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● *Paper*

PARTITIONING OF ^{238}Pu, ^{239}Pu AND ^{241}Am IN SKELETON AND LIVER OF U.S. TRANSURANIUM REGISTRY AUTOPSY CASES

R. L. Kathren*
Pacific Northwest Laboratory, P.O. Box 999, Richland, WA 99352 and U.S. Transuranium Registry, P.O. Box 100, Richland, WA 99352

and

J. F. McInroy**
Los Alamos National Laboratory, P.O. Box 1760, Los Alamos, NM 87545

and

M. M. Reichert†
Northwest College and University Association for Science (University of Washington), 100 Sprout Road, Richland, WA 99352

and

M. J. Swint*
U.S. Transuranium Registry, P. O. Box 100, Richland, WA 99352

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***Abstract*—The content of ^{238}Pu, ^{239}Pu and ^{241}Am in the liver and skeleton was estimated from radiochemical analysis of human liver and bone samples obtained at autopsy from former actinide workers whose occupational histories were suggestive of chronic inhalation exposures, with minor skin contamination and wounds documented in a few individuals. For times estimated to be several years to a few decades post intake, 75.8 ± 15.3% of the total ^{241}Am in the skeleton and liver was found in the skeleton (25 cases) as compared with 63.4 ± 24.1% for ^{238}Pu (36 cases) and 53.2 ± 18.2% for ^{239}Pu (43 cases). These differences are significant at the 95% confidence level. Of these cases, 34 included data on both ^{238}Pu and ^{239}Pu and were divided into high and low activity subgroups. The difference in the fractionation of the two Pu isotopes was apparent only in the low activity subgroup, suggesting that the difference observed between the Pu isotopes may be an artifact of the data. The different partitioning of these three nuclides suggests that the ALIs for ^{238}Pu and ^{241}Am may be high by about 25–50% if only the dose to bone is considered and may be high by 12–13%, based on the weighted committed dose equivalent in target organs or tissues.**

INTRODUCTION

THE U.S. TRANSURANIUM REGISTRY (USTR) studies the distribution and concentration of the transuranic elements in humans through radiochemical analysis of autopsy tissues donated voluntarily by occupationally exposed persons (Sw87). This report describes the results of the evaluation of data from registrants with measurable tissue concentrations of Pu and Am who had contributed bone and liver specimens to the registry.

Plutonium and the higher actinides were first identified as bone seekers by Hamilton (Ha47). Since that pioneering report, the tendency of Pu and Am to concentrate in the skeleton and the liver has been confirmed by numerous studies, largely conducted with experimental animals. The biokinetic models proposed by the International Commission on Radiological Protection (ICRP) in *ICRP Publication* 30 (ICRP30) assume that Pu and Am absorbed into the bloodstream will be deposited principally in the skeleton and liver: 45% in each of these

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† Present address: University of Florida, Department of Nuclear Engineering, Gainesville, FL 32611.

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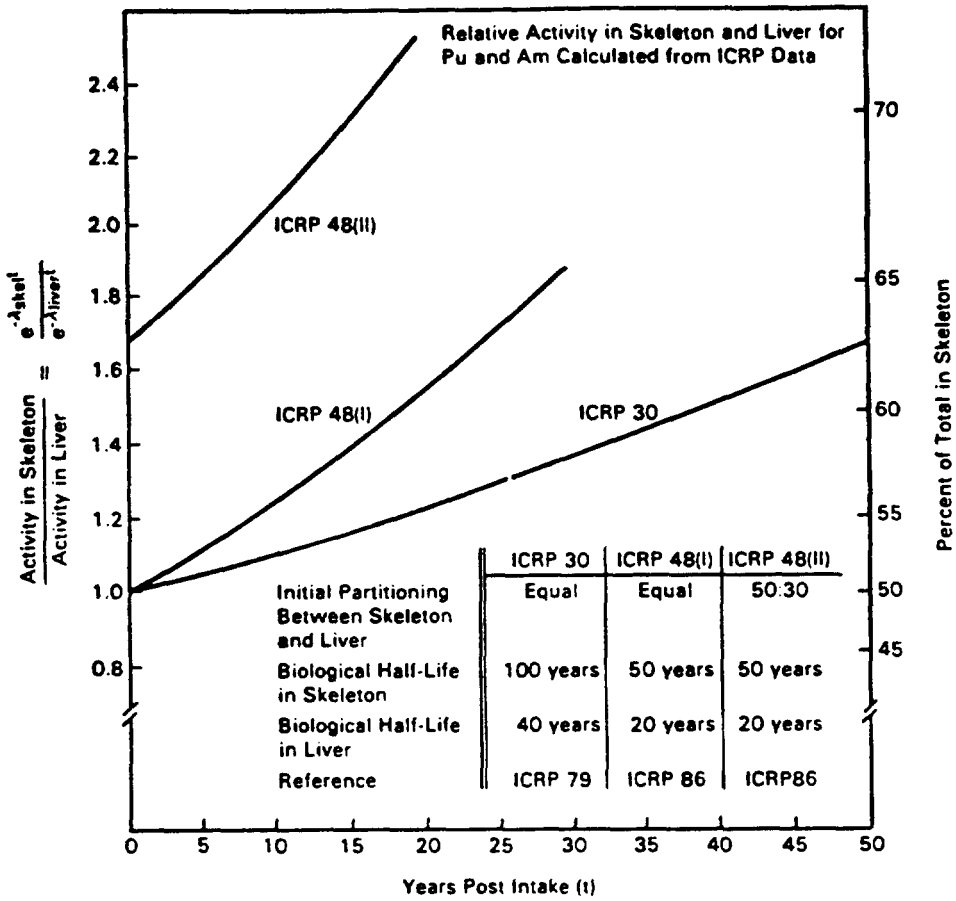

