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## Enhanced radiosensitivity and radiation-induced apoptosis in glioma CD133-positive cells by knockdown of SirT1 expression

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## Abstract

CD133-expressing glioma cells play a critical role in tumor recovery after treatment and are resistant to radiotherapy. Herein, we demonstrated that glioblastoma-derived CD133-positive cells (GBM-CD133<sup>+</sup>) are capable of self-renewal and express high levels of embryonic stem cell genes and SirT1 compared to GBM-CD133<sup>-</sup> cells. To evaluate the role of SirT1 in GBM-CD133<sup>+</sup>, we used a lentiviral vector expressing shRNA to knock-down SirT1 expression (sh-SirT1) in GBM-CD133<sup>+</sup>. Silencing of SirT1 significantly enhanced the sensitivity of GBM-CD133<sup>+</sup> to radiation and increased the level of radiation-mediated apoptosis. Importantly, knock-down of SirT1 increased the effectiveness of radiotherapy in the inhibition of tumor growth in nude mice transplanted with GBM-CD133<sup>+</sup>. Kaplan–Meier survival analysis indicated that the mean survival rate of GBM-CD133<sup>+</sup> mice treated with radiotherapy was significantly improved by Sh-SirT1 as well. In sum, these results suggest that SirT1 is a potential target for increasing the sensitivity of GBM and glioblastoma-associated cancer stem cells to radiotherapy.

**Keywords:** Glioblastoma; SirT1; CD133; Radiotherapy

## Article Outline

### Materials and methods

### Results

Isolation and characterization of CD133-positive cells from glioblastoma

Knock-down of SirT1 expression enhanced radiosensitivity and radiation-induced apoptosis in GBM-CD133<sup>+</sup>

Knock-down of SirT1 in GBM-CD133<sup>+</sup> enhanced the effectiveness of IR treatment in vivo

### Discussions

### Acknowledgements

### Appendix A. Supplementary data

### References



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