FACT SHEETS
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ARGONNE
NATIONAL
LABORATORY

INTRA-LABORATORY MEMO

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November 6, 1981

TO: All CHR staff, temporary employees and consultants

FROM: J. Rundo

SUBJECT: Fact Sheets

I attach for your retention a copy of each of the eleven Fact Sheets prepared by CHR staff for the AUA Review Committee members. They provide useful summaries of many of the facets of our work.

Many thanks to those who prepared these, and also to those who provided me with information and slides for my overview of the Center's work. Drs. Matanosky and Sagan have been very interested in all that they have seen and heard.

lw

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CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Late Excretion Rates of ^{226}Ra Following Occupational or Iatrogenic Exposure
(Total effort 1.5 p-y)

Knowledge of the excretion rates of ^{226}Ra from persons with significant amounts of this nuclide is needed for evaluation of their radiation doses. These data are useful in establishing the parameters of the nuclide retention functions used in estimation of body content of and doses to contaminated subjects over long periods of time.

Previous studies have reported some excretion rate values, but to obtain a more complete picture of the metabolic parameters we have determined the ^{226}Ra excretion rates for a larger and more diverse group of subjects with long-term ^{226}Ra body contents.

The urinary and fecal excretion rates of ^{226}Ra were determined for 53 subjects: 25 had been radium dial painters, 16 were iatrogenic (medical) cases and 12 were former radium chemists. Data on the subjects, such as body content of ^{226}Ra, and times and dates of exposure, were obtained from the CHR annual report.[1] The time between first exposure and sample collection ranged from 20 to 60 yr with a mean of 40 yr, and the body content of ^{226}Ra at the time of collection ranged from 11 to 8600 nCi.

The mean coefficients of elimination, CE (fraction of the body content excreted annually), are shown in the table. That of the dial painters of 2.23% per yr was

Mean coefficients of elimination (unweighted), % ^{226}Ra content/yr ± SE

Group	Sex	Urinary(U)	Fecal(f)	Total[a]	Ratio, U/F
Dial Painters	25F	0.11 ± 0.02	2.12 ± 0.39	2.23	0.052 ± 0.013
Medical Cases	7M, 9F	0.025 ± 0.003	0.95 ± 0.10	0.98	0.026 ± 0.004
Radium Chemists	9M, 3F	0.067 ± 0.012	1.23 ± 0.23	1.30	0.054 ± 0.014
				Weighted mean	0.030 ± 0.007

[a] The SE values for these results are essentially identical to those for the feces.

significantly greater than those of the medical cases and chemists, 0.98 and 1.30% per yr, respectively. The higher value for the dial painters may be due to the existence of two populations, a large one with values below 3.5% per yr and a smaller one with values above about 3.9% per yr. It could also be a single population with a broad lognormal distribution as shown in the diagram overleaf.

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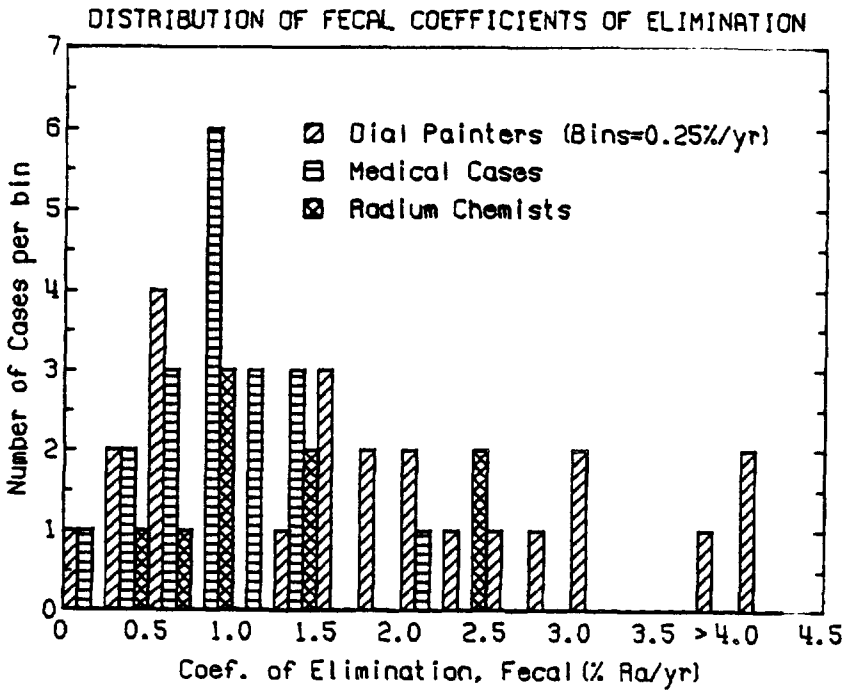

These values of CE correspond to instantaneous values for the dial painters, medical cases and chemists, respectively of 0.92, 0.41 and 0.34 for the exponent, b, in the power function retention equation, $R = At^{-b}$. The first of these values differs significantly from the Norris value of 0.52. The mean ratio of urinary-to-fecal excretion rates was (3.0 ± 0.7)% (± S.E.).

The results of the measurements made here are useful in the dosimetry of the radium patients in that the parameter b in the power function equation may be obtained for a given individual from the CE. This value is similar to that derived from serial whole body counting measurements made over 10 to 15 yrs.

Reference

1. Center for Human Radiobiology, Appendix A, Exposure data for radium patients, in Radiological and Environmental Research Division Annual Report, July 1979-June 1980, ANL-80-115, Part II, pp. 184-251.

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CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Inhalation Exposures at Lindsay Chemical Company
(Total effort 1.5 p-y)

The Center for Human Radiobiology is currently engaged in an assessment of possible health effects of employment in the thorium refining industry. An important part of this study is the determination of thorium and rare earth exposure conditions at a thorium refinery located in West Chicago, Illinois, and the resultant uptake in the workers.

The West Chicago plant was operated by the Lindsay Light Company and its corporate successors Lindsay Chemical Company and Kerr-McGee Chemical Corporation from 1932 until 1973. It is presently an empty shell being stripped and decontaminated for eventual demolition. Fortunately we have obtained approximately 10,000 air filter samples which were collected by Lindsay from 1962-1973. The sampling date and location (i.e. floor and building) of each filter are known. We have also obtained the alpha counting records and airborne thorium calculations corresponding to each filter. In theory these records provide a survey of the time and location distribution of airborne thorium and in some cases thoron as well. However, some unjustified assumptions gave calculated values of thorium and especially thoron which are much too low.

