Space nuclear power system accidents: Doses from Pu-238 and Am-241 inhalation

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To address the shortage of availability of Pu-238 for space missions, while new initiatives for Pu-238 production are being undertaken, there is a need for exploration of the use of Am-241 as a possible replacement for Pu-238 since the stockpile of Am-241 from the nuclear weapons program has remained relatively intact. Previously, there have been studies of the risks and consequences of Pu-238 release in postulated accidents including, for example, the Final Safety Analysis Report (FSAR) for the Galileo Mission. Since this report used an ICRP-30 based model, and a later ICRP-66 model has become available, it is of interest to re-evaluate the previous results for Pu-238 and obtain new results for Am-241. We are reporting here the following results of calculations for inhalation doses using our own computational programs (as based on different models). The results include committed equivalent doses for Pu-238 particles using the Galileo FSAR model, the original ICRP-30 model, and the ICRP-66 model. We also calculated committed equivalent dose for Am-241 using the ICRP-66 model. The ICRP-30 and ICRP-66 results were obtained using assumptions of committed time and resuspension taken from the FSAR. We have found that the ICRP-66 predicts lower doses for Pu-238 than those predicted by the Galileo FSAR or ICRP-30. Also we have found that the Am-241 lung doses are lower than those of Pu-238 because of greater clearance of Am-241 from the lungs as compared with Pu-238.

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1. Introduction

NASA’s supply of radioisotopes for Radioisotope Heat Units (RHU) and Radioisotope Thermoelectric Generator (RTG) power sources (we will refer to these together as Radioisotope Power Systems - RPSs) is facing a crisis due to shortages of Pu-238 for future missions. To address this shortage while new initiatives for Pu-238 production are effected, there is a need for exploration of the use of Am-241 as a possible replacement for Pu-238 since the Am-241 stockpile from the nuclear weapons program has remained relatively intact. It is imperative that the safety of Am-241 and its interactions with the environment be assessed to certify its use in RPS units. This assessment will require:

- Investigation of release and transport mechanisms of Am-241 in the environment and understanding receptor pathways for dose assessments as part of nuclear risk assessments, and
- Developing approaches and methodologies for nuclear risk assessment of space radioisotope power system applications.

The risk from a hazard (potential of an activity to cause harm to an entity or simply exposure) can be defined (Hines et al., 1993; McCormick, 1981; Rasmussen, 1981) as:

\[ R \left[ \text{harm/unit time} \right] = f \left[ \text{events/unit time} \right] C \left[ \text{harm/event} \right] \]  

or

\[ R \left[ \text{exposure(s)/unit time} \right] = f \left[ \text{events/unit time} \right] C \left[ \text{exposure(s)/event} \right] \]  

Mathematically, considering \( n \) events, 1, 2, ..., \( n \), we can write the total risk \( R \) from these events as:

\[ R \left[ \text{harm/unit time} \right] = \sum_{i=1}^{n} f \left[ \text{events/unit time} \right] C \left[ \text{harm/event} \right] \]  

or

\[ R \left[ \text{exposure(s)/unit time} \right] = \sum_{i=1}^{n} f \left[ \text{events/unit time} \right] C \left[ \text{exposure(s)/event} \right] \]
where \( R_i = f_i C_i, f_i \) is the frequency of the specific \( i \)-type of event, and \( C_i \) is its associated consequence. For example, relating to use of RPSs, for an individual subjected to exposure from an accident involving a spacecraft carrying a Pu-238 RPS (Frank, 1999; Goldman et al., 1991; Kastenberg and Wilson, 2004), one could find:

\[
\begin{align*}
    f &= 10^{-6} \quad \text{(RPS accident with significant release/launch)} \\
    C &= 10^{-5} \quad \text{(Excess cancer over the lifetime to an exposed individual/RPS accident)} \\
    R &= 10^{-11} \quad \text{(Excess cancer over the lifetime to an exposed individual/launch)}
\end{align*}
\]

By most standards, such a value of \( R \) would be considered insignificant considering all of the other risks from various hazards that the individual would be exposed to. Assuming this \( R \) to be a mean value for all exposed persons (say about 100,000 near the accident or even spread out worldwide), the excess cancer risk to this total population then be about \( 10^{-6} \) to 0.05 over the lifetimes of all exposed individuals and is, again, most likely insignificant. However, there are admittedly large uncertainties in estimations of both \( f \) and \( C \). In launches to date (about 27 launches carrying 46 RPSs, see NAS study (National Research Council Radioisotope Power Systems Committee, 2009)), there has been only one RPS accident involving any release of Pu-238 (Transit 5BN-3 spacecraft). Thus, \( f = 10^{-6} \) is just an estimate based on the likely event tree and fault tree analysis methods (Frank, 1999; Goldman et al., 1991). The estimation of \( C \) is likewise based on assumptions regarding release, dispersion, aerosol and dust interactions with Pu-238, inhalation and ingestion of Pu-238, related doses to critical organs, and cancer/dose relationships.

