ISSN: 2157-2518
Journal of Carcinogenesis & Mutagenesis

OPEN ACCESS Freely available online

Opinion Article

Radiation-Induced Mutations in Carcinogenesis

Joel Kim*

Department of Environmental Health Sciences, Graduate School of Public Health, Seoul National University, Seoul, Republic of Korea

DESCRIPTION

Radiation-induced mutations represent a critical area of study in genetics and cancer biology, as exposure to ionizing and non-ionizing radiation can lead to significant alterations in the genetic material of cells. These mutations arise when radiation interacts with DNA either directly or indirectly, resulting in structural and functional damage that, if unrepaired or misrepair, can contribute to carcinogenesis. Understanding the mechanisms underlying radiation-induced mutations is essential for evaluating cancer risk, improving therapeutic strategies, and developing protective measures against radiation exposure.

Ionizing radiation, such as X-rays, gamma rays, and particle radiation, possesses sufficient energy to remove tightly bound electrons from atoms, creating ions. This process can directly break the DNA backbone, causing Single-Strand Breaks (SSBs) and Double-Strand Breaks (DSBs), which are among the most lethal forms of DNA damage. Double-strand breaks are particularly significant because incorrect repair can lead to chromosomal aberrations such as deletions, inversions, and translocations. These cytogenetic alterations are commonly observed in radiation-associated cancers and serve as biomarkers of exposure.

In addition to direct DNA damage, radiation can also exert indirect effects through the radiolysis of water molecules, generating Reactive Oxygen Species (ROS) such as hydroxyl radicals. These highly reactive molecules can attack DNA bases and sugar moieties, leading to base modifications, abasic sites, and cross-linking. The accumulation of such damage increases the likelihood of mutations during DNA replication. Oxidative stress induced by radiation further amplifies genomic instability, contributing to long-term cellular consequences.

Non-ionizing radiation, including Ultraviolet (UV) radiation, primarily affects DNA by inducing the formation of pyrimidine dimers, particularly thymine dimers. These lesions distort the DNA helix and interfere with replication and transcription. If not repaired by nucleotide excision repair mechanisms, these distortions can result in point mutations. UV-induced mutations are strongly associated with skin cancers, highlighting the role of environmental radiation in mutagenesis.

Cells have evolved complex DNA repair mechanisms to counteract radiation-induced damage. These include base excision repair, nucleotide excision repair, homologous recombination, and non-homologous end joining. While these systems are generally effective, errors during the repair process can introduce mutations or chromosomal rearrangements. The fidelity of these repair pathways is a key determinant of cellular response to radiation and influences individual susceptibility to radiation-induced cancers.

Radiation-induced mutations also play a significant role in medical applications, particularly in radiation therapy for cancer treatment. While the primary goal of radiotherapy is to induce lethal DNA damage in cancer cells, surrounding normal tissues may also be affected, leading to secondary malignancies. Advances in targeted radiation techniques aim to minimize such risks by delivering precise doses to tumor tissues while sparing healthy cells. Nevertheless, the mutagenic potential of radiation remains a concern in clinical settings.

Cytogenetic analysis has been instrumental in detecting and characterizing radiation-induced chromosomal abnormalities. Techniques such as karyotyping, fluorescence in situ hybridization, and micronucleus assays are widely used to assess DNA damage and chromosomal instability. These methods are valuable not only in clinical diagnostics but also in radiation biology research and environmental monitoring.

The study of radiation-induced mutations has also contributed to our understanding of dose-response relationships and thresholds for safe exposure. Factors such as radiation type, dose, duration of exposure, and individual genetic background influence the extent of mutagenesis. This knowledge is essential for establishing safety guidelines in medical, occupational, and environmental contexts.

Correspondence to: Joel Kim, Department of Environmental Health Sciences, Graduate School of Public Health, Seoul National University, Seoul, Republic of Korea, E-mail: joel@kim23435.kr

Received: 31-Jan-2026, Manuscript No. JCM-26-31153; **Editor Assigned:** 02-Jan-2026, Pre QC No. JCM-26-31153 (PQ); **Reviewed:** 16-Jan-2026, QC No. JCM-26-31153; **Revised:** 23-Jan-2026, Manuscript No. JCM-26-31153 (R); **Published**: 30-Jan-2026, DOI: 10.35248/2157-2518.26.17.499

Citation: Kim J (2026). Radiation-Induced Mutations in Carcinogenesis. J Carcinog Mutagen. 17:499.

In conclusion, radiation-induced mutations are a major contributor to genomic instability and cancer development, arising from both direct DNA damage and indirect oxidative mechanisms. Continued research in this field is vital for improving radiation safety standards, enhancing therapeutic outcomes, and deepening our understanding of the molecular basis of mutagenesis.