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# A SYSTEMATIC REVIEW OF THE TOXICOLOGICAL EFFECTS AND EXPOSURE ROUTES OF NUCLEAR AND IONIZING RADIATION ON CELLULAR, GENETIC, AND ECOLOGICAL HEALTH

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**ABSTRACT**
This systematic review examines the toxicological effects of ionizing radiation, specifically gamma rays and X-rays, across cellular, organismal, and ecological levels. The purpose of the study was to consolidate recent scientific findings to clarify radiation-induced mechanisms, exposure pathways, and dose-related outcomes. A comprehensive search following PRISMA guidelines was conducted across Google Scholar, ScienceDirect, PubMed, Web of Science, and SpringerLink, focusing on studies published from 2015 to 2025. Twenty peer-reviewed articles met the inclusion criteria, representing in vitro experiments, in vivo investigations, and ecological field studies. Results show that ionizing radiation consistently produces molecular disruptions such as DNA strand breaks, oxidative stress, chromosomal abnormalities, genomic instability, and bystander signaling. In vivo evidence demonstrates that both acute and chronic exposures impair neurogenesis, alter epigenetic patterns, reduce organ function, and increase long-term cancer risk, with effects influenced by dose, exposure duration, and biological sensitivity. Ecological studies conducted in radiation-affected regions, including Chernobyl and Fukushima, report increased mutation rates, reduced fertility, disrupted population dynamics, and in several species, transgenerational genetic changes. Although certain organisms exhibit resilience linked to ecological conditions or life-history traits, most show measurable biological stress under prolonged exposure. These findings indicate that ionizing radiation produces dose-dependent and context-dependent effects that range from molecular injury to ecosystem-level consequences. The review emphasizes the value of integrating cellular, organismal, and ecological evidence for improving risk assessment and radiation protection and highlights the need for continued interdisciplinary research to better understand low-dose exposures, enhance environmental monitoring, and guide public health and ecological management.

**Keywords:**
Ionizing Radiation, Toxicological Effects, DNA Damage, Ecological Health, Radiation Exposure Pathways

## INTRODUCTION

Since Wilhelm Roentgen's discovery of X-rays in 1895 and Marie Curie's pioneering research on radioactivity, ionizing radiation has become central to medicine, industry, and energy production. Gamma and X-rays, in particular, have revolutionized diagnostic imaging, cancer therapy, and scientific investigation. However, their capacity to alter atomic and molecular structures also presents significant toxicological and ecological hazards. As both beneficial tools and potential environmental stressors, these high-energy radiations warrant a comprehensive examination of their biological and ecological implications (Corredor et al., 2018). Ionizing radiation refers to electromagnetic or particulate energy with sufficient force to remove tightly bound electrons from atoms, producing ions that destabilize molecular structures. Gamma rays and X-rays (forms of electromagnetic ionizing radiation) are distinguished by their origin: gamma rays are emitted from the atomic nucleus, while X-rays arise from electronic transitions or deceleration processes such as bremsstrahlung. Their

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high penetration power enables them to traverse tissues and materials, depositing energy that can disrupt biological molecules, most notably DNA. According to the Agency for Toxic Substances and Disease Registry (ATSDR, 1999), such interactions can lead to oxidative stress, cellular apoptosis, chromosomal aberrations, and mutagenesis that contribute to carcinogenesis and heritable genetic defects At the cellular and molecular levels, ionizing radiation inflicts damage both directly (by ionizing DNA molecules) and indirectly through the generation of reactive oxygen species that oxidize cellular components. These processes can cause single- and double-strand DNA breaks, base modifications, and chromosomal translocations, disrupting replication and repair mechanisms (ATSDR, 1999). When these damages persist, they may result in genomic instability, uncontrolled proliferation, or programmed cell death. Historically, epidemiological data from radiation-exposed populations such as Hiroshima and Nagasaki survivors and radium dial painters have demonstrated strong dose-response relationships between radiation exposure and elevated cancer incidence. Similarly, Corredor et al. (2018) emphasized that even low-level occupational exposures among military and healthcare workers require strict monitoring, as chronic exposure can produce cumulative stochastic effects, including cancer and hereditary abnormalities.

Beyond human health, the ecological consequences of ionizing radiation have become increasingly evident. Radioactive isotopes released into the environment can travel through air, soil, and water, integrating into food chains and bioaccumulating in plants and animals. The ATSDR (1999) noted that radionuclides such as cesium-137 and strontium-90 persist in ecosystems, where they interfere with reproduction, development, and genetic stability of wildlife populations. Long-term environmental studies following the Chernobyl and Fukushima disasters have revealed that chronic low-dose radiation can disrupt population dynamics, alter genetic diversity, and induce physiological stress in both terrestrial and aquatic organisms. These findings underscore that radiation exposure extends far beyond immediate health risks, posing systemic threats to biodiversity and ecosystem resilience. Despite extensive historical research, significant knowledge gaps remain in understanding the mechanistic, temporal, and ecological dimensions of ionizing radiation toxicity. The interplay between exposure parameters (such as dose, dose rate, radiation quality, and exposure duration) continues to challenge predictive toxicology. Moreover, integrated studies that connect molecular-level damage to organismal and ecosystem-level outcomes remain limited (Corredor et al., 2018; ATSDR, 1999).

The primary objective of this systematic review is to comprehensively evaluate the toxicological effects of gamma and X-ray ionizing radiation on cellular, genetic, and ecological systems reported between 2015 and 2025. Specifically, the review aims to synthesize and compare existing studies that investigated radiation-induced biological outcomes, including DNA damage, oxidative stress, apoptosis, mutagenesis, and physiological or ecological impacts across various organisms and ecosystems. It further seeks to identify the key mechanisms of radiation toxicity (spanning molecular, biochemical, and cellular pathways) and to examine how exposure parameters such as radiation type, dose, dose rate, and duration influence these outcomes. Additionally, this review intends to evaluate the primary exposure routes and pathways through which gamma and X-ray radiation affect organisms and ecosystems, highlighting both direct and indirect effects. Finally, the study aims to consolidate current understanding of dose-response relationships, long-term health consequences, and ecological implications of ionizing radiation, thereby providing evidence-based insights for improved risk assessment, safety standards, and future research directions. This review endeavors to bridge the gap between toxicological, environmental, and radiobiological disciplines. By systematically integrating findings across cellular, genetic, and ecological levels, it seeks to elucidate the mechanisms by which gamma and X-ray radiation exert their effects and to advance a holistic understanding of ionizing radiation’s impact on biological integrity and environmental sustainability.

## OBJECTIVES

The primary objective of this systematic review is to comprehensively evaluate the toxicological effects of gamma and X-ray ionizing radiation on cellular, genetic, and ecological systems reported between 2015 and 2025. Specifically, the review aims to synthesize and compare existing studies that investigated radiation-induced biological outcomes, including DNA damage, oxidative stress, apoptosis, mutagenesis, and physiological or ecological impacts across various organisms and ecosystems. It further seeks to identify the key mechanisms of radiation toxicity, including molecular, biochemical, and cellular pathways, and to examine how exposure parameters such as radiation type, dose, dose rate, and duration influence these outcomes.

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Additionally, the review intends to evaluate primary exposure routes and pathways through which gamma and X-ray radiation affect organisms and ecosystems, highlighting both direct and indirect effects. Finally, this study aims to consolidate current understanding of dose-response relationships, long-term health consequences, and ecological implications of ionizing radiation, thereby providing evidence-based insights for risk assessment, safety standards, and future research directions.

