Introduction
The protection quantity effective dose was devised by the International Commission on Radiological Protection (ICRP) as a measure of radiation detriment. It takes into account the different sensitivities of different organs and tissues to the induction by radiation of stochastic effects. However, it is now clear that, in the case of exposure to radon decay products, effective dose is used differently. Without being explicitly acknowledged, effective dose is used as a measure of detriment from the combined effects of two separate carcinogenic agents: radiation (from radon decay products) and tobacco smoke.

With recent epidemiological studies now able to estimate the risk of lung cancer as a function of both exposure to radon decay products and exposure to tobacco smoke, it has become evident that the major contributor to this hybrid form of effective dose is tobacco smoke. In the absence of smoking, the dose conversion convention – from radon decay product exposure to effective dose – would be several times smaller than the value recommended by ICRP.

Discussion
The pooled European study of residential radon and lung cancer (Darby et al 2006) suggests that the excess relative risk (ERR_{smk}) per unit exposure to radon is largely independent of smoking status. This implies a multiplicative relationship between risk from radon and risk from tobacco smoke:

\[ \Delta R = R_{0} \times ERR_{Rn} \times RR_{smk} \times \Delta R_{Rn} \]  

where \( \Delta R \) is the increase in cumulative risk to attained age arising as a consequence of an increment in indoor radon concentration \( \Delta R_{Rn} \), and where \( R_{0} \) is the baseline risk of lung cancer in the absence of both tobacco smoke and radon, and \( RR_{smk} \) is the relative risk from a given level of smoking.

Eq.1 has been used to calculate the risk values in Table 1, with \( R_{0} \) (0.59%), \( ERR_{Rn} \) (0.00084 per Bq m\(^{-2}\)) for measured indoor radon, and \( RR_{smk} \) taken from Darby et al 2006. For smokers of 15 or more cigarettes per day, the risk of lung cancer is about 30 times greater than for a never-smoker. For a ‘population average’ level of smoking, the risk is about 5 to 6 times greater than for a never-smoker.

To the extent that cumulative risk to age 80 behaves similarly with regard to smoking status as lifetime excess absolute risk (LEAR), it is clear that when recommending a population average value for LEAR, the ICRP includes a large factor attributable to smoking.

Further, when deriving a dose conversion convention – by aligning the radon LEAR with the nominal risk coefficient for ionizing radiation – the smoking factor is carried over into the quantity effective dose. Effective dose then becomes a measure of the combined detriment from tobacco smoke and radiation, with tobacco smoke being the dominant component. In the process, tobacco smoke risk is turned into millisievert. Most of the magnitude of the currently recommended dose conversion convention – 5 mSv/WLM – is actually due to tobacco smoke.

Unfortunately, the recent updating of ICRP recommendations for exposure to radon (ICRP 2011) does not address the problem; rather, it perpetuates it.

Incorrectly attributing tobacco risk to radiation is not just bad science. It leads to misguided decision making in optimizing protection. It unnecessarily exacerbates public fear of radiation. And it means that recorded doses in millisievert become ambiguous: how much reflects radiation detriment and how much is due to tobacco smoke?

A solution
In future, whether using a dose conversion convention derived from epidemiology or a dose coefficient from dosimetric modelling, calculated effective doses should reflect radiation detriment only. The risk from tobacco smoke should also be dealt with, but not by turning it into millisievert.

It has been suggested that using population average values, whereby including tobacco smoke detriment in the evaluation of effective dose, is necessary to provide protection for the population as a whole. But the same end can be achieved by using population average risk values for smoking and radon combined. The same values of reference levels and derived constraints would be obtained, and the same degree of protection provided.

There is no need to attribute detriment to the wrong carcinogen in order to implement the ICRP system of protection in a manner that provides protection for everyone.

References: