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14 to 17 support the values of "background" concentrations chosen in these calculations. Measurements are in progress to estimate further the ^{226}Ra content and also to assess the emanation rate of the ^{222}Rn daughter from the wood, which determines the fraction of the ^{226}Ra forming ^{210}Pb in the wood.

Since these results indicate that the circulation of lead in heartwood is small after a certain time, if any significant portion of the lead was derived either from direct atmospheric uptake or from increased concentration in the soil from atmospheric fallout, the wood might show the effects of changes in exposure over the years. Some measurements of the stable Pb concentration in these trees made by Dr. Ter Haar are shown in Figure 38.[12] Although environmental lead may be increasing, the few points available indicate a lower concentration of stable lead in trees in more recent times. This effect may be caused by the weighting of the data by the low values in the outer rings (near $t = 0$), which because of metabolic activity may be unrepresentative of the remainder of the wood. Also, as the tree ages and roots become deeper, the availability to the trees of stable lead may decrease relative to that of ^{210}Pb. Thus, increased lead in the atmosphere and soil over the years may not be available to the wood, and so the effects are not seen in this type of measurement.

Both the ^{226}Ra and stable lead data are consistent with those of the ^{210}Pb (^{210}Po) in that the concentrations in the hickory are much higher than in the other woods by factors of 2 to 10. The reasons for these variations are unknown, but they could be caused by basic metabolic differences, the higher ash content of the hickory, or to differing environmental levels to which our particular specimens were exposed. The latter case seems unlikely since one would not expect all three materials to increase simultaneously.

In summary, the decrease of the ^{210}Pb concentration with a 21.4-year half-life shows there is little circulation of lead in hickory heartwood more than 20 years old, while in oak there is little circulation even in wood less than 5 years old (but with less certainty). The uncertainties in these conclusions are caused by the low concentration of ^{210}Pb and the presence of ^{226}Ra in the oaks. The ^{210}Po does not appear to circulate, either. Decreased, rather than increased, concentration of stable lead in recent times is evidenced by these data. However, more detailed measurements are necessary to check this point.

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THE CONCENTRATION OF LEAD IN HUMAN BONE

R. B. Holtzman, H. F. Lucas, Jr., and F. H. Ilcewicz

The concentration of lead in bone from humans whose ages ranged from newborn to 85 years was found to increase with age at a rate of 0.6 μg (g ash)$^{-1}$ yr^{-1} and to be 8.7 μg (g ash)$^{-1}$ at birth. These results corroborate previous reports of increases up to age 30, and they show a continuing and similar increase in the group over 35 years of age. This rate represents an increase in skeletal content of about 4 μg/day, which is about 1% of the daily intake. The biological half-life of lead implied from the data from this study ranges from 70 to 90 years and is longer than previously reported. The concentrations of lead in the group over 30 apparently consisted of two normally-distributed populations, which may reflect differences in the cigarette smoking habits of the subjects. Further studies are required to more accurately evaluate the effects of smoking

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and to determine whether the accumulation of lead in adults simply reflects a long biological half-life or is caused by changing levels of intake of lead.

INTRODUCTION

Lead, because of its wide commercial application, ubiquity in the environment, and high toxicity, has probably been the subject of more toxicological studies than any other single substance. Consequently, the toxicity is well known for high levels of acute and chronic exposure to lead. Patterson[1] and Hardy[2] are very concerned about a possible health hazard to persons not occupationally exposed to lead, since in urban areas, the average lead concentration in blood is as high as one-third to one-half that at which symptoms of toxicity may be clinically apparent following an acute exposure.[3]

Despite extensive toxicological, metabolic, and environmental studies,[2] much still remains to be learned about the metabolic parameters of this element and its distribution in man and the environment. There is also disagreement about the variation of lead in man as a function of age, residence history, and occupation in other than industrial exposure to this element.[4]

Part of the controversy on the toxicity of lead concerns the ability of the body to compensate for increased intake levels, that is, the tendency for excretion rates of lead to approach asymptotically those of intake.[1, 4] The change in lead content of the human body as a function of age may, therefore, be a sensitive method of assessing the degree of this compensation.

