

Temozolomide sensitivity

Temozolomide sensitivity reported in comparable studies was not consistent between or within malignant glioma cell lines. Drug discovery science performed on these models cannot reliably inform clinical translation. A consensus model of reporting can maximise reproducibility and consistency among in vitro studies ¹⁾.

Circular RNAs have been reported to play key roles in the progression of various cancers, including gliomas. Yuan et al. designed a study to investigate the role of [hsa_circ_0072309](#) in autophagy and temozolomide sensitivity in glioblastoma (Glioblastoma).

The effect of [hsa_circ_0072309](#) on autophagy and TMZ sensitivity were examined by [GFP-RFP-LC3](#), [transmission electron microscopy](#) (TEM), [flow cytometry](#), [Western blot](#), and [immunofluorescence](#). The mechanism of [hsa_circ_0072309](#) regulating the [p53 signaling pathway](#) was analyzed using Western blot, IP, and rescue experiments.

Low [hsa_circ_0072309](#) expression predicts poor prognosis for glioma patients. The regulation of [hsa_circ_0072309](#) on autophagy and TMZ sensitivity depends on the status of p53. [Hsa_circ_0072309](#) promoted autophagy by p53 signaling pathway and enhanced sensitivity of glioblastoma to temozolomide (TMZ) in p53 wild-type Glioblastoma, but not in p53 mutant Glioblastoma. [Hsa_circ_0072309](#) inhibits p53 ubiquitination and increases the stability of p53 protein in the context of p53 wild-type. MiR-100 mediates [hsa_circ_0072309](#) regulating p53. P53 inhibitor or autophagy inhibitor could reverse the effect of [hsa_circ_0072309](#) on TMZ sensitivity in p53 wild-type Glioblastoma.

This study revealed a function of [hsa_circ_0072309](#) promoting autophagy by p53 signaling pathway and enhancing TMZ sensitivity. These findings demonstrated that [hsa_circ_0072309](#) may be a potential and promising target in designing the treatment strategy for Glioblastoma. ²⁾

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