The works by Goldman et al. (1991), Frank (1999), and Kastenberg and Wilson (2004) are particularly informative. Goldman et al.’s panel, which was sponsored by several government agencies, studied potential health risks from postulated accidents involving the Pu-238 RTG on the Ulysses solar exploration mission launched on October 6, 1990. The RTG contained 24.2 pounds of polished cylinders of radioactive Pu-238 (in oxide form) in 18 packaged modules. They addressed the question of “what might have happened to the Pu-238 if an explosion worse than that of Challenger ripped apart this shuttle and caused the Ulysses spacecraft to disintegrate across the sky. Also considered was what might have happened if, after leaving the shuttle’s bay, Ulysses accidently reentered the atmosphere and smashed into something as hard as granite?” The authors discuss various accident scenarios and the subsequent release of Pu-238 dioxide, its environmental transport, inhalation and ingestion (inhalation appears to be about 300 times more likely than ingestion), and its health effects. The explanation is bit involved, but Pu-238 leads to smaller particles that dissolve more rapidly in water than those resulting from Pu-239 and, hence, requires a different health effects model than Pu-239. Some of the main understandings from that study can be summarized as:

1. Concerns for the safety of RTGs has always been a part of the U.S. space program, and design of the RTGs has evolved from a health protection philosophy of dilution and dispersion to one of containment. An earlier event involving a Navy navigational satellite (Transit 5BN-3 mission) released about 3 pounds of Pu-238 (17,000 Curies) into the atmosphere in a dilute band around the Earth after accidental atmospheric reentry and burnup. The burnup of the Transit 5BN-3 satellite prompted the development of a four-layer containment system for Pu-238: an iridium jacket around the fuel, further ensconced by two graphite jackets, ultimately placed inside a modular container to provide further protection. Two subsequent accidents, the Nimbus B-1 weather satellite and the Apollo 13 mission, appear to have confirmed the efficacy of this multi-barrier containment philosophy as no subsequent releases occurred.
2. Out of the nine hypothesized accident scenarios, the authors concluded that only two worst case scenarios could (realistically) release Pu-238.
3. In the first scenario (the shuttle exploding on or close to the launch pad with metal shards slicing into RTG container) a small fraction of the Pu-238 would consist of dust-sized small particles (up to 10 to 20 \( \mu \)m in diameter) that would be transported through the atmosphere to be either inhaled or incorporated into the food chain. In the second scenario (metal shards slicing into the RTG at an altitude of about 10,000 ft), some of the Pu-238 would be released in a plume, and by location and design, most of the Pu-238 would likely fall into deep ocean. However, the possibility of release in a heavily populated area does still exist.
4. The second scenario would involve a maximal release of about 380 Curies. Fifty Curies (about one ounce) of Pu-238 in the air and 330 Curies (about 6.6 ounces) in a four-meter puff two feet off the ground. Approximately 700,000 people might be exposed, receiving a collective dose of 3000 person-rem in the first year, and with a committed (50 year) dose of 4100 person-rem. Statistically, one could expect 0.9 excess cancers (to one single person) in a population of 700,000, or, approximately one excess lifetime cancer in a million-people exposed to the radiation, such that \( C = 10^{-6} \). We have not found estimates for the second scenario, because it is even less consequential.

Frank expanded upon this work regarding the Cassini mission and provided an in-depth analysis of the frequency of these scenarios using event tree methods and associated uncertainties. Kastenberg and Wilson applied the results of Goldman et al., Frank, and several other government panel reports to the risk equation (perspective) that we have discussed earlier, and emphasized the overall smallness of an \( R = 10^{-11} \) excess lifetime cancer risk to an individual from the launch. They also noted that the probabilities for two terms (\( f \) and \( C \)) are truly independent with each being about 1 in a million, thus the resultant risk is inherently minuscule.

Although the frequency \( f \) is very small, and the estimated consequence \( C \) is also very small, the perceived societal risk is often not small for nuclear accidents. The focus often remains more on the consequence \( C \), and it becomes necessary to improve our understandings and estimations of it. We should note that although considerable information on the modeling of \( C \) is available, it is scattered through several government reports and notes, and is not easily retrievable. Also, as noted by Kastenberg and Wilson, even contemporaneous reports have used different models and data (ICRP-30 in one case and ICRP-60 in another), and it would be useful to have all this information expressed in a single framework.

We have explored this last aspect in some detail in this paper. Of particular interest to us is the Final Safety Analysis Report (FSAR) for the Galileo Mission (General Electric Company, 1988; NUS Corporation, 1989) which describes the consequence modeling in some detail. Therein, it was assumed that Pu particles can be released in accidents through cracks in the RTG containment. The released amounts and particle size distributions were modeled using some small-scale laboratory experiments as guides. For the modeling of the atmospheric transport, the amount of material and its radioactivity available for inhalation by a subject population (directly and from resuspension of particles deposited on ground)
were estimated. A simplified version of the ICRP-30 model was used. The Galileo FSAR gives dose conversion (committed equivalent dose per unit activity) factors for three different Activity Median Aerodynamic Diameter (AMAD) values: 0.12, 3, 5.2, and 8.4 μm, with specific removal half-times and compartmental fractions for Pu-238 dioxide, and extends the dose commitment period from 50 to 70 years. A resuspension factor was included for continuous exposure to the particles (aerosols). The FSAR considered two cases in the estimation of dose conversion factors: Single instantaneous inhalation in which all material is inhaled at time \( t = 0 \), and long-term inhalation. Long-term inhalation assumes an initial resuspension factor of \( 10^{-5} \) that decreases exponentially to \( 10^{-9} \) after two years, and remains constant at \( 10^{-9} \) thereafter. There have been more recent missions but either their FSAR is not readily available or the dosimetry model data used by them has less information than the Galileo report to be useful for our purposes here.

