

Supplementary material

Black tea theaflavins inhibit formation of toxic amyloid-beta and alpha-synuclein fibrils

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Supplementary Figures

Supplementary Figure 1: A β peptide binding. (A) A β peptide sequences in SPOT peptide arrays.

Peptide numbers represent the starting amino acid of 10-mer peptides. Each peptide sequence is shifted by one amino acid towards the C-terminus of the A β 42 sequence. (B) Incubation of a 10-mer peptide array with TF3, EGCG, or CR (300 μ M). Compound binding to peptides was quantified colorimetrically directly (CR) or after NBT staining (EGCG, TF3). Two potential binding regions in A β peptides were detected (peptides 8-16 and 23-28). (C) Binding of EGCG, TF3 and CR to A β 16-22 and scrambled A β 16-22 was visualized colorimetrically. EGCG and TF3 were stained by NBT.

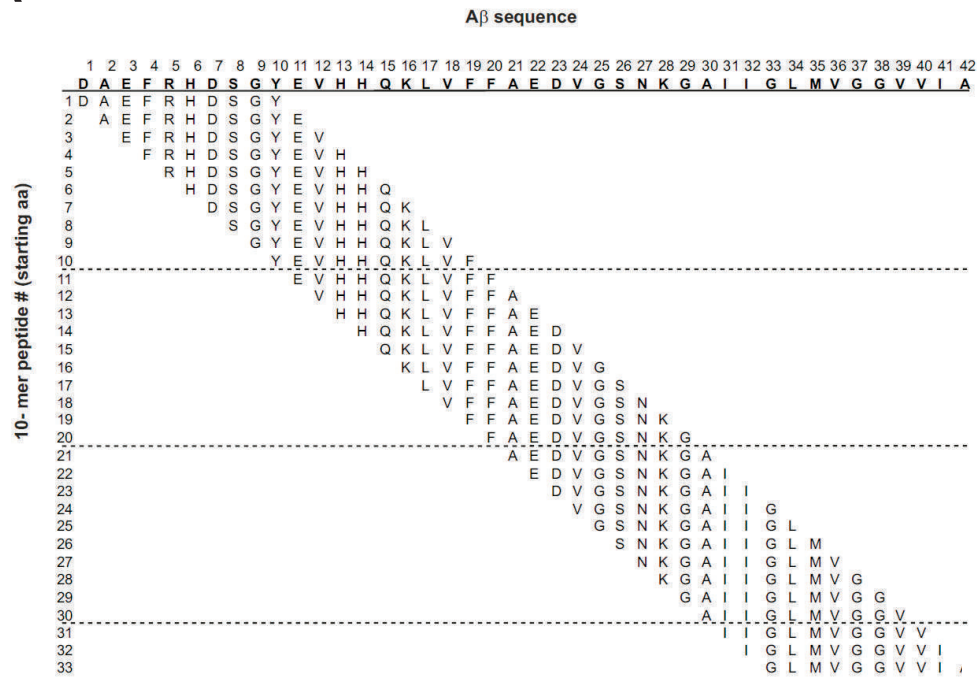
Supplementary Figure 2: Effect of filtration and BSA on TF3. (A) ThT fluorescence measurement of

A β ₄₀ (15 μ M) aggregation kinetics in the presence of TF3 (30 μ M) and BSA (30 μ M). (B) A β ₄₂ (15 μ M) aggregation kinetics in the presence of TF3 (30 μ M) and BSA (30 μ M). (C) A β ₄₂ (15 μ M) aggregation kinetics in the presence of TF3 (15 μ M) that had been pre-filtered through a 3 kD cut-off membrane filter.

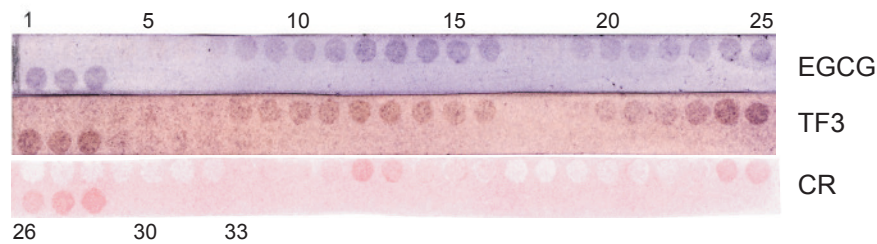
Supplementary Figure 3: EGCG and theaflavins induce SDS-resistant α S aggregates.