We have data from over 12,000 air filter samples which were alpha counted four times after filtration, at 1 hr, 10.6 hr, 106 hr and 465 hr. Using a more realistic model for radioactive growth and decay than did Lindsay and new data on the characteristics of the filters, we have calculated the concentrations in the air of thorium, thoron, lead-212 and bismuth-212. The results for more than 12,000 of these filters have been stored in a computer file.

Lindsay Chemical reported the concentrations of lead-212 as thoron on the assumption that thoron and lead-212 in the air were in equilibrium. Because of ventilation it is very likely that the thoron levels were much larger. Using the bismuth-212 to lead-212 ratios, we calculated an "effective age" of the air and then calculated the proper thoron levels. These were typically 20 times higher than previously reported. In addition, our values for bismuth-212 and for thorium are typically 15% and 35% higher, respectively.

The airborne radioactivity levels were particularly high on the third floor of building 9. For example in 1964 the geometric means of airborne radioactivity concentrations (32 filters) for thorium, thoron, lead-212, and bismuth-212 were 0.058, 4100, 203, and 60 pCi/L, respectively. These values can be compared to the maximum permissible concentrations[1] in air of these isotopes of 0.03, 300, 20, and 100 pCi/L, respectively. Thus the workers involved here were exposed to concentrations of thorium, thoron and lead-212 well in excess of present limits. Although high thorium levels were also reached in other years we do not have enough information to determine the thoron and daughter concentrations.

Since the plant also produced rare earth chemicals (in fact almost exclusively in its last years), we have measured the airborne rare earth concentrations. We have utilized energy dispersive x-ray fluorescence spectrometry to determine simultaneously lanthanum, cerium, praseodymium, and neodymium directly on the

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air filter samples. To date we have analyzed a total of 14 filters from 1967 and 1970. The relative amounts were usually in the order La > Ce > Nd > Pr. The airborne concentrations of 44-340, 25-680, 25-76, and 32-330 ng/L for La, Ce, Pr, and Nd, respectively, were 3 to 4 orders of magnitude greater than those of the respective elements in urban aerosols of 5, 10, 3, and 6 pg/L.[2] The rare earth contents of the filters did not correlate with the radioactivity measurements. Thus we are probably looking at different types of dusts.

The concentrations of thorium-232 were determined in the tissues of two workers in this plant from whom we had obtained tissues from autopsy, and the concentrations of rare earth elements were determined in the tissues of one of these subjects, as shown in the table. The thorium levels in case L-2107 with a 33-yr exposure history were substantially greater than those of the other case, L-2932, with only a 3-yr exposure. Both were higher than the control levels, except for the liver and kidney of the latter subject. The levels of La and Ce in lung and kidney appear to be very high relative to those for control samples.[3]

Concentrations in Tissues (ppm)

Case (yrs exp)	Element	Lung	Lymph Nodes	Liver	Spleen	Kidney
L-2107 (33)	La	8.56 ±0.88		3.53 ±0.39	3.65±0.37	0.041 ±0.007
	Ce	39.4 ±2.1		9.41 ±0.52	10.0 ±1.8	0.096 ±0.025
	Sm	0.9 ±0.25		0.15 ±0.04		
	Nd	7.1 ±2.1		1.7 ±0.5		
	^{232}Th	6.18 ±0.30*	17.94 ±0.91	0.22 ±0.01*	1.23 ±0.05*	0.016 ±0.003*
L-2932 (3)	^{232}Th	0.085±0.005	0.93 ±0.06	0.0035±0.0011		0.0019±0.0006
Controls+	^{232}Th	0.004	0.079	0.001	0.001	0.002

*Multiple samples

+Data from Ref. 4

References

1. U. S. Nuclear Regulatory Commission, Rules and regulations, Title-10-Chapter 1, Code of Federal Regulations, Part 20, Standards for protection against radiation, U. S. Government Printing Office, Washington, D.C. (codified and reissued September 1978).

2. J. A. Cooper, Battelle Northwest Laboratories Report BNWL-SA-4690.

3. H. J. M. Bowen, Environmental Chemistry of the Elements (Academic Press, London, 1979), pp. 103-104.

4. M. E. Wrenn, N. P. Singh, S. A. Ibrahim, N. Cohen and G. Saccomanno, Thorium in human tissues, in Proc. of the Natural Radiation Environment Meeting III, Houston, Texas, April 23-28, 1975, p. 783.

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CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Alpha-Particle Doses to Soft Tissues from Internally-Deposited ^{226}Ra and ^{228}Ra

(Total effort 0.5 p-y)

An excess of breast cancer has been described among female radium dial workers. The tumor incidence appears to vary with total radium intake to blood but not with the duration of exposure. This suggests that internal rather than external radiation is the principal carcinogen and has stimulated interest in soft tissue dosimetry.

Alpha-particle doses in a 50-yr period following a single systemic intake of ^{226}Ra or ^{228}Ra have been estimated for 31 organs and tissues of the adult human. The dose estimates were derived primarily from the ICRP alkaline earth model[1] fitted to data on retention of ^{226}Ra in soft tissues and bone,[2] combined with reported ratios of ^{226}Ra to calcium in soft tissues and bone at natural levels[3] and the distribution of calcium in the tissues of Reference Man.[4]

The median of the 31 organ and tissue doses from the alpha-particles of ^{226}Ra itself is 0.08 rad per μCi systemic intake. An additional average dose of 0.01 rad per μCi ^{226}Ra systemic intake is delivered by ^{226}Ra daughters produced in soft tissue or transferred from bone to soft tissue. Soft-tissue doses from alpha particles of the ^{228}Ra decay series are about six times those from ^{226}Ra alpha particles for equal systemic intakes of ^{226}Ra and ^{228}Ra, with the assumption that ^{228}Ra daughter products do not transfer from the organ in which they are produced.