Fig. 1. Relative activity in skeleton and liver for Pu and Am calculated from *ICRP Publication* 30 data.

organs and the remaining 10% in the rest of the body and early excreta (ICRP79). In its most recent publication dealing with the metabolism of Pu and related elements, the ICRP indicates that although there is wide variation in the partitioning of Pu and Am among individual cases, and although the most likely average deposition in skeleton and liver is 50% and 30%, respectively, the ICRP 30 model, ". . . remains an adequate assumption" (ICRP86). Retention half-times of 50 and 20 y for skeleton and liver, respectively, are proposed for Pu, but specific values are not given for Am. Thus, as shown in Fig. 1, based on the ICRP models, the ratio of Pu or Am in bone to that in liver is expected to be unity soon after intake and to increase slowly with time because of a faster clearance from liver than from bone. More recently, McInroy et al. (Mc85) examined a whole body and found (about 25 y after intake) that the distribution of ^{241}Am differed markedly from this model with approximately 80% of the systemic ^{241}Am in the bone and only 6% in the liver.

METHODS

To evaluate the relative distribution of Pu isotopes and ^{241}Am in skeleton and liver, USTR cases with the following criteria were selected: (1) Both bone and liver specimens had been collected at autopsy. (2) These tissues had detectable quantities of one or more of the following nuclides: ^{238}Pu, ^{239}Pu and ^{241}Am.

Individuals in this study typically had 20 or more years of employment at facilities where Pu and Am were processed. For nearly all of the cases, exposure was assumed to have occurred from chronic low-level inhalation over a period of several to many years, since there was, except in a few cases, no history of confirmed accidental acute inhalation, ingestion or a wound except in a few cases. Tissue samples were collected at autopsy from USTR registrants and forwarded to Los Alamos National Laboratory for radiochemical analysis. In general, tissue samples were collected in accordance with the USTR tissue selection protocol (Br81). With few exceptions, the entire liver was collected, along with samples of vertebral body and ribs, and in most cases the sternum. The combined wet weight of the bone samples was typically a few hundred grams. For a few cases, the patella and occasionally samples of other bone (femur, skull, clavicle) were also taken and analyzed. The analytical procedure involved drying and ashing of tissues, dissolution in nitric acid and removal of an aliquot for analysis. After the ad-

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dition of ^{242}Pu and ^{243}Am tracers, separation and isolation of Pu and Am were accomplished by anion exchange followed by electroplating onto stainless steel disks for quantitative α spectrometry. The detailed radiochemical procedures have been described by Boyd, Eutsler and McInroy and by McInroy et al. (Bo81; Mc85).

Concentrations of ^{239}Pu in samples of bone and liver were measured for 43 cases. Similar data for ^{241}Am and ^{238}Pu were available for 25 and 36 cases, respectively. Intake of the three nuclides probably occurred simultaneously as a mixture, although radiochemical data are not available for all three nuclides in all cases. The concentration data were converted to organ content and normalized to a constant base by assuming the organ mass to be that given for Reference Man: 2.8 kg for bone ash and 1.8 kg wet mass for liver (ICRP75). This was done for all cases except for Case 102 for which weights were available for both the entire skeleton and liver (Mc85).

