0	Z9C	0	0	0	0
10-864	M	1906		01	1949	156	1979	0	C6	0.0	Z9C	0	0	0	0
10-866	F	1900		01	1920	12	1979*	8	G4	0.00775	Z2	3	26	41	398
10-867	F	1915		07	1929	209	1974	0	B6	0.0	Z9B	0	0	0	0
10-869	F	1912		01	1927	132	1974	2	C6	0.00181	Z9B	1	1	9	8
10-870	F	1911	1978	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	1983	1935	02	1930	40	1975	27	A1	0.0	Z9	2	0	8	0
10-890	F	1912		01	1927	2	1979	0	B6	0.00181	Z9B	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
10-901	F	1910		01	1924	3	1975	0	B6	0.01160	Z2B	0	0	0	0
10-903	F	1909		01	1943	2	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	1969	0	1976	1	B6	0.0	Z9C	0	0	0	0
10-907	F	1910		01	1946	5	1979	1	C6	0.0	Z9C	0	0	2	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

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DISE	EXP	YEAR	EXP	YEAR	PA226	RA226	RA228	RA228	INPUT	INPUT	CUM	CUM
				TYPE	FIRST	WKS	OF	NCI	METHOD	TO RA226	METHOD	RA226	RA228	RADS	RADS
					EXP		MEAS		+ ERR	RATIO	+ ERR	UCI	UCI	RA226	RA228
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	B6	0.0	Z9C	0	0	0	0
10-921	F	1905		01	1923	1	1977	0	B6	0.00907	Z2	0	0	0	0
10-928	M	1918		07	1948	0	1958	1	B6	0.0	Z9	0	0	1	0
10-931	M	1911		01	1946	1040	1979	4	C3	0.0	Z9C	1	0	5	0
10-932	M	1903		76	1919	208	1979	15	B2	0.0	Z9B	5	0	54	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	1979	4	C3	0.0	Z9C	1	0	11	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	Z9B	0	0	4	4
10-953	F	1908		01	1923	49	1973*	15	G6	0.00770	Z2	5	12	71	478
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	9	0
10-962	F	1916		07	1934	27	1978	0	B6	0.0	Z9B	0	0	0	0
10-963	F	1901		01	1919	10	1975	647	B1	0.00170	B3B	209	318	3240	4784
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	1971	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	16	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP	YR	EXP	YR	RA226	RA226	RA226	RA226	RA226	RA226	RA226	RA226
								NCI	METHOD	RATIO	METHOD	UCI	UCI	CUM	CUM
									+ ERR		+ ERR			RADS	RADS
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	1958	3	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	1979	2	C6	0.0	Z9C	0	0	2	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	1979	3	C3	0.0	Z9C	1	0	9	0
10-996	F	1920		07	1943	260	1979	1	B6	0.0	Z9B	0	0	3	0
10-997	F	1926		07	1945	572	1979	0	B6	0.0	Z9	0	0	0	0
10-998	F	1929		07	1942	588	1978	0	B6	0.0	Z9B	0	0	0	0
11-002	F	1919		01	1941	728	1979	0	B6	0.0	Z9	0	0	0	0
11-003	F	1919		07	1942	40	1974	3	G6	0.0	Z9	1	0	8	0
11-004	M	1924		01	1946	702	1979	1	B6	0.0	Z9	0	0	1	0
11-005	M	1926		17	1948	1612	1979	3	B6	0.0	Z9	0	0	2	0
11-009	F	1913		07	1942	884	1979	0	B6	0.0	Z9B	0	0	0	0
11-010	F	1922		07	1942	598	1979	0	B6	0.0	Z9	0	0	0	0
11-015	F	1907		01	1925	2	1976	0	G6	0.01000	Z2	0	0	0	0
11-016	F	1926		01	1924	17	1978	24	C3	0.00803	Z2	0	0	112	642
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	Z9B	0	0	0	0
11-021	F	1907		07	1931	282	1978	0	C6	0.00203	Z3	0	0	0	0
11-023	F	1911		17	1927	2	1975	0	B6	0.00290	Z9B	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	1975	71	1948	312	1978	0	B6	0.0	Z9	0	0	0	0
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	336	1974	3	B3	0.0	Z5C	0	0	1	0
11-033	M	1951		06	1971	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	48	0
11-035	M	1949		07	1973	60	1977	0	C6	0.0	Z9C	0	0	0	0
11-036	M	1916		07	1946	1716	1979	6	C3	0.0	Z9C	1	0	3	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADON PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SFX	BORN	DIED	EXP. TYPE	YEAR	AGE	DATE	RA226	RA226	RA226	METHOD	RA226	RA226	CUM	RA226
								NCI	RA226	RA226	RA226	RA226	RA226	RA226	RA226
11-859	F	1923		01	1941	18	1978	1	B6	0.0	29B	0	0	0	0
11-861	F	1922		01	1941	18	1977	0	B6	0.0	29B	0	0	0	0
11-863	F	1916		01	1942	17	1977	0	B6	0.0	29B	0	0	0	0
11-865	F	1920		01	1942	17	1977	0	B6	0.0	29B	0	0	0	0
11-866	F	1927		01	1942	15	1977	0	B6	0.0	29B	0	0	0	0
11-871	F	1925		01	1940	17	1977	5	B3	0.0	29B	1	0	14	0
11-875	F	1923		01	1941	16	1977	1	B6	0.0	29B	0	0	3	0
11-916	F	1918		01	1941	10	1975	1	B6	0.0	29B	0	0	3	0
11-923	F	1924		01	1942	10	1975	1	B6	0.0	29B	0	0	2	0
11-924	F	1920		01	1941	10	1978	1	B6	0.0	29B	0	0	2	0
11-925	F	1920		01	1941	17	1975	0	B6	0.0	29B	0	0	0	0
11-938	F	1931		01	1951	16	1975	0	B6	0.0	29B	0	0	0	0
11-947	F	1925		01	1942	16	1975	4	B3	0.0	29B	1	0	3	0
11-957	F	1925		01	1942	16	1979*	1	B6	0.0	29B	0	0	0	0
11-959	F	1912		01	1941	20	1977	0	B6	0.0	29B	0	0	0	0
11-960	F	1924		01	1942	16	1975	0	B6	0.0	29B	0	0	0	0
11-962	F	1922		01	1942	13	1979*	0	B6	0.0	29B	0	0	0	0
11-971	F	1923		01	1944	15	1979*	0	B6	0.0	29B	0	0	0	0
11-973	F	1919		01	1950	10	1975	0	B6	0.0	29B	0	0	0	0
11-974	F	1917		01	1944	10	1977	0	B6	0.0	29B	0	0	0	0
11-982	F	1922		01	1942	20	1976	0	B6	0.0	29B	0	0	0	0
11-989	F	1921		01	1943	15	1977	0	B6	0.0	29B	0	0	0	0
11-991	F	1924		01	1942	16	1975	0	B6	0.0	29B	0	0	0	0
12-012	F	1918		01	1941	11	1977	0	B6	0.0	29B	0	0	0	0
12-016	F	1919		01	1941	11	1977	0	B6	0.0	29B	0	0	0	0
12-022	F	1924		01	1942	15	1978	0	B6	0.0	29B	0	0	0	0
12-025	F	1914		01	1951	18	1975	0	B6	0.0	29B	0	0	0	0
12-026	F	1914		01	1942	16	1976	0	B6	0.0	29B	0	0	0	0
12-033	F	1925		01	1951	15	1975	3	B3	0.0	29B	1	0	6	0
12-040	F	1921		01	1942	15	1976	1	B6	0.0	29B	1	0	8	0
12-043	F	1921		01	1942	18	1978	2	B6	0.0	29B	1	0	5	0
12-045	F	1925		01	1942	16	1977	0	B6	0.0	29B	0	0	3	0
12-061	F	1920		01	1942	16	1975	1	B6	0.0	29B	0	0	3	0
12-064	F	1924		01	1942	15	1979*	0	B6	0.0	29B	0	0	0	0
12-074	F	1923		01	1943	10	1977	1	B6	0.0	29B	0	0	3	0

