Supplemental Figure 1. Characterization of the Aβ species used in the experiments.
A. ThT assay detected no fibrillar species of $A\beta_{(1-40)}$ at 5 μM concentration. However, 5 μM $A\beta_{(1-42)}$ had fibrillar species present, even after ultracentrifugation, suggesting a high-propensity for aggregation. **B.** The oligeomeric species were separated by size exclusion chromatography and analyzed by western blot analysis of the 50 fractions eluted from the column. The oligomeric size was approximated by co-elution with markers of the known size, and by dividing the molecular weight of the oligomer by the size of the monomeric Aβ peptide.

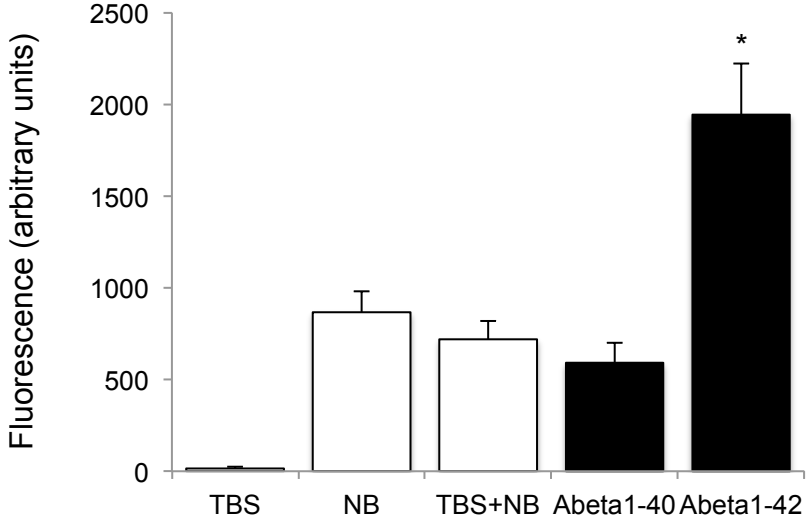
Supplemental Figure 2. Four point dose-response curves for selected compounds.
Neurons were treated with Aβ in the presence of 2.5 nM, 25 nM, 250 nM, and 2.5 μM of the indicated compound. These data were used to calculate the an approximate EC50 values in Fig. 2.

Supplemental Figure 3. COX1 and COX2 inhibitors do not alter neurite length. Neurons treated with 10 μM of the COX1 inhibitor FR122047, or the COX2 inhibitor, CAY10404, did not significantly alter neurite length in the absence of $A\beta_{(1-40)}$.

Supplemental Figure 4. Inhibition of $A\beta_{(1-40)}$ and $A\beta_{(1-42)}$ -induced neurite loss by NSAIDs.
A. Representative images $A\beta_{(1-42)}$-induced neurite loss at 1 μM and 5 μM concentrations. **B.** Similar to $A\beta_{(1-40)}$, $A\beta_{(1-42)}$-elicited reduction of neurite length was markedly attenuated in the presence of 10 μM of various NSAIDs (ibuprofen, Ibu; naproxen, Napr; nabumetone, Nabu).

