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● *Paper*

COMPARISON OF ESTIMATES OF SYSTEMIC Pu FROM URINARY EXCRETION WITH ESTIMATES FROM POST-MORTEM TISSUE ANALYSIS

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***Abstract*—Urinalysis data for 17 individuals with known exposure to Pu were supplied to six laboratories, each of which made an estimate of systemic deposition. In general, the evaluation was done with the Langham model or one of its derivatives, and the values obtained by the laboratories for any single case were typically within a factor of two of the mean for all laboratories. The estimates made from urinalysis data typically were several-fold greater than those made from autopsy data. The results indicate that estimates based on urinary excretion overestimate systemic deposition and tend to confirm previous observations that the Langham equation underestimates urinary excretion.**

INTRODUCTION

SYSTEMIC deposition of Pu in occupationally exposed individuals is commonly assessed by the application of various biokinetic models to specific urinary excretion data of an exposed individual. Although a large number of biokinetic models have been proposed and thus are available for this purpose, most are either based on or are mathematically similar to the original power function excretion curve derived by Langham on the basis of his observations in 18 human subjects experimentally injected with plutonium citrate (Langham et al. 1950, Langham 1956, Langham 1957). The basic Langham equation is

$$Y(u) = 0.002t^{-0.74},$$

in which $Y(u)$ refers to the daily fractional excretion of the injected dose and t the number of days after intake.

Modifications to the original Langham model have been made by Healy (Healy 1957), and later by Beach and Dolphin (Beach and Dolphin 1964), Nelson (Nelson 1967), Leggett (Leggett 1984), Jones (Jones 1985) and others, to account for the continuous infusion into the bloodstream from Pu deposited in a metabolic pool such as the lungs, and by Lawrence (Lawrence 1962) and Snyder (Snyder 1962) to account for multiple rather than single intakes. Over the years, various others including Durbin (Durbin 1972) and the International Commission on Radiological Protection (ICRP) (ICRP 1969) have also made adaptations of the basic Langham model based on specific, albeit limited observations of workers with known exposure to Pu.

Despite widespread application, the Langham model and its derivatives have not been fully satisfactory for estimating systemic depositions of Pu in humans. In a reanalysis of the experimental data originally reported by Langham and his coworkers (Langham et al. 1950), Robertson and Cohn (Robertson and Cohn 1964) found slightly different coefficients for the power function, but in particular noted a shallower slope for urinary excretion in Los Alamos workers at times ranging from 140 to 1750 d postintake. In her detailed examination of the same 18 cases used by Langham plus additional data from persons with long-term occupational exposures, Durbin (Durbin 1972) computed higher daily excretion rates and total excretion than those predicted from the Langham model.

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She attributed this difference primarily to the inclusion of data from only normally functioning excretory systems and derived a new excretion equation based on a combination of five exponentials. The differences were most apparent at times longer than 1500 d postexposure.

More recently, Rundo (Rundo 1981) was able to follow up three of the original 18 cases and observed a significantly greater excretion at 10,000 d postinjection than predicted by either the Langham or Durbin models. In one subject, marked increase in the Pu excretion rate was seen at about 6000 d postexposure, peaking at about 9500 d and thereafter declining. Rundo (Rundo 1981) observed that the ICRP model grossly underestimates the excretion rate at early times postexposure, but by 1500 d overestimates it by greater than a factor of 2. The crossover point occurs at about 104 d postexposure. Moss and Gautier (Moss and Gautier 1985), in a reevaluation of the model and its original data sources, have identified possible errors in the original data which resulted in flaws in the original model.