We first attempted to replicate the Galileo FSAR results using its model, but some assumptions about the rate of inhalation of activity in the report are not very clear and this has led to some differences between our estimates and the report’s results. We have then used both the full ICRP-30 and ICRP-66 models to obtain improved estimates for \( 238\text{PuO}_2 \). Results show that use of ICRP-66 leads to lower doses than those obtained from the ICRP-30 and FSAR models. Finally, we have used ICRP-66 to estimate doses from Am-241, making assumptions like those used for Pu-238. We found that the committed equivalent dose in lungs for inhaled \( 241\text{AmO}_2 \) is lower than the committed equivalent dose for \( 238\text{puO}_2 \) aerosols, mainly due to differing absorption of these compounds from the respiratory tract to blood. The Am compound is cleared faster from the respiratory tract.

2. ICRP model families

Internal exposure to a radionuclide may be modeled using a compartmental technique. These models abstract various regions or tissues of the body into different compartments. The compartments are connected through various biochemical as well as physical transport mechanisms, facilitating mass transfer between the different compartments. Often the mass transfer is facilitated by a transport compartment, an abstraction of the circulatory system. In many models, however, there are other direct mechanisms that allow mass to be transported among compartments.

Two popular compartment models are the Medical Internal Radiation Dose (MIRD) model and the International Commission on Radiological Protection (ICRP) model. The MIRD modeling methodology is geared towards the use of medical physicists and physicians, while the ICRP methodology is well suited to the needs of health physicists. We have explored ICRP internal exposure models, as these allow for more direct computation of the risks faced by populations in general, and will allow for a more direct comparison with the FSAR of the Galileo mission.

The ICRP models for internal exposure include models for the respiratory tract, the alimentary (digestive) tract, and the metabolism (biokinetics) of the radionuclide in question. These models are loosely grouped into sets of standards. The ICRP-30 (1979) family of models is the workhorse, so to speak, of the civilian U.S. nuclear fleet and its use is mandated by USNRC regulations (10 CFR 52) for estimating exposures to the public. The ICRP-60 (1991) guidelines include revisions to the radiation weighting factor and tissue weighting factors which are used in ICRP-66. ICRP-66 introduced a new and more detailed human respiratory tract model with more compartments than its predecessor to consider the differences in uptake and clearance for different regions and a new method to calculate the time dependent uptake. The ICRP-103 family of models is the most refined family of ICRP models. It includes the updated respiratory model from publication 66 and an updated alimentary tract model detailed in publication 110. Different systemic biokinetic models have been used to describe the metabolism of different radiochemical species within the different families of models. For certain transuranic species, it has been noted in the literature that the older families of models significantly overestimate committed dose to exposed individuals.

Hence, we will consider different models for the human respiratory tract as described in the literature to facilitate both comparison with existing data, and refined data that is expected to describe the internal dosimetry of the radionuclides in question. The following pages detail the ICRP models we are using, and hopefully provide a clear picture of the actual meaning of the model.

3. Equations and model parameters

3.1. The ICRP-30 model

The ICRP-30 model is shown in Fig. 1. It has ten compartments which correspond to four different anatomical regions: The nasal passage \((NP)\) with compartments \(A\) and \(B\); the trachea and bronchial tree \((TB)\) represented by compartments \(C\) and \(D\); the pulmonary parenchyma \((P)\) represented by compartments \(E, F, G,\) and \(H\); and the pulmonary lymphatic system \((L)\) represented by compartments \(I\) and \(J\). When particles are inhaled they deposit in the NP, TB, and \(P\) regions according to the fractions \(D_{NP}, D_{TB},\) and \(D_P\) which depend on the AMAD of the particle distribution (see Fig. 2.) Each compartment has associated removal half-times and compartmental fractions: \(D\) for less than 10 days, \(W\) from 10 to 100 days, and \(Y\) for more than 100 days. Table 1 shows the removal half-times and compartmental fractions: \(D\) for less than 10 days, \(W\) from 10 to 100 days, and \(Y\) for more than 100 days. Table 1 shows the removal half-times and compartmental fractions for the \(Y\) material that is the classification for \(\text{PuO}_2\) compounds.