In recent work by Nusbaum et al.[5] the lead concentration in calvarium and rib bone from subjects in the Los Angeles area was slightly higher for subjects over 20 years of age than below. Above 30 years of age the concentration did not appear to change. The lead concentration in lung, bone, kidney, pancreas, liver, and aorta was shown by Schroeder and Balassa's analyses of Tipton's data[6] to increase up to ages of at least 30–40 years. More recently Schroeder and Tipton[7] reported that the concentrations of lead and calcium in aortas increased with age, and that the lead concentration increased faster than calcium. Horiuchi et al.[8] in Japan found a similar increase in rib, vertebra, and femur up to age 40, and the concentrations of lead and calcium in the various bones were correlated at the 0.05 level of significance. They estimated that the total body lead content increased from about 78 mg in adolescence to about 131 mg at age 50. The similarity between these estimates and the 111-mg total body content[9] estimated from the data of Tipton et al.[10] and more recent estimates of 131 mg by Tipton and Schroeder[7] indicates a similarity between the U. S. and Japanese populations. However, there is still some question about the total mass of soft tissue and the sampling of the wet bone in the Japanese study.[8]

This increase of skeletal lead content with age implies, as stated by Schroeder and Tipton,[7] that the body is not in a steady state with respect to lead, that is, the human body retains a portion of all lead ingested. Thus, the increase in lead content of about 50 mg in 30 years (Horiuchi) represents a retention of about 4 μg/day, or about 1% of the 400 μg ingested daily.[9, 11] This low level of retention is well within the experimental error of most metabolic balance studies. Accumulation could be due to the long biological half-life of lead in the skeleton or to changes in dietary and smoking habits on reaching adulthood. Smoking, as shown by Nusbaum et al.,[5] may increase skeletal lead by as much as 30%.

Corroboration of increases in skeletal lead with age is shown in previously reported data of ^{210}Pb, a naturally-occurring radioactive nuclide with a 22-year half-life, which is also ubiquitous in humans and their environment.[12]

The purpose of this study is to determine the lead concentration in bone from a large number of persons for whom age, sex and residential histories were available. Occupational histories were also available for 85 of the 105 subjects.

EXPERIMENTAL METHOD

The bone samples were obtained during normal surgical or autopsy procedures. No two samples were from the same subject, and the cause of death or basis for surgery was known. None are believed to have been exposed to lead occupationally.

Lead concentration was also determined in teeth from 29 other subjects from Chicago and neighboring regions of Illinois. These teeth were pooled in 8 age groups of 2 to 4 teeth each.

The concentration of lead was determined by the spectrophotometric method of Ilcewicz et al.[13] In this method the bone is ashed 8 hr at 600° C and dissolved in 9 M HCl at a concentration of up to 0.2 g ash/ml. After extraction with triisooctylamine to remove interfering elements, mainly iron, the lead is determined from the absorbance at a wave length of 271 mμ. Comparison of dry-ashed with identical wet-ashed specimens showed no loss of lead due to heating or to the solvent extraction procedure. Because of the variability of bone weights, as discussed elsewhere, and the high likelihood of lead being associated with the mineral fraction of bone, the concentrations are given as a function of the ash weight of bone.[14] The overall analytical errors are estimated to be less than 5%.

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RESULTS AND DISCUSSION

The concentrations of lead in the various specimens in units of $\mu g/g$ bone ash, along with the age of the subject are presented in Table 18. The specimens are tabulated by bone type, i.e., rib, vertebra and cortical (femur and tibia), and by sex from subjects without known bone disease ("normal" bone). In addition, data from a previously published paper are included for uninvolved cortical bone (femur or tibia) from subjects with osteogenic sarcomas ("sarcoma" bone).[15]

The concentration is plotted as a function of age of the subject in Figure 39. The linear least squares line is shown for each group. For the "normal" cases the equation is

$$Y = (3.6 \pm 4.3) + (0.60 \pm 0.09)t$$

and for the "sarcoma" cases

$$Y = (8.6 \pm 2.7) + (0.37 \pm 0.08)t,$$

where Y is the lead concentration ($\mu g/g$ ash) and t is the age in years.