It is important to examine results obtained with models using urinary excretion data and compare them with direct measurement of the Pu content of the tissues after death. Snyder (Snyder 1962) reported data for a single case in which the postmortem estimate was 642 Bq (17.5 nCi). This compared quite favorably with the estimate of 682 Bq (18.6 nCi) obtained with PUQFUA, a FORTRAN program developed at Los Alamos to compute body burdens from urinary excretion data (Lawrence 1962). Both the PUQFUA and Snyder estimates were within a factor of 2 of the 1.17 kBq (32 nCi), calculated with the Langham equation. This is not too surprising as PUQFUA was based on the Langham model (Lawrence 1978). A larger comparison involving 39 cases was reported in preliminary form by the United States Transuranium Registry (USTR), and was later expanded to 41 cases (Breitenstein et al. 1981, Heid 1983). The USTR study compared the *in vivo* estimates of systemic burdens made from bioassay measurements with autopsy estimates for these cases and found considerable variation between the two (Heid 1983). For all but two cases, the health physics estimates made during life were on the average 6.3 times greater than those made on the basis of postmortem tissue analysis. Another study was carried out by Schofield (Schofield 1982) in the United Kingdom in which the systemic Pu depositions made from excretion and autopsy analyses were compared for 21 cases with similar findings. Nonetheless, Schofield concluded that further and more careful investigation was needed, and that the studies thus far did not seem to warrant a change in the methods used for estimating Pu deposited systemically.

In this study, a comparison is made of 17 individuals using tissues collected at autopsy as part of the program of the USTR. A preliminary study comparing systemic burdens at autopsy with bioassay estimates made during life using some of these same cases has been reported previously (Heid 1983). This prior study did not compare the urinalysis estimates from various laboratories nor did it provide the depth of evaluation reported herein. Moreover, because the cases were not specifically identified in the prior study, it is not possible to specify in a convenient manner which are common to both.

EXPERIMENTAL PROCEDURE

Where available, urinary excretion and other data pertaining to acute accidental exposure that were collected during life for 17 USTR autopsy cases with known exposure to ^{239}Pu were reviewed and analyzed. These cases represented a diversity of exposure conditions and a wide range of estimated systemic deposition (Tables 1 and 2). All of the individuals had long-term chronic inhalation potential, and some were known to have been involved in one or more acute inhalation or skin contamination incidents, or to have incurred possible contaminated wounds. Of the group, Cases 4 and 7 had received DTPA treatment for contaminated wounds.

Each of the six participating laboratories was sent the following available urinary excretion data:

- activity of ^{239}Pu in a daily urine sample
- number of days between the last negative and the first positive urine sample
- number of days between the first positive urine sample and the end of sampling period.

Other relevant information (e.g. whether and when the individual had been involved in an incident and if DTPA was administered) was supplied for most but not all cases, but only in very general terms. For example, a statement might be made that the individual had been involved in an incident at a particular time, but the type of incident and other details were typically not provided. The information in the "Comments" column of Table 1 was not provided as such. Each of the participating laboratories was requested to estimate systemic deposition by application of a biokinetic model of its choice (i.e. the biokinetic model used operationally), making any needed assumptions based on its own individual program and internal practices whenever data essential to the systemic burden assessment, such as the precise date of intake, were missing. No estimate of error was made or requested for the individual values reported by the participating laboratories. To ensure the promised anonymity, neither the laboratories nor the specific method each used to provide the estimated systemic depositions have been identified.

Tissue samples were collected at autopsy from each of the 17 cases and analyzed radiochemically for ^{239}Pu content of Los Alamos National Laboratory. In general the tissue samples were collected in accordance with the USTR autopsy tissue selection protocol (Breitenstein 1981). In all cases, samples of the major reservoir organs for Pu, i.e. liver and skeleton, were obtained along with kidney, spleen, and the lungs and associated lymphatics, although the latter was excluded from the autopsy estimate of systemic deposition. For most cases, heart and/or muscle tissue samples were also available. The details of the radiochemical procedure which involve dry ashing and

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Table 1. Summary of cases.

Case #	Years at Risk	No. Urinalysis Since First Positive	Days from First Positive Urine to End of Sampling	Days Between Last Negative & First Positive Urine	Years Between Autopsy & Last Urinalysis	Comments
1	25	66	6110	60	0	10 possible acute inhalations 1952-67. Wound to index finger, 1969; no detectable activity.
2	20	27	1820	30	7	Acute inhalations 1959 & 1961.
3	25	44	3825	100	3	Acute inhalation $Pu(NO_3)_3$ 1955.
4	28	46	2970	60	2	Acute inhalations 1952, 1953, 1966. Wounds 1960, 1961, 1962 with DTPA treatment each time
5	10	35	3137	27	0	No acute exposures documented.
6	13	31	1413	48	0	31 possible acute inhalations 1965.
7	15	55	3543	~90	0	Wounds 1960, 1963, 1965; latter treated by excision and DTPA; 870 Bq (19 nCi) estimated remaining in wounds
8	15	20	3596	~90	3	No acute exposures documented.
9	18	68	5290	44	0	Wound 1962.
10	10	16	1482	~90	0	Chronic exposure to high fired oxide.
11	9	22	378	263	4	Acute inhalation 1965.
12	27	26	9669	?	0	No acute exposures documented.
13	26	82	8460	?	0	Wounds 1969 & 1970; possible acute inhalation.
14	1	9	10720	27	0	Acute inhalation 1945.
15	14	32	4739	?	0	No acute exposure documented.
16	14	20	2661	?	9	No acute exposure documented.
17	5	54	3248	?	11	Also exposed to Po.