The clearance of inhaled material from the lungs in the ICRP-30 model is described by the following set of interlinked first order differential equations (ICRP, 1979):

\[
\frac{d}{dt}q_A(t) = I(t)D_{NP}F_A - (\lambda_A + \lambda_R)q_A(t) \quad (4)
\]

\[
\frac{d}{dt}q_B(t) = I(t)D_{NP}F_B - (\lambda_B + \lambda_R)q_B(t) \quad (5)
\]

\[
\frac{d}{dt}q_C(t) = I(t)D_{TB}F_C - (\lambda_C + \lambda_R)q_C(t) \quad (6)
\]

\[
\frac{d}{dt}q_D(t) = I(t)D_{TB}F_D + \lambda_Fq_F(t) + \lambda_Gq_G(t) - (\lambda_D + \lambda_R)q_D(t) \quad (7)
\]

\[
\frac{d}{dt}q_E(t) = I(t)D_PF_E - (\lambda_E + \lambda_R)q_E(t) \quad (8)
\]

\[
\frac{d}{dt}q_F(t) = I(t)D_PF_F - (\lambda_E + \lambda_R)q_F(t) \quad (9)
\]

\[
\frac{d}{dt}q_G(t) = I(t)D_PF_G - (\lambda_G + \lambda_R)q_G(t) \quad (10)
\]

\[
\frac{d}{dt}q_H(t) = I(t)D_PF_H - (\lambda_H + \lambda_R)q_H(t) \quad (11)
\]
where \( q_A(t), q_B(t), \text{etc.} \) are the number of disintegrations per Bq in
compartments A, B, etc. at time t, \( \dot{I}(t) \) is the rate of inhalation of the radionuclide in disintegrations/(Bq day), \( \lambda_A \) to \( \lambda_I \) are the biological clearance rates of compartments A to I which are obtained from the values of \( T \) in Table 1 by use of the equation \( \lambda = \ln(2)/T \). \( \lambda_R \) is the radioactive decay constant of the radionuclide, \( F_A \) to \( F_I \) are the fractions of material entering the various compartments which are obtained from the values of \( F \) shown in Table 1; and \( D_{NP}, DTB, \) and \( D_P \) are the regional depositions dependent on the AMAD shown in Fig. 2.

For a single instantaneous intake, \( \dot{I}(t) \) is set equal to zero for \( q_A(t) \) to \( q_I(t) \). For both single instantaneous and continuous intake, the initial condition for equations (4)–(11) is set equal to the number of disintegrations per Bq instantaneously deposited in each compartment (I) times the regional depositions (\( D_{NP}, DTB, \) and \( D_P \)) times the compartmental fractions (\( F_A, F_P, \ldots, F_I \)) using the same order as in the first term of the right-hand side of equations (4)–(11). For equations (12) and (13), the initial condition is zero. For long-term intakes, a resuspension factor can be included in \( \dot{I}(t) \).

3.2. The Galileo FSAR model

This lung dosimetry model is basically a simplification of the ICRP-30 model using the same regional depositions, but it has only five compartments and these use different removal half-times and compartmental fractions as shown in Fig. 3.

The set of differential equations which we used to describe the Galileo FSAR lung model are:

\[
q_{NP}(t) = \dot{I}(t)R_f(t)D_{NP} - (\lambda_{NP} + \lambda_R)q_{NP}(t) \tag{14}
\]

\[
q_{TB}(t) = \dot{I}(t)R_f(t)D_{TB} + 0.1\lambda_P q_{P1}(t) + 0.25\lambda_P q_{P2}(t) - (\lambda_{TB} + \lambda_R)q_{TB}(t) \tag{15}
\]

\[
q_{P1}(t) = 0.5\dot{I}(t)R_f(t)D_{P1} - (\lambda_{P1} + \lambda_R)q_{P1}(t) \tag{16}
\]

\[
q_{P2}(t) = 0.5\dot{I}(t)R_f(t)D_{P2} - (\lambda_{P2} + \lambda_R)q_{P2}(t) \tag{17}
\]

\[
q_{LN}(t) = 0.15\lambda_P q_{P2}(t) - (\lambda_{LN} + \lambda_R)q_{LN}(t) \tag{18}
\]

where \( q_{NP}(t), q_{TB}(t), q_{P1}(t), q_{P2}(t), \) and \( q_{LN}(t) \) are functions representing the number of disintegrations per Bq in compartments NP, TB, P1, P2, and TBLN at time t, respectively. \( R_f(t) \) is a factor related to the amount of particles that are re-suspended as a function of time which is initially \( 10^{-5} \) (t = 0) and decreases exponentially to \( 10^{-9} \) after two years, remaining at the latter value thereafter, see equation (19), \( \lambda_{NP}, \lambda_{TB}, \lambda_{P1}, \lambda_{P2}, \) and \( \lambda_{LN} \) are the biological clearance rates of compartments NP, TB, P1, P2, and TBLN, respectively which are obtained from the values shown in Fig. 3 by use of the equation \( \lambda = \ln(2)/T \), and the other terms are already defined in the ICRP-30 model.

\[
R_f(t) = \left\{ \begin{array}{ll}
(1 \times 10^{-5})\exp(-1.46029 \times 10^{-7}t), & 0 \leq t < 63072000 \\
1 \times 10^{-9}, & t \geq 63072000
\end{array} \right. \tag{19}
\]

where \( t \) is in seconds.

