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SCIENTIFIC NOTE

ARE RADON GAS MEASUREMENTS ADEQUATE FOR EPIDEMIOLOGICAL STUDIES AND CASE CONTROL STUDIES OF RADON-INDUCED LUNG CANCER?

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The lung dose derived from radon is not attributed to the radon gas itself, but instead to its short-lived progeny. However, in many epidemiological studies as well as in case control studies of the radon risk, the excess number of cancers are related to the radon gas exposure, and not to the radon progeny exposure. A justification for such an approach has resorted to the assumption that there is self-compensation between the radiation doses from the unattached and attached fractions. In the present study, we used the Jacobi model to calculate the radon progeny concentrations in a room by varying the attachment rate and then calculated the resulting lung dose. It was found that self-compensation was not fully realised, and the effective dose can vary by a factor up to ~2 for the same radon gas concentration. In conclusion, the radon gas concentration alone does not provide adequate information on the effective dose.

INTRODUCTION

It is now well established that the short-lived radon progeny contributes about half of the total exposure of human beings to ionising radiation. It is also well known that the radon dose delivered to the lungs is not attributed to the radon gas itself, but instead to its short-lived progeny. Exposure to radon progeny (usually expressed in the traditional unit called Working Level Month or WLM) is measured through the product of PAEC × t, where PAEC is the potential alpha energy concentration (usually expressed in the traditional unit called Working Level or WL) and t is the exposure time. To relate the radon progeny exposure and the effective dose, the so-called dose conversion coefficient (DCC) is used. The DCC value derived dosimetrically, i.e. based on lung modelling, is ~15 mSv per WLM.

However, in many epidemiological studies as well as in case control studies of the radon risk, the excess number of cancers are related to the radon gas exposure (given in Bq m^{-3}), and not to the radon progeny exposure. A justification for such an approach has resorted to the assumption that there is self-compensation between the radiation doses from the unattached and attached fractions, i.e. with an increase in the aerosol concentration, the attached fraction of PAEC and the equilibrium factor increase while the unattached fraction of PAEC decreases. With this argument, it is asserted that the total radiation dose should be relatively constant and independent of the unattached fraction and the equilibrium factor, so that the risk can be effectively surrogated by the radon gas concentration alone. However, such an approach has not been rigorously examined. The purpose of this paper is to study such an approach in detail, and to make a conclusion in the end.

METHODOLOGY

The following three steps of calculations were performed. First, the Jacobi model was used to calculate the radon progeny concentrations in a room. The Jacobi model was extended so that the three modes of attached radon progeny could be studied(1). The obtained results give the ratios between the concentrations of radon progeny and radon gas in different modes (including the unattached fraction as well). All the parameters of the Jacobi room model were fixed except the attachment rate λ_a. The fixed parameters were set as follows: the ventilation rate was $\lambda_v = 0.55\ h^{-1}$, the deposition rate of the unattached and attached progeny were $\lambda_d^u = 20\ h^{-1}$ and $\lambda_d^a = 0.2\ h^{-1}$, respectively, and the recoil factor was $p_1 = 0.83$. The attachment rate λ_a was varied between 10 and 110 h^{-1} with steps of 10. This range was considered typical for the parameter. Varying the attachment rate is a good method to obtain different unattached fractions and equilibrium factors. In reality, different attachment rates are expected in various environments with different aerosol concentrations. The PAEC (in WL) was also calculated for a radon gas concentration

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of 3700 Bq m^{-3}. The results of these calculations are described in the following section.

In the second step, the obtained results for progeny concentrations were used as inputs in the home written computer program, LUNGDOSE.F90$^{(2)}$, which calculated DCC. This program followed the human respiratory tract model published by the International Commission on Radiological Protection$^{(3)}$. Additional aerosol parameters and subject-related parameters are needed for the DCC calculations.

Here, the best estimations of parameters given by Marsh and Birchall$^{(4)}$ were adopted in the calculations. The subject-related parameters used in the calculations were as follows: breathing rate, 0.78 m^3 h^{-1}; tidal volume, 0.866 litre per breath; and functional residual capacity, 3300 ml. The aerosol parameters were as follows: density of unattached particles, 1 g cm^{-3}; density of attached particles, 1.4 g cm^{-3}; shape factors are 1 and 1.1 for unattached and attached particles, respectively; median diameters (with geometrical standard deviations given in parentheses) are 0.9 (1.3) nm, 50 (2) nm, 250 (2) nm and 1500 (1.5) nm for unattached, nucleation, accumulation and coarse modes, respectively; and partitioning between different attached modes are 0.28, 0.70 and 0.02 for nucleation, accumulation and coarse modes, respectively.

By using the program LUNGDOSE.F90, the DCC (mSv per WLM) was calculated as a function of the attachment rate. The results are also given in the following section.

In the third step, the radon gas concentration was considered as 3700 Bq m^{-3} and the dose values were determined for each of the considered cases.

RESULTS

In Figure 1, the ratios of the concentrations of the unattached progeny, F_i^u, to that of ^{222}Rn are shown. With an increase in the attachment rate, all F_i^u values decrease. The values for the unattached ^{218}Po are two orders of magnitude larger than those for the unattached ^{214}Bi. Figure 2 presents the values for the attached progeny in three different modes, namely nucleation, accumulation and coarse modes. The values for all modes increase with the attachment rate.

Figure 3 shows the variation of the unattached fraction, i.e. the ratio between the unattached fraction of PAEC (PAEC^u) to the total PAEC, and the equilibrium factor as a function of the attachment rate. As expected, the unattached fraction decreases and the equilibrium factor increases with increasing attachment rate. Here, some self-compensation for the dose value is realised, i.e. the dose from the

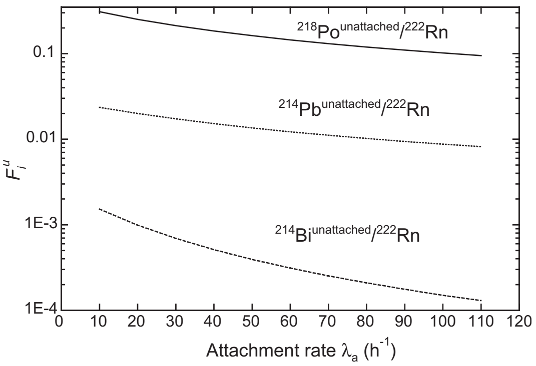

Figure 1. Ratios between concentrations of different unattached progeny and radon gas concentration as a function of the attachment rate. Solid line, ^{218}Po; dotted line, ^{214}Pb; and dashed line, ^{214}Bi.

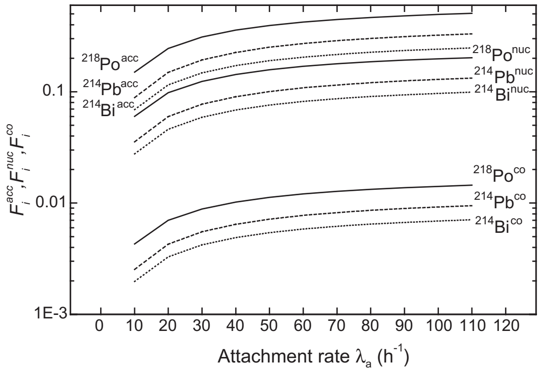

Figure 2. Ratios of progeny concentrations to radon concentration in three different attached modes as functions of the attachment rate (nuc, nucleation mode; acc, accumulation mode; and co, coarse mode). The values for all modes increase with the attachment rate.