wet ashing, have been described by Boyd et al. (Boyd et al. 1981). After dissolution of the ash in nitric acid, an aliquot was taken for analysis. Plutonium-242 was added as a tracer and the Pu was then separated by anion exchange and electroplated onto a stainless steel planchet for quantitative α spectroscopy (Boyd et al. 1981).

Once the concentration of Pu had been obtained for a given tissue or organ, the total Pu content was determined by multiplying the concentration by the actual mass (wet weight) of the organ, if available, or by the Reference Man value (ICRP 1975). The systemic Pu in the whole body was then obtained by summing the amounts determined for the various organs, excluding, of course, pulmonary deposition or deposition at a wound site. This method is subject to potentially large sources of error since only portions of various tissues and organs were collected and analyzed, and not all tissues and organs in the body were sampled.

The largest potential source of uncertainty in the autopsy estimates is probably associated with the estimate of skeletal deposition. Since the entire skeleton was never collected, the actual weight was unknown, and could have differed from the Reference Man value of 10 kg wet and 2.8 kg ash by as much as 30% (ICRP 1975). Moreover, the concentration of Pu throughout the skeleton is not constant, but highly variable (Durbin 1973, Kathren et al. 1987).

Another potential source of error is the use of wet weights to estimate skeletal deposition. In this study, the autopsy estimates of skeletal deposition were made using wet weight since ashed weights were not available in all cases. In a comparison of 13 cases for which both ash and

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Table 2. Estimated systemic burdens.

Case No.	Estimate Based on Urine Excretion Data, kBq: Participating Laboratory A	B	C	D	E	F	Mean All Labs*	Std. Dev.	Estimate Based on Autopsy Data, kBq	Ratio of Mean Urinalysis Estimate to Autopsy Estimate
1	0.51	0.40	0.18	0.29	0.33	0.40	0.35	0.11	0.09	3.9
2	0.51	0.40	0.29	0.08	0.37	0.51	0.36	0.16	0.40	0.9
3	1.4	1.5	1.1	0.95	0.95	1.1	1.17	0.23	0.77	1.5
4	0.22	0.12	<0.09	0.18	0.15	0.18	0.14	0.08	0.13	1.1
5	0.22	0.15	0.08	0.26	0.18	0.22	0.19	0.06	0.03	6.3
6	0.15	0.12	0.08	0.18	0.26	0.66	0.24	0.21	0.06	4.0
7	7.6	6.6	6.1	3.7	6.0	3.8	5.63	1.57	0.36	15.6
8	0.37	0.18	0.15	0.37	0.26	0.33	0.28	0.10	0.05	5.4
9	0.51	0.37	0.29	0.81	0.37	0.51	0.48	0.19	0.11	4.4
10	0.18	0.12	0.08	0.18	0.08	0.22	0.14	0.06	0.01	10.8
11	0.07	0.08	0.04	0.08	0.08	0.15	0.08	0.04	0.04	2.0
12	0.29	0.18	<0.04	0.26	0.22	0.29	0.21	0.11	0.03	7.1
13	1.6	1.3	0.92	1.3	0.84	1.1	1.18	0.28	0.34	3.5
14	1.6	0.55	0.04-0.81**	0.15	0.59	0.22	0.59	0.53	0.19	3.2
15	0.15	0.15	<0.09	0.33	0.04	0.26	0.16	0.13	0.03	5.3
16	0.22	0.15	0.04	0.04	0.15	0.37	0.16	0.12	0.01	21.7
17	3.5	3.5	3.6	0.62	2.1	5.5	3.14	1.64	0.51	6.1

*<Values taken as 0.
**Reported as range; arithmetic mean of values (1.6) used to compute mean.