Table 1. Estimated organ content of ^{238}Pu, ^{239}Pu and ^{241}Am at autopsy based on radiochemical analyses.

	^{238}Pu (Bq)		^{239}Pu (Bq)		^{241}Am (Bq)	
Case Number	Liver	Skeleton	Liver	Skeleton	Liver	Skeleton
13	0.28	0.35	4.29	3.62	0.68	3.17
16	0.003	0.09	0.06	0.27		
18	0.25	0.81	23.5	26.7	--	--
20	0.05	0.22	0.10	0.29	0.02	0.04
21	0.08	0.11	4.51	4.67	0.34	0.81
22	0.05	0.43	2.99	2.27	--	--
58	0.07	0.10	2.04	3.47	--	--
60	0.23	0.32				
79	0.01	0.28	0.36	0.38		
82	0.007	0.35	0.05	0.15	0.17	2.98
90	0.005	0.35	0.10	0.36		
93	0.79	0.42	4.30	1.98		
94	0.05	0.54	0.10	0.28		
100	0.12	0.09	0.59	0.66	--	--
101	0.005	0.01	0.28	0.38		
102	0.03	0.77	1.37	2.29	554.0	6623.1
104	0.04	0.15	0.27	0.36	0.13	0.37
106	14.8	6.97	0.15	4.23		
107	--	--	2.04	3.46	0.20	0.99
108	4.04	1.12	145.3	54.7	9.82	21.7
141	28.2	16.4	414.8	326.7		
142	0.59	0.65	27.7	23.3	17.5	52.4
143	0.06	0.05	5.50	2.42	--	--
144	0.007	0.005	0.38	0.61	0.01	0.14
145	0.65	2.49	48.8	81.1	19.4	23.5
147			0.04	0.20	0.02	0.24
148	1.40	0.71	70.3	27.7	7.16	9.50
149	0.04	0.04	2.78	1.93	0.17	0.46
150	0.06	0.15	0.69	2.67	0.26	2.08
151	0.28	0.42	13.7	19.5	--	--
152	4.48	1.14	168.4	100.9	6.50	18.4
153	0.21	0.05	5.34	1.49	0.92	0.86
154	0.005	0.04	0.09	0.34	0.02	0.21
155	0.03	0.04	1.69	1.44	0.13	0.29
156	0.02	0.10	0.11	0.05		
157			0.14	0.20	0.28	0.32
160	--	--	0.94	0.32	0.04	1.00
187	0.05	0.52	1.43	4.28	0.93	10.5
188	0.65	0.63	28.9	14.2	3.75	5.25
190	--	--	0.02	0.07	0.05	0.12
192			1.53	0.98		
193	6.33	3.53				
195			0.09	0.11	--	--
197			0.46	0.38	0.03	0.33
198			0.22	0.32		

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The estimated organ content for each case is given in Table 1.

As full organ weights were not available (except as noted for one case), the estimated organ content expectedly would have some error associated with it—in some cases this would probably be considerable. The largest potential source of error in the estimates of the total organ content is probably associated with the estimate of skeletal deposition. Since the entire skeleton was not collected, the actual weight was unknown, and could have differed from the Reference Man values by as much as 25% (ICRP75). Moreover, as the concentration of Pu throughout the skeleton varies as a function of the fraction of cancellous bone (Du85a) and the ash content of the bone (Ka87), errors in estimating the Pu content of the skeleton could conceivably be as great as an order of magnitude depending on how closely the bone samples were representative of the mean concentration of Pu in the skeleton at death.

The bone samples for these cases were largely ribs and vertebrae, for which the concentration (i.e. activity per gram of ash) would be at least initially expected to be greater than the skeletal average, leading to estimates of skeletal burden that will be too large (ICRP86). Animal studies suggest that at long times after intake, the differences between the actinide concentrations in these bone samples and the mean concentration in the skeleton as a whole will diminish as a result of differential losses from trabecular and compact bone and recirculation of actinide released from liver and bone. This conclusion is supported by human data in two cases which indicate that at long times after intake, the distribution of actinide shifts from a surface deposition to a volume-type distribution (Sc76; Sc81; Du85b). In a recent study of two human half-skeletons from males at long times after intake, which included one of the cases previously mentioned, the concentration in the ribs and vertebral bodies was found to be approximately representative of the skeletal mean, rather than the extremes (Ka87). Hence, the error in the estimate of skeletal content for any one of these long-term deposition cases is not expected to exceed a factor of 2, with the average of all the cases somewhat better. It should be noted in interpreting the results that the same method of determining organ content was consistently applied to all three radionuclides, so regardless of the error in estimating total skeletal content, comparisons within data sets are likely to be valid.

