FK506 Reduces Amyloid Plaque Burden and Induces MMP-9 in AβPP/PS1 Double Transgenic Mice

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Supplementary Figure S1. Mouse body weight monitoring during experimental period. Mice bodyweights were measured once a week. There was no significant body weight change during experimental periods (7 weeks) in AβPP/PS1 mice (left panels), wild type littermate mice (right panels), vehicle (upper panels) or FK506-injected mice (lower panels).

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Supplementary Figure S2. Aβ1–40 levels in FK506-injected mouse brains. Human Aβ1–40 levels are decreased in FK506 treated mice brain, although not reaching statistical significance (Veh; Vehicle injected, FK506; FK506 injected).

Supplementary Figure S3. AβPP/PS mice brains exhibit amyloid plaques and GFAP positive astrocytes (middle and right panels). But both of amyloid plaques (green, arrow) and GFAP-positive Immunostaining analysis of Aβ and GFAP astrocytes (red) were reduced in FK506 injected mouse brains (right panel) compared to vehicle injected mice (middle panel). Wild type litter mate mouse brain showed no amyloid plaque staining (left panel).
Supplementary Figure S4. Correlation analysis among CaN activity, MMP-9, synaptophysin, PSD-95, GFAP, and plaque burden. There is significant correlation between GFAP and amyloid load. For MMP-9 level and amyloid load, it shows a tendency to negative correlation, even though it is not statistically significant. We did several correlation analyses for various pairs. Briefly, when calcineurin activity was lowered by FK506, MMP-9, synaptophysin, PSD-95 levels were increased, while GFAP level and plaque burdens were decreased when CaN activity was low.