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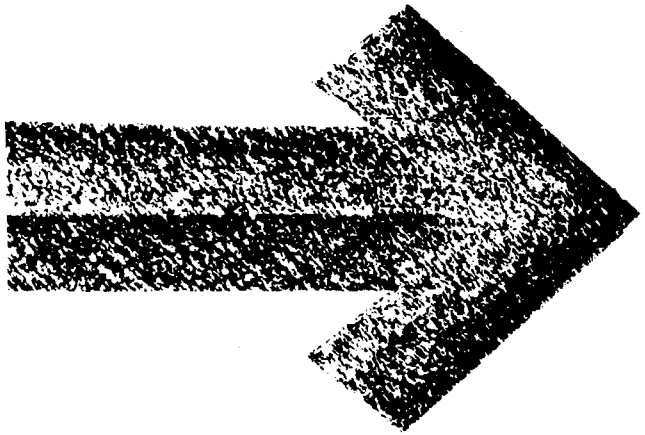
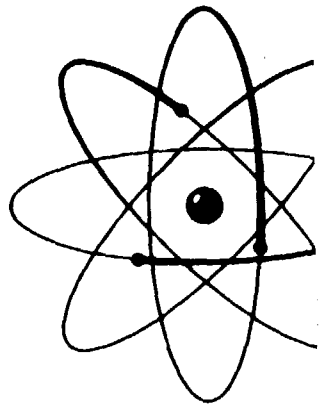


TID-17191

Report Number

"Natural Contents of Rad (Ra^{210}) and RaF (Po^{210}) in the Human Body"



United States Atomic Energy Commission
Division of Technical Information

OTI-131(May 1962)

USAEC Division of Technical Information Extension, Oak Ridge, Tennessee

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1 OF 1

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For pres. at Symp. on Radioactivity in Man, Sept. 5-7, 1962, Chicago, Ill.

ANC-6793

The Natural Contents of RaD (Pb^{210}) and RaF (Po^{210}) in the Human Body*

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aug. 1962

I. Introduction

In order to assess the dose due to the natural radiation in the human environment, the internal radioactivity of the body must be determined along with that of the external radiation. Much effort has been expended in determination of the artificial background from bomb fallout, whereas on a much smaller scale this has been done on many of the naturally occurring radionuclides, such as C^{14}, K^{40}, Ra^{226} and Ra^{228} (1-4). C^{14} accounts for little of the dose, although because of its location in the cell nucleus, it may be quite important. K^{40}, on the other hand, accounts for an appreciable fraction of the internal absorbed dose in the body,(5) but its biological importance is considerably reduced if an estimated RBE of 4 for alpha particles is assumed.

One purpose of these studies is to locate large human population groups which are identical to other groups, except for differences in exposure to natural radiation. Thus, there is the possibility of studying low-level radiation effects on humans. Natural radiation is of particular interest because it may allow the discovery of very large populations (that is, 100,000 or more) which have experienced known and constant lifetime exposures. Such groups have been found in certain

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* Work performed under the auspices of the U. S. Atomic Energy Commission.

sections of Illinois in which the Ra^{226} skeletal levels in the population are 10 times those of their neighbors. The differences are caused by variations in the Ra^{226} concentrations in the drinking water.(1)

RaD is another such nuclide. As shown in Fig. 1 (Slide 1), RaD is a Ra^{226} decay product, but one does not expect it to follow the parent because of their different chemistries and the long half life of RaD. The most important difference is that RaD is derived from the rare gas intermediate, Rn^{222}, which is easily translocated from the parent Ra^{226}.

Only in the last few years has naturally occurring RaD been studied. Dudley,(6) estimated the body content of this nuclide to be about 10 times that of Ra^{226}. Considering the amount of information available at that time, this conclusion was quite reasonable when compared to the subsequent measured values of Black(7), of Hill(8) and of this laboratory(9) using small sections of bone. On the other hand, Hursh(10) measuring whole body ash, found the RaD content to be only about 1/2 that of the Ra^{226}.

II. Experimental Technique

For this study particular effort was devoted to the determination of RaD, RaF and Ra^{226} in human specimens. These consisted of surgical and autopsy specimens of soft tissue and bone obtained from medical sources.