TABLE 1 (CONT.) RADIOTHERAPY DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2) (3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	EXP	YEAR	EXP	YEAR	FA226	FA226	FA226	FA226	FA226	FA226	FA226
				TYPE	FIRST	DUR	CF	METHOD	RATIO	METHOD	INPUT	INPUT	CUM	CUM
					EXP	HRS	WKS	NCI	ERR	+ PRR	RA226	RA226	RA226	RA226
12-075	F	1923		01	1941	208	1977	1	B6	0.0	0	0	3	0
12-086	F	1925		07	1942	156	1977	2	B6	0.0	0	0	5	0
12-089	F	1924		01	1943	52	1974	0	R6	0.0	0	0	0	0
12-094	F	1929		01	1946	4	1975	3	R6	0.0	1	0	6	0
12-095	F	1927		01	1947	1	1974	0	R6	0.0	0	0	1	0
12-096	F	1921		01	1946	22	1973	2	C6	0.0	1	0	6	0
12-098	F	1930		01	1951	52	1974	1	B6	0.0	0	0	1	0
12-099	F	1929		07	1951	18	1976	0	B6	0.0	0	0	0	0
12-102	F	1951		07	1972	0	1978	1	C6	0.0	0	0	0	0
12-108	F	1915		01	1942	23	1974	0	B6	0.0	0	0	0	0
12-110	F	1927		01	1946	1	1976	0	B6	0.0	0	0	0	0
12-111	F	1929		01	1947	19	1974	4	B3	0.0	1	0	9	0
12-113	F	1915		01	1942	19	1975	0	B6	0.0	0	0	1	0
12-115	F	1953		07	1972	52	1975	0	B6	0.0	0	0	0	0
12-117	F	1914		01	1943	3	1979	0	C6	0.0	0	0	0	0
12-118	F	1932		16	1954	2	1977	1	B3	0.0	0	0	2	0
12-119	F	1933		17	1947	41	1975	1	B6	0.0	0	0	0	0
12-123	F	1924		01	1945	17	1976	1	B3	0.0	0	0	3	0
12-127	F	1917		01	1941	17	1975	0	B6	0.0	0	0	0	0
12-128	F	1920		01	1943	20	1978	2	C6	0.0	1	0	6	0
12-129	F	1927		01	1946	4	1976	0	B6	0.0	0	0	1	0
12-130	F	1924		01	1947	2	1976	5	B2	0.0	1	0	11	0
12-133	F	1926		01	1946	7	1976	1	B3	0.0	0	0	3	0
12-134	F	1927		01	1946	1	1975	0	B6	0.0	0	0	0	0
12-136	F	1929		07	1966	4	1975	1	P6	0.0	0	0	0	0
12-141	F	1925		01	1943	3	1978	3	C3	0.0	1	0	10	0
12-142	F	1922		01	1942	8	1976	0	B6	0.0	0	0	0	0
12-143	F	1924		01	1942	56	1975	1	B6	0.0	0	0	2	0
12-145	F	1921		01	1941	25	1975	0	B6	0.0	0	0	0	0
12-146	F	1920		01	1943	32	1977	0	B6	0.0	0	0	1	0
12-148	F	1925		01	1946	4	1975	0	B6	0.0	0	0	0	0
12-150	F	1914		01	1943	104	1976	0	B3	0.0	1	0	10	0
12-155	F	1929		01	1954	34	1976	0	B6	0.0	0	0	1	0
12-163	F	1920		01	1942	78	1974	4	B3	0.0	1	0	10	0
12-164	F	1920		01	1943	13	1975	0	B6	0.0	0	0	1	0

