## SUPPORTING INFORMATION

## Design, characterization, and use of a novel amyloid β-protein control for assembly, neurotoxicity, and gene expression studies

Ghiam Yamin<sup>a, b</sup>, Giovanni Coppola<sup>b, c</sup>, and David B. Teplow<sup>b\*</sup>

**Table S1.** Microsoft Excel file containing: (1) Differential analysis for all the comparisons reported in the main text (Figure 6 and 7A); (2) gene ontology annotation for each of the comparisons (Figure 7B); (3) WGCNA gene list and module membership (Figure S2); and (4) gene ontology annotation of yellow and orange modules (Figures 8 and 9). Raw and normalized gene expression data have been deposited within the Gene Expression Omnibus (GEO) repository (<u>www.ncbi.nlm.nih.gov/geo</u>), accession number: GSE85952.

**Table S2.** Commercially available scrambled Aβ peptides.

Fig. S1. Phase-contrast microscopy image of primary hippocampal neurons after 7 days in culture.Red scale bar is 91 μm.

**Fig. S2.** Relationship between module eigengene (first principal component, corresponding to the weighted summation of expression across all the probes included in a given module) and treatment (A $\beta$  assembly type). The orange and yellow modules, which are independent from one another, showed the greatest correlation with fibrillar A $\beta$ 40 and A $\beta$ 42 treatment among the total of 13 modules defined. Cont: control; S40 and S42: sA $\beta$ 40 and sA $\beta$ 42; T40 and T42: LMW A $\beta$ 40 and LMW A $\beta$ 42, XL40 and XL42: cross-linked A $\beta$ 40, cross-linked A $\beta$ 40 and A $\beta$ 42; F40 and F42: fibrillar A $\beta$ 40 and fibrillar A $\beta$ 42, respectively.