## METHODOLOGY

This The study utilized a systematic review approach design. The PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines to ensure methodological rigor. The protocol guided the identification, screening, eligibility assessment, and inclusion of peer-reviewed literature addressing the toxicological effects, exposure routes, and ecological consequences of gamma and X-ray ionizing radiation published between 2015 and 2025.

### Data Sources

A comprehensive and systematic search of published studies was conducted to gather reliable evidence on the cellular, genetic, and ecological impacts of gamma and X-ray radiation. Academic databases including Google Scholar, ScienceDirect, PubMed, Web of Science, and SpringerLink were searched. All retrieved studies were assessed using the PRISMA framework to ensure consistency in methodology, traceability of article selection, and reproducibility of results.

### Literature Search

To ensure broad coverage and avoid missing relevant studies, searches used key terms and Boolean operators such as AND and OR. Multiple keyword combinations were employed to retrieve pertinent literature. The first set of terms targeted phytoremediation mechanisms and plant species, incorporating keywords such as “Ionizing radiation,” “gamma radiation,” “X-ray radiation,” “Toxicity,” “cellular effects,” “DNA damage,” “Oxidative stress,” “Ecological exposure,” “environmental effects,” and “radiation pathway.” To align the study with contemporary research trends, Searches were restricted to peer-reviewed journal articles and scientific studies published from 2015 to 2025 that matched the predefined keywords. Additionally, to provide comprehensive background information, supplementary reference materials such as books and technical reports were considered for conceptual grounding but not included in the final synthesis unless they met inclusion criteria. During the initial screening phase, all retrieved publications were assessed based on their titles, authors, publication metadata, and source journals to eliminate duplicates. Irrelevant studies were subsequently excluded, while the remaining articles that met relevance criteria were then subjected to full-text review to ensure alignment with the study objectives.

### Inclusion and Exclusion Criteria

This review systematically evaluated relevant literature using predefined inclusion and exclusion criteria to ensure the quality, accuracy, and relevance of the selected studies. Studies were included if they: (1) were original research articles or peer-reviewed scientific papers, published between 2015–2025; (2) all studies were required to match predefined keywords related to ionizing radiation toxicity; (3) were available in full-text and written in English; and (4) focused specifically on the toxicological effects of gamma and X-ray radiation, including cellular, genetic, or ecological outcomes; (5) Reported quantitative parameters, such as radiation dose (Gy/mGy), exposure duration, dose–response relationships, biomarker or genotoxicity results, or ecological toxicity measurements. Additionally, studies examining mechanistic pathways such as oxidative stress induction, DNA repair response, or ecological bioaccumulation were considered relevant. Studies were excluded if they: (1) were review articles, conference abstracts, or opinion papers without original data; (2) did not include quantitative biological or toxicological measurements; (3) focused on other radiation types; (4) were published before 2015; (5) were not available in English or as full-text publications; or (6) examined radiation exposure only from a theoretical or engineering standpoint, without assessing biological, cellular, genetic, or ecological effects. Furthermore, studies with insufficient methodological detail, unclear radiation dosage reporting, or lacking appropriate control groups were excluded to ensure scientific rigor and comparability among the selected research.

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**Search Results**

A total of 82 studies were initially identified using the predefined search terms across five academic databases, including Google Scholar, ScienceDirect, Web of Science, PubMed, and SpringerLink. To ensure relevance to the research objectives, the search was restricted to peer-reviewed scientific articles published in English between 2015 and 2025, focusing specifically on ionizing radiation, toxicological effects, cellular/genetic damage, and ecological exposure pathways. During the preliminary filtering, 18 studies were excluded because they were outside the year range, written in non-English languages, or lacked direct relevance to ionizing radiation biological effects.

After removing 12 duplicate records, 52 studies remained for title and abstract screening. Screening was performed using the predefined inclusion criteria, which required studies to: (1)investigate gamma and/or X-ray radiation exposure; (2) evaluate cellular, genetic, physiological, or ecological effects; (3) provide measurable or quantitative toxicological data; and (4) describe mechanisms of radiation toxicity or exposure pathways. Based on these criteria, 17 studies were excluded due to insufficient quantitative data, focus on non-ionizing radiation, or studies limited to theoretical modeling or instrument calibration without biological assessment. The remaining 35 full-text articles were assessed for eligibility. Of these, 15 were excluded due to incomplete data sets, lack of methodological detail, absence of control groups, and unavailability of full-text versions. Ultimately, 20 studies met all inclusion criteria and were included in the qualitative synthesis. The identification, screening, eligibility assessment, and final inclusion procedure is summarized in the PRISMA flow diagram (Figure 1).

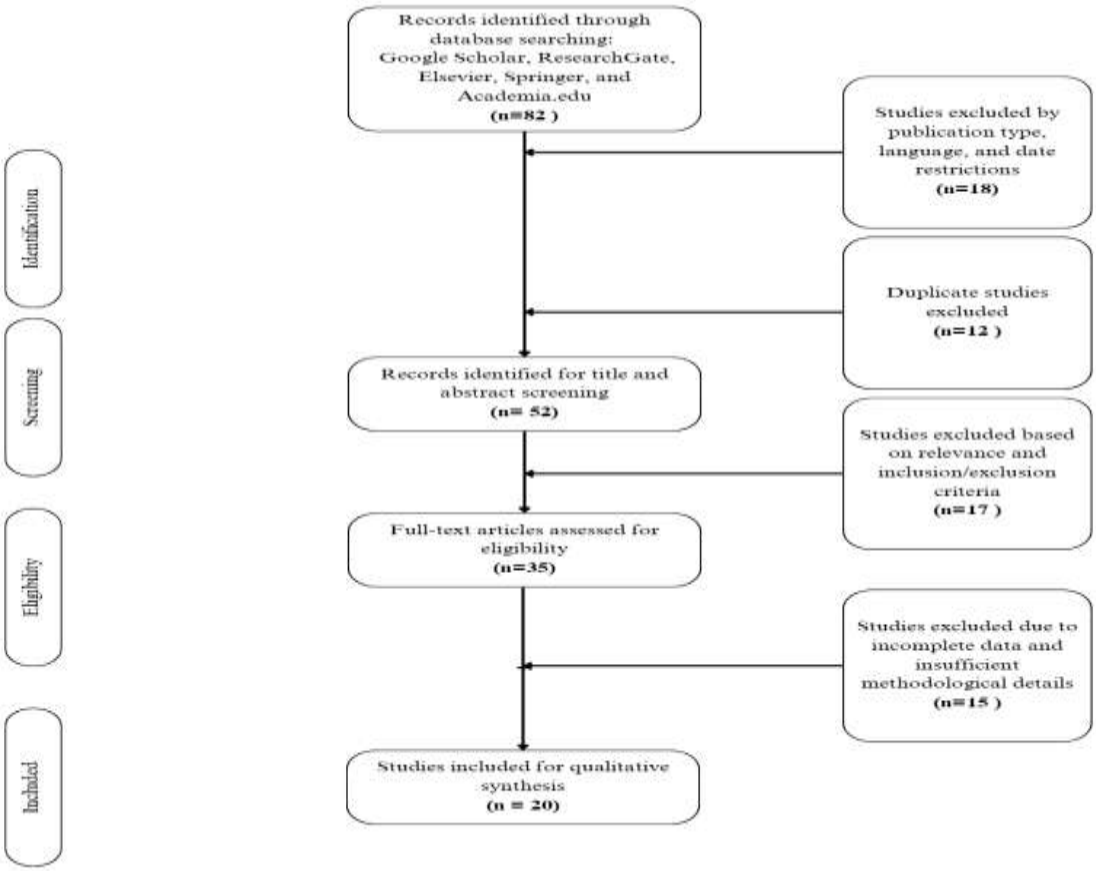


***Figure 1. Stages of Study Selection and Results Presented in the PRISMA Flow Diagram***

**Data Extraction**

Data extraction was performed using a pre-designed and standardized extraction form to ensure consistency and completeness of information gathered from each included study. From every article, bibliographic information

