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Evidence of galectin-1 involvement in glioma chemoresistance

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Abstract

Glioblastomas (GBMs) are resistant to apoptosis but less so to autophagy; a fact that may at least partly explain the therapeutic benefits of the pro-autophagic drug temozolomide in the treatment of GBM patients. Galectin-1 (Gal1) whose expression is stimulated by hypoxia is a potent modulator of GBM cell migration and a pro-angiogenic molecule. Hypoxia is also known to confer cancer cells with resistance to chemotherapy and radiotherapy and to modulate the unfolded protein response (UPR) during endoplasmic reticulum (ER) stress. The present study investigates whether decreasing Gal1 expression (by means of a siRNA approach) in human Hs683 GBM cells increases their sensitivity to pro-autophagic or pro-apoptotic drugs. The data reveal that temozolomide, the standard treatment for glioma patients, increases Gal1 expression in Hs683 cells both *in vitro* and *in vivo*. However, reducing Gal1 expression in these cells by siRNA increases the anti-tumor effects of various chemotherapeutic agents, in particular temozolomide both *in vitro* and *in vivo*. This decrease in Gal1 expression in Hs683 cells does not induce apoptotic or autophagic features, but is found to modulate p53 transcriptional activity and decrease p53-targeted gene expression including DDIT3/GADD153/CHOP, DUSP5 ATF3 and GADD45A. The decrease in Gal1 expression also impairs the expression levels of seven other genes implicated in chemoresistance: ORP150, HERP, GRP78/Bip, TRA1, BNIP3L, GADD45B and CYR61, some of which are located in the ER and whose expression is also known to be modified by hypoxia. This novel facet of Gal1 involvement in glioblastoma biology may be amenable to therapeutic manipulation.

Keywords: Galectin-1; Chemoresistance; Glioma

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