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A comparison of $^{239+240}$Pu post-mortem measurements with estimates based on current ICRP models

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Measurements of $^{239+240}$Pu in human tissues, from nuclear weapons testing, provide an invaluable source of data for verifying the uptake and distribution of radionuclides in the body. Measured concentrations of $^{239+240}$Pu in lung, tracheobronchial lymph nodes, liver and skeleton have been compared with concentrations calculated using estimated plutonium intakes, the ICRP Publication 66 Respiratory Tract Model and the ICRP Publication 67 biokinetic model for plutonium. Measurement data tend to fall between the concentrations estimated on the basis of Type M and Type S absorption parameters. This indicates that the models represent the movement of plutonium through the body reasonably well.

Introduction

It is important to compare human tissue data with model estimates to ensure that the models correctly represent the processes involved. Measurements of nuclides in humans resulting from atmospheric testing have been carried out since the late 1950's[1,2] and modelling of the behavior of the nuclides since the early 1960's.[3,4] Post-mortem data of fallout nuclides are an invaluable source of information for studying the uptake and movement of radionuclides through the human body since exposures have occurred on a world-wide scale, thereby allowing measurements to be carried out in large numbers of individuals.

In 1976 BENNETT,[5] using the model developed by the International Commission on Radiological Protection (ICRP) Task Group on Lung Dynamics[6] together with calculated $^{239+240}$Pu intakes, estimated the $^{239+240}$Pu concentrations in various tissues. He used the lung model parameters, i.e., assumed that fallout $^{239+240}$Pu behaved with Class Y lung clearance characteristics and the biokinetic models recommended in ICRP Publication 19.[7] MCINROY[8] compared these computed estimates of $^{239+240}$Pu with measured concentrations of these radionuclides from an extensive post-mortem programme carried out in the United States. This study found reasonable agreement between estimated and measured values for lung and liver. However, estimated values for skeleton and lung-associated lymph nodes (LALN) did not fit the measured data so well. MCINROY[8] suggested that this resulted from insufficient sensitivity of the analytical procedures and the non-uniform distribution of $^{239+240}$Pu throughout the skeleton. MCINROY[8] stated that the extrapolation of the activity concentration of the whole skeleton from a specimen of, usually trabecular, bone could lead to large errors. The estimated LALN concentrations, in particular, did not tie in with the measured data, i.e., an order of magnitude difference. An explanation suggested by ICRP in its Publication 48[9] is that humans accumulate less $^{239+240}$Pu in the LALN than the previous ICRP models[6,7] had suggested.

Since the work of BENNETT[5] and MCINROY,[8] ICRP has updated its respiratory tract model[10] and its biokinetic model for plutonium.[11] The objective of the study reported here was to compare $^{239+240}$Pu post-mortem data with the expected concentrations in tissues estimated using the new ICRP models. The post-mortem data used in the previous comparative study[8] were augmented with more recent measurements. By examining data that spans from 1953 to 1985, the reliability of the models over extended periods of time can be more accurately gauged.

Experimental

Inhalation of plutonium was the only route of intake considered, this being the predominant exposure pathway. Although higher levels of $^{239+240}$Pu enter the human body via ingestion than inhalation, the very low fractional absorption across the gastrointestinal tract, $5 \cdot 10^{-4}$,[12] means that inhalation remains the major source of intake even when fallout $^{239+240}$Pu concentrations in surface air are low.[13]

Modelling of tissue activity concentrations

Air concentrations were estimated for all relevant years for each location where tissue measurements were available. Direct measurements of $^{239+240}$Pu concentrations in air were not routinely made until the late 1960's,[14–19] so where none were available, other methods were used to estimate surface air concentrations. The methods used are given below in order of descending preference:

(1) The ratio of $^{239+240}$Pu concentration in the air to the deposition rate of $^{239+240}$Pu reported by BENNETT[1] was used to estimate $^{239+240}$Pu air concentrations where only deposition rates[15,16] were available.

(2) The average ratio of $^{239+240}$Pu to ^{90}Sr or ^{137}Cs air concentration[15,22] was calculated from years where both sets of measurements are available. Then, for years where no $^{239+240}$Pu air concentrations were available, the

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concentrations were estimated using the product of the ratio and the ^{90}Sr or ^{137}Cs air concentrations.

(3) The average ratio of ^{90}Sr air concentrations to the yearly depositions was calculated from years where both sets of data were available. The years where there were no ^{90}Sr air concentrations available were completed by multiplying the ^{90}Sr deposition[15,23] by the ratio. The $^{239+240}$Pu air concentrations were then calculated using the method given above.

(4) Plutonium-239,240 air concentration measurements or predictions made at a similar latitude.

