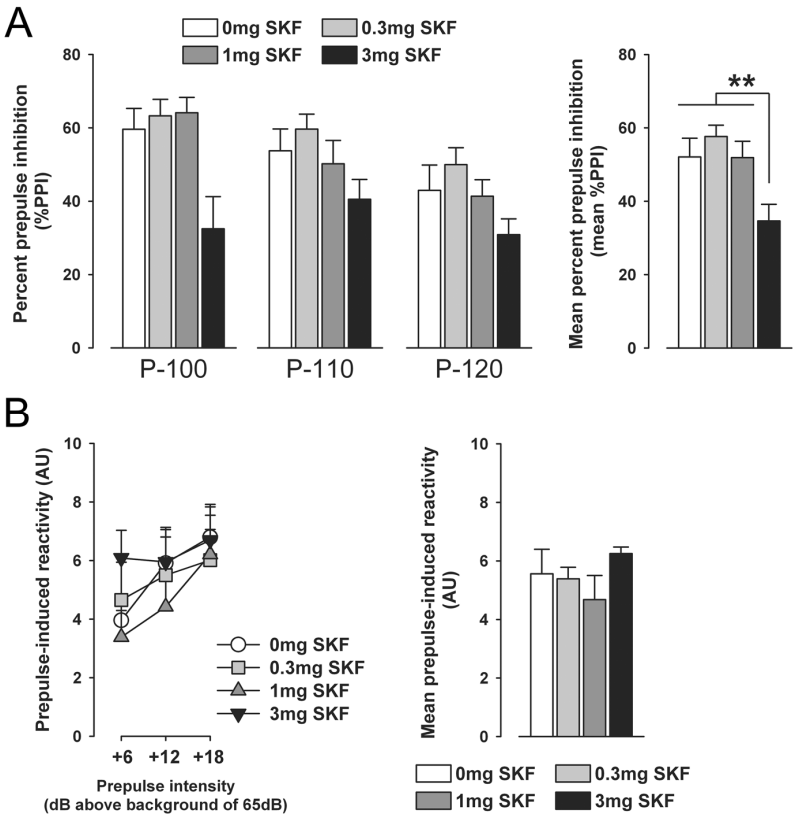
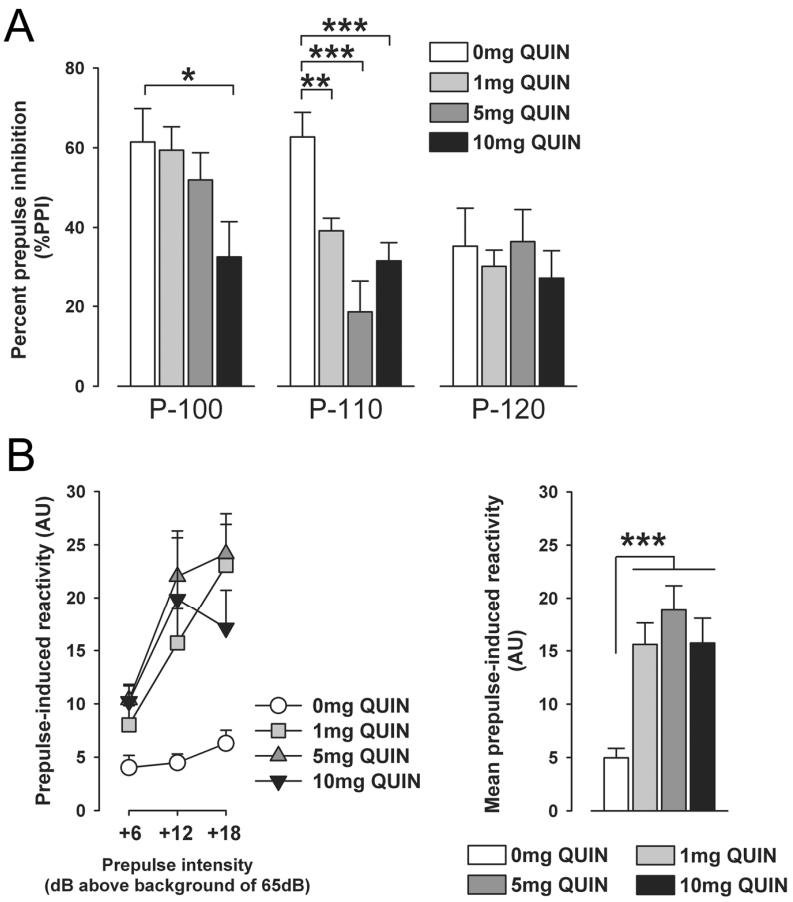
Supplemental material



Supplemental Figure 1. Effects of pharmacological dopamine D1 receptor (D1R) stimulation on prepulse inhibition and prepulse-induced startle reactivity in adult C57BL6/mice. Adult (postnatal day 70) C57BL/6 male mice were treated with the selective D1R agonist SKF38393 (SKF) at a dose of 0 (=vehicle; NaCl solution), 0.3, 1, or 3 mg/kg (i.p.) 10 min before the commencement of prepulse inhibition (PPI) testing. **(A)** The graphs show percent prepulse inhibition (%PPI) as a function of three pulse intensities (100dB$_A$ = P-100, 110dB$_A$ = P-110, and 120dB$_A$ = P-120) and the mean %PPI across all three pulse levels. Administration of the highest dose of SKF38393 (3 mg/kg) led to a significant reduction in % PPI, which was noticeable in all three pulse conditions, leading to a significant overall reduction in mean %PPI. $^{**}P < 0.01$, based on Fisher's LSD post-hoc group comparisons following presence of a significant [$F(3,28) = 5.23$, $P < 0.01$] effect of treatment in the initial 4 × 3 × 3 (treatment × prepulse level × pulse level) ANOVA. **(B)** The line plot depicts prepulse-induced reactivity (in arbitrary units, AU) as a function of the three prepulse levels (+6, +12 and +18 dB$_A$ above a background level of 65dB$_A$); the bar plot shows the mean prepulse-induced startle reactivity across all three prepulse levels. SKF38393 treatment did not significantly affect prepulse-induced startle reactivity compared to vehicle treatment. The drug did also not significantly affect reactivity to pulse-alone stimuli (data not shown). All values in (A) and (B) are means±S.E.M. N=8 in each drug condition.



