|| ISSN(online): 2589-8698 || ISSN(print): 2589-868X ||

Available online on http://www.ijmbs.info

International Journal of Medical and Biomedical Studies; 2025; 9(2); 1-5

Original Research Article

# Biological Effects of Radiation Therapy on Cancer Cells

**Harihar Nath Tiwari[1], Subhadra Choubey[2], Ravi Byahut[3], Rajesh Kumar Singh[4], Seema Devi[5]**

[1]Senior Resident, Department of Radiotherapy, Patna Medical College & Hospital, Patna, Bihar, India
[2]Senior Resident, Department of Radiotherapy, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India
[3]Professor & HOD, Department of Radiotherapy, Patna Medical College & Hospital, Patna, Bihar, India
[4]Professor & HOD, Department of Radiotherapy, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India
[5]Professor, Department of Radiotherapy, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India

**Received: 24-12-2024 / Revised: 11-01-2025 / Accepted: 17-02-2025**
**DOI: https://doi.org/10.32553/ijmbs.v9i2.2967**
**Corresponding author: Subhadra Choubey**
**Conflict of interest: No conflict of interest**

**Abstract:**

**Background:** Radiation therapy damages tumour cells' DNA and induces apoptosis, a common cancer treatment. The biological response to radiation depends on tumour type, genetics, and microenvironment. This study examines radiation therapy's effects on cancer cells and tumour regression and survival.

**Methods:** A prospective observational study of 150 radiation therapy patients was done at Patna Medical College and Hospital (PMCH) from April 2022 to March 2024. The biological response was evaluated using γ-H2AX DNA damage markers, caspase-3 apoptosis markers, and tumour regression rates. We also examined treatment-related toxicity and survival.

**Results:** Of the patients, 85% demonstrated a strong DNA damage response (γ-H2AX expression) and 70% indicated enhanced apoptosis (caspase-3 activation). Head and neck (75%) and cervical (70%) malignancies had the highest tumour regression rates (68%). Patients had 40% treatment-related toxicities, 10% of which were severe (Grade 3–4). PFS was 65% and OS was 72% at 1 year.

**Conclusion:** Radiation therapy effectively induces DNA damage and apoptosis, leading to significant tumor regression. However, tumor resistance remains a challenge, necessitating further research into radiosensitizers and combination therapies to enhance treatment efficacy.

**Keywords:** Radiation therapy, DNA damage, apoptosis, tumor regression, survival outcomes



## Introduction

A key component of cancer treatment is radiation therapy, which uses ionising radiation to target and kill cancerous cells while causing the least amount of harm to nearby healthy tissues [1]. Radiation dose, cell type, tumour microenvironment, and inherent genetic traits are some of the variables that affect the complicated biological response of cancer cells to radiation [2]. The main way that radiation

works is by causing DNA damage, which can result in cell cycle arrest, apoptosis, necrosis, or mitotic disaster. Radiation can also affect tumour vasculature and cause immunological reactions, which can further affect tumour control [3,4].

Understanding the mechanisms underlying the biological response of cancer cells to radiation is crucial for optimizing treatment efficacy and minimizing adverse effects [5]. Research in this field focuses on enhancing radiosensitivity through combined therapies, identifying biomarkers for radiation response, and developing personalized treatment strategies [6,7].

This study aims to evaluate the biological response of cancer cells to radiation therapy, exploring the mechanisms of DNA damage, repair pathways, and cellular outcomes to improve therapeutic strategies and enhance treatment effectiveness.

## Materials and Methods

### Study Design

This is a prospective observational study conducted to assess the biological response of cancer cells to radiation therapy.

### Study Setting

The study was carried out at Patna Medical College and Hospital (PMCH), Patna, over a period of two years from April 2022 to March 2024.

### Study Population

A total of approximately 150 patients diagnosed with various malignancies and undergoing radiation therapy were included in the study.

### Inclusion Criteria

- Patients diagnosed with histologically confirmed malignancies.
- Patients undergoing radiation therapy as a part of their treatment plan.
- Patients willing to provide informed consent for participation in the study.

### Exclusion Criteria

- Patients with a history of prior radiation therapy for the same malignancy.
- Patients with severe comorbidities that could influence radiation response.
- Patients lost to follow-up during the study period.

### Data Collection and Analysis

- **Clinical and Demographic Data**: Patient demographics, tumor type, staging, and treatment history were documented.
- **Radiation Treatment Details**: Radiation dose, fractionation schedule, and concurrent therapies were recorded.
- **Biological Response Assessment**:
    - **Cellular Response**: Evaluation of DNA damage markers (e.g., γ-H2AX), apoptosis, and cell cycle arrest.
    - **Tumor Response**: Radiological and clinical assessment of tumor regression.
    - **Biomarker Analysis**: Expression of radiation-induced stress markers and immune response indicators.
- **Follow-up**: Patients were monitored for acute and late radiation-induced effects, along with overall treatment response.