The variation of the lead content of different bones with age was evaluated and the coefficients of the linear least squares fits to the data are summarized in Table 19. The linear fit was chosen as the simplest to describe the data; no significant improvement of the variance was given by a second order function ($P > 0.05$),[16] except in "normal" female vertebra ($P < 0.05$).

The zero intercepts, A, (concentration at birth) ranged from -2 to $13\ \mu g/g$ ash with large standard deviations. At the 5% levels of significance or better, only 2 groups, sarcoma "all" and sarcoma "female," had intercepts significantly greater than zero ($P < 0.01$). However, a mean value of $8.7\ \mu g/g$ ash was obtained for bone from three stillbirths and a 6-month-old child. This is consistent with the data of Horiuchi et al.[8] who found the lead concentration of fetal bone to increase with age from $0.5\ \mu g/g$ ($3\ \mu g/g$ ash if the wet-to-ash ratio in fetal bone is 6) in a 5-month fetus to $1.5\ \mu g/g$ ($9\ \mu g/g$ ash) at 10 months. Schroeder and Tipton (their Table 5)[7] found less than $4\ \mu g/g$ ash in bone in their 0-to-1-year old subjects (apparently the detection limits of their measurements). The apparently negative intercept in the normal cortical bone is consistent with that of Horiuchi et al.[8] for femur bone.

The slopes of the regression curves of the various data groups are significantly greater than zero ($P < 0.01$), except for female rib ($P \approx 0.07$). The slopes of the lines for the different groups are not significantly different from that of the total "normal" of $0.60\ \mu g$ (g ash)$^{-1}$ yr^{-1}, except those of the female rib and "sarcoma" subjects which are significantly lower ($P < 0.01$ and < 0.025, respectively). However, these differences appear to result from an excess of high values at the younger ages. If the zero intercept, A, is fixed at zero, the slopes of the regression lines are no longer significantly different.

That the slopes are not zero is also shown by the significant correlation coefficients in Table 19 of about 0.5 to 0.7. While bone from female rib increases at only about one-half the rate of male rib, this difference is not significant ($P \approx 0.10$). The slope for "normal" male cortical bone appears to be different from that

TABLE 18. CONCENTRATION OF LEAD IN BONE ASH AS A FUNCTION OF AGE OF SUBJECT

Rib[a]		Vertebra[a]		Cortical[a]		Cortical[b]	
Age, yr	Concentration, μg Pb/g ash	Age, yr	Concentration, μg Pb/g ash	Age, yr	Concentration μg Pb/g ash	Age, yr	Concentration μg Pb/g ash
Male							
0	16.9	0.5	8.5	8	3.9	9	7.0
3	<1.0	28	24.7	13	3.3	12	3.5
8	3.6	28	35.5	14	12.6	13	7.3
27	23.2	44	19.4	19	2.8	13	7.5
30	11.0	53	61.2	20	4.0	16	7.6
32	15.4	57	47.9	42	38.9	17	30.5
36	13.2	59	41.9	48	37.2	17	13.0
37	19.5	71	41.6	49	24.0	23	17.7
37	25.5			53	34.5	27	13.7
37	39.4			56	43.5	32	16.3
38	19.7			59	79.3	44	17.3
38	7.7			61	31.5	47	31.8
41	59.4			65	96.5	58	50.0
42	57.2			66	30.8	59	38.6
44	50.8			72	43.3	63	19.0
48	25.8			85	40.5	64	33.9
59	49.6					67	28.2
68	48.1					68	33.4
74	21.7						
Female							
8	7.2	0	6.0	13	6.2	7	22.5
18	9.7	0	3.5	61	22.2	11	3.6
25	15.0	9	12.2	72	33.8	12	14.7
32	16.6	34	28.8			12	18.1
32	25.7	46	44.3			14	14.7
34	13.2	55	67.2			15	24.7
35	8.4	65	65.2			15	16.9
37	13.5	65	82.5			15	16.4
38	21.6	85	60.5			23	24.0
40	17.9	85	18.3			30	19.6
41	22.5					46	25.9
42	24.1					47	10.1
43	13.0					50	22.0
46	56.9					62	40.0
49	16.5						
65	13.4						
68	25.6						

[a] Subjects having no known bone disease.
[b] Subjects having osteogenic sarcoma.

