COMPARISON OF ICRP 30 MODELS TO NEWER MODELS

D. Allen, J. W. Neton, L. J. Elliott
National Institute for Occupational Safety and Health, 4676 Columbia Parkway, Cincinnati, Ohio, United States of America, 45226 Fax: 513-458-7125

Abstract

To estimate the probability of causation in an occupational radiation exposure compensation program, it is necessary to reconstruct the dose for the tissue or organ that was diagnosed with a primary cancer. Since the United States regulatory bodies use ICRP 30 recommendations for internal dosimetry, it was unclear whether ICRP 30 or the more current recommendations of the ICRP should be used. Since the Energy Employees Occupational Illness Compensation Program evaluates the radiogenic origin of a cancer by using probability of causation methods, the annual dose rather than the committed dose is required to correctly associate the dose with a particular cancer. With that in mind, the ICRP 30 and the newer ICRP 66/67 models were compared to evaluate any differences between the two models. This paper compares differences in calculated annual doses delivered to several organs over a period of 50 years as a result of inhalation exposures as a function of both intake and uptake. The major factors that contribute to the observed differences is discussed.

1. Introduction

Under the U.S. Energy Employees Occupational Illness Compensation Program Act [1], the National Institute for Occupational Safety and Health (NIOSH) is tasked with reconstructing internal and external organ doses for certain U.S. Department of Energy (DOE) facility workers who are covered under the provisions of the Act. As part of this effort, it was necessary to select an internal dose model that could be used to calculate the annual dose delivered to organs as a result of intakes of radioactive material. In the United States, the current internal dose models used in Federal occupational exposure regulations are those contained in ICRP Publication 30 [2]. While these models may be suitable for regulatory compliance purposes, there are more recent ICRP publications that provide a number of features that are desirable under a worker's compensation program. This paper presents a comparison between internal dose results obtained from selected exposure scenarios using either the ICRP 30 model or those contained in ICRP publication 66 and 67 [3].

Since inhalation is the most likely route of exposure in an occupational setting, the starting point for this comparison was to calculate the annual dose per unit inhalation intake to several organs using both models. For this paper, insoluble ^{239}Pu was chosen as a representative actinide. Based on the metabolic behavior of plutonium, the dose to the lung and liver were selected for comparison. The default particle size recommended by publications 30 and 66 were used (i.e. 1 micron AMAD for ICRP 30 and 5 micron AMAD for ICRP 66). The relative annual dose equivalent per unit intake delivered to the lung and liver is provided in Figures 1 and 2, respectively.

Inspection of the plots indicates that the ICRP 66 model produces a lower lung dose than ICRP 30 for each year after inhalation. It also clearly indicates that the ICRP 30 model exhibits a decrease in annual dose to a constant dose rate, not to zero. This is due to ICRP 30 model's inclusion of a lymph node compartment with an infinite half-life. This effect dominates the lung dose within 20 years of inhalation. The ICRP 66 model on the other hand provides

physiological clearance rates for each compartment and then assigns the absorption rate the same for each (with the exception of one of the extra-thoracic compartments). For solubility class S material the absorption rate of 99.9% of the material correlates to a half-life of about 6930 days. This is much longer than the longest clearance rate of ICRP 30 (with the exception of the infinite half-life compartment). This implies that the ICRP 66 model delivers the material to the transfer compartment slower than ICRP 30.

Much of the difference in the lung dose is due to the continuous exposure from the small amount of the inhaled material in the lymph nodes that does not clear the lungs. The additional plot (labeled "no lymph node") in Figure 1 shows the ICRP 30 lung dose if the final dose-rate from the lymph nodes are subtracted for each year. With this line superimposed on the graph, the differences in the remaining clearance rates are clear.

The plot of the annual liver dose equivalent over time provided in Figure 2 indicates that the ICRP 30 model exhibits a steep build-up over the first 10 years, primarily due to the 1000 day half-life of the lymph node compartment. Once the activity is depleted from lung and built up in liver, the liver clearance rate begins to dominate, causing a steady decrease in liver dose. The ICRP 66/67 metabolic models provide a slower absorption rate from the lung but a faster clearance rate from the liver. This newer model also provides one more important difference. The new model accounts for material re-incorporated into tissues after it is released from an organ. ICRP 30 simply assumed any material released from an organ was immediately eliminated from the body, while ICRP 67 allows for recycling of material that is cleared form an organ. The effect of this recycling is evident in Figure 3 which provides a plot of the annual dose equivalent to the liver per unit of ^{239}Pu injected directly into the blood stream. Even though the liver clearance rate for ICRP 30 is much slower than that of ICRP 67, the inclusion of a recycling component produces a longer effective clearance rate for the ICRP 67 model.