### Statistical Analysis

Data were analyzed using appropriate statistical methods to determine the correlation between radiation exposure and biological responses in cancer cells. Survival outcomes, tumor regression rates, and treatment-related toxicities were assessed.

## Results

According to clinical and radiological evaluations, 68% of cases had partial or whole tumour regression; head and neck cancers had the highest response rates (75%) and cervical cancers (70%). 18% of individuals had tumour progression, mostly those with radioresistant tumour subtypes or advanced illness. 40% of patients experienced radiation-induced toxicities, with 30% experiencing grade 1–2 toxicities (fatigue, mucositis, dermatitis) and 10% experiencing grade 3–4 toxicities (severe

mucositis, pneumonitis, myelosuppression). With a 1-year progression-free survival (PFS) of 65% and an overall survival (OS) of 72%, the trial also assessed long-term therapy outcomes. Tumour regression and survival outcomes were better for patients with substantial DNA damage responses and apoptotic markers ($p < 0.05$) than for those with low biomarker expression.

Overall, the study highlights that radiation therapy effectively induces DNA damage and apoptotic cell death in cancer cells, with a significant correlation between biological response and treatment outcomes. However, variations in tumor radiosensitivity and toxicity profiles suggest the need for personalized treatment approaches to optimize therapeutic efficacy.

**Table 1: Patient Demographics and Tumor Distribution**

| Characteristic | Number of Patients (n=150) | Percentage (%) |
|---|---|---|
| **Mean Age (years)** | 54.2 ± 10.8 | - |
| **Gender** | | |
| Male | 90 | 60% |
| Female | 60 | 40% |
| **Tumor Type** | | |
| Head and Neck Cancer | 53 | 35% |
| Cervical Cancer | 38 | 25% |
| Lung Cancer | 30 | 20% |
| Gastrointestinal Cancers | 29 | 20% |

**Table 2: Radiation Dose and Biological Response**

| Parameter | Value | Percentage (%) |
|---|---|---|
| **Mean Radiation Dose (Gy)** | 50 ± 5 | - |
| **DNA Damage Response (γ-H2AX Positive Cases)** | 128 | 85% |
| **Apoptosis Response (Caspase-3 Activation)** | 105 | 70% |

**Table 3: Tumor Response to Radiation Therapy**

| Response Category | Number of Patients | Percentage (%) |
|---|---|---|
| **Complete or Partial Tumor Regression** | 102 | 68% |
| Head and Neck Cancer | 40 | 75% |
| Cervical Cancer | 27 | 70% |
| Lung Cancer | 18 | 60% |
| Gastrointestinal Cancer | 17 | 58% |
| **Tumor Progression** | 27 | 18% |
| **Stable Disease** | 21 | 14% |

**Table 4: Radiation-Induced Toxicities**

| Toxicity Grade | Number of Patients | Percentage (%) |
|---|---|---|
| **Grade 1–2 (Mild-Moderate)** | 45 | 30% |
| Dermatitis | 20 | 13% |
| Mucositis | 15 | 10% |
| Fatigue | 10 | 7% |
| **Grade 3–4 (Severe)** | 15 | 10% |
| Severe Mucositis | 7 | 5% |
| Pneumonitis | 5 | 3% |
| Myelosuppression | 3 | 2% |

**Table 5: Survival Outcomes**

| Outcome | Value | Percentage (%) |
|---|---|---|
| **1-Year Progression-Free Survival (PFS)** | - | 65% |
| **Overall Survival (OS)** | - | 72% |
| **Correlation of Biomarkers with Tumor Regression ($p < 0.05$)** | Significant | - |

## Discussion

Radiation therapy is still one of the best ways to treat a variety of cancers because it damages DNA and sets off cellular reactions that cause tumour regression or apoptosis. Over the course of two years (April 2022–March 2024), 150 patients at PMCH had their biological response to radiation therapy evaluated. The results showed that 70% of patients had increased apoptotic activity (caspase-3 activation) and 85% of patients had a significant DNA damage response (γ-H2AX expression), which were associated with better tumour regression and survival outcomes. Our research supports the findings of Joiner and van der Kogel (2009), who found that radiation-induced DNA damage causes cancer cells to undergo necrosis, apoptosis, and mitotic disaster, all of which ultimately contribute to tumour reduction [8]. Additionally, a study by Loeffler and Durante (2020) highlighted that the main mechanism of radiation-induced cytotoxicity is double-strand DNA breakage [9].

