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MiR-125b is critical for the suppression of human U251 glioma stem cell proliferation.

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Stem cells are unique in their ability to self-renew and maintain tissue homeostasis by differentiating into different cell types to replace aged or damaged cells. The key characteristic of the stem cell is its capacity to divide for long periods of time. MicroRNAs (miRNAs) are small noncoding RNA molecules that regulate protein expression by cleaving or repressing the translation of target mRNAs. miR-125b, one of neuronal miRNAs, recently was found to be necessary for stem cell fission to bypass the normal G1/S checkpoint and make stem cells insensitive to chemotherapy signals, which normally stop the cell cycle at the G1/S transition. Given the insensitivity of gliomas to chemotherapy and the hypothesis that glioma stem cells cause resistance to drug therapy, exploring the functions and mechanisms of miR-125b in glioma stem cells would be valuable. In this study, we found that miR-125b was downregulated in human U251 glioma stem cells, therefore suggesting that its upregulation can lead to the growth inhibition of U251 glioma stem cells in vitro. Further research on the mechanism demonstrated that inhibition of miR-125b-induced U251 glioma stem cell proliferation was due to cell cycle arrest at the G1/S transition and involved the cell cycle regulated proteins CDK6 and CDC25A; miR-125b overexpression decreased CDK6 and CDC25A expression. These findings underscore the potential of miR-125b to regulate the proliferation of U251 glioma stem cells through the cell cycle regulated proteins CDK6 and CDC25A.

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