



Proceeding Paper

Radon Assessment: An Overview of Concentration Variability and Synergies with Other Health Risk Factors in Indoor Air [†]

Evangelos Batris ^{1,*}, Dimitrios Nikolopoulos ², Ioannis Valais ¹ and Konstantinos Moustris ³

¹ Department of Biomedical Engineering, University of West Attica, 12210 Athens, Greece; valais@uniwa.gr

² Department of Industrial Design and Production Engineering, University of West Attica, 12241 Egaleo, Greece; dniko@uniwa.gr

³ Department of Mechanical Engineering, University of West Attica, 12241 Egaleo, Greece; kmoustris@uniwa.gr

* Correspondence: ebatris@uniwa.gr; Tel.: +30-6946337330

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Abstract: Indoor Radon is the second cause of deadly lung cancer and is suspected of inflicting further health risks and synergies with other air pollutants. Radon mapping is based on long-term average concentrations, but the spatiotemporal concentration variability can be very high, delivering higher radiation doses than expected. Continuous monitoring, appropriate indices, and reliable models, aided by digital technologies may improve the assessment and management of indoor Radon risk, especially for vulnerable populations. Further epidemiological studies, research and modelling in dosimetry, new monitoring technologies and methods, and synergies with air quality research, are expected to contribute towards a unified strategy for Radon risk assessment, management, and perception.

Keywords: radon assessment; variability; indoor air quality; health risks; monitoring; indices

1. Introduction

Radon is recognized as the second, after smoking, most important risk factor for lung cancer [1–3]. It is also being investigated for further health risks [4–10]. It is a radioactive noble gas originating from the underground radioactive decay of heavier radionuclides, and reaching the surface of the Earth's crust through cracks, pores, leaks, and water [2]. Radon itself decays further, yielding solid radioactive short-lived daughters, which are direct sources of harmful ionizing radiation to the lungs [11]. In outdoor air, Radon is not considered a serious health risk, as it is sufficiently diluted, and people spend less time outdoors [12]. But indoors, in dwellings, workplaces, caves, mines, etc., with limited ventilation, hence, limited dilution, the concentration of Radon is higher [3] and people reside there longer, leading to appreciable effective doses of radiation (half of the effective dose from all natural sources [13]). Reference levels for Radon are set [14] to assess health risks.

The average indoor Radon concentration over longer periods has been the leading indicator of Radon risk. Lengthy measurements with passive dosimeters [15] are used to create regional Radon maps [16]. The constant monitoring of Radon concentration indoors is viable; however, with low-cost commercial continuous monitoring instruments [17], Radon concentration may exhibit significant variability [6,18–20]. Even at low average concentrations, the actual concentration at certain times or in certain spots may exceed reference levels, raising Radon risk concerns for vulnerable people (children, pupils, patients, the elderly, etc.). Constant monitoring would be ideal, but long-term Radon risk assessment requires models. Modelling the effective dose and the resulting health risk is demanding [12,18,21–24]. In locations with high Radon concentrations (usually with Uranium or granite-rich soil [3]), such models are expected to substantially improve risk assessment and management, aided by digital technologies [16,25,26].



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2. The Source of Indoor Radon and Its Concentration Variability

The earth's crust is rich in long half-life radionuclides; therefore, their decay products are continuously formed, and effectively remain, underground. But, Rn-222 (Radon), a radioactive noble gas (the heaviest of all noble gases and nine times denser than air), is invisible, tasteless, and odorless, hence totally untraceable by humans without appropriate instrumentation. It is formed underground from the decay of Ra-226 (Radium), which, in turn, is formed by the decay of U-238 (Uranium), and reaches the surface of the Earth, entering enclosed spaces (caves, mines, and buildings) through various paths. Due to its long half-life of 3.8 days, it is further diffused into the air from the soil and from waters, and then convected to other locations, thus becoming abundant. Through alpha decay, it yields several radioactive products (known as Radon daughters or Radon progeny) of various half-lives: Po-218, Bi-214, Pb-214, and Po-214. These four nuclides are solid particles with half-lives shorter than that of Radon (For Po-214, only a few minutes, making it a fast tracer of Radon) and can attach themselves to airborne particles that may stick to the surface of bronchial tissues, or form clusters and remain unattached, but still become trapped in the lungs. So, it is mainly Radon progeny that are the actual irradiators of the respiratory tract and pulmonary tissues.

Rn-220 (Thoron) is a Radon isotope formed by the alpha decay of Ra-224, which, in turn, is a decay product of Th-232 (thorium). Its very short half-life of just 56 s means that it does not reach as high concentrations as Radon. It is not diffused into the air from the soil in significant quantities, but it is exhaled by building materials. So, its concentration is considered noteworthy only near the walls, the floor, or the ceiling of a building. Nevertheless, its own short-lived progeny Po-216 and Ra-220 can reach significant concentrations and interact with the lungs after being inhaled [13] in a fashion similar to Radon progeny.