TABLE 1 (CONT.) EXPOSURE DATA FOR SADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIE	EXP	YR	EXP	YR	PA226	PA226	PA226	RA226	INPUT	INPUT	CUM	FADS
				TYPE	EXP	YR	YR	NCI	RA226	RA226	RA226	RA226	RA226	RA226	RA226
12-397	M	1916		06	1947	520	1979	15	C3	0.0	Z9B	3	0	22	0
12-421	M	1940		07	1968	260	1978	2	C6	0.0	Z9C	0	0	1	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
12-426	M	1923		07	1946	18	1975	1	B6	0.0	Z9B	0	0	2	0
12-428	F	1907		01	1922	13	1978	190	C2	0.0	Z9C	61	0	920	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	1996		01	1918	26	1975	1	B6	0.0	Z9C	0	0	4	0
12-437	F	1926		01	1943	104	1975	1	B6	0.0	Z9C	0	0	4	0
12-438	M	1942		06	1964	122	1977	1	C6	0.0	Z9C	0	0	1	0
12-442	M	1945		07	1971	56	1978	1	C6	0.0	Z9C	0	0	0	0
12-443	M	1919	1978	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
12-448	M	1923		06	1967	624	1979*	1	B6	0.0	Z9C	0	0	0	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
12-453	M	1914		06	1929	156	1979*	9	B2	0.0	Z9C	2	0	19	0
12-455	M	1943		06	1970	87	1979*	3	B3	0.0	Z9C	0	0	1	0
12-456	M	1918		06	1938	364	1976	249	B1	0.0	Z9C	62	0	509	0
12-460	M	1923		17	1945	1092	1975	0	B6	0.0	Z9B	0	0	0	0
12-499	P	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
12-515	P	1917		01	1941	52	1978	0	C6	0.0	Z9C	0	0	0	0
12-516	F	1918		01	1941	4	1979*	3	B3	0.0	Z9C	1	0	9	0
12-518	M	1899		07	1941	104	1979*	0	C6	0.0	Z9C	0	0	0	0
12-522	F	1921		01	1941	30	1977	0	B6	0.0	Z9C	0	0	1	0
12-523	P	1923		01	1941	104	1977	0	C6	0.0	Z9C	0	0	1	0
12-528	F	1917		01	1943	156	1979*	1	B6	0.0	Z9C	0	0	3	0
12-529	F	1920		01	1941	104	1977	0	C6	0.0	Z9C	0	0	1	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	TYPE	EXP	YFAR	EXP	YEAR	FA226	FA226	FA226	RA226	IMPDI	INPUT	CUH	RADS
								NCI	FA226	RA226	RA226	RA226	RA226	FA226	RA226
12-650	M	1926	16	1955	16	1955	13 1977	2	B3	0.0	79C	0	0	2	0
12-651	F	1946	31	1965	31	1965	13 1977	0	B6	0.0	79C	0	0	0	0
12-665	F	1925	37	1971	37	1971	26 1977	1	B6	0.0	79C	0	0	0	0
12-659	M	1957	37	1974	37	1974	24 1977	0	B6	0.0	79C	0	0	0	0
12-672	M	1929	31	1951	31	1951	52 1977	1	B3	0.0	79C	0	0	2	0
12-672	F	1920	31	1942	31	1942	80 1979*	2	B6	0.0	79C	1	0	6	0
12-675	F	1931	31	1952	31	1952	30 1978	0	C6	0.0	79C	0	0	1	0
12-688	F	1917	31	1944	31	1944	17 1977	1	B6	0.0	79C	0	0	2	0
12-693	F	1922	31	1942	31	1942	0 1979*	0	B6	0.0	79C	0	0	1	0
12-694	F	1931	31	1943	31	1943	13 1976	0	B6	0.0	79B	0	0	0	0
12-695	F	1926	31	1951	31	1951	133 1979*	1	C6	0.0	79C	0	0	2	0
12-700	F	1929	31	1952	31	1952	52 1978	2	C6	0.0	79C	0	0	4	0
12-702	F	1918	31	1942	31	1942	160 1977	1	B6	0.0	79C	0	0	3	0
12-709	F	1925	31	1952	31	1952	121 1976	0	B6	0.0	79C	0	0	0	0
12-710	F	1911	31	1952	31	1952	104 1976	0	B6	0.0	79C	0	0	1	0
12-729	F	1904	31	1944	31	1944	6 1978	0	C6	0.0	79C	0	0	0	0
12-738	F	1922	31	1949	31	1949	32 1979*	0	C6	0.0	79C	0	0	0	0
12-739	F	1914	31	1954	31	1954	17 1978	3	C6	0.0	79C	1	0	5	0
12-746	F	1913	31	1942	31	1942	124 1976	0	B6	0.0	79B	0	0	0	0
12-748	F	1911	31	1944	31	1944	13 1978	0	C6	0.0	79C	0	0	0	0
12-757	F	1922	31	1941	31	1941	104 1976	1	B3	0.0	79C	0	0	3	0
12-764	F	1924	31	1952	31	1952	104 1977	1	B3	0.0	79C	0	0	3	0
12-765	F	1921	71	1945	71	1945	1352 1976	0	B6	0.0	79C	0	0	0	0
12-771	F	1930	31	1949	31	1949	936 1976	0	B6	0.0	79C	0	0	0	0
12-779	F	1929	31	1952	31	1952	52 1976	0	B6	0.0	79C	0	0	0	0
12-784	F	1930	31	1953	31	1953	17 1977	5	C6	0.0	79C	1	0	10	0
12-788	F	1918	31	1951	31	1951	163 1979*	0	C6	0.0	79C	0	0	0	0
12-791	F	1920	31	1943	31	1943	17 1979*	2	B3	0.0	79C	0	0	4	0
12-795	F	1918	31	1949	31	1949	17 1977	1	B6	0.0	79C	0	0	1	0
12-797	F	1922	31	1951	31	1951	184 1979*	2	C6	0.0	79C	0	0	3	0
12-802	F	1906	31	1943	31	1943	14 1978	2	C6	0.0	79C	1	0	6	0
12-810	F	1910	31	1943	31	1943	104 1977	0	B6	0.0	79C	0	0	0	0
12-826	F	1926	31	1943	31	1943	8 1977	2	C6	0.0	79C	1	0	6	0
