



Radiation Mutagenesis and Its Impact on Genetic Stability

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DESCRIPTION

Radiation mutagenesis refers to the process by which ionizing or non-ionizing radiation induces alterations in the genetic material of living organisms. These alterations can result in a wide range of genetic changes, from point mutations and chromosomal rearrangements to large-scale deletions or duplications. The study of radiation-induced mutations has been instrumental in understanding the mechanisms of Deoxyribonucleic Acid DNA damage, repair and genomic instability. It has also provided valuable insights into the evolution of organisms, the development of cancer and the risks associated with environmental and occupational exposure to radiation.

Radiation mutagenesis is a double-edged phenomenon; while it poses significant health hazards, it has also been harnessed in experimental genetics and crop improvement programs. Ionizing radiation, including X-rays, gamma rays and high-energy particles, is the primary source of radiation-induced mutations. These forms of radiation possess sufficient energy to ionize atoms and molecules within cells, generating reactive free radicals that interact with DNA. The resulting damage can include single-strand breaks, double-strand breaks, base modifications and cross-linking. Among these, double-strand breaks are particularly critical because they are difficult for the cell to repair accurately and often lead to chromosomal aberrations.

Non-ionizing radiation, such as ultraviolet light, primarily induces mutations through the formation of pyrimidine dimers, which distort the helix and interfere with replication and transcription. Failure to repair these lesions can result in permanent mutations that may alter gene function. Cells possess multiple repair mechanisms to counteract the effects of radiation-induced damage. Base excision repair, nucleotide excision repair and mismatch repair address specific types of lesions, while homologous recombination and non-homologous end joining repair double-strand breaks. The efficiency and accuracy of these repair pathways determine the likelihood that a mutation will become permanent. Mutagenesis is not random but is influenced by the type of radiation, its energy, the phase of

the cell cycle and the chromatin structure surrounding the nucleus.

Highly condensed chromatin regions may be more resistant to damage but less accessible for repair, whereas actively transcribed genes may be more vulnerable to mutational events. From a human health perspective, radiation mutagenesis is a major concern due to its association with cancer and heritable genetic disorders. Exposure to high doses of ionizing radiation, such as during nuclear accidents or medical imaging procedures, can significantly increase the risk of malignancies. Mutations in oncogenes or tumor suppressor genes can disrupt normal cell growth regulation, leading to uncontrolled proliferation and tumor formation. Similarly, germline mutations induced by radiation can be transmitted to offspring, potentially causing developmental defects or predisposition to disease.

Epidemiological studies of populations exposed to radiation, including survivors of atomic bombings and patients undergoing radiotherapy, have provided valuable data on dose-response relationships and the long-term consequences of mutagenic exposure.

Radiation mutagenesis is also relevant to environmental and occupational health. Workers in nuclear power plants, radiology departments, or space missions are exposed to varying levels of radiation, necessitating strict safety protocols and monitoring. Regulatory agencies have established permissible dose limits and guidelines to minimize risk, while research continues to develop protective measures, such as radio protective agents that scavenge free radicals or enhance repair. In addition, understanding radiation-induced mutagenesis contributes to risk assessment in situations involving environmental contamination, medical imaging and cosmic radiation.

Recent advances in molecular biology and genomics have expanded our understanding of radiation mutagenesis. High-throughput sequencing technologies enable precise mapping of radiation-induced mutations at the nucleotide level, revealing mutation signatures unique to different types and doses of radiation. These findings have implications for cancer research, as specific mutational patterns can indicate prior exposure and

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guide treatment decisions. Furthermore, gene editing technologies have allowed experimental manipulation repair pathways to study their role in mutagenesis, opening avenues for potential therapeutic interventions that could reduce the harmful effects of radiation exposure. A comprehensive understanding of radiation mutagenesis not only informs public health and safety measures but also provides valuable insights into the fundamental processes of genome stability, evolution and disease prevention.

In conclusion, radiation mutagenesis is a fundamental biological process with far-reaching implications for genetics, medicine and

environmental health. It arises from cell damage caused by ionizing and non-ionizing radiation and is influenced by cellular repair mechanisms, chromatin structure and exposure conditions. While radiation mutagenesis poses significant risks, including cancer development and heritable genetic defects, it has also been harnessed as a tool in experimental genetics and plant breeding. Ongoing research into the molecular mechanisms underlying radiation-induced mutations, along with the development of protective strategies and advanced genomic techniques, continues to enhance our understanding of this phenomenon.