Table S1. Excitation and emission maxima of the LCOs free in solution (PBS) or bound to recombinant Aβ 1-42 fibrils, Aβ plaques or NFTs.

LCO	Excitation maximum, Exc_{max} (nm)	Emission maximum, Em_{max} (nm)	Exc_{max} bound to Aβ1-42 fibrils (nm)	Em_{max} bound to Aβ1-42 fibrils (nm)	Em_{max} bound to Aβ plaques (nm)	Em_{max} bound to NFTs (nm)
t-HTAA	373	502	418*,437	488	488	488
q-HTAA	374	503	416*,437	489	489	489
q-FTAA	373	504	402,430*,452	505	505	505
p-HTAA	386	520	418,446*	490,523	494,531	497,531
p-FTAA	403	529	429,458*,486	506, 541	517,553	517,553
hx-HTAA	418	526	468*,498	521, 558	521,558	521,561
hx-FTAA	421	528	453,482*,512	535, 574	538,581	542,581
h-HTAA	422	542	455,485*,517	538, 578	545,586	545,586
h-FTAA	426	572	460,489*,521	547, 585	557,594	561,599

*Main excitation peak

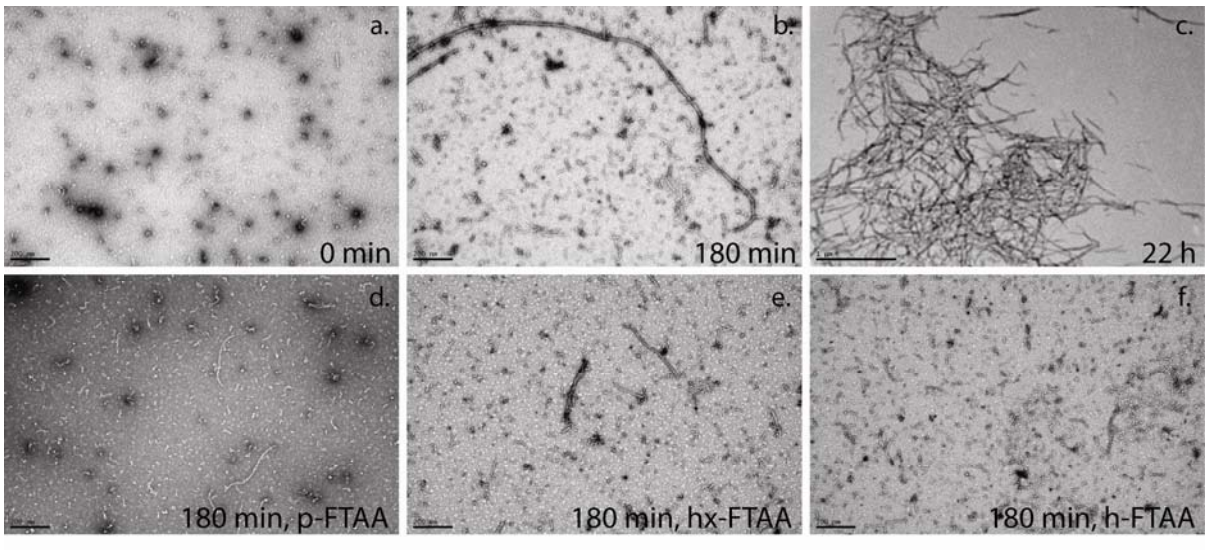

Fig. S1 Transmission electron microscopy (TEM) images of recombinant Aβ 1-42 that has been allowed to aggregate for 0 min, 180 min or 22 h in absence of LCOs (a, b, c) or for 180 min in presence of p-FTAA (d), hx-FTAA (e) or h-FTAA (f). Scale bars represent 200 nm (a, b, d, e, f) or 1 μm (c).

