RADIATION EXPOSURE STANDARDS FOR RADON DAUGHTER BASED ON LUNG CANCER RATES IN NONSMOKERS*

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Introduction:

Risk based standards for allowable emissions of radionuclides to the environment require risk estimates which are as precise and accurate as possible. For radon and its associated decay products or "daughters" two studies of uranium miners, one from the United States (1, 2) and one from Czechoslavakia (3,4), are of sufficient size and duration to yield reasonably precise estimates of health effects, but their accuracy when applied to the general nonmining population is questionable because the principal health effect associated with radon daughter exposure is lung cancer and the principal cause of lung cancer is cigarette smoking. This would not present a problem, if smoking and radon daughters act additively in the induction of lung cancer, but the results of two recent analyses of the American miners data (5,6) as well as our review of the Czech studies suggest multiplicative interaction. The following discussion shows that, if smoking and radon act multiplicatively: (1) the smoking status of the population at risk in large part determines the magnitude of one's risk estimate, and (2) risk based standards for radon based on the general population contain a "hidden subsidy" for smokers. For these reasons, we argue that radon health effects coefficients and resulting risk based standards should be based on relative risk models applied to nonsmoking populations.

The Evidence for Multiplicative Interaction

Until recently, work on the American miners data seemed to suggest that though radon induced lung cancer might occur somewhat sooner (i.e., shorter latency) in smokers than in nonsmokers, the ultimate risk of lung cancer per unit of radon daughter exposure (commonly expressed as working level months (WLM) (7)) was comparable (2). However, a later study (6) using a Cox proportional hazard model (8) showed the interaction between smoking and radon daughter exposure was best expressed by a multiplicative model of the form:

$$R_{se} = R_s \cdot R_e \qquad (1)$$

where R_{se} is the risk in individuals exposed to both radon and smoking, R_s is the risk in those exposed to only smoking, R_e is the risk in those exposed to only radon, all relative to risk in those exposed to neither. A still more recent evaluation of the same data (5) verified that equation (1) gives a good fit to the American miners data and used an extension of the Cox model (9) to formally reject an additive model.

The Czech miner data has not had the benefit of analysis by the newer procedures mentioned above, but BEIR III (10) notes that relative risks of 4.7 and 5 are seen in radon exposed smokers and nonsmokers respectively, assuming that background lung cancer rates in Czech smokers are 10 times those in nonsmokers. Thus, it appears that multiplicative interaction is the rule in these data also.

Calculating the Influence of Smoking

Actuarial populations of "pure" smokers and nonsmokers were developed using the 1969 United States abridged life tables for white males and females (11), lung

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cancer rates for nonsmokers taken from the American Cancer Society Study of nonsmokers (12), and general smoker mortality data from the 1979 Surgeon General's Report (13). This calcuation assumes known age specific lung cancer death probabilities for smokers, a constant all cause standardized mortality ratio (SMR) (14) for smokers versus nonsmokers for ages 40 and above (we assume 1.75 for males and 1.5 for females), and known age specific percentages of smokers in the base population (we assume 10-45% in males and 10-33% in females, depending on age). This information is used to separate the general population life table into two life tables, one for smokers, the other for nonsmokers. Details of the procedure are given in Ginevan (15).

Given these life tables we can calculate health risk for a particular level and duration of radon daughter exposure and either a relative risk (RR) model of the form:

$$Q_{ex} = B \cdot d_x \cdot Q_{cx} \qquad (2)$$

where Q_{ex} is the age specific excess probability of dying of lung cancer, B is a risk coefficient with units of percent increase per WLM, d_x is the effective exposure in WLM at age x, and Q_{cx} is the baseline probability of dying of lung cancer at age x, or an absolute risk model (AR) of the form:

$$Q_{ex} = C \cdot d_x \qquad (3)$$

where C is a risk coefficient with units of cancers per year per WLM. The modeling approach is a cohort life table (16) which calculates effects in terms of premature deaths per 100,000 persons and months of lost life expectancy. The model itself is described in detail in Ginevan (15).

