mir 203

MicroRNA-203 (miR-203) has been shown to function as an important regulator of tumor progression at various stages.

Liu et al. investigated the effect of miR 203 expression and bufalin treatment on glioma cell proliferation and stem cell-like phenotypes.

They used cell viability assay, colony formation assay, cell apoptosis assay and neurosphere formation assay to dectect the treatment effect of bufalin on U251 and U87 cells. Cells were transfected with the miR-203 mimic without bufalin treatment or cells were transfected with anti-miR-203 under bufalin treatment, the above expreiments were repeated. RT-PCR was employed to quantify miR-203 expression. Western blot was performed to detect the stem cell-like (CSC) markers, OCT4 and SOX2. Luciferase activity assay was used to determine whether the SPARC is the target of miR-203.

Bufalin treatment inhibited cell proliferation, colony formation, and CSC phenotypes and increased cell apoptosis and expression of miR-203. Furthermore, overexpression of miR-203 led to similar outcomes as bufalin treatment with respect to the cell viability, colony formation, cell apoptosis and the phenotypes of glioma cells. While anti-miR-203 attenuated the inhibitory effects of bufalin as promoting cell proliferation, colony formation and CSC phenotyes and inhibiting cell apoptosis. In addition, we identified SPARC as a novel target gene of miR-203. CONCLUSIONS: These findings suggest that miR-203 plays an important role in bufalin's ability to inhibit the growth of glioma cells and the development of stem cell-like phenotypes [1)].

Miroshnikova et al., found that glioma aggression and patient prognosis correlate with HIF1A levels and the stiffness of a tenascin C (TNC)-enriched ECM. Gain- and loss-of-function xenograft manipulations demonstrated that a mutant IDH1 restricts glioma aggression by reducing HIF1α-dependent TNC expression to decrease ECM stiffness and mechanosignalling. Recurrent IDH1-mutant patient gliomas had a stiffer TNC-enriched ECM that the study attributed to reduced miR-203 suppression of HIF1α and TNC mediated via a tension-dependent positive feedback loop. The work suggests that elevated ECM stiffness can independently foster glioblastoma aggression and contribute to glioblastoma recurrence via bypassing the protective activity of IDH1 mutational status [2)].

1)
Liu T, Wu C, Weng G, Zhao Z, He X, Fu C, Sui Z, Huang SX. Bufalin Inhibits Cellular Proliferation and Cancer Stem Cell-Like Phenotypes via Upregulation of MiR-203 in Glioma. Cell Physiol Biochem. 2017 Nov 23;44(2):671-681. doi: 10.1159/000485279. [Epub ahead of print] PubMed PMID: 29169175.
2)
Miroshnikova YA, Mouw JK, Barnes JM, Pickup MW, Lakins JN, Kim Y, Lobo K, Persson AI, Reis GF, McKnight TR, Holland EC, Phillips JJ, Weaver VM. Tissue mechanics promote IDH1-dependent HIF1α-tenascin C feedback to regulate glioblastoma aggression. Nat Cell Biol. 2016 Nov 7. doi: 10.1038/ncb3429. [Epub ahead of print] PubMed PMID: 27820599.