Alpha-particle doses to the intestinal mucosa from ^{226}Ra and ^{228}Ra decay series activity in the contents of the gastrointestinal tract and to the red marrow of bone from activity in trabecular bone were estimated with the use of ICRP dosimetric models.[5]

Dose estimates for several of the organs and tissues are listed in the table on the reverse side. For subjects with low occupational intakes of radium acquired over lengthy employment periods, the more significant soft-tissue doses probably resulted from exposure to external radiation and from inhalation of ^{222}Rn and its short-lived decay products. The soft-tissue dosimetry of the occupational environment and that of the internally-emitted gamma rays and beta particles of the ^{226}Ra and ^{228}Ra decay series are currently being assessed.

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Organ or Tissues	Fifty-yr alpha-particle dose per μCi systemic intake. ^{226}Ra (rad)	^{228}Ra (rad)
Eye	0.66	3.8
Red Marrow	0.55	1.1
Thyroid	0.34	2.0
Intestinal Mucosa		
ULI (ingestion)	0.040	0.048
(injection)	0.003	0
LLI (ingestion)	0.12	0.14
(injection)	0.009	0
Intestine, whole		
ULI	0.11	0.63
LLI	0.076	0.44
Ovary	0.082	0.45
Testis	0.046	0.24
Breast	0.045	0.15
Pancreas	0.039	0.20
Total Soft Tissue	0.045	0.21

References

1. International Commission on Radiological Protection, Alkaline Earth Metabolism in Adult Man, ICRP Publication 20, Pergamon Press, Oxford (1973).

2. Schlenker, R. A., Keane, A. T., and Holtzman, R. B., The retention of ^{226}Ra in human soft tissue and bone: Implications for the ICRP alkaline earth model, Health Physics (in press).

3. Rajewsky, B., Belloch-Zimmerman, U., Löhr, E., and Stahlhofen, W., ^{226}Ra in human embryonic tissue, relationship of activity to the stage of pregnancy, measurement of natural ^{226}Ra occurrence in human placenta, Health Phys. 11, 161 (1965).

4. International Commission on Radiological Protection, Report of the Task Group on Reference Man, ICRP Publication 23, Pergamon Press, Oxford (1975).

5. International Commission on Radiological Protection, Limits for intakes of radionuclides by workers, ICRP Publication 30, Part I, Pergamon Press, Oxford (1979).

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CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Studies of the Gastrointestinal Absorption of Plutonium in Mammals
(Total effort 2.0 p-y)

To a major degree, the gastrointestinal absorption factor, f_1, for plutonium (and hence the MPC for plutonium in drinking water) is based on the results of a comprehensive investigation by Weeks et al.[1] in which solutions of Pu(IV) were administered to rats for nearly a year. The plutonium concentration ranged from a factor of 10 lower than the present MPC to a factor of 10^5 higher than MPC; the medium was 0.01 M nitric acid. The mean of their values for percent absorption was 0.003%.

In our investigation of the G.I. absorption of plutonium, single administrations have been made of solutions whose weight concentrations were the same as that for ^{239}Pu at MPC, 8×10^{-11} g/mL (5 pCi/mL). The medium has been 0.01 M sodium bicarbonate solution (which simulates many natural waters with respect to both anionic composition and pH), and the plutonium has been in either the VI state (to simulate chlorinated drinking water[2]) or in the IV state (to simulate unchlorinated drinking water). Other media have been citrate buffer and 0.01 M nitric acid. These were used to obtain absorption values for Pu(IV) that could be compared with those obtained by earlier investigators. The animal used in most of our studies has been the mouse, but studies have also been made with the rat (to ensure comparison with other rat data) and the dog (to obtain values for a larger animal). The values we have obtained for plutonium absorption are presented in the following table along with those obtained by Weeks et al.

Gastrointestinal Absorption of Plutonium in Mice, Rats, and Dogs

Medium	Animal	Pu Oxid. State	Feeding Regimen	No. of Adms.	Conc., g/mL	Absorp., %
			This Study			
0.01 M $NaHCO_3$	Mouse	VI	Fasted	Single	8×10^{-11}	0.15 ± 0.03
0.01 M $NaHCO_3$	Mouse	IV	Fasted	Single	8×10^{-11}	0.20 ± 0.02
0.01 M HNO_3	Mouse	IV	Fasted	Single	8×10^{-11}	0.17 ± 0.03
0.1 M citrate	Mouse	IV	Fasted	Single	8×10^{-11}	0.24 ± 0.05
			Weeks et al.			
0.01 M HNO_3	Rat	IV	Fed	~400	10^{-12}-10^{-6}	0.003± 0.001
0.01 M HNO_3	Rat	IV	Fasted*	Single	5×10^{-4}	0.003± 0.001
0.1 M HNO_3	Rat	IV	Fasted*	Single	5×10^{-4}	0.30 ± 0.10
0.1 M citrate	Rat	IV	Fasted*	Single	5×10^{-4}	0.30 ± 0.10
0.1 M HNO_3	Rat	VI	Fasted*	Single	5×10^{-4}	1.7 ± 0.5
			This Study			
0.01 M $NaHCO_3$	Rat	VI	Fasted	Single	8×10^{-11}	0.32 ± 0.04
0.1 M citrate	Rat	IV	Fasted	Single	8×10^{-11}	0.38 ± 0.06
0.01 M $NaHCO_3$	Dog	VI	Fasted	Single	8×10^{-11}	0.067± 0.014

*This is based on discussions of these experiments that were held with one of the authors (R. C. Thompson). The text of the paper does not provide this information.

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There were two possible explanations for the difference between the values obtained by Weeks et al. and by ourselves: (1) either the animals in all their experiments were fed while ours were fasted, or (2) their animals that received single administrations were fasted and the Pu(IV) in their 0.01 M nitric acid solutions had inadvertently become polymeric. The first explanation would also require that the absorption of plutonium be dependent on feeding regimen when the medium is 0.01 M nitric acid, but not dependent on it when the medium is either 0.1 M nitric acid or citrate buffer. With respect to the second explanation Weeks et al. provided no information to indicate whether or not the plutonium in their solutions was monomeric or polymeric; all of our solutions were tested to ensure that they were monomeric.