In these models, the committed equivalent dose to an organ is the product of two factors: the total number of nuclear transformations over a period of time \( \tau \) (\( U_s(\tau) \)), and the energy absorbed (Joules) per kg in the region of interest (specific effective energy, SEE). Therefore, the committed equivalent dose for a single radionuclide is given by the equation:

\[
H_s = SEE \sum U_s(\tau) \tag{20}
\]

\( U_s(\tau) \) is determined integrating the solution of the different \( q \) functions in the compartmental models from 0 to \( \tau \) and adding the results to get the total number of nuclear transformations, as it is shown in equation (21). The lungs are comprised of the compartments C to J in the ICRP-30 model and by compartments TB, P1, P2, and TBLN in the Galileo FSAR model, the nasopharyngeal region is excluded in both. The time of integration suggested by ICRP-30 is 50 years but the Galileo FSAR extended it to 70 years to account for the age span of the population.
where $Y_i$ is the yield of radiations of type $i$ per transformation, $E_i$ (in Joules) is the energy of radiation $i$, $AF(T-S)_i$ is the fraction of energy absorbed in target organ $T$ per emission of radiation $i$ in the source organ which, in this case as the lungs are both the source and target organ, means $AF(T-S)_i = 1$, $Q_i$ is the quality factor appropriate for radiation type $i$ (we are considering only alpha particles and $Q_\alpha = 20$), and $M_I$ (in kg) is the mass of the target organ which, for the mass of the lungs in a reference man, is taken as 1 kg. The alpha particle energies and yields considered for the isotopes of interest, Pu-238 and Am-241, are shown in Table 2.

### 3.3. The ICRP-66 model

The latest model proposed by ICRP for the respiratory tract is described in ICRP publication 66 (see Fig. 4). The anatomical regions considered are: anterior nasal, naso-oropharynx/larynx, bronchi, bronchioles, and alveolar interstitium. These regions are represented by 14 compartments of which 13 have two states to represent time-dependent uptake: initial state and transformed state. From the “initial” state particles dissolve at a constant rate $s_p$, and are simultaneously converted to a “transformed” state at a rate $s_{pt}$ where they have a different dissolution rate $s_t$. The dissolution mentioned above is the process to dissociate particles into material that can be absorbed into the blood (ICRP, 1994). The compartment names are numbered from 1 to 14 and their respective names in order are: $A1, A2, A3, bb_1, bb_2, bb_{seq}, BB_1, BB_2, BB_{seq}, LN_{th}, ET_2, ET_{seq}, LN_{gt}$, and $ET_1$. All of them have a corresponding transformed state compartment apart from $ET_1$. The fractional deposition of particles (for a normal nose breather) to the different regions of the respiratory tract is obtained from Annex F of ICRP publication 66 (1994). It is given for a range of AMAD from 0.5 μm to 20 μm. There are three types of default absorption behaviors labeled F (fast), M (moderate), and S (slow) which are related to the classification of materials as D, W, and Y in ICRP-30. Table 3 shows the values for $s_p$, $s_{pt}$, and $s_t$ for the three types of absorption. These are used only in the absence of absorption rate values for specific radionuclide compounds. Compounds with Pu and Am which are important for this work fall into types S and M, respectively (Blanchardon et al., 2014; Davens et al., 2010). It is important to note that “whereas D, W, and Y defined overall clearance, F, M, and S refer only to absorption into blood” (ICRP, 1994).

Particle transport rates do not change for the three types of absorption behaviors, their values in units of d⁻¹ are shown in Fig. 4.

To determine the retention of inhaled materials in the ICRP-66 model, the following systems of interlinked first order differential equations are used (Ishigure and Inaba, 1996):

$$A1\frac{dq_{01}(t)}{dt} = \dot{l}(t)DE_{01} - (m_{1,4} + s_p + s_{pt} + \lambda_R)q_{01}(t)$$  \hspace{1cm} (23)

$$A1_{T}\frac{dq_{01T}(t)}{dt} = -(m_{1,4} + s_t + \lambda_R)q_{01T}(t) + s_{pt}q_{01}(t)$$  \hspace{1cm} (24)

$$A2\frac{dq_{02}(t)}{dt} = \dot{l}(t)DE_{02} - (m_{2,4} + s_p + s_{pt} + \lambda_R)q_{02}(t)$$  \hspace{1cm} (25)

$$A2_{T}\frac{dq_{02T}(t)}{dt} = -(m_{2,4} + s_t + \lambda_R)q_{02T}(t) + s_{pt}q_{02}(t)$$  \hspace{1cm} (26)

$$A3\frac{dq_{03}(t)}{dt} = \dot{l}(t)DE_{03} - (m_{3,4} + m_{3,10} + s_p + s_{pt} + \lambda_R)q_{03}(t)$$  \hspace{1cm} (27)

$$A3_{T}\frac{dq_{03T}(t)}{dt} = -(m_{3,4} + m_{3,10} + s_t + \lambda_R)q_{03T}(t) + s_{pt}q_{03}(t)$$  \hspace{1cm} (28)

---

### Table 2

<table>
<thead>
<tr>
<th>Radionuclide</th>
<th>Energy (MeV)</th>
<th>Yield (%)</th>
<th>Avg. Energy per disintegration (MeV)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pu-238</td>
<td>5.5</td>
<td>70.9</td>
<td>5.48</td>
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<tr>
<td></td>
<td>5.46</td>
<td>29</td>
<td></td>
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<td></td>
<td>5.49</td>
<td>84.7</td>
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<td>5.44</td>
<td>13</td>
<td></td>
</tr>
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<td></td>
<td>5.39</td>
<td>1.6</td>
<td></td>
</tr>
<tr>
<td>Am-241</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Fig. 4. ICRP Publication 66 Lung Model. The ICRP Publication 66 respiratory tract model is shown (ICRP, 1994). This model is more complex than the ICRP publication 30 model using 14 compartments of which 13 have two states (initial and transformed). This figure shows the particle transport rates between different compartments.

