RADON RISK IN URANIUM MINING AND ICRP

D. Chambers*, R. Stager* and N. Harley**

*SENES Consultants 121 Granton Drive Unit 12, Richmond Hill Ontario, Canada L4B 3N 4
** New York University School of Medicine, Dept. of Environmental Medicine, 550 First Avenue, New York, NY 10016

Corresponding author: Doug Chambers; Email: dchambers@senes.ca

Abstract

In 2009, the ICRP issued a radon statement indicating that the dose conversion convention (DCC) for radon progeny, an epidemiologic based convention, would likely double and that the calculation of risk from radon should move to a fully biologically based dosimetric approach, from the long standing epidemiological approach. Both issues have important implications for uranium miners. This paper first examines the epidemiological basis for the ICRPs recommendations and provides a detailed discussion of the effect of smoking prevalence on lifetime excess absolute risk and consequently on the DCC. The paper also examines the capability of current dosimetric models to address the risk from smoking and exposure to radon and observes that results of dosimetric models do not match sufficiently well with those from epidemiology, presumably because smoking is not relevant to biokinetic dosimetric models. The paper also briefly reviews the measurements needed to implement a dosimetric based system of protection for radon. It is important to understand that mine environments vary widely not only between mines but also within mines and with time. This variability presents challenges to the development of measurement programs and dosimetric evaluations. At this time, data for mines are very limited and there needs to be further improvements in the modelling, measurement techniques, and understanding of modern workplace conditions. Overall, given the current "notional" 30% prevalence of smoking and the apparent trend in decreasing smoking prevalence (eventually leading to lower baseline rates), a nominal DCC developed by epidemiology with value of the order of 6-7 mSv/WLM corresponding to the current lung cancer mortality rates would seem to be protective of miners, representative of current smoking prevalence, likely "conservative" for future reference populations, consistent with the ICRP's use of nominal conversion coefficients, and suitable as a nominal dose conversion coefficient.

Key Words

Radon; epidemiology; dose conversion convention; dosimetry

Introduction

Until recently, epidemiological studies of miners provided the main basis for estimating the risks from exposure to radon (e.g., UNSCEAR 2006 Annex E). Today, case-control studies of residential exposure to radon also show a risk of lung cancer increasing with increasing exposure to radon. (e.g., Darby *et al.*, 2006; Krewski *et al.*). The traditional metric for exposure to miners has been the working level month (WLM), a measure of exposure to the short-lived progeny of radon [hereafter radon].

To implement its system of radiological protection, the International Commission on Radiological Protection (ICRP) has established a dose conversion convention (DCC) to allow doses from exposure to radon to be added to doses from other sources of radiation exposure. ICRP 65(1993) provided an epidemiologically based DCC (i.e., mSv per WLM) calculated by dividing the risk of lung cancer derived from epidemiological studies of miners by the detriment from external radiation based primarily on data from follow-up of Japanese atomic bomb survivors[1].

In its November 2009 statement on radon, the ICRP indicated that, based on their recent re-estimation of lifetime excess absolute risk (LEAR) based on epidemiological studies of miners (especially French and Czech miners), that the nominal risk coefficient for exposure is about a factor of two higher than the current risk factor. Since the DCC is proportional to the LEAR, the corresponding DCC is also expected to increase by a factor of about two. ICRP 115 (ICRP 2010) confirmed this increase in risk and also that the ICRP proposes moving from the current epidemiologically based approach to treat radon in the same way as other radionuclides within the ICRP's system of protection and to publish dose coefficients calculated using dosimetric models.