In our study of the effect of feeding regimen, a solution of Pu(VI) in 0.01 M $NaHCO_3$ was administered to mice that were fed ad libitum. The absorption of plutonium was 0.015 ± 0.001%. This is a factor of about 10 lower than our values for fasted mice but a factor of about 5 higher than the values obtained by Weeks et al. in their long-term study. Sullivan et al.[3] have recently reported absorption values of 0.45 and 0.024% in fasted and fed rats, respectively, when a 0.0005 g/mL Pu(VI), 0.01 M nitric acid solution was administered. In our study of the properties of a 1×10^{-6} g/mL Pu(IV), 0.01 M nitric acid solution (the highest concentration used in the long-term experiments of Weeks et al.), it was found that 45% was polymeric (as established by ultrafiltration) at the time of solution preparation, despite the care taken to avoid this. Over a period of 200 days the degree of polymerization decreased from 45 to 13%, the percentage of Pu(VI) (from the oxidation of Pu(IV) by atmospheric oxygen) increased from 3% to 48%, and the G.I. absorption in fed mice increased from 0.0045% to 0.013%.

From the data we have obtained in this investigation, it appears that (1) the absorption of plutonium in fasted mice is independent of both its oxidation state and the administration medium, but is reduced by a factor of about 10 when the mice are fed, and (2) the values obtained by Weeks et al. for 0.01 M nitric acid solutions were for solutions of polymeric rather than monomeric plutonium. In light of our data, and the fact that the consumption of drinking water and food are frequently not concurrent, it would appear that the present value for the gastrointestinal absorption factor of plutonium in man, 0.01% (as adopted by the ICRP), is a factor of about 20 too low. A paper containing all the information when using rodents (save that on the effect of feeding) was published recently.[4] A paper on plutonium absorption in dogs has been submitted for publication.

References

1. M. H. Weeks et al., Radiat. Res. 4, 339 (1956).

2. R. P. Larsen and R. D. Oldham, Science 201, 1008 (1978).

3. M. F. Sullivan, Radiat. Res. 80, 116 (1979).

4. R. P. Larsen et al. Radiat. Res. 87, 37 (1981).

0019802

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CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Theory of the Induction of Bone Cancer by Ionizing Radiation

(Total effort 1.5 p-y)

In our attempt to find a model of bone cancer induction which is consistent with known data, we have extended the Marshall-Groer model of 1977 (two initiating events in one cell) to the level of the DNA molecule in order to test for consistency with fundamental mechanisms of ionization and deletion of information.

Extension to the level of DNA suggests that we calculate the probability of clusters of ionizations in or near the molecule sufficient to disrupt it. Extending a bookkeeping technique developed by Wideroe and using the method developed by Kim for extrapolation from existing data for ionization cross section in water vapor, we have developed an electron delta ray tree down to fourth order which matches the Combecher data for energy per ion pair. We have derived a degradation spectrum for all energies of incident electrons; the curve for 10 kev electrons agrees with the curve from Paretzke's Monte Carlo analysis.

The alpha particle tree involves consideration of the changing charge state of the alpha due to pickup and loss of electrons as it slows down, the stripping of the electron from the He^{+} state, the convoy electrons (electrons moving at almost the same velocity as the alpha after a collision with a water molecule), as well as the hard Rutherford collisions and the soft dipole collisions. A new set of ionization cross section data for He^{++} and He^{+} ions incident on water vapor was given to us by Toburen, Wilson and Popovich. Also, a new set of data on energy per ion pair has been received from a German group (R. Huber) which extends the energy region downwards by an order of magnitude.

We are now generating a more accurate alpha particle track structure from these new data. First by fitting the different processes, hard, soft, convoy and Auger electrons to the He^{++} data, then by fitting the He^{+} data by addition of a stripping term, we have obtained representative fits to all the data. We obtain the energy dependence of the charge equilibria from Armstrong's data. This gives us an effective cross section for ionization as the alpha slows down. We have computed new values for the number of ionizations per KeV per micron. The higher orders are under consideration pending the numerical evaluation of some near-singular integrals for which we have designed special methods.

We would like to acknowledge the continuing help provided us by Mitio Inokuti and Yong-Ki Kim of the Fundamental Molecular Physics and Chemistry section of RER.

Prepared by J. H. Marshall and A. Pagnamenta Argonne National Laboratory

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CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Dosimetry of ^{224}Ra in Bone
(Total effort 0.4 p-y)

Introduction

Since the early 1940's, intravenous injections of ^{224}Ra have been used in Europe as a palliative treatment of ankylosing spondylitis and bone tuberculosis. Several thousand patients have been treated and a number of them have contracted osteogenic sarcoma. Because ^{224}Ra is an alpha emitter which decays predominantly on bone surfaces, it has been considered an analog for ^{239}Pu, which behaves in much the same way. A major effort has been made by German scientists to establish the medical history of the subjects following their treatment, and recognition of the carcinogenic side effects has brought a significant reduction in the amounts injected.

Despite extensive knowledge of injection levels, the dosimetry is quite uncertain. This stems from the fact that ^{224}Ra heads a lengthy decay chain which ends in ^{208}Pb. Proper dosimetry of the target cells requires knowledge, not only of ^{224}Ra retention but also of the daughter product retention. Fortunately, attention can be focused on just two daughters, ^{220}Rn and ^{212}Pb, because they determine, to a large extent, the retentions of all other daughters.

In collaboration with the Radiobiology Laboratory, University of Utah, a study of ^{220}Rn and ^{212}Pb retention on bone surfaces of young adult beagle dogs has been undertaken to gain a better understanding of ^{224}Ra dosimetry.

Three dogs were each injected with about 100 μCi ^{224}Ra nearly in equilibrium with its daughters. Another dog was injected with about 100 μCi of ^{226}Ra and a fifth dog was injected with a similar amount of ^{212}Pb. All five dogs were sacrificed 24 hours after injection. Retention was studied by direct alpha spectrometry of the endosteal bone surfaces under vacuum.

^{220}Rn

Because of its 56-second half-life, the ^{220}Rn retained in life quickly disappears after death and only the retention in vitro can be determined. However, a rule exists, supported by numerous data, that radon retention by bone in vivo never exceeds that in vitro. Data from our ^{226}Ra dog exemplify this. The fractional retention of ^{222}Rn in vivo at the endosteal surfaces of 7 bone samples ranged from 0.026 to 0.035; after 13 days in vacuum, ^{222}Rn activity ranged between 0.29 and 0.52 of the ^{226}Ra activity, an increase by an order of magnitude. The initial fractional retention of ^{220}Rn for 7 bone samples under vacuum ranged between 0.4 and 0.7 and averaged 0.6. Using the above rule, these values are interpreted as upper limits to the retention in vivo.