RESULTS AND DISCUSSION

Figures 2a, 2b and 2c show the number of cases and the corresponding percentage of ^{238}Pu, ^{239}Pu and ^{241}Am in skeleton relative to the total amount in skeleton plus liver. Table 2 summarizes the data for each radionuclide. The ICRP 30 model predicts equal amounts of these nuclides will be initially deposited in the skeleton and liver from the transfer compartment (i.e. blood), with the ratio of activity in the skeleton to that in the liver at subsequent time determined by the specific clearance rate constants, as shown in Fig. 1.

In the 43 cases for which data were available for ^{239}Pu, the amount in the skeleton ranged from 23% to 97% of the total in the skeleton plus liver, with a mean of 53.2 ± 18.2%. This mean is not significantly different from that predicted by the ICRP 30 model, but does not support the 70:30 partitioning ratio between skeleton and liver proposed by Thomas, Healy and McInroy (Th84) or the 50:30 partitioning ratio suggested by *ICRP Publication* 48 (1986). In other words, the ratio of the amount in the bone to the amount in the liver was 1.14. This observation is wholly consistent with rate constants and initial equal

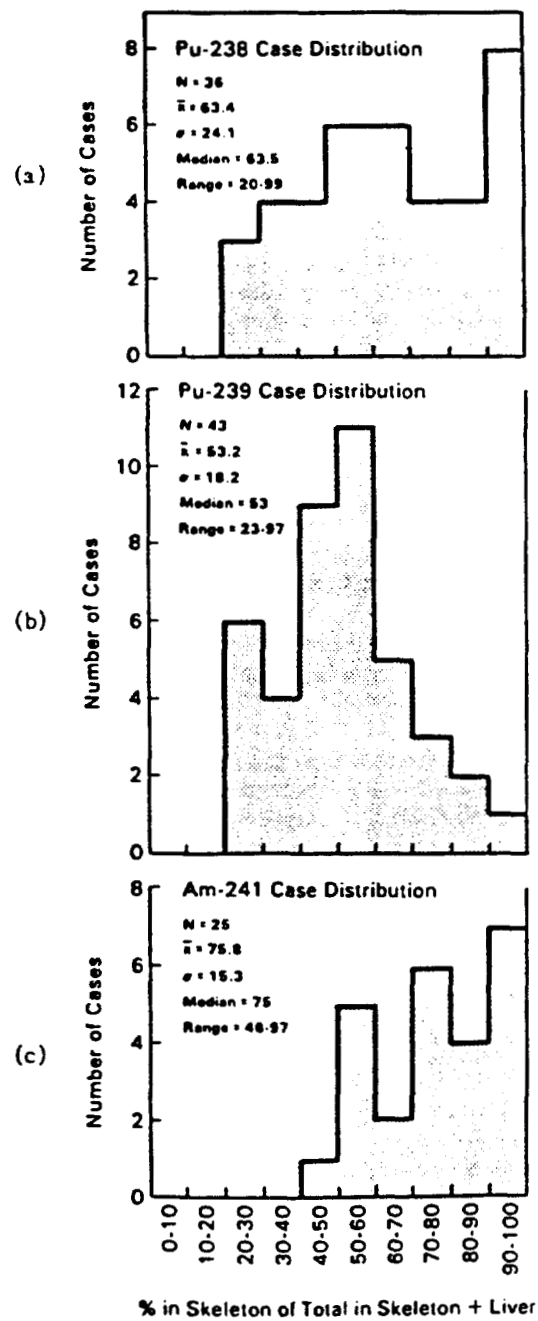

Fig. 2. Case distribution for (a) ^{238}Pu, (b) ^{239}Pu and (c) ^{241}Am.

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Table 2. Summary of observations for the partitioning of ^{238}Pu, ^{239}Pu and ^{241}Am between skeleton and liver in U.S. Transuranium Registry autopsy cases.

