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Impact of radiation on humans

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ABSTRACT

Humans are continuously exposed to radiation from natural sources (notably indoor radon), medical procedures, occupational settings, and occasional nuclear accidents. In this review we discuss the mechanisms of ionizing radiation injury and summarize major clinical syndromes across organ systems. Ionizing radiation deposits energy that directly damages DNA or indirectly generates hydroxyl radicals, producing single- and double-strand breaks and oxidative base lesions; higher linear energy transfer tends to create complex damage that is harder to repair. These lesions activate apoptosis, mitotic catastrophe, and inflammatory cell-death pathways, with tissue responses shaped by dose, irradiated volume, fractionation, oxygenation, and cell-cycle sensitivity. At whole-body doses of roughly 1–8 Gy, hematopoietic acute radiation syndrome (ARS) causes early lymphopenia followed by neutropenia and thrombocytopenia, increasing risks of infection and bleeding. At approximately 6–15 Gy, gastrointestinal ARS results from intestinal crypt stem-cell loss, leading to severe diarrhea, barrier failure, sepsis, and malnutrition; at very high doses, neurovascular collapse and cerebral edema can be fatal. The skin is also a critical target: cutaneous radiation injury has a threshold near 2 Gy and progresses from erythema to desquamation and ulceration with late fibrosis, and pain may be opioid-resistant. Thoracic exposure can cause pneumonitis progressing to fibrosis, while central nervous system (CNS) injury includes acute encephalopathy and delayed radionecrosis/cognitive impairment. Practical preparedness includes early triage (time-to-emesis, serial lymphocyte counts, biodosimetry when available) and prevention through justification, optimization, dose limitation, and radon mitigation.

Keywords: Radiation; ionizing, acute radiation syndrome, DNA damage, radiation injuries, radiobiology.

INTRODUCTION

Radioactive atomic nuclei release energy into their surroundings to reach a more stable structure; this energy is known as radiation**.** In daily life, we are exposed to radiation in many areas without even realizing it. Natural radiation refers to radiation that occurs spontaneously in nature and is encountered by all living beings. Cosmic rays, radon gas, gamma emissions, or internal radiation that occurs when radioactive substances enter the body through inhalation or digestion can be given as examples of natural sources. The most important source of natural radiation is radon gas. It is found abundantly in rocks and soil within the Earth's crust. By leaking into indoor environments through gaps or cracks in building foundations, it can pose risks to human health.[1,2]

Radiation is broadly classified into two main groups: ionizing and non-ionizing radiation. Ionizing radiation produces ions by removing electrons from atoms or molecules. It is divided into two categories: particles (alpha particles, beta particles, neutrons) and electromagnetic waves (X-rays, gamma rays). Non-ionizing radiation does not cause ion formation in the atoms or molecules it interacts with. Because its energy is lower than that of ionizing radiation, its harmful effects are more limited. Visible light, ultraviolet, infrared radiation, radio frequencies, and microwaves are examples of this group.[3,4]

During nuclear decay, alpha (α) particles, beta (β) particles, and frequently gamma (γ) rays are released. Ionizing radiation refers to nuclear radiation that carries sufficient energy to remove electrons from atoms or molecules, resulting in their ionization. Ionizing radiation spans high-energy electromagnetic waves—most notably gamma rays and X-rays—and it also includes the higher-energy end of ultraviolet light. Ionization can also be triggered by particulate radiation such as alpha and beta particles, as well as by neutrons. When primary cosmic rays strike molecules in Earth’s atmosphere, they create cascades of secondary particles, including muons, mesons, and positrons.[1,4] These interactions also lead to the formation of naturally occurring radionuclides (for example, carbon-14), which later release ionizing radiation as they undergo radioactive decay.[5]

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The harmful health effects of ionizing radiation were recognized shortly after the discovery of X-rays in 1895. For instance, hair loss (epilation) was reported in 1896, and skin burns were soon described. Moreover, studies conducted in 1928 demonstrated that X-rays induced germline mutations in fruit flies.[6]

On August 6, 1945, humanity encountered nuclear weapons for the first time when an atomic bomb was dropped on Hiroshima, Japan; a second bomb was dropped on Nagasaki three days later. These explosions generated powerful shock waves, extreme heat, and the release of ionizing radiation. In addition to injuries caused directly by blast pressure and thermal effects, the intense radiation exposure led to acute dose-dependent effects, including deaths resulting from gastrointestinal and bone marrow damage, as well as non-fatal manifestations such as epilation. Among the survivors, the long-term effects of radiation resulted in various health problems in the years that followed.[7]

