401352

DRAFT

COMPARISON OF PLUTONIUM CONCENTRATIONS IN DIETARY AND INHALATION PATHWAYS AT BIKINI AND NEW YORK AND THEIR RELEVANCE TO Pu URINE CONCENTRATIONS AND BODY BURDENS

— W.L. Robison and V.E. Noshkin

INTRODUCTION

In 1970 analyses for plutonium 239 and 240 in pooled urine samples from Bikini by the HASL-ERDA Laboratory (Health and Safety Laboratory, New York, N.Y.) showed an average concentration of 0.007 pCi/ℓ[1]. In 1971 the average $^{239,240}Pu$ concentration of 3 urine samples from Bikini was 0.004 pCi/ℓ, but in 1974 the average in 10 samples increased to 0.013 pCi/ℓ.[1] (No errors are attached to the values reported in Ref. 1. The average value for any year was obtained by multiplying the Pu concentration of each sample by its volume, summing these products, and dividing by the total volume). In 1975 the average $^{239,240}Pu$ concentration in a pooled 9-ℓ urine sample from Bikini was reported[2] as 0.011 pCi/ℓ. This concentration is similar to the value found in 1974 but is 10 times higher than the concentration (0.001 pCi/ℓ)[2] reported for a 1975 representative population sample from New York.

Because of the toxicity of plutonium and the belief that urine concentrations are a direct indicator of plutonium burdens in the body, the increase in Bikini plutonium levels from early to mid 1970's and the large difference between the concentration found in samples from New York and Bikini is cause for concern.

[redacted]

5009716

DRAFT

The purpose of this report is to present comparative data on Bikini and New York plutonium pathways to man now available from the recent LLL 1975 Bikini survey[3, 4, 5] and other studies. Although a comparison of the pathways is not sufficient to clarify issues on plutonium concentrations in body tissues or excretions, it does show that a Bikini population is exposed to higher plutonium levels through dietary and inhalation pathways than a New York population. We acknowledge that the excretion rates of plutonium and the quantities excreted may differ significantly depending upon routes of entry into the body and that the assessment of these rates and quantities is further complicated by dissimilar physico-chemical forms of plutonium in the environment. We suggest that differences in concentrations in various pathways could account for the relative difference in urinary levels presently found for the two populations. Computed annual plutonium urinary concentrations, however, are very different than reported values[2] from New York and Bikini. Several interpretations are proposed for this noted discrepancy. Furthermore, if the concentrations found in pathways are directly related to the concentrations excreted, then the urine plutonium levels will, with time, become increasingly higher and differ even more from future control samples from New York as the Bikinians rely increasingly on dietary components from their atoll.

PATHWAY ANALYSIS

Inhalation Pathway

The contribution from the inhalation pathway to plutonium concentration in urine, especially at Bikini, is very hard to quantify. In addition to the different activity levels from fallout present in the air at Bikini and New York, resuspension processes that contribute airborne plutonium at New

and Bikini are very difficult but important processes to assess to estimate the cumulative exposure to plutonium from inhalation. Bennett[7] has recently concluded that plutonium from resuspended fallout at New York presently contributes 0.3% and will ultimately contribute an additional 0.2% of the intake that occurs during the original deposition of the fallout debris during any time period. The soil plutonium levels on Bikini Island[5] are certainly, on the average, higher than fallout levels deposited in the United States[8]. Visual observations indicate that the formation of dust clouds or resuspension of surface materials by people or vehicles at Bikini is slight even during very long dry spells. However the available comparative aerosol data shows that some locations on Bikini Island have higher levels of plutonium in the air than expected from world wide fallout and higher levels than those encountered in New York City during comparable periods. We shall show in a following section that resuspension may also contribute elevated plutonium levels to the ingestion pathway at Bikini.

