
Estimation of Lifetime Lung Cancer Risk from Radon Inhalation in PNG

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Abstract. There are several known factors causing lung cancer. Exposure to nanoparticles, polycyclic aromatic hydrocarbons (PAHs) and inhalation of radioactive radon gas are the major sources. The World Health Organization (WHO) estimates that radon exposure accounts for approximately 3% to 14% of all lung cancer cases, depending on factors like average radon levels and smoking habits. Research in a Southeast Asian country, estimated 26% of lung cancer deaths in males and 28% in females. These were attributed by indoor radon exposure, with variations depending on local radon concentrations. Radon is the second leading cause of lung cancer in the United States, after cigarette smoking. The EPA estimates that about 21,000 people die each year as a result of radon-related lung disease. Radon is radioactive and can decay into cancer-causing radioactive progeny. These progenies stick onto the Trachea Bronchi region of lung and continuously irradiate the lung tissues unless they are translocated by some physiological process. Radon need to be measured for a long time in the indoor atmosphere to obtain a time averaged estimate of the inhalation dose to the inhabitants. Active radon measurements are more accurate even if they are difficult to deploy for measurements. In the present study, results of measurements of nearly a year long continuous indoor radon measurements at different locations in the city of Lae in PNG will be presented. Using the results of the measurement of indoor radon activity, excess lifetime risk due to the inhalation of radon will also be presented.

Key words: Radon, inhalation, radiation dose, excess cancer risk

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Dr Jojo has nearly 32 years of research and teaching experience in India and abroad. He has successfully carried out 10 funded research projects in India, one in Malaysia and 3 in Papua New Guinea. Successfully supervised 15 doctoral degrees, 4 M Phils and several Postgraduate students. He also has two patents to his credit. Published about 110 papers in referred journals and taken part in more than 100 conferences in various countries. He is the executive editor of the Interdisciplinary Journal of Papua New Guinea University of Technology and reviewer to several renowned journals. He is a member of several professional bodies like Institute of Physics (IoP, UK), South Pacific Environmental Radiation Association (SPERA, Australia), Hong Kong Chemical, Biological & Environmental Engineering Society (HKCBEEES, Hong Kong), The New York Academy of Sciences, (US), Malaysian Institute of Science (Malaysia), African Physical Society, (Ghana), All Kerala Astro-sciences Society (AKAS, India) and Nuclear Tracks Society of India. He has won several awards for teaching and research from different countries.

1. INTRODUCTION

Radon is the only radioactive gas in the uranium series and is a known indoor radioactive pollutant. Radon is produced from the radioactive decay of uranium (^{238}U), which is found in almost all natural materials. Radon is also found in water and escapes from the ground into the indoor air, where it decays into solid progenies. Radon supports 7 alpha active radio isotopes before it decays finally to ^{206}Pb . The building materials, the water supply, soil air, and natural gas can all be sources of radon inside homes (Nguyen Dinh Chau, 2019). The basements of the buildings allow for more opportunity for radon in soil gas to enter into the living space. Exposure to excess radon gas can result in lung cancer. Radon progenies become airborne attached to the aerosol particles in the air we breathe. When inhaled the daughter products of radon can get attached to the lung tissues. When the radioisotopes decay inside the lungs, radiation from these particles can damages lung tissues, and can lead to lung cancer. The onset of lung cancer would usually occur after several years of exposure. Although lung cancer can be treated, the survival rate is one of the lowest among other types of cancers.