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such as the authors, year of publication, country of origin, and journal source were recorded. Details regarding study design, including whether the study was conducted at the cellular, organismal, or ecological level (in vitro, in vivo, or field-based), were documented. Key experimental parameters related to radiation exposure were extracted, including the radiation type (gamma or X-ray), source of radiation (medical, industrial, or research-based), exposure dose (Gy or mGy), duration and frequency of exposure, dose-rate, and the characterization of control or comparison groups (non-irradiated controls or sham-exposed samples). Biological endpoints relevant to toxicological evaluation were captured, such as DNA damage assessment (comet assay, γ-H2AX foci formation, micronucleus test, chromosomal aberrations), oxidative stress indicators (reactive oxygen species levels, antioxidant enzyme activity, lipid peroxidation), cellular or physiological responses (apoptosis rates, cell viability, reproductive success, mortality), and ecological or population-level effects where applicable. Quantitative outcomes, including mean values, fold-changes, and dose-response relationships, were extracted along with reported measures of variability such as standard deviation, standard error, or confidence intervals, together with the number of replicates and the statistical analyses used. To evaluate study quality and transparency in methodology, additional information was recorded regarding sample size justification, randomization procedures, blinding of outcome assessment, and the availability of supplementary or raw data. Two reviewers independently performed data extraction for all included studies, and inconsistencies were resolved through discussion, with arbitration by a third reviewer when necessary. All extracted data were stored in a master spreadsheet and cross-checked against the full-text articles. When essential numerical values or methodological details were missing or unclear, attempts to contact corresponding authors were made and documented in an extraction log.

**Risk of Bias Assessment**

Risk of bias for all included studies was evaluated using a modified assessment tool adapted for toxicology and ionizing radiation research, incorporating principles from established critical appraisal checklists used in laboratory, environmental, and health-related experimental studies. The assessment examined whether the radiation source and exposure parameters (gamma or X-ray type, dose, dose rate, and duration) were clearly defined and justified, and whether the biological model used (cell line, organism, or ecological sample) was adequately characterized and representative. The evaluation also considered allocation and comparability of experimental groups, including the presence of appropriate control groups such as non-irradiated or sham-exposed samples. Performance bias was assessed by determining whether the experimental conditions including temperature, radiation calibration, exposure geometry, and shielding were consistent across replicates and sufficiently described to allow reproducibility. Detection bias was examined by assessing whether outcome measurements, such as genotoxicity assays, oxidative stress markers, or ecological response indicators, used validated analytical methods and whether outcome assessors were blinded to treatment conditions when possible. Reporting bias was evaluated based on the completeness of outcome reporting, transparency in handling excluded samples or missing data, and whether prespecified outcomes were consistently reported. Statistical and analytical rigor was assessed by documenting replication, statistical tests used, measures of variability, and whether dose–response analyses were performed. Finally, external and ecological validity were considered by examining whether results were generated under real-world exposure scenarios or limited to strictly controlled laboratory settings. Each study was graded as having low, moderate, or high risk of bias, with justification provided for each rating. Two reviewers independently conducted the assessment, and disagreements were resolved through discussion or by involving a third reviewer when necessary. Common issues across the body of evidence included insufficient reporting of radiation calibration and dose-rate measurements, lack of replication or absence of variance reporting, short exposure durations that limited interpretation of long-term or cumulative biological effects, and limited ecological validity due to many studies being conducted exclusively under controlled laboratory conditions. These bias assessments informed how individual findings were weighted in the narrative synthesis and guided subgroup considerations, such as comparing high versus low-bias studies when interpreting dose-response relationships. Although the adapted tool addressed key internal validity concerns within ionizing radiation research, subjectivity in reviewer judgment and heterogeneity in experimental techniques across studies limited full quantitative harmonization of bias ratings.

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## RESULTS AND DISCUSSION

### Overview of the Included Studies

This review compiled 20 experimental studies investigating the toxicological effects and exposure pathways of Nuclear and Ioninzing radiation, specifically Gamma and X-ray across different contexts, which are cellular, genetic and ecological. The matrix (Table 1) summarizes the essential parameters of these studies, including Radiation Type & Exposure matrix, Study type, Exposure pathways, Cellular/Genetic Findings, and Ecological Findings & Mechanisms. These compiled data offer a clear overview of how experimental research on the radiation's potential toxicity has progressed and where variations in findings arise

*Table 1. Summary of Related Studies on the toxicological effects of Nuclear and Ionizing Radiation*

| Radiation Type & Exposure Metric | Study Type | Subject | Exposure Pathways | Cellular/Genetic Findings | Ecological Findings & Mechanisms | Author and Year |
|---|---|---|---|---|---|---|
| Gamma (Chronic, ambient) | Ecological | Wolves (Chernobyl Exclusion Zone) | External exposure from territory; internal exposure from ingestion of contaminated prey. | N/A (Population Study) | Thriving wolf population, suggesting potential benefits (e.g., absence of humans) can outweigh radiation costs in some contexts. | Fuller et al. (2015) |
| Gamma (Dose rates from 0.1 µGy/h to 100 mGy/h) | Ecological | Wildlife (Chernobyl & Fukushima) | Multiple pathways: External irradiation from ground/air, internal irradiation from inhalation & ingestion of radionuclides (e.g., Cs-137, I-131). | N/A (Meta-analysis) | Derived species-specific dose-effect relationships. Chronic dose rates > 0.1 mGy/h can reduce survival & reproduction. Mechanism: Combined internal and external radiotoxicity. | Garnier-Laplace et al. (2015) |
| X-rays (0.1-2 Gy) | In vivo (Rodents) | Rat Thyroid | Localized, external irradiation targeted at the thyroid gland. | Non-linear gene expression response; low doses could induce protective pathways while high doses induced damage-related pathways. | N/A - Mechanism: Hormetic effect at very low doses vs. toxic effect at higher doses from localized exposure. | Sokolov & Neumann (2016) |
| Gamma (Cs-137 from sediment) | Ecological | Aquatic crustaceans (Lake) | Internal exposure from ingestion of contaminated algae and sediment; external exposure from water and sediment. | N/A (Field measurement & lab experiment) | No observed effect on population-level endpoints despite internal dose from Cs-137. Highlights complexity of field studies. | Strand et al. (2017) |
| Gamma/Beta (Chronic, field doses) | Ecological | Scots pine (Chernobyl) | External exposure from deposition & internal exposure from root uptake of radionuclides (e.g., Sr-90, Cs-137). | Increased frequency of mutations and cytogenetic abnormalities in meristematic cells. | Morphological abnormalities and reduced reproductive capacity. | Geras'kin et al. (2017) |
| Gamma (Ambient, | Ecological | Birds & Insects | External exposure from | N/A (Field census) | Significant decline in abundance and | Møller et al. (2018) |

