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Pathogenesis of Changes Developing in the Human Body Under the Effect of Ionizing Radiation

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Abstract: Ionizing radiation is a powerful physical factor capable of inducing complex biological changes in the human body. Depending on the dose, duration, and type of exposure, ionizing radiation may lead to acute tissue damage as well as long-term pathological consequences. The primary target of radiation is the cellular genome, where direct DNA damage and indirect effects mediated by reactive oxygen species initiate a cascade of molecular and cellular disturbances. These early events contribute to oxidative stress, mitochondrial dysfunction, inflammatory responses, and disruption of immune regulation. As a result, structural and functional damage develops at the tissue and organ levels, particularly in radiosensitive systems such as the hematopoietic and gastrointestinal systems. Long-term exposure is associated with genomic instability, impaired tissue regeneration, and an increased risk of malignant transformation. Understanding the pathogenetic mechanisms underlying radiation-induced changes is essential for improving prevention strategies, early diagnosis, and the development of effective radioprotective and therapeutic approaches.

Keywords: ionizing radiation, pathogenesis, DNA damage, oxidative stress, radiation injury.

Introduction

Relevance of the Topic: Ionizing radiation is widely used in diagnostic radiology, radiation therapy, nuclear energy, and industry. At the same time, epidemiological data indicate a persistent increase in radiation-associated health risks worldwide. The World Health Organization and UNSCEAR reports emphasize that understanding pathogenetic mechanisms is crucial for risk assessment and prevention of radiation-related diseases[1].

Purpose of the Study: To analyze modern scientific data on the pathogenetic mechanisms of changes developing in the human body under the influence of ionizing radiation.

Materials and Methods: A narrative review of scientific publications from 2020 to 2025 was conducted using PubMed, Scopus, Web of Science, and official reports of international organizations. Experimental, clinical, and epidemiological studies focusing on molecular and systemic radiation effects were analyzed[2] (Figure 1).

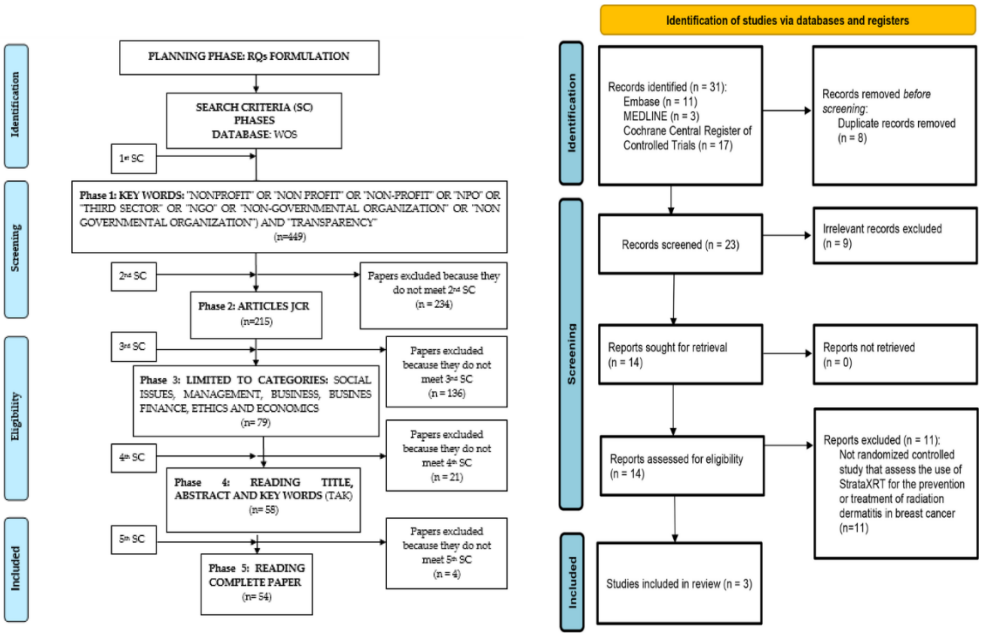

Figure 1. Schematic representation of the process of selecting and analyzing radiobiological studies.

These figures show the stages of selection, filtering, and analysis of scientific articles. Experimental, clinical, and epidemiological studies are evaluated together in the study of radiation pathogenesis[3].