The importance of smoking has been noted by the ICRP who state that "*Furthermore, radon appears to act in a more multiplicative than additive manner on the underlying rates of lung cancer of the exposed population. Thus, for the same radon exposure, the risk of lung cancer from radon for smokers is substantially greater than that for non-smokers*". (Guest Editorial to ICRP 115, 2010) This is an important observation as current radon risk projection models are all relative risk models and thus, the

---

[1] It should be noted that since the risk from radon exposure is to certain cells in the respiratory tract, the comparison is, in effect, a comparison of risk of lung cancer and risk of cancer from whole body exposure.

estimation of LEAR, and in turn DCC, is strongly influenced by smoking prevalence in the reference populations[2].

This paper examines the role of smoking on the estimation of risk from exposure to radon and suggests an approach to addressing smoking prevalence in the application of the ICRPs proposed new approach to protection against radon exposure. A short commentary on radon dosimetry and smoking is also provided.

Epidemiological Based Dose Conversion Convention

ICRP 65 (ICRP 1993) adopted a DCC based on comparison of risks from exposure to radon derived from studies of miners exposed to high levels of radon and decay products in the past and risk from exposure to external whole body radiation as illustrated in the following equation:

$$DCC\,(\,mSv\,/\,WLM\,)=\frac{risk\,(\,LEAR\,)\,/\,WLM}{risk\,/\,mSv}\,[1]$$

Where:
- DCC is dose conversion convention,
- risk refers to lifetime excess absolute risk (LEAR) per WLM, and
- risk per mSv refers to the ICRP's total detriment per mSv as defined in ICRP 103 which has been revised since ICRP 65. This was based on data for atomic bomb survivors.

While the dose conversion convention (DCC) has units of dose, it is simply, an epidemiological risk ratio and not related biokinetic dose modeling. The risk from exposure to radon – namely, lifetime excess absolute risk or LEAR – is a key factor. The LEAR is estimated using risk projection models from various epidemiology studies and baseline mortality and cancer data which are combined in life tables for calculation purposes [e.g., Tomasek *et al.* (2008); ICRP 115 (2010); Chambers and Stager (2011)].

Epidemiological models are inherently uncertain. This arises from a combination of factors, including the form of the risk projection model(s), underlying biological processes, unavoidable variability as may arise from individual smoking habits for example, and uncertainty in the inputs including physical and

---

[2] That is, the epidemiologically based radon risk models are applied to the risks of lung cancer in the underlying, baseline or reference populations. In other words, the increased risk assigned to radon is actually the risk arising from the combination of underlying lung cancer rates in the reference population which arise primarily from smoking and exposure to radon.

3

biological parameters. Judgemental factors, namely, radiation weighting and tissue weighting factors also affect the conversion of LEAR to mSV. The importance of smoking characteristics is illustrated in Table 1 which shows the results of applying seven risk projection models to mining workforce with annual exposure of 2 WLM/yr from 18 to 64 years and a range for smoking prevalence, from 100% never smokers to 50% never smokers.

As illustrated by the data in Table 1, the nominal DCC risk to a population of lifelong smokers from radon and smoking (based on estimates of LEAR) can be more than 10 times larger than the nominal risk of lung cancer from radon in a population of never-smokers with the remaining assumed to be smokers. Excluding the values for GSF (ICRP 65) and Darby (2006), the median DCC over range of smoking prevalence shown in Table 1 is about 6-7 mSv/WLM with a 20-fold range from about 1 mSv/WLM (for Ontario miners) to 21 mSv/WLM (from Eldorado miners)) depending on the smoking prevalence.

Table 1   DCC (mSv per WLM) as Function of Prevalence[3] of Non-Smokers

	% Non-smokers					
	100	**90**	**80**	**70**	**60**	**50**
GSF (ICRP 65)	2	3	4	6	7	8
BEIR VI	3	6	8	11	14	16
FrenchCzech	3	5	8	10	12	15
Ontario	1	3	4	5	6	7
Eldorado	3	7	10	14	17	21
Wismut	2	3	5	6	8	9
Darby[4]	1	3	4	5	6	8

As illustrated in Figure 1, at present, an overall smoking prevalence for males and females combined is generally in the range of 20 to 30%, and for males only, generally in the range of from 20 to 40%. Smoking prevalence appears to be decreasing world-wide[5] and it seems reasonable to assume that in time, the rates of lung cancer in the reference populations will also decrease eventually, assuming continued use

---

[3] Estimates of LEAR are made using life table methods which account for age-dependent rates of lung cancer and competing causes of mortality in addition to taking account of the seven different risk projection models. See Chambers and Stager (2011) for details of calculations.

