# Biological response to radiation therapy

Isha Jagat

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## 1 INTRODUCTION

The body's reaction to DNA and linage. The present perspective on studies of DNA damage and cell cycle response following ionising radiation (IR) and its application in radiation oncology has been greatly influenced by the avalanche of information in cellular biochemistry and molecular biology. When DNA is damaged by IR, mammalian cells respond by activating two critical physiological functions: cell cycle regulation and DNA repair. Most cells have repair pathways that are always active and are controlled by cell cycle checkpoints. The cell must choose between DNA repair and cell death after recognising damaged DNA and assessing the damage. The goal is to protect the genome's integrity. Radiation response to DNA linage. radiation (IR) and its use in radiation oncology. When DNA is damaged by IR, mammalian cells respond by activating two critical physiological functions: cell cycle regulation and DNA repair. Most cells have repair pathways that are always active and are controlled by cell cycle checkpoints. The cell must choose between DNA repair and cell death after recognising damaged DNA and assessing the damage. The goal is to protect the genome's integrity.

### 1.1 DNA repair:

Effective DNA repair is critical for the genome's long-term stability. The DNA-dsb is the most major DNA damage linked to reproductive cell death in response to IR. Dsb can also be generated by normal endogenous mechanisms such oxygen free radicals, DNA replication, or topoisomerase failure, in addition to IR. Mis rejoining events such as dicentric chromosomes and ring chromosomes, both of which lead to a mitotic catastrophe within 1-2 turns of the cell cycle, are linked to persistent damage. The dsb could also result in significant translocations or the insertion of genetic material outside of its original place. These key events, which may include oncogene activation and deletion or down-regulation of housekeeping genes, are often persistent during the next cell divisions. Biophysical experiments reveal a link between radiosensitivity and both unrejoined dsb fractions and rejoining kinetics. Even while the majority of cells that progressively rejoin the breaks appear to be IR sensitive, other radiosensitive cell lines rejoin the breaks regularly.

## 1.2 Apoptotic:

In recent years, the manner of cell death caused by DNA damage triggered by anticancer treatments, including radiotherapy, has been highlighted. The discovery of the function of wild-type TP53 in inducing apoptosis in response to DNA damage has sparked a huge interest in programmed cell death. There is clear doubt that after DNA damage that is dependent on wild-type TP53, lymphoid tissues and lymphoma cells die a quick apoptotic death. New ideas have arisen as a result of the fact that apoptosis is thought to be linked to genetically specified pathways. One of these ideas is that the genotype of normal or tumour cells can predict DNA-damaging chemical susceptibility.

### 1.2.1 Clonogenic:

In a Clonogenic experiment, both genotypes may demonstrate similar cell survival. If clonogenic survival is considered as the endpoint for cell death, neither TP53 status nor the ability of the cells to undergo apoptosis appears to play a significant impact in the susceptibility of these cells to DNA-damaging chemicals, according to the findings of this review. A recent review looked at the ambiguous role of apoptosis in non-lymphoid normal tissues for maintaining the balance between cell production and cell loss. Apoptosis contributes just a small amount to cell turnover in mammalian normal tissues, with a few exceptions. Several processes control cell death in tumours, such as necrosis produced by insufficient angiogenesis.

## 1.3 Critical issues:

New pathways involved in cell cycle checkpoints and DNA repair will almost certainly be discovered in the near future. However, converting this knowledge into clinically viable medicines faces numerous challenges. —The application of recent understanding, as seen above, to the in vivo and human condition is still largely unknown. —It is necessary to determine the relative importance of abnormalities in the various signalling pathways involved in cell cycle checkpoints and DNA repair for radiosensitivity. The radiosensitivity of people who are heterozygous or have polymorphisms for some of the genes involved in the complex machinery of cell cycle checkpoints and DNA repair differs from the general population. It is necessary to gain a better understanding of how common these abnormalities are among cancer patients.

# 2 CELL AND TISSUE RESPONSE TO DOSES ABOVE AND AROUND 1 GY

# 3 CELL AND TISSUE RESPONSE TO LOW RADIATION DOSES—HRSIIRR