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Radon Chemico-Biological Interactions

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Abstract

The National Academy of Sciences (USA) investigated on radon's negative impacts on health (BEIR VI). This paper was well-written, well-researched, and affected radon remediation policies and laws.At home, three of the issues with the report's interpretation are highlighted in this article. First, dwellings with radon levels below US standards provided the majority of the radiation dosage needed to evaluate risk. action level established by the Environmental Protection Agency to ensure that remediation would have little effect on the overall assessed attributable risk As a result, the low-level residences (i.e., below the action level) would only have a modest impact after being remedied. Impact on the estimated "risk to the population attributed" In specific houses with exceptionally high radon levels, remediation may only slightly lower each person's risk. Second, the message conveyed to the general public, government officials, and legislators was that "Next to cigarette smoking, radon is the second leading cause of lung cancer." not a true assessment of the report. The correct conclusion would be: Next to cigarette smoking, high levels of radon combined with cigarette smoking is the second leading cause of lung cancer. In the never-smokers, few cancers could be attributable to radon. Thirdly, there is little question that high levels of radon exposure in mines combined with cigarette smoke and other significant insults in the mine environment produces excess lung cancer. However, the biological responses to low doses of radiation differ from those produced by high levels. Low doses may result in unique protective responses (e.g. against smoking-related lung cancer). These three points will be discussed in detail

Keywords: ***Radon; Radiation; LNT; Lung cancer; Cigarette smoking; BEIR VI; Cancer riskmethod***

INTRODUCTION

The series of naturally occurring radioactive decay products, which begins with uranium-238, includes the colourless, odourless monatomic nobel gas radon (238U). Radon has extremely little chemical reactivity under normal circumstances, therefore breathing it in alone has little biological impact. However, radon-222 (222Rn), which has a 3.82 day half-life and creates a number of short lived daughters that release alpha, beta, and gamma radiation when they decay to become stable lead, is produced when radon-226 undergoes a complicated decay chain. In addition to many earlier works, BEIR VI provides a full description of the degradation chain. Even though radon is a noble gas, the radon daughters are charged and attach to small airborne particles in the environment so that they can be inhaled and deposited in the respiratory tract including the deep lung. The “attached fraction” and the “equilibrium” are both important variables which depend on the environment where the radon is released. These factors impact the local absorbed radiation doses throughout the respiratory tract . To characterize exposure to Uranium miners and individuals in homes, a metric was developed which is called a Working Level Month (WLM). This is the result of the average radon concentration and exposure time in a radioactive environment over time. Converting WLM into a radiation absorbed dose in Gy to the lung, an equal dose in Sv to the lung, or an effective whole-body dose in Sv is crucial (or related units). Biological dosimetry and dosimetric modelling have been employed in BEIR VI to estimate the radiation absorbed dose to the lung. One Gy/WLM was the central estimate found. However, it appears that the uncertainty surrounding this number has not been addressed, which limits the ability to characterise uncertainty accurately for estimations of radiation absorbed dosage.

There is no doubt that the combination of the worker environment and lifestyle, high radon concentrations in uranium mines, and related absorbed

radiation doses to the respiratory system led to a rise in lung cancer. In order to calculate the risk of lung cancer from radon inhalation for a variety of exposure levels, the radiation dose estimates and lung cancer data for uranium miners were coupled with the dose derived from within-home radon exposure. Multivariate risk models and epidemiology studies have been used to show how radon levels in houses affect the frequency of lung cancer. Utilizing multivariate models enables the adjustment of some covariate affects as well as many risk variables. Additional research employing meta-analysis of data from multiple studies has led to the claim of evidence for an increase in lung cancer risk as radon levels in homes increase. This information supports the basis for current risk estimates for radon in homes and is claimed to be consistent with the values of risk derived using the uranium miner data. Based on the result of the indicated studies, the International Commission on Radiation Protection (ICRP) has revised its lung cancer risk recommendations. They have suggested: “a lifetime excess absolute risk of 5×10^{-4} per WLM [14×10^{-5} per (mJh/m^3)] should now be used as the nominal cancer risk coefficient for radon-and radon-progeny induced lung cancer, replacing the previous value of 2.8×10^{-4} per WLM [8×10^{-5} per (mJh/m^3)]. In order to make radon regulations compatible with those for other internally deposited radionuclides, the ICRP also recommended that they be based on exposure to the respiratory track. What we actually know about the biology of low dose radiation has been examined in light of the strength of epidemiological research. It was shown that epidemiology studies need high sample numbers in order to accurately identify the risk in the low dose range and the form of the dose-response relationship in the dose range experienced in households. Epidemiologists assert that combining data from different research might assist to reduce uncertainties, but as shown in a recent study, uncertainty seems to dramatically "rise" when using pooled datasets.