In our study, 68% of patients showed either full or partial tumour response, with head and neck cancers showing the highest response (75%) and cervical malignancies showing the highest response (70%). Because of their radiosensitivity, head and neck malignancies responded to fractionated radiation therapy with a 70–80% response rate, according to research by Bentzen (2006) [10]. Furthermore, our study's 1-year overall survival (OS) of 72% and progression-free survival (PFS) of 65% are in line with a meta-analysis by Overgaard et al. (2011) that showed radiation therapy for cervical cancer patients had a 5-year survival advantage of roughly 60–70% [11]. The impact of radiation-induced toxicity was also emphasised in the study. Treatment-related toxicities affected 40% of patients, with 10% suffering from severe (Grades 3–4) side effects such as myelosuppression, pneumonitis, and mucositis. These results are in line with those of Gupta et al. (2012), who found that between 30 and 50 percent of patients with head and neck cancer experience radiation-induced toxicities, including mucositis [12]. Radiation therapy has advanced, although tumour resistance still exists. Due to the significant radioresistance of gastrointestinal and lung malignancies, our study revealed that 18% of patients experienced tumour development despite radiation therapy. This is consistent with the findings of the Baumann et al. (2016) study, which showed that tumours with hypoxic conditions and those with malfunctioning apoptotic pathways react poorly to radiation treatment [13]. To increase the effectiveness of radiation, new approaches like immunotherapy, targeted medicines, and radiation sensitizers are being investigated. Combining radiation with immune checkpoint inhibitors (e.g., anti-PD-1/PD-L1) may enhance tumour responses by promoting anti-tumor immunity, according to studies by Formenti and Demaria (2013) [14].

## Conclusion

With the greatest results in head and neck and cervical malignancies, this study showed that radiation therapy efficiently causes DNA damage, apoptosis, and tumour regression. Radiation resistance is still an issue, though, especially with

gastrointestinal and lung cancers, therefore more research into combination treatments and radiosensitizers is required. Personalised radiation therapy techniques require further multi-institutional investigations with molecular profiling, longer follow-up periods, and larger sample sizes.

## References


1. Wang H, Mu X, He H, Zhang XD. Cancer radiosensitizers. Trends Pharmacol Sci. 2018;39(1):24-48.
2. Rödel F, Keppner S, Capalbo G, et al. Survival of irradiated tumor cells under hypoxia and the role of DNA repair. Radiother Oncol. 2011;99(3):377-382.
3. Barker HE, Paget JT, Khan AA, Harrington KJ. The tumour microenvironment after radiotherapy: mechanisms of resistance and recurrence. Nat Rev Cancer. 2015;15(7):409-425.
4. Lee CL, Blum JM, Kirsch DG. Role of p53 in regulating tissue response to radiation. J Clin Oncol. 2019;37(27):239-247.
5. Chatterjee A, Dasgupta S, Sidransky D. Molecular markers for cancer detection and radiation response. Nat Rev Cancer. 2018;18(8):557-567.
6. Hall EJ, Giaccia AJ. Radiobiology for the radiologist. 7th ed. Philadelphia: Lippincott Williams & Wilkins; 2012.
7. Verheij M, Dewit LG, Boomgaard MN, et al. Radiation-induced apoptosis in relation to cell proliferation in murine lymphoma. Int J Radiat Biol. 2019;95(3):348-358.
8. Joiner MC, van der Kogel A. Basic clinical radiobiology. 4th ed. London: Hodder Arnold; 2009.
9. Loeffler JS, Durante M. Charged particle therapy—optimization, challenges and future directions. Nat Rev Clin Oncol. 2020;17(6):361-370.
10. Bentzen SM. Preventing or reducing late side effects of radiation therapy: radiobiology meets molecular pathology. Nat Rev Cancer. 2006;6(9):702-713.
11. Overgaard J, Hansen HS, Specht L, Overgaard M. The role of radiotherapy in cervical cancer treatment: results from meta-analyses. Radiother Oncol. 2011;100(2):117-126.
12. Gupta T, Agarwal J, Jain S. Radiation-induced mucositis: current treatment and emerging therapies. J Cancer Res Ther. 2012;8(3):203-210.
13. Baumann M, Krause M, Overgaard J, et al. Radiation oncology in the era of precision medicine. Nat Rev Cancer. 2016;16(4):234-249.
14. Formenti SC, Demaria S. Combining radiotherapy and cancer immunotherapy: a paradigm shift. J Natl Cancer Inst. 2013;105(4):256-265.