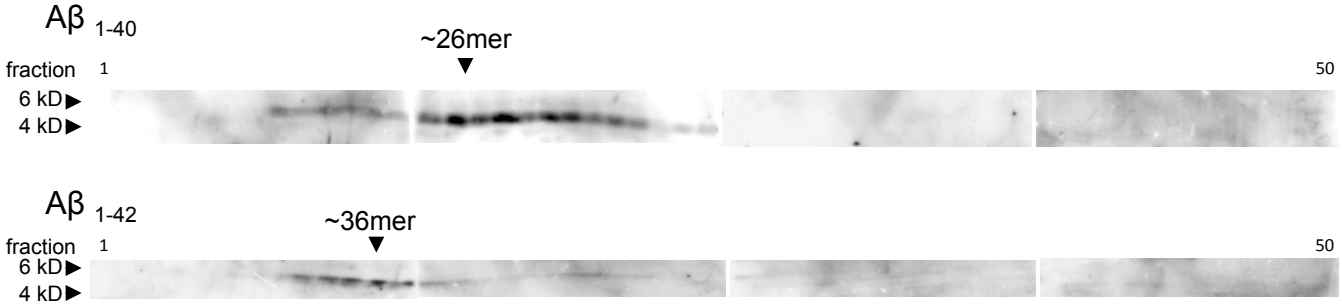
A

ThT assay

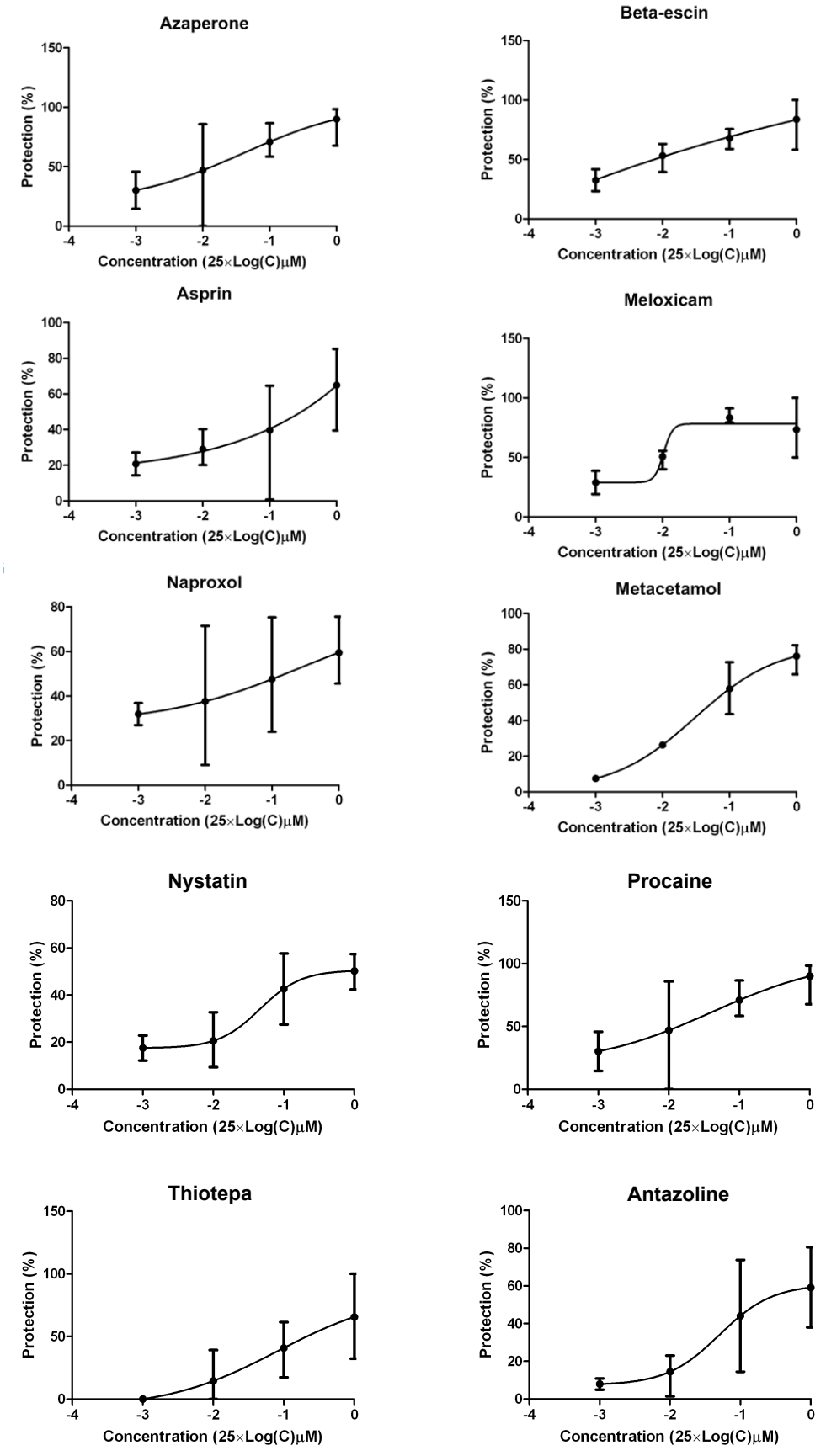


B

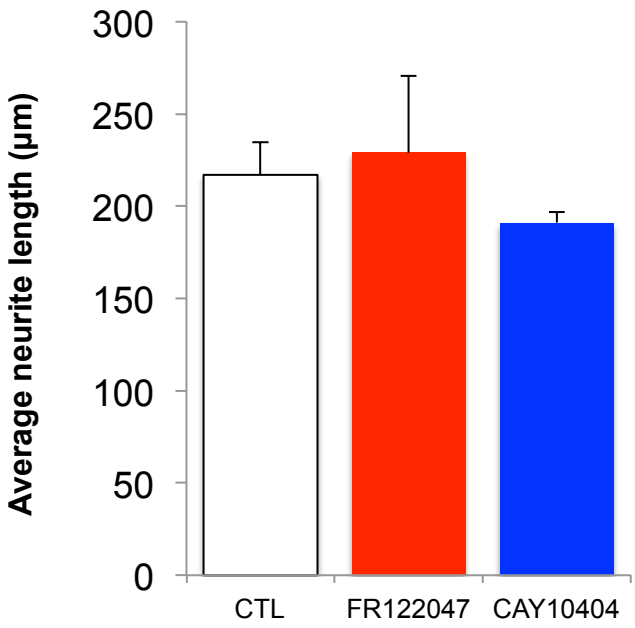
Size-exclusion chromatography



