

Despite the well-characterized [expression profile](#) of [miR-351](#) in the [nervous system](#), its molecular mechanisms in [glioma](#) still remain elusive.

Wu et al. intended to assess the regulatory function of [miR 351](#) on [nuclear apoptosis-inducing factor 1 \(NAIF1\)](#) and, thereby, modulation of [cancerous](#) behaviors of human [glioma cell lines](#). Two human glioma [cell lines](#) ([U87](#) and [U251](#)) and normal human [astroglia](#) (NHA) cell line were cultured. The [cell lines](#) were prepared and transfected with mimic, inhibitor, and negative controls (NCs) of [miR-351](#), then MTT and wound healing assays were performed. They extracted the total protein for [western blotting](#) assay and isolated the total [RNA](#) for real-time [PCR](#). The [miR-351](#) expression was significantly decreased in U87 and U251 cell lines compared with the NHA cell line ($P < 0.05$). NAIF1 expression was significantly higher in [glioma cell lines](#) compared with the NHA cell line ($P < 0.05$). Moreover, the NAIF1 expression showed a negative correlation with [miR-351](#) ($P = 0.005$, $r = -0.522$). [Apoptosis](#) was significantly decreased in both cell lines transfected with [miR-351](#) mimics compared with the NC group at 72 and 96 h after transfection ($P < 0.05$) and significantly increased in the transfected group with [miR-351](#) inhibitors compared with the NC group at 72 and 96 h after transfection ($P < 0.05$). According to our results, after 24 and 48 h, migration was increased in the mimic group compared with the [miR-351](#) NC group and decreased in the inhibitory group compared with the [miR-351](#) NC group in the U251 cell line. The findings provide theoretical evidence that [miR-351](#), which targets NAIF1, could be considered an important marker in the pathogenesis of [glioma](#). Furthermore, [miR-351](#) has valuable potential to serve as a new prognostic and diagnostic biomarker and could be considered a potential target for the treatment of this cancer in the near future ¹⁾.

¹⁾

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