\[
BB_{seq}: \frac{dq_{09T}(t)}{dt} = -(m_{0.10} + s_T + \lambda_R)q_{09T}(t) + s_{pt}q_{09}(t)
\]  

\[
LN_{th}: \frac{dq_{10}(t)}{dt} = -(s_p + s_{pt} + \lambda_R)q_{10}(t) + m_{3.10}q_{03}(t) + m_{6.10}q_{06}(t) + m_{9.10}q_{09}(t)
\]

(40)
or more source tissues, the relationship between sources and targets is critical. Functional ET$_{1}$, which represent the energy deposited per unit of mass of sixteen chemical forms and the corresponding retention and absorption parameters. The dissolution rate of the transformed state (representing time-dependent uptake), $s_t$, is given by:

$$ L_{NNP}: \frac{dq_{10}(t)}{dt} = - (s_t + \lambda_R)q_{10}(t) + m_{3.10}q_{053}(t) + m_{6.10}q_{067}(t) + m_{9.10}q_{097}(t) + s_p q_{10}(t) $$

(42)

$ET_{2}$: $$ \frac{dq_{11}(t)}{dt} = \bar{l}(t)E_{11} \left( \frac{m_{11.15} + s_p + s_{pt} + \lambda_R}{q_{11}(t)} \right) + m_{7.11}q_{07}(t) + m_{8.11}q_{08}(t) $$

(43)

$ET_{2}$: $$ \frac{dq_{117}(t)}{dt} = - (m_{11.15} + s_t + \lambda_R)q_{117}(t) + m_{7.11}q_{077}(t) + m_{8.11}q_{087}(t) + s_p q_{11}(t) $$

(44)

$ET_{seq}$: $$ \frac{dq_{12}(t)}{dt} = \bar{l}(t)E_{12} \left( \frac{m_{12.13} + s_p + s_{pt} + \lambda_R}{q_{12}(t)} \right) $$

(45)

$ET_{seq}$: $$ \frac{dq_{127}(t)}{dt} = - (m_{12.13} + s_t + \lambda_R)q_{127}(t) + s_p q_{12}(t) $$

(46)

$LN_{et}$: $$ \frac{dq_{13}(t)}{dt} = - (s_t + \lambda_R)q_{13}(t) + m_{12.13}q_{13}(t) $$

(47)

$LN_{et}$: $$ \frac{dq_{137}(t)}{dt} = - (s_t + \lambda_R)q_{137}(t) + m_{12.13}q_{137}(t) + s_p q_{13}(t) $$

(48)

$E_{1}$: $$ \frac{dq_{14}(t)}{dt} = \bar{l}(t)E_{14} \left( \frac{m_{14.16} + \lambda_R}{q_{14}(t)} \right) $$

(49)

where $q_{01}(t), q_{02}(t), \ldots, q_{14}(t)$ are the number of disintegrations per Bq in the 14 initial state compartments at time $t$, $q_{017}(t), q_{027}(t), \ldots, q_{137}(t)$ are the number of disintegrations per Bq in the 13 transformed state compartments at time $t$, $DE_{01}$, $DE_{02}, \ldots, DE_{09}$, $DE_{11}$, $DE_{12}$, and $DE_{14}$ are the fractional deposition of particles to the different compartments in the ICRP-66 model. These 12 fractions are obtained multiplying the values found in Annex F of ICRP-66 by some factors as it is shown in Table 4. The coefficients $m_j$ are the particle transport rates from compartment $i$ to compartment $j$; their values are shown in Fig. 4. The absorption parameters $s_p$, $s_{pt}$, and $s_t$ are as shown in Table 3. The other terms in equations (23)–(49) are explained in the ICRP-30 model.

In the ICRP-66 model, the committed equivalent dose ($H_E$) is obtained using equation (50). Note that unlike equation (20) where there was only one value of $SEE$, now there is a set of values for $SEE$ which represent the energy deposited per unit of mass of sixteen source tissues into eight target regions. Each target region has one or more source tissues, the relationship between sources and targets is shown in Table 5.

### Table 3
Absorption parameters for type F, M, and S materials. The dissolution rate of the transformed state (representing time-dependent uptake), $s_t$, has no value for the F type because nothing is transferred to the transformed state compartments so there is no need to have a dissolution rate.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>F (fast)</th>
<th>M (moderate)</th>
<th>S (slow)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$s_p$ (day$^{-1}$)</td>
<td>100</td>
<td>10</td>
<td>0.1</td>
</tr>
<tr>
<td>$s_{pt}$ (day$^{-1}$)</td>
<td>0</td>
<td>90</td>
<td>100</td>
</tr>
<tr>
<td>$s_t$ (day$^{-1}$)</td>
<td>0.005</td>
<td>0.0001</td>
<td></td>
</tr>
</tbody>
</table>

### Table 4
Fractional deposition of particles. The values for regions $Al$, $bb_{fast}$, $bb_{slow}$, $BB_{fast}$, $BB_{slow}$, $ET_{2}$, and $ET_{1}$ can be found in the tables of Annex F of ICRP publication 66. The coefficients in the third column were obtained from the example calculation of respiratory tract doses in section 9.4 of ICRP-66.