Comparable data on plutonium concentrations in the air exists only for the periods of late May to early June 1970[9] and May 1972[10]. During the 1970 sampling period, $^{239,240}Pu$ levels in surface air were determined at five locations on Bikini Island. The aeolian concentrations during this period of 29 May to 2 June 1970 ranged from 60 to 540 aCi/m^3 (aCi = attocurie).[9] The mean air concentration at 4 sites on the island of Eneu (Bikini Atoll) was only 40 aCi/m^3 during a comparable period.[9] Plotted in Fig. 1 as a function of latitude, are the average plutonium concentrations at ground level at air sampling stations of the HASL sampling network during June 1970.[11] The

concentration-latitude profile was obtained by simply drawing a continuous curve through the available data points.

From this curve the average fallout concentration expected in ground level air at the latitude of Bikini Island (11^{o} 37' N) should be approximately 32 aCi/m^3. This value cannot be considered to be in disagreement with the mean concentration detected on the lesser contaminated island of Eneu.[4, 5] However, it is not only well below the log normal median (134 aCi/m^3) or mean (186 aCi/m^3) concentrations on Bikini Island but is well below the range found over the entire Island. There is little question that the plutonium levels in air during June 1970 were above fallout background levels for the latitude of Bikini Island. The only mechanism by which these levels could have been attained was through resuspension processes on the Island. The median value at Bikini is very nearly equivalent to the mean air concentration at New York during this period. It should be remembered that during 1970 the urine concentrations from Bikini and New York were also comparable. Except for water, no other indigenous material was consumed at Bikini during the two years before 1970. From this comparison of New York and Bikini, the 1970 data strongly suggests there was a close correlation between aeolian plutonium levels and urine concentrations.

In May 1972, air samplers were again operated on Bikini Island at four different locations. The $^{239,240}Pu$ air concentration during this period ranged from less than to 6 to 80 aCi/m^3.[10] The log normal median concentration over the Island was 21 aCi/m^3 and the mean concentration was 34 aCi/m^3. In Fig. 2 are plotted the HASL plutonium air concentrations during May 1972 as a function of latitude.[11] A smooth continuous curve

[redacted]

was again drawn through the data points. The air fallout concentration expected at the latitude of Bikini during May 1972 is approximately 16 aCi/m^3. This concentration is only slightly less than the median value found and only half the mean plutonium air concentration detected at Bikini during this period. In May 1972, 37 aCi/m^3 were detected in the air over New York. Again we find a similarity between the plutonium in Bikini and New York air, and the Bikini mean concentration is still higher than world wide fallout concentrations predicted for this latitude. The 1972 data on air concentration at Bikini also showed a strong geographical correlation.[10] The plutonium air concentrations increased in the samplers from the N.W. to S.E. along the length of the Island. There are, therefore, regions of island that have higher plutonium aerosol concentrations than others. The individual inhalation exposure then must also depend on the time spent working or living in a specific region of the Island.

In summary, without more detailed data from Bikini and without any knowledge on the difference between concentrations from large volumes of open air and the concentrations in an individual's immediate environment resulting from resuspension by a person during his daily routine, we can at least safely assume that the inhalation pathway for plutonium is comparable to New York. The available data strongly suggests that over certain regions of the Island the aeolian concentrations are significantly higher than New York. If resuspension by human activity is also important, there is a much greater chance of higher exposure at Bikini through the inhalation pathway.

[redacted]

Drinking Water

The primary source of drinking and cooking water for Bikini inhabitants is unprocessed rain water obtained from cisterns attached to the newly constructed buildings along lagoon road. The cisterns collect water drained from the windward roof of each building. Ground water has also been used for drinking purposes in periods of drought and will be used in the future when ever cistern water is unavailable. There is presently a high demand for the ground water for agriculture on Bikini Island.

