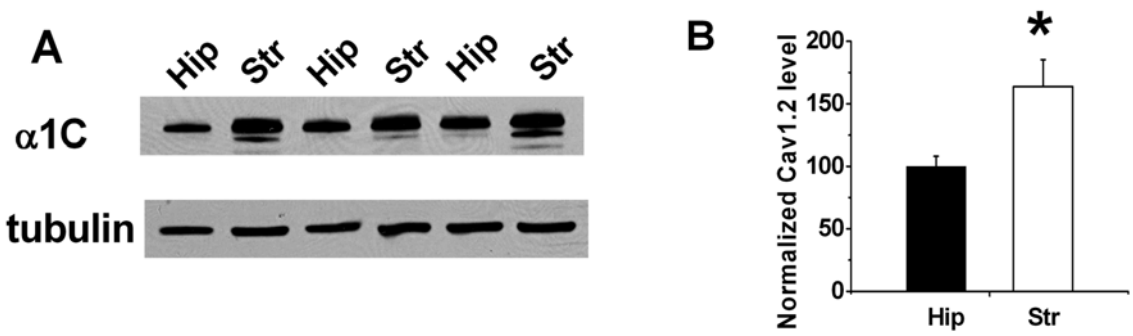

Figure S1. Higher $Ca_v1.2$ levels in striatum compared to hippocampus. A. Immunoblots of hippocampal or striatal lysates from one-month old WT mice probed with anti-α1C antibody. α-tubulin was used as loading control. B. Quantitative analysis of data in (A), $Ca_v1.2$ level was significantly higher (*$p<0.05$) in striatum compared to hippocampus. Each value represents the mean ± SEM (N=3).