[4] The GSF results are provided for context and comparison to previous work of the ICRP 65 but are quite comparable to the results for the Ontario model. The Darby model (2006), based on residential radon, is provided for further context.

[5] See for example the WHO and the World bank:

WHO Tobacco Control Database: http://data.euro.who.int/tobacco/?TabID=2402,

WHO (MPOWER Package): http://www.searo.who.int/LinkFiles/Tobacco_Free_Initiative_mpower_report_full_2008.pdf, and

http://web.worldbank.org/WBSITE/EXTERNAL/TOPICS/EXTHEALTHNUTRITIONANDPOPULATION/EXTETC/0,,contentMDK:20225516~menuPK:478889~pagePK:148956~piPK:216618~theSitePK:376601,00.html

of relative risk projection models, this will also lead to an eventual reduction in both the LEAR and the associated DCC.

For smoking prevalence in this latter range, i.e., 20 to 40%, the median DCC is about 6-7 mSv/WLM. It may be that a "nominal" population average DCC of about 6-7 mSv/WLM corresponding to a "nominal" smoking prevalence of 30% seems reasonable for radiological protection purposes. The use of such a "notional" radon DCC would seem to be reasonably protective for the majority of the population. For smokers, the average radon risk factor is not as protective, but their overall risk for lung cancer is dominated by smoking. The simple reality is that smokers can substantially reduce their risk from premature death from lung cancer by giving up smoking, much more than by reducing their radon exposure.

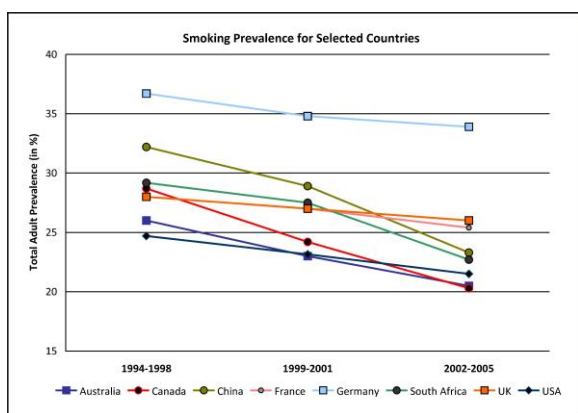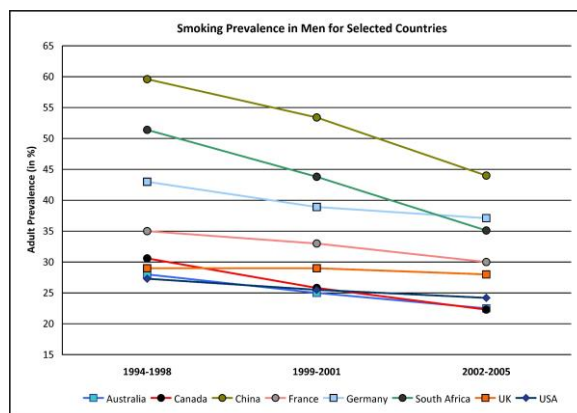Figure 1(a)   Smoking Prevalence (Country)   Figure 1(b)   Smoking Prevalence (Male)

Dosimetric Considerations

The risk of lung cancer arising from exposure to radon and its decay products arises from the deposition of the alpha particle emitting radon decay products on the bronchial airways. In general terms, two types of radon decay product bronchial dose models have been published. One type is biologically based, calculating airway deposition using data from hollow casts of the human bronchial tree. The other type assumes decay product deposition fractions in several compartments that represent sections of the bronchial tree. The alpha dose is calculated to basal or mucous cells at specific depths in the bronchial epithelium from the estimated activity on the airways or in compartments.