(5) Plutonium-239,240 air concentrations predicted using the atmospheric transport model developed by BENNETT.[24]

The estimated air concentrations used here are based on intakes estimated for Akita, Japan. Post-mortem data were available from locations that vary from latitudes 34.5° N to 60° N, with most data coming from within a few degrees of 40° N. Since the air concentrations are linked significantly to latitude, intakes estimated for Akita, at 39′ 40″ N, provide a reasonable estimate of the $^{239+240}$Pu inhalation intakes. The yearly intakes were then calculated from the product of the air concentrations and the annual inhalation rate given in ICRP Publication 66[10] for sedentary male workers.

An in-house computer program, LUDEP (Lung Dose Evaluation Program),[25] which implements the new ICRP Respiratory Tract Model,[10] was modified to include the most recent ICRP biokinetic model for plutonium.[11] It was then used to calculate the $^{239+240}$Pu activity concentrations in lung, tracheobronchial lymph nodes (TBLN), liver and skeleton from the estimated $^{239+240}$Pu intakes. This study uses the term TBLN which is assumed to refer to both LALN, as used in the ICRP Publication 48[9] and thoracic lymph nodes (LN_{TH}) given in the ICRP Publication 66.[10] Two sets of values were calculated based on (1) slow (Type S) and (2) moderate (Type M) default ICRP Publication 66[10] absorption parameters from lung to blood. The activity median aerodynamic diameter (AMAD) was assumed to be 1 μm, the default recommended by ICRP[10] for environmental exposures in the absence of specific information.

Post-mortem data

Post-mortem data from individuals with no known occupational exposure were collated from many sources.[3,14,26–39] Required information was the age of the individual at death, the residence of the individual at death and the $^{239+240}$Pu concentrations of the various organs. In total 2633 measurements were used, taken from 15 studies. Of these, 960 were measurements of $^{239+240}$Pu concentrations in liver, 529 in bone, 786 in lung and 358 in TBLN. Some of the studies reviewed presented measurements in "bone", whereas other studies gave details of the bone type. For the purposes of this comparison all bone types were taken to be "skeleton". All activity concentrations were converted to mBq · kg^{-1} fresh weight. Where this conversion required organ masses, which were not provided in a study, those of ICRP Publication 23 Reference Man[40] were used.

This study has been based only on adult post-mortem data as there is evidence that there are significant differences in the biokinetics of plutonium between children and adults.[11] There were insufficient data to permit independent analyses on children. Therefore, individuals were excluded if they had not reached adulthood when atmospheric nuclear weapons testing began, the assumptions being that adulthood was 20 years old and the commencement of large scale weapons testing was 1952.[41] Median activity concentrations were calculated in terms of year and location. The median rather than the mean was used as it is unaffected by extreme values.

Ratios of tissue concentrations

The ratios of concentrations in different tissues are very useful as they enable the uncertainties in the assessment of intakes to be eliminated and thereby allow the robustness of the models themselves to be more thoroughly examined. Since, as noted above, there had been discrepancies in the modelling of plutonium transfer from lung to TBLN, the ratios of TBLN to lung concentrations were calculated. The ratios of lung to combined liver and skeleton activities were also calculated. ICRP Publication 67[11] states that it can be assumed that the liver and skeleton contain 80% of plutonium that has been absorbed into the systemic circulation. Therefore, the ratio of lung to combined liver and skeleton activities gives a good indication of the absorption rate of $^{239+240}$Pu from the lung to the rest of the body.

Results and discussion

Comparison of measurements and estimated tissue concentrations

Figures 1 to 4 show the comparison of measured and estimated $^{239+240}$Pu activity concentrations in the liver, skeleton, lung and TBLN.

For liver (Fig. 1) the estimated values assuming Type M absorption parameters provide a better fit than those using Type S, although both sets are slightly lower than the measurements. For skeleton (Fig. 2) the measurements tend to lie between the values assuming Type M and Type S absorption parameters but, as for liver, with Type M providing the slightly better fit. For the lung (Fig. 3) the measurements lie in general between Type M and Type S. However, in contrast to the liver and skeleton the estimates using Type S absorption parameters provide the better fit. The trend of the measured data with time also shows good agreement with Type S. For the TBLN (Fig. 4) there is wide variability in the data but estimated concentrations assuming Type S give the better fit.