The Fukushima Daiichi Nuclear Power Plant accident resulted in low-dose internal or external radiation exposure for approximately two million residents of the surrounding region. In contrast, the Chernobyl Nuclear Reactor accident released nearly ten times more radioactive material than Fukushima, dispersing across many European countries. Millions of children in Ukraine, Belarus, and Russia were exposed to the isotopes Cs-137 and I-131. These radioisotopes caused moderate radiation accumulation, particularly in thyroid tissue, with doses exceeding 100 mSv in a subset of affected children. Over the past 25 years, approximately 7,000 cases of thyroid cancer (mostly papillary type) have been reported, and 20 children have died due to metastatic disease.[8,9]

This review aims to provide a comprehensive overview of the biological mechanisms, organ-specific effects, clinical manifestations, and current management of ionizing radiation injury. The literature search was conducted in PubMed, Scopus, and Web of Science, primarily focusing on studies published between 2020 and 2026, while also including relevant earlier landmark studies published since 2005.

ABSORBED DOSE AND EFFECTIVE DOSE

Absorbed dose, expressed in gray (Gy), represents the amount of ionizing radiation energy deposited per unit mass of tissue. Effective dose, expressed in sievert (Sv), accounts for both the type of radiation and the differing sensitivities of exposed organs and tissues, and is mainly used to estimate the overall long-term stochastic health risk, such as cancer.[10]

RADIATION EFFECT AT THE CELLULAR LEVEL

Cell death caused by radiation is commonly divided into two main categories: interphase death and reproductive death. Interphase death describes cells that stop functioning normally after irradiation and die within a few hours rather than continuing to divide. It is thought to result from severe damage to intracellular components after exposure to high radiation doses, along with the activation of nucleases and proteolytic enzymes that cause chromatin breakdown and release of histones. These events are followed by nuclear condensation and destruction, rapid loss of normal nuclear structure, and eventual cell death. In addition, radiation-related membrane damage and impaired cellular energy metabolism are also important contributors to interphase death.[11,12]

The majority of cells die through proliferative death. This form of death usually occurs via mitotic catastrophe, a process triggered by the buildup of chromosomal damage and incorrect repair of DNA double-strand breaks caused by radiation. After completing one or more rounds of cell division, the affected cells eventually lose their capacity to keep proliferating and then die.[12,13] It is widely accepted that cellular responses, including cell death, are influenced not only by the radiation dose but also by the specific type of cell and its stage in the cell cycle (Table 1).

Table 1. Radiation-related cell-death patterns

Radiation-related trigger	Cell-death pattern
Receptor signaling or intracellular stress (DNA/ER stress)	Apoptosis
Severe structural or bioenergetic damage	Necrosis
Widespread subcellular damage and need for turnover	Autophagy (excessive)
Lipid peroxidation overload	Ferroptosis
Inflammasome-driven inflammatory signaling	Pyroptosis
Danger signals and antigen exposure	Immunogenic cell death
Checkpoint, chromosome, or mitotic apparatus damage	Mitotic catastrophe

DNA: Deoxyribonucleic acid, ER: Endoplasmic reticulum

Recent advances show that radiation responses extend beyond direct DNA damage. Ionizing radiation activates the DNA damage response, particularly the ataxia-telangiectasia mutated (ATM) and ATM- and Rad3-related pathways, which regulate DNA repair, cell-cycle arrest, apoptosis and radiosensitivity.[14,15] Unrepaired deoxyribonucleic acid fragments and micronuclei can activate the cyclic GMP–AMP synthase–stimulator of interferon genes pathway, inducing type I interferons, dendritic-cell activation and tumor-specific T-cell responses.[16,17] Radiation may also induce immunogenic cell death through the release of damage-associated molecular patterns, which promote antigen presentation and antitumor immunity.[18] Alternatively, irradiated cells may become senescent and develop a senescence-associated secretory phenotype, contributing to inflammation and tissue injury.[19] Signals released from irradiated cells can damage neighboring non-irradiated cells, producing the bystander effect, whereas systemic immune activation may cause regression of distant, non-irradiated tumors, known as the abscopal effect.[20]

Various types of DNA damage can occur during radiation therapy. The most harmful are double-strand breaks and oxidative base damage. These lesions kill cancer cells and underlie the therapeutic effect of radiotherapy.[3] The fundamental feature of ionizing radiation is that it induces DNA breaks, directly or indirectly, thereby driving the cell toward apoptosis and cell death.[21] Radiation exerts biological effects on the DNA molecule by acting directly on structures that carry genetic information or on components essential for cell survival.[22,23]