Pathogenesis of Radiation-Induced Changes:

1. Primary Physical and Chemical Effects. The initial stage of radiation injury begins with the absorption of ionizing energy by tissues. This causes direct ionization of biomolecules and radiolysis of intracellular water, leading to the formation of reactive oxygen species (ROS). Hydroxyl radicals and hydrogen peroxide are considered the main mediators of early cellular damage (Figure 2)[4].

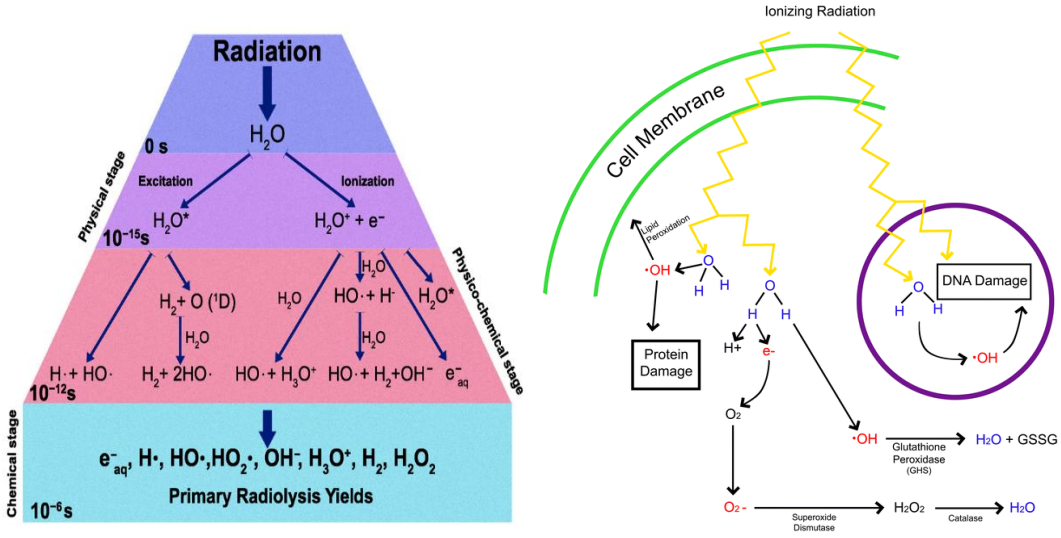

Figure 2. Radiolysis of water molecules and the formation of free radicals under the influence of ionizing radiation.

Ionizing radiation induces double-strand breaks in DNA, which are particularly dangerous. Improper repair leads to genomic instability and mutations.

2. DNA Damage and Genomic Instability. DNA is the primary biological target of ionizing radiation. The most severe lesions include double-strand breaks, which are strongly associated with mutagenesis and chromosomal aberrations. Recent molecular studies demonstrate that incorrect DNA repair results in persistent genomic instability and plays a key role in radiation-induced carcinogenesis[5] (Figure 3).

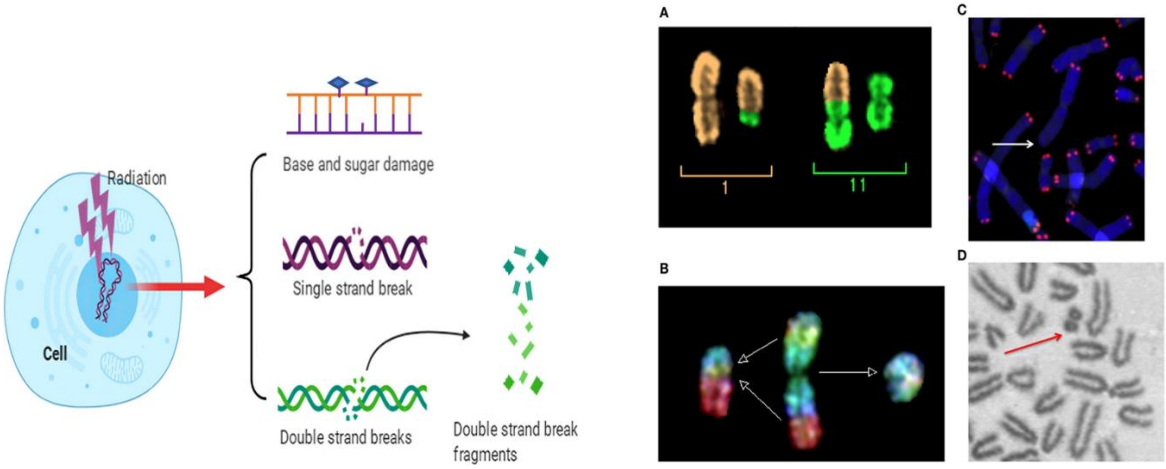

Figure 3. DNA double-strand breaks and chromosomal aberrations.