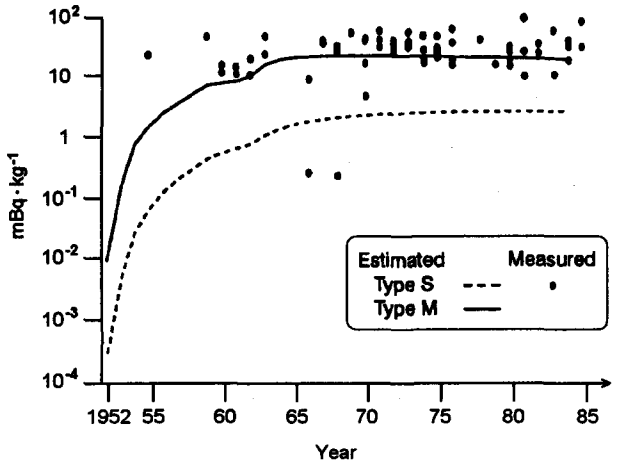

Fig. 1. Plutonium liver activity concentrations

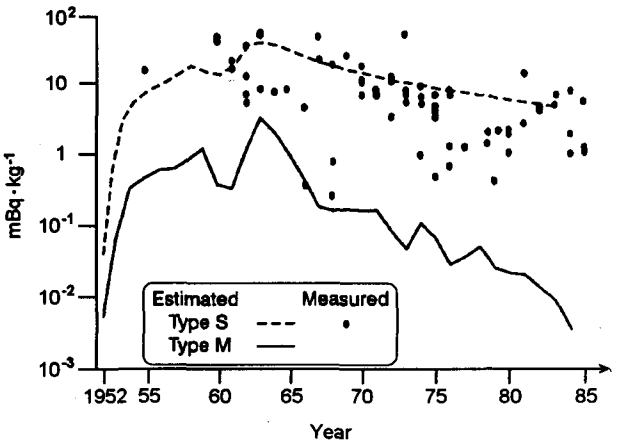

Fig. 3. Plutonium lung activity concentrations

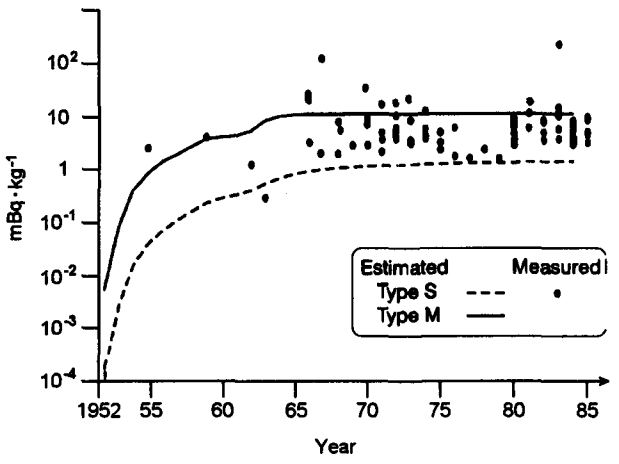

Fig. 2. Plutonium skeleton activity concentrations

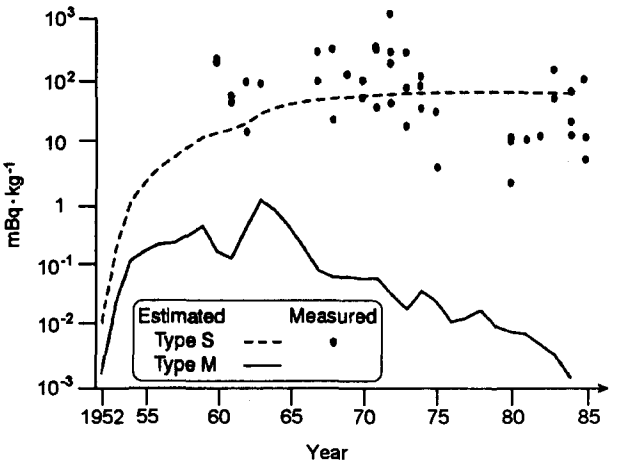

Fig. 4. Plutonium TBLN activity concentrations

Tissue concentration ratios

Figure 5 shows the ratio of TBLN to lung concentrations for measured and estimated data. Again there are great ranges in the values based on measurement data but the data are fitted reasonably well by Type S. Figure 6 shows the ratio of lung to summed liver and skeleton activities. All the measurements with one exception lie between Type M and Type S with the data following a similar trend over time as Type S.

ICRP, in its Publication 71,[42] recommends that environmental plutonium be assumed to be Type M in the absence of specific information. It also states that $^{239+240}$Pu, resulting from nuclear weapons testing, seem to be broadly consistent with the previous ICRP classification of Class Y, which generally corresponds to Type S. These judgements are supported by the findings of this study which shows that most of the measurement data fall between the estimates calculated assuming Type M and Type S absorption parameters.

The comparison for lung and TBLN data indicates that environmental $^{239+240}$Pu, when taken into the body, exhibits Type S characteristics. However, a considerable number of measurement data fall between estimates made using Type S and Type M. There are difficulties with the measurements of $^{239+240}$Pu in TBLN in that the samples are small and the levels of $^{239+240}$Pu low that can result in large errors on the measurements. In addition, there could have been incomplete separation of the lung and TBLN that would result in the concentration in the lung being too high and that in the TBLN being too low. However, the estimated concentrations for TBLN fit the measured data much more closely than the values previously estimated using the ICRP models. This may be accounted for by the revision of the ICRP's Respiratory Tract Model[10] to give slower transfer rates from the lung to TBLN and to include absorption to blood from the TBLN.