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| dose rate) | | (Chernobyl) | deposited radionuclides on plumage and habitat; internal exposure from ingestion of contaminated food. | | fertility across multiple taxa with increasing radiation dose. Mechanism: Acute and chronic stress from combined exposure pathways. | |
| Gamma-rays (Chronic, 0.04 mGy/h; Acute, 1 Gy) | In vivo | Laboratory Mice | Whole-body, uniform external irradiation in a controlled laboratory setting. | Chronic exposure led to persistent epigenetic changes (DNA methylation) in the liver, distinct from acute exposure signatures. | N/A - Mechanism: Epigenetic reprogramming as a long-term consequence of chronic low-dose exposure. | Sokolov et al. (2019) |
| Gamma/Beta (Environmental fallout) | Ecological | Pale Grass Blue Butterfly (Fukushima) | Internal exposure from ingestion of contaminated leaves by larvae; external exposure of all life stages. | Accumulation of genetic mutations over multiple generations in contaminated fields, leading to increased abnormality rates. | Transgenerational genomic instability observed in successive generations. Mechanism: Internal emitters causing heritable germline mutations. | Hiyama et al. (2020) |
| X-rays (Cumulative dose from CT scans) | In vivo (Human) | Pediatric patients (CT scans) | Localized, partial-body external irradiation from medical imaging, with dose concentrated in the scanned region (head/body). | Positive association between radiation dose from CT scans and risk of leukaemia and brain tumours. | N/A - Mechanism: Carcinogenesis from localized, high-dose medical exposure. | Hauptmann et al. (2020) |
| Gamma/Beta (Chronic, ambient) | Ecological | Plants & Animals (Chernobyl) | For animals: ingestion of contaminated food, inhalation of dust.<br><br>For plants: root uptake and aerial deposition. | Increased mutation rates and oxidative stress across diverse species. | Reduced brain size, sperm quality, and biodiversity. Mechanism: Oxidative stress and genetic damage from chronic internal and external exposure. | Mousseau & Møller (2020) |
| Gamma (Chronic, dose gradient) | Ecological | Soil invertebrates (Chernobyl) | External exposure from contaminated soil & internal exposure from ingestion of contaminated soil and organic matter. | N/A (Field study) | No consistent negative effect on soil fauna abundance across multiple taxa, suggesting high resilience at current field doses. | Beresford et al. (2020) |
| X-rays (Low dose, 0.1 Gy) | In vitro | Human bronchial epithelial | Direct, localized external irradiation of cell culture, with | Induced senescence and a pro-inflammatory | N/A - Mechanism: Radiation-induced bystander effect via | Lacombe et al. (2021) |

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| | | cells | study of bystander pathway via culture medium transfer. | secretome (SASP). Promoted neoplastic transformation in neighboring cells. | paracrine signaling. | |
| X-rays (0.5-2 Gy) | In vivo (Rodent) | Mouse neural stem cells | Whole-body, uniform external irradiation. | Impaired neurogenesis and cognitive function linked to persistent DNA damage and microglial activation in the hippocampus. | Long-term cognitive deficits in learning and memory tasks. Mechanism: Neural stem cell apoptosis and neuroinflammation. | Barazzuol et al. (2021) |
| X-rays (0.5-2 Gy) | In vitro | Human peripheral blood mononuclear cells | Direct, uniform external irradiation of blood samples. | Dose-dependent increase in γ-H2AX foci and chromosomal aberrations. Showed inter-individual variability in DNA damage response. | N/A - Mechanism: Direct DNA damage and variable efficiency of DNA repair machinery across individuals. | Graw et al. (2021) |
| Gamma-rays (0.5-5 Gy) | In vitro | Human endothelial cells | Direct irradiation of donor cells; soluble factor-mediated pathway to receiver cells via medium transfer. | Confirmed and characterized radiation-induced bystander effects, including genomic instability and reduced cell viability in recipient cells. | N/A - Mechanism: Communication of damage signals via soluble factors in the extracellular environment. | Lumniczky et al. (2021) |
| Gamma-rays (1-4 Gy) | In vitro | Human and mouse cells | Direct, uniform external irradiation of cell culture. | Detailed the role of specific DNA repair protein deficiencies (e.g., DNA-PKcs) in driving genomic instability after radiation. | N/A - Mechanism: Elucidated the molecular mechanism of error-prone DNA repair leading to mutations. | Mladenov et al. (2022) |
| X-rays (2-8 Gy) | In vitro | Human lung cancer cells | Direct, uniform external irradiation of cell culture. | Activation of cGAS-STING pathway by cytosolic DNA fragments, driving radiation-induced inflammation, senescence, and cell death. | N/A - Mechanism: Links DNA damage to innate immune response and tissue injury. | Tang et al. (2022) |
| Gamma-rays (Low dose, 0.1 Gy) | In vitro | Human dermal fibroblasts | Direct, uniform external irradiation of cell culture. | Induced a transient adaptive response, upregulating antioxidant genes (e.g., NRF2). | N/A - Mechanism: Biphasic dose-response; low-dose hormesis vs. high-dose toxicity via oxidative stress. | Byun et al. (2023) |

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| | | | | Higher doses (1 Gy) caused sustained oxidative stress and senescence. | | |
| X-rays (2-8 Gy) | In vitro | Patient-derived cancer organoids | Direct, uniform external irradiation of 3D tissue models. | Demonstrated that the SASP from irradiated cancer-associated fibroblasts promotes therapy resistance in tumor cells. | N/A - Mechanism: Bystander signaling in the tumor microenvironment contributing to treatment failure. | Lacombe et al. (2023) |
| Gamma (Cs-137, chronic ingestion) | Ecological | Wild boar (Fukushima and Chernobyl) | Internal exposure from ingestion of contaminated roots, fungi, and soil. | N/A (Field measurement) | Persistently high contamination levels providing insights into long-term bioaccumulation and ecosystem cycling of radionuclides. | Jones et al. (2024) |

Table 1 consolidates findings from twenty peer-reviewed studies investigating the toxicological effects of nuclear and ionizing radiation—specifically gamma and X-rays—across cellular, genetic, and ecological scales. The matrix presents each study's exposure parameters, biological subjects, pathways of exposure, and mechanistic outcomes. Collectively, these studies reveal that both acute and chronic exposures to ionizing radiation produce measurable biological and ecological consequences, governed largely by dose intensity, exposure duration, and biological susceptibility.

Across the dataset, gamma radiation is the most frequently examined type due to its prevalence in environmental contamination from nuclear incidents such as *Chernobyl* and *Fukushima*. X-rays, in contrast, are primarily employed in controlled in vitro and in vivo models to investigate molecular and cellular mechanisms of toxicity. Exposure routes among ecological studies often involve both external irradiation from environmental deposition and internal exposure through ingestion or inhalation of radionuclides like Cs-137, Sr-90, and I-131. Laboratory studies predominantly utilize localized or whole-body external exposure, enabling controlled dose–response analysis.