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TABLE 19. LINEAR REGRESSION PARAMETERS FOR VARIOUS DATA GROUPINGS OF LEAD CONCENTRATION IN BONE

Type (No. of samples)	Linear regression coefficients		Correlation coefficient, r	Maximum probability, $r = 0$
	Intercept	Slope		
	$A \pm$ S.D., μg Pb (g ash)$^{-1}$	$B \pm$ S.D., μg Pb yr^{-1} (g ash)$^{-1}$		
All samples (105)	4.9 ± 2.9	0.552 ± 0.065	0.70	0.0005
Normal				
All (73)	3.63 ± 4.13	0.605 ± 0.089	0.62	0.0005
Cortical, M + F (19)	−1.70 ± 9.48	0.709 ± 0.183	0.71	0.0005
Trabecular, M + F (54)	5.53 ± 4.63	0.562 ± 0.103	0.64	0.0005
Vertebra, M + F (18)	11.04 ± 7.73	0.600 ± 0.151	0.67	0.005
Rib, M (19)	7.06 ± 7.98	0.536 ± 0.194	0.45	≈0.025
Rib, F (17)	8.25 ± 7.67	0.276 ± 0.187	0.33	0.10
Osteogenic sarcoma (cortical)				
All (32)	8.62 ± 2.67	0.371 ± 0.078	0.69	0.0025
Male (32)	4.47 ± 3.96	0.456 ± 0.094	0.77	0.005
Female (14)	13.15 ± 3.56	0.248 ± 0.115	0.50	0.05
Other data				
Horiuchi et al.[a]	−0.65	0.61	—	—
Schroeder and Tipton[b]				
Rib, 0–49 yr	(0.00)[c]	1.03 ± 0.10	—	—
Rib, 0–69 yr	9.3 ± 9.8	0.59 ± 0.26	—	—

[a] Reference 8.
[b] Reference 7, Table 5.
[c] Forced zero intercept.

for the "sarcoma" bone, but it is not significant at the 5% level of confidence. The concentration of lead in bone of female sarcoma cases increases more slowly than "normal" cortical bone ($P < 0.05$) and is essentially identical to the "normal" female rib bone.

The lead concentration for subjects over age 30 shows a similar increase with age. For "normal" subjects the rate of increase is $b = (0.56 \pm 0.17)$ μg Pb g^{-1} yr^{-1}, and for the "sarcoma" cases, $b = (0.50 \pm 0.21)$ μg Pb g^{-1} yr^{-1}.

The rate of increase of concentration estimated in "normal" bone of about 0.6 μg g^{-1} yr^{-1} bone ash, is equivalent to the 0.37 μg g^{-1} yr^{-1} in wet bone (femur) found by Horiuchi et al.[8] if one assumes that femur has about 60% ash content.[14] Schroeder and Tipton's data in their Table 6 give estimated rates by weighted least squares analysis of 1.03 μg g^{-1} yr^{-1} in the 0–49 year group and 0.59 g^{-1} yr^{-1} in the 0–69 group (Table 19). Their higher values may be caused by some high measurements in the 40- to 59-year group which range to 265 μg g^{-1}. The yearly increases observed by Horiuchi, et al.[8] and in this study amount to an accumulation in "normal" subjects of about 1.9 mg of lead per year in a "Standard Man" with 2600 g of total skeletal ash. The estimated total skeletal lead of 96 mg in a "Standard Man," aged 50, in this study, compares favorably with the 92 mg of skeletal lead from a previous report[9] and a little less favorably with the 110 mg of Schroeder and Tipton based on their median concentrations of 43 ppm of ash and 2600 g of skeletal ash.