12-829	F	1922	31	1949	31	1949	18 1977	0	C6	0.0	79C	0	0	0	0
12-831	F	1918	31	1940	31	1940	207 1978	1	B6	0.0	79B	0	0	3	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BOEN	DIED	EXP	YEAR	EXP	OF	RA226	RA226	RA226	RA226	RA226	RA226	RA226	RA226
				TYPE	EXP	TYPE	MEAS	NCI	RA226	RA226	RA226	RA226	RA226	RA226	RA226
12-841	F	1922		01	1952	1	1977	0	C6	0.0	Z9C	0	0	0	0
12-843	F	1916		01	1952	30	1979*	0	B6	0.0	Z9C	0	0	0	0
12-849	F	1916		01	1941	208	1977	0	C6	0.0	Z9C	1	0	8	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
12-858	F	1917		01	1951	22	1978	0	C6	0.0	Z9C	0	0	0	0
12-863	F	1929		01	1953	11	1978	2	C6	0.0	Z9C	0	0	3	0
12-864	F	1919		01	1952	34	1978	0	C6	0.0	Z9C	0	0	0	0
12-875	F	1921		01	1953	13	1979*	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	R3	0.0	Z9C	0	0	3	0
12-885	F	1918		01	1945	4	1978	2	C6	0.0	Z9C	1	0	6	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
12-891	F	1920		01	1946	40	1979*	3	B3	0.0	Z9C	1	0	7	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	5	0
12-918	F	1918		01	1940	208	1977	1	B6	0.0	Z9C	0	0	3	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
12-933	F	1923		01	1944	52	1979*	2	B3	0.0	Z9C	0	0	4	0
12-942	F	1898		01	1944	40	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
12-953	F	1920		01	1942	104	1979*	1	R3	0.0	Z9C	0	0	4	0
12-965	F	1924		01	1945	52	1977	2	R3	0.0	Z9C	0	0	5	0
12-967	F	1913		01	1953	12	1979*	0	B6	0.0	Z9B	0	0	0	0
12-977	F	1920		01	1943	7	1978	1	C6	0.0	Z9C	0	0	2	0
12-978	F	1919		08	1919	39	1976	0	B6	0.0	Z9B	0	0	0	0
12-981	F	1907		01	1921	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	13	0
12-985	F	1934		08	1934	39	1976	1	B6	0.0	Z9B	0	0	3	0
12-986	F	1932	1976	08	1932	39	1976	6	B3	0.0	Z9B	2	0	15	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BOBN	DIED	EXP	YEAR	EXP	YEAR	FA226	RA226	BA226	RA226	INPUT	INPUT	CUM	RADS
				TYPE	EXP	HKS	DEAS	NCI	ERR	RA226	ERR	RA226	RA226	RA226	RA226
13-002	F	1901		01	1923	462	1977	0	B6	0.0	Z9C	0	0	0	0
13-007	M	1911		01	1951	676	1976	1	B6	0.0	Z9E	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	1943	39	1979	0	B6	0.0	Z9	0	0	0	0
13-015	F	1910	1970	01	1954	884	1976	1	B6	0.0	Z9C	0	0	1	0
13-019	F	1915		01	1942	124	1977	0	B6	0.0	Z9B	0	0	0	0
13-021	F	1914		01	1942	104	1979*	0	B6	0.0	Z9B	0	0	0	0
13-022	F	1920		01	1942	69	1979*	0	C6	0.0	Z9C	0	0	1	0
13-025	F	1914		01	1940	32	1977	0	C6	0.0	Z9C	0	0	0	0
13-026	F	1921		01	1941	26	1977	0	C6	0.0	Z9C	0	0	0	0
13-027	F	1922		01	1942	155	1977	1	C6	0.0	Z9C	0	0	4	0
13-044	F	1954		07	1977	40	1977	0	B6	0.0	Z9C	0	0	0	0
13-050	M	1932		07	1977	40	1977	1	B6	0.0	Z9C	0	0	0	0
13-051	F	1978	1962	04	1925	+	0 1349	700	G4	0.0	Z9	145	0	1648	0
13-055	F	1908		07	1923	11	1978	0	B6	0.00800	Z2	0	0	0	0
13-056	M	1958		06	1976	52	1977	3	C6	0.0	Z9C	0	0	0	0
13-057	F	1922		07	1976	104	1978	0	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
13-059	M	1910		07	1933	2184	1978	1	B6	0.0	Z9B	0	0	1	0
13-063	F	1908		07	1933	1976	1978	0	B6	0.0	Z9B	0	0	0	0
13-064	F	1912		07	1959	102	1978	0	B6	0.0	Z9B	0	0	0	0
13-067	F	1917		01	1942	39	1978	0	B6	0.0	Z9B	0	0	0	0
13-071	F	1923		01	1942	78	1978	1	B6	0.0	Z9B	0	0	3	0
13-078	F	1908		07	1942	1300	1978	0	B6	0.0	Z9E	0	0	0	0
13-090	F	1921		07	1939	312	1978	0	B6	0.0	Z9B	1	0	0	0
13-082	F	1920		01	1942	52	1978	2	B6	0.0	Z9B	1	0	6	0
13-095	F	1918		07	1942	936	1978	0	B6	0.0	Z9B	0	0	0	0
13-087	F	1925		01	1942	8	1978	0	B6	0.0	Z9B	0	0	0	0
13-088	F	1922		01	1942	8	1978	0	B6	0.0	Z9B	0	0	0	0
13-089	F	1923		01	1942	104	1978	0	B6	0.0	Z9B	0	0	0	0
13-092	F	1917		07	1952	1106	1979*	0	B6	0.0	Z9B	0	0	0	0
13-102	F	1912		01	1928	936	1970*	4	B6	0.0	Z9	1	0	14	0
13-107	F	1904		17	1936	1920	1978	5	B3	0.0	Z9B	1	0	8	0
13-108	F	1907		17	1942	1612	1978	2	B6	0.0	Z9B	0	0	3	0
13-109	F	1910		01	1943	1	1970*	7	G6	0.0	Z9	2	0	20	0

