Correction

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-porter assay kit (Promega, Madison, WI, USA). Or the HEK293 cells post the transfection for 24 h was lyzed for western blot analysis.

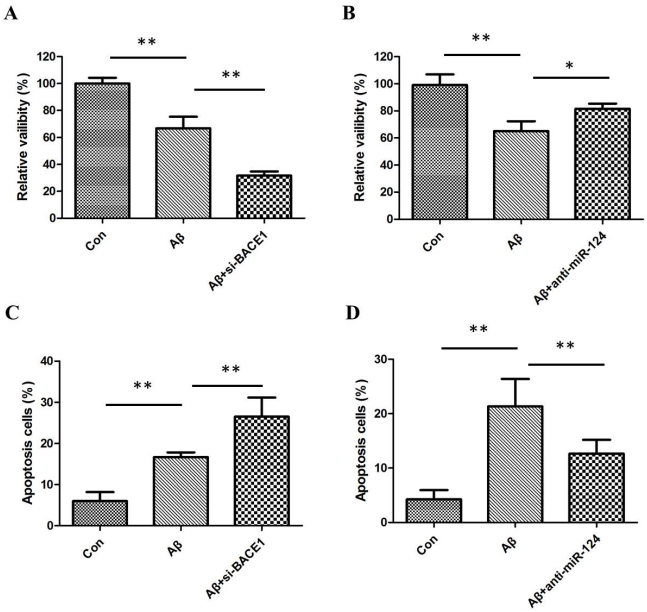

Figure 2: (**A**) MTT assay results showed that Aβ inhibited the viability of SH-SY5Y cells and downregulation of BACE1 enhanced the inhibitory effects of Aβ; (**B**) downregulation of miR-124 relieved Aβ-induced viability inhibition of SH-SY5Y cells; (**C**) flow cytometric analysis results showed that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

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