Three of the cisterns were first sampled in June 1975 and analyzed for ^{137}Cs, ^{90}Sr, and plutonium radionuclides. The results[4] are abstracted and shown in Table 1. From an examination of the fallout in rainfall at other Pacific Islands over the period of 1968 to 1974, it was concluded[4] that the ^{90}Sr and by anaology, ^{137}Cs and $^{239,240}Pu$ concentrations in the cistern water did not result solely from world wide fallout. The cisterns contained levels of radionuclides that were locally derived. In support of this contention, two water samples collected in October 1975 from the drinking water tanks on the ERDA supported Marshall Island Research vessel, the R.V. Liktanur, contained 0.6 ± 0.2 pCi/ℓ of $^{239,240}Pu$ and 0.09 ± .04 pCi/ℓ of ^{137}Cs. This water comes from the rain water supply collected at Kwajalein Atoll. The $^{239,240}Pu$ and ^{137}Cs concentrations in Marshall Island rainfall are then approximately 1/20 of the concentrations in the Bikini cisterns. We therefore conclude that the Bikini cistern water contains small, but nevertheless significantly elevated, levels of plutonium radionuclides above those expected from world wide fallout. The higher concentrations could originate from leaching of the concrete cisterns

[redacted]

(the concrete used for the cistern construction was locally derived) or, alternatively, from resuspended airborne labeled particles accumulated on the drainage surface of the roofs and washed into the cisterns with the next rain. If the latter mechanism is correct, resuspension processes contribute plutonium not only to the inhalation pathway but to the ingestion pathway as well.

Bennett[7] has recently published data on fallout $^{239,240}Pu$ in 1972 dietary components in New York that included from 1973 a mean tap water concentration of 0.3 pCi/ℓ. Other data appropriate for comparative purposes are fallout levels in untreated surface water of the Great Lakes.[12, 13] These data are summarized in Table 2 along with the mean and range of plutonium concentrations in cistern and ground water from Bikini. Assuming that water consumption rates for individuals at Bikini and New York are similar, there can be little question that Bikinians experience a higher plutonium body burden from cistern or ground water ingestion than populations in New York. We assume here, of course, that Bikini Island water is the only available source for the present population. It follows that urine levels in the Bikini population would exceed those in a New York population even if this were the only pathway involved with all other pathways contributing similar levels of Pu to the blood stream at both locations.

Dietary Intake

Terrestrial Food Products

The diet for the people on Bikini Island consists of foods imported from the United States and foods grown and obtained locally from Bikini Atoll. The imported foods, on the average, should contain fallout levels

[redacted]

of plutonium similar to those consumed by New Yorkers. Plutonium concentrations in the New York diet are abstracted from a report by Bennett[7] (see Table 3).

Some recent plutonium data from the June 1975 Bikini Survey[5] and earlier data[6] obtained for terrestrial food items are given in Table 4. Only upper plutonium detection limits (with 95% confidence) were available for some samples because of the limited sample size that could be obtained from the existing inventory of food products on the Island. However, from those samples where there was sufficient material to obtain a real number (papaya, pig muscle), it is clear that the Pu concentrations are twice as high as any values reported for terrestrially derived food products in the New York diet.

It is not yet clear just how much of the different food products grown on Bikini Island are actually used in the diet. However, whatever the use (and there is undoubtedly some) and whatever increased future use there may be would lead to higher body burdens and therefore higher urine concentrations of Pu in the Bikini population over the New York population from the food pathway.

For example, if one assumes an average Pu concentration of 0.6 pCi/kg fresh weight in all the food products on Bikini Island and a combined intake of all foods of 100 g/d or 36.5 kg/y, then the yearly plutonium intake would be 21.9 pCi compared to the 1.46 pCi (1.6 pCi minus the shellfish and water) estimated by Bennett[7] for New York. Plutonium levels in a diet entirely derived from Bikini terrestrial foods are 15 times the levels in a terrestrial diet from New York.