TABLE 1 (CONT.) EXPOSURE DATA FOR RADIUM PATIENTS TO END OF 1979

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)
CASE	SEX	BORN	DIED	TYPE	EXP	YRPF	EXP	YFAR	FA226 METHOD + ERR	RA226 RATIO + ERR	FA226 METHOD + ERR	INPUT RA226 UCI	INPUT RA226 UCI	RA226 RADS	CUH FADS RA226
13-113	F	1906		01	1928	2080	1978	2	B6	0.0	Z9B	0	0	5	0
13-127	P	1914		07	1942	260	1978	1	B6	0.0	Z9B	0	0	3	0
13-132	F	1905		07	1932	1976	1978	3	B3	0.0	Z9B	1	0	6	0
13-136	F	1909		07	1942	130	1978	0	B6	0.0	Z9B	0	0	0	0
13-138	F	1907		07	1942	520	1979*	0	B6	0.0	Z9B	0	0	0	0
13-139	F	1922		01	1944	130	1978	4	B3	0.0	Z9B	1	0	10	0
13-145	P	1920		17	1937	468	1976	2	B6	0.0	Z9B	1	0	6	0
13-146	P	1921		01	1942	52	1978	1	B6	0.0	Z9B	0	0	3	0
13-147	F	1900		17	1939	204	1979*	1	B6	0.0	Z9B	0	0	3	0
13-151	F	1904		07	1927	036	1978	1	B6	0.0	Z9B	0	0	3	0
13-152	M	1901		07	1941	228	1978	3	C6	0.0	Z9C	1	0	6	0
13-153	M	1908		07	1939	1352	1978	1	B6	0.0	Z9B	0	0	1	0
13-154	P	1905		07	1941	1248	1978	0	B6	0.0	Z9B	0	0	0	0
13-158	F	1920		01	1944	52	1979*	1	B6	0.0	Z9B	0	0	3	0
13-161	P	1948		01	1969	8	1978	2	C6	0.0	Z9C	0	0	1	0
13-165	P	1917		01	1943	104	1979*	1	B3	0.0	Z9C	0	0	4	0
13-167	P	1928		07	1953	260	1979*	0	B6	0.0	Z9B	0	0	0	0
13-170	F	1923		01	1943	104	1979*	0	B6	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 50 bone sarcoma cases and 29 sinus or mastoid carcinoma cases among the 2223 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 84 measured persons considered to have radium-induced malignancies. 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.