Ionizing radiation induces double-strand breaks in DNA, which are particularly dangerous. Improper repair leads to genomic instability and mutations[6].

3. Oxidative Stress and Mitochondrial Dysfunction. Radiation-induced mitochondrial damage leads to sustained overproduction of ROS, forming a self-perpetuating cycle of oxidative stress. This mechanism contributes to lipid peroxidation, protein oxidation, and activation of apoptotic pathways [7](Figure 4).

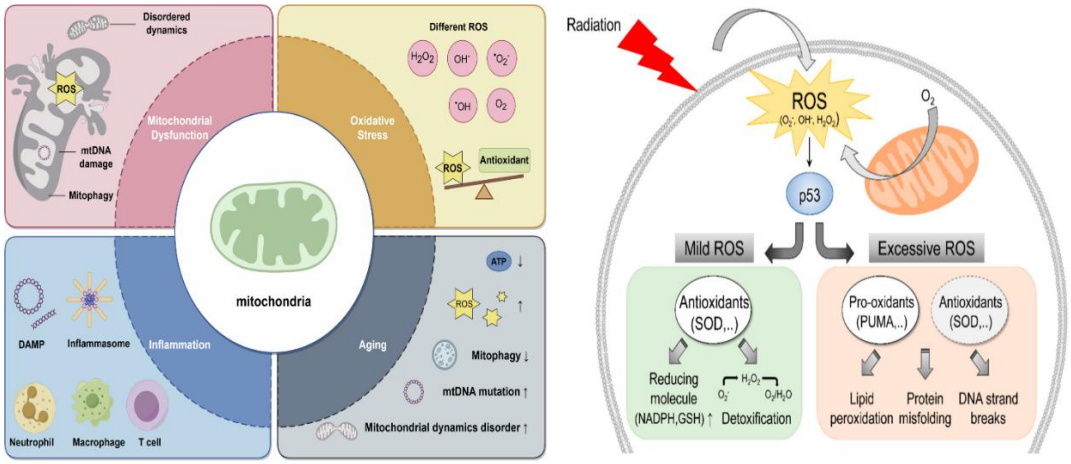

Figure 4. Mitochondrial damage and excessive ROS production.

Radiation damages mitochondrial membranes and increases ROS production. This process creates persistent oxidative stress within the cell[8].

4. Inflammatory and Immune Responses. Ionizing radiation activates inflammatory signaling pathways, including NF-κB and pro-inflammatory cytokines such as IL-6 and TNF-α. Acute exposure often results in immunosuppression, whereas chronic low-dose exposure may cause immune dysregulation and autoimmune reactions[9] (Figure 5).

