microRNA (miR)-328 has been reported to be implicated into tumorigenesis and tumor progression in human gliomas. However, there were controversial study results in relation to its expression pattern as well as functions in this disease. The aim of the current study was to determine the clinical significance of miR-328 expression in patients with gliomas and its effect in tumor cell malignant phenotypes.

Quantitative real-time PCR was performed to detect the expression levels of miR-328 in 116 glioma and 15 non-neoplastic brain tissues. Then, the correlations of miR-328 expression with selected clinicopathologic parameters and clinical outcome of glioma patients were statistically evaluated. Moreover, CCK-8 and transwell assays were performed to investigate the functions of miR-328 in cell proliferation, invasion and migration, respectively.

Compared to non-neoplastic brain tissues, the expression levels of miR-328 were significantly downregulated in glioma tissues ($p < 0.001$). In addition, miR-328 downregulation was significantly associated with WHO grade ($p < 0.001$) and Karnofsky performance status score ($p = 0.02$). Moreover, glioma patients with low miR-328 expression exhibited markedly shorter overall survival than those with high expression ($p < 0.001$). Furthermore, functional assays in vitro system demonstrated that enforced expression of miR-328 could notably attenuate cell proliferation, invasion and migration of two glioma cell lines, including U251 and U87.

The data offer the convincing evidence that loss of miR-328 expression may stimulate advanced tumor progression and adverse outcome via promoting cellular proliferation and invasion. We propose a tumor suppressive role of miR-328 and its potential therapeutic value in human glioma [1)].

1)
Yuan J, Zheng Z, Zheng Y, Lu X, Xu L, Lin L. microRNA-328 is a favorable prognostic marker in human glioma via suppressing invasive and proliferative phenotypes of malignant cells. Int J Neurosci. 2016 Feb;126(2):145-153. Epub 2015 Jul 20. PubMed PMID: 25562367.