<table>
<thead>
<tr>
<th>Regions</th>
<th>Deposition Compartment</th>
<th>Deposition Fraction</th>
</tr>
</thead>
<tbody>
<tr>
<td>$Al$</td>
<td>$ET_{1}$</td>
<td>$DE_{01}$ = 0.34</td>
</tr>
<tr>
<td></td>
<td>$ET_{2}$</td>
<td>$DE_{02}$ = 0.64</td>
</tr>
<tr>
<td></td>
<td>$ET_{3}$</td>
<td>$DE_{03}$ = 0.18</td>
</tr>
<tr>
<td>$bb_{fast}$</td>
<td>$bb_{fast}$</td>
<td>$DE_{04}$ = 0.593</td>
</tr>
<tr>
<td></td>
<td>$bb_{slow}$</td>
<td>$DE_{05}$ = 0.4</td>
</tr>
<tr>
<td>$BB_{fast}$</td>
<td>$BB_{fast}$</td>
<td>$DE_{06}$ = 0.007</td>
</tr>
<tr>
<td></td>
<td>$BB_{slow}$</td>
<td>$DE_{07}$ = 0.663</td>
</tr>
<tr>
<td>$ET_{2}$</td>
<td>$ET_{seq}$</td>
<td>$DE_{08}$ = 0.33</td>
</tr>
<tr>
<td></td>
<td>BBseq</td>
<td>$DE_{09}$ = 0.007</td>
</tr>
<tr>
<td>$ET_{1}$</td>
<td>ETseq</td>
<td>$DE_{10}$ = 0.9995</td>
</tr>
<tr>
<td></td>
<td>ET$_{1}$</td>
<td>$DE_{11}$ = 0.0005</td>
</tr>
</tbody>
</table>

### Table 5
Relation of target regions and source tissue to calculate $SEE$. Several source regions may contribute to each target and each combination has an associated absorption fraction.

<table>
<thead>
<tr>
<th>Source Target</th>
<th>ET$_{1}$</th>
<th>ET$_{2}$</th>
<th>ET$_{seq}$</th>
<th>LN$_{et}$</th>
<th>BB$_1$</th>
<th>BB$_2$</th>
<th>BBseq</th>
<th>AI</th>
<th>Al</th>
<th>LN$_{inh}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Source Target</td>
<td>BB$_{fast}$</td>
<td>BB$_2$</td>
<td>BB$_{seq}$</td>
<td>BB$_{fast}$</td>
<td>BB$_{slow}$</td>
<td>BB$_{slow}$</td>
<td>ET$_{1}$</td>
<td>ET$_{2}$</td>
<td>ET$_{seq}$</td>
<td>LN$_{et}$</td>
</tr>
</tbody>
</table>

### Equation (50)

$$ H_E = \sum \lambda_R \cdot U_i(\tau) $$

Each combination of source tissue and target region has a fraction of absorbed energy associated, $AF(T-S_i)$, which is dependent on the energy of radiation $i$. Each $SEE$ is calculated using equation (22), taking the values for $AF(T-S_i)$ from Table H.1 and the values for the masses of each target from Table 5 (both tables are in the ICRP-66 publication, 1994). Values not found in Table H.1 like $AF(LN_{et} \leftarrow LN_{et})$, $AF(AI \leftarrow Al)$, and $AF(LN_{inh} \leftarrow LN_{inh})$ are equal to 1. Then, the eighteen values of $SEE$, must be multiplied by the total number of transformations in the committed time that take place in the source tissue, $U_i(\tau)$. The function $U_i(\tau)$ is already defined in equation (21). The function $q_i(t)$ will be the sum of the functions corresponding to initial and transformed state compartments with two exceptions: $ET_{1}$, which has no transformed state compartment, and $AI$ where functions of initial and transformed states for $AI_{1}$, $AI_{2}$, and $AI_{3}$ will be added together into one $q_{AI}(t)$. As mentioned above, the commitment period is usually 50 years but we will extend it to 70 years to compare it with the other models. Finally, to obtain the committed equivalent dose in the lungs, we exclude the extra-thoracic regions $ET_{3}$, $ET_{2}$, and $LN_{et}$.
the committed equivalent dose. The works of Davesne et al. (2010) and Blanchardon et al. (2014) provide specific absorption parameters for PuO₂ and AmO₂ compounds, these are based on reviews of several absorption experiments. Table 6 shows these values together with the default absorption parameters proposed in ICRP-66.

