Electronic Supplementary Information (ESI) for Nanoscale

Copper inducing Aβ42 rather than Aβ40 nanoscale oligomer formation is the key process for Aβ neurotoxicity

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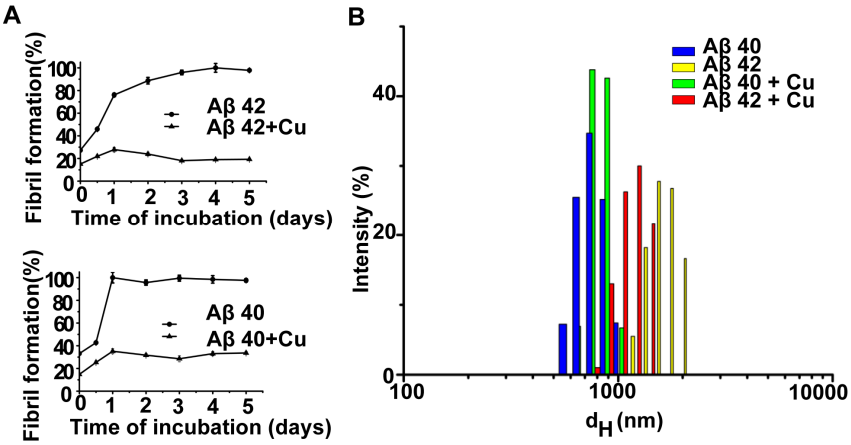

Fig. S1 Copper modulated the aggregation of Aβ42 and Aβ40. (A) Effects of Cu(II) on Aβ fibril formation assessed by the ThT fluorescence assay for 5 days. Aβ was incubated at 10 μM alone or with 10 μM Cu(II). The data are shown as means±SD; n=3. (B) DLS analysis of 10 μM Aβ42 or Aβ40 incubated alone or with 10μM Cu(II) for 24 hours at 37 °C. Different columns show the evolution of the multimodal size distribution peaks.

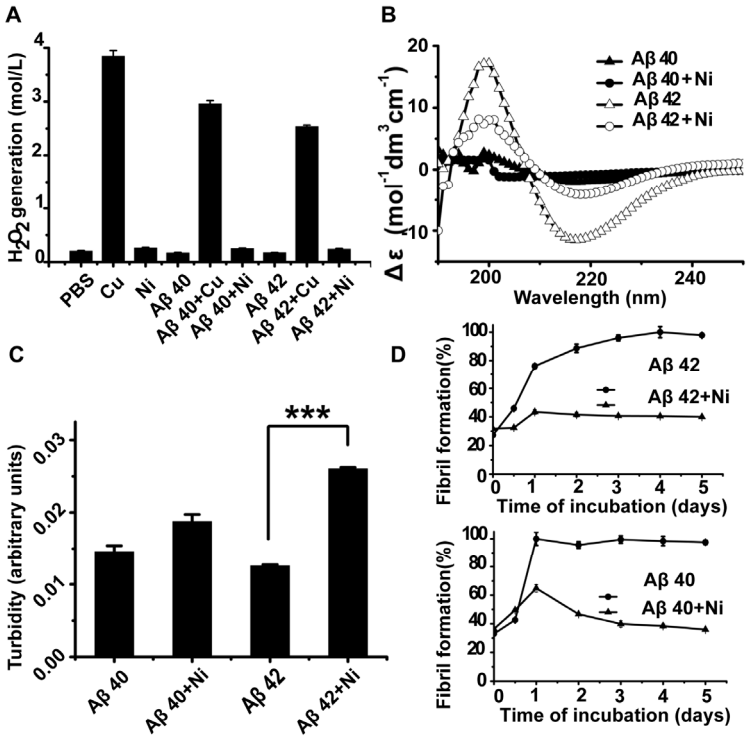

Fig. S2 Nickel did not produce H_2O_2, but recurred copper's effects on Aβ42 and Aβ40 conformation and aggregation. (A) The H_2DCF-DA test for H_2O_2 generation was performed in solutions of 10 μM Cu(II), Ni(II) or 10 μM Aβ alone, or 10 μM Cu(II) or Ni(II) plus 10 μM Aβ with 5 mM reducing agent dopamine, 100 mM deacetylated H_2DCF, and 1mM horseradish peroxidase. (B) CD spectra of Ni(II)-induced Aβ42 and Aβ40 conformation changes. Curves are against blank and repeated for 5 times. 20 μM Aβ40 or Aβ42 was incubated at pH 7.4 at 37 °C for 1 hour with or without 20 μM Ni(II). (C) Turbidity measurements of 10 μM Aβ incubated with or without 10 μM Ni(II) for 3 days. The data are shown as means±SD; n=3. ***: $p < 0.0005$ (by two-tailed Student's T-test). (D) Effects of Ni(II) on Aβ fibril formation assessed by the ThT fluorescence assay for 5 days. Aβ was incubated at 10 μM alone or with 10 μM Ni(II). The data are shown as means±SD; n=3. (B) (C) (D) experiments were carried out at the same time with corresponding experiments of copper in Figure 3.