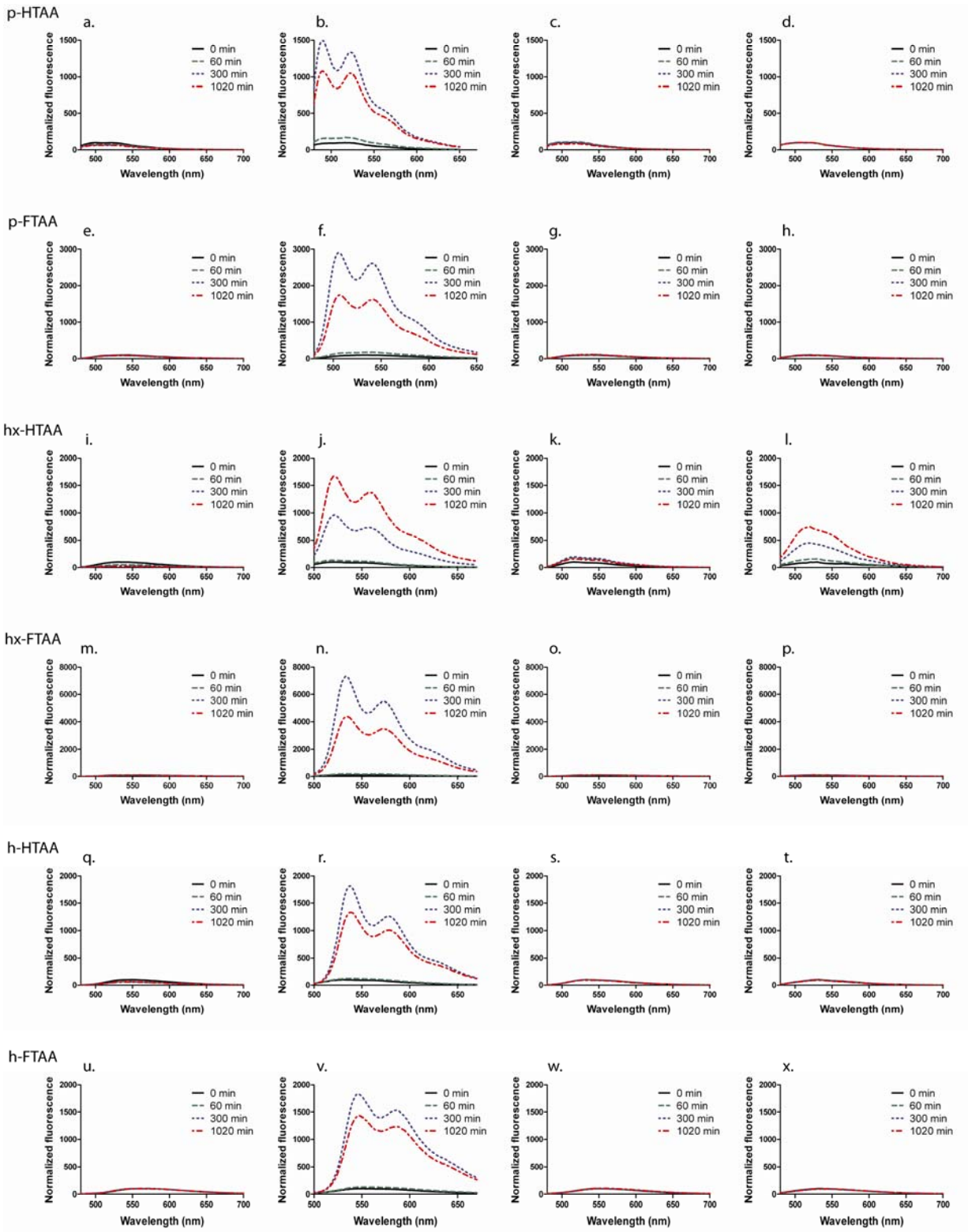

Fig S2. The emission spectrum of the indicated LCO in PBS (a, e, i, m, q, u) or mixed with 10 μM Aβ1-42 (b, f, j, n, r, v), 100 μM bovine insulin (c, g, k, o, s, w) or 10 μM bovine serum albumin (d, h, l, p, t, x) in PBS incubated at 37°C for 0 minutes (black solid line), 60 minutes (green dashed line), 300 minutes (blue dotted line) or 1020 minutes (red dot-dashed line). All probes were excited at 450 nm.