Marine Food Products

Marine food products from Bikini Atoll supply a substantial portion of plutonium to the diet for Bikinians. Some Pu concentrations in fish at Bikini Atoll have been published by Nevissi and Schell.[14] These data are abstracted and given in Table 5 along with some data on marine invertebrates.[18] Using both data for fish muscle or eviscerated whole fish and the detection limits as real numbers and weighting by the number of fish in a sample, the mean Pu concentration for fish at Bikini Atoll is 2.2 pCi/kg wet weight. Concentrations in invertebrate muscle average 1 pCi/kg. Assuming an average daily intake of 600 g (or 219 kg/y),[15] the total annual plutonium intake would be 482 pCi.

For comparison, the data listed in Table 3 show a concentration in New York shellfish and fish of 0.011 pCi/kg and 0.0016 pCi/kg, wet weight, respectively. The total annual intake of Pu from New York marine products is 0.024 pCi. The ratio of Pu intake through marine food pathway for Bikini compared to New York is 2×10^4.

DISCUSSION

The estimated annual intake of Pu through various pathways is given in Table 6. With the possible exception of the inhalation pathway, available data indicate that all exposure pathways will contribute a higher Pu body burden to Bikinians than to New York residents. Since surface soil concentrations at Bikini are much higher than New York and the resuspended material created by a person in his immediate environment as a result of daily activities may be a more important factor for estimating the intake by resuspension inhalation than open air Pu concentrations, we believe that the Pu intake via inhalation at Bikini Atoll would exceed that in New York.

An inhalation pathway analysis similar to the one used for the Enewetak Atoll dose assessment[15], based upon a mass loading concept, also indicates that higher intake at Bikini by inhalation is possible. Using a mass loading of 100 $\mu g/m^3$ as in the Enewetak analysis[15], an average 0- to 5-cm Bikini Pu soil concentration of approximately 9 pCi/g, and a 20 m^3/day breathing rate, the annual intake via inhalation would be 6.6 pCi compared with the estimate of 0.2 pCi based on aerosol measurements.

Inhalation experiments[16] and dose models[7,16,17] indicate that approximately 0.1% of the activity inhaled will be excreted in the urine. Assuming an annual intake of 0.2 pCi for both Bikini and New York via inhalation, only a total of 2×10^{-4} pCi of Pu would be found in the urine during the course of a year.

The transfer coefficient across the gut into the blood is assumed to be 3×10^{-5} for ingested Pu[16]. At Bikini this would mean that 15.4×10^{-3} pCi would be transferred to the blood. Of this amount approximately 8%[16,17] or 1.2×10^{-3} pCi would be transferred to the urine. We find from this analysis that the plutonium ingestion pathway contributes more than the inhalation pathway to man at Bikini. In New York the concentration to the total urine level of Pu through ingestion (1.6 pCi $\times$ $3 \times 10^{-5} \times 0.08 = 3.8 \times 10^{-6}$ pCi) is negligible compared to the inhalation route. Therefore the total annual estimated Pu in urine would be 2×10^{-4} pCi for New York residents and $2 \times 10^{-4} + 12 \times 10^{-4} \cong 1.4 \times 10^{-3}$ pCi for Bikini residents. This computation shows that plutonium levels in the urine could be at least 7 times higher at Bikini than New York. This ratio is very similar to the ratio reported to us[2].

These analyses indicate that the differences observed in the Pu concentrations in urine of New York and Bikini populations can be accounted for

by the measured differences of Pu intake via food, water, and air, and are therefore valid within the assumptions for dietary and inhalation intake. However, the absolute quantities predicted by models[2,16,17], based upon the same dietary intake values (Table 6), do not correspond with the recently reported urine concentrations[2]. Table 7 contains data comparing the reported[2] urine concentrations with the quantities predicted from the pathways and models at New York and Bikini.