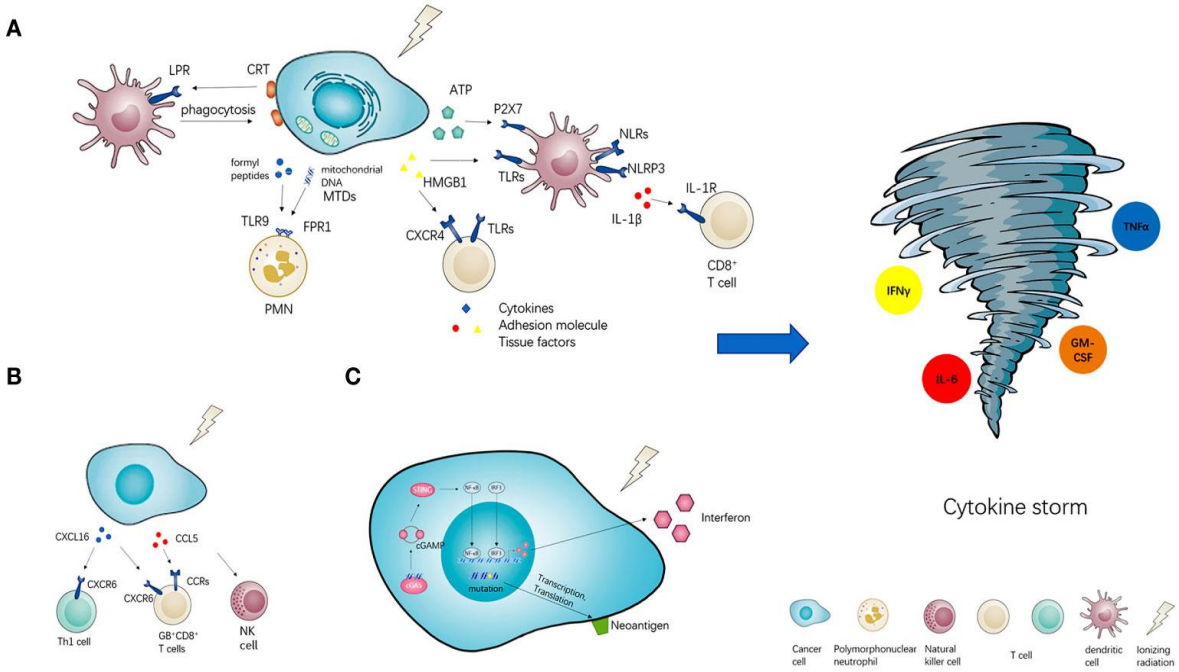

Figure 5. Radiation-induced activation of inflammatory cytokines and the NF-κB pathway.

Ionizing radiation activates inflammatory mediators. This can lead to suppression or inappropriate activation of the immune system.

5. Tissue and Organ-Level Changes. Highly radiosensitive tissues include bone marrow, gastrointestinal epithelium, skin, and gonads. Damage to stem cell populations leads to hematopoietic failure, gastrointestinal syndrome, and impaired tissue regeneration. Late radiation effects are characterized by fibrosis, vascular sclerosis, and progressive organ dysfunction[10] (Figure 6).

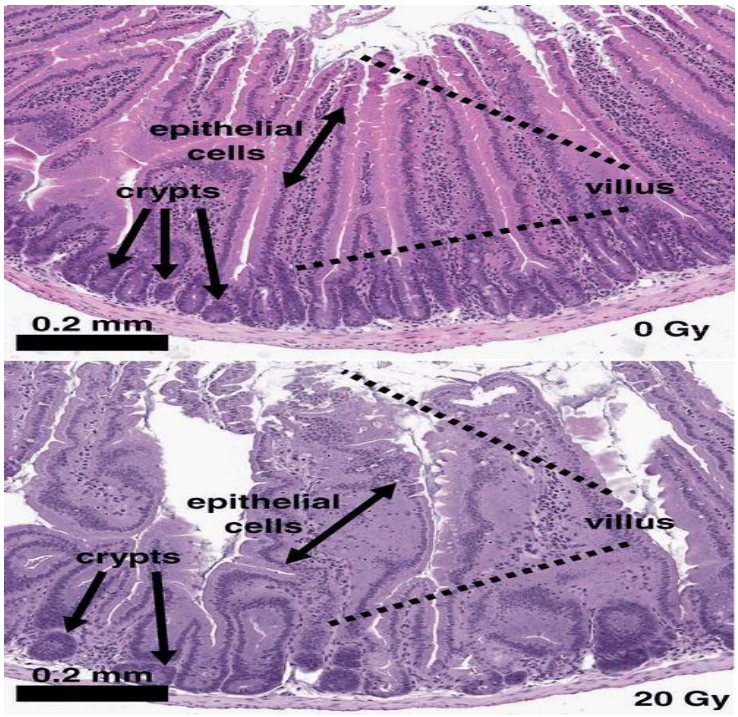

Figure 6. Radiation damage to the intestinal epithelium.

The most sensitive tissues to radiation are those with rapidly dividing cells. The loss of stem cells leads to organ failure.

6. Long-Term Effects and Carcinogenesis. Long-term epidemiological studies confirm an increased incidence of leukemia and solid tumors following radiation exposure. Recent research highlights the importance of epigenetic alterations, radiation-induced bystander effects, and chronic inflammation in delayed carcinogenesis[11] (Figure 7).

