

Biological Impact of Radon Gas on Juvenile Cells and Children

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Received: 10.06.2025 / Accepted: 16.07.2025 / Published: 17.07.2025

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DOI: [10.5281/zenodo.16017798](https://doi.org/10.5281/zenodo.16017798)

Abstract

Review Article

Radon gas (Rn-222) is a naturally occurring radioactive noble gas produced by the decay of uranium and thorium in the earth's crust. It is the leading contributor to natural background radiation exposure indoors. Due to their developing tissues and higher mitotic rates, children and juvenile cells are particularly susceptible to radon-induced damage. This article reviews the biological mechanisms of radon toxicity, its impact on juvenile cells and children, epidemiological findings, and public health considerations.

Keywords: Radon Gas, Rn-222, Radioactive Gas, Uranium Decay, Thorium Decay, Background Radiation, Juvenile Cells, Children, Radon Toxicity, Public Health, Epidemiology.

Citation: Murad, A. K., Alajeely, A. A. A., Kadhim, Q. S., Ali, A. L., & Salman, E. F. (2025). Biological impact of radon gas on juvenile cells and children. *GAS Journal of Clinical Medicine and Medical Research*, 2(6), 24-26, ISSN: 3049-1568.

1. INTRODUCTION

1.1 Radiation: Definition and Classification

Radiation is the emission and propagation of energy in the form of waves or particles through space or a medium. It can be broadly categorized into ionizing and non-ionizing radiation based on the energy carried and its ability to ionize atoms or molecules. Ionizing radiation carries sufficient energy to remove electrons from atoms, creating ions and causing molecular damage. It includes alpha particles, beta particles, gamma rays, X-rays, and neutrons [1,2].

Non-ionizing radiation, such as ultraviolet light (lower energies), visible light, infrared radiation, microwaves, and radio waves, does not have enough energy to ionize atoms but can induce thermal or photochemical effects [1]. The distinction between these two types is critical due to the potential for DNA damage and biological harm associated primarily with ionizing radiation.

Humans are exposed to radiation from natural sources, including cosmic rays and terrestrial radionuclides like uranium and thorium decay products (notably radon gas), as well as artificial sources such as medical imaging and industrial applications [3]. The interaction of ionizing radiation with

biological tissues depends on radiation type, energy, and linear energy transfer (LET). Alpha particles from radon progeny are high LET radiation, causing dense ionization and significant biological damage [4].

Children's developing cells are more radiosensitive due to rapid division and developmental processes, increasing the biological impact of radiation exposure, particularly from radon [5,6].

1.2 Physical Characteristics and Sources of Radiation

Alpha particles, emitted during radon decay, are heavy and highly ionizing but have low penetration power, stopped by skin or paper; however, inhaled radon progeny pose serious internal risks [7]. Beta particles are lighter, penetrate more, while gamma rays and X-rays have deeper tissue penetration but lower ionization density [8].

Radon gas originates from uranium decay in soil and rocks, accumulating indoors where ventilation is poor. It attaches to aerosols and dust, which when inhaled deposit radioactive particles in the lungs, causing alpha radiation exposure to lung tissue. Radon is the second leading cause of lung cancer worldwide, after smoking [9].



2. BIOLOGICAL MECHANISMS OF RADON TOXICITY

Radon gas itself is chemically inert, but its radioactive progeny, including polonium-218 and polonium-214, emit alpha particles during decay. These alpha particles have high LET, producing dense ionization tracks that induce significant DNA damage, including double-strand breaks, base modifications, and chromosomal aberrations [10,11].

Inhaled radon progeny deposit in the respiratory tract, where alpha radiation induces DNA damage both directly and indirectly through reactive oxygen species (ROS) generated by water radiolysis. This damage triggers cellular responses such as activation of DNA repair pathways, cell cycle arrest, or apoptosis. Failure to repair can result in mutations and carcinogenesis [12].

Due to high LET, even low doses of radon progeny can produce substantial biological effects, especially in lung stem and progenitor cells critical for tissue regeneration [13].

3. SUSCEPTIBILITY OF JUVENILE CELLS AND CHILDREN

Children's cells are especially vulnerable to radiation damage due to:

- **High mitotic rates:** Rapidly dividing cells increase the likelihood of replication of damaged DNA [14].
- **Developing DNA repair systems:** Immature repair mechanisms may be less efficient or error-prone [15].
- **Organ development:** Critical periods of organogenesis make structural and functional defects more likely [16].
- **Respiratory system differences:** Smaller airway size and different airflow lead to deeper deposition of radon progeny and higher doses to sensitive lung tissues [17].

These factors collectively heighten children's susceptibility to radon's adverse effects, including increased lifetime cancer risk [18].

4. EPIDEMIOLOGICAL EVIDENCE

Multiple epidemiological studies confirm residential radon exposure is linked to increased lung cancer risk in adults. Combined analyses indicate a linear dose-response relationship with no safe threshold [19]. Although pediatric-specific data are limited due to long latency periods and lower lung cancer incidence in children, mechanistic studies and dosimetric models suggest children bear higher relative risks due to biological susceptibility and longer expected lifespan [20,21].

5. HEALTH EFFECTS BEYOND CANCER

Emerging research suggests radon exposure may also impact immune function and exacerbate respiratory diseases like asthma in children, though evidence remains preliminary and requires further investigation [22].

6. PREVENTION AND MITIGATION

Mitigating radon exposure involves testing indoor air levels, improving ventilation, sealing foundation cracks, and installing radon reduction systems, especially in homes and schools to protect vulnerable populations [23].

7. CONCLUSION

Radon gas presents a significant natural radiation hazard, particularly to children whose juvenile cells exhibit enhanced radiosensitivity. Understanding radon's biological impacts supports public health efforts to reduce exposure and protect children's health.

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