The reported concentration Pu in urine from the New York population is 10^{-3} pCi/ℓ[2]. Assuming a urine excretion of 1 ℓ/d per person the total Pu excreted via urine per year would then be 0.365 pCi (see Table 7). Bennett[7] estimates the total annual intake of Pu via food, water, and inhalation to be approximately 1.8 pCi for a person in New York. These values then suggest that 20% of the intake is appearing in the urine. This is a much higher percentage than has ever been reported[16] and is higher than percentages normally used for model predictions[7,17]. Only 8% of the amount of Pu entering the blood reaches the urine[17] while 90% of the Pu in the blood is equally partitioned to both liver and bone[7,16,17]. Using the latter values would mean that the bone and liver burdens should increase by 2.1 pCi annually. This quantity, however, is nearly equal to present New York total body burden accumulated since 1954[7]. The Pu concentrations reported for the urine of the Bikini population would, of course, indicate body burdens 10 times higher than those of the New York population.

Bennett's data[7] can also be used to predict the quantity of Pu expected in the urine as a result of the body burden accumulated since 1954. The major source of input to the blood, and subsequently to the urine, is from turnover in the lung and lymph nodes which have half times of 500 and 1000

days, respectively. His data show a lymph nodes burden of 0.40 pCi and a lung burden of 0.12 pCi for the year 1974. The Pu appearing in the urine from these two compartments during the next year would be 0.013 pCi. The contribution to the urine from inhalation and ingestion of Pu during the current year would be 0.0002 pCi (see Table 7). Therefore, the expected annual excretion from previous body burdens and present ingestion pathways is approximately 0.012 pCi compared with the computed annual amount of 0.365 pCi[2]. These results suggest several possible interpretations:

- The urine samples were contaminated at the time of collection, and the Pu concentrations are significantly below those reported.
- The transfer coefficient across the gut for biologically complexed Pu is much higher than 3×10^{-5}, the value developed from animal studies with various Pu compounds.
- The transfer to urine from the blood for ingested Pu is greater then 8%.
- The direct transfer of Pu to blood from the upper respiratory tract is greater than 1%.
- The estimated intake values through food, water, and air are incorrect.
- Any combination of the above.

Interestingly, the plutonium body distribution models[7,16,17] and the Bikini pathway data (Tables 6 and 7) show that the major fraction of Pu presently entering the urine is by ingestion. The absolute quantity of Pu predicted to reach the urine as a result of this annual intake is 1.4×10^{-3} pCi instead of the annual 3.65 pCi computed from the reported data[2]. There would, of course, be an additional contribution to urine Pu levels due to

transport from deep lung and lymph node burdens resulting from previous inhalation exposure. However, the magnitude should be similar to that previously estimated for New York and would therefore lead to total annual urine levels of approximately 0.015 pCi. The reported urine concentrations[2] lead to an annual quantity of 3.65 pCi. If we accept the reported urine concentrations for Bikini then it would seem that the transfer coefficient of Pu across the gut for Pu incorporated in food products must be much higher than 3×10^{-5}.

However, the New York data indicate that the major contribution to the Pu urine concentrations is via the inhalation pathway. If the Pu concentrations reported for urine samples from New York are accepted, the data suggest that the parameters for transfer of Pu to blood must be considerably higher than those presently used. An increase, however, in both the amount transferred from the upper respiratory tract to the blood and the amount transferred from the blood to the urine, which are reasonable for physiological function and chemical transport, still cannot account for 20% of the total annual intake appearing in the urine. Because of this seemingly large fraction of the total intake appearing in the urine, it is difficult to evaluate whether this human data indicates that transfer to urine is greater for human populations than previously assumed based upon animal studies.

SUMMARY

Bikini Atoll may be the only global source of data on humans where intake via ingestion is thought to contribute the major fraction of plutonium body burden. It is possibly the best available source of data for evaluating the transfer of Pu across the gut wall after being incorporated into biological systems. If the plutonium urine data for

the Bikini population is correct, and the estimated dietary intakes are reasonable, it appears that the transfer across the gut wall of Pu incorporated into food products is greater than previously expected.

The New York data, where the major intake is via the inhalation pathway, if accepted as reported[2] also lead to altered conclusions regarding the physiological transport of plutonium. The reported levels in urine would account for as much as 20% of the total estimated annual intake of Pu. This is a fraction that is much higher than believed possible. If, however, the estimated annual intake of Pu and the reported urine concentrations for New York are correct, then Pu is eliminated more rapidly through the urine than previously estimated. This would indicate that lesser body burdens would be expected from intake of Pu.