Dosimetric models show that the dose per unit intake of radon decay products depends on the site of deposition in the respiratory tract. In turn, the site of deposition depends strongly on the particle size distribution of the radon decay products, especially small sized (ultra-fine) particles below about 10 nm

5

diameter (commonly referred to as the "unattached fraction") which is a critical factor in the modeling of radiation dose to the tracheobronchial tree.

In the ICRP's system of radiological protection, the effective dose (E) is used as a "*protection quantity*". The effective dose[6] depends on the absorbed dose (a physical quantity) adjusted by a radiation-weighting factor (w_R) to account for relative biological effectiveness of different radiation and a tissue-weighting factor (w_T) to account for the different radiosensitivities of various organs and tissues. These factors were most recently reviewed by the ICRP in Publication 103.

Tissue weighting factors are derived from the calculations made for radiation detriment as described in ICRP Publication 103. The detailed method and process in calculating radiation detriment is outlined in ICRP Publication 103 (e.g., A140-152 and Box A.1). The radiation detriment associated with each type of cancer is normalized to a sum and the relative radiation detriment is determined and used for tissue-weighting factors. The ICRP currently uses a nominal radiation weighting factor of 20 and a tissue-weighting factor for the whole lung of 0.12 (and 0.08 for the bronchial tree).

Other (judgemental) factors might be considered. Brenner *et al.* (1995) for example, suggest that a radiation-weighting factor of 20 may be too large for radon and recommended a value of 10 for residential exposure. The ICRP (11/07/2011) acknowledges that *"It is possible that the dosimetric estimates are generally higher because they include a radiation weighting factor of 20 for alpha particles, chosen for use in the calculation of the ICRP quantities, equivalent and effective dose. It may be that the relative biological effectiveness (RBE) of alpha particles compared to gamma rays for lung cancer induction, included implicitly in the dose conversion convention is closer to 10 than 20."* Similarly, other tissue weighting factors have been reported as for example, by Harley *et al.* (1996) who estimate the dose specifically to the bronchial region and use a w_T, of 0.08 for the bronchial tree.

A recent paper by Baias, *et al*. (2010) reports biokinetic based dose conversion factors DCFs (in units of mSv/WLM) for four different categories of smokers. Physiological and morphological changes to the lung induced by smoking were accounted for, using aerosol parameter values fixed for a mine atmosphere[7]. DCFs reported by these authors include a DCF of 7.2 mSv/WLM for non-smokers with a range of from 1.74 mSv/WLM for heavy short-term smokers (and light long-term smokers) to a

---

[6] The radon decay products are relatively short-lived and only affect cells and tissues at the surface of the lung. This raises the question of whether the dosimetric approach is fully suitable here, as the concept of effective dose is based on (equivalence to) uniform whole body radiation.

[7] Baias *et al.* 2010 cite Marshall, Birchall and Davis, (2002) in this regard.

maximum of 13.34 mSv/WLM for heavy long-term smokers. The observation that the DCF for a non-smoker is higher than for a short term heavy smoker or a long-term light smoker, does not comport with observations from epidemiology. Moreover, it is well established that the risk to smokers, compared to non-smokers, can be 20 or more times larger than the risk to non-smokers (see for example, Darby *et al.* (2006)). Epidemiology suggests a much larger (>10 fold) range of risks than would be implied by biokinetic based dose models such as that of Baias *et al*. This difference is perhaps explained by biology, in particular, non-specific sensitivity of key cells/tissues arising from smoking.