For the reasons given above the TBLN to lung concentration ratios estimated in this study are about an

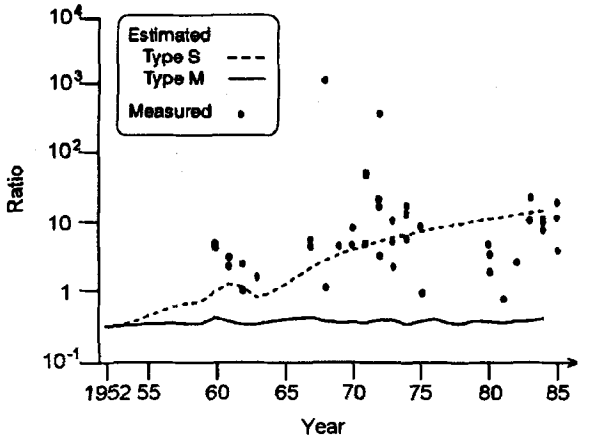

Fig. 5. Ratio of TBLN to lung plutonium activity concentration

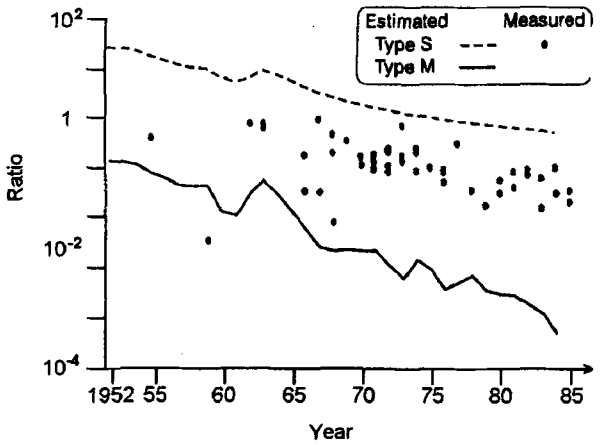

Fig. 6. Radio of lung to liver and skeleton plutonium activities

order of magnitude lower than those estimated by BENNETT, e.g. for 1976 using the ICRP Publication 19[7] model and assuming Class Y parameters the ratio is approximately 290 whereas using ICRP Publication 66,[10] with Type S parameters, gives a ratio of about 10. As can be seen from Fig. 5 there is now much closer agreement between the measured and estimated data that indicates that the new model more adequately represents the transfer of plutonium from lung to TBLN.

The measurements of $^{239+240}$Pu concentrations for liver are slightly higher than the estimates made assuming Type M characteristics whereas the skeleton data tend to be marginally lower than Type M. This seems to suggest that the ratio of partition of plutonium between the liver and skeleton is higher than that predicted by the ICRP biokinetic model.[11] ICRP Publication 56[43] discusses the large variability in the distribution of plutonium between the liver and skeleton. Fox et al.[44] found a significantly correlated increase in the concentration of $^{239+240}$Pu in liver with age at death and a correlated decrease in skeletal content. In ICRP Publication 48[9] this was considered to be suggestive of plutonium transfer from skeleton to liver throughout life. Since the data reported here excluded younger subjects, the age at death tends to be at least over sixty years old, *i.e., it would* be expected that some of the initially deposited $^{239+240}$Pu could have transferred from the skeleton to the liver. This results in higher amounts in the liver compared to the skeleton.

Since all the ratios of lung to liver and skeleton activities for the measured data, with one exception, fall between estimates made assuming Type M and Type S parameters this indicates that the ICRP models work well. Preliminary work indicates that by increasing the fraction of $^{239+240}$Pu that is rapidly dissolved, f_R, whilst keeping the other parameters (fast dissolution rate, s_R and slow dissolution rate, s_s) as for Type S, the fit of the estimates to the ratios based on measurements can be greatly improved.

BENNETT assumed that the AMAD of fallout $^{239+240}$Pu was 0.4 μm. More recent work suggests that 0.6 μm is appropriate for aged fallout particles.[45] For this study the sensitivity of levels of $^{239+240}$Pu concentrations in the tissues according to AMAD was considered. Assuming values of 0.4, 0.6 or 1 μm does not create significant differences in the estimated concentrations especially when considering the variability in the measurement data.

Conclusions

The measurement data indicates that inhaled $^{239+240}$Pu resulting from nuclear weapons testing exhibits behavior which is intermediate between concentrations estimated assuming Type M and Type S absorption parameters. It is concluded that considering the many inherent uncertainties the models represent the movement of $^{239+240}$Pu throughout the body reasonably well.

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