ACKNOWLEDGMENT

This work was performed under the auspices of the U. S. Energy Research and Development Administration under Contract No. W-7405-ENG-48.

REFERENCES

1. R. A. Conard, A Twenty-Year Review of Medical Findings in a Marshallese Population Accidentally Exposed to Radioactive Fallout, Brookhaven National Laboratory, Report BNL-50424 (1975).
2. T. F. McCraw, USERDA Headquarters, private communications (1976).
3. P. H. Gudiksen, T. R. Crites, and W. L. Robison, External Dose Estimates for Future Bikini Atoll Inhabitants, Lawrence Livermore Laboratory, Rept. UCRL-51879 Rev. 1 (1976).
4. V. E. Noshkin, K. M. Wong, R. J. Eagle, and G. Brown, Preliminary Evaluation of the Radiological Quality of the Water on Bikini and Eneu Islands, Lawrence Livermore Laboratory, Rept. UCRL-51971 (1975).
5. W. L. Robison, unpublished data.
6. V. Nelson, University of Washington, unpublished data.
7. B. G. Bennett, "Transuranic Element Pathways to Man," in Int. Symp. Transuranium Nuclides Environ., (Int. Energy Agency, Vienna, 1976), p. 367.
8. E. P. Hardy, P. W. Krey, and H. L. Volchok, "Global Inventory and Distribution of Fallout Plutonium," Nature 241, 444 (1973).
9. A. E. Smith, and W. E. Moore, Report of the Radiological Clean-Up of Bikini Atoll, Southwestern Radiological Health Laboratory, Rept. SWRHL-111r (1972).
10. O.O.T. Lynch, T. F. McCraw, V. A. Nelson, and W. E. Moore, Radiological Resurvey of Food, Soil and Groundwater at Bikini Atoll, 1972, Energy Research and Development Administration, Rept. ERDA-34, UC-41 (1975).
11. Health and Safety Laboratory Fallout Program, Quarterly Environmental Appendix, HASL-306, Append. July, 1976.

[redacted]

12. M. A. Wallgren, and D. M. Nelson, Plutonium in the Five Great Lakes; Comparison of Surface Waters, Argonne National Laboratory Annual Rept. ANL-8060 Part III, (January-December, 1973) p. 93.

13. J. G. Farmer, V. T. Bowen, and V. E. Noshkin,"Long Lived Artifical Radionuclides in Lake Ontario, Part I. Supply from Fallout, and Concentration in Lake Water of Plutonium, Americium, Strontium 90 and Cesium 137," submitted to Limnology and Oceanography (1976).

14. A. Nevissi, and W. R. Schell, "^{210}Po and ^{239}Pu, ^{240}Pu in Biological Water Samples from the Bikini and Enewetak Atolls," Nature 255, 321 (1975).

15. Enewetak Radiological Survey, Vol. 1, 1973.

16. ICRP Publication 19, The Metabolism of Compounds of Plutonium and Other Actinides, (Pergamon Press, 1972).

17. W. E. Martin, and S. G. Bloom, "Plutonium Transport and Dose Estimation Model," in Int. Symp. Transuranium, Nuclides Environ. (International Atomic Energy Agency, Vienna, 1976), p. 385

18. V. Noshkin, unpublished data.

Table 1

Radionuclide concentrations in Bikini cisterns

pCi/ℓ

Building	^{137}Cs	^{90}Sr	$^{239,240}Pu$
5	2.5	1.1	7.9×10^{-3}
24	1.8	1.9	13.7×10^{-3}
School	1.7	1.4	29.0×10^{-3}

[redacted]

