A study investigated the activity of FBXL7 and miR-152-5p in glioma. Levels of microRNA-152-5p (miR-152-5p) and the transcript and protein of FBXL7 were assessed by real-time PCR and Western blotting, respectively. The migratory and invasive properties of cells were measured by Transwell migration and invasion assay and their viability were examined using CCK-8 assay. Further, the putative interaction between FBXL7 and miR-152-5p were analysed bioinformatically and by luciferase assay. The activities of FBXL7, TMZ and miR-152-5p were analysed in vivo singly or in combination, on mouse xenografts, in glioma tumorigenesis. The expression of FBXL7 in glioma tissue is significantly up-regulated, which is related to the poor prognosis and the grade of glioma. TMZ-induced cytotoxicity, proliferation, migration and invasion in glioma cells were impeded by the knock-down of FBXL7 or overexpressed miR-152-5p. Furthermore, the expression of miR-152-5p reduced remarkably in glioma cells and it exerted its activity through targeted FBXL7. Overexpression of miR-152-5p and knock-down of FBXL7 in glioma xenograft models enhanced TMZ-mediated anti-tumour effect and impeded tumour growth. Thus, the miR-152-5p suppressed the progression of glioma and associated tumorigenesis, targeted FBXL7 and increased the effect of TMZ-induced cytotoxicity in glioma cells, further enhancing our knowledge of FBXL7 activity in glioma [1)].

MiR-152 regulated cell proliferation and apoptosis of glioma mediated by Runx2, while the mechanism of down regulated miR-152 in glioma tissues and cells was its hypermethylation [2)].

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miR-152-3p can inhibit glioma cell proliferation and invasion activities by decreasing DNMT1. The restoration of miR-152-3p may have therapeutic application in the treatment of GBM [3)].

1)
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