	No. of Cases	Activity in skeleton / Activity in skeleton + liver %	Standard Deviation	Median %	Range %
ICRP 30 Model (^{238}Pu, ^{239}Pu, ^{241}Am)	---	50	---	---	---
^{238}Pu	36	63.4	24.1	63.5	20-99
^{239}Pu	43	53.2	18.2	53	23-97
^{241}Am	25	75.8	15.3	75	48-97

distribution of the ICRP 30 model; it does not fit with the ICRP 48 model (ICRP86) or that of Thomas, Healy and McInroy (Th84), which would predict the ratios to initially be 1.67 and 2.33, respectively, and further increasing with time because of the longer half-time in the skeleton.

In the 36 cases where ^{238}Pu data were available (Table 2), the amount in the skeleton ranged from 20–99% of the total amount in both the skeleton and liver, with a mean of 63.4% and a standard deviation of the mean of 24.1%. Thus, the mean ratio of the amount in the bone relative to the amount in the liver was 1.73.

The difference between these means for the Pu isotopes is significant at the 95% confidence level ($p < 0.05$), and it suggests that following inhalation a greater proportion of ^{238}Pu than ^{239}Pu may be deposited in the skeleton. This observation is in agreement with the work of various investigators including Park et al. (Pa80), Muggenburg et al. (Mu80) and Mewhinney and Diel (Me83) in which enhanced dissolution, translocation, and excretion of ^{238}Pu were observed in beagle dogs exposed to $^{238}PuO_2$ aerosols compared to other studies where animals were exposed to $^{239}PuO_2$ (Me83; Gu84; Gu87). However, the observation differs from the ICRP models which do not differentiate biokinetically between the two plutonium isotopes (ICRP79, ICRP86).

The amount of ^{241}Am in the skeleton relative to the total in the skeleton plus liver ranged from 48% to 97% for the 25 cases, with a mean of 75.8 ± 15.3%. Thus, the ratio of the amount in the skeleton relative to the amount in liver was 3.13. The difference between the mean percentage of ^{241}Am in the skeleton and that of the two Pu isotopes was significant at the 99% confidence level ($p < 0.01$), which is consistent with more rapid loss of Am from liver than bone and supports the models put forth by Griffith and coworkers and more recently by Durbin and Schmidt in which the half-life for ^{241}Am in liver is calculated to be about 2 y (Du85b; Gr83). It would also tentatively support a model in which a greater fraction of ^{241}Am than ^{239}Pu is deposited or taken up in the bone.

The 25 ^{241}Am cases were part of the total of 43 ^{239}Pu cases mentioned previously. Therefore, it was possible to compare the relative fractionation of ^{241}Am and ^{239}Pu between bone and liver for these 25 cases. These data are shown in Fig. 3 where the percentage of ^{241}Am and ^{239}Pu in the skeleton (relative to the total in the skeleton and liver) is plotted. The percentages of ^{239}Pu and ^{241}Am in the skeleton tend to follow each other, suggesting that qualitatively the two nuclides are handled similarly within the body.

It is reasonable to assume that the Pu and Am exposure routes were the same in these cases (i.e. low-level inhalation over a period of years). The logical conclusion appears to be that for these long-term exposure cases, a smaller percentage of Am than Pu is retained in the liver. However, the specific details of the exposure of these cases are not known at this time; it is likely that at least some individuals were exposed to a mixture of Pu isotopes, including ^{241}Pu, the parent of ^{241}Am. Thus, at least some of the ^{241}Am in the bone might have resulted from decay of this relatively short-lived (13-y half-life) isotope of Pu. However, decay of ^{241}Pu in bone alone would produce only a relatively small amount of ^{241}Am because of its much longer (458 y) half-life and is insufficient to explain the relatively large fraction of Am in the skeleton. Moreover, the results of the single whole body analyzed by the USTR suggest that about 80% of the systemic burden of Am was in the skeleton at the time of death some 25 y post-exposure (Du85b; Mc85). The data in this paper seem to be more consistent with a model in which the half-life of Am in the liver is relatively short, perhaps on the order of 2 y, as suggested by Griffith et al. and Durbin and Schmidt and certainly less than the 40 or 20 y proposed by the ICRP (Gr83; Du85b; ICRP79; ICRP86).