Note: Slight differences in ratio of urinalysis to autopsy estimates are attributable to rounding.

wet weights were available, Heid (Heid 1983) observed that deposition estimates made on the basis of wet weights averaged about two times those made using ash weights. The reason for this difference was not stated, but in reviewing the specific radiochemical procedures used, it seems likely to be in some measure attributable to inaccurate or inconsistent determinations of weights. The estimates of deposition were made by determining the concentration in either the wet or dry bone samples and scaling up to Reference Man values. Low wet bone weights may have resulted from desiccation and high dry weights from incomplete ashing. These, in combination, could account for some, perhaps a major portion, or even all of the difference in the estimates made with the wet weights as opposed to those made with the dry weights.

Overall, given the several relatively good bone samples for these cases and considering all possible sources of error, the total uncertainty in the estimate of skeletal content for any individual case is not expected to exceed a factor of 2, including small errors which might have been introduced by residual soft tissue adherent to the bone sample before analysis.

For all tissues, in addition to the normal errors associated with radiochemical analysis, errors could also be introduced by a variation in the sample weight from dehydration prior to analysis, non-representativeness of the sample, and from differences between the actual organ weights and Reference Man values. The latter two are of greater potential significance than the former. Non-representativeness of the sample could result in errors in the estimate of the activity in a particular tissue or organ. However, in the case of the liver, which along with the skeleton is the site of initial deposition and ultimately the reservoir for virtually all of the systemic Pu (ICRP 1979), all or a significant fraction was collected and analyzed thereby minimizing uncertainty from these potentially large sources of error. Considering all potential factors, the uncertainty in the estimated content of the liver is not

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not expected to exceed ±30%. For the other tissues and organs, the uncertainty from all sources is not expected to exceed ±50%.

Assuming that only a small fraction of the systemic Pu was resident outside the skeleton and liver (ICRP 1960, ICPP 1979), the total uncertainty or effect on the autopsy estimates would be small. Most of the uncertainty in the autopsy estimates would thus be associated with the skeleton and liver. Assuming the uncertainties are additive, and that 45% of the systemic Pu is resident in bone, 45% in liver, and 10% in all other tissues (ICRP 1979), the total uncertainty for the autopsy estimates is not expected to exceed +100, −50%, i.e. a factor of 2, at the outside.

RESULTS AND DISCUSSION

Table 2 provides the estimates of systemic Pu burden from bioassay data obtained from each participating laboratory along with those made from the postmortem tissue analyses for each of the 17 cases. Examination of the data in Table 2 generally reveals relatively good agreement among the estimates for any given case by the six participating laboratories irrespective of the specific model and assumptions used. Although there are occasional outliers and exceptions, for any given case the range of values reported by the six participating laboratories typically fell within a factor of 2 from the mean. To some extent, this is not too surprising since all of the participating laboratories used similar models with the original Langham power function as their basis. There were no obvious biases among the laboratories, and the results from each laboratory appear to be internally consistent. However, it also should be noted that there were slight differences in what the laboratories calculated and reported. One laboratory reported individual organ depositions which were summed to get the total deposition for the purposes of this study. Five of the six laboratories reported systemic deposition as of the date of the last urine sample. The other reported committed systemic deposition for soluble Pu, which would be greater than estimates made of systemic burden at the time of the last urinalysis. Despite this difference, this laboratory did not always report the highest values.

Comparison of the values obtained from the urinalysis data with those obtained from autopsy tissue samples revealed a somewhat different picture. Agreement of the mean value of the six laboratories with the value estimated from the autopsy tissue samples was within a factor of 2 in only four of the 17 cases—Cases 2, 3, 4 and 11. Examination of the available records for all the cases revealed nothing to account for the agreement in these four cases, or conversely, the high estimates in the other cases with the exception of Case 7. Strikingly high estimates of systemic deposition were made by all six participating laboratories for Case 7, as compared with the estimate of systemic deposition made for this case from the autopsy tissue analyses. Unlike the other cases, Case 7 had a significant residual deposition in the fingers resulting from three separate contaminated wounds. The first of these occurred approximately 10.5 y before death and was estimated to have initially contained 436 Bq (12 nCi), which was reduced to an estimated 160 Bq (4.4 nCi) through wound cleansing. The second contamination event occurred about 3.5 y before death and involved an initial deposition of 33 Bq (0.9 nCi) which was reduced to an estimated 7 Bq (0.2 nCi) by routine cleansing.