Rn-219 (Actinon), a decay product of U-235 (Actinium), has a half-life of just 3 s. So, although traceable, it is not diffused into the atmosphere from the soil or exhaled from materials in any meaningful quantity, and the concentration of its progeny is not significant. So, Radon, the most abundant among all its isotopes, is responsible for about half of the annual effective dose of radiation from natural sources [27]. A small, observable fraction comes from Thoron and its progeny. Other isotopes are not significant, for that matter.

Radon and Thoron concentrations behave differently due to their dissimilar half-lives. Tokonami et al. [13,28] showed that not only is the concentration of Thoron and its progeny unrelated to Radon, but it can be higher than that of Radon near the walls, even as much as one meter or more. So, by attributing the usual concentration measurements solely to Radon, the contribution of Radon and progeny to the health risk tends to be overestimated, and, subsequently, so does the respective contribution of Thoron and progeny. This is addressed by measuring far from the walls, or by using instruments that discriminate Thoron.

Radon enters an indoor environment from geogenic sources (soil and water), where its concentrations can be very high [3], and from anthropogenic sources (building materials, water, and gas supplies). Stratigraphy, geological, and hydrogeological factors influence Radon activity concentrations in soil and water, while the structural and plant features of a building influence the accumulation of Radon [29] indoors. Radon is soluble in cold water, so when the temperature rises, it is released from water into the air. Evidently, the concentration of Radon can highly vary throughout the day and from place to place, even room to room [19,20,23,26,30–32]. Temperature exhibits a clear correlation with Radon concentration; humidity appears also to correlate, and indications exist that some air quality parameters may also correlate with Radon concentration [26,33,34], while other air parameters, such as pressure, show some association but need further investigation [26,31]. Occupancy, usage profile, insulation, and ventilation also appear to correlate with Radon concentration [26,35]. Seasonal studies show that indoor Radon concentration increases in the winter months (decreased air temperature and natural ventilation), while it decreases in the summer months (increased air temperature and natural ventilation) [26]. Diurnal studies show Radon concentrations rise in the early morning, then decrease until an evening

or night peak occurs. Variations can be large; a variability of, for example, $\pm 50\%$ is not uncommon [26,36].

3. The Health Effects of Radon

Radon was originally considered harmful only for Uranium miners, who were proven to get lung cancer from it, and harmless for the general population, or even therapeutic, especially for pain reduction, [2,7] at various SPAs. But in the 1980s, it started to be recognized as a carcinogenic substance, one of the main causes of the most common form of cancer among men (14% [3]) and second among women (11.4% against 11.7% of breast cancer [3]), lung cancer. This form of cancer is the deadliest of all [3] and is caused primarily by smoking. For non-smokers, Radon is the first cause of lung cancer (15%–25%) [1–3,27]. As alpha radiation from Radon decay can easily be absorbed by external body tissues and other common obstacles or respiratory protective equipment [8], and Radon itself is rather stable during the very brief inhalation–exhalation cycle, it is not so much Radon itself, but rather its solid radioactive progeny, also alpha emitters, that induce carcinogenic effects. They enter the respiratory tract, and remain in suspension, either unattached (with diameters $\sim 0.5\text{--}5$ nm in size) or attached to aerosol, dust, smoke, or moisture particles. The attached daughters, at an amount depending on ambient conditions, comprise three modes: the nucleation mode with activity median diameters (AMD) between 10 and 100 nm, the accumulation mode with most of the potential alpha energy concentration (PAEC) and AMD values of 100–450 nm, and a coarse mode with an AMD > 1 μm [37].

Radon daughter particles irradiate the bronchial epithelium and secretory cells with ionizing alpha radiation, initiating carcinogenesis. These short-lived decay products are present in the air as unattached particles (with diameters $\sim 0.5\text{--}5$ nm in size) or can attach to existing particles in the atmosphere forming the so-called attached progeny. The attached progeny may consist of three modes, designated as the nucleation mode with activity median diameters (AMD) between 10 and 100 nm, the accumulation mode with AMD values of 100–450 nm, and a coarse mode with an AMD > 1 μm . Most of the potential alpha energy concentration (PAEC) is in the accumulation mode. The magnitude of the unattached fraction depends mainly on the ambient particle concentration, which depends on local conditions.

Radon, by its progeny, is responsible for about 3–14% of the diagnosed lung cancer cases, being second in place only behind smoking, but reason number one for never-smokers [38]. In addition, smokers are 25 times more at lung cancer risk from Radon than non-smokers. The risk of lung cancer increases proportionally with increasing Radon exposure [1], by about 16% per 100 Bq/m³ increase in long time average Radon concentration.