The biological impact also depends on radiation quality. High-linear-energy-transfer (LET) radiation (e.g., alpha

particles, some neutrons, and heavy ions) tends to produce clustered, complex DNA damage that is harder to repair, while oxygenation increases fixation of radiation damage (oxygen effect) and can modify radiosensitivity.[3]

Indirect DNA breaks occur when low-energy radiation enters the cell and strikes a water molecule, resulting in the formation of hydroxyl radicals (free radicals derived from water).[24] If these radicals are close to DNA, they can cause a break in one strand of the helix. Depending on the extent of the damage to the helix, the cell may repair this damage or undergo apoptosis.[25]

Radiation primarily affects multiple systems—mainly the hematologic, gastrointestinal, and neurovascular systems—and it can also significantly affect the skin.[26] As illustrated in Figure 1, ionizing radiation causes direct and reactive oxygen species (ROS)-mediated DNA damage, triggering cellular repair or death pathways that lead to tissue and organ injury and ultimately result in acute and chronic clinical manifestations.

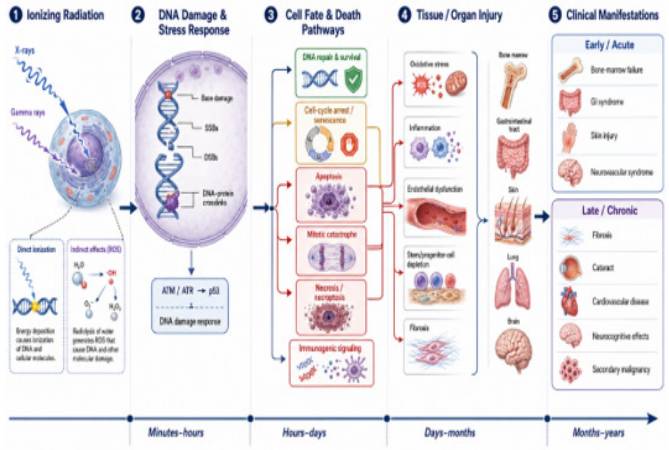


Figure 1. Molecular, cellular, tissue, and clinical consequences of ionizing radiation exposure
DNA: Deoxyribonucleic acid, ATM: Ataxia-telangiectasia mutated

In suspected ARS, early triage is aided by time-to-emesis, serial absolute lymphocyte counts, and, where available, biodosimetry methods such as dicentric chromosome assays or other cytogenetic/biomarker approaches to estimate whole-body dose and guide escalation of supportive care.[27] ARS classically progresses through four phases. The prodromal phase develops within minutes to days after exposure and commonly includes nausea, vomiting, fatigue, and diarrhea. This is followed by a latent phase, during which symptoms temporarily improve despite ongoing cellular and tissue injury. The manifest illness phase subsequently appears with clinical findings determined by the radiation dose and the predominantly affected organ system, particularly the hematopoietic, gastrointestinal, or neurovascular system. Finally, patients enter either the recovery phase, which may last weeks to months, or experience progressive organ failure and death after severe exposure.[26,27]

In mass-casualty scenarios, identifying individuals exposed to ionizing radiation and stratifying them into treatment-relevant groups is a major challenge for medical response teams. For example, individuals with minimal exposure (<2 Gy) may not require emergency care. Those exposed to moderate (2–6 Gy) or high (6–10 Gy) doses are likely to benefit from timely treatment. However, individuals exposed to supralethal doses (>10 Gy), for whom survival is not achievable, should receive only palliative care. Defining these subcategories is crucial for conserving the limited resources expected to be available in any mass-casualty situation.[28]

ORGAN-SPECIFIC EFFECTS OF IONIZING RADIATION

Cutaneous Radiation Injury

Cutaneous radiation injury (CRI) is a deterministic effect that occurs in the skin and subcutaneous tissues after radiation exposure. The skin is radiosensitive due to both rapidly renewing cells (basal keratinocytes) and microvascular structures. The threshold dose is 2 Gy, and the severity increases with higher doses.[29] Cutaneous radiation injury typically results from radioactive fallout, radiotherapy, or medical radiological procedures. The Hiroshima, Nagasaki, and Chernobyl incidents are historical examples of this type of injury.[9,30] The cutaneous manifestations that develop according to increasing severity are shown sequentially in Figure 2. Very high localized doses can cause necrosis/radionecrosis and chronic, non-healing wounds.[31]

