

Modeling Plutonium Decorporation in Female Nuclear Worker Treated with Ca-DTPA

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The present work models plutonium (Pu) biokinetics in a female former nuclear worker. Her bioassay measurements are available at the U.S. Transuranium and Uranium Registries. The worker was internally exposed to a plutonium-ameridium mixture via acute inhalation at a nuclear weapons facility. She was medically treated with injections of 1 g Ca-DTPA on days 0, 5, and 14 after the intake. Between days 0 and 20, 13 fecal and 24 urine samples were collected and analyzed for ²³⁹Pu and americium (²⁴¹Am). Subsequently, she was followed-up for bioassay monitoring over 14 years, with 13 additional post-treatment urine samples collected and analyzed for Pu. The uniqueness of this dataset is due to the availability of: (i) both early and long-term bioassay data from a female with Pu intake; (ii) data on chelation therapy for a female; and (iii) fecal measurement results. Chelation therapy with DTPA is known to aid in reducing the internal radiation dose by enhancing the excretion of Pu from the body. Such enhancement affects Pu biokinetics in the human body, posing a challenge to the internal dose assessment. The current dose assessment practice is to exclude the data affected by DTPA from the analysis. Using this standard approach, i.e., only using data obtained 100 days after the last DTPA administration, the worksite's Radiation Protection personnel estimated the ²³⁹Pu intake to be 73 Bq, with a committed effective dose equivalent to the whole-body of 16 mSv and a committed organ dose equivalent to the bone surfaces of 340 mSv. The present analysis is the first attempt to model explicitly the combined biokinetics of Pu and DTPA by using a newly developed chelation model. The bioassay data collected during and after the DTPA administrations were used for biokinetic modeling and dose assessment. The Markov Chain Monte Carlo method was used to investigate model parameter uncertainty, given the bioassay data and assumed prior probability distributions. Preliminary results of this study show that the worker's Pu intake was 21 Bq, with a committed effective dose to the whole-body of 2.31 mSv and a committed equivalent dose to the bone surfaces of 66.7 mSv. Differences in results are expected not only because of the different dosimetric systems used, but also because this analysis includes chelation-affected bioassay data and uses a biokinetic model that accounts for the effect of chelation therapy in removing Pu from the body.

USTUR-0655-23A