^{212}Pb

The bone surface activity of ^{212}Pb is determined from the activity of its alpha-emitting granddaughter, ^{212}Po. For the 7 bone samples, $^{212}Pb/^{224}Ra$ at death

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ranged from 0.3 to 1.1 and averaged 0.5.

Little bone surface activity was found in the ^{212}Pb dog; therefore, the substantial amounts of ^{212}Pb on the samples from the ^{224}Ra dog result from decay of ^{224}Ra in vivo rather than deposition of injected ^{212}Pb.

Dosimetry

Until now, it has been common to assume that ^{224}Ra daughter products are completely retained on bone surfaces. This assumption is proven incorrect by the results of our experiment. Using the average values found for fractional retention, the alpha particle energy released at bone surfaces per disintegration of ^{224}Ra is about 14 MeV or just a little more than one-half of the energy which would be released if the ^{224}Ra daughter products were completely retained. Endosteal cell dose rates would also be about one-half those for complete retention. This result will have a significant impact on the calculation of dose for human ^{224}Ra patients.

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CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Dosimetry of Mastoid Epithelium
(Total effort 2.0 p-y)

Carcinomas arising from the mucous membranes of the paranasal sinuses and mastoid air cells are a well-known late effect of radium deposition in humans. The tumors are produced by alpha particle bombardment from two locations on opposite sides of the epithelium: bone, the site of radium deposition, and the air spaces, where radon gas accumulates. Mastoid carcinomas outnumber sinus carcinomas 18 to 11 and, because of structural differences between the mastoids and sinuses, mastoid dosimetry is the more tractable problem. It has been the focus of our attention recently.

Because humans are exposed to a variety of bone-seeking radionuclides with no gaseous daughter products, it is essential to determine the relative importance of irradiation from the air spaces and irradiation from bone. The ratio of dose rates,

$$\frac{D_{bone}}{D_{air}} = \left(\frac{C_{Ra}^{bone}}{C_{Rn}^{air}}\right)\left(\frac{S_{bone}}{S_{air}}\right)$$

is directly proportional to the ratio of radium and radon concentrations. The value of C_{air}^{bone} shortly before death can be determined by post-mortem autoradiographic measurements on mastoid bone but there is no practical way to measure the concentration in vivo of air space radon. Therefore a model of radon accumulation has been formulated for the prediction of radon concentration, C_{Ra}^{air}.

The factors S_{bone} and S_{air} are proportional to the doses delivered per alpha particle emitted in bone and air space, respectively. They include the effects of energy absorption by tissue through which the alpha particle must pass to reach the epithelium, the limitation on dose imposed by the smallness of alpha particle ranges (30-80 μm in tissue) and the effect of air cavity size. With the knowledge of mucosal structure gained from histomorphometry of the mastoid in our laboratory, S_{bone}/S_{air} has been calculated.

Steady State Model

The model is designed to predict radon concentrations in steady state. The time dependence of C_{Rn}^{air} during the approach to steady state has been ignored. The mastoid air cells with their bony septa and the surrounding bone cortex are assumed to constitute a closed system bounded by reflecting walls. Radon is continuously generated by radium decay and is continuously swept away by blood. With a time invariant radium concentration the amount of radon in the system assumes a steady state value equal to the rate of production divided by the rate of removal. This amount is partitioned between bone and air space according to Henry's Law for the partial pressure of a solvent in the gas phase above a solution. The model provides an equation for the ratio $C_{Ra}^{bone}/C_{Rn}^{air}$. With values for C_{Ra}^{bone} measured by autoradiography, values for C_{Rn}^{air} may be obtained, a few examples of which are presented in

Prepared by R. A. Schlenker 0019806 Argonne National Laboratory

the following table.

Case #	Body Ra, μCi	C_{Ra}, pCi/cm^3	C_{Rn}, pCi/liter
01-014	2.2	280	680
03-240	4.3	500	1200
03-209	1.1	760	1800
00-006	2.6	1220	3000

Within the range shown there is no correlation between body Ra content and radon concentration. For one subject, 03-209, a measurement of radon exhaled in the breath was made in vivo, which gave 12 pCi/liter demonstrating that the air space radon concentration greatly exceeds the breath concentration. This is because the air in the mastoid cells tends to stagnate, i.e. the air cells are not well ventilated.

D_{bone}/D_{air}

The ratio of dose rates is a function of several anatomical, physiological and physical-chemical variables which enter the radon accumulation model and the calculation of S_{bone}/S_{air}. The most important of these is the thickness of the lamina propria, the connective tissue layer which lies between bone and epithelium in the mucous membrane. A factor of 2 change in this thickness changes D_{bone}/D_{air} by more than a factor of 5, as shown by the table below for a mastoid air cell diameter of 1 mm.

D_{bone}/D_{air}	Thickness, μm
1.4	20*
.50	30
.18	40
.054	50
.0036	60
0	> 66.8

Quite accurate values of D_{bone}/D_{air} for other air cell diameters can be obtained from the table by assuming inverse proportionality to diameter.

More than 75% of the lamina propria is thinner than 66.8 μm and the average thickness is probably between 20 and 66.8 μm. For an air cell of 1 mm diameter, which is typical of the mastoid, it appears likely that D_{bone}/D_{air} is less than 1 and that the air space dose rate predominates. When the diameter is smaller than this such a conclusion is less certain.

Because the mastoid contains a great number of air cells with widely varying diameters it is probable that the air space dose predominates for the smaller ones and the bone dose predominates for the larger ones.

* Thinnest lamina propria so far observed in mastoid.

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CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Radium Exposure Registry
(Total effort 2.0 p-y)

Industrial and medical usage of radium in the U.S. began about 1913. Workers were heavily exposed in the radium dial and other industries until 1925, when radium's toxic effects became known and safety measures were adopted. Numerous therapeutic exposures occurred through 1930, along with some continuing occupational exposures. The radium dial industry revived during World War II, creating a large cohort of individuals exposed at lower levels. The early radium cohort is unique in that it is the only large group of humans exposed (non-therapeutically) to internally deposited alpha-emitting radionuclides at levels leading to unequivocal health effects. This permits estimation of dose-response relationships. The later cohorts both validate the lower-portion of the dose-response relationships and are proving invaluable as low-dose controls in the analysis of cancers of other soft-tissues.