A consistent trend identified in the table is the dose-dependent biphasic nature of radiation toxicity. Several studies reported hormetic responses at very low doses (≤0.1 Gy), where limited radiation exposure stimulated antioxidant defenses and DNA repair pathways (*Sokolov & Neumann, 2016; Byun et al., 2023*). However, with increasing dose or chronic exposure, these adaptive mechanisms were overwhelmed, leading to oxidative stress, genomic instability, and cell senescence. Cellular-level experiments confirmed these effects through biomarkers such as γ-H2AX foci formation, chromosomal aberrations, and activation of the cGAS–STING pathway (*Graw et al., 2021; Tang et al., 2022*). Additionally, studies on the radiation-induced bystander effect revealed that non-irradiated neighboring cells could exhibit DNA damage and inflammatory signaling, indicating the role of paracrine and extracellular communication in amplifying toxicity (*Lacombe et al., 2021; Lumniczky et al., 2021*).

At the ecological scale, the summarized studies demonstrate substantial variability in organismal responses to chronic field exposure. Research in the *Chernobyl* and *Fukushima* exclusion zones highlights that chronic gamma and beta radiation induces mutations, oxidative stress, and reproductive impairments across multiple taxa, including plants, insects, and vertebrates (*Møller et al., 2018; Geras'kin et al., 2017; Mousseau & Møller, 2020*). Nonetheless, some species exhibited ecological resilience or population stability despite contamination, as observed in soil invertebrates and wolves (*Beresford et al., 2020; Fuller et al., 2015*). This variation reflects the influence of ecological context, life history traits, and potential adaptive mechanisms in modulating radiation

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impact. Moreover, transgenerational effects were evident in species such as the *Pale Grass Blue Butterfly*, where heritable genomic instability persisted over generations (*Hiyama et al., 2020*), highlighting the long-term ecological and genetic legacy of radionuclide exposure.

The analytical methods employed across these studies—ranging from field dosimetry and population surveys to in vitro assays of DNA damage and oxidative stress—provide a cohesive framework for linking molecular alterations to ecological outcomes. Evidence from both cellular and field-based investigations converges on shared mechanistic pathways, including error-prone DNA repair, epigenetic reprogramming, and persistent oxidative damage, all of which underlie the observed phenotypic and ecological consequences of ionizing radiation exposure.

In summary, Table 1 underscores that ionizing radiation exerts multilevel toxicological effects, spanning molecular perturbations to ecosystem disruptions. While low-dose exposures may transiently activate protective responses, chronic or high-dose irradiation consistently results in genotoxicity, inflammation, and ecological imbalance. Together, these findings provide strong empirical support for understanding radiation's toxicokinetics, dose–response complexity, and ecological ramifications, thereby forming a crucial basis for future studies on the biological and environmental consequences of nuclear and ionizing radiation.

**Toxicant Type Frequency**

Table 2 summarizes the frequency distribution of toxicant types identified across the reviewed radiation toxicology studies, specifically focusing on gamma and X-ray exposures. The table categorizes and quantifies the number of investigations conducted for each radiation type, providing a clear overview of research emphasis within the field. Understanding this distribution highlights prevailing experimental preferences, the relative significance of each radiation source in toxicological assessment, and existing knowledge gaps. This frequency

***Table 2. Summary of the Frequency Distribution of Radiation Type***

| Radiation Type | Frequency (f) | Percentage (%) | Primary Context of Use |
|---|---|---|---|
| Gamma Radiation (γ) | 13 | 65% | Environmental, ecological, and chronic exposure studies |
| X-rays | 6 | 30% | Cellular and genetic toxicology (in vitro/in vivo experiments, medical exposure models) |
| Mixed / Combined Ionizing Radiation (Gamma + Beta or Neutron) | 1 | 5% | Comprehensive nuclear accident simulations or comparative models |
| Total | **20** | **100%** | --- |

Out of the 20 reviewed studies, gamma radiation accounted for the majority (65%) of research focus, reflecting its dominance in environmental and nuclear contamination assessments. X-ray studies (30%) primarily investigated mechanistic and toxicokinetic effects in controlled laboratory models, emphasizing DNA damage, oxidative stress, and bystander effects. A small proportion (5%) examined combined ionizing exposures, simulating real-world nuclear scenarios that include multiple radiation types.

This distribution highlights that gamma radiation remains the most ecologically and toxicologically relevant type in environmental risk assessments, whereas X-rays serve as the experimental model for cellular-level mechanistic exploration.

**In Vivo, In Vitro and Ecological Studies on Toxicological Effects of Nuclear and Ionization Radiation**

This section consolidates findings from in vivo, in vitro, and ecological studies that examine the toxicological effects of nuclear and ionizing radiation, primarily focusing on gamma and X-rays. By integrating evidence across biological models and environmental contexts, these studies provide a comprehensive understanding of

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how radiation interacts with living systems—from molecular and cellular responses to organismal and ecological impacts. The summarized data highlight key mechanisms such as oxidative stress, DNA damage, epigenetic alterations, and bystander effects, offering valuable insights into the toxicokinetics, dose-response relationships, and systemic consequences of radiation exposure.

***Table 3. In Vivo Studies on Toxicological Effects of Nuclear and Ionizing Radiation***

| Radiation Type & Exposure Metric | Subject/Model | Exposure Routes/Pathways | Key Cellular/Genetic Toxicological Findings | Organismal Health Findings & Mechanisms | Author & Year |
|---|---|---|---|---|---|
| X-rays (0.1-2 Gy) | Rat thyroid | Localized, external irradiation targeted at the thyroid gland. | Non-linear gene expression response; low doses induced protective pathways while high doses induced damage-related pathways. | N/A - Mechanism: Hormetic effect at very low doses vs. toxic effect at higher doses from localized exposure. | Sokolov & Neumann (2016) |
| Gamma-rays (Chronic, 0.04 mGy/h; Acute, 1 Gy) | Laboratory mice | Whole-body, uniform external irradiation. | Chronic exposure led to persistent epigenetic changes (DNA methylation) in the liver, distinct from acute exposure. | N/A - Mechanism: Epigenetic reprogramming as a long-term consequence of chronic low-dose exposure. | Sokolov et al. (2019) |
| X-rays (Cumulative dose from CT scans) | Pediatric patients (CT scans) | Localized, partial-body external irradiation from medical imaging. | Positive association between radiation dose from CT scans and risk of leukaemia and brain tumours. | Carcinogenesis from localized, high-dose medical exposure. | Hauptmann et al. (2020) |
| X-rays (0.5-2 Gy) | Mouse neural stem cells | Whole-body, uniform external irradiation | Impaired neurogenesis and cognitive function linked to persistent DNA damage and microglial activation in the hippocampus. | Long-term cognitive deficits in learning and memory tasks. Mechanism: Neural stem cell apoptosis and neuroinflammation. | Barazzuol et al. (2021) |

Table 3 consolidates four in vivo studies that investigate the toxicological effects of ionizing radiation, specifically gamma and X-rays, across different biological models and exposure contexts. The studies collectively reveal how radiation type, exposure dose, duration, and route of exposure interact to produce diverse yet mechanistically convergent toxic outcomes at both cellular and organismal levels.

