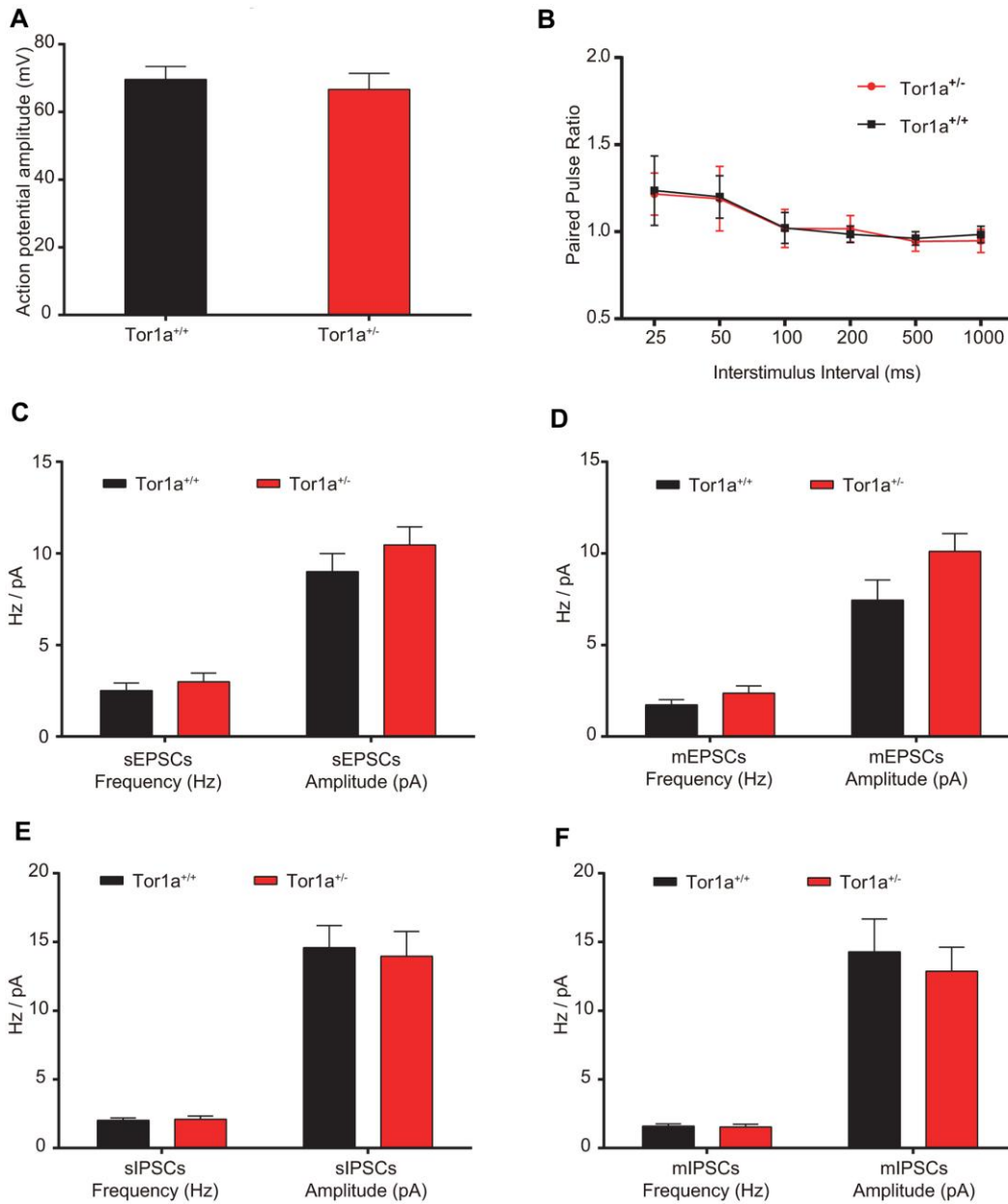
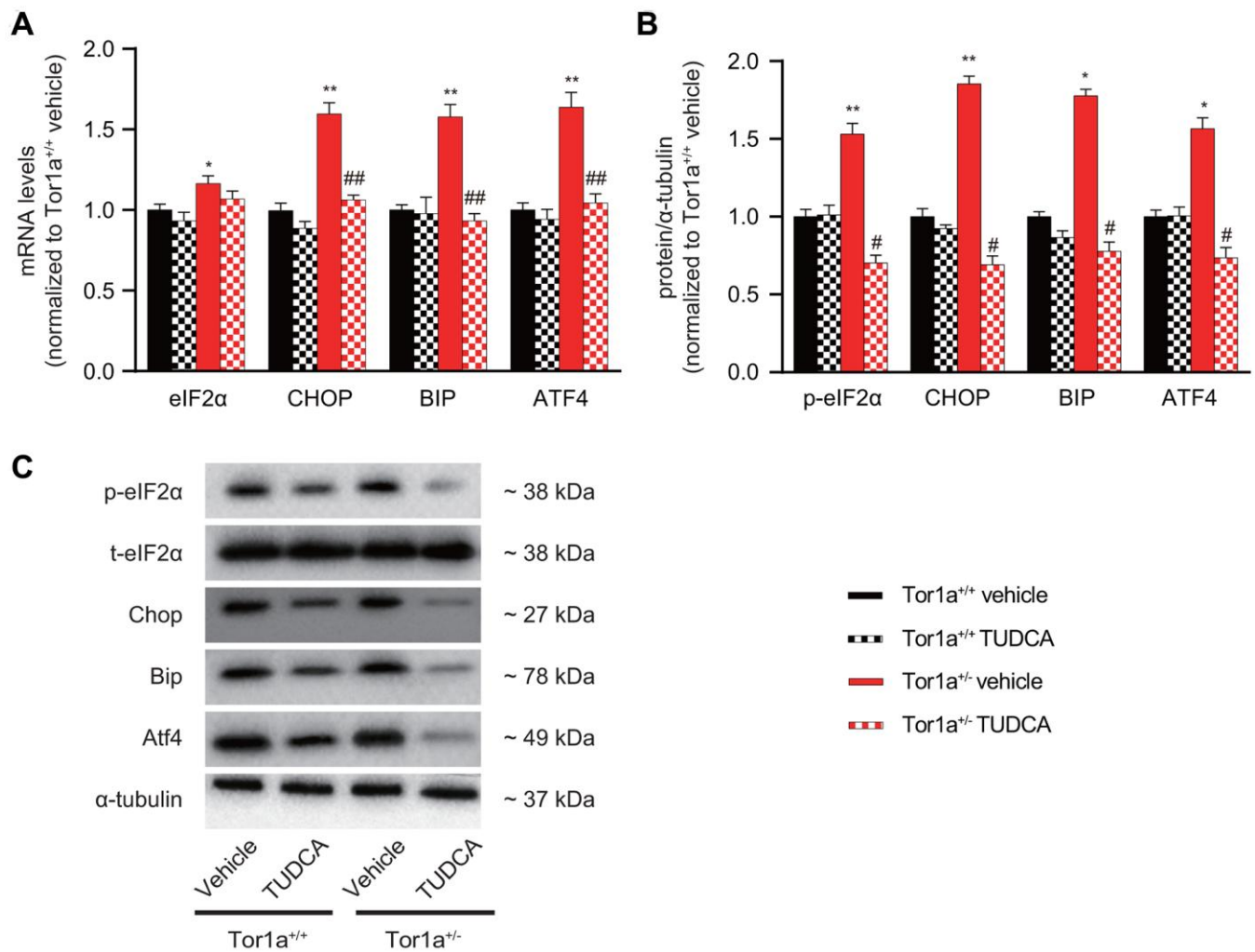


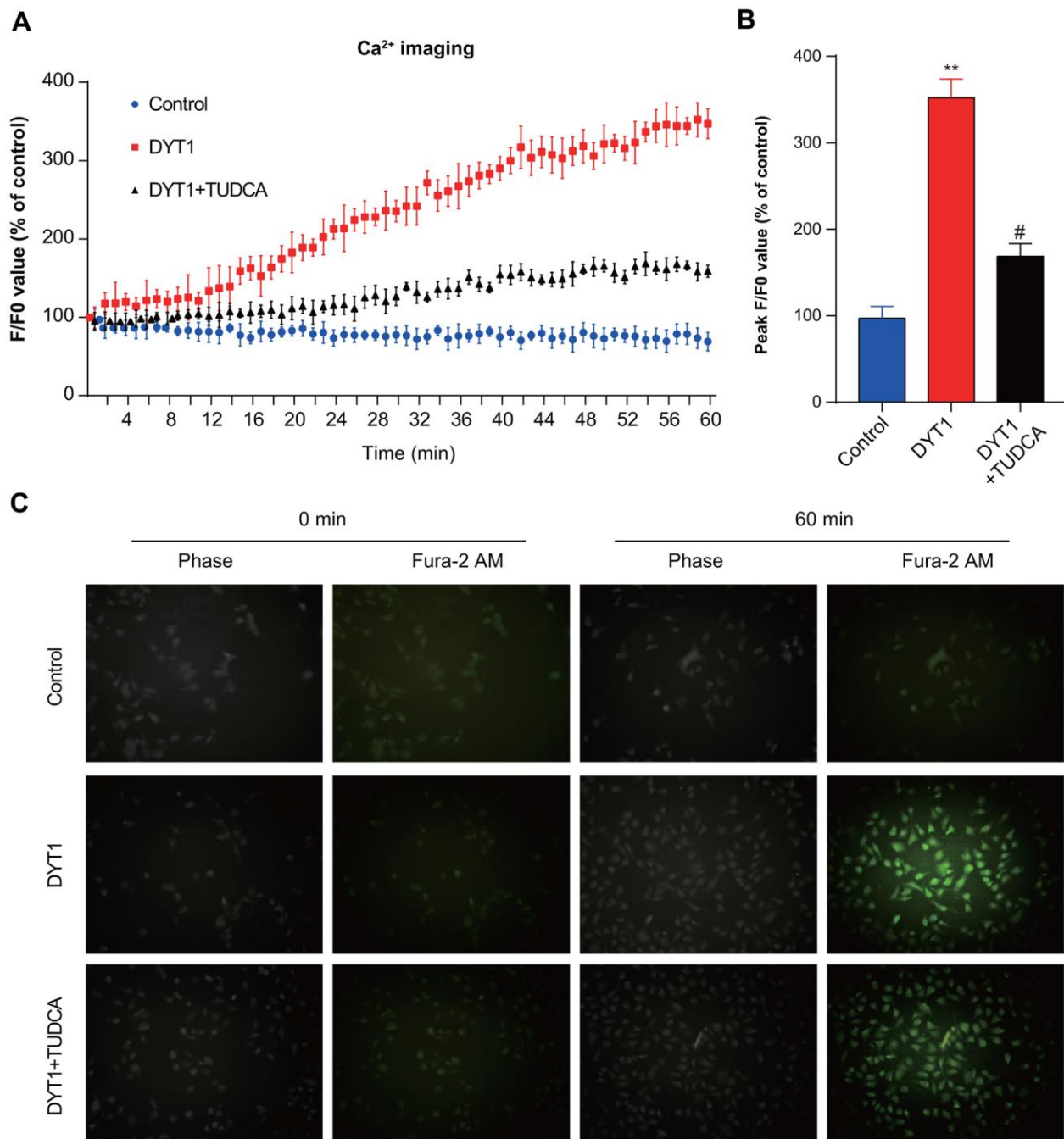
**SUPPLEMENTARY FIGURES**



**Supplementary Figure 1. Intrinsic and synaptic properties of SPNs from juvenile mice.** (A) Depolarizing (+600 pA) and hyperpolarizing (200 pA) current steps caused tonic action potential discharge in SPNs recorded from Tor1a<sup>+/+</sup> (black) and Tor1a<sup>+/-</sup> (red) mice. (B) Paired-pulse ratio (PPR) showed similar facilitation in both genotypes. Short ISI (25-50 ms) of paired synaptic stimulation could induce PPF in both genotypes ( $P < 0.05$ ), whereas longer ISI (100-1000 ms) failed ( $P > 0.05$ ). (C) Glutamatergic sEPSCs recordings in PTX from SPNs of Tor1a<sup>+/+</sup> and Tor1a<sup>+/-</sup> mice showed no significant difference between genotypes in frequency and amplitude (both  $P > 0.05$ ). (D) Glutamatergic mEPSCs recordings in PTX plus TTX from SPNs of Tor1a<sup>+/+</sup> and Tor1a<sup>+/-</sup> mice showed no significant difference