MicroRNA-361-5p

MicroRNA (miR)-361-5p has been studied to suppress gliomas development. Based on that, an insight into the regulatory mechanism of miR-361-5p in gliomas was supplemented from E3 ubiquitin ligase component N-recognin 5 (UBR5)-mediated ubiquitination of ataxia-telangiectasia mutated interactor (ATMIN). miR-361-5p, ATMIN, and UBR5 levels were clinically analyzed in gliomas tissues, which were further validated in gliomas cell lines. Loss/gain-of-function method was applied to determine the roles of miR-361-5p and UBR5 in gliomas, as to cell viability, migration, invasion, colony formation ability, and apoptosis in vitro and tumorigenesis in vivo. The relationship between miR-361-5p and UBR5 was verified and the interaction between UBR5 and ATMIN was explored. It was detected that reduced miR-361-5p and ATMIN and enhanced UBR5 levels showed in gliomas. Elevating miR-361-5p was repressive in gliomas progression. UBR5 was directly targeted by miR-361-5p. UBR5 can ubiquitinate ATMIN. miR-361-5p suppressed gliomas by regulating UBR5-mediated ubiquitination of ATMIN. Downregulating UBR5 impeded gliomas tumor growth in vivo. Upregulating miR-361-5p targets UBR5 to promote ATMIN protein expression, thus to recline the malignant phenotype of gliomas cells [1)].

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Jia J, Ouyang Z, Wang M, Ma W, Liu M, Zhang M, Yu M. MicroRNA-361-5p slows down gliomas development through regulating UBR5 to elevate ATMIN protein expression. Cell Death Dis. 2021 Jul 28;12(8):746. doi: 10.1038/s41419-021-04010-1. PMID: 34321465.