The overall effect on the annual liver dose equivalent is that the absorption rate from the lung and the clearance rate from the liver are both reduced, causing a slower build up to a dose-rate that is sustained much longer. Over a 50 year time period, the difference is a factor of about 8, however, this difference continues to shrink as additional years are added.

2. Comparison Using Bioassay Measurements

The information above assumes the actual inhalation intake is known, and not estimated from bioassay samples. Since this is not normally the case, it is important to evaluate the two models starting from a bioassay sample. The differences in dose per unit intake discussed above is caused by the slower lung clearance rate and the recycling in the ICRP 66/67 model. This implies a slower overall clearance rate from the body, which in turn implies lower bioassay concentrations for the same intake. When starting with a bioassay sample, the model is worked in reverse to determine an intake. Therefore, ICRP 66/67 will calculate a higher intake than ICRP 30 from the same bioassay sample. This affect will act to counter the lower dose per unit intake from ICRP 66/67 model. To demonstrate this effect, Figures 4 and 5 provide plots of the annual dose per unit activity detected in a urine sample collected 30 days after an intake. Again, an additional curve is shown on the Figure 4 that shows the result of subtracting the dose caused by the infinite half-life of the lymph node

By comparing Figures 1 and 2 to figures 4 and 5, it is clear that the differences in these two models diminish if the starting point is a bioassay result. The 50 year committed dose to the lung is now approximately 25% lower for ICRP 66/67 instead of a factor of 7.2. Likewise, the

committed dose to the liver is now approximately 30% lower instead of a factor of 7.8 observed earlier.