Table 2

Comparative plutonium water concentrations

Location	$^{239,240}Pu$ (fCi/ℓ) Mean	Range	Reference
Bikini			
Cistern water (1975)	17	8-29	4
Ground water (1975)	44	6-122	4
New York			
City tap water (1973)	0.3		7
Great Lakes (1973)			
Superior	0.63		12
Michigan	0.73		12
Huron	0.63		12
Erie	0.17		12
Ontario	0.25		13

DRAFT

Table 3

Fallout $^{239,240}Pu$ in diet - New York 1972

Food product	Consumption kg/yr	Concentration pCi/kg (Fresh)	Intake pCi/yr
Shellfish	1	0.011	0.011
Bakery products	44	0.0085	0.37
Whole grain products	11	0.0060	0.066
Fresh fruit	59	0.0051	0.30
Dry beans	3	0.0048	0.014
Fresh vegetables	48	0.0043	0.21
Root vegetables	10	0.0035	0.035
Poultry	20	0.0033	0.066
Flour	34	0.0028	0.095
Meat	79	0.0026	0.20
Fresh fish	8	0.0016	0.013
Rice	3	0.0016	0.005
Potatoes (peeled)	38	0.0014	0.053
Eggs	15	0.0012	0.019
Macaroni	3	0.0012	0.004
Canned vegetables	22	0.0009	0.019
Milk	200	< 0.0003	< 0.06
Fruit juice	28	< 0.0003	< 0.007
Canned fruit	11	< 0.0002	< 0.002
Tap water (1973)	511	0.0003	0.13
		TOTAL	1.6 pCi

[redacted]

Table 4

$^{239,240}Pu$ in foods grown on Bikini Island

Food product	Concentration pCi/kg (fresh)
Bikini, June 1975	
Pandanus	< 2.7
Breadfruit	< 3.6
Papaya	0.67
Coconut	< 0.27
Squash	< 3.6
Pig muscle	0.72
Chicken flesh	< 6.3
Banana*	< 3.6
Papaya*	< 1.8
Squash*	< 9.0
Pandanus*	< 1.8

Detection limit values = <.

*Data from ref. 6

[redacted] 5009135

Table 5

$^{239,240}Pu$ in Bikini fish and invertebrate muscle

Fish species	No. of samples	pCi/kg wet	Reference
Surgeon fish	3	< 0.45	14
Surgeon fish	1	8.1	14
Convict surgeon	39	< 0.45	14
Convict surgeon	4	12.6	14
Convict surgeon	1	4.5	14
Convict surgeon	4	7.7	14
Panulirus (lobster)	8	< 0.4	18
Grapsus (crab)	5	1.7 ± 0.5	18

[redacted]

Table 6

Estimated annual intake of Pu[+]

Pathway	Bikini pCi/yr	New York pCi/yr	Ratio Bikini/New York
Inhalation	$\geq$ 0.2	0.2	$\geq$ 1
Drinking water[*]	8.7	0.13	58
Terrestrial foods	21.9	1.4	15
Marine foods	482	0.024	2×10^4

[+]See text for discussion and assumptions for each pathway.

[*]Assuming cistern water only. Any use of ground water would increase this estimate.