TABLE 20. CONCENTRATION OF LEAD IN TEETH

No.	Mean age,[a] yr	No. of teeth in pool	Pb, μg/g ash
1	4	2	9.6
2	6	3	2.9
3	24	4	21.4
4	26	4	17.2
5	43	4	40.5
6	44	4	22.7
7	62	4	21.9
8	64	4	14.2
Mean (ages 4–6)			6.3
Mean (ages 24–64)			23.0 ± 9.2 (S.D.)

[a] Mean of age at time of tooth extraction.

Unlike that in bone, the lead concentration in teeth did not increase with age after the second decade. As shown in Table 20, the concentration appears to level off at 23 ± 9 μg/g ash, which is equivalent to that reached in bone at about age 27. The constant and low concentration is to be expected because of the greatly reduced mineral metabolism in the teeth of adults. The low values in the deciduous teeth are consistent with

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the significant but low concentrations observed in bone from fetuses and young children.

The validity of the confidence limits depends on the statistical distribution of the data. The total data and the subgroupings given in Table 19 were normally distributed as shown by a cumulative probability test. However, since the concentration increases with age, the distribution could be biased by the sample age distribution, that is, by the number of samples from younger subjects relative to those from older ones. This age effect was removed by testing the distribution of the residuals (the deviations of the data points from the least squares line). A histogram for all 105 samples is given in Figure 40. A best-fit gaussian curve for these data is shown by the solid line. The distribution of the deviation from the least-squares line appears to be skewed and a 2-gaussian-fit reduces the variance ($P < 0.10$). This skewness is attributed, at least in part, to the additional lead intake by smokers.[5] The effect of smoking on lead content of bone would be most apparent in subjects over 30 years of age. The histogram for these 41 trabecular "normal" cases is given in Figure 41. The solid lines represent two normal curves. These two curves give a significant variance reduction ($P < 0.05$) over a single normal curve. The smaller curve, centered on + 11.2 μg/g ash represents 29% of the total area. This value, while lower than the 44% of the smokers in the adult population (above 17 years

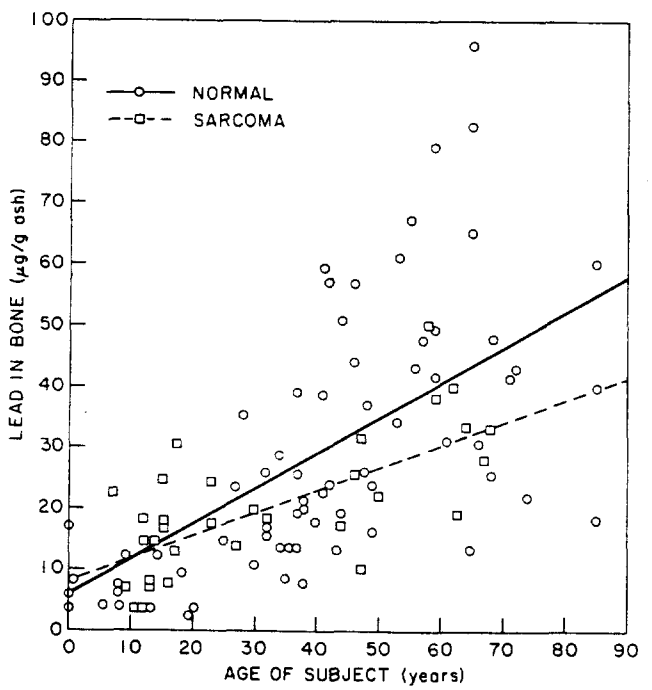

FIG. 39.—Concentration of lead in human bone ash versus age of subjects for "normal" and "sarcoma" cases. Lines are linear least squares fit to the data.

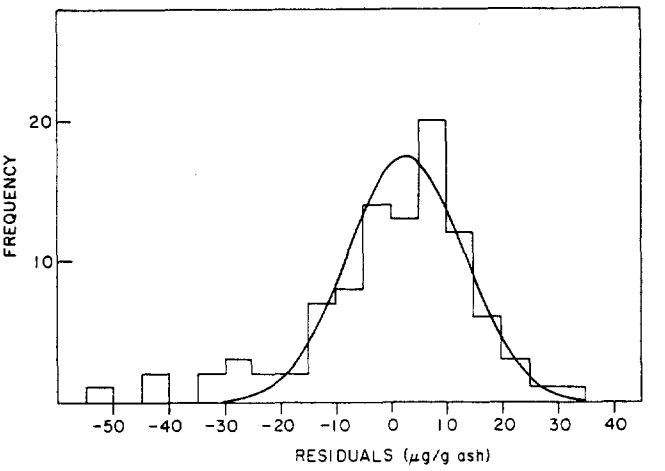

FIG. 40.—Deviation of lead concentration from least squares fit for all data. Gaussian fit of the distribution is also given.