Supplemental Figure 2. Effects of pharmacological dopamine D2-like (D2-like) receptor stimulation on prepulse inhibition and prepulse-induced startle reactivity in adult C57BL6/mice. Adult (postnatal day 70) C57BL/6 male mice were treated with the D2R-like agonist quinpirole (QUIN) at a dose of 0 (=vehicle; NaCl solution), 1, 5, or 10 mg/kg (i.p.) 10 min before the commencement of prepulse inhibition (PPI) testing. **(A)** The graphs show percent prepulse inhibition (%PPI) as a function of three pulse intensities ($100dB_A$ = P-100, $110dB_A$ = P-110, and $120dB_A$ = P-120). Administration of the highest dose of QUIN (10 mg/kg) significantly reduced % PPI in conditions, in which a stimulus of low (P-100) or middle (P-110) intensity served as the pulse stimulus; QUIN at low (1 mg/kg) or middle (5 mg/kg) dose significantly decreased %PPI in conditions, in which a stimulus of middle (P-110) intensity was used as the pulse stimulus; QUIN did not significantly affect the expression of %PPI in conditions, in which a stimulus of high (P-120) intensity served as the pulse stimulus. $*P < 0.05$ and $**P < 0.01$, based on Fisher's LSD post-hoc group comparisons restricted to each pulse condition following presence of a significant [$F(3,28) = 5.42$, $P < 0.01$] main effect of treatment [$F(6,56) = 2.82$, $P < 0.05$] and significant treatment × pulse level interaction in the initial 4 × 3 × 3 (treatment × prepulse level × pulse level) ANOVA. **(B)** The line plot depicts prepulse-induced reactivity (in arbitrary units, AU) as a function of the three prepulse levels (+6, +12 and +18 dB_A above a background level of $65dB_A$); the bar plot shows the mean prepulse-induced startle