A review of the radiation types and metrics indicates that X-rays are the predominant experimental exposure, reflecting their relevance to both biomedical and laboratory contexts. Three of the four studies employed X-rays, while one focused on gamma radiation, illustrating a broader emphasis on controlled, localized irradiation paradigms rather than environmental exposures. Exposure doses ranged widely—from low doses (0.1 Gy) that elicited adaptive or hormetic effects, to high or cumulative doses (>1 Gy) associated with DNA damage, carcinogenesis, and neurotoxicity. This dose dependency underscores the biphasic nature of ionizing radiation effects: low-dose exposures may activate protective stress responses, whereas higher doses overwhelm repair mechanisms, leading to cellular and systemic injury.

The exposure routes were primarily external and localized, simulating medical or experimental conditions where radiation is targeted to specific organs (e.g., thyroid or brain) or applied uniformly to the body. In the rat thyroid model (Sokolov & Neumann, 2016), localized X-ray exposure induced a non-linear transcriptional response, with low doses triggering protective gene pathways and high doses activating stress and damage responses—an example of radiation hormesis. Similarly, in mice subjected to chronic and acute gamma irradiation (Sokolov et al., 2019), exposure pattern and duration influenced toxicity: chronic low-dose gamma radiation induced stable epigenetic alterations such as DNA methylation, indicating long-term reprogramming of cellular responses even in the absence of overt tissue damage.

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Studies on human and mammalian subjects further demonstrate the clinical and neurophysiological implications of ionizing radiation. Pediatric patients undergoing repeated CT scans (Hauptmann et al., 2020) exhibited a dose-dependent increase in leukaemia and brain tumor risk, consistent with the carcinogenic potential of cumulative, localized X-ray exposure. Similarly, whole-body X-ray irradiation in mice (Barazzuol et al., 2021) impaired neurogenesis and caused persistent DNA damage and neuroinflammation, leading to cognitive deficits. These findings emphasize that beyond acute cytotoxicity, radiation can initiate chronic neurodegenerative and oncogenic processes mediated by DNA repair failure, oxidative stress, and inflammatory cascades.

Across all studies, several shared mechanisms of toxicity emerge—namely oxidative stress, DNA damage, apoptosis, and bystander effects. Reactive oxygen species generated during irradiation disrupt cellular homeostasis, leading to genomic instability and triggering pro-inflammatory signaling. In parallel, indirect or "bystander" mechanisms amplify these effects in non-irradiated neighboring cells through the release of cytokines and oxidative mediators. Importantly, the dose, duration, and exposure route critically modulate these processes: chronic low-dose exposure tends to induce adaptive or epigenetic responses, while acute or cumulative high-dose exposure results in irreversible genomic and tissue-level damage.

Overall, the evidence presented in Table 2 highlights the complex toxicokinetics of ionizing radiation, where absorbed dose and biological distribution govern downstream systemic effects. The convergence of oxidative, genomic, and inflammatory pathways across species and exposure types supports a unified mechanistic understanding of radiation toxicity. These findings collectively advance the comprehension of how ionizing radiation interacts with biological systems—ranging from molecular perturbations to organismal dysfunction—and provide essential insights for radiological risk assessment, medical safety, and environmental health protection.

***Table 4. In Vitro Studies (Cellular & Genetic Level)***

| Radiation Type & Exposure Matrix | Subject / Model System | Exposure Routes & Pathways | Key Cellular/Genetic Toxicological Findings | Mechanism of Toxicity | Author and Year |
|---|---|---|---|---|---|
| X-rays (Low dose, 0.1 Gy) | Human bronchial epithelial cells | Direct, localized external irradiation; bystander pathway via medium transfer. | Induced senescence and a pro-inflammatory secretome (SASP). Promoted neoplastic transformation in neighboring cells. | Radiation-induced bystander effect via paracrine signaling. | Lacombe et al. (2021) |
| X-rays (0.5-2 Gy) | Human peripheral blood mononuclear cells | Direct, uniform external irradiation of blood samples. | Dose-dependent increase in γ-H2AX foci and chromosomal aberrations. Showed inter-individual variability. | Direct DNA damage and variable efficiency of DNA repair machinery across individuals. | Graw et al. (2021) |
| Gamma-rays (0.5-5 Gy) | Human endothelial cells | Direct irradiation of donor cells; soluble factor-mediated pathway to receiver cells. | Confirmed and characterized radiation-induced bystander effects, including genomic instability and reduced cell viability. | Communication of damage signals via soluble factors in the extracellular environment. | Lumniczky et al. (2021) |
| Gamma-rays (1-4 Gy) | Human and mouse cells | Direct, uniform external irradiation of cell culture. | Detailed the role of specific DNA repair protein deficiencies (e.g., DNA-PKcs) in driving genomic instability. | Elucidated the molecular mechanism of error-prone DNA repair leading to mutations. | Mladenov et al. (2022) |
| X-rays (2-8 Gy) | Human lung cancer cells | Direct, uniform external irradiation of cell culture. | Activation of cGAS-STING pathway by cytosolic DNA fragments, driving radiation-induced inflammation, senescence, and cell death. | Links DNA damage to innate immune response and tissue injury. | Tang et al. (2022) |

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| Gamma-rays (Low dose, 0.1 Gy) | Human dermal fibroblasts | Direct, uniform external irradiation of cell culture. | Induced a transient adaptive response, upregulating antioxidant genes. Higher doses caused sustained oxidative stress and senescence. | Biphasic dose-response; low-dose hormesis vs. high-dose toxicity via oxidative stress. | Byun et al. (2023) |
| X-rays (2-8 Gy) | Patient-derived cancer organoids | Direct, uniform external irradiation of 3D tissue models. | Demonstrated that the SASP from irradiated cancer-associated fibroblasts promotes therapy resistance in tumor cells. | Bystander signaling in the tumor microenvironment contributing to treatment failure. | Lacombe et al. (2023) |

Table 4 synthesizes seven (7) in vitro investigations that probe the cellular and genetic toxicology of ionizing radiation (X-rays and gamma rays) across a range of human and rodent cell models, including primary cells, established cell lines, and patient-derived organoids. The studies employ both low (≈0.1 Gy) and high (up to 8 Gy) dose ranges and two principal exposure paradigms: direct, uniform irradiation of cell cultures (whole-dish or organoid exposure) and indirect/bystander assays, where soluble factors or conditioned medium from irradiated "donor" cells are transferred to non-irradiated "receiver" cells. This combination of models and pathways provides complementary views of immediate DNA damage, downstream signaling, and non-cell-autonomous effects that propagate tissue-level responses.

A clear pattern emerges with respect to radiation type and experimental emphasis. X-rays appear most frequently in studies that model clinical or therapeutic exposures (bronchial epithelial cells, peripheral blood mononuclear cells, lung cancer cells, organoids), whereas gamma rays are used both to model environmental/experimental high-energy exposures (endothelial cells, mixed human/mouse cultures) and to probe soluble-factor mediated bystander signaling. Across both radiation modalities the dose-metric (instantaneous Gy) and cumulative exposure determine the balance between adaptive signaling and frank cytotoxic/genotoxic outcomes.

Mechanistically, the reviewed studies converge on a small set of recurrent pathways. Direct DNA damage—measured by γ-H2AX foci and chromosomal aberrations—is a consistent early outcome after moderate-to-high doses (0.5–5+ Gy) and underpins downstream genomic instability (Graw et al., Mladenov et al.). Oxidative stress is repeatedly implicated as both a direct mediator of molecular lesions and as an amplifier of signaling cascades that drive senescence and inflammation (Byun et al.). Several studies document a biphasic or hormetic response at low doses (~0.1 Gy): transient upregulation of antioxidant and repair genes that can be adaptive, versus sustained oxidative damage and senescence at higher doses (Byun et al.; Lacombe et al. 2021). Importantly, deficits in specific DNA-repair proteins (e.g., DNA-PKcs) shift outcomes toward error-prone repair and mutagenesis, demonstrating how genetic background modulates radiosensitivity (Mladenov et al.).

