

YTHDF2

[YTH protein domain](#) family member 2 (YTHDF2) is highly expressed in Glioblastoma tissues, while the mechanism of YTHDF2 in [Temozolomide resistance](#) in Glioblastoma remains not fully elucidated.

The YTHDF2 expression in TMZ-resistant tissues and cells was detected. Kaplan-Meier analysis was employed to evaluate the prognostic value of YTHDF2 in Glioblastoma. Effect of YTHDF2 in TMZ resistance in Glioblastoma was explored via corresponding experiments. RNA sequence, FISH in conjugation with fluorescent immunostaining, RNA immunoprecipitation, dual-luciferase reporter gene and immunofluorescence were applied to investigate the mechanism of YTHDF2 that boosted TMZ resistance in Glioblastoma.

YTHDF2 was up-regulated in TMZ-resistant tissues and cells, and patients with high expression of YTHDF2 showed lower survival rate than the patients with low expression of YTHDF2. The elevated YTHDF2 expression boosted TMZ resistance in Glioblastoma cells, and the decreased YTHDF2 expression enhanced TMZ sensitivity in TMZ-resistant Glioblastoma cells. Mechanically, YTHDF2 bound to the N6-methyladenosine (m6A) sites in the 3'UTR of EPHB3 and TNFAIP3 to decrease the mRNA stability. YTHDF2 activated the PI3K/Akt and NF-κB signals through inhibiting expression of EPHB3 and TNFAIP3, and the inhibition of the two pathways attenuated YTHDF2-mediated TMZ resistance.

YTHDF2 enhanced TMZ resistance in Glioblastoma by activation of the PI3K/Akt and NF-κB signalling pathways via inhibition of [EPHB3](#) and [TNFAIP3](#) ¹⁾.

¹⁾

Chen Y, Wang YL, Qiu K, Cao YQ, Zhang FJ, Zhao HB, Liu XZ. YTHDF2 promotes temozolomide resistance in glioblastoma by activation of the Akt and NF-κB signalling pathways via inhibiting EPHB3 and TNFAIP3. Clin Transl Immunology. 2022 May 9;11(5):e1393. doi: 10.1002/cti2.1393. PMID: 35582627; PMCID: PMC9082891.

From:

<https://neurosurgerywiki.com/wiki/> - **Neurosurgery Wiki**

Permanent link:

<https://neurosurgerywiki.com/wiki/doku.php?id=ythdf2>

Last update: **2024/06/07 02:59**