Conclusions

The ICRP now recommends a doubling of its nominal risk coefficient for radon-induced lung cancer. In addition, the ICRP proposes to treat radon in the same fashion as it deals with other radionuclides in its system of radiological protection, namely, using ICRP's biokinetic dose models and that it will provide "nominal" dose coefficients calculated using its biokinetic dose models and "nominal" parameter values.

In considering the use of biokinetic dose conversion factors (DCFs), it is important to understand that mine environments vary widely not only between mines but also within mines and with time. This variability presents challenges to the development of measurement programs and dosimetric evaluations and determination of the dose "averaged" over the varying mine conditions. There are difficulties in measuring these conditions for exposures within a mine environment so that the average dose over the exposure period can be determined.

Tabulations of published biokinetic model based dose factors are given in UNSCEAR (2006, Annex E) and ICRP Publication 115 (ICRP 2010). Figure 2 shows the range of epidemiological DCCs reported earlier in this paper along with the range of biokinetic dosimetric dose factors (DCFs) from Table B.1 of ICRP 115 (2010) and those reported by Baias *et al.* (2010). The ranges of DCCs and DCFs overlap. Interestingly, the dose conversion factor for non-smokers reported by Baias *et al.* (2010) is about the same as the epidemiologically based DCC for 30% smoking prevalence. ICRP 115 (2010) indicates that the epidemiological based dose conversion convention gives nominal values close to 10 mSv/WLM and that this is consistent with the dosimetric based factor. We suggest that this "coherence" may well be fortuitous and that further consideration of the DCC would be appropriate.

As indicated earlier, using a mixed population of smokers and non-smokers to determine the average radon risk factor would seem to be reasonably protective for the majority of the population. Given current smoking prevalence and a nominal DCC value of the order of 6-7 mSv/WLM corresponding to

7

the current "notional" 30% prevalence, we conclude that a "notional" value is reasonable and reasonably close to the current DCC of 5 mSv/WLM.

Moreover, given the apparent decreasing trend in smoking prevalence, such a value, *i.e.*, a radon dose conversion convention (DCC) of 6-7 mSv/WLM would seem to be reasonably protective of miners, appropriate for current smoking prevalence and baseline risks of lung cancer, likely "conservative" for future reference populations, and consistent with the ICRP's use of nominal conversion coefficients.

Figure 2   Ranges of Radon Dose Conversion (DCC) and Dose Conversion Factors (DCF)
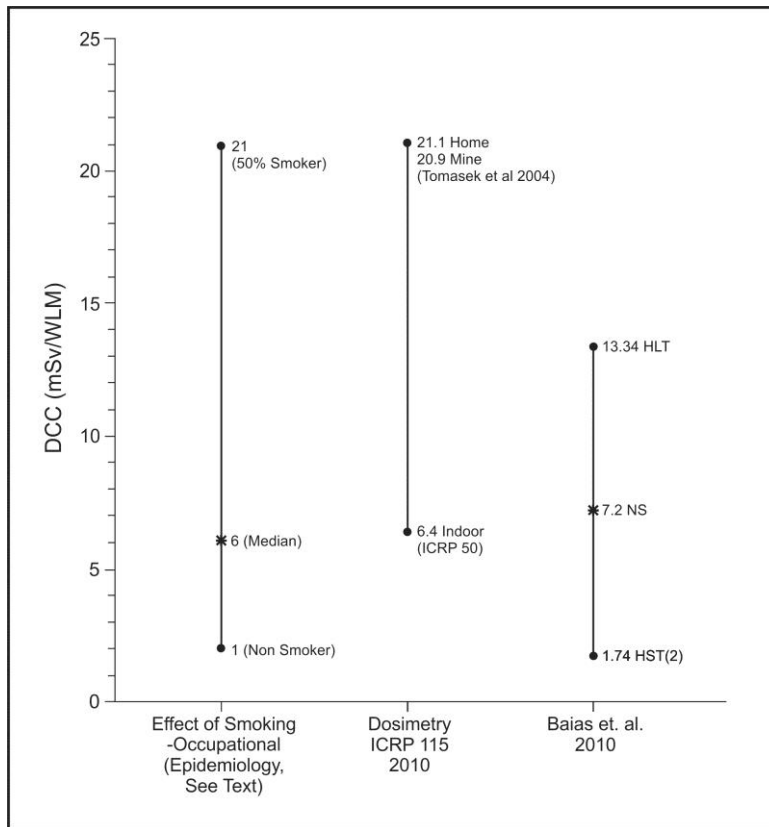
HLT = heavy long-term smoker; NS = never smoker; HST = heavy short-term smoker

