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THE INFLUENCE OF ENVIRONMENTAL TOBACCO SMOKE ON RADON DOSIMETERY

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Environmental tobacco smoke (ETS) can affect the radiation dosimetry of indoor radon by modifying the behaviour of its decay products. Firstly, ETS significantly enhances the concentration of airborne particles, thereby reducing the unattached fraction. However, the lower mobility of ETS particles leads to an increase in airborne radioactivity by a factor of 2.5. Calculations presented here suggest that this is likely to give a protective effect, although an increased dose can result, depending upon how low the unattached fraction is in the absence of ETS. Clearly, this merits further study. Secondly, natural radioactivity in tobacco means that ETS enhances the airborne concentrations of the long-lived decay products 210Pb and 210Po by a factor of up to 2.4 for the latter nuclide. However, this does not significantly increase the overall dose due to radon. Finally, it has been suggested that the act of smoking can enhance the deposition of the attached fraction in the bronchial region; this hypothesis has been discounted.

INTRODUCTION

Radon is the single most important source of irradiation of the general population (1). Most of this dose derives from the deposition in the respiratory tract of the short-lived decay products of radon (218Po 214Pb, 214Bi and 214Po), two of which are alpha-emitters (2). When radon decays, the products are present as free ions, typically 3 nm in size (3). These have a high mobility, due to Brownian diffusion, with the result that they deposit to a surface, be it the fabric of a room, an ambient airborne particle or the surface of the respiratory tract. The airborne fraction present on ambient particles, which have an average size of about 130 nm (3), is termed 'attached', the remainder being 'unattached'. The unattached fraction deposits more efficiently in the respiratory tract than the attached; the site of deposition is also shifted from the pulmonary (attached) to bronchial (unattached) regions of the lung (4). The International Commission on Radiological Protection (ICRP) have recently calculated that the dose to the respiratory tract is dominated by that to the bronchial region, with that due to the unattached fraction being at least an order of magnitude greater than that due to the attached (4)
Environmental tobacco smoke (ETS) can affect dosimetric calculations of indoor radon decay products in a number of ways. Firstly, cigarette smoking significantly enhances the concentration of

airborne particles. This will have the effect of reducing the unattached fraction, thereby reducing the resulting radiological dose. However, the mobility of such particles is much less than for the unattached fraction, with the result that more of the decay products remain airborne for longer periods. Thus, the presence of ETS may either reduce or enhance the dose, depending on the prevailing conditions in the absence of ETS. Secondly, it is well documented that tobacco contains the two long-lived radon decay products, 210Pb and 210Po, the latter being an alpha-emitter (5). Both are relatively volatile metals, and so may significantly increase the ambient levels of these nuclides during tobacco combustion. Lastly, it is known that the deposition of tobacco tar during the act of smoking does not follow predictions based on particle size (6). Should this also apply to ETS, or if a substantial fraction of decay products be inhaled during the act of smoking, a revision in dosimetric estimates is required (7). This paper considers each effect in turn.

EFFECTS OF ETS ON SHORT--LIVED DECAY PRODUCTS

ETS significantly increases the number of airborne particles present in indoor atmospheres. For example, a cigarette contributes about 10 mg of particles of median size 200 nm in ETS (8); assuming such particles are unit density spheres, the concentration in an unventilated 14 m3 room would be 9.10^4 cm-3, well above normal ambient levels. Clearly, such an increase in particle concentration will reduce the unattached fraction. Normally, 7-15 % of the decay products are unattached, but this drops to less than 5 % in the presence of a source of airborne particles such as ETS (9). However, such attachment means that the decay products will remain airborne longer, since the mobility of the particles due to Brownian diffusion will be much less than that of the free ions. This, in turn, will lead to an increase in the levels of total airborne radioactivity; under experimental conditions, it has been demonstrated that the smoking of cigarettes leads to an increase in the concentration of decay products by a factor of 2.5 (10).

The current ICRP lung model (11) does not apply to radionuclides with short half-lives, resulting in the critical dose to the bronchial region being underestimated (4). This situation is -being rectified (4); following unit exposure, calculated doses to the bronchial region are 150 nGy for 3 nm particles (unattached), 11 nGy for 130 nm particles (ambient attached) and 7 nGy for 200 nm particles (ETS). [Unit exposure is defined as 1 Bq h m-3 EER; EER is the equilibrium equivalent concentration of radon, i.e. the activity concentration of radon in equilibrium with its decay products which has the same potential alpha-particle energy as the actual non-equilibrium mixture of decay products.] Thus, for an ambient atmosphere of 130 nm particles with an unattached particle size of 3 nm, and fraction (F), the dose per unit exposure (in nGy per Bq h m-3) is:-

$$D = 150F + 11(1-F)$$

$$= 139F + 11 \qquad (1)$$

In the presence of ETS particles of 200 nm, this equation becomes:-

$$D = .143F + 7 \qquad (2)$$

Thus, the presence of ETS reduces the overall dose for a given exposure and degree of attachment. Against this, it should be noted that the presence of cigarette smoke enhances the airborne concentration by a factor of 2.5, with the result that exposures will be 2.5 times greater (10), thereby increasing the dose for a given degree of attachment. However, the degree of attachment is a strong function of the airborne particle concentration (12).