The RaD was determined by analysis of its decay product RaF (Po^{210}) (Fig. 1)(Slide 1). The analyses were performed by first wet ashing the samples with nitric and perchloric acids. Next, the nitrates were destroyed by repeated fumings with hydrochloric acid, and finally, after adjusting to pH 0.3, the Po^{210} was plated onto a silver disk at 90° C.

It was then alpha counted.(7,9)

Ra^{226} was determined by the radon emanation method of Lucas.(11) The ash content was determined from the calcium, expressed as calcium phosphate, which, because of a series of compensating factors, is equivalent to the ash.(9)

III. Results and Discussion

Interpretation of these measurements to obtain meaningful conclusions involved certain problems. The first, was that of the sampling procedure, that is, to determine if a particular sample is representative of the whole. Only small samples were available and even if larger ones had been available, processing would have been difficult. Thus, it was necessary to determine how well they represented the entire skeleton and body. This was done by the use of sets of bone sections, tibia, skull, mandible and either rib or joint bone, in which each set was from a single individual. Data from 8 sets are shown in Table I (Slide 2). Within a factor of 2 or so (with a few exceptions) any given bone appears to be representetive. Table 2(Slide 3) shows the Ra^{226} concentrations to be relatively constant within a given individual, again with a few notable exceptions. Because of the fairly high probability that a given bone represents the total skeleton (as defined by a few samples), and for lack of a better hypothesis, it has been assumed that the distribution of RaD is uniform within the skeleton.

The second problem considered was the whole-body distribution of RaD. The results in Table 3 (Slide 4) show about 60% of the activity to be in the skeleton, a result also in agreement with that calculated from the metabolic parameters of stable lead.(12,13) Since the skeleton constitutes about 10 to 15% of the total body weight, and contains more

than 60% of the RaD, the dose rate to the skeleton is about 10 times that to the other parts of the body.

Finally, the problem of the RaF-RaD equilibrium in the skeleton was considered. This is important because the dose is essentially due to the alpha-emitting RaF. The results were obtained by analyzing the RaF within a few weeks after surgery and then again after several months. Table 4 (Slide 5) shows the RaF to RaD ratio to be 1.0 ± 0.2 and thus the metabolic properties are essentially controlled by those of RaD, that is, on the average, there is little excess or deficiency of RaF over RaD.

Fig. 2 (Slide 6) is a plot of the RaD vs. the Ra^{226} concentrations in 128 samples from about 100 individuals. There is little correlation and in a given Ra^{226} interval of 0.01 pc/g ash, the RaD concentrations may range over a factor of 6. The RaD does appear to increase with Ra^{226} concentration and all points, except two, lie above the line:

$$(RaD) = 0.56\ (Ra^{226})^{.71}$$

where the parentheses refer to the concentration in units of pc/g ash of the enclosed species. The significance of this line is unknown. The two points below the curve are less significant than the others because they are data from specimens obtained from children and there is some evidence that children have lower RaD concentrations than do adults.(9) The overall average of RaD was 0.146 ± 0.020 pc/g ash and of Ra^{226} 0.037 ± 0.007 pc/g ash.

Table 5 (Slide 7) shows the RaD concentration in the trabecular bone samples to average 0.18 pc/g ash, about 75% greater than that in cortical bone samples. The average RaD concentration in men, 0.16 pc/g ash, was 35% higher than the 0.119 pc/g ash in women. While the

trabecular and cortical samples are not uniformly distributed between the two sexes, the average for each respective type of bone is higher in men than in women. While there are many questions involved in the sampling procedures, these results do indicate the RaD concentration is higher in trabecular than in cortical bone and is higher in men than in women.

On a regional basis, there is no apparent correlation in the measured population as shown in Table 6 (Slide 8). This is a series of measurements on rib specimens from 14 people who lived in Chicago 15 years or more. The concentrations range over a factor of 6, but average 0.177 pc/g ash, which is slightly higher than the overall average of 0.146 pc/g ash but it is about the same as the average trabecular bone. The Ra^{226} values average 0.015 pc/g ash, which is about that expected in Chicago residents.(11)

We have also tried to determine the source of RaD in the body and, as shown in Table 7 (Slide 9) six sources are considered: Ra^{226} and Rn^{222} in the body, RaD in potable water, RaD in the atmosphere, short lived Rn^{222} daughters in the air and RaD in food.