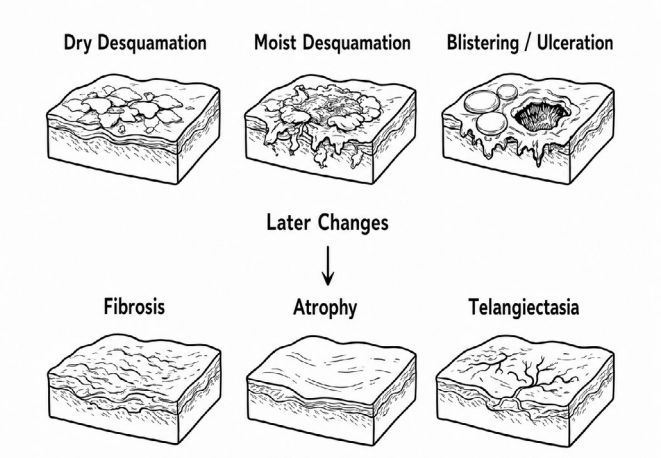


Figure 2. Skin manifestations of cutaneous radiation injury

Radiation-induced skin pain is often resistant to opioid therapy. For diagnosis, a detailed medical history, physical examination, complete blood count, ultrasound, and thermography are useful. Although there is no specific approved treatment, management requires a multidisciplinary approach. First-line treatment includes pain control, infection prevention, and psychological support. Topical corticosteroids and a combination of pentoxifylline and α-tocopherol may be beneficial.[32]

When necessary, surgical interventions such as excision, flap coverage, or amputation may be performed. Hyperbaric oxygen therapy has shown promising results. In addition, mesenchymal stem cell (MSC) applications contribute to healing through anti-inflammatory and regenerative effects.[33]

Effects of Radiation on the Respiratory System

Ionizing radiation to the chest (most often during thoracic radiotherapy, but also after high-dose accidental exposure) can injure the respiratory system through direct cellular damage and an amplified inflammatory cascade. The major toxicity is radiation-induced lung injury, which typically evolves from an early inflammatory phase (radiation pneumonitis) to a later fibrotic remodeling phase (radiation-

induced pulmonary fibrosis).[34] Mechanistically, injury to alveolar epithelium and pulmonary microvascular endothelium promotes capillary leak, disturbed surfactant, and cytokine signaling (including pro-inflammatory and pro-fibrotic mediators such as IL-1, IL-6, TNF-α, and TGF-β), leading to edema and impaired gas exchange.[35] Clinically, pneumonitis often appears weeks to months after exposure with dry cough, dyspnea, low-grade fever, and radiographic ground-glass opacities or consolidation; untreated or severe cases can progress to chronic fibrosis with reduced lung compliance and diffusion capacity.[36] Risk increases with larger irradiated lung volumes and higher dose-volume metrics, as well as patient factors and concurrent systemic therapies. Management focuses on ruling out infection or tumor progression, providing supportive care, and using corticosteroids for symptomatic pneumonitis; prevention relies on modern planning techniques and adherence to lung dose constraints.[37] In extreme exposures, acute respiratory distress and pleural effusions may occur, requiring oxygen and close monitoring.

Effects of Radiation on the Nervous System

Until recently, the nervous system was thought to be relatively insensitive to radiation. However, more recent research has shown that the central nervous system (CNS) can react even to low levels of radiation, and these effects often lead to tissue injury.[38] The harmful effects of radiation on the CNS are particularly important in the contexts of cancer radiotherapy and space research.[39] The brain and spinal cord are late-responding tissues, and low-dose irradiation of large volumes may cause long-term complications.[40] Acute effects include cerebral edema, encephalopathy, and seizures, whereas delayed effects comprise demyelination, white matter loss, and cognitive impairment.[41] In addition, pseudoprogression and brainstem syndrome may develop after radiotherapy.[42] Within minutes to hours, manifestations may include severe headache, dizziness, altered consciousness, ataxia, seizures, and agitation; coma may develop later. Prominent early nausea/vomiting, profound fatigue, and hypotension can accompany the picture. Prognosis is generally poor, with high mortality at very high whole-body doses despite supportive care.[26]

