



**Citation:**

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2d). I.e., in glioblastoma, miR-21 had the opposite effect on differentiation in NSPC. In the end, we demonstrated that miR-21 could promote the differentiation of NSPC.

### MiR-21 inhibits the proliferation of NSPCs

To determine the effect of miR-21 on the proliferation of NSPC, we first performed cell viability assay using MTT assay. Single NSPCs were cultured in 1-D-1 in the presence of glioblastoma cells, 24-h cell viability assay was performed. As a result, we observed that miR-21 significantly reduced the proliferation of NSPC, and the cell viability was significantly inhibited by MTT assay. We observed that knockdown of miR-21 could enhance the cell viability of NSPC. Obviously, the proliferation of NSPCs was significantly inhibited by miR-21 at 12 h, and the cell viability was significantly reduced, each time for 24 h. In contrast, miR-21 significantly enhanced the cell viability of NSPC. Cell viability was significantly increased by miR-21 for the first 24 h, and the cell viability was significantly increased (Figure 3a). Therefore, we infer that miR-21 promotes NSPC proliferation, while miR-21 inhibits the differentiation of NSPC. In addition, we observed that miR-21 significantly enhanced the cell viability of NSPC. In addition, we observed that miR-21 significantly enhanced the cell viability of NSPC. In addition, we observed that miR-21 significantly enhanced the cell viability of NSPC.

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### MiR-21 in uence on the activation of AKT and GSK-3 signaling pathway

To determine the effect of miR-21 on the activation of AKT and GSK-3 signaling pathway, we first performed cell viability assay using MTT assay. Single NSPCs were cultured in 1-D-1 in the presence of glioblastoma cells, 24-h cell viability assay was performed. As a result, we observed that miR-21 significantly reduced the proliferation of NSPC, and the cell viability was significantly inhibited by MTT assay. We observed that knockdown of miR-21 could enhance the cell viability of NSPC. Obviously, the proliferation of NSPCs was significantly inhibited by miR-21 at 12 h, and the cell viability was significantly reduced, each time for 24 h. In contrast, miR-21 significantly enhanced the cell viability of NSPC. Cell viability was significantly increased by miR-21 for the first 24 h, and the cell viability was significantly increased (Figure 3a). Therefore, we infer that miR-21 promotes NSPC proliferation, while miR-21 inhibits the differentiation of NSPC.



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