Supporting information

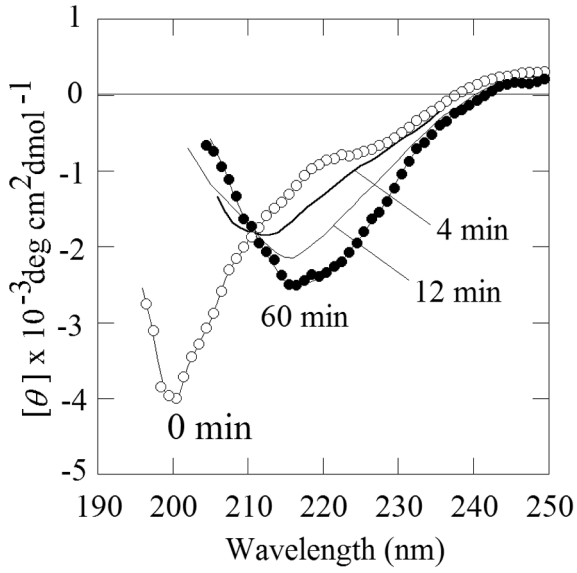

Figure S1. Effect of temperature on the secondary structure of αAC(71-88). Far-UV CD spectra of 500 μg/ml αAC(71-88) in 10 mM sodium phosphate (pH 7.5) with 100 mM NaCl was measured at 48°C.

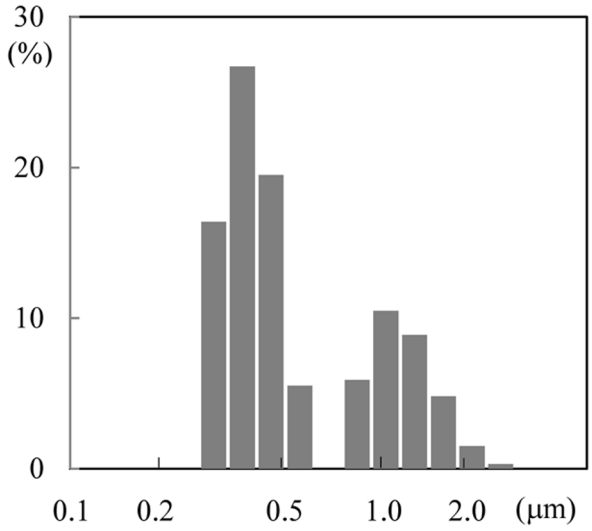

Figure S2. The distribution of diameter size of the heat-induced aggregate of ADH obtained by DLS measurement. ADH (150 μg/ml) in 10 mM sodium phosphate (pH 7.5) with 100 mM NaCl was incubated at 44°C for 1 hr.

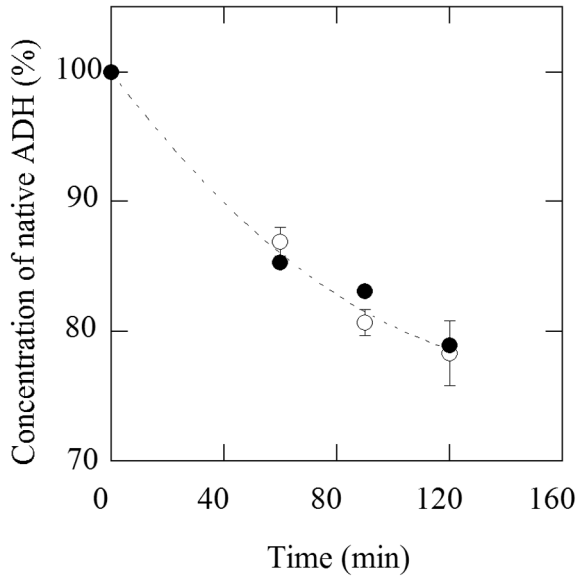

Figure S3. Effect of the αAC(71-88) amyloid fibrils on the concentration change of non-aggregated ADH in the solution incubated at 44 °C. ADH (150 μg/ml) in 10 mM sodium phosphate (pH 7.5) with 100 mM NaCl was incubated at 44°C in the presence (○) and absence (●) of the αAC(71-88) amyloid (10 μg/ml). The mixture was incubated for a given time and then centrifuged at 12,000 *g* to remove the ADH aggregates. The absence ADH aggregate in the supernatant was confirmed by gel filtration chromatography. The concentration of non-aggregated ADH in the supernatant was then determined from the A_{280}.

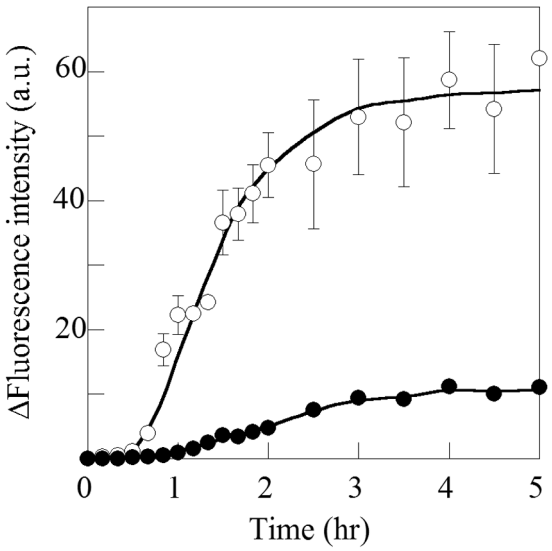

Figure S4. Fluorescence assay to analyze the effect of the αAC(71-88) amyloid on the fibril formation of Aβ(1-40). ThT fluorescence intensity 100 μg/ml Aβ(1-40) in 50 mM sodium phosphate (pH 7.5) with 100 mM NaCl was monitored at 37°C with shaking at a rotation rate of 200 rpm. ○, no additives; ●, + 400 μg/ml αAC(71-88) amyloid. The time trace of ThT fluorescence of the solution of Aβ(1-40) showed a characteristic sigmoidal curve with a lag phase of ~1 hr followed by a rapid fibril growth phase.