Unmeasured Persons

Tables 3 and 4 list exposed persons with unknown or uncertain radium content who had probable or confirmed bone sarcomas and probable or confirmed paranasal sinus or mastoid carcinomas, respectively. There are 24 probable or confirmed bone sarcoma cases and 5 probable or confirmed sinus or mastoid carcinoma cases among the approximately 1400 radium cases with unmeasured body burdens for whom medical data are available. We have evidence that eight 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.

During the past year one person was added to the list of unmeasured bone-sarcoma cases and one was deleted, so that Table 3 contains the same number of

cases as the corresponding table in the 1979 annual report.¹ A copy of the death certificate for newly added case 05-534, obtained in 1979, indicated that the cause of her death in 1939 was a carcinoma (sic) of the right humerus of two years' duration. This person was a dial worker who mixed luminous paint at a plant in New York from 1917 to 1919.

Case 03-779 was deleted from the list of unmeasured bone-sarcoma cases after the exhumed remains were examined. Sections of a fascial sarcoma of the left thigh were examined microscopically six weeks prior to death in 1942, the pathologist concluding that "in all probability the tumor had arisen from the soft tissues, although there was a possibility of its having been osteogenic in origin." Radiographic examination of the skeletal remains in 1979 revealed mild radium changes in several bones and a large area of cortical erosion in the proximal left femur apparently attributable to pressure from the overlying soft-tissue neoplasm of the thigh. Because the remains did not support a diagnosis of bone malignancy, case 03-779 has not been included in the list of measured bone sarcoma cases. Exposure and dosimetric data for this case are listed in Table 1 of Appendix A of this report. The cumulative skeletal dose at the time of death was 2650 rad.

Reference

1. Radium-induced malignancies, Radiological and Environmental Research Division Annual Report, July 1978-June 1979, ANL-70-65, Part II, pp. 213-219

Table 1. Bone Sarcomas in Persons with Known Radium
Body Content as of 31 December 1979

CASE	SEX	B.C.F.N	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	9272	1960
01-562	F	1901	1931	1920	7143	1931
01-103	F	1903	1946	1922	7025	1946
00-023	F	1900	1929	1917	6928	1929
03-115	M	1896	1971	1925	6860	1957
01-031	F	1906	1934	1925	6824	1934
03-401	F	1900	1963	1923	6781	1962
01-011	F	1872	1937	1919	6678	1936
00-005	F	1901	1939	1917	6643	1939
05-953	F	1902	1978	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
03-118	F	1898	1955	1931	5159	1955
00-007	F	1903	1935	1919	5046	1934
00-027	F	1902	1942	1919	4995	1942
03-429	F	1908	1976	1923	4387	1967
01-051	F	1904	1977	1923	4265	1972
01-024	F	1901	1956	1916	4085	1956
03-234	F	1890	1965	1915	3810	1964
05-281	F	1898	1964	1916	3804	1956
03-402	F	1905	Live	1923	3761	1953

Table 1 (cont.)

<u>CASE</u>	<u>SEX</u>	<u>BORN</u>	<u>DIED</u>	<u>EXPOSED</u>	<u>CUM. RADS</u>	<u>DIAGNOSED</u>
01-179	F	1890	1966	1924	3642	1943
01-239	F	1901	1958	1917	3153	1955
01-520	F	1862	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
01-025	F	1886	1952	1924	2497	1950
03-212	F	1902	1951	1927	2412	1951
03-210	M	1906	1958	1926	2396	1956
01-613	F	1906	1936	1923	2319	1935
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	Live	1922	1445	1934
03-106	F	1876	1959	1931	1323	1957
01-439	F	1880	1953	1922	886	1949

Table 2. Carcinomas of the Paranasal Sinuses and Mastoid Air Cells in Persons with Known Radium Body Content as of 31 December 1979

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	1979	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	7656	1974
01-014	F	1901	1949	1916	6799	1949
03-676	F	1897	1977	1924	6433	1976
01-179	F	1890	1966	1924	6319	1965
03-425	F	1908	1976	1923	4785	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

^aCarcinoma 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
00-030	F	1903	1924	1918	1923
00-031	F	1903	1940	1920	1938
00-035	F	1900	1941	1917	1941
01-088	F	1906	1931	1923	1931
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
03-800	F	1908	1945	1924	1944
03-806	F	1896	1956	1922	1956
03-848	F	1903	1958	1922	1958
05-534	F	1897	1939	1917	1937
05-987	F	1901	1962	1918	1962
09-087	M	1891	1934	1912	1933

^aAll were dial painters except cases 01-387 (iatrogenic, i.v. and per os), 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

^aAll were dial painters.

