

SUPPLEMENTAL FIGURE LEGENDS

Supplemental Figure 1: Changes in motor performance during the immunization

The line graph represents the changes of motor performance in control, sham-treated or active immunization group over the 3 months of treatment period.

Supplemental Figure 2: Histopathological changes of α B-crystallin and MHC class I in skeletal muscle fibers.

(a) Immunofluorescent staining of α B-crystallin in skeletal muscle fibers shows upregulation of the protein in untreated and sham-treated muscle, whereas the upregulation of α B-crystallin is markedly reduced in actively immunized mice. Quantitative measurement of the steady-state levels of α B-crystallin by western blot confirms a reduced level of the protein in the skeletal muscle of actively immunized mice. $p < 0.05$ compared to untreated or sham group. (b) Immunofluorescent staining of MHC class I reveals no obvious difference among all groups.

Supplemental Figure 3: Correlation between plasma anti-A β antibody concentration and unbound plasma A β levels in actively immunized mice

In the actively immunized mice, decreased unbound (or free) plasma A β levels are found with increased plasma anti-A β antibody levels, suggesting good affinity of endogenously generated antibody on A β following active immunization.

Supplemental Figure 4: A11-positive oligomers colocalize with A β antibody

(a) Double immunofluorescent staining of skeletal muscle of MCK-APP/PS1 mice shows that A11-positive immunoreactivity colocalizes or is in the vicinity of 6E10-positive amyloid deposits. Some intracellular amyloid deposits are not immunoreactive to the A11 antibody. TOTO-3 was used as a nuclear counter-stain. (b) Immunoprecipitation with A11 or 6E10 antibody further confirms A β oligomers in skeletal muscle of MCK-APP/PS1 mice. Note that active immunization group had reduced A β oligomers in skeletal muscle.

Supplemental Figure 5: Analysis of oligomeric and fibrillar A β preparation and its cytotoxicity on C2C12 cells

(a) The assembly state of A β in the solution following oligomer or fibrillar preparation was determined by dot blot analysis using 6E10 or A11 antibody. (b) Representative phase-contrast images of undifferentiated C2C12 cells exposed to A β oligomers for 24 hrs with or without anti-A β antibodies purified from immunized mice. (c) Representative phase-contrast images of differentiated C2C12 cells exposed to A β oligomers for 24 hrs.