The final contamination event occurred approximately 5 y before death and resulted in an estimated initial deposition of 47 kBq (1.3 μCi) in the tip of the middle finger of the right hand. This was reduced to an estimated 14–17 kBq (380–460 nCi) by cleansing and excision the day of the wound. Daily treatment with 1 g of DTPA was instituted that same day and continued for 8 d following. About 22 mo after the contamination, a second surgical excision was performed, removing an additional 35 kBq (950 nCi) of Pu, and leaving an estimated residual in the wound of about 700 Bq (19 nCi). Thus, at the time of death, the three wound sites contained no more than about 870 Bq (23.6 nCi) or about one-sixth the estimated systemic deposition made from the urinalysis estimates.

Comparison of the deposition estimates made from the urinalysis data with those made from postmortem radiochemical analysis of tissues collected at autopsy is shown graphically in Fig. 1. With the exception of Case 7, which was excluded from the comparison, the urinalysis estimates are consistently higher than those made from the tissues collected at autopsy, particularly at lower levels of Pu. A similar observation was made by both Schulte (Schulte 1975) and Schofield (Schofield 1982). Schulte reported on nine cases, and in all instances the estimates made from urinalysis data exceed those made from postmortem tissue analysis. In two cases, the systemic deposition at autopsy was below detection limits. In Schulte's series of 21 cases, the ratio of urinary estimates to autopsy estimates based on ash weights of tissue was higher than unity in 19 cases, ranging from 0.7 to 18 with a mean of 4.4. The higher ratios were observed typically with smaller estimated depositions. The ratios were even greater, ranging from 2 to 37 when the autopsy estimates were based on wet tissue weights, and averaging 7.5. In another study, Ohlenschlaeger and coworkers (Ohlenschlaeger et al. 1984) evaluated a single case with four different biokinetic models and found that the systemic deposition was overestimated by a factor of 11–16.

Analysis of the data in this, and other studies indicates that the urinalysis results are not a simple multiple of the autopsy results. Rather, there seems to be convergence of the urinalysis and autopsy estimates at the higher levels. This was true with both the 17 cases in this study and the cases of Schulte (Schulte 1975), Schofield (Schofield 1982), and Ohlenschlaeger et al. (Ohlenschlaeger et al. 1984), which have been evaluated in the same way and are included in the presentation in Fig. 1. If a line is fitted to the data, the point of convergence is about 1 kBq. The apparent convergence may be interpreted as indicative of overestimates resulting from the limitations of the Langham equation. At the lower levels, the degree of overestimation is significantly greater, possibly because of