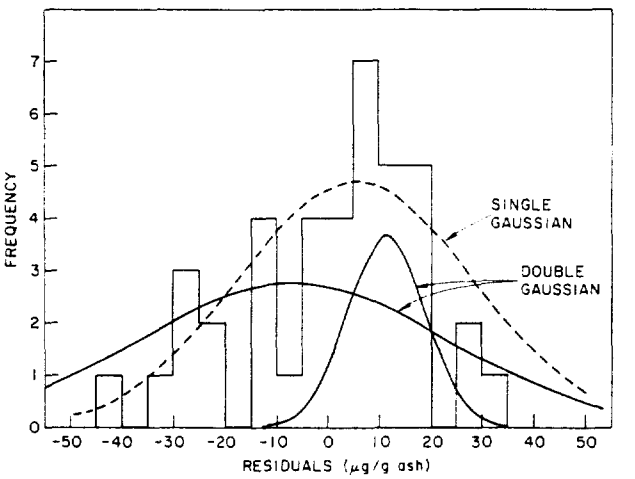

FIG. 41.—Deviations of lead concentrations from least squares fit for "normal" cases over 30 years of age. Best fit for 2 gaussians also shown.

of age), is comparable to the fraction of those smoking more than 11 cigarettes per day.[17]

If one assumes the exponential model of mineral metabolism given in the ICRP Report,[11] and that lead intake is constant over the lifetime, the body should reach equilibrium within a period of time equal to several half-lives of lead in the skeleton. Thus, from the previously estimated biological half-life of about 15 years,[9] the content should level off at about 50 years. This value of the half-life is inconsistent with the data. The slopes of the linear regression curves derived for the concentration versus age data for specimens from people above 30 years of age are very similar to those of the whole group, but with larger variances. However, these slopes are still significantly greater than zero ($P < 0.01$).

The half-life of lead in the body can be estimated if

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one assumes that the half-life and rates of intake and excretion are constant. A single exponential model leads to the equation

$$C = C_\infty(1 - e^{-\lambda t}), \qquad (1)$$

where C_∞ is the skeletal concentration of lead at long times, λ is the decay constant (0.693/half-life), and t is the time in years. An iterative procedure to estimate the parameters of the above equation (Davidon's variable metric minimization)[18] gives a half-life of 71 ± 12 years and a content at long times of 91 ± 13 μg/g ash for the "normal" subjects. This half-life is substantiated to some extent in data on ^{210}Pb excretion rates in radium dial painters in which the biological half-life (which would appear to apply to stable lead, also) is about 57 years.[19]

The increase in stable lead with age is also consistent with previously published data on ^{210}Pb in 128 samples from about 100 subjects from an unexposed midwestern U. S. population.[12] A linear regression of the variation of specific activities in pCi ^{210}Pb/g bone ash with age t was

$$Y = (0.068 \pm 0.024) + (0.0015 + 0.004)t. \qquad (2)$$

The rate of increase is significant ($P < 0.005$), although as with stable lead in subjects over 30 years of age, the coefficient was smaller and not significant at the 5% level, in agreement with the data of Hunt et al.[20] For the radioactive lead this decrease in slope is probably caused by the radioactive decay half-life of 21.4 years, which limits the effective (observed) half-life in the body to a maximum of about 15 years.