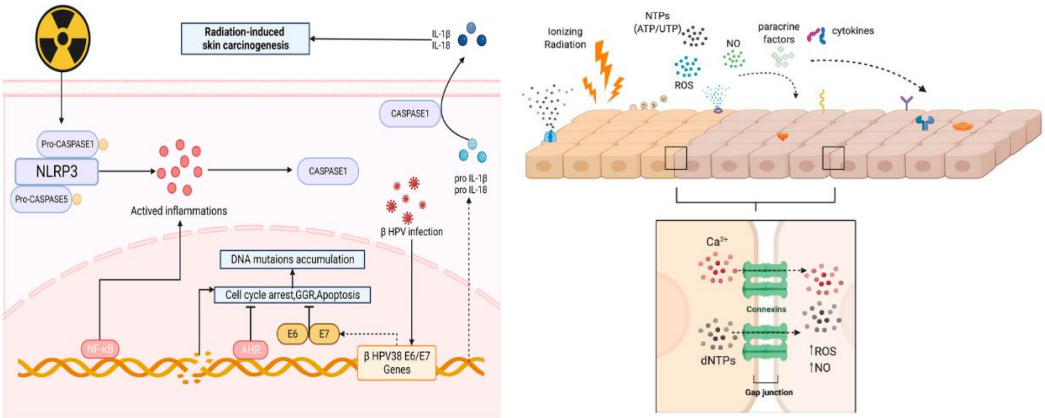

Figure 7. Mechanisms of carcinogenesis and bystander effect under radiation.

Radiation directly affects damaged cells, but also neighboring cells. This causes the development of delayed tumor processes.

Results of Recent Studies

Experimental models published between 2021 and 2024 demonstrate that even low-dose radiation exposure can induce long-lasting molecular and epigenetic changes[12]. Clinical studies report that biomarkers of oxidative stress and DNA damage correlate with disease severity and prognosis in irradiated individuals. Novel radioprotective agents and antioxidant therapies have shown promising protective effects in preclinical trials [13] (Figure 8).

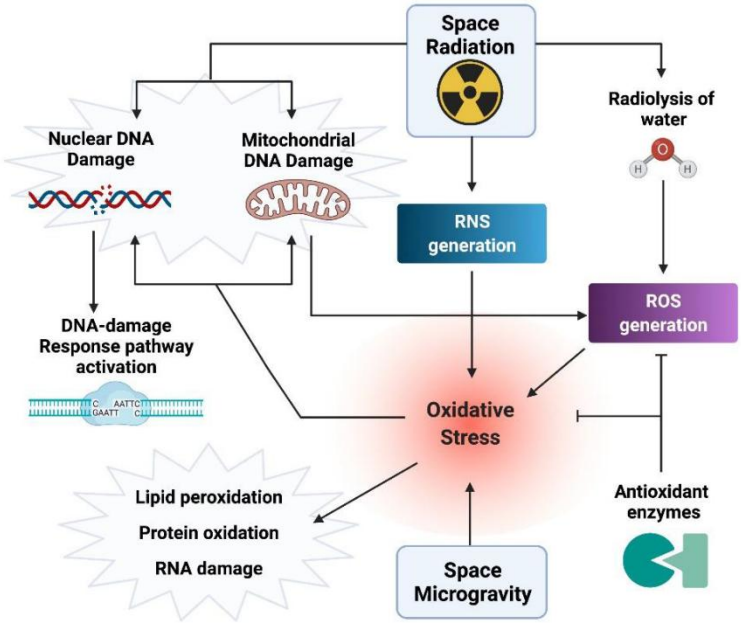

Figure 8. Biomarker changes in radiation exposure and the effects of radioprotectors[14].

Modern studies show that biomarkers of oxidative stress and DNA damage are associated with clinical conditions. The effectiveness of radioprotective agents is being evaluated in experimental models[15].

Conclusion

The pathogenesis of ionizing radiation involves a complex transition from initial atomic ionization to persistent systemic alterations. Modern evidence (2020–2025) reveals that the clinical outcome of exposure is determined not only by direct DNA damage but also by genomic instability, the bystander effect, and chronic oxidative stress.These mechanisms drive the progression from acute cellular injury to long-term consequences such as fibrosis and malignancy. A deeper understanding of these pathways is vital for developing targeted radioprotective strategies and improving safety protocols in both medical and industrial settings. Consequently, future research should focus on molecular biomarkers to better predict and mitigate late radiation effects.

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