Suppl. Figure 1

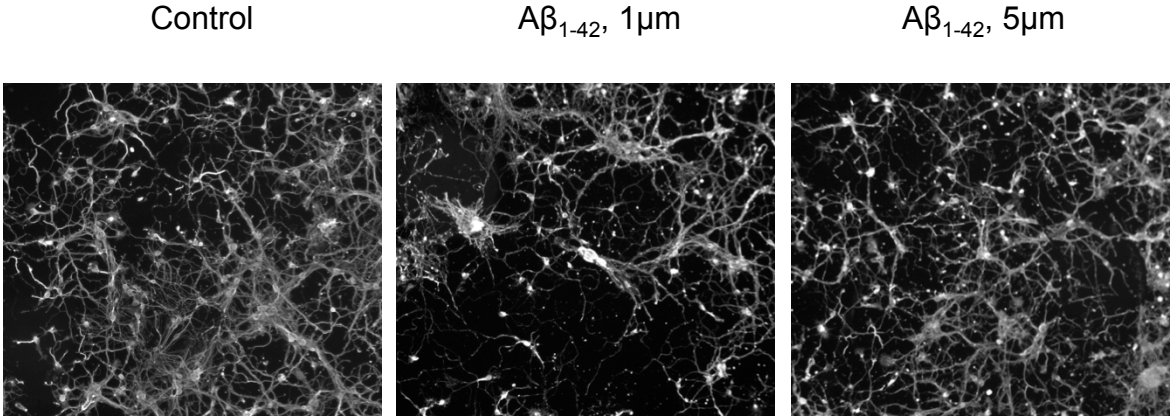


Suppl. Figure 2

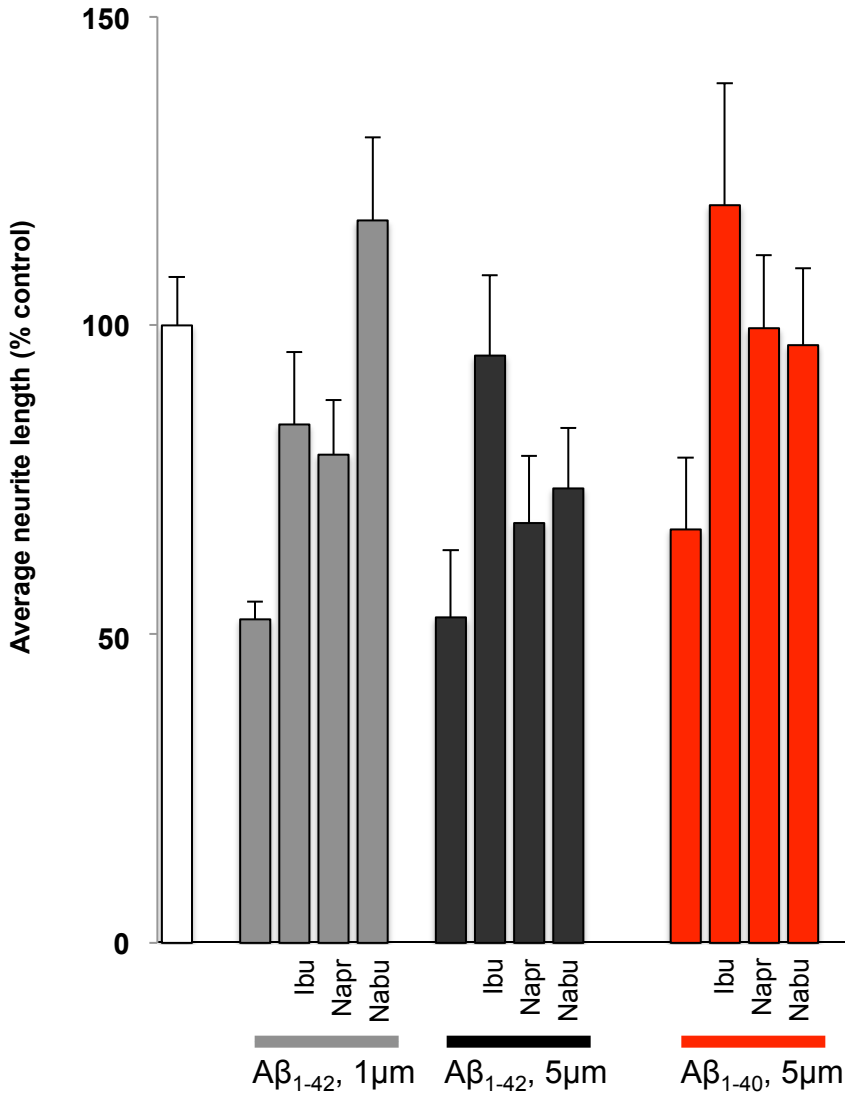


Suppl. Figure 3

A



B



Suppl. Figure 4