It has been found from a study of German homes, that the particle concentration could increase 100-fold during the act of cigarette smoking, remaining elevated for up to 5 hours afterwards (9). Correlating with this, the average unattached fraction dropped from an average of 0.12 to 0.02 (9), whilst the average radon concentrations increased from 160 Bq m-3 EER to 295 Bq m-3 EER. Thus, from equation (1), one hour's exposure would lead to a dose of 4.4 µGy prior to smoking, but only 2.9 µGy after smoking (equation (2)), a drop of 34 %. Making the simplified assumptions that smoking always reduces the unattached fraction to 1/6 of its previous value (9), but that the overall concentration of radioactivity rises by a factor of 2.5 (10), then the presence of cigarette smoke will lead to a dose reduction, when the original unattached fraction is greater than a value, F, found by solving the equation:-

$$139F + 11 = (143F/6 + 7) \times 2.5 \qquad (3)$$

i.e. for values of the unattached fraction prior to smoking of greater than 8.2 %. This value is well below the average 18 % observed in UK dwellings (3), and below the average 10 % in German houses without obvious aerosol sources such as smoking or cooking (9).

In view of these calculations, it is perhaps surprising that there have not been more investigations of the effects of the indoor aerosol on the attached fraction and, hence, dose. Some limited evidence to support these calculations comes from a comparative study of the levels of 214Bi in smokers and nonsmokers, determined using whole-body monitoring (13). Non-smokers were found to have 2-4 times the levels of smokers. Unfortunately, the data were not described in terms of whether the nonsmokers lived with another non-smoker, or a smoker. However, given that smokers must live in an atmosphere where ETS is present, these data would suggest that the dose from the shortlived decay products is indeed lower.

EFFECTS OF ETS ON LONG-LIVED DECAY PRODUCTS

Tobacco, as any other plant, has a tendency to incorporate radionuclides in the environment. Two nuclides of particular dosimetric significance are 210Pb and 210Po (14). Both are relatively volatile

metals, with the result that a significant proportion of the cigarette content may become airborne during combustion, thereby raising the concentration of these nuclides. Certainly, it is well documented (e.g. 15) that cigarette smokers have higher burdens of these nuclides as a result of their intake through smoking.

On average, cigarette tobacco contains 17 mBq g-1 of 210Po; this is supported by an equal activity of the parent nuclide (5). Estimates suggest that between 24 and 46 % of the cigarette 210Po is transferred to ETS (16,17,18); 210Pb has about half the volatility of 210Po (19,20). Assuming that a cigarette contains 0.8 g of tobacco, there will be approximately 4.8 mBq Of 210Po and 2.4 mBq of 210Pb generated in ETS. Thus, if 20 cigarettes are smoked over the course of 16 hours in a 14 m3 room, being ventilated at 3 air changes per hour, then average levels generated of 210Po and 210Pb will be 0.14 and 0.07 mBq m-3 respectively.

To put these values into context, the average 210Pb concentration in air is estimated to be 0.5 mBq m-3 (21). As the ratio of 210po to 210Pb is about 0.2 (1), the concentration Of 210Po is 0.1 mBq m-3. Thus, the addition of ETS will only increase the levels of 210Pb by 14 %, but those Of 210Po by 140 %, to 0.24 mBq m-3. Since the latter nuclide is an alpha-emitter, it dominates the dose arising from inhalation of these two nuclides, contributing 86 % of the total (1). Using the current ICRP model (11), and assuming 0.2 µm ETS particles with pulmonary clearance half-times of half with 0.2 d and half with 0.01 d (Class W), the committed effective dose equivalent for 210Po is 3.8 µSv Bq-1. Further assuming a breathing pattern of 0.75 m3 hr-1, then the total intake per year is 1.6 Bq, i.e. a dose of 6 µSv per year. Under the proposed new model, the clearance rates are slightly faster (22) and the deposition a factor of 3 lower (4), so the calculated dose will be much smaller. Since the total dose due to the inhalation of radon and decay products is about 1000 µSv per year (1), it can be seen that the contribution from natural radioactivity in ETS is insignificant.

THE DEPOSITION OF TOBACCO SMOKE

The above calculations assume that the current and proposed deposition models apply to tobacco smoke. Many factors are known to influence deposition other than size and breathing pattern, including physique, disease and particle solubility (23). In particular, the deposition of tar from cigarette smoke during the act of smoking is much greater, and more central within the lung, than would have been predicted from its particle size; in fact, it deposits like a particle some 10 times larger (6). However, the limited data on the deposition of ETS would suggest that the processes of aging and dilution change the nature of the smoke, resulting in a deposition pattern like any other non-hygroscopic 200 nm particle (24).

Finally, it has been suggested that the particles of ambient air, whether ETS or not, could be drawn through a cigarette during the act of puffing. This could modify their deposition pattern as outlined above, thereby contributing to 'hot' spots of activity in the lung (7). However, this hypothesis has been discounted, since

in deposition of a factor of 10 will not affect the overall dose significantly (25).

CONCLUSIONS

The presence of environmental tobacco smoke may modify the dose due to the inhalation of radon decay products in one of three ways, namely a) modification of the concentration and degree of attachment of the short-lived products, b) acting as an additional source of the long-lived products and, c) modifying the deposition pattern. Of these factors, the last two may be dismissed as not of dosimetric significance. The first, however, does have a significant effect. Despite significantly elevating the airborne levels of radioactivity, the increased degree of attachment will reduce the dose under most normal domestic situations. It is estimated that the dose reduction could be about 35 %, based upon data generated in a German survey. Clearly, this merits further investigation.

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