Using the exponential model for excretion and the parameters for lead and RaD metabolism in the "Standard Man"(9,12,13) based on those given in the Report of the International Commission of Radiation Protection, we have estimated the contributions of these various sources to the body content. The contribution of drinking water appears significant, but the value used is extremely high,(9) about 10 times the average, and consequently, few people would be exposed to this. The Rn^{222} value of 3 pc/l is also high.(14) We conclude, therefore, that in most individuals the only significant sources of RaD to the body are food and air. The general agreement of the calculated with the measured values is significant, although, because of the assumptions in the

calculations and the great range of the experimental data, the close agreement is fortuitous.

Finally, the data presented here may be used to estimate the internal dose rate due to RaD and its daughters relative to that from the Ra^{226} chain, Fig. 1 (Slide 1). The effective dose is given by the product of the average energies of the emitted particles, an RBE of four for alpha particles and the fractional retention of the nuclides. For RaD and its daughters and for Ra^{226}, the retention is one; for Rn^{222} and its daughters, it is 0.3.[15] Consequently, for a given activity of each parent nuclide, the ratio of effective dose rates of RaD to Ra^{226} is 0.5. Table 8 (Slide 10) shows the average RaD concentration of 0.146 pc/g ash is equivalent to a Ra^{226} concentration of 0.073 pc/g ash. This is about twice the measured Ra^{226} concentration in our samples. It must be noted that this factor of 2 is actually a minimum since these samples include a relatively large fraction from people residing in areas with high Ra^{226} drinking water. Most people live in low-level areas, such as Chicago.[11] Consequently, over the whole country the RaD dose-rate levels are actually about 5 times those of Ra^{226}. Since the dose due to Ra^{228} in the body is about equal to that of Ra^{226},[16] the RaD dose is about 2-1/2 times that of the two Ra nuclides.

The radiation levels of RaD may be also compared to those of Sr^{90}. If an RBE of 4 is assumed for alpha particles relative to that of betas, and the Sr^{90} concentration is about 0.5 pc/g ash[17], then the RaD dose is about 10 times that due to Sr^{90}.

IV. Conclusion

It must be concluded that because of the difficulties in determining the RaD and RaF contents of the skeleton, the total dose-rate therein may be difficult to estimate. Moreover, because of the lack of correlation between the RaD and the Ra^{226} concentrations, the relative dose rates cannot be defined as a function of geography. The statistical uncertainties in any associated epidemiological study would thereby be increased.[18] Some of the difficulties might be alleviated if further studies make other correlations apparent.

Acknowledgements

Special thanks are due to Dr. A. J. Finkel and Mr. H. F. Lucas, Jr. for obtaining the majority of the samples and to Dr. J. B. Hursh for the sets of bone specimens. Mr. Lucas also kindly supplied much of the Ra^{226} data and his discussions were very helpful. Technical assistance was supplied by Messieurs F. W. Ilcewicz and R. P. Burke. I am particularly indebted to Mr. L. D. Marinelli for the many helpful suggestions and discussions during the preparation of this paper.

REFERENCES

1.* A. F. Stehney. Radioisotopes in the Biosphere, (University of Minnesota, Minneapolis, 1960), p. 366.

2.* H. Muth, B. Rajewsky, H. J. Hantke, and K. Aurand. Health Physics 2, 239 (1960).

3. H. F. Lucas and D. P. Krause. Radiology 74, 114 (1960).

4. D. P. Krause. Argonne National Laboratory Radiological Physics Division Semiannual Reports, ANL-6049 (October, 1959), p. 51, and ANL-5967 (May, 1959) p. 126.

5. R. A. Dudley. Low Level Irradiation, (American Association for the Advancement of Science, Washington, D.C., 1959), p. 7.

6.* R. A. Dudley. The Toxicity of Skeletal Irradiation at Naturally Occurring Radiation Levels, April, 1957 (unpublished).

7. S. C. Black. Health Physics 7, 87 (1961).

8. C. R. Hill. Health Physics 8, 17 (1962).

9. R. B. Holtzman. Argonne National Laboratory Radiological Physics Division Semiannual Report, ANL-6199 (August 1960) p. 94.

10. J. B. Hursh. Science 132, 1666 (1960).

11. H. F. Lucas, Jr. Argonne National Laboratory Radiological Physics Division, ANL-6297 (September, 1961), p. 55.

* These papers contain extensive reference to the literature in this field.

REFERENCES (continued)

12. International Commission on Radiological Protection, Committee II on Permissible Dose for Internal Radiation (1959). Health Physics 3, 1-380 (1960). Modifications to these data are presented in Reference 13.