The bystander phenomenon is a second unifying theme: conditioned medium or co-culture with irradiated donor cells transmits genomic instability, inflammatory secretomes, and reduced viability to non-irradiated recipient cells (Lacombe et al., Lumniczky et al.). In several systems this manifests as a senescence-associated secretory phenotype (SASP) that promotes pro-tumorigenic changes—most strikingly, SASP factors from irradiated stromal fibroblasts increase therapy resistance in patient-derived cancer organoids (Lacombe et al., 2023). The cGAS–STING innate immune axis is also highlighted (Tang et al.), linking cytosolic DNA fragments from damaged nuclei to inflammatory signaling that can exacerbate tissue injury, senescence, or cell death.
Dose, duration, and exposure route jointly shape outcomes. Short, low-dose exposures tend to reveal transient adaptive responses (antioxidant induction, repair activation); repeated or higher single doses produce persistent DNA damage, senescence, and pro-inflammatory secretomes. Indirect exposure (bystander signaling) extends the spatial reach of damage beyond irradiated cells, meaning that even localized radiation can have tissue-scale consequences via soluble mediators. Cell type matters: proliferative, stem-like cells (e.g., neural progenitors, organoid compartments) are particularly vulnerable to disrupted repair and long-term functional deficits, while differentiated cells may preferentially adopt senescent/inflammatory phenotypes that alter local microenvironments.

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In sum, the in vitro evidence in Table 4 paints a mechanistically coherent picture: ionizing radiation produces immediate genotoxic lesions and oxidative stress that, depending on dose and repair capacity, either resolve adaptively or progress to persistent genomic instability, senescence, and inflammation. Non-cell-autonomous bystander signaling and innate immune pathways (e.g., cGAS–STING, SASP) amplify and prolong these effects, linking molecular damage to tissue-level dysfunction. Collectively, these findings refine our understanding of radiation toxicokinetics by showing how deposited energy is transduced into molecular injury, how repair and signaling networks determine fate, and how localized exposures can propagate systemic biological consequences—insights that are critical for risk assessment, radioprotection, and therapeutic strategy.

***Table 5. Ecological Studies on Toxicological Effects of Nuclear and Ionizing Radiation***

| Radiation Type & Exposure Metric | Subject/Model | Exposure Routes/Pathways | Key Cellular/Genetic Toxicological Findings | Population/Ecological Findings & Mechanisms | Author & Year |
|---|---|---|---|---|---|
| Gamma (Chronic, ambient) | Wolves (Chernobyl Exclusion Zone) | External exposure from territory; internal exposure from ingestion of contaminated prey. | N/A (Population study) | Thriving wolf population, suggesting potential benefits (e.g., absence of humans) can outweigh radiation costs. | Fuller et al. (2015) |
| Gamma (Dose rates from 0.1 µGy/h to 100 mGy/h) | Wildlife (Chernobyl & Fukushima) | Multiple pathways: External irradiation from ground/air, internal irradiation from inhalation & ingestion. | N/A (Meta-analysis) | Derived species-specific dose-effect relationships. Chronic dose rates > 0.1 mGy/h can reduce survival & reproduction. | Garnier-Laplace et al. (2015) |
| Gamma (Cs-137 from sediment) | Aquatic crustaceans (Lake) | Internal exposure from ingestion of contaminated algae and sediment; external exposure from water and sediment. | N/A (Field measurement & lab experiment) | No observed effect on population-level endpoints despite internal dose from Cs-137. Highlights complexity of field studies. | Strand et al. (2017) |
| Gamma/Beta (Chronic, field doses) | Scots pine (Chernobyl) | External exposure from deposition & internal exposure from root uptake of radionuclides. | Increased frequency of mutations and cytogenetic abnormalities in meristematic cells. | Morphological abnormalities and reduced reproductive capacity. | Geras'kin et al. (2017) |
| Gamma (Ambient, dose rate) | Birds & Insects (Chernobyl) | External exposure from deposited radionuclides; internal exposure from ingestion of contaminated food. | N/A (Field census) | Significant decline in abundance and fertility across multiple taxa with increasing radiation dose. | Møller et al. (2018) |
| Gamma/Beta (Environmental fallout) | Pale Grass Blue Butterfly (Fukushima) | Internal exposure from ingestion of contaminated leaves by larvae; external exposure of all life stages. | Accumulation of genetic mutations over multiple generations in contaminated fields. | Transgenerational genomic instability observed in successive generations. | Hiyama et al. (2020) |
| Gamma/Beta (Chronic, ambient) | Plants & Animals (Chernobyl) | Multiple pathways: Ingestion, inhalation, root uptake, and aerial deposition. | Increased mutation rates and oxidative stress across diverse species. | Reduced brain size, sperm quality, and biodiversity. *Mechanism: Oxidative stress and genetic damage.* | Mousseau & Møller (2020) |

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| Gamma (Chronic, dose gradient) | Soil invertebrates (Chernobyl) | External exposure from contaminated soil & internal exposure from ingestion of contaminated soil. | N/A (Field study) | No consistent negative effect on soil fauna abundance across multiple taxa, suggesting high resilience at current field doses. | Beresford et al. (2020) |
| Gamma (Cs-137, chronic ingestion) | Wild boar (Fukushima and Chernobyl) | Internal exposure from ingestion of contaminated roots, fungi, and soil. | N/A (Field measurement) | Persistently high contamination levels providing insights into long-term bioaccumulation and ecosystem cycling of radionuclides. | Jones et al. (2024) |

Table 5 consolidates nine ecological studies investigating the toxicological effects of nuclear and ionizing radiation—primarily gamma and gamma/beta exposures—across diverse wildlife, plant, and soil invertebrate populations. These studies collectively highlight how chronic environmental radiation interacts with multiple organisms and ecosystems, providing a broad understanding of population- and community-level responses to radioactive contamination in real-world contexts, particularly in the Chernobyl Exclusion Zone and Fukushima.
A clear pattern emerges in radiation type and exposure metrics: gamma radiation dominates these ecological investigations, either as ambient chronic exposure or through localized deposition of radionuclides (Cs-137, Sr-90), while combined gamma/beta exposures are examined to capture fallout scenarios. The dose rates reported vary widely, from low chronic background levels (≈0.1 μGy/h) to substantial environmental contamination, emphasizing the gradient of real-world exposures experienced by organisms. These studies span multiple taxa, including mammals (wolves, wild boar), birds, insects, aquatic crustaceans, plants (Scots pine), and soil invertebrates, reflecting a comprehensive ecological scope.

Exposure pathways are multifaceted. External irradiation from habitat or environmental deposition interacts with internal exposures arising from ingestion of contaminated food, water, or soil. In aquatic systems, bioaccumulation through algae and sediments serves as a significant route, whereas terrestrial species are affected via root uptake in plants or dietary intake in herbivores and omnivores. The studies collectively demonstrate that both direct and indirect exposure routes influence organismal health outcomes and ecosystem integrity.

