# Article information:

RBBP4 regulates the expression of the Mre11-Rad50-NBS1 (MRN) complex and promotes DNA double-strand break repair to mediate glioblastoma chemoradiotherapy resistance - ScienceDirect
<https://www.sciencedirect.com/science/article/pii/S0304383523000290>

# Article summary:

1. RBBP4 is an effective modulator of radiosensitivity and chemosensitivity in MGMT-negative GBM.

2. RBBP4 regulates the expression of the Mre11-Rad50-NBS1 (MRN) complex and the level of DNA-DSB repair, which are closely associated with recovery from TMZ- and radiotherapy-induced DNA damage in U87MG and LN229 glioblastoma cells.

3. Disruption of RBBP4 induced GBM cell DNA damage and apoptosis in response to TMZ and radiotherapy, enhancing radiotherapy and chemotherapy sensitivity by the independent pathway of MGMT.

# Article rating:

May be slightly imbalanced: The article presents the information in a generally reliable way, but there are minor points of consideration that could be explored further or claims that are not fully backed by appropriate evidence. Some perspectives may also be omitted, and you are encouraged to use the research topics section to explore the topic further.

# Article analysis:

The article “RBBP4 regulates the expression of the Mre11-Rad50-NBS1 (MRN) complex and promotes DNA double-strand break repair to mediate glioblastoma chemoradiotherapy resistance” provides a detailed overview of how RBBP4 can modulate glioblastoma resistance to chemotherapy and radiotherapy by recruiting transcription factors and epigenetic regulators that bind to their promoters to regulate the expression of the Mre11-Rad50-NBS1 (MRN) complex and the level of DNA double strand break repair. The article is well written, providing a comprehensive overview of its topic, supported by evidence from clinical studies as well as experiments conducted on U87MG and LN229 glioblastoma cells.

The article does not appear to be biased or one sided, presenting both sides equally with no promotional content or partiality. It also mentions potential risks associated with disruption of RBBP4, such as increased sensitivity to TMZ plus RT in vitro and in vivo. However, there are some missing points that should be considered when evaluating this article’s trustworthiness. For example, it does not explore any counterarguments or provide evidence for its claims made about RBBP4’s role in mediating glioblastoma chemoradiotherapy resistance. Additionally, it does not discuss any potential limitations or drawbacks associated with its findings or conclusions.

In conclusion, while this article provides a comprehensive overview of its topic with evidence from clinical studies as well as experiments conducted on U87MG and LN229 glioblastoma cells, it lacks exploration into counterarguments or evidence for its claims made about RBBP4’s role in mediating glioblastoma chemoradiotherapy resistance as well as discussion about potential limitations or drawbacks associated with its findings or conclusions.

# Topics for further research:

* Glioblastoma chemoradiotherapy resistance
* RBBP4 counterarguments
* Evidence for RBBP4 role in glioblastoma
* Limitations of RBBP4 research
* Potential drawbacks of RBBP4 findings
* Clinical implications of RBBP4 research

# Report location:

<https://www.fullpicture.app/item/6889821f65ddc75313aad7f03390ad43>