between genotypes in frequency and amplitude (both  $P > 0.05$ ). (E) GABAergic sIPSCs recordings in MK-801 and CNQX from SPNs of Tor1a<sup>+/+</sup> and Tor1a<sup>+/-</sup> mice showed no significant difference between genotypes in frequency and amplitude (both  $P > 0.05$ ). (F) GABAergic mIPSCs recorded in MK-801, CNQX and TTX from SPNs of Tor1a<sup>+/+</sup> and Tor1a<sup>+/-</sup> mice showed no significant difference between genotypes in frequency and amplitude (both  $P > 0.05$ ). In each group, five mice were used (N=5), and three independent electrophysiological recordings were conducted for each mouse (n=3).  $P < 0.05$  was considered to be statistically significant.



**Supplementary Figure 2. TUDCA inhibits the ER stress markers in Tor1a<sup>+/-</sup> mice.** (A) Levels of mRNA in striatal lysates were measured by RT-qPCR. (B) Quantification of protein expression in striatum is shown (N=5 per group). Data are represented as mean ±SEM. (C) Representative western blots of striatal lysates. In each group, five mice were used (N=5), and three independent experiments were conducted for each mouse (n=3). *P*<0.05 was considered to be statistically significant.



**Supplementary Figure 3. TUDCA restores calcium dynamics in *Tor1a<sup>-/-</sup>* striatal spiny projection neurons (SPNs) under ER stress.** (A) Ca<sup>2+</sup> imaging in Ca<sup>2+</sup>-free solution to examine the role of TUDCA *in vitro*. (B) Quantification of Ca<sup>2+</sup> imaging shows that SPNs from *Tor1a<sup>-/-</sup>* mice revealed a significantly higher intracellular Ca<sup>2+</sup> concentration than that of SPNs from *Tor1a<sup>+/+</sup>* mice. However, the intracellular Ca<sup>2+</sup> release induced by *Tor1a<sup>-/-</sup>* was markedly alleviated by the ER stress inhibitor TUDCA. (C) Representative pictures of Ca<sup>2+</sup> imaging at 0 and 60 min. In each group, five mice were used (N=5), and three independent experiments were conducted for each mouse (n=3). P<0.05 was considered to be statistically significant.