Among late complications, the most important is radionecrosis, which arises from glial and endothelial injury and disruption of the blood–brain barrier.[40] Very high doses can cause endothelial injury, capillary leak, cerebral edema, microvascular failure, and a severe inflammatory response.[43] Secondary CNS tumors such as meningiomas, gliomas, and sarcomas are more frequently observed in individuals exposed to radiation.[44] NASA has identified cancer, CNS performance loss, and ARS as critical risks for astronauts. For long-duration spaceflight, exposure to galactic cosmic rays and sporadic solar particle events involves a mixed field that includes high-energy heavy ions; these components have higher LET and may pose distinct carcinogenic and neurocognitive risks compared with terrestrial low-LET exposures, motivating mission-specific risk modeling.[45]

Hematopoietic Syndrome

Because the hematopoietic system contains rapidly dividing stem and progenitor cells, it is among the most radiosensitive targets. After whole-body exposure to radiation doses of approximately 1–8 Gy, hematopoietic ARS may develop following a latent phase. DNA damage and apoptosis in bone marrow lead to an early drop in lymphocytes (first days), followed by neutropenia and thrombocytopenia (days to weeks).[46]

As a result of radiation-induced damage, bone marrow failure develops and blood cells can no longer be produced. This may lead to aplastic anemia, myelodysplastic syndrome, erythrocyte aplasia, megakaryocytic thrombocytopenia, and chronic neutropenia.[47] Consequently, hematopoietic organs (bone marrow, thymus, spleen) are suppressed, and serious, life-threatening conditions such as anemia, leukemia, secondary infections, and immunodeficiency may occur.[48]

Effects of Radiation on the Gastrointestinal System

Gastrointestinal ARS is characterized by intestinal dysfunction that develops after exposure to high radiation doses of 6–15 Gy. GI injury usually appears at higher doses than the hematopoietic syndrome and is closely related to loss of intestinal crypt stem cells. Shortly after exposure, nausea, vomiting, and diarrhea occur; following a latent phase, bloody diarrhea, sepsis, and risk of death increase. Hematopoietic syndrome often coexists and worsens the prognosis. With modern treatment, the survival rate is around 50%.[48] Radiotherapy damages not only cancer cells but also rapidly dividing tissues such as the intestines.[49] Salivary gland damage can lead to hyposalivation, mucositis, and functional impairment.[50]

High-dose radiation causes complications such as ulcers, malnutrition, and diarrhea.[51] Fractionated treatments, protection of radiosensitive regions, and the use of hydrogels are recommended for radioprotection.[52]

Finally, radiation induces apoptosis, ferroptosis, and inflammation in intestinal cells. Inhibition of these processes and supplementation with tetrahydrobiopterin (BH4) can reduce intestinal ischemia and radiation enteritis.[53] The major organ-specific effects of radiation, including typical dose thresholds, clinical manifestations, diagnostic approaches, and management strategies, are summarized in **Table 2**.

Effects of Radiation on Other Systems: Cardiovascular, Endocrine, and Reproductive Effects

Ionizing radiation can also cause clinically important cardiovascular, endocrine, and reproductive toxicity. Cardiac and vascular exposure promotes endothelial injury, inflammation, oxidative stress, and fibrosis, increasing the long-term risks of coronary artery disease, cardiomyopathy, valvular dysfunction, and pericardial disease.[54] Endocrine effects are generally dose- and site-dependent; irradiation of the hypothalamic–pituitary region may cause growth hormone, thyroid, adrenal, or gonadotropin deficiencies, whereas direct thyroid exposure can result in hypothyroidism.[55] Reproductive tissues are particularly radiosensitive: testicular irradiation may impair spermatogenesis and reduce testosterone production, while ovarian exposure can deplete the follicular reserve, leading to premature ovarian insufficiency, infertility, and early menopause.[56,57] Therefore, long-term cardiovascular, hormonal, and reproductive surveillance should be considered in exposed individuals.