Modeling Results and Discussion

Our radon risk evaluation assumed the parameters given below:

Period of exposure - lifetime; exposure per year-1 WLM; $^{+}$latency-10 years; *age at first risk-30; relative risk coefficient - 0.01/WLM; absolute risk coefficient - 1×10^{-5} lung cancers per year/(WLM.person).

The results of our risk modeling are shown in Table 1. (We note the differences between smokers and nonsmokers in terms of both life expectancy and risk of lung cancer are in accord with those given in the Surgeon General's Report (13)). Under the relative risk model there are dramatic differences in the impact of radon exposure, depending on the smoking status of the population at risk. For the excess death measure, the ratio of the highest risk (smoking males) to lowest risk (nonsmoking females) populations is about 13.1. For loss in life expectancy the same ratio is about 11.7. Absolute risk models appear to provide a way out in that the same ratios are 1.5 for excess deaths and 1.8 for loss in life expectancy. However, this ignores the fact that our risk coefficients were derived from miner populations, over half of whom are smokers, and as shown earlier absolute risk models do not fit these data.

+Assumes dose must be received at least 10 years in the past to affect present lung cancer risk.

*Assumes lung cancer risk is zero before age 30 regardless of dose.

The "hidden subsidy" mentioned earlier arises because a given amount of radon daughter exposure is about 4 times more hazardous if one takes the general population rather than nonsmokers as a baseline. This difference is entirely attributable to smoking. For the same degree of protection a risk based standard for the general population is therefore about 4 times lower than one for nonsmokers. The cost of this fourfold reduction (which might be small or large depending on the scenario) is our "hidden subsidy."

One might argue that this subsidy is justifiable, but this contention is vitiated by considering the health risk voluntarily assumed by smokers. Reducing risk in nonsmokers to a level of 10^{-5} to 10^{-6} per person per year would limit exposure to about 0.1 WLM per year (17). An additional reduction to about 0.025 WLM per year would be required if general population risks are considered. The health benefit to smokers of this 0.075 WLM difference is about 250 excess lung cancer deaths per 100,000 or about .4 months of life expectancy. Compared to nonsmokers, smokers' excess lung cancer risk is about 5280 cases per 100,000 and their loss in life expectancy is about 4.6 years. Therefore the health benefit is either 5 percent (excess deaths) or 0.7 percent (life expectancy) of the risk smokers have voluntarily assumed. Thus, if the cost of the fourfold reduction in radon daughters dictated by the presence of smokers in the population is even moderately large, it seems difficult to justify.

Conclusions

Radiation protection standards for radon daughters based on the general population contain a "hidden subsidy" because of enhanced risk in radon daughter exposed smokers. While the cost of this subsidy is variable, the health benefits to smokers are small relative to the risk they have voluntarily assumed. The principal conclusion of this exercise is therefore that radiation protection policy for radon daughters should be based nonsmokers, except where the "hidden subsidy" for smokers, inherent in standards based on the general population, is negligible. Validation of such a policy will require careful consideration of what "reference" nonsmoking populations should be assumed. Likewise, careful consideration should be given to both new analyses of existing data sets, and if possible studies of new populations, to rigorously test the assumptions put forward here. We feel that such efforts will allow development of a radiation protection policy for radon daughters which is both equitable and cost effective.

TABLE 1: LIFE EXPECTANCY (LE) LOSS IN LIFE EXPECTANCY, LIFETIME RISK OF LUNG CANCER AND EXCESS LUNG CANCER DEATHS (GIVEN AS DEATHS PER 100,000 AT RISK), FOR SIX POPULATIONS EXPOSED TO 1 WLM PER YEAR LIFETIME, UNDER THE RR AND AR MODELS.

	MALES			FEMALES		
	GENERAL POPULATION	NON-SMOKERS	SMOKERS	GENERAL POPULATION	NON-SMOKERS	SMOKERS
LE	67.82	70.29	64.81	75.23	76.48	72.69
LE Loss (Months)						
RR	3.94	0.56	6.55	1.20	0.56	2.29
AR	3.95	4.52	3.26	5.45	5.78	4.78
Expected Deaths	4662	683	9529	1157	623	2344
Excess Deaths						
RR	2641	426	5213	669	398	1295
AR	1714	1874	1519	2152	2238	1977

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