α S (50 μ M) was incubated for 26 h at 37°C in SP-buffer in the presence of equimolar concentrations of TF1, TF2a, TF2b, TF3, TF3 + DTT (1 mM), EGCG and EGCG + DTT (1 mM) and analyzed by FRA using anti- α S antibody. All compounds induced the formation of SDS-stable aggregates, even under reducing conditions.

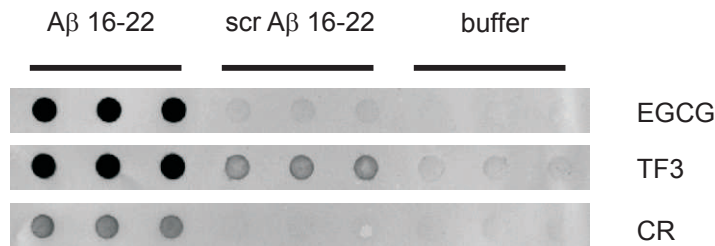
A



B

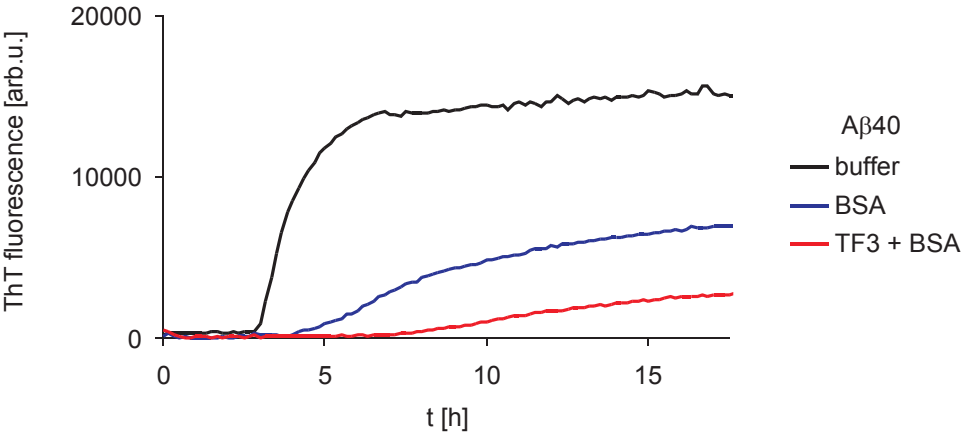


C

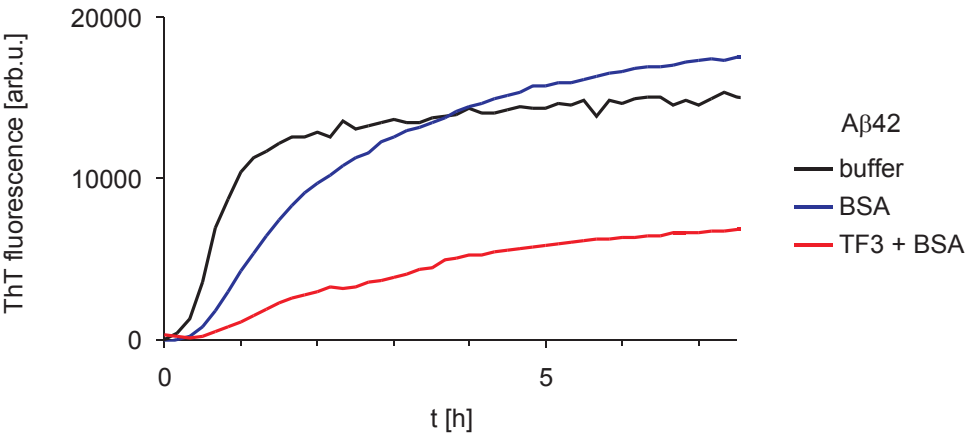


Supplementary Fig S2

A



B



C

