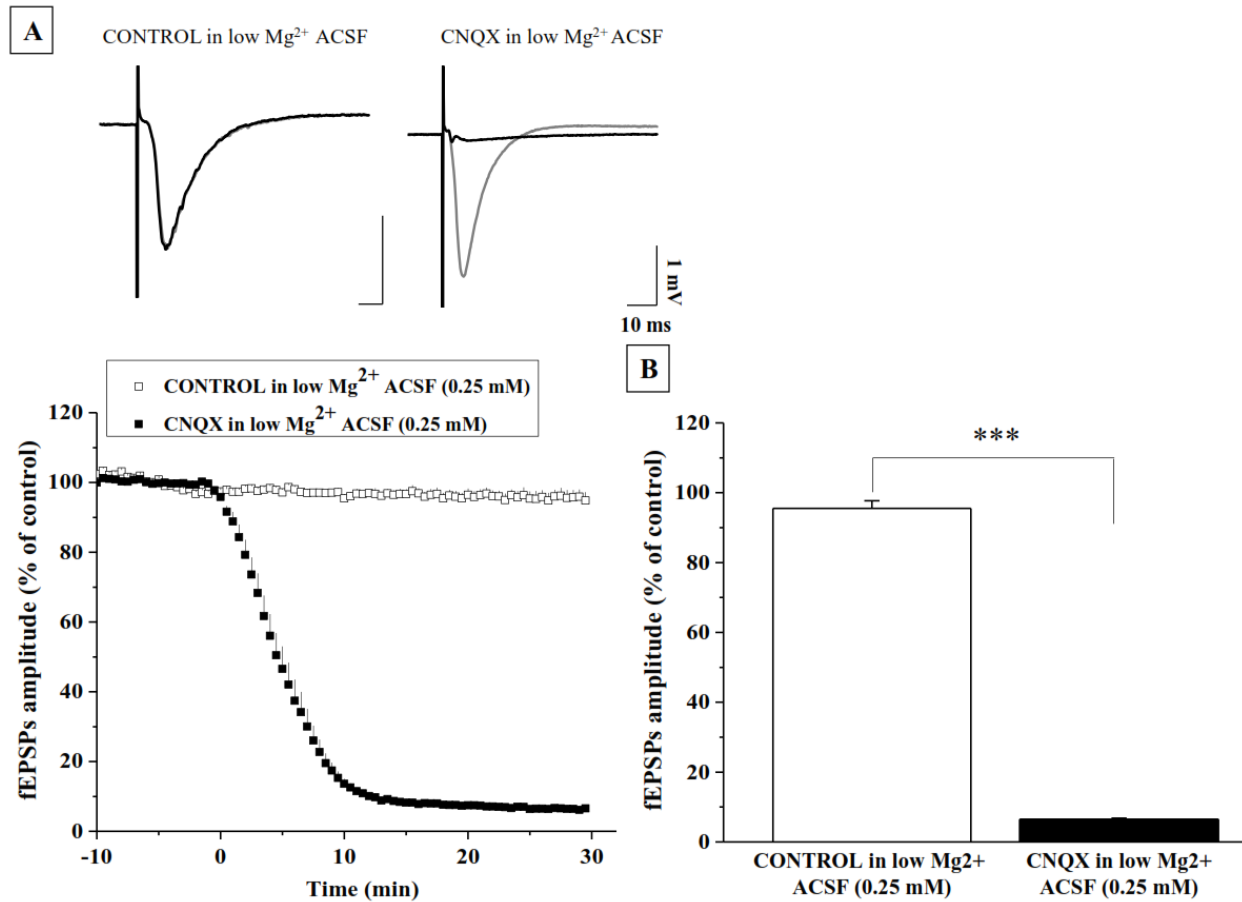
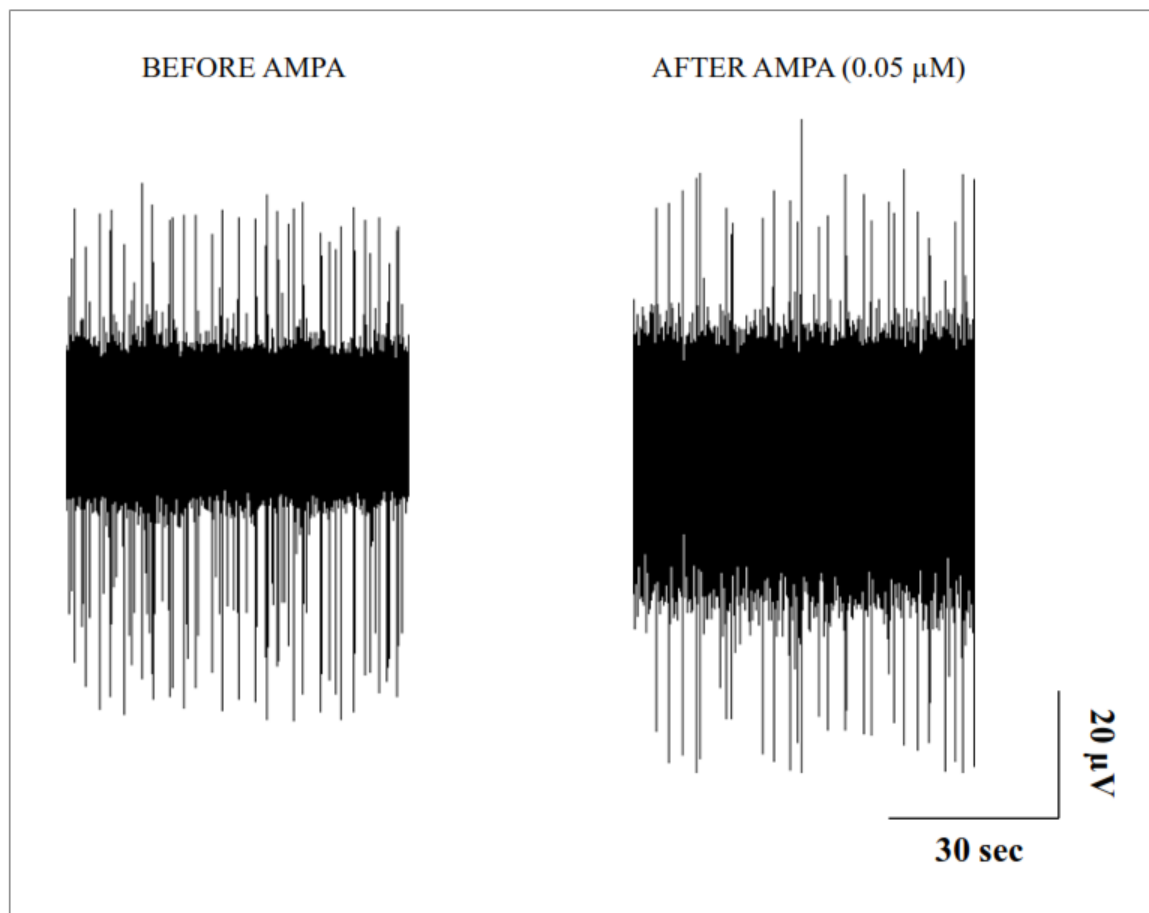


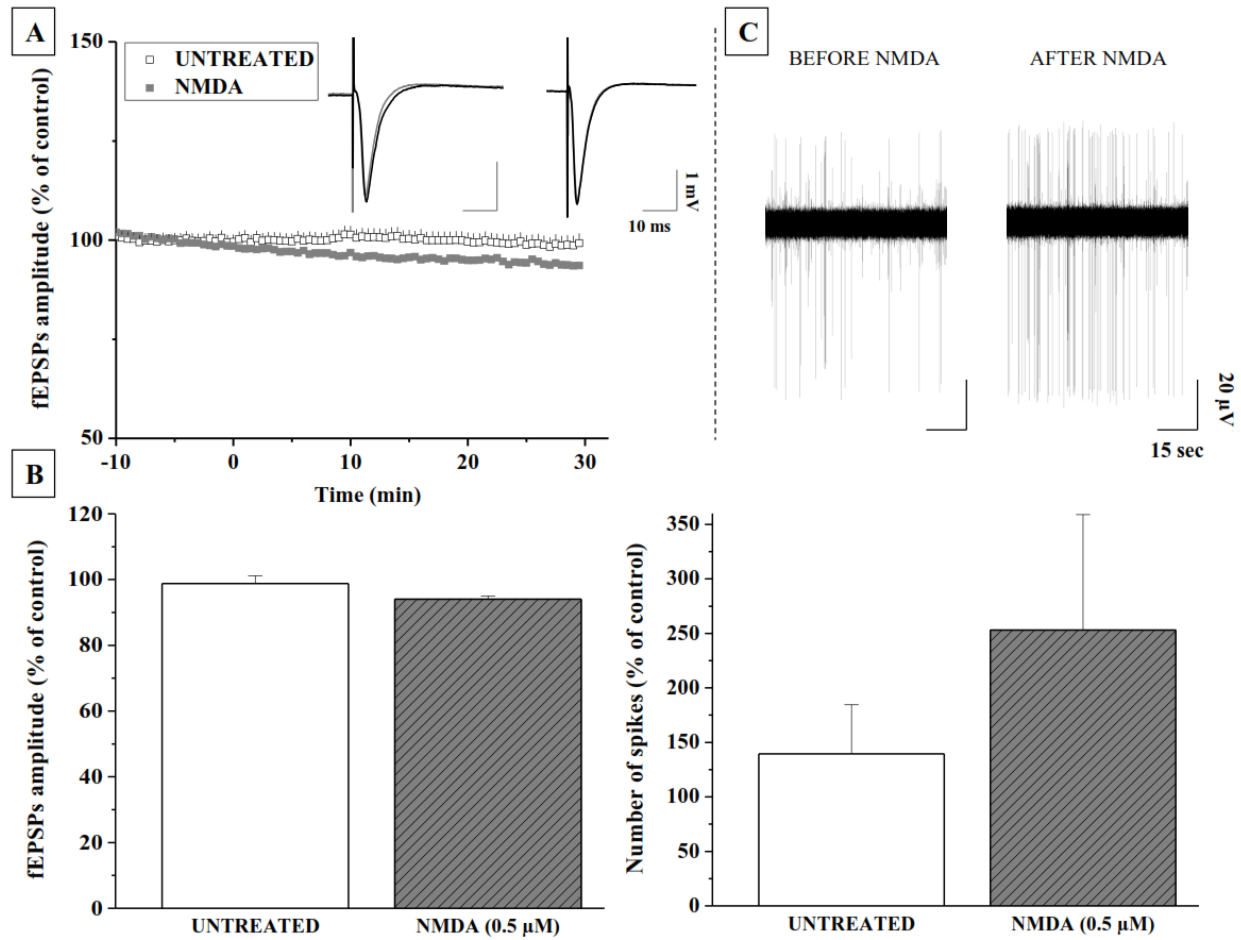
Supplementary material



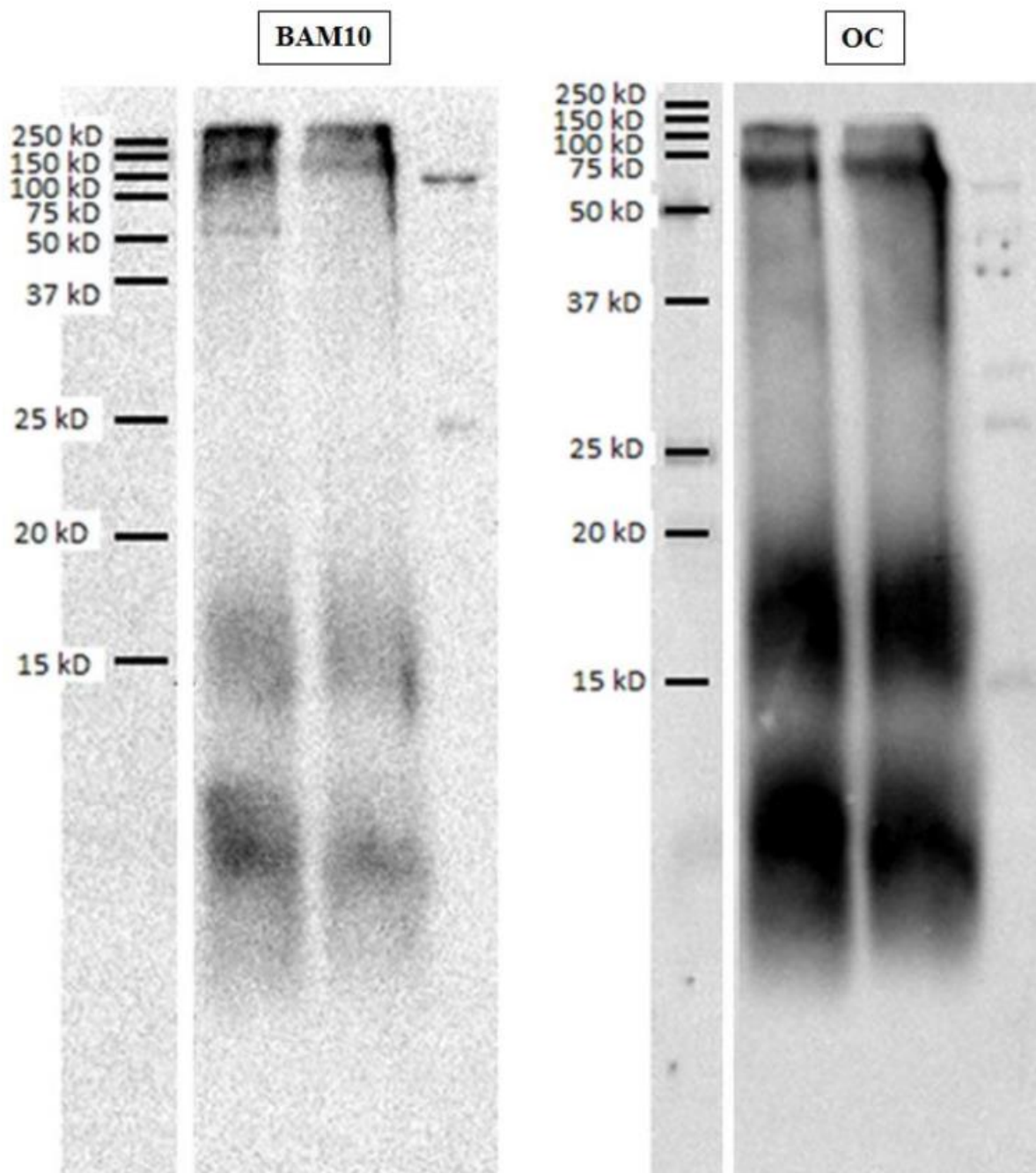
Supplementary Figure 1. CNQX abolished evoked fEPSPs even under condition that promotes NMDAR activation (in low Mg^{2+} ACSF) (**A**). Upper panel shows representative fEPSPs before (gray) and after (black) treatment. Bar graphs show the average of the fEPSPs amplitudes of the 25-30 min period after treatment (**B**). Error bars show SEM; *** $p \leq 0.001$.



Supplementary Figure 2. Low concentration of AMPA (0.05 μ M) increased basal activity which hindered the effective detection of spikes. Figure shows representative traces before (left) and after (right) AMPA application.



Supplementary Figure 3. Small concentration of NMDA (0.5 μ M), which was reported to activate extrasynaptic NMDARs, did not affect fEPSPs (**A**). Inset shows representative fEPSPs before (grey) and after (black) treatment. Bar graphs show the average of the fEPSPs amplitudes of the 25-30 min period after treatment (**B**). Low concentration of NMDA elevated spiking frequency, although not significantly (**C**). Inset shows representative spike trains before and after treatment. Error bars show SEM.



Supplementary Figure 4. Abeta(1-42) samples used for the recordings contained clearly detectable SDS-stable trimers (≈ 15 kDa) and higher molecular weight protofibrils. Staining with OC antibody, which is specific for species having beta-sheet conformation, shows that the small low-n oligomers are of prefibrillar nature.