Supplementary Figure 1

Steady decline of transmission rate of the 16p11.2 df/+ allele.

The percentage of the heterozygous 16p11.2 df/+ mutant mice, including both male and female, gradually declines across multiple generations during backcrossing to C57BL/6J. The number of 16p11.2 df/+ and total mice for each generation are: N5: 21/53, N6: 26/118, N7: 41/250, N8: 57/434, N9: 16/202, and N10: 8/102.
Supplementary Figure 2

Basal synaptic transmission is normal in the 16p11.2 df/+ mice.

(a) Input-output functions, plotted as fEPSP slope versus stimulus intensity, are not different between the WT (n = 14 animals) and mutant mice (n = 14 animals). Repeated measures one-way ANOVA, p = 0.92.

(b) Paired-pulse facilitation is comparable between the WT (n = 16 animals) and mutant (n = 17 animals) mice across multiple stimulus intervals (10, 20, 50, 100, 200, 300, 500 ms). Repeated measures one-way ANOVA, p = 0.76. All data are plotted as mean ± SEM.
Presynaptic LTD is independent of genotype and is not affected by cycloheximide treatment. DHPG increases paired-pulse facilitation in both the WT and 16p11.2 df/+ mutant slices and this effect is not affected by cycloheximide. PPF is calculated at a 50 ms inter-stimulus interval: WT baseline: 1.44 ± 0.019, n = 17 animals, 37 slices, WT DHPG: 1.55 ± 0.035, n = 17 animals, 18 slices, WT DHPG+CHX: 1.55 ± 0.027, n = 17 animals, 19 slices. Mut baseline: 1.45 ± 0.023, n = 16 animals, 42 slices, Mut DHPG: 1.56 ± 0.053, n = 16 animals, 21 slices, Mut DHPG+CHX: 1.53 ± 0.038, n = 16 animals, 21 slices. Statistical analyses are performed using unpaired t-tests. All data are plotted as mean ± SEM.