Figure 1
Annual Lung dose per Bq intake

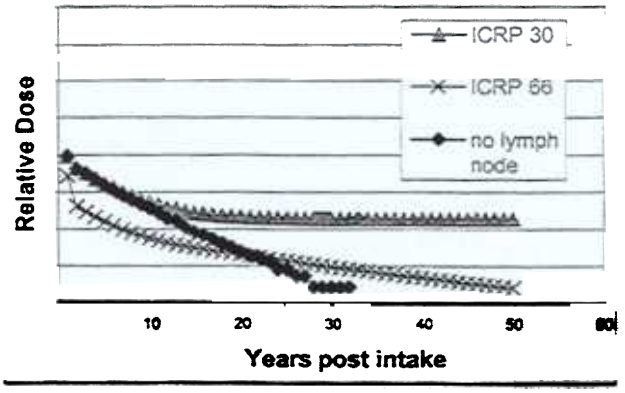


Figure 2
Annual Liver dose per Bq intake

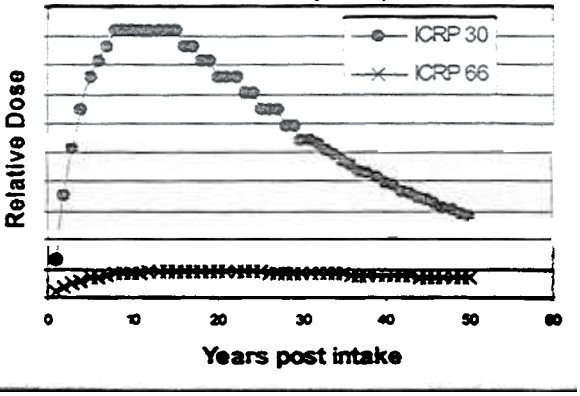


Figure 3
Annual liver dose per Bq uptake

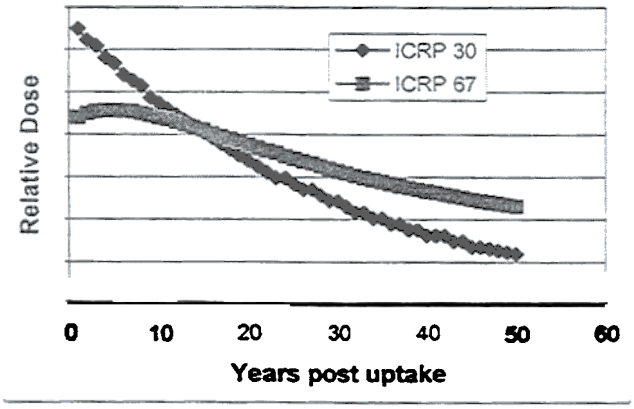


Figure 4
Annual lung dose per Bq in sample

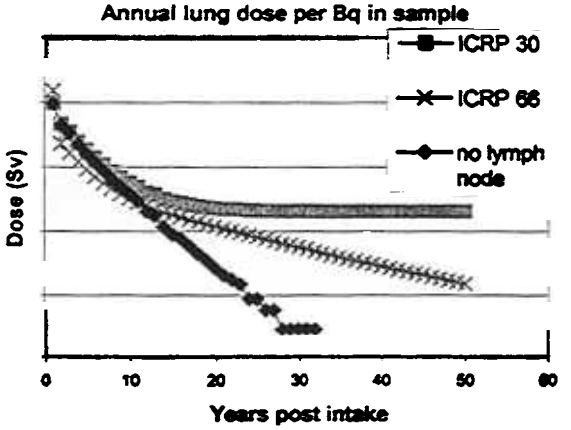
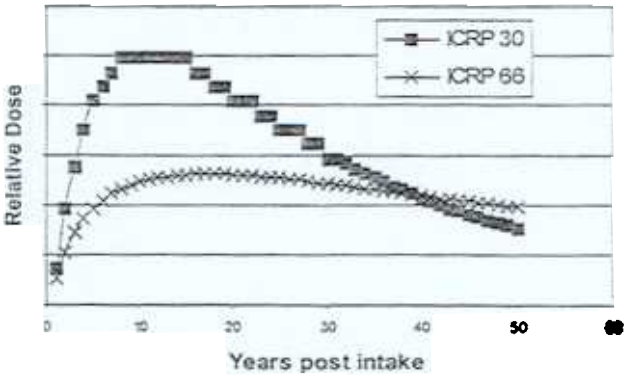


Figure 5
Annual liver dose per Bq in sample



3. Comparison of Other Solubility Classes

The infinite half-life compartment for the lymph nodes in the ICPR 30 model does not exist for other solubility classes. This tends to even out the large difference noted in lung dose observed in Figure 1. In fact for class W solubility material, the lung is cleared in only a few years under the ICRP 30 model. Likewise under the ICRP 66 model, the lung is cleared quickly, however, it continues to accumulate dose due to the incorporation of additional material from recycling. The overall affect is that the 50 year committed lung dose associated with the ICRP 66 model is 25% higher than the ICRP 30 model. The liver dose on the other hand has a lower 50 year committed dose (approximately 44% lower than ICRP 30). This is due primarily to the faster liver clearance function. The recycling of material limits this difference, but not as much as the slow lung clearance for class Y material.

Again the difference associated with using bioassay as a starting point causes a higher calculated dose for the newer models. The 50 year committed dose to the lung is 150% higher under ICRP 66 and the committed liver dose is 13% higher than ICRP 30 methods.

4. Conclusions

Because the newer ICRP models are more realistic in their approach to calculating internal dose, there are differences in the annual dose to organs given the same intake of radioactive material. These differences are most evident when intakes to insoluble compounds are involved. Where intakes are estimated from bioassay data, however, these differences are somewhat diminished. The newer ICRP models tend to smooth out the dose and deliver it over a longer period of time. This is primarily due to the addition of a recycling component to the newer model which more accurately depicts the metabolic behavior of material deposited in the body.

References

[1] The Energy Employees Occupational Illness Compensation Program Acy, Public Law 106-398, 114 Stat. 1654, 1654A-1231 (October 2000), enacted as Title XXXVI of the Floyd D. Spence National Defense Authorization Act for Fiscal Year 2001.

[2] International Commission on Radiological Protection. Limits for Intakes of Radionuclides by Workers, Part 1, Oxford: Pergamon Press, ICRP Publication No. 30, (1979).

[3] International Commission on Radiological Protection. Human Respiratory Tract Model for Radiological Protection. Oxford: Pergamon Press; ICRP Publication No. 66; (1994).

[4] International Commission on Radiological Protection. Age-dependent Doses to Members of the Public from Intake of Radionuclides, Part 2, Oxford: Pergamon Press; ICRP Publication No. 67; (1994).