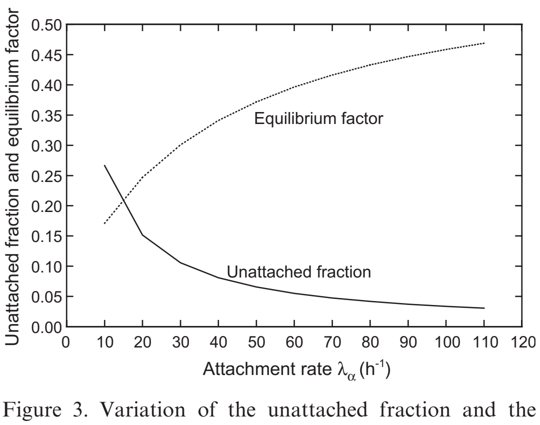

Figure 3. Variation of the unattached fraction and the equilibrium factor with the attachment rate.

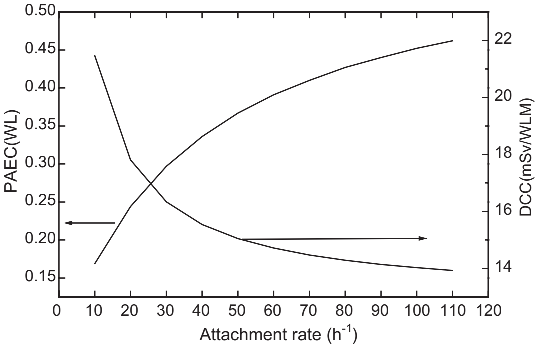

Figure 4. The variation of DCC (right vertical axis) and PAEC (left vertical axis) for 3700 Bq m^{-3} of ^{222}Rn as a function of the attachment rate.

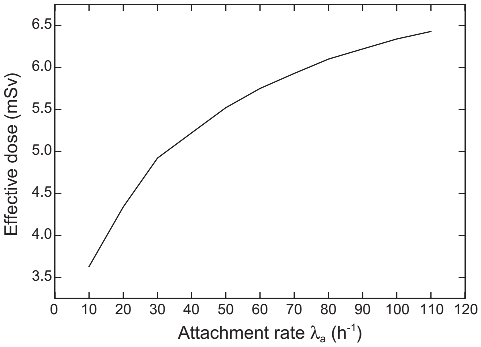

Figure 5. Effective dose as a function of the attachment rate (for a radon concentration of 3700 Bq m^{-3}).

unattached progeny decreases whereas the dose from the attached progeny increases.

Figure 4 shows the DCCs calculated for different attachment rates given by the right vertical axis. The DCC decreases with the attachment rate in the range from 22 to 14 mSv per WLM. Also shown in Figure 4, by the left vertical axis, is the total PAEC for a radon concentration of 3700 Bq m^{-3}. This value was chosen because, for this radon concentration, PAEC = 1 WL if equilibrium is achieved between radon and its progeny. When equilibrium is not achieved, PAEC is below 1 WL. Finally, the effective doses for 1 WLM were recalculated as

$$E = \mathrm{PAEC(WL)} \times \mathrm{DCC}\left(\mathrm{mSv\ WLM}^{-1}\right) \times t \quad (1)$$

and the results are shown in Figure 5. The effective dose increases from 3.6 up to 6.4 mSv.

CONCLUSIONS

Figure 5 shows that the effective dose varies for different environmental conditions and the targeted self-compensation is not fully realised. For the same radon gas concentration, the effective dose can vary by a factor up to ~2. Therefore, it is concluded that knowing the radon gas concentration alone does not provide adequate information on the effective dose or the attributed risk.

Many previous epidemiological and case control studies on the radon risk were based on the measurements of the radon gas concentrations, and the risk coefficients were expressed for a unit activity concentration (Bq m^{-3}) of the radon gas. Such an approach is shown here to be inadequate, and the dose determined based on radon gas measurements alone can be wrong by a factor of ~2. The only way to obtain accurate dose values has to rely on actual radon progeny measurements. Nikezic *et al.*(5) has proposed a convenient method for long-term measurements of radon progeny concentrations using the LR 115 solid-state nuclear track detector.

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