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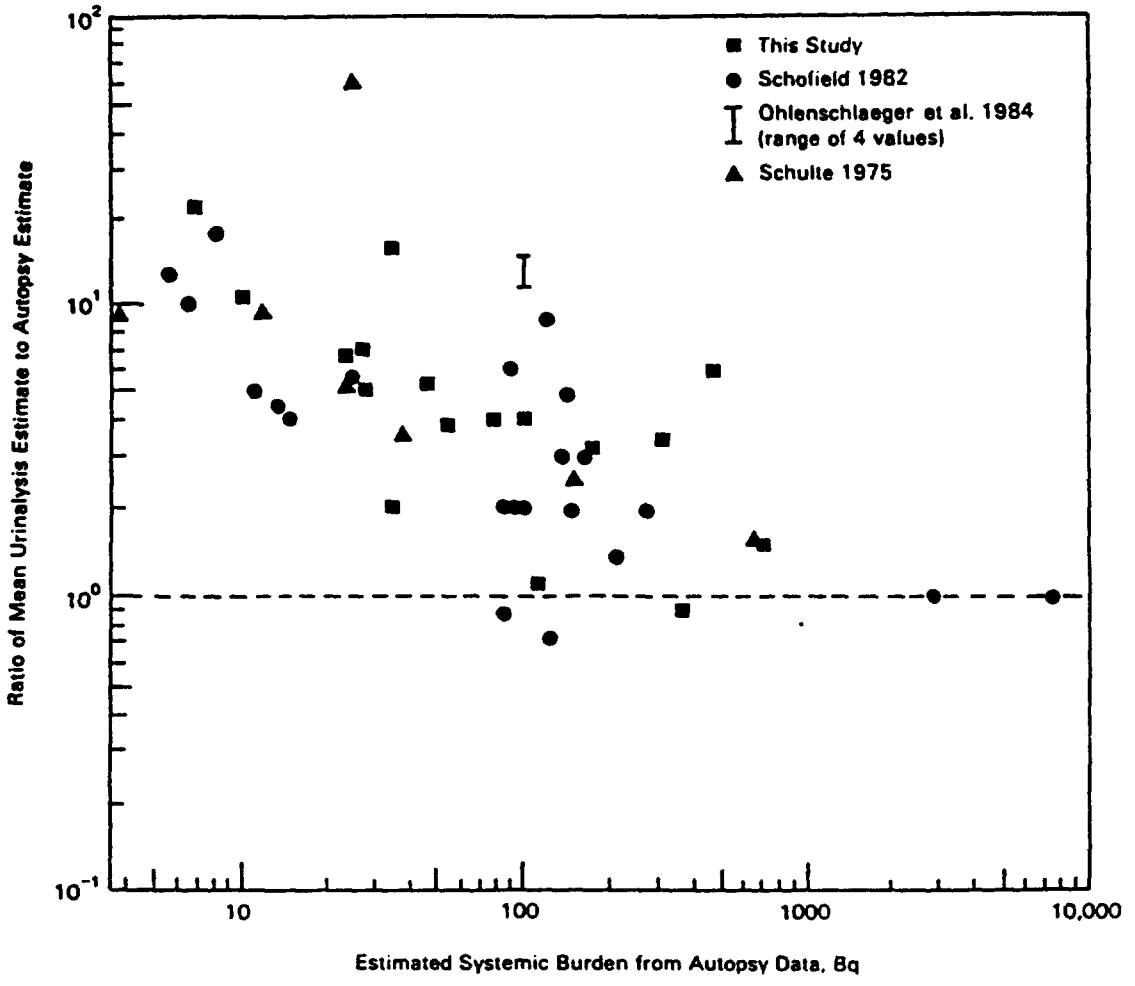

Fig. 1. Comparison of systemic Pu estimated from urinalysis and autopsy data.

limit of detection difficulties or perhaps from interferences associated with incomplete chemical separation of naturally occurring radionuclides. Another possible explanation is that the fractional excretion of Pu is dose dependent, with a greater fraction excreted at the higher doses than at the lower ones.

The higher estimates of systemic deposition made from the evaluation of the urinalysis data relative to estimates made from evaluation of postmortem tissue analysis support the observations of others (Durbin 1972, Leggett 1984, Robertson and Cohn 1964, Rundo 1981) that indicate that the Langham model typically underestimates urinary excretion over long periods of assay and hence overestimates residual systemic deposition. Unlike the previous analyses of both Rundo (Rundo 1981) and Durbin (Durbin 1972) discussed above, this conclusion seems to be independent of the time since intake of Pu. However, the number of cases is not sufficient to support a viable analysis of the time variable.

CONCLUSIONS

The findings in this study of 17 cases show that urinalysis derived *in vivo* estimates of systemic deposition of Pu made from urinalysis data typically exceed those based on radiochemical analysis of tissues obtained at autopsy. In only four cases did the autopsy and urinalysis estimates agree within a factor of 2.

Comparison of estimates of systemic deposition made by six different laboratories from urinalysis results revealed relatively good agreement (typically within a factor of 2) among the laboratories for any individual case. Although each participating laboratory used its own model and assumptions for determining its estimates, there were no obvious biases or systematic errors associated with any one laboratory. The results suggest that the Langham equation and its subsequent modifications may underestimate urinary excretion of systemic Pu, resulting in an overestimate of systemic burden when the equations are used for this purpose.

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