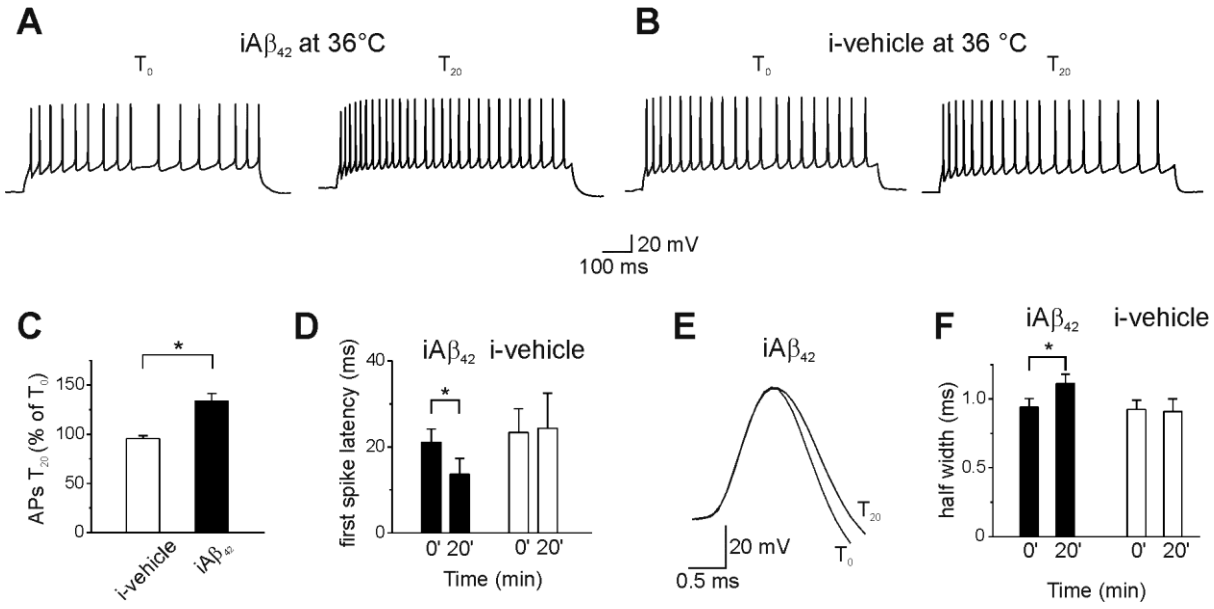
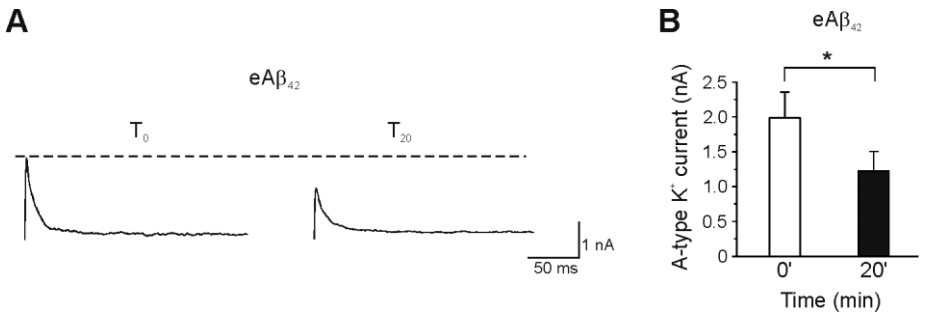
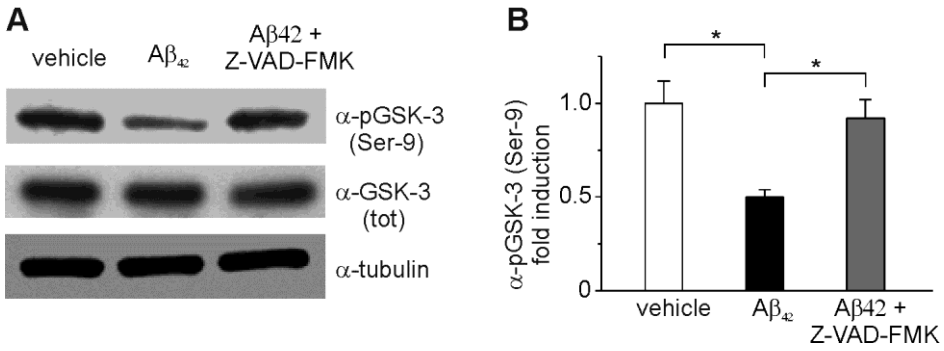