Depending on the location, construction type and ventilation conditions radon may get accumulated. The majority of radon exposure in the indoor spaces is from radon entering the rooms from the ground. As a result, radon concentrations are found to be highest in the lowest levels of homes. Long-term exposure to elevated radon levels is the second leading cause of lung cancer after smoking (Dhanya Balakrishnan, 2021). The risk increases significantly for smokers exposed to radon. Based on the geological variability, some areas are naturally predisposed to higher radon levels due to the composition of soil and rocks beneath buildings. Therefore, monitoring ensures compliance with safety standards and helps to take mitigation measures like improving ventilation or sealing foundation cracks to reduce exposure and safeguard health. Estimation of the risk can essentially help to identify, evaluate, and manage potential hazards that could negatively impact individuals. Risk assessment can also guide to develop strategies to eliminate or minimize risks from exposure to higher concentrations of radon. The proportion of lung cancers ascribed to radon has been estimated in the range from 3 to 14%. With respect to the radon level and smoking habits, the risk of lung cancer increases approximately by 16% per 100 Bq m⁻³ for long-term exposure to indoor radon concentration (Morawska L, 2021).

2. METHODOLOGY

Indoor radon varies widely depending on several parameters related to the building materials, geology, atmospheric conditions and ventilation of the room. There are several factors determine the risk of lung cancer caused by radon exposure. Primarily, the concentration of radon in the indoor atmosphere where you spend most of our time. The amount of time one spends in the room, presence and nature of aerosols like cigarette of kitchen smoke, water droplets from air cooler and the like are the other confounding factors. The aerosols facilitate radon progeny to get attached to and become airborne. These progeny goes into the lungs while we inhale. To arrive at a reasonably good representative rate of radon concentration, time averaged long-term measurements are necessary.

There are several methods for long term assessment of indoor radon. Because of simplicity and ease of operation, in most investigations, passive alpha radiation detectors are being used. This method needs no supporting power supply or complicated methods of analysis. At the same time there are arguments that these methods lack reproducibility and precision.

In the present study, we have adopted active continuous measurement of indoor radon concentrations (Becquerels per cubic meter). Ten different locations in the city of Lae were chosen for the study. The device makes hourly measurements of indoor radon concentrations and stores in the data logger. Using the radon data, annual exposure to radon, inhalation dose and the inhalation cancer risks were determined.

3. EXPERIMENTAL

Radoneye+2 Smart radon monitors manufactured by Radon FTLab, Republic of Korea were used for the measurements of radon in the indoor atmosphere. RadonEye+2 device utilizes a pulsed ionization chamber equipped with a high-precision detection circuit to measure radon concentrations. It detects alpha particles emitted during the radioactive decay of radon gas, providing a real-time reading of radon activity concentration. It has a sensitivity of 0.5counts per minute per pCi per litre and has accuracy $\pm 10\%$ at 10pCi per litre. It automatically

takes measurement at every hour and has a storage capacity of data for one year. The device has inbuilt database working with Wi-Fi and Bluetooth connectivity.

Ten indoor locations in the city of Lae were selected to expose the Radoneye+2 detectors. Houses, offices, workplaces and labs were there among the exposure sites. The device measures radon, temperature and humidity of the indoor atmosphere, simultaneously.

The measurements were carried out from June 2024 to January 2025 in all the locations. The results of the continuous measurements were accessed using Bluetooth connectivity of a smartphone. The downloaded data was analyzed using Atmos Light -1.4.2.0 software to obtain the results.

The assessment of radon inhalation risk is based on the air exposure to decay products in units of potential alpha energy concentration (PAEC) in the environment. The unit of potential alpha energy exposure is the working level month (WLM). For domestic (i.e., residential) exposures, 1 WLM is equivalent to an indoor radon concentration of exposure at 185 Bq m^{-3} for a full year with 70% of time spent in the home. The SI unit of exposure replacing PAEC is the joule hour per cubic meter (J hr m^{-3}) and 1 WLM equals $0.0035 \text{ J h m}^{-3}$ (NRC., 1999)

