Supplementary Data

Peripheral Administration of Antisense Oligonucleotides Targeting the Amyloid-β Protein Precursor Reverses AβPP and LRP-1 Overexpression in the Aged SAMP8 Mouse Brain

Michelle A. Erickson^{a,b}, Michael L. Niehoff^{c,d}, Susan A. Farr^{c,d}, John E. Morley^{c,d}, Lucy A. Dillman^a, Kristin M. Lynch^a and William A. Banks^{a,*}

^aGRECC, Veterans Affairs Puget Sound Health Care System and University of Washington School of Medicine, Division of Gerontology and Geriatric Medicine, Department of Internal Medicine, Seattle, WA, USA

^bSaint Louis University, Department of Pharmacology and Physiology, St. Louis, MO, USA

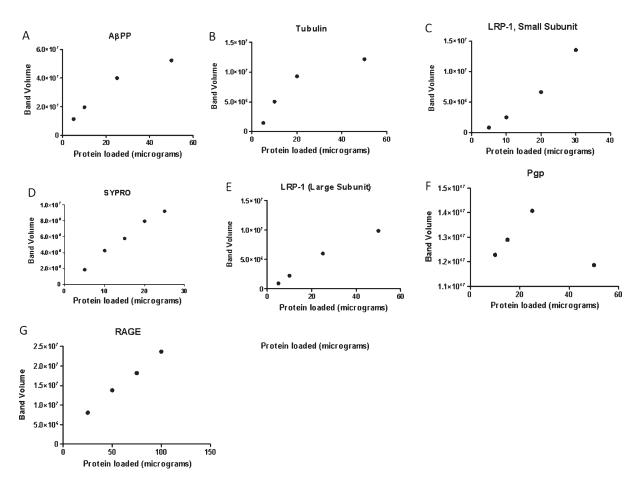
^cVeterans Affairs Medical Center-St. Louis, St. Louis, MO, USA

^dSaint Louis University School of Medicine, Division of Geriatrics, Department of Internal Medicine, St. Louis, MO, USA

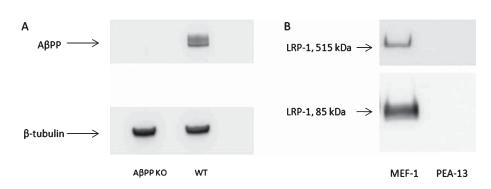
Handling Associate Editor: D. Allan Butterfield

Accepted 4 November 2011

^{*}Correspondence to: William A. Banks, Bldg. 1, Rm. 810A, 1660 Columbian Way, Seattle, WA 98108, USA. Tel.: +1 206 764 2701; Fax: +1 206 764 2569; E-mail: bankswa@slu.edu.



Supplementary Figure 1. Confirmation of immunoblot signal linearity for all antibodies and loading controls used in this study. Band volumes were plotted as a function of protein loading amount per well for A) A β PP, B) β -tubulin, C) LRP-1 small subunit, D) SYPRO, E) LRP-1 large subunit, F) Pgp, and G) RAGE.



Supplementary Figure 2. Confirmation of signal specificity for antibodies. A) A β PP signal in brain tissue of knockout mice and wild-type controls, B) LRP-1 signal in knockout mouse embryonic fibroblast (PEA-13) cell lines and wild-type (MEF-1) control.