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Author Correction: Glycosylation Significantly Inhibits the Aggregation of Human Prion Protein and Decreases Its Cytotoxicity

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This Article contains an error in the order of the Figures. Figures 8 and 9 were published as Figures 9 and 8 respectively. The correct Figures 8 and 9 appear below as Figures 1 and 2. The Figure legends are correct.

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Figure 1. N-linked glycosylation deficiency enhances PrP toxicity in RK13 cells induced by the toxic prion peptide PrP 106–126. RK13 cells stably expressing wild-type PrP (a), V180I (b), N197D (c), or the double mutant N181D/N197D (d) were cultured for 3 days and incubated with 60 μ M PrP 106–126 for 2 days. The percentage of apoptotic cells was determined by flow cytometry as described in the legend of Fig. 7.

Figure 2. N-linked glycosylation deficiency enhances PrP toxicity in SH-SY5Y cells induced by the toxic prion peptide PrP 106–126. SH-SY5Y cells transiently expressing wild-type PrP (a), V180I (b), N197D (c), or the double mutant N181D/N197D (d) were cultured for 2 days and incubated with 60 μ M PrP 106–126 for 2 days. The percentage of apoptotic cells was determined by flow cytometry as described in the legend of Fig. 7.

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