Despite variability in dose, species, and ecological niche, common mechanisms of toxicity emerge. Several studies report genomic instability, mutation accumulation, and oxidative stress across taxa, particularly in plants and invertebrates, linking chronic radiation exposure to cellular-level damage that scales to population- and community-level effects (Geras'kin et al., 2017; Mousseau & Møller, 2020; Hiyama et al., 2020). Bystander and transgenerational effects are also evident: the Pale Grass Blue Butterfly exhibits heritable genomic instability over successive generations, demonstrating that radiation impacts can persist beyond immediately exposed individuals (Hiyama et al., 2020). In mammals, population-level observations reveal nuanced outcomes: wolves in Chernobyl thrived despite contamination, likely due to reduced human interference, whereas wild boar show persistent internal contamination, reflecting bioaccumulation and highlighting long-term ecological cycling of radionuclides (Fuller et al., 2015; Jones et al., 2024). Birds and insects exhibit dose-dependent declines in abundance and fertility, underscoring the sensitivity of reproductive endpoints to chronic low-level radiation (Møller et al., 2018).

The findings emphasize that dose, duration, and species-specific sensitivity collectively shape ecological outcomes. Low to moderate chronic doses may have subtle or negligible population effects in resilient taxa (soil invertebrates), whereas cumulative exposure or higher doses exacerbate genotoxicity, oxidative stress, and reduced reproductive fitness in sensitive species. Furthermore, transgenerational and indirect exposure mechanisms expand the ecological footprint of radioactive contamination, revealing complex interplays between environmental persistence, bioaccumulation, and organismal response.

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In conclusion, the ecological evidence summarized in Table 5 highlights the systemic and long-term effects of nuclear and ionizing radiation across diverse ecosystems. By linking radiation exposure to genomic instability, oxidative stress, reproductive impairment, and population dynamics, these studies elucidate the toxicokinetics of radiation in real-world ecological contexts, providing essential insights for environmental risk assessment, conservation management, and understanding the broader biological consequences of chronic ionizing radiation.

**Synthesis and Implications**

The collective evidence from in vitro, in vivo, and ecological studies underscores the multifaceted toxicological impact of ionizing radiation, particularly gamma rays and X-rays, across biological scales. In vitro studies reveal consistent cellular and genetic responses, including DNA damage, oxidative stress, senescence, and bystander effects, demonstrating how even localized irradiation can propagate molecular dysfunction beyond directly exposed cells. Mechanistic pathways such as activation of the cGAS–STING axis, SASP, and error-prone DNA repair are recurrently observed, illustrating the molecular underpinnings of radiation-induced cellular injury. Dose, exposure duration, and cell type strongly influence these outcomes, highlighting the importance of context in interpreting radiation toxicity.

In vivo investigations expand these molecular insights to organismal effects, revealing how radiation exposure affects specific organs and systemic health outcomes. Studies in rodents and pediatric patients show that low-dose exposures can elicit adaptive, protective responses, whereas higher or cumulative doses induce persistent DNA damage, epigenetic modifications, impaired neurogenesis, and increased cancer risk. Whole-body versus localized irradiation paradigms further illustrate how exposure route modulates both immediate and delayed toxic effects. These findings bridge molecular damage observed in vitro with clinically relevant physiological and neurological consequences, providing a critical link between cellular mechanisms and organismal health.

Ecological studies provide a complementary perspective by situating radiation toxicity within real-world environmental contexts. Populations of plants, invertebrates, and vertebrates in Chernobyl and Fukushima exhibit varying resilience, with genomic instability, oxidative stress, reproductive impairment, and transgenerational effects reported across taxa. Observed outcomes are shaped by dose gradients, chronicity, exposure pathways, and species-specific sensitivity, with some populations (e.g., wolves) thriving under chronic contamination due to ecological factors such as human absence. These studies highlight the systemic and long-term ecological implications of radiation, extending the scope of toxicokinetic understanding from individual organisms to populations and ecosystems.

A cross-cutting theme across these studies is the dose- and context-dependent nature of radiation toxicity. At low doses, organisms may activate repair and adaptive pathways that mitigate damage, whereas higher or prolonged exposures overwhelm repair mechanisms, leading to cumulative DNA lesions, oxidative stress, and systemic dysfunction. The bystander effect, transgenerational instability, and epigenetic reprogramming illustrate that radiation effects are not confined to directly exposed cells or individuals, emphasizing the broader systemic and ecological reach of ionizing radiation. These mechanisms collectively inform both human health risk assessment and environmental management of contaminated areas.

Overall, the integration of molecular, organismal, and ecological evidence advances a holistic understanding of radiation toxicokinetics and systemic impacts. The findings have significant implications for radiation protection, medical imaging and therapy, environmental monitoring, and conservation strategies. Recognizing the interplay between exposure dose, route, duration, and organismal sensitivity is critical for predicting outcomes and mitigating risks. This synthesis emphasizes the necessity of multi-level, interdisciplinary approaches to fully capture the complex biological consequences of nuclear and ionizing radiation.

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## CONCLUSION

This systematic review provides strong evidence that ionizing radiation, particularly gamma rays and X-rays, affects biological systems across multiple levels of organization. Findings from cellular and molecular studies show that radiation consistently causes DNA strand breaks, oxidative stress, chromosomal abnormalities, and disruptions in the mechanisms responsible for maintaining genomic stability. These effects activate pathways related to senescence, inflammation, and modified gene expression, while also influencing how cells communicate with one another through bystander signaling. The reviewed studies make it clear that dose, exposure duration, and biological sensitivity are central factors determining whether cells mount adaptive responses or progress toward irreversible damage. At moderate to high doses, the capacity for repair is generally exceeded, leading to persistent genetic instability and increased likelihood of carcinogenic and degenerative processes.

In vivo research further expands this understanding by demonstrating how these cellular effects translate into measurable physiological and developmental outcomes. Rodent models show that both chronic low-dose and acute high-dose exposures can alter neurogenesis, modify epigenetic patterns, impair organ function, and increase susceptibility to disease. Human-based evidence, particularly from pediatric imaging studies, confirms that repeated or concentrated exposure elevates long-term health risks such as cancer and neurological impairment. The diversity of these results reflects the complexity of ionizing radiation's action in whole organisms, where factors such as tissue type, developmental stage, and exposure route shape the severity and nature of toxic effects. Together, the in vivo findings demonstrate that the impacts of radiation extend well beyond immediate cellular injury, influencing systemic processes and long-term health trajectories.

Ecological studies add another dimension by showing how ionizing radiation affects entire populations and ecosystems over extended periods. Research conducted in Chernobyl, Fukushima, and other contaminated environments reveals that plants, insects, birds, mammals, and aquatic organisms experience increased mutation rates, reduced reproductive success, altered behavior, and in some cases transgenerational genetic changes. While certain species have maintained stable populations due to ecological advantages or reduced human disturbance, many others show clear signs of biological stress and disrupted population dynamics. These ecological patterns, when viewed alongside molecular and organismal data, highlight radiation as a stressor capable of shaping both short-term biological responses and long-term ecological outcomes. Overall, the combined findings of this review demonstrate the far-reaching effects of gamma and X-ray radiation and point to the need for continued interdisciplinary research, improved environmental monitoring, and refined radiological protection strategies to safeguard human health and ecological stability.

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