Knowledge of the effects of alpha-emitters is vital in predicting health effects in the nuclear power industry, especially with respect to fuel reprocessing, and in the nuclear defense industry. Exposure standards for plutonium and other alpha-emitters have been set on the basis of human experiences with radium. Radium itself is currently of concern as a trace substance in water supplies and in relict tailings piles.

Current findings. Radium deposits mostly in bone, and the principal effects are associated with bone and bone surfaces. These are osteosarcomas, carcinomas of the paranasal sinuses and mastoids, and skeletal changes such as bone necrosis and coarsening of trabeculation. Over 80 cases of bone sarcoma and over 30 cases of sinus or mastoid tumor have been identified, most in measured cases.

Excess multiple myeloma among the early dial workers has been demonstrated,[1] and an excess of breast cancer has been suggested.[2] Recent results suggest that any breast cancer excesses may not be related to radiation.[3] There is no clear indication that the moderate excess of colon cancer in the population is related to radium or external radiation,[3] but further investigations of all soft-tissue cancers are required.

A very preliminary investigation of fertility among a subset of radium workers suggests a relationship between radium body burdens and subsequent fertility.[4]

Status of fieldwork and follow-up. From records of earlier radium research projects and search efforts of the Center since 1969, over 5000 persons exposed to radium have been identified. Most were in the radium dial industry, but some were chemistry research or therapeutic exposures. Table 1 gives a summary of current status of follow-up:

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Table 1. Follow-up status of radium cases

	Living		Dead			
	Measured	Unmeasured	Measured	Unmeasured	Unlocated	Total
Dial workers	1370	646	424	713	754	3907
All registrants	1559	729	699	963	1185	5135

The numbers above reflect increases of 50 in the number of newly measured cases, and of 117 in the number of newly located cases. The majority of examinations, of course, are re-examination.

Future plans. We will continue to register all authenticated cases of radium exposure, so potential for future investigations is maintained. Emphasis now is on soft-tissue effects, with respect to analysis. Analytic goals can be subdivided into short-term goals and long-term goals: the three analyses which are short-term priorities are investigation of effects of radium on reproduction, so far as that can be determined from data in the medical records; a case-control study of breast cancer in the population using previously uncoded information; and a mortality analysis of cancer in the male workers and radium chemists. The latter is a small population, but worth the limited effort of analysis.

Two long-term investigations need to be furthered: statistical reconstruction of underlying populations to resolve the "objective-nonobjective" ascertainment question, and epidemiologic investigations into factors associated with radium retention beyond duration of employment. Preliminary analyses suggest that current radium levels may show associations with factors other than duration and external radiation. In addition, a study of mortality and of congenital abnormalities (to the extent that these can be studied in subjects now of late middle age) is being planned for the children of the dial painters.

References

1. J. Cuzick, Radiation-induced myelomatosis, New Eng. J. Med., 304, 204 (1981).

2. E. E. Adams, and A. M. Brues, Breast cancer in female radium dial workers first employed before 1930, J. Occup. Med. 22, 583 (1980).

3. A. F. Stehney, H. F. Lucas, Jr., and J. H. Stebbings, Soft-tissue cancer mortality among female radium dial workers, in preparation.

4. A. P. Polednak, Fertility of women after exposure to internal and external radiation, J. Environ. Path. Toxicol., 4, 457 (1980).

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CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Occupational Epidemiology of Thorium Workers

(Total effort 2.0 p-y)

The objective of this program is to determine the health effects of industrial exposure to thorium and the long-term pattern of deposition of inhaled thorium and its daughter products in humans. As an energy resource, thorium-232 is of interest as a source of fissile uranium-233, a much less toxic nuclear fuel than plutonium. Studies of thorium are also relevant to the assessment of health risks from other alpha-emitters, such as plutonium, important in fuel reprocessing and breeder reactor technologies.

Health endpoints in this study include: cause-specific mortality, with the emphasis on cancer mortality; morbidity as determined by medical examination of living workers; and changes in physiological and biochemical parameters suggesting adverse effects upon bone marrow, liver, lung, and other organs.

Summary of efforts to date

The study populations are derived from the employment records of the Lindsay Chemical Company and its corporate successors. The company was primarily involved in the extraction of thorium and rare earth chemicals from monazite ores and in the production of thorium-impregnated mantles for gas lamps. From all available records, dating back to 1925, 3542 male and 1040 female employees have been identified, but records prior to 1940 are incomplete. Our studies deal mostly with 3200 men who worked at a plant site in West Chicago, Illinois, at any time from 1940 to shutdown in 1973.

Three health effects analyses have been completed and presented (in October, 1981): mortality in over 3000 workers; the complete blood count in 275 workers examined clinically and counted for body radioactivity, and liver function in those same 275 workers.

Provisional conclusions are that blood counts and liver function are marginally impaired in workers with higher body burdens of radionuclides. The mortality results have been previously reported.[1] No clear evidence for an effect of thorium exposure on mortality has been obtained. However, the slightly elevated mortality from all cancers and non-significant excesses of lung cancer and pancreatic cancer justify a second mortality follow-up study.

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Future plans

Most efforts during 1982 will be directed toward the analysis of existing data sets. The detailed analyses of blood counts and hepatic function tests will be finalized, and detailed analyses of the relationship of pulmonary function measurements, including flow and volume measurements from spirometry, and diffusing capacity, will be carried out on the 286 thorium workers who have been examined living.

A biostatistical analysis of factors which affect the relationship of exhaled thoron (^{220}Rn) to thorium daughters *in vivo* is also planned, with emphasis on factors relating to respiratory physiology.

A second round of mortality follow-up is also planned, but for the following year.

Reference

1. A. F. Stehney, A. P. Polednak, J. Rundo, A. M. Brues, H. F. Lucas, Jr., B. C. Patten, and R. E. Rowland, Report on health status and body radioactivity of former thorium workers. NUREG/CR-1420, ANL-80-37, Jan. 1980.

