

Editorial



Radon and Lung Cancer: Disease Burden and High-risk Populations in Korea

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- ▶ See the article “Radon Exposure-induced Genetic Variations in Lung Cancers among Never Smokers” in volume 33, e207.
- ▶ See the article “The Disease Burden of Lung Cancer Attributable to Residential Radon Exposure in Korean Homes” in volume 33, e223.

Radon, a well-known human carcinogen, is a radioactive gas naturally occurring from uranium and thorium in the ground. Among the 34 known radioactive isotopes of radon, ^{222}Rn and ^{220}Rn (thoron) were found at significant concentrations in the human environment with half-lives of 3.82 days and 55.6 seconds, respectively. Thus, outside air typically contains very low levels of radon; however, when contained in a closed environment, it builds up to higher concentrations.

In Korea, a local company has reportedly sold beds with radioactive materials, which is a monazite containing radon and thoron, in an anion powder formed inside the mattress covers, emitting radiation above the dose limit of 1 mSv/year for the public; consequently, health concerns from people who have used these mattresses are becoming a social issue.

Lung cancer is the most evident health effect from radon exposure, and radon is stipulated as the second leading cause of lung cancer, after smoking by the World Health Organization.¹ Radon can spontaneously decay or change to other atoms, called radon progeny (or daughter), which are electrically charged and can attach to tiny dust particles in the air. When the dust particles are inhaled, they can adhere to the epithelia of the lungs. The deposited atoms decay or change by emitting alpha radiation (alpha particles), which can potentially damage cells or DNA in the lungs. Because the alpha radiation travels only short distances (a few centimeters) and is not able to penetrate human skin, it cannot reach any other organs, which resulted in the lung cancer as the only potentially important cancer hazard posed by radon. Although some studies indicated that radon exposure was associated with diseases other than lung cancer such as leukemia and skin cancer, these findings were not consistent with other studies and lacked biological feasibility.

The contribution of radon to lung cancer can vary among nations and individual characteristics. In Korea, about half of the natural background radiation is from radon, and its concentrations are higher than ones in the global average.² However, information based on the Korean population on those who are at high risk at the individual level and how the disease burden in the population level are limited. In this regard, the recent two papers by

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Choi et al.³ and Kim and Ha,⁴ published in a timely manner, give us a better understanding of genetic variations in lung cancers and the disease burden associated with radon exposure in Korea.

In order to more effectively reduce the individual risk from radon exposure, knowing who is more susceptible for radon-induced lung cancer is necessary. Exploring genetic susceptibility for the risk of lung cancer due to radon exposure can provide some clues on who are at higher risk, because of the limited information on risk factors associated with radon-related lung cancer. Even common risk modifiers for radiation-related cancer (mostly exposure to X-ray and gamma ray) such as age at exposure and gender have not been observed clearly as risk of lung cancer associated with radon exposure. Several genome-wide association studies investigated the association of genetic polymorphisms with the risk of lung cancer due to radon exposure, suggesting possible biological mechanisms including gene mutations and chromosome alterations.^{5,6} In addition, lung cancer in never smokers (LCINS) is known to be a disease independent of smoking-associated lung cancer. Because radon is the second leading cause of LCINS and its ethnic and genetic attributes are still unknown, genetic variations in LCINS should be investigated with human data. Choi et al.³ identified several common genetic alterations (CHD4 rs74790047, TSC2 rs2121870, and AR rs66766408) both lung cancer patients and normal individuals exposed to high levels of residential radon, requiring further study to determine whether these genes play a role in developing LCINS after exposure to residential radon. As the genetic alterations for susceptibility to LCINS due to radon exposure remain unclear, further studies should be continuously conducted, taking into account the biological and carcinogenic pathways to induce lung cancer.

For public policy purposes, disease burden is an important measure of population health, often quantified in terms of disability-adjusted life years (DALYs), the combining estimated years of life lost and lived with disabilities. In general, disease burden attributable to radon exposure can be calculated by multiplying the population attributable fraction (PAF) for lung cancer, defined as the proportion of lung cancer attributable to radon exposure by the DALY in lung cancer. Kim and Ha⁴ estimated the disease burden associated with lung cancer based on the national radon survey in 2011–2012. The PAF for lung cancer due to exposure to residential radon was 6.7% for men and 4.7% for women, and the total burden of lung cancer was 12,750 DALY for men and 4,022 DALY for women in 2013. These values were higher than the global average in which PAF was approximately 3.5% for men and 3.3% for women.⁷ The major contribution of higher PAF in Korea than the global average was relatively higher radon concentration. Given that the radon concentrations were likely to be higher in socioeconomically disadvantaged populations,⁴ a radon control policy consistent with health promotion in those populations as a priority should be developed and deployed. And most of all, stop smoking is necessary to reduce overall disease burden because the majority of radon-induced lung cancers are among those also exposed to tobacco smoke.

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