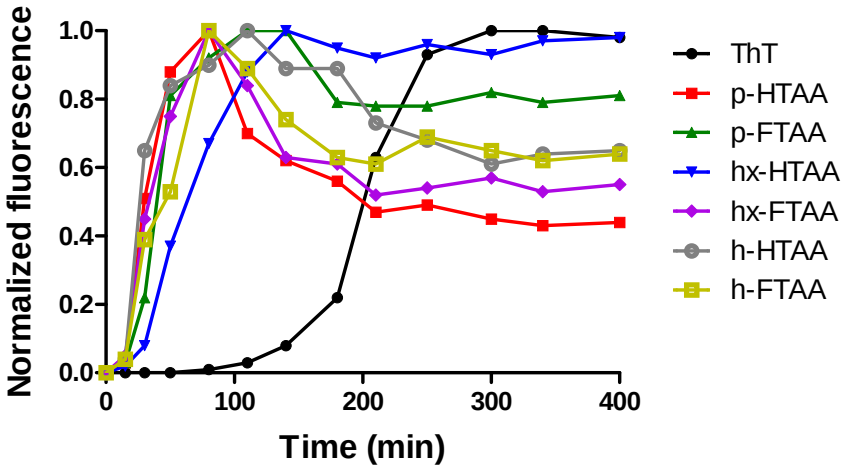

Fig. S3 The fibrillation kinetics of insulin monitored with ThT, p-HTAA, p-FTAA, hx-HTAA, hx-FTAA, h-HTAA or h-FTAA fluorescence. All LCOs detect early fibrillar species not detected by ThT.

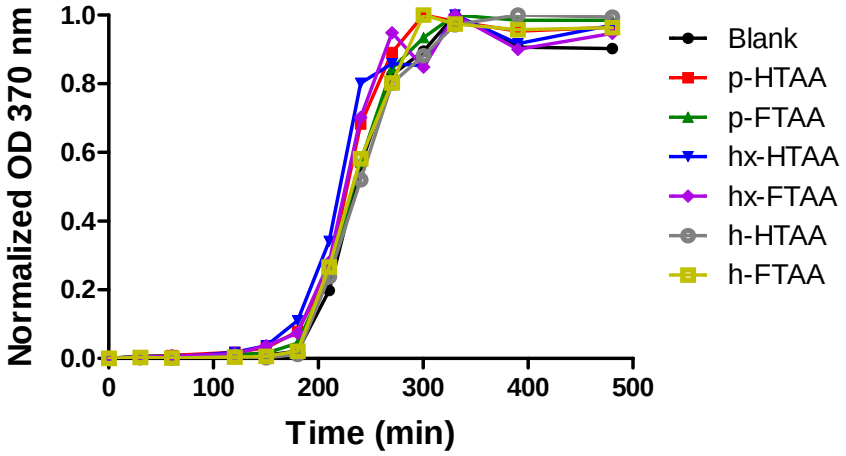

Fig. S4 The fibrillation of insulin in the absence or presence of the indicated LCOs followed with turbidity measurements. Samples containing LCOs displayed the same optical density as the blank sample verifying that none of the probes accelerated the fibrillation rate of insulin.

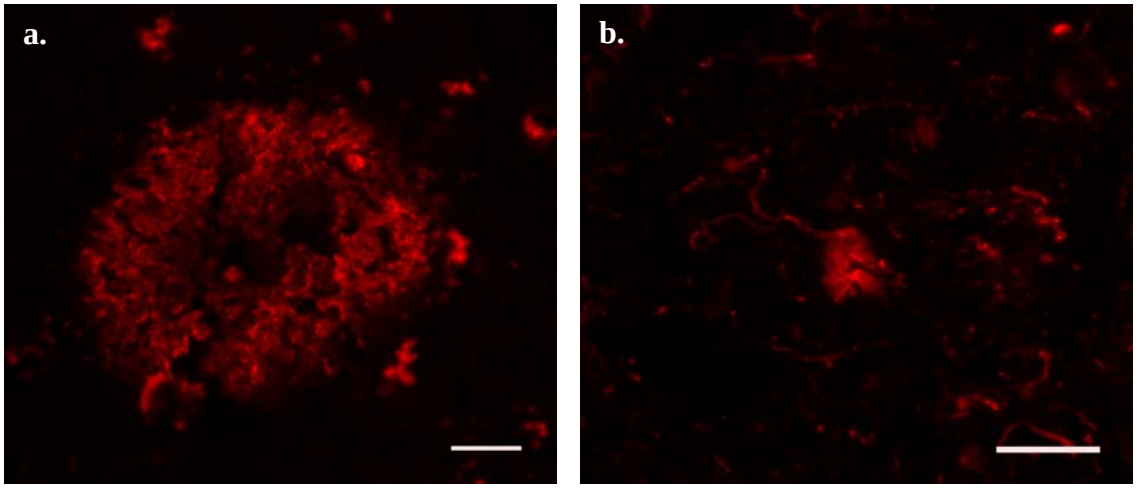

Fig. S5 Fluorescence images of Alzheimer´s disease brain tissue stained with antibody against Aβ (a) or phosphorylated tau (b). Scale bars represent 20 μm.