0019811

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CENTER FOR HUMAN RADIOBIOLOGY

Fact Sheet on

Radon in Houses
(Total effort 1.2 p-y)

A study is under way of the levels of naturally-occurring radon (the decay product of radium) and its short-lived radioactive daughter-products in houses in the Chicago area. The daughter-products are responsible for the largest dose of radiation from natural sources to any part of the body, i.e. lung. The most reasonable calculations seem to indicate about 10^4 fatalities per year in the United States due to lung cancer induced by radon daughters. Reduction of ventilation rates of houses with outside air to conserve energy can only increase this number. Thus, there is an urgent need for extensive data on the actual levels in houses of radon and its daughter-products, their variation with time, the sources of radon and the fate of its daughter-products under normal conditions in ordinary houses. In the past year, special emphasis has been placed on measurements of the equilibrium factor, or working level ratio, and determining those parameters which govern its value.

Survey Results to Date

A total of 109 buildings has now been investigated and a summary of the results is set out in the table below. These data include results for 46 houses in a group of about 120 in an area of about 0.2 km^2 (50 acres) which are being investigated systematically. At least one sample of air was taken from each level in the house, during the months of August 1980 to March 1981. Some houses had windows open when the samples were taken, while in others ventilation with outside air was at a minimum, so that the results are not strictly comparable. However the data suggest that the distributions of the concentrations for each level are approximately log-normal with geometric mean values of 1.6 pCi L^{-1}, 0.5 pCi L^{-1} and 0.7 pCi L^{-1} for lower level (usually basement or crawl space), first floor (entrance level) and second floor respectively. The corresponding geometric standard deviations were 2.4, 3.3 and 2.4 respectively. The limited data on radon in earth-shielded houses suggest that the average concentration may be higher than in the group of conventional houses.

	Number	Level	Conc'n. range, pCi L^{-1}
All houses	87	0 (Lower)	0.2 - 52
	64	1	0.1 - 26
	46	2	0.1 - 55
With unpaved crawl space	29	0	0.3 - 36
	10	1	0.3 - 19
	6	2	0.2 - 55
Earth-shielded	6	1	1.2 - 16

0019812

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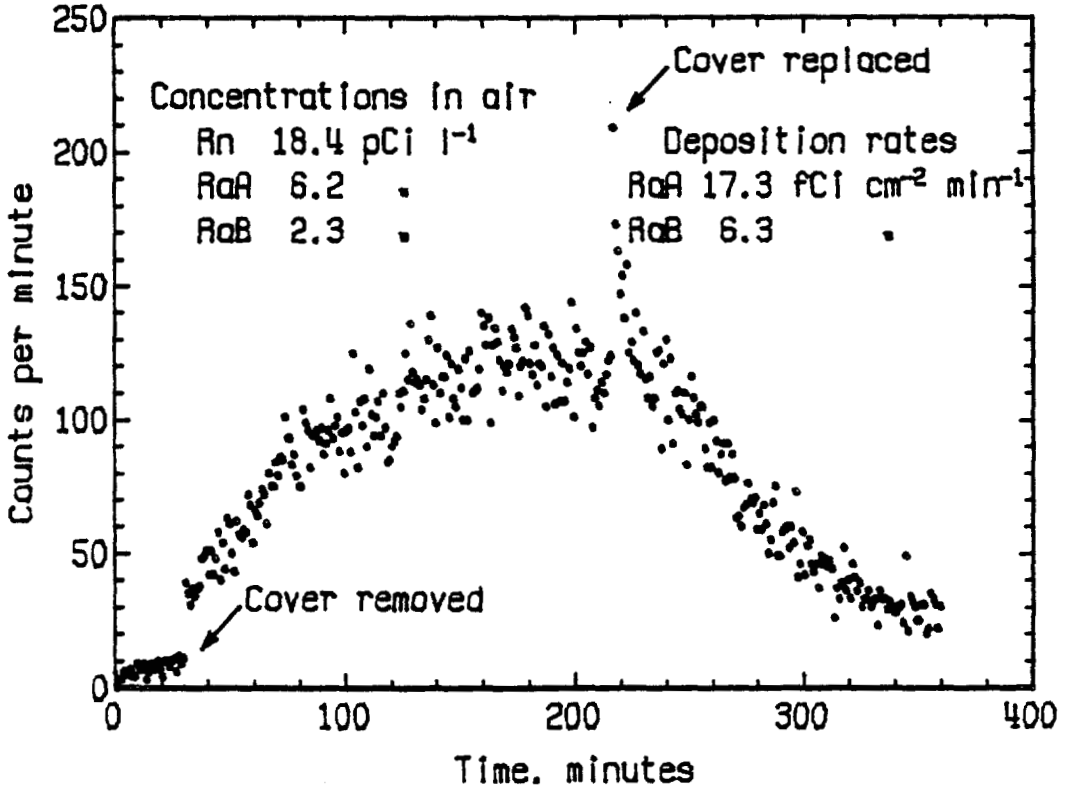
Equilibrium Factor Measurements

Our earlier data on equilibrium factors which gave values rather lower than those reported by others have been reinvestigated and extended. In all cases where we observed values for F of < 0.3, the houses were closed and the heating or air conditioning systems were running, with consequent filtration of the circulating air. Laboratory experiments elsewhere[1] have shown that the aerosol concentration is the single most important parameter determining the equilibrium factor. When dust particles are removed from the air by filtration, radon daughters cannot attach to them, and instead, the daughters plate-out on surfaces, resulting in a low value of F.

The plot below shows what we believe to be the first direct measurements of radon daughter plate-out in a house. When the cover was removed from the window of a 316 cm^2 area gas-flow proportional counter, the counting rate immediately increased due to the detection of radon and daughter decays in the air within α-particle range of the window. The counting rate continued to increase due to the deposition of radon daughters on the window, and the results of a least squares fit to determine the deposition rates are shown. Note that the ratios of RaA to RaB equal 2.7 for both the air concentrations and the deposition rates. When the cover was replaced, the counting rate doubled, since daughters were also deposited on the cover itself, and then decayed at a rate characteristic of a mixture of radon daughters.



0019813

Reference

1. A. C. George, E. O. Knutson, and K. W. Tu, Radon Daughter Plateout, Part I: Measurements. Sub. to Health Physics, 1981.