[redacted] 5009737

Table 7

Concentration of Pu in Urine —

Measured vs Predicted

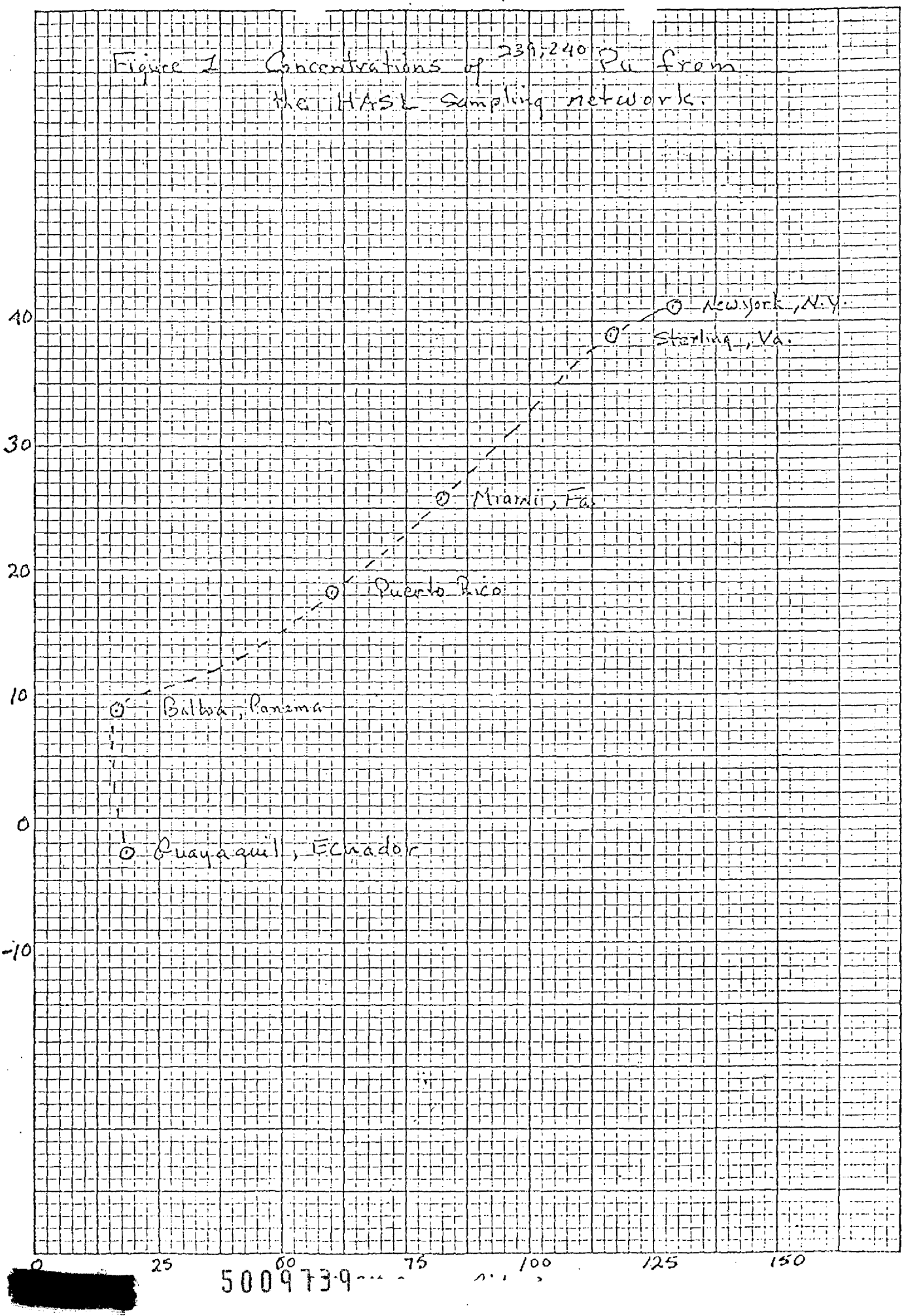
Location	Estimated annual intake in pCi		Annual predicted Pu in urine via ingestion and inhalation from estimated annual intake†			Annual quantity of Pu excreted* pCi
	Inhalation	Ingestion	Total pCi	% Ingestion	% Inhalation	
New York	0.2	1.6	0.0002	3	97	0.365
Bikini	0.2	513	0.0014	86	14	3.65

*Computed from assumed excretion rate of 1 ℓ/d and concentrations reported in Ref. 2.

†Model and parameters are those summarized in Ref. 7 and 17.

[redacted] 5009738

Figure 1 Concentrations of $^{239,240}Pu$ from the HASL sampling network.



KOE 5 X 5 TO THE ½ INCH 359-6 KEUFFEL & ESSER CO. MADE IN U.S.A.

[redacted] 5009739

Figure 2 Concentrations of $^{239,240}Pu$ in air from the HASL Sampling network.