References

Baias, P. *et al.* 2010. *Lung Dosimetry for Inhaled Radon Progeny in Smokers.* Radiation Protection Dosimetry (2010), Vol. 138, NO. 2, pp. 111-118.

Brenner, D.J., R.C. Miller, Y. Huang *et al*. 1995. *The Biological Effectiveness of Radon-Progeny Alpha Particles.* III. Quality Factors. Radiation Research, 142 (1): 61-69.

Chambers, D.B. and R.H. Stager 2011. *Prediction of the Variation in Risks from Exposure to Radon at Home or at Work.* Radiation Protection Dosimetry, Vol. 146, No.1-3, pp. 34-37.

Darby, S., D. Hill, A. Auvinen *et al*. 2005. *Radon in Homes and Risk of Lung Cancer: Collaborative Analysis of Individual Data from 13 European Case-control Studies*. Br. Med. J. 330(7485): 223 (2005).

Harley, N.H., B.S. Cohen, E.S. Robbins 1996. *The Variability in Radon Decay Product Bronchial Dose.* Environ. Int. 22 (Suppl. 1), S959–S964.

Harrison and Marsh 2011. *Radon Dosimetry.* Presentation to the ICRP 2011 International Symposium, Bethesda, USA, October 2011.

International Commission on Radiological Protection (ICRP) 2011. *Lung Cancer Risk from Radon and Progeny and Statement on Radon*. ICRP Publication 115; Ann ICRP 40 (1).

International Commission on Radiological Protection (ICRP) 2009. *Statement on Radon.* November.

International Commission on Radiological Protection (ICRP) 2007. *The 2007 Recommendations of the International Commission on Radiological Protection.* Annals of the ICRP, ICRP Publication 103, Vol. 37, Nos. 2-4.

International Commission on Radiological Protection (ICRP) 1994. *Human Respiratory Tract Model for Radiological Protection.* ICRP Publication 66. Ann. ICRP, **24**(1-3) (Oxford: Pergamon Press) (1994).

International Commission on Radiological Protection (ICRP) 1993. *Protection Against Radon-222 at Home and at Work*. Annals of the ICRP, ICRP Publication 65, Vol. 23, No. 2.

Krewski, D., J. Lubin, J.M. Zielinski *et al.* 2005. *Risk of Lung Cancer in North America Associated with Residential Radon.* Epidemiology 16(2): 137-145 (2005).

Marsh, J.W., A. Birchall and K. Davis 2005. *Comparative Dosimetry in Homes and Mines: Estimation of K-factors.* In: Proceedings of Seventh Symposium – The Natural Radiation Environment, Rhodes, May 2002 (Amsterdam: Elsevier) pp. 290-298.

NAS-NRC 1999. Committee on the Biological Effects of Ionizing Radiation (BEIR VI) 1999. *The Health Effects of Exposure to Radon.* National Research Council (NRC). Washington, DC: National Academy Press.

National Council on Radiation Protection and Measurements (NCRP) 1987. *Exposure of the Population in the United States and Canada from Natural Background Radiation.* NCRP Report NO. 94 (National Council on Radiation Protection and Measurements, Bethesda, Maryland).United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) 2006. *Effects of Ionizing Radiation.* UNSCEAR 2006 Report, Volume II, Scientific Annex E.