Radon atoms and their short-lived progeny have high mobility. Radon's short lived radioactive daughter products get attached onto atmospheric aerosols (forming the "attached fraction" with a size from a few nanometers to a micrometer). Radon daughters, regardless of whether attached to aerosols or not, diffuse in the atmosphere. They may ultimately get deposited onto the ground or walls of buildings. Practically, the progenies never attain radioactive equilibrium with radon inside the room, completely. The equilibrium fractions of radon's short half-lived progeny are described by the "equilibrium factor", which ranges from 0 (when radon present in the air having no progeny) to 1 (when radon's short half-lived progenies are in the same concentration of radon). When a person is exposed to radon and its short half-lived progenies, whether attached to aerosols or not, the inhaled progeny may get deposited in the respiratory tract and irradiate it. The delivered dose depends on the location of the deposit, which itself is a function of particle size. Finer particles (in particular the unattached fraction) can reach the sensitive cells of the bronchial epithelium and the pulmonary alveoli of lungs and thereby deliver larger doses. Radon being a gas, is largely exhaled and therefore, it only weakly irradiates the lung tissues. Since 1987, the World Health Organization has recognized radon as a known cause of human lung cancer. Epidemiological studies on uranium miners have shown a risk of excess mortality due to lung cancer associated with radon exposure. More recent studies in the general population have shown that this risk is significant for continuous domestic exposure to radon at concentrations above approximately 200 Bq m^{-3} . The action of radon and its progenies, combined with smoking, leads to a higher lung cancer risk somewhere between the sum and the product of the two relative risks (BEIR IV, 1999).

3.1 Estimation of Radon Progeny concentrations

Inhalation of radon gas contributes relatively less dose to the lung as compared with its progeny. Inhalation of alpha emitting short-lived radon progeny and the subsequent deposition of this progeny on the bronchial tree of lungs delivers most of the radiation dose. Therefore, the health risk associated with the inhalation of radon gas depends on radon progeny levels. Radon progeny levels are determined using the equilibrium equivalent concentration (EEC) which is estimated from the measured radon gas concentration (Bq m^{-3}) and the equilibrium factor (F_{eq}). In the present study, we have applied radon equilibrium factor, 0.5 (Reshma Bhaskaran, 2017).

3.2 Lifetime lung cancer risk (LLCR)

There are several radon risk models in the literature. In all risk models, lifetime risk of radon-induced lung cancer increases with increased exposure to radon progeny. The lifetime risk estimates could vary significantly between the various risk models considered.

The alpha dose delivered to target cells in bronchial epithelium arises mainly from the short-lived decay products deposited on the bronchial airway surfaces. The decay products, however, are on the airway surfaces within about 20 to 30 μm of these target cells, and thus have a higher probability of hitting a target-cell nucleus.

The annual weighted equivalent dose from radon gas decaying in the lung has been calculated for the whole lung and bronchial epithelium (Sowby, 1981) (NCRP, 1987). The Annual effective dose gas dose itself is $7 \times 10^{-3} \text{ mSv y}^{-1}$ per Bq m^{-3} (ICRP 1981) or $5 \times 10^{-3} \text{ mSv y}^{-1}$ per Bq m^{-3} (NCRP 1987). The dose from ^{222}Rn gas is

lower by about a factor of 10 compared to the bronchial dose from the decay products deposited on the airways.

For the estimation of Lifetime Lung Cancer Risk (LLCR), the conversion factor proposed by fourth National Research Council Committee on Biological Effects of Ionizing Radiations (BEIR IV) was used.

Excess Lifetime Lung Cancer Risk of 2% per 148 Bqm⁻³ (=0.56WLM) was used (BEIR IV, 1988).

4. RESULTS AND DISCUSSION

Figure 1 shows a typical spectrum of radon measurement by the Radoney+2 detector. The graph shows the variation of radon concentration measured in Bqm⁻³, continuously from mid-June 2024 to April 2025. There are incidences of sporadic spikes of radon concentration which is due to the environmental and subsurface soil radon emanation. The instrument is capable of continuous measurement of temperature and humidity which are not shown in the graph.

