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Inverse Correlation between RAD51 Expression and Survival in Glioblastoma Patients

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Introduction

Glioblastoma (GBM) is the most aggressive and lethal primary brain tumor, characterized by rapid progression, resistance to therapy, and poor survival outcomes. Despite advances in surgical resection, radiation therapy, and chemotherapy, the median survival of GBM patients remains limited to approximately 12–15 months post-diagnosis. Recent research has focused on molecular biomarkers that influence tumor progression and patient prognosis. One such biomarker is RAD51, a crucial protein involved in Homologous Recombination (HR) repair of DNA double-strand breaks. Studies indicate that elevated RAD51 expression is associated with increased tumor aggressiveness and therapy resistance, leading to poorer survival outcomes in GBM patients. RAD51 plays an essential role in maintaining genomic stability by facilitating the error-free repair of DNA damage. It is a key component of the HR repair pathway, allowing cells to recover from DNA damage that would otherwise result in cell death. However, in cancer, the overexpression of RAD51 provides tumor cells with an enhanced ability to repair therapy-induced DNA damage, thus conferring resistance to radiation and chemotherapy. This mechanism enables GBM cells to evade standard treatments, promoting tumor recurrence and worsening patient prognosis.

Description

Clinical studies have demonstrated an inverse correlation between RAD51 expression levels and survival in GBM patients. High RAD51 expression has been linked to increased tumor proliferation, enhanced DNA repair capacity, and reduced sensitivity to therapeutic interventions. By analyzing patient tumor samples, researchers have found that individuals with elevated RAD51 expression tend to have significantly shorter overall survival and progression-free survival compared to those with lower RAD51 levels. The correlation remains significant even when accounting for variables such as age, tumor grade, and treatment regimen, emphasizing the independent prognostic value of RAD51 in GBM. Mechanistically, the upregulation of RAD51 in GBM may be driven by various oncogenic signaling pathways, including the activation of the phosphoinositide 3-kinase (PI3K)/Akt pathway, which has been implicated in promoting DNA repair and cell survival. Additionally, hypoxic conditions within the tumor microenvironment may induce RAD51 expression, further enhancing the resilience of GBM cells to therapy-induced DNA damage. These findings suggest that targeting RAD51-mediated DNA repair could represent a promising therapeutic strategy for overcoming resistance in GBM [1].

Preclinical studies have explored the potential of RAD51 inhibitors as a means to sensitize GBM cells to radiation and chemotherapy. Small-molecule inhibitors targeting RAD51 or its interacting partners have shown promise in reducing tumor cell viability and enhancing the effectiveness of DNA-damaging agents. By impairing the HR repair pathway, these inhibitors increase the accumulation of DNA damage, leading to tumor cell apoptosis. Furthermore, the combination of RAD51 inhibition with existing treatments such as temozolomide (TMZ), the standard chemotherapeutic agent for GBM, has demonstrated synergistic effects in preclinical models, supporting the rationale for clinical translation. Given the critical role of RAD51 in GBM progression, the development of targeted therapies against this protein is an area of active investigation. Strategies to downregulate RAD51 expression, such as RNA interference (RNAi) and CRISPR-based gene editing, are being explored as potential approaches to enhance treatment efficacy. Moreover, the identification of biomarkers that predict RAD51 dependency in GBM could help stratify patients who would benefit most from RAD51-targeted therapies. Personalized treatment approaches incorporating RAD51 inhibitors in combination with conventional therapies could potentially improve survival outcomes in GBM patients [2,3].

While RAD51 has emerged as a promising therapeutic target, challenges remain in translating these findings into clinical applications. One major hurdle is the potential toxicity associated with RAD51 inhibition, as normal cells also rely on HR repair for genomic stability. Therefore, selective targeting of RAD51 in tumor cells while minimizing adverse effects on healthy tissues is a key consideration for drug development. Additionally, resistance mechanisms to RAD51 inhibition may arise, necessitating the exploration of combination therapies to prevent treatment escape. The inverse correlation between RAD51 expression and GBM patient survival highlights the need for continued research into DNA repair mechanisms as potential therapeutic targets. Further studies are required to validate the prognostic value of RAD51 in larger patient cohorts and to establish its role as a predictive biomarker for treatment response. Advancements in molecular profiling and precision oncology will be instrumental in guiding the development of novel therapeutic strategies aimed at improving outcomes for GBM patients [4,5].

Conclusion

In conclusion, RAD51 expression levels serve as a critical determinant of GBM prognosis, with higher expression correlating with poorer survival outcomes. The ability of RAD51 to enhance DNA repair and confer resistance to standard therapies underscores its role as a key driver of tumor progression. Targeting RAD51 and its associated pathways presents an opportunity to overcome therapy resistance and improve the efficacy of GBM treatments. Future research efforts focused on RAD51 inhibition and personalized treatment strategies hold promise for transforming the therapeutic landscape of GBM, offering hope for better patient survival and quality of life.

Acknowledgement

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Conflict of Interest

None.

References

1. Louis, David N., Arie Perry, Guido Reifenberger and Andreas Von Deimling, et al. "The 2016 World Health Organization classification of tumors of the central nervous system: A summary." *Acta Neuropathol* 131 (2016): 803-820.

2. Stupp, Roger, Sophie Taillibert, Andrew A. Kanner and Santosh Kesari, et al. "Maintenance therapy with tumor-treating fields plus temozolomide vs temozolomide

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alone for glioblastoma: A randomized clinical trial." *Jama* 314 (2015): 2535-2543.

3. Cloughesy, Timothy F. and Andrew B. Lassman. "NovoTTF: Where to go from here?." *Neuro-Oncol* 19 (2017): 605-608.

4. Hegi, Monika E., Annie-Claire Diserens, Thierry Gorlia and Marie-France Hamou, et al. "MGMT gene silencing and benefit from temozolomide in glioblastoma." *N Engl J Med* 352 (2005): 997-1003.

5. Bar, Eli E. "Glioblastoma, cancer stem cells and hypoxia." *Brain Pathol* 21 (2011): 119-129.

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