13. R. B. Holtzman. Argonne National Laboratory Radiological Physics Division Semiannual Report, ANL-6297 (February, 1961), p. 67, and Health Physics 8, 315 (1962).

14. H. Moses, A. F. Stehney, and H. F. Lucas, Jr. J. of Geophysical Research 65, 1223 (1960).

15. W. P. Norris, T. W. Speckman and P. F. Gustafson. Am. J. Roentgenol. Rad. Therapy and Nuclear Med. 73, 785 (1955).

16. H. F. Lucas, Jr. Second International Conference of Radiation Research, Harrogate, Yorkshire, England, August 5-11, 1962, p. 102 (Abstract).

17. J. L. Kulp and A. R. Schulert, Science 136, 619 (1962). The value 0.5 pc/g ash is a rough estimate from this paper for people about 13 years of age.

18. L. D. Marinelli. American Journal of Roentgenology and Radium Therapy, LXXX, 729 (1958).

FIGURE 2

RaD CONCENTRATION AS A FUNCTION OF Ra^{226} CONCENTRATION IN HUMAN BONE SAMPLES

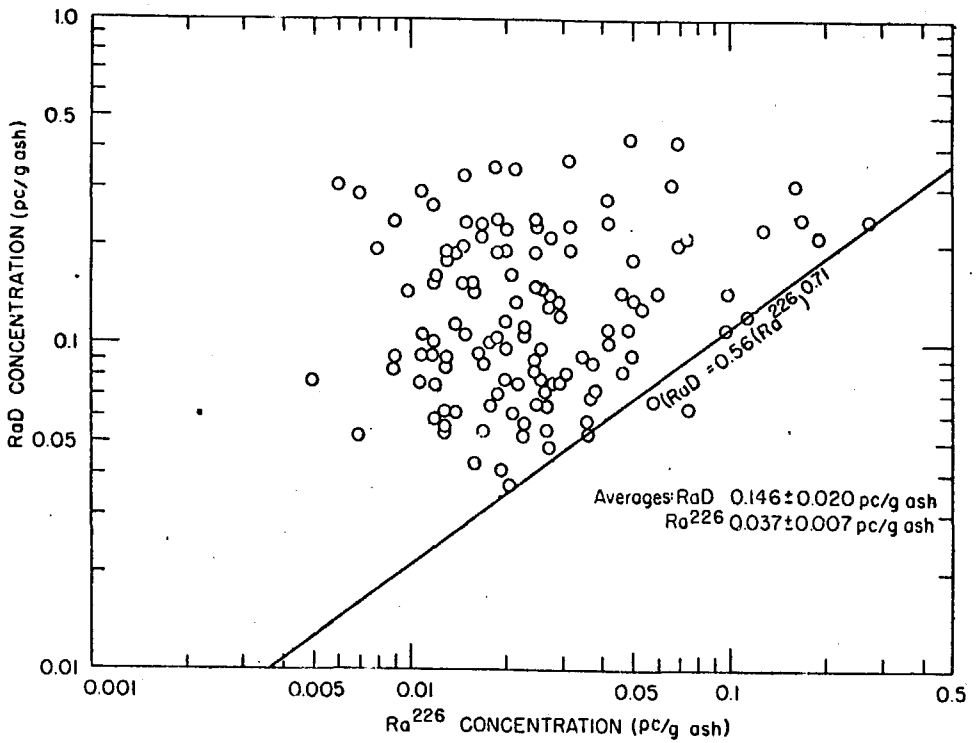

TABLE I

RaD CONCENTRATIONS IN BONE

SUBJECT*

Bone	40 M	43 F	49 F	52 F	63 M	68 M	77 M	90 F
Tibia	0.075	0.090	0.081	{0.078 {0.132	0.085	{0.269 {0.247	0.117	0.070
Mandible	0.105	0.049	0.081	0.077	0.055	0.093	0.225	0.061
Skull	0.102	{0.092 {0.146	0.110	{0.366 {1.33	0.091	{0.201 {0.419	0.191	0.086
Rib	0.142	-	0.129	-	0.150	-	0.453	0.066
Joint (Bone)	-	0.083	-	0.071	-	0.152	-	-