^bDeath certificate lists paranasal sinus carcinoma as cause of death; histologic diagnosis from biopsy tissue was rhabdomyosarcoma of the maxillary antrum.

PUBLICATIONS BY THE STAFF OF THE CENTER FOR HUMAN RADIOBIOLOGY
FOR THE PERIOD JULY 1979 THROUGH JUNE 1980

- C. G. Cacic, R. E. Toohey, R. D. Oldham, and R. P. Larsen, The concentration of plutonium in hair following intravenous injection, Twenty-Fourth Annual Meeting of the Health Physics Society, Philadelphia, PA, July 8-13, 1979; Health Phys. 37, 820 (December 1979).
- M. J. Harris and R. A. Schlenker, A histological basis for dosimetry in radium-induced head carcinomas, Twenty-Fourth Annual Meeting of the Health Physics Society, Philadelphia, PA, July 8-13, 1979; Health Phys. 37, 808 (December 1979).
- M. J. Harris, Morphometry of the mucous membranes and the human paranasal sinuses and mastoid cavities, 93rd Annual Meeting of the American Association of Anatomists, Omaha, Nebraska, April 28-May 1, 1980; Anat. Rec. 196, 73-74a (March 1980).
- C. B. Henning and E. L. Lloyd, An electron microscope study of a fibrosarcoma and associated bone cells in a radium dial painter, 37th Annual Meeting of the Electronmicroscopy Society of America, San Antonio, Texas, August 13-17, 1979, pp. 218-219.
- R. B. Holtzman, P. W. Urnezis, A. Padova, and C. M. Bobula III, Contamination of the human food chain by radionuclides from uranium mill tailings — Progress report, Twenty-Fourth Annual Meeting of the Health Physics Society, Philadelphia, PA, July 8-13, 1979; Health Phys. 37, 850-851 (December 1979).
- A. T. Keane, R. B. Holtzman, and R. A. Schlenker, A review of data on the retention and distribution of radium in human soft tissues, Twenty-Fourth Annual Meeting of the Health Physics Society, Philadelphia, PA, July 8-13, 1979; Health Phys. 37, 819 (December 1979).
- E. L. Lloyd, M. A. Gemmell, C. B. Henning, D. S. Gemmell, and B. J. Zabransky, Transformation of mammalian cells by alpha particles, Int. J. Radiat. Biol. 36, 467-478 (1979).
- L. F. Mausner, M. A. Moinester, J. D. Knight, S. Cochavi, and S. Friedland, In vivo beam localization by positron activation in pion therapy, Radiat. Res. 80, 10-23 (1979).
- L. F. Mausner, R. A. Schlenker, and B. G. Oltman, Spectrometry applied to bone surface alpha dosimetry, Twenty-Fourth Annual Meeting of the Health Physics Society, Philadelphia, PA, July 8-13, 1979; Health Phys. 37, 808-809 (December 1979).

- R. A. Naumann, G. Schmidt, J. D. Knight, L. F. Mausner, C. J. Orth, and M. E. Schillaci, Negative muon capture ratios for alloys and alkali halide solid solutions, *Phys. Rev. A* 21, 639 (February 1980).
- A. P. Polednak and J. E. Farnham, Osteometry and autoradiography of the long bones of skeletons of women exposed to internal radiation at 13-19 years of age, *Am. J. Phys. Anthropol.* 52, 335-340 (1980).
- R. E. Rowland, The risk of bone sarcoma from plutonium-239, Biological Implications of Radionuclides Released from Nuclear Industries, Proc. Symp., Vienna, March 26-30, 1979, Vol. II, Int. Atomic Energy Agency, Vienna, pp. 211-224 (1979).
- R. E. Rowland, Statement of R. E. Rowland (regarding radium and barium in drinking water), Safe Drinking Water — Oversight, Hearing before the Subcommittee on Health and the Environment of the Committee on Interstate and Foreign Commerce, House of Representatives, Ninety-Sixth Congress, November 5, 1979, U.S. Government Printing Office, pp. 117-129 (1980).
- J. Rundo, F. Markun, N. J. Plondke, and D. J. Keefe, Delinquent radon daughters in the home, Twenty-Fourth Annual Meeting of the Health Physics Society, Philadelphia, PA, July 8-13, 1979; *Health Phys.* 37, 801 (December 1979).
- J. Rundo, F. Markun, and N. J. Plondke, Some determinations at Argonne National Laboratory of radon in houses, Radon in Buildings, NBS Publication 581, U.S. Department of Commerce, National Bureau of Standards, Washington, D.C., p. 63 (1980) (abstract).
- R. A. Schlenker and M. J. Harris, Dosimetry of head carcinomas in radium cases, Twenty-Fourth Annual Meeting of the Health Physics Society, Philadelphia, PA, July 8-13, 1979; *Health Phys.* 37, 808 (December 1979).
- R. A. Schlenker, Dosimetry of paranasal sinus and mastoid epithelia in radium-exposed humans, Twenty-Eighth Annual Meeting of the Radiation Research Society, New Orleans, LA, July 1-5, 1980; *Radiat. Res.* 83, 401-402 (August 1980).
- C. S. Serio, C. B. Henning, and E. L. Lloyd, Mitogenic stimulation of peripheral lymphocytes from radium workers, Federation of American Societies on Experimental Biology, Mtg., Anaheim, CA, April 4-8, 1980; *Fed. Proc.* 39, 909 (March 1980).
- H. Spencer, Sr. M. C. Rusin, R. B. Holtzman, and L. Kramer, Metabolic balances of cadmium, copper, manganese, and zinc in man, *Am. J. Clin. Nutr.* 32, 1867-1875 (September 1979).