4. Results

4.1. Committed equivalent dose from Pu-238 particles

The Galileo FSAR gives dose conversion (committed equivalent dose per unit activity) factors for three AMADs: 0.12, 3, 5.2, and 8.4 μm. As mentioned before, the model used to obtain them is basically the one found in ICRP publication 30 but with the following modifications: specific removal half-times and compartmental fractions for PuO₂, the extension of dose commitment period from 50 to 70 years, and a resuspension factor included for continuous exposure to the aerosol. To account for different types of exposures FSAR considers two cases in the estimation of dose conversion factors: Single instantaneous inhalation in which all material is inhaled at time \( t = 0 \), and long-term inhalation. Long-term inhalation assumes an initial resuspension factor of \( 10^{-3} \) that decreases exponentially to \( 10^{-9} \) after two years and remains at \( 10^{-8} \) thereafter. We reproduced the FSAR model in Mathematica® with the intention of comparing the dose eventually with the newer respiratory tract model proposed in ICRP publication 66 and the results are shown in Table 7. Some assumptions in the FSAR about the rate of inhalation of activity are not very clear and this may lead to the differences in the doses between FSAR and our model. The main outliers are the values for 0.12 and 8.4 μm AMAD in both cases (short and long term). The difference in the result for 0.12 μm can be attributed to the fact that the values for fractional deposition in the different regions of the respiratory tract (Fig. 2) are intended for the different regions of the respiratory tract (Fig. 2) are intended for the ICRP-30 model considering the same assumption in the report of 70 years of commitment time and the resuspension factor of equation (19).

To get more insight about this problem we also programmed the ICRP-66 model in Mathematica® and this program was benchmarked to the example shown in Table 24 of ICRP publication 66.

Table 6

<table>
<thead>
<tr>
<th>Parameter (day(^{-1}))</th>
<th>PuO₂</th>
<th>AmO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Default type S</td>
<td>Davesne et al.</td>
<td>Default type M</td>
</tr>
<tr>
<td>( s_p )</td>
<td>0.1</td>
<td>0.0019</td>
</tr>
<tr>
<td>( s_p )</td>
<td>100</td>
<td>1.6</td>
</tr>
<tr>
<td>( a_l )</td>
<td>0.0001</td>
<td>0.0001</td>
</tr>
</tbody>
</table>


table shows how inhaled AmO₂ particles produce a smaller committed equivalent dose than PuO₂ particles.

4.2. Committed equivalent dose from Pu-238 and Am-241 particles

Now using the ICRP-66 model, we compare the committed equivalent dose from Pu-238 and Am-241 in Fig. 7, considering them as type S and type M materials, respectively. First we evaluated the model with the same absorption parameters (S and M) for both radionuclides and the difference was negligible. Based on this observation we conclude that the half-lives of these radionuclides and their average alpha energies do not influence the deposited dose considerably but the absorption parameters do. This figure shows how inhaled AmO₂ particles produce a smaller committed equivalent dose than PuO₂ particles.

To provide a better estimation of the committed equivalent dose we calculated it again using specific absorption parameters for PuO₂ and AmO₂ taken from Table 6, the results are shown in Fig. 8.
For Pu, the committed equivalent doses using default and specific absorption parameters are practically the same while for Am using the specific absorption parameters results in a reduced committed dose. As the Am compound is cleared faster from the respiratory tract, it deposits a lower committed dose.

5. Discussion and conclusions

The overestimation of lung dose by ICRP-30 model over the ICRP-66 model is mostly due to both the different regional deposition of particles and the absorbed fractions in these models. A recent revision to the ICRP Publication 66 human respiratory track model (Smith et al., 2013) suggests some changes for extra-thoracic compartments and new default absorption parameters. The former has no significant effect on our calculation because we are considering only the thoracic dose. We have addressed the latter by using specific absorption parameters for each material.

In the ICRP-66 model, the three main parameters influencing dose calculation for different radionuclides are: radiological half-life, energies of alpha particles, and the retention and absorption parameters for the chemical form of the aerosol. As both half-lives, for Pu-238 and Am-241, are large compared to the biological clearance rates their effect is not significant. The alpha energies do not represent a significant difference in dose because their average energy per disintegration is very similar between these two radionuclides, being 5.48 and 5.44 MeV, respectively. The factors that have the greatest influence are the retention and absorption parameters for each material. As Am-241 particles are cleared at a faster rate from the respiratory tract, the deposited dose by unit activity inhaled of Pu-238 particles in lungs is on the average 2.6
times the dose caused by Am-241 particles (using specific absorption parameters).

Although Am-241 seems less risky for humans than Pu-238 per unit of activity inhaled (a factor of about 1/2.6 ≈ 0.4) for the same activity, the mass of Am-241 required is about 5 times that of Pu-238. An RTG using Am-241 will experience a slower power decline than one using Pu-238 due to the longer half-life of Am-241. However, the power density and efficiency will be smaller for a system powered by Am-241. Assuming the same density, a device powered by Am-241 would have 5 times the volume than a device powered by Pu-238. Thus, dimensionally, it would be 1.7 times larger in every direction and have almost 3 times the surface area. The same power distributed over 3 times the surface area would seem to generally correspond to a lower surface temperature which will reduce the voltage generated by the RTG (this is very design dependent as it is a problem of thermal transport). Overall, thus while the specific dose from Am-241 is about 1/2.6 of that due to Pu-238, the lung dose from Am-241 may be about a factor 2 higher over a same power Pu-238 RTG.

We should note also that after Am and Pu particles are cleared from the respiratory tract they are transported to blood and the gastro-intestinal tract, and will impact other parts of the human body. The present work should be extended to estimation of the risks involved by using a whole-body dosimetry model. Additionally, it would be also important to elucidate the comparative particle releases (Pu-238 and Am-241) from postulated accidents of
specific designs, transport and transformations of the radionuclides in the atmosphere, and availability for inhalation.

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References