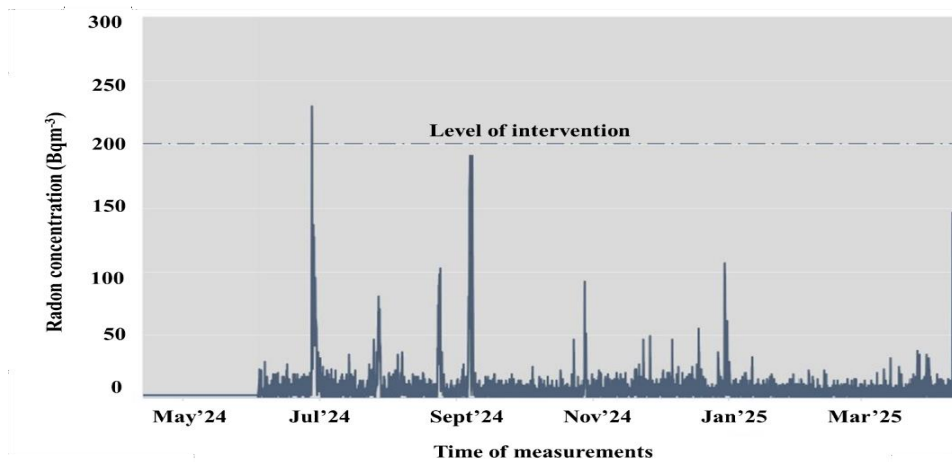


Figure 1 Spectrum of continuous radon measurement by Radoney+2

The data obtained from all the ten devices were compiled and used for determining the Lung Cancer Risk.

Table 1. Radon concentration, Annual Effective Dose and Lifetime Cancer Risk

| Location | Av. Radon Concentration (Bqm ⁻³) | AED (× 10 ⁻³ mSv y ⁻¹) | | Lifetime Lung Cancer Risk Percentage (%) |
|---------------|--|---|-------------|--|
| | | Lungs | Bronchial | |
| 1. House | 10 | 50 | 70 | 0.14 |
| 2. House | 14 | 70 | 98 | 0.19 |
| 3. House | 13 | 65 | 91 | 0.18 |
| 4. Office | 8 | 40 | 56 | 0.11 |
| 5. Office | 10 | 50 | 70 | 0.14 |
| 6. Office | 10 | 50 | 70 | 0.14 |
| 7. Office | 14 | 70 | 98 | 0.19 |
| 8. Workplace | 16 | 80 | 112 | 0.22 |
| 9. Workplace | 12 | 60 | 84 | 0.16 |
| 10. Workplace | 18 | 90 | 126 | 0.24 |
| A.M. | 12.5 | 62.5 | 87.5 | 0.17 |
| S. D. | 3.1 | 15.5 | 21.7 | 0.04 |

The results indicate that the average radon concentrations measured in the locations are that of a normal radiation environment, even if infrequent spikes were observed during measurements. The mean value of radon concentration at the locations (12.5 ± 3.1 Bq m⁻³) was much lower than the global average of 40 Bq m⁻³. For the

estimation of AED, the conversion factors, 7×10^{-3} mSv y^{-1} per Bq m^{-3} and 5×10^{-3} mSv y^{-1} per Bq m^{-3} were used for Bronchial epithelium and the whole lungs respectively. Conversion factor indicates that Bronchial epithelium is more vulnerable for the radiation dose with respect to the whole lungs in general.

The percentile life time cancer risk was estimated assuming 80% occupancy and average longevity of 70 years. The estimated Lifetime Lung Cancer risk was 0.17 ± 0.04 . The estimate risk factors agree with the results of other investigations conducted in various countries.

According to the WHO, the risk of lung cancer due to indoor radon exposure increases by 16% for each 100 Bq m^{-3} , and the dose-response correlation is linear. According to the US EPA, lifetime exposure to 74 Bq m^{-3} lead to 32 and 4 lung cancer cases per 1000 population, for smokers and non-smokers respectively.

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