Zhao et al. found that miR-524-3p and miR-524-5p were suppressed in the classical molecular subtype of glioblastoma (GBM) from Chinese Glioma Genome Atlas (CGGA) data, and the suppression was associated with EGFR overexpression and EGFRvIII mutation. These two miRNAs improved overall survival time of patients with glioma, and their overexpression could restrain glioma cell migration, proliferation, and cell cycle, and control tumor formation in vivo. Interestingly, both of the miRNAs had a synergistic inhibitory effect on glioma cells. Furthermore, we confirmed that EGFR amplification/EGFRvIII mutation can repress the expression of Pri-miR-524 by histone modification. MiR-524-3p and miR-524-5p inhibited TGF/β, Notch and the Hippo pathway by targeting Smad2, Hes1 and Tead1, respectively; these pathways repressed their common downstream transcription factor, C-myc. More interestingly, C-myc bound to the promoter region of EGFR/EGFRvIII and activated its expression. These findings indicate that miR-524 mediates the EGFR/EGFRvIII stimulating effect. It may serve as a potential therapeutic agent and classical-specific biomarker for the development of glioma [1)].

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Zhao K, Wang Q, Wang Y, Huang K, Yang C, Li Y, Yi K, Kang C. EGFR/c-myc axis regulates TGFβ/Hippo/Notch pathway via epigenetic silencing miR-524 in gliomas. Cancer Lett. 2017 Aug 1. pii: S0304-3835(17)30458-5. doi: 10.1016/j.canlet.2017.07.022. [Epub ahead of print] PubMed PMID: 28778566.