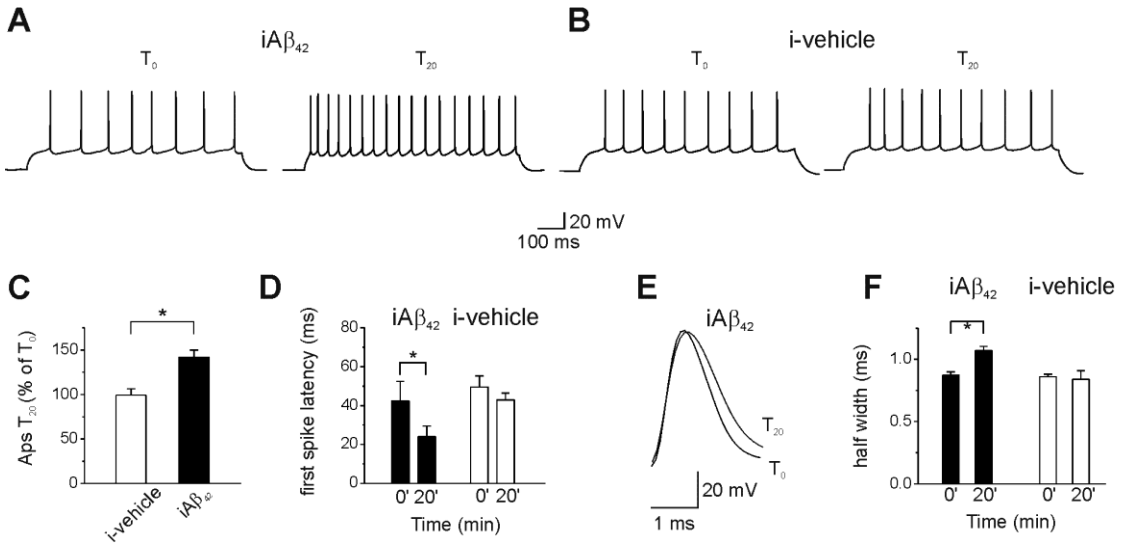
Supplementary figure 1. Intracellular perfusion of Aβ42 increases primary hippocampal neuron excitability also at more physiological temperature (36°C). (**A**) Examples of whole-cell recordings at 36°C. Twenty min of intracellular perfusion of Aβ42 via the patch-pipette produced an increase in APs number comparable to what observed at room temperature and reported in figure 1. (**B**) Intracellular vehicle perfusion had no effect on neuronal excitability. (**C, D and F**) Bar graphs showing the effects of intracellular perfusion of Aβ42 and vehicle (at 36°C) in terms of: *i*) Percent changes in APs number (Aβ42: T_0 = 21.7 ± 1.9; T_{20}: 29.3 ± 3.4; n = 6; $p < 0.05$; paired t-test); *ii*) first spike latency (Aβ42:T_0 = 21.1 ± 3.0 ms; T_{20} = 13.7 ± 3.6 ms; n = 6; $p < 0.05$; paired t-test) and *iii*) half width (Aβ42: T_0 = 0.9 ± 0.1 ms; T_{20} = 1.1 ± 0.1 ms; n = 6; $p < 0.05$; paired t-test; see also representative traces in **E**).



Supplementary figure 2. Extracellular $A\beta_{42}$ reduces transient A-type K^+ currents in primary mouse hippocampal neurons. (**A**) Representative traces showing the reduction of transient A-type K^+ currents after 20 min of extracellular application of $A\beta_{42}$. (**B**) Bar graph summarizing data from experiments shown in A (n = 6).



Supplementary figure 3. Caspase inhibition prevents the $A\beta_{42}$-induced decrease in phosphorylated GSK-3 at Ser-9. Representative Western blots of hippocampal proteins showing immunoreactivity for phosphorylated GSK-3 at Ser-9 following 40 min of $A\beta_{42}$ stimulation (alone or after 30 min of pre-treatment with caspase inhibitor Z-VAD-FMK). Densitometry for the blots probed with Ser-9 phosphorylated GSK-3 normalized to ~ total GSK-3 is shown (n = 3; $p < 0.05$; statistics by Mann-Whitney test).



Supplementary figure 4. Intracellular perfusion of Aβ42 increases CA1 hippocampal pyramidal neuron excitability in acute slices preparation. (**A**) Representative current clamp traces, recorded in CA1 hippocampal pyramidal neurons from non-Tg slices, showing the increase in APs number after 20 min of intracellular perfusion of Aβ42 via patch-pipette. (**B**) Intracellular vehicle perfusion had no effect on neuronal excitability. (**C**) Bar graph summarizing data from experiments shown in A and B (Aβ42 $n = 6$; vehicle $n = 5$; $p < 0.05$). (**D**) Bar graph showing first spike latency in the two experimental conditions (Aβ42 $n = 6$; vehicle $n = 5$; $p < 0.05$). (**E**) Representative traces showing the AP width in Aβ42–injected neuron. (**F**) Bar graph showing mean values of spike half-width after 20 min of intracellular perfusion of Aβ42 ($n = 6$; $p < 0.05$) and vehicle ($n = 5$; $p > 0.05$).