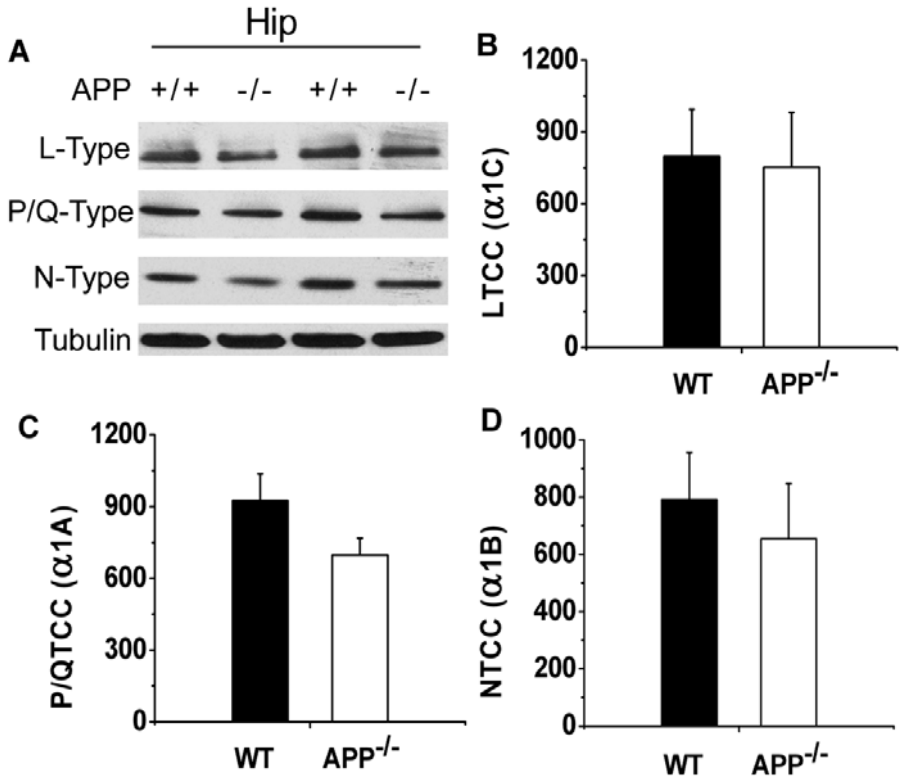

Figure S2. Similar LTCC, P/QTCC and NTCC expression levels in the $APP^{-/-}$ and WT hippocampus. A. Immunoblots of hippocampal extracts from 3 week-old $APP^{-/-}$ and WT littermates. The blots were probed with anti-α1C, α1A and α1B subunits antibodies representing LTCC, P/QTCC and NTCC, respectively. Anti-α-tubulin was used as loading control. Quantification of blots shown in A, revealing that no significant differences were found in the expression of LTCC (B), P/QTCC (C) and NTCC (D). Each value represents the mean ± SEM (N=3).

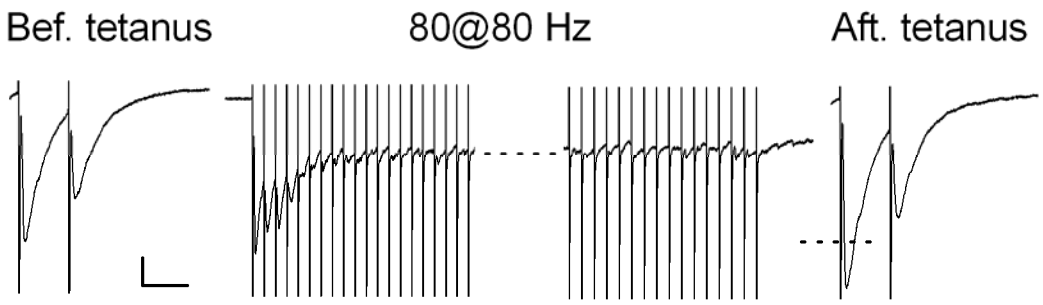

Figure S3. Induction of GABAergic PTP in WT hippocampal neurons by high frequency stimulation (1 sec at 80 Hz). IPSC1 amplitude increased 29 ± 5% after tetanus compared to before tetanic control (N=3). Scales: 100 pA/50 msec.