*Subjects identified by age and sex

TABLE 2

Ra^{226} CONCENTRATIONS IN BONE

SUBJECT*

Bone	40 M	43 F	49 F	52 F	63 M	68 M	77 M	90 F
Tibia	0.022	0.025	0.047	0.026 0.030	0.013	0.012 0.019	0.014	0.019
Mandible	0.015	0.028	0.032	0.030	0.020	0.012	0.020	0.021
Skull	0.018	0.051 0.100	0.049	0.032 0.027	0.009	0.070 0.070	0.014	0.017
Rib	0.016	-	0.053	-	0.025	-	0.020	0.025
Joint (Bone)	-	0.025	-	0.027	-	0.016	-	-

* Subject identified by age and sex

TABLE 3

DISTRIBUTION OF RaD IN VARIOUS HUMAN ORGANS

Organ (Ratio: organ wt. to body wt., %)	Average Concentration of RaD (pc/g wet)*	Fraction of Total Activity (%)
Liver (2.4%)	0.011 ± 0.003	1.7
Muscle (43%)	0.006 ± 0.004	17
Bone (Rib) (4.0% of ash) (10% of total body)	0.235 ± 0.036	63
Other tissues (45%)	0.006**	18

*bone given per gm ash

**other tissues assumed to have same average concentrations as muscle

TABLE 4

RADIOACTIVE EQUILIBRIUM BETWEEN RaD AND RaF IN HUMAN BONE *in vivo*

Sample No.	Type of Tissue	Ratio RaF/RaD
115	Iliac Crest	0.81
131	Rib	0.77
135	Vertebra	0.79
140	Joint	1.00
186	Rib	1.43
245	Rib	1.17
Average		1.0 ± 0.2

TABLE 5

AVERAGE RaD CONCENTRATIONS

Bone	Sex*		
	Male (pc/g ash average ± E)	Female (pc/g ash average ± E)	All Subjects (pc/g ash average ± E)
Trabecular	0.196 ± 0.023 (47)	0.156 ± 0.040 (20)	0.184 ± 0.029 (67)
Cortical	0.115 ± 0.021 (36)	0.090 ± 0.020 (25)	0.105 ± 0.021 (61)
All Bone	0.161 ± 0.022 (83)	0.119 ± 0.030 (45)	0.146 ± 0.020 (128)

*The number of samples in each group is given in the parentheses.
E is the 90% confidence interval of the mean.

TABLE 6

RaD AND Ra^{226} CONCENTRATIONS IN RIB BONES OF CHICAGO RESIDENTS

RESIDENCE TIME >15 YEARS

Sample	Ra^{226} (pc/g ash)	RaD (pc/g ash)
1	.014	.063
2	.005	.078
3	.009	.084
4	.025	.096
5	.022	.134
6	.028	.138
7	.010	.144
8	.021	.160
9	.020	.190
10	.008	.195
11	.009	.243
12	.007	.290
13	.006	.308
14	.022	.355
	.015 ±.003	.177 ±.040
	(± 20%)	(± 23%)

TABLE 7

POSSIBLE SOURCES OF THE RaD CONTENT OF THE HUMAN SKELETON

Source	Contribution to RaD Concentration (pc/g ash)
Ra^{226} in Bones	0.004
Rn^{222} Dissolved in Body (Equivalent to 50 l of air at 3.0 pc Rn^{222}/l)	0.007
Potable Water (Maximum Observed, 0.4 pc RaD/l)	0.022
RaD in Atmosphere (0.03 pc RaD/l)	0.073
RaD from Short-Lived Activities (Rn^{222} daughters)	0.004
Food	0.066
Total	0.176
Total (less Rn^{222} in body and potable water)	0.148
Measured RaD Concentration (Average)	0.145 ± 0.020

TABLE 8

DOSE RATES OF RaD AND Sr^{90} RELATIVE TO THOSE OF Ra^{226} AND $Ra^{226} + Ra^{228}$

Nuclide	Concentration (pc/g ash)	Dose Rate (Relative to Ra^{226})	Dose Rate* (Relative to $Ra^{226} + Ra^{228}$)
Ra^{226} (all subjects)	0.037	1	0.5
RaD	0.146	2.0	1.0
Sr^{90}	0.5	0.3	0.15
Ra^{226} (Chicago)	0.015	1	0.5
RaD	0.146	5	2.5
Sr^{90}	0.5	0.7	0.35

*Assume Ra^{228} dose rate equals Ra^{226} dose rate

END