A similar evaluation was made of the 34 cases in which both ^{238}Pu and ^{239}Pu data were available. The data points are plotted in Fig. 4 and show that the percentage of the two Pu isotopes in the skeleton track together, although there is apparently a greater percentage of ^{238}Pu in the skeleton than ^{239}Pu.

The observed differences between the two Pu isotopes may, in fact, be an artifact of the data and thus may be

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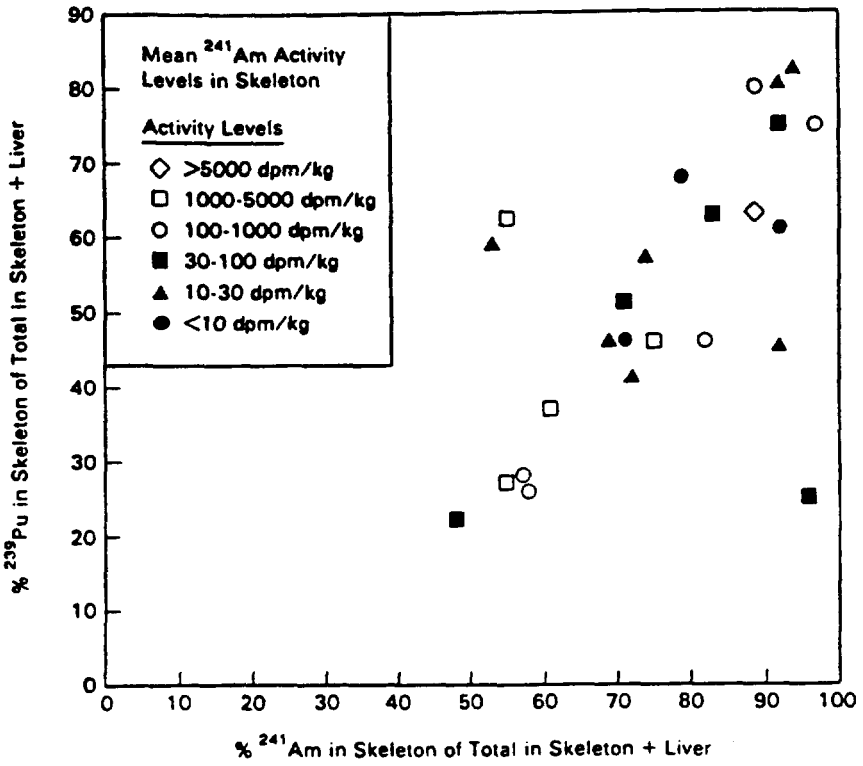

Fig. 3. Fraction of ^{241}Am and ^{239}Pu in bone.

apparent rather than real. Examination of Fig. 4 reveals that those cases with the lowest concentrations show the greatest percentage of Pu in the skeleton. For all 34 cases, the mean percentages of ^{238}Pu and ^{239}Pu in the skeleton of the total activity in the liver and skeleton combined were 63.6 ± 26.6 (median = 58.5, range = 20–99) for ^{238}Pu and 53.9 ± 19.0 (median = 55, range = 22–96) for ^{239}Pu. These means are significantly different at the 95% confidence level (one-tailed t test), and expectedly do not differ appreciably from the values obtained for these isotopes shown in Table 2 for all cases for which data were available for either Pu isotope.

However, if the 34 cases for which both ^{238}Pu and ^{239}Pu data are available are arbitrarily divided into two subgroups on the basis of estimated organ activity, a new and intriguing situation appears. The division into the subgroups was made by including in the low activity subgroup all cases in which the estimated activity in the liver or the skeleton of either Pu isotope was <0.1 Bq. This subgroup included 21 cases. The other subgroup of 13 cases—the so-called high activity subgroup—included all the other cases. Only the low activity subgroup showed a significant difference in the partitioning between the two isotopes. For the 21 cases in this subgroup, means ± standard deviations of 73.1 ± 22.4 and 58.6 ± 17.8 were obtained for ^{238}Pu and ^{239}Pu, respectively; the difference between these means is significant (one-tailed t test, $p < 0.05$). For the 13 cases assigned to the high subgroup, the mean percentages of Pu in the skeleton of the total in the skeleton plus liver were 48.2 ± 19.8 for ^{238}Pu and 46.2 ± 18.3 for ^{239}Pu, clearly not significantly different from each other. Intragroup comparison of the high and low subgroups for both Pu isotopes revealed a significantly greater percentage of skeletal Pu ($p < 0.05$) in the low activity subgroup in both instances. The difference becomes even more striking if a lower cutoff point—e.g. 0.05 Bq—is used to divide the data into high and low groups.