Although the half-life estimated here is similar to that found previously, the large variances are a strong indication of the necessity for further examination of the assumptions, particularly those of constant intake, and of the model itself. Thus, the intake of Pb may vary drastically at various times in life or with social change. A particularly large increase may occur in the late teens, because of an increased exposure to cigarette smoke and auto exhaust. Smoking alone may increase the intakes of stable lead by 30%[5] and of ^{210}Pb by 100%.[21]

In contrast to the possible increased exposure to young adults, a decreased exposure above age 70 seems likely. The fraction of male smokers drops from 55.9% in the 17 to 44-year group to 28.4% in the over 65-year group. For women, an even greater reduction in the percent smoking is observed.[17] This means that persons reaching the older ages would be partially selected by smoking habit from a lower lead intake group. This selectivity would result in the reduced number of high values at the older ages as shown in Figure 39, and as noted by others.[5-8]

CONCLUSION

The data presented here on both stable lead and on ^{210}Pb are consistent with those of Horiuchi et al.[8] and of Schroeder and Tipton[7] and demonstrate an increase in the skeletal concentration of lead with age of about 0.6 μg (g ash)$^{-1}$ yr^{-1}. This increase with age requires that 1% of the daily intake be permanently bound by bone, and indicates that the body is not in equilibrium with environmental lead. The soft tissue concentrations of lead were constant with age in non-U.S. subjects.[7, 8] In contrast, Schroeder and Tipton showed a positive correlation between skeletal and soft tissue concentrations in U.S. subjects. Thus, while the skeletal lead may not be toxic and bone may act as a detoxifying "sink" in cases of lead poisoning,[22] the skeletal concentration is an indication of the total exposure. Smoking appears to increase the daily intake of lead. Since the percentage of the human population so exposed is lowest in the very young and the very old, smoking will affect the correlation between age and the concentration of lead in the bone. Further studies of this nature combined with extensive, well-controlled metabolic balance studies are indicated. In particular, the lead concentrations in bone from smokers and non-smokers need further investigation.

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*NON-UNIFORMITY IN THE RETENTION OF THE ALKALINE EARTHS IN ANIMALS AND MAN**

Elizabeth Lloyd

In order to summarize some of the experimental information on which the model of bone turnover being developed for the ICRP must be based, measurements of the uptake and loss of Ca^{45}, Sr^{90}, and Ra^{226} in different bones and in different parts of bone of rabbits, dogs, and man are reviewed.

RETENTION IN TRABECULAR BONE AND CORTICAL BONE VS. WHOLE SKELETON

^{90}Sr in Different Rabbit Bones

The alkaline earths are taken up in different concentrations in different bones. In general, trabecular bone appears to take up more radioisotope than cortical bone but releases it faster. Figure 42 shows the specific activity of ^{90}Sr in different bones in the adult rabbit relative to the mean value for the whole skeleton at different times after a single intravenous injection. This shows about a fivefold difference in the specific activity of the lumbar vertebrae compared with the midportion of the tibia at 10 min after injection. This difference drops to about a factor of two at 460 days when both portions of bone approach the mean values for the whole skeleton.

^{226}Ra in Different Human Bones

In man the pattern of distribution of radium in individual bones as a function of time after intake appears to be somewhat similar to that shown above for the rabbit. Figure 43 shows a linear-linear plot of the concentration of ^{226}Ra in different human bones. This graph summarizes the data available from the MIT studies on human radium.[1] There is a wide spread in the different values for different bones, and for the sake of clarity, the individual points have been omitted here. Figure 44 shows a typical spread of the experimental values for the vertebrae where a straight line on a log-linear plot appears to give a slightly better fit to the data than the linear-linear plot in Figure 43.

In Figure 43 the results have been further subdivided to show the difference in persons who were exposed before age 20 and those exposed after 20 years of age. The duration of exposure varied from 0.1 year to 31 years, but 18 out of 24 cases had a duration of exposure less than 5 years. In addition, it is probable that even in the cases having a long duration of exposure, the most significant exposure took place in the earlier years before more strict regulations were imposed. Figure 44 does, however, show a larger spread in the values for the cases which were exposed at age greater than 20 years. This is in agreement with the findings of Fletcher et al.[2] from ^{90}Sr fallout studies.

* This is a synopsis of a contribution made as a member of the ICRP Committee on the Local Retention Function of Bone-Seeking Isotopes.

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