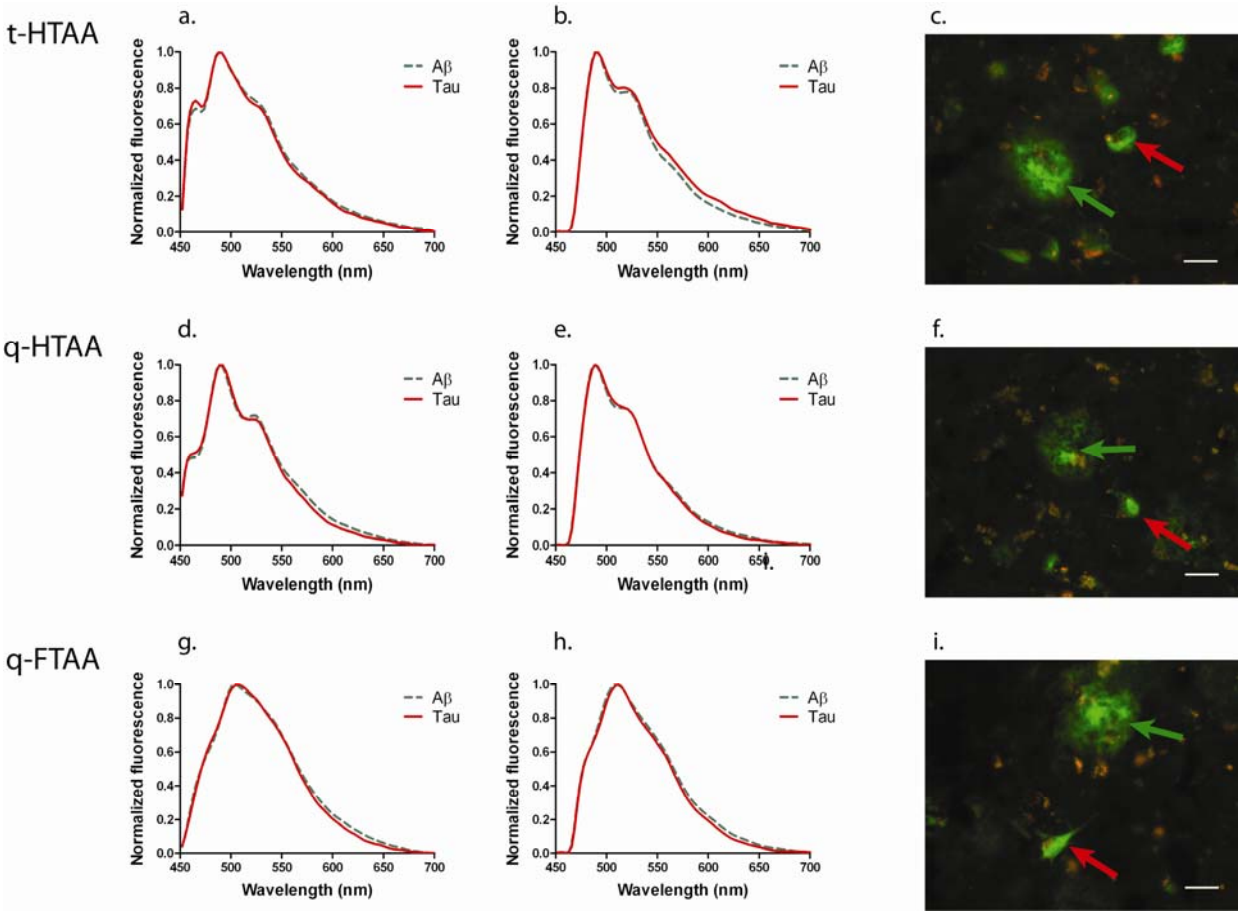

Fig. S6 Emission spectra and fluorescence images of t-HTAA, q-HTAA and q-FTAA bound to Aβ plaques and NFTs in brain tissue from patient with Alzheimer´s disease. (a, d, g) The emission spectra of the indicated LCO bound to Aβ plaques (green dashed line) or NFTs (red solid line) when excited at 405 nm. (b, e, h) The emission spectra of the indicated LCO bound to Aβ plaques (green dashed line) or NFTs (red solid line) when excited at 436 nm. (c, f, i) Fluorescence images of human AD brain section stained with t-HTAA (c), q-HTAA (f) or q-FTAA (i). The tetrameric probes can not be used to spectrally discriminate between Aβ plaques (green arrow) or aggregated tau (red arrow).