

Correction: MiR-124 acts as a target for Alzheimer's disease by regulating BACE1

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This article has been corrected: The correct Materials and Methods and Figure 2 are given below:
The authors declare that these corrections do not change the results or conclusions of this paper.

Luciferase reporting assay

The 3' UTR of BACE1 and the CMV promoter were amplified from human chromosomal DNA and pcDNA3.1 (+) and cloned into the pGL3-luciferase basic vector (Promega, Madison, WI, USA). Sequences of primers and cloning strategy are available on request. For the luciferase assays, 50 nM of miR-124 mimics or scrambled RNA were co-transfected with the reporter vector and the Renilla control vector (Promega, Madison, WI, USA) into the HEK293 cells by Lipofectamine 2000 (Invitrogen, Carlsbad, CA, USA). 24 h post transfection, the measurements were performed using the Dual luciferase re-reporter assay kit (Promega, Madison, WI, USA). Or the HEK293 cells post the transfection for 24 h was lysed for western blot analysis.

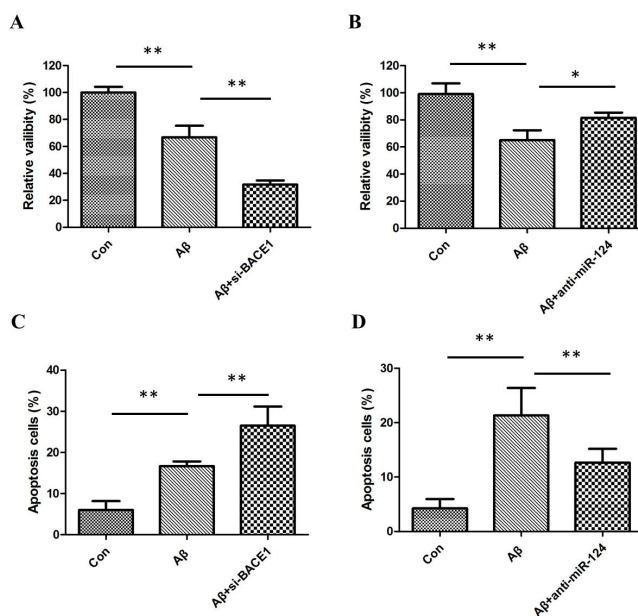


Figure 2: (A) MTT assay results showed that A β inhibited the viability of SH-SY5Y cells and downregulation of BACE1 enhanced the inhibitory effects of A β ; (B) downregulation of miR-124 relieved A β -induced viability inhibition of SH-SY5Y cells; (C) flow cytometric analysis results showed that A β -induced apoptosis of SH-SY5Y cells and downregulation of BACE1 enhanced the induced effects of A β ; (D) downregulation of miR-124 decreased apoptosis of SH-SY5Y cells in the presence of A β .

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