The reason for this apparent difference between the high and low activity subgroups is obscure. The differences could simply be attributable to a data artifact or chance and hence not real or to the greater uncertainty associated with the measured values in the low group. It is even possible that the difference may truly be somehow attributable to or a real function of deposition level. However, regardless of the reasons, the observation suggests that differences between the partitioning of the two Pu isotopes observed with the entire body of cases may in fact not be real, and in any case bears additional study.

CONCLUSIONS

On the basis of the partial human body cases analyzed by the USTR, it appears that, at least for chronic exposures and/or long burden times, the partitioning of the three actinide nuclides ^{238}Pu, ^{239}Pu and ^{241}Am between the skeleton and liver in male human adults is not the same. For ^{239}Pu, there was approximately equal partitioning between the skeleton and the liver, while for ^{238}Pu there was about 1.7 times more in the skeleton than in

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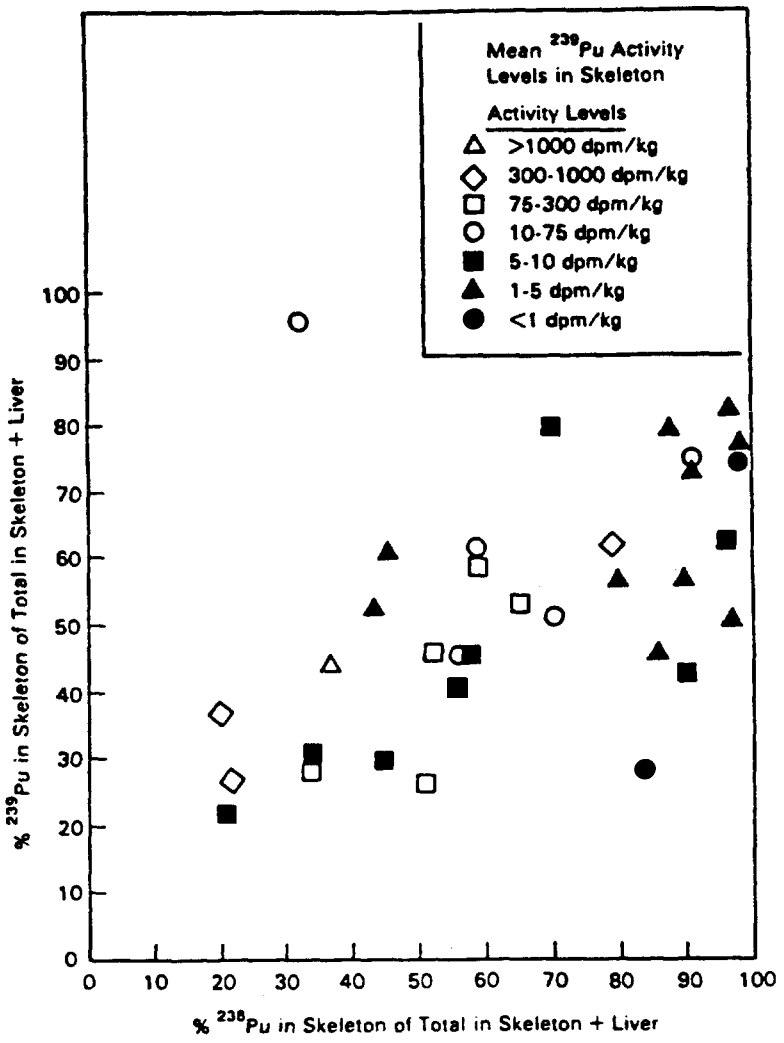

Fig. 4. Fraction of ^{238}Pu and ^{239}Pu in skeleton.

the liver. For ^{241}Am the amount in the skeleton was about 3 times that in the liver.

This difference in partitioning is of significance in biokinetic modeling and the establishment of permissible intake limits for these nuclides. The partitioning factors observed in this report indicate that the annual limits on intake (ALI) for ^{238}Pu and ^{241}Am put forth by the ICRP (ICRP79), which assumed initially equal partitioning between bone and liver, may be high by a factor of approximately 25% and 50%, respectively, considering only the dose to bone. However, these ALIs are only about 12–13% high based on the weighted committed dose equivalent in target organs or tissues following intake by inhalation.

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