- A. F. Stehney, A. P. Polednak, J. Rundo, A. M. Brues, H. F. Lucas, Jr., B. C. Patten, and R. E. Rowland, Health status and body radioactivity of former thorium workers. Interim report, Argonne National Laboratory Report ANL-80-37, NUREG/CR-1420 (January 1980).
- K. L. Swithin, W. J. Bair, P. N. Dean, J. Rundo, and F. K. Tomlinson, Status and trends in the external counting of inhaled heavy elements deposited in vivo, Health Phys. 37, 641-657 (November 1979).
- R. E. Toohey, R. E. Walkup, R. P. Larsen, and R. D. Oldham, Urinary excretion rate of ^{241}Am produced in vivo, Twenty-Fourth Annual Meeting of the Health Physics Society, Philadelphia, PA, July 8-13, 1979; Health Phys. 37, 820 (December 1979).
- R. E. Toohey and M. A. Essling, Measurements of ^{241}Am in vivo at long times after inhalation, Health Phys. 38, 139-145 (February 1980).
- R. E. Toohey, M. A. Essling, and D. R. Huff, Retention and gross distribution of ^{75}Se following intravenous injection of ^{75}Se -selenomethionine, Health Phys. 37, 395-397 (September 1979).

PAPERS ACCEPTED FOR PUBLICATION

- E. E. Adams and A. M. Brues, Breast cancer in female radium dial workers first employed before 1930, J. Occup. Med.
- A. M. Brues, The long-term follow-up of radium dial painters and thorium workers, Int. Conf. on the Medical Basis for Radiation Accident Preparedness, Oak Ridge Associated Universities, Oak Ridge, Tennessee, October 18-20, 1979.
- M. J. Harris and R. A. Schlenker, Quantitative histology of the accessory nasal sinuses and mastoid cavities, Ann. Otol., Rhinol., and Laryngol.
- R. B. Holtzman, Normal dietary levels of ^{226}Ra , ^{228}Ra , ^{210}Pb , and ^{210}Po for man, Proc. Conf. on the Natural Radiation Environment, III, Houston, Texas, April 24-28, 1978.
- A. P. Polednak, Fertility of women after exposure to internal and external radiation, J. Environ. Pathol. Toxicol.
- A. P. Polednak, Thyroid tumors and thyroid function, J. Environ. Pathol. Toxicol.
- J. Rundo, Radioactivity in man: Levels, effects, and unknowns, Public Meeting on Federal Research into Biological Effects of Ionizing Radiation, National Institutes of Health, Bethesda, Maryland, March 10-11, 1980.
- C. S. Serio, C. B. Henning, R. E. Toohey, and E. L. Lloyd, Mitogenic stimulation of peripheral lymphocytes of radium workers, Int. J. Radiat. Biol.

R. E. Toohey, C. G. Cacic, R. P. Larsen, and R. D. Oldham, The concentration of plutonium in hair following intravenous injection, Health Phys.

Seventh Symposium on Microdosimetry, Oxford, England, Sept. 8-12, 1980

J. H. Marshall and A. Pagnamenta, Toward a theory of the initiation of cancer by ionizing radiation

A. Pagnamenta and J. H. Marshall, Track structure analysis for electrons and alpha particles in water

Workshop on Measurement and Interpretation of Actinide Accumulation by Man, Snowbird, Utah, Oct. 14-18, 1979

R. P. Larsen, R. D. Oldham, M. H. Bhattacharyya, E. S. Moretti, and D. J. Austin, On the gastrointestinal absorption of plutonium

R. P. Larsen, R. D. Oldham, and R. E. Toohey, Macrodistribution of plutonium in the human skeleton

J. Rundo, The late excretion of plutonium following acquisition of known amounts

J. Rundo, D. R. Brewster, M. A. Essling, and J. Y. Sha, Radioactivity in former workers at a thorium refinery

R. A. Schlenker and B. G. Oltman, Plutonium microdistribution in human bone

R. A. Schlenker and B. G. Oltman, Uranium concentrations in human bone

R. E. Toohey, The distribution of ^{241}Am in the human body as determined by external counting

PAPERS SUBMITTED FOR PUBLICATION

R. P. Larsen, R. D. Oldham, M. H. Bhattacharyya, E. S. Moretti, and D. J. Austin, Gastrointestinal absorption of plutonium

E. L. Lloyd and C. B. Henning, Cells at risk for the production of bone tumors in radium exposed individuals: An electron microscope study