Radon has been investigated for inducing, or at least correlating with, a spectrum of further health risks, especially in high Radon concentration areas [6]. Alpha radiation from Radon progeny in the respiratory tract cannot penetrate the tissues to significant depths of the body at large, but there are other ways for Radon and its progeny to get incorporated and distributed into the body, causing other health risks.

Some studies show non-carcinogenic irritative effects of Radon on the skin, but they are inconclusive, and there is no conclusive evidence for harmful effects after treatment with Radon at SPAs, because of the big latency between irradiation and development of malignancies [7]. A correlation between high Radon concentration and high air pollution (high AQI—Air Quality Index) appears in studies of non-malignant brain tumors [8] and COVID-19 mortality (for unknown reasons) [5]. It seems that Radon exhibits correlations with other contaminants for several diseases [3,27,39], which is not surprising, given that progeny particles get attached to other particles, for example, contaminants, thus allowing possible synergies. The synergy of Radon with other causes of cancer exhibits cumulative effects [27], so its carcinogenic effect increases in the presence of other carcinogens.

Some indications connect Radon to cerebrovascular disease [9], Alzheimer's and Parkinson's disease [7], and stomach cancer [4,10]. Associations of Radon with child leukemia [7] and multiple sclerosis [1] have been reported. Due to various biases, such

findings do not prove that Radon is the cause, and further studies are imperative. No conclusive evidence correlates Radon with cardiovascular diseases [1], breast, and kidney cancer [10].

4. Indoor Radon Risk Assessment and Mitigation—Perspectives

There is no “safe” amount of Radon that can be inhaled since its effects are cumulative [1–3]. To estimate the health risk imposed by a certain concentration of Radon, the resulting equivalent dose must be estimated [37]. Radon is assumed to be in equilibrium with its progeny [1,2], yielding a total Potential Alpha Energy (PAE) and a Potential Alpha Energy Concentration (PAEC—PAE per unit volume). The Potential Alpha Energy Exposure (PAEE) is the product of the PAEC and the duration of the exposure to the radiation from the Radon and progeny mixture. The Equivalent Dose determines the biological effect of the radiation exposure [37]. This is the product of the PAEE with the Dose Coefficient (DC), which is a correlation factor between the offered PAEE and its biological impact. DC depends on various factors, including the nature of the exposed tissue [24,37,40,41].

The actual risk of carcinogenesis is estimated by combining the dosimetric and biological considerations with epidemiological data and medical surveys investigating the health effects of Radon [27,37], especially since there is significant latency between exposure to radiation and the emergence of carcinogenesis. As stated earlier, Radon affects human health in more than one way and degree of severity. Therefore, its synergies with other contaminants and possible cumulative effects make Radon assessment and management a high priority for civil engineering and medical authorities. A first coarse assessment can be done by setting reference levels for the long-term average Radon concentration. International organizations (WHO, etc.) issue recommendations for such Radon reference levels [1,2]. If these reference levels are exceeded, a series of remedial actions is justified. As expected, the reference levels are evaluated in accordance with relevant ongoing scientific research. So, there is a widely accepted reference level of the long-term concentration average at 300 Bq/m^3 (Upper Reference Level—URL [37]), which should initiate a series of remedial measures. Many states have adopted this limit and base long-term strategies and programs for the mitigation of Radon on them. An optional warning value of 100 Bq/m^3 (148 Bq/m^3 in the USA) is also favorable among various organizations [2,37]. At this value, simpler remedial measures are advised to reduce the Radon concentration.

Traditional Radon mitigation techniques are the ventilation of indoor spaces and insulation of floors and walls [2]. Filtration [42] by personal respiratory protective equipment (RPE), such as face masks [11], is shown to be very effective, also by HEPA filters in air purifiers, coatings, covering layers, and dedicated devices [42,43]. In addition, smart monitoring, modelling and the use of indices expand our options in developing Radon mitigation strategies and programs. Mancini et al. [29] investigated the use of indices (GRI—soil, I_{RP112Rn} —building materials, and C_w —water) to support the assessment of Radon hazard, and proposed their improvement. Lopes et al. [25] proposed a new index (IRREI—Indoor Radon Risk Exposure Indicator) for better management and perception of Radon risk.

Our understanding of Radon risk continues to develop, along with new technologies and materials to assist in the mitigation of Radon hazards. Strategies and solutions are not regulated, but ongoing investigations may lead to vital improvements and a harmonized global methodology. Research could focus on (a) modelling Radon risk in finer spatial and temporal detail to facilitate targeted solutions and interventions; (b) integrating Radon with other air quality indicators to provide a unified approach to indoor air quality; (c) investigating new materials, methods, technologies (for example, IoT—Internet of Things, machine learning, AI), and devices to mitigate Radon; (d) converge with the research on other carcinogens, as Radon appears to form synergies; (e) develop educational programs to enhance the perception of the Radon risk, especially by young people and workers.

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