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CENTER FOR HUMAN RADIOBIOLOGY

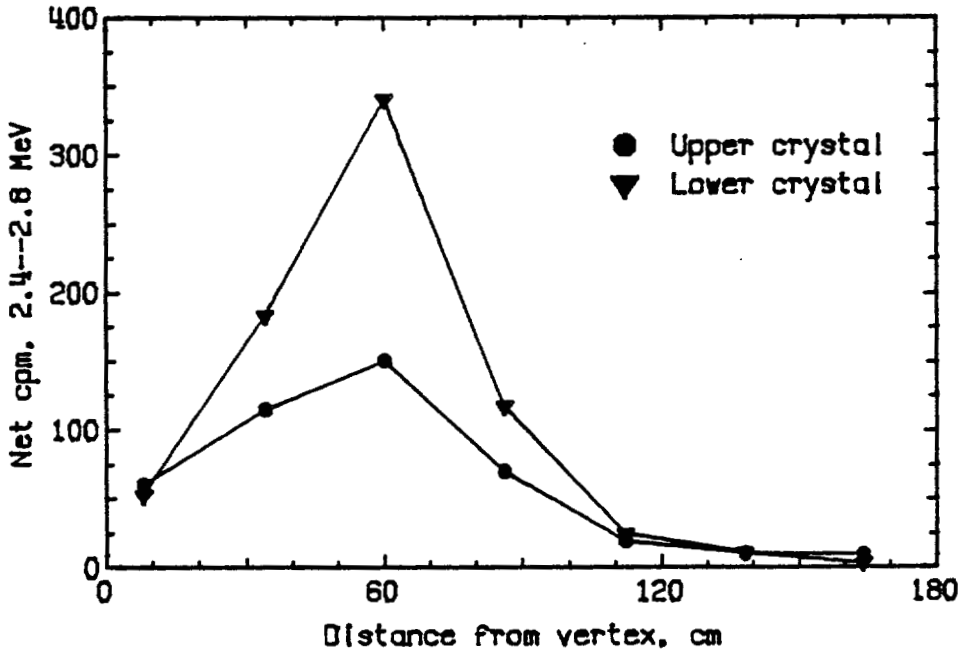
Fact Sheet on

Measurements of Thorotrast in Vivo
(Total effort 0.1 p-y)

Thorotrast is a colloidal suspension of thorium dioxide which was used as a contrast medium in radiography from 1928 until the mid-1950's. During the past year we measured the body content of radioactivity of a Thorotrast case and the method of data analysis employed is applicable to our measurements of thorium in former workers at a thorium refinery.

Distribution Measurements

The case was a 65-year-old white male who had had Thorotrast myelography in the early 1940's. We first established the location of the activity by means of 7-position scans with large uncollimated NaI(Tl) crystals arranged above and below the supine subject. The results shown below indicated that there was a sharp maximum in the response at a little less than 60 cm from the vertex, and that the center of activity was closer to the dorsal surface than the ventral. A transverse profile scan with a slit-collimated detector centered at 60 cm from the vertex established that the activity was localized in a small area centered about 3 cm to the left of midline.



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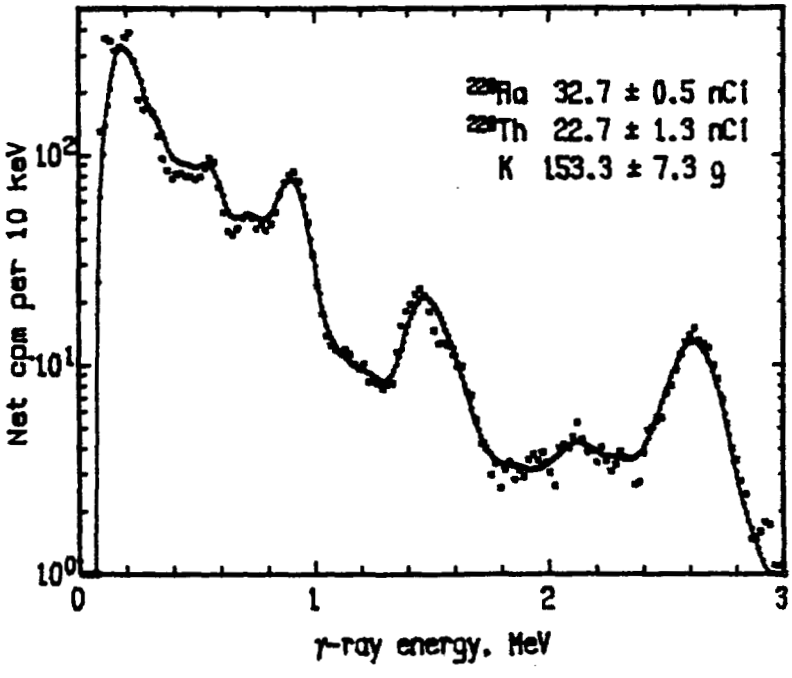
γ-ray Spectroscopy

^{228}Ac, ^{212}Bi, and ^{208}Tl emit a number of γ-rays resulting in a quite complicated spectrum. However, once the spectrum is analyzed, some information on the degree of equilibrium of the daughters among themselves can be obtained, and this in turn yields information on their differential metabolism. The γ-ray spectrum obtained with the lower crystal at 60 cm from the vertex was fitted by a computerized method of least squares with three standards: ^{228}Ra, with a ^{228}Th activity equal to 96% that of the ^{228}Ra; ^{228}Th with subsequent daughters in equilibrium; and potassium. The sources were placed in a tissue-equivalent phantom and their positions adjusted until the counting rate ratio of the upper and lower crystals matched that in vivo. The experimental spectrum and the fitted curve are shown in the figure below. The limits of the fit were from 0.8 to 2.8 MeV, since the energy region below 0.8 MeV is extremely sensitive to small differences in the configuration of the standard sources in a phantom vs. that of the activity in vivo. As shown on the figure, the amount of ^{228}Th present (22.7 ± 1.3 nCi) was considerably less than that of ^{228}Ra (32.7 ± 0.5 nCi). This is due primarily to the loss of ^{224}Ra from its site of formation and also to the exhalation of ^{220}Rn. The same also applies to ^{228}Ra, so all that can be said of the ^{232}Th content is that it must be greater than 32.7 ± 0.5 nCi (303 ± 5 mg).



Although this method of spectral analysis can only yield a lower limit for ^{232}Th content, it is still extremely important to apply it to the γ-spectra obtained from the population of thorium workers under study at CHR, since very little is known about the metabolism of thorium daughters following exposure by inhalation.