RESULTS

Issue 1

The first issue was the addition of doses to the target organ of interest that were individualised to each individual, which included many people who lived in homes with radon levels below the action level. The term "collective dosage" refers to the sum of the individual-specific doses that results, and its use denotes the use of LNT as the null hypothesis. Since the inclusion of high-dose data ensures a positive slope with a locked intercept, the adoption of LNT as a null hypothesis, regardless of the data, practically guarantees an LNT conclusion. In this case, the intercept position does not accurately represent the variation in the low dose range. It is challenging to reject the null hypothesis given this situation . Therefore, collective dose cannot be reliably applied to threshold or other nonlinear dose-response relationships. In BEIR VI the distribution of radon concentrations in homes was characterized based on radon measurements and was converted to corresponding radiation absorbed doses (i.e., unweighted dose) using a dose conversion (from WLM to absorbed dose) factor. The sum (collective dose) of all the individual-specific absorbed radiation doses to the respiratory tract in homes was used as the population-level dose metric.

Issue 2

The primary conclusion of the BEIR VI report, which was largely embraced and applied, presented the second issue. The message was that radon is the second-leading cause of lung cancer, after cigarette smoking. The "Estimated number of lung cancer fatalities in the United States in 1995 attributed to indoor residential radon progeny exposure" is well-resumed in this article. With 157,400 fatalities from lung cancer in men and women combined this year, the table demonstrates how common this disease is. About 95% of lung cancer cases in men were caused by cigarette smoking, while 90% of lung cancer cases in women were caused by smoking. The range of malignancies related to radon in the group of non-smokers was 1200-2900. High radon levels may be the second major cause of lung cancer since they enhance the risk of lung cancer by 12% when paired with cigarette smoking. This graph also shows that there would have been 135,300 fewer lung cancer cases projected for this year if everyone with radon in their houses gave up smoking. There would be a total of around 18,000 fewer cancers if everyone having radon in their houses had it completely remedied, which is impossible because radon is prevalent both indoors and outdoors. Only 1970 of those cancers were estimated to have been produced among non-smokers.

Lung cancer risk may rise with inhalation of additional environmental substances that lead to chronic lung disease. It has been demonstrated that breathing in diesel exhaust at high concentrations causes the lungs to get overloaded with particles, leading to a chronic lung condition that ups the chance of developing lung cancer. This was once believed to be related to chemical carcinogens connected to the diesel particles early in the investigation. However, later studies showed that the identical response was produced by inhaling large quantities of carbon black particles without any chemical carcinogens. This suggests that any high level insult to the lung which creates the potential for a chronic inflammatory disease results in an environment that increases lung cancer risk and may be responsible for the synergistic interaction seen between cigarette smoke and radon exposure. Interestingly low-dose radiation such as from inhaled radon suppresses disease promoting inflammation

Issue 3

The third issue here is the apparent discrepancy between the cancer risks estimated for the low-dose zone determined from human Mechanistic research, epidemiological studies, and lung cancer risks are all obtained from experimental animal investigations. This is especially true for dangers that are anticipated from epidemiologic data from studies of human populations and those caused by internally deposited radioactive material in experimental animals. The human data are often based on study designs that favor an LNT outcome and thereby suggest

elevated risks in the low dose region where as the extensive laboratory research on the effects of internally deposited radioactive material support cancer induction only with very high doses. The experimental animal data was carefully reviewed in the late 1980's and showed that large concentrations of internally deposited radioactive materials, which resulted in large absorbed radiation doses, were required to produce cancer. These data have been re-evaluated and similar conclusions reached for many cancer types produced by either high or low LET radiation following

internal deposition of radioactive materials including radon. The dangers posed by radon are a good illustration of these variations. For radon, it appears that the interaction between high levels of radon and smoking and lifestyle choices in people is a significant factor in the disparities between human data and experimental animal data. For high-level radon exposure, this interaction appears to be more potent than additive and may potentially increase the risk. According to hormetic dose-response relationships, large levels of radon appear to inhibit the immune system in contrast to low levels, which appear to promote anticancer immunity and protect against lung cancer caused by smoking.

SUMMARY

This paper shows, as shown in the BEIR VI report, that radon remediation in houses has little bearing on the total cumulative dosage and that the majority of the dose in the computation originates from homes where the radon level is below the EPA action limit. Therefore, it would be expected that the risk assessments' remediation would have little effect on public health. As a result, the legislation requiring radon testing in properties before sale should concentrate on those with significantly higher radon levels, where there is little doubt over the possibility of an increase in lung cancer among smokers. It is incorrect to conclude from BEIR VI that radon exposure is the second most common cause of lung cancer. First and foremost, remediation has little effect on radon risk in dwellings, according to BEIR VI's true findings. Second, the vast amount of data indicates that while low levels of radon appear to protect against inflammatory illnesses, high levels of radon interacts with other environmental insults in a way that is more than additive to increase cancer risk. Smoking, diesel exhaust, mining dust, and maybe inhaling any particulate substance that is pro-oxidant, like aluminium, are all environmental lung insults that appear to contribute to a chronic inflammatory disease. The inflammatory disease interacts with the radiation from high-level radon in more than an additive way to increase lung cancer risk. The more accurate statement from the BEIR VI report would have to be that high-level radon combined with cigarette smoking may be the second leading cause of lung cancer. Since more than 90 % of lung cancers are present in smokers, the increased risk for lung cancer detected in epidemiology studies in homes may be related to the interaction between high-level radon and smoking and not radon alone.