Table 2. Radiation effects, summary

	Cutaneous radiation injury	Effects of radiation on the respiratory system	Effects of radiation on the nervous system	Hematopoietic syndrome	Effects of radiation on the gastrointestinal system
Typical dose/ threshold	Threshold dose>2 Gy Severity increases with higher dose	Risk rises with larger irradiated lung volumes and higher dose-volume metrics	Low-dose large-volume exposure may cause long-term complications Very high whole-body doses cause acute neurovascular picture	Hematopoietic syndrome typically at 1–8 Gy whole-body exposure	GI-ARS typically at 6–15 Gy (often with hematopoietic syndrome)
Clinical features	Dry desquamation → moist desquamation Blistering/ulceration, late fibrosis/atrophy/ telangiectasia	Dry cough, dyspnea, low-grade fever Imaging: ground-glass or consolidation Late: reduced compliance and diffusion	Headache, dizziness, altered consciousness, ataxia, seizures Delayed: demyelination, white matter loss, cognitive impairment, radionecrosis	Early lymphopenia (days) followed by neutropenia and thrombocytopenia (days–weeks) anemia, infections, bleeding	Early nausea/vomiting/ diarrhea, later bloody diarrhea, sepsis, malnutrition, ulcers and radiation enteritis
Diagnosis	Detailed history+physical exam Ultrasound and thermography may help	Differentiate from infection/tumor progression	Neurologic exam+imaging monitor for pseudoprogression and brainstem syndrome after radiotherapy	Serial CBC with differential; consider marrow evaluation if needed	Clinical course+laboratory examination for hydration/ electrolytes Evaluate for sepsis Imaging/endoscopy as indicated
Management	Pain control, infection prevention, psychological support Topical steroids Pentoxifylline+alpha-tocopherol	Supportive care Corticosteroids for symptomatic pneumonitis Prevention via modern planning and lung dose constraints	Supportive care Manage delayed complications (radionecrosis/ cognitive decline) Spaceflight adds high-LET particle risk considerations	Supportive care, antimicrobials as needed, transfusions and hematopoietic growth-factor support when indicated Manage marrow failure complications	Supportive care and radioprotection strategies (fractionation, sparing, hydrogels) Target ROS/RNS-driven injury Inhibition of apoptosis/ ferroptosis and BH4 supplementation mentioned

GI-ARS: Gastrointestinal acute radiation syndrome, CBC: Complete blood count, LET: Linear-energy-transfer, GI: Gastrointestinal, ROS: Reactive oxygen species, RNS: Reactive nitrogen species, BH4: Tetrahydrobiopterin

RADIATION CARCINOGENESIS

Radiation carcinogenesis is a major long-term stochastic effect of ionizing radiation; its probability increases with dose, whereas cancer severity is not dose-dependent. Radiation-induced DNA damage, genomic instability, and mutations may initiate malignancy after a latency ranging from several years to decades.[58] Leukemia generally appears earlier than most solid tumors and shows a clear dose–response relationship.[59] The thyroid is especially radiosensitive during childhood, as demonstrated by the increased incidence of thyroid cancer after exposure to radioactive iodine following the Chernobyl accident.[60] Breast cancer risk is also elevated after irradiation, particularly among females exposed at a young age,[61] while lung cancer risk is influenced by both radiation dose and smoking.[62] In addition, radiotherapy may produce secondary malignancies within or near the irradiated field, including lung, esophageal, thyroid, bone, and soft-tissue cancers, often after a prolonged latency.[63]

The health consequences of low-level radiation exposure remain unclear, largely because existing studies lack sufficient statistical strength. As a result, the linear no-threshold (LNT) model continues to serve more as a cautious framework than a fully validated biological principle. Future research should focus on developing dependable molecular biomarkers to assess radiation exposure. Integrative approaches such as multi-omics combined with machine learning could accelerate and personalize biodosimetry. Although new radioprotective compounds show potential, current findings are predominantly derived from animal experiments. To bring these innovations into clinical practice, large-scale, multicenter studies in humans are essential.[64,65]

CONCLUSION

As a result, this review highlights that radiation exposure is not limited to rare disasters; people encounter it routinely through natural sources such as cosmic rays and especially indoor radon that can enter buildings through cracks and gaps, creating a preventable public-health risk. It also emphasizes why ionizing radiation matters biologically: by damaging cellular components—most critically DNA—it can trigger cell death pathways and, depending on dose and tissue sensitivity, lead to clinically important outcomes. Historical events (e.g., Hiroshima/Nagasaki) and large-scale nuclear accidents illustrate both the acute deterministic injuries seen at high doses and the longer-term health consequences that may appear years later, reinforcing the importance of risk communication and preparedness.

Clinicians must be aware of both the short-term and long-term health consequences of radiation exposure to enable timely diagnosis, effective treatment, and ongoing patient monitoring. To reduce unnecessary risks, strict compliance with radiation protection principles—such as justification of use, dose optimization, and proper shielding—remains vital. Robust emergency preparedness requires coordinated multidisciplinary protocols, rapid dose evaluation, efficient triage systems, and the presence of skilled personnel with adequate medical resources.

ETHICAL DECLARATIONS

Peer Review Process

This review was externally peer-reviewed.

Conflict of Interest

The author declare no conflicts of interest.

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Author Contributions

The author is solely responsible for the conception, data collection, analysis, and writing of this manuscript.

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