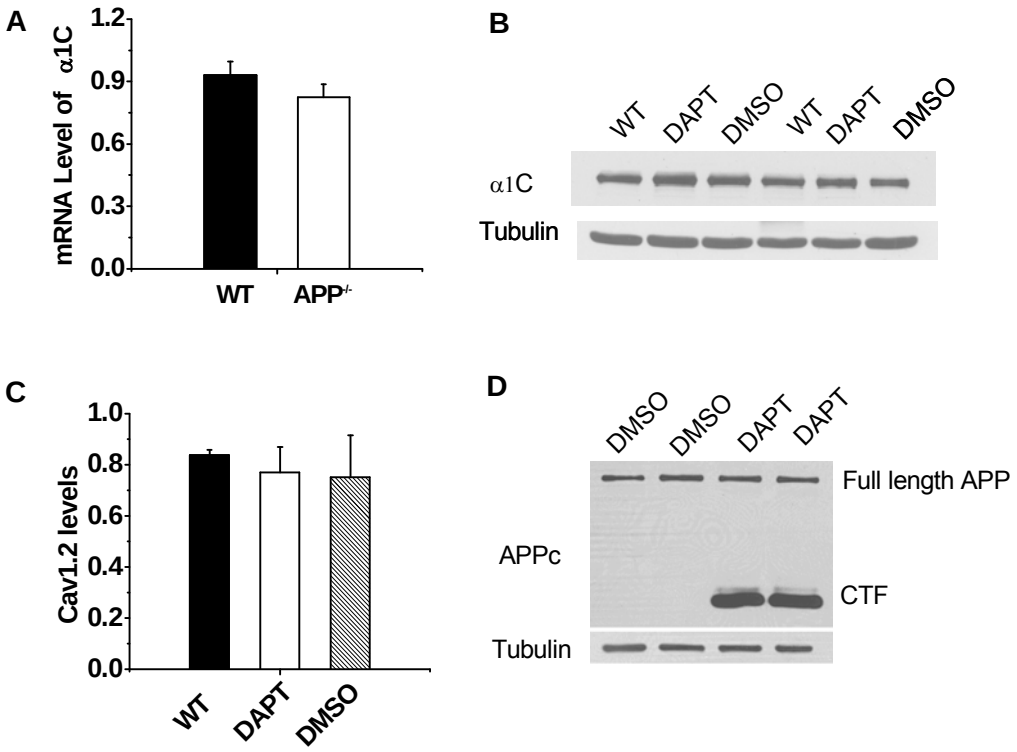

Figure S4. Similar α1C mRNA expression in WT and APP$^{-/-}$ striatum and unaffected α1C protein level by γ-secretase inhibitor. A. Quantitative RT-PCR analysis of α1C mRNA levels in wild-type and APP$^{-/-}$ striatum (values normalized to GAPDH control). B. Representative immunoblots of α1C protein from striatal cultures with or without γ- secretase inhibitor. WT: untreated; DAPT: γ- secretase inhibitor 1 μM DAPT treated; DMSO: DMSO treated (vehicle control). α-tubulin was used as loading control. C. Quantification of α1C/tubulin. Each value represents the mean ± SEM (N=3). D. Accumulation of APP-CTF in the presence of 1μM DAPT, cleavage products of APP by α and β secretases and the lack of γ- secretase activity.

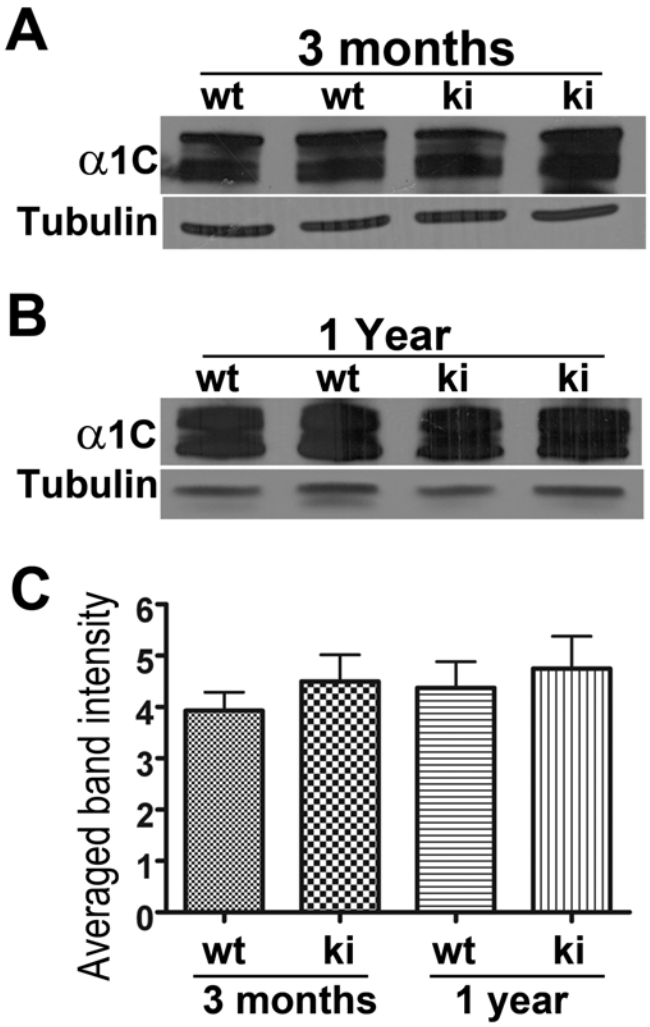

Figure S5. Indistinguishable $Ca_v1.2$ levels in striatum of wild type (WT) and homozygous *APP* knock-in mice containing human Aβ sequence and the Swedish/London FAD mutations (ki). A and B. Immunoblots of striatal lysates from 3 months (A) and one year (B) old WT and ki mice probed with anti-α1C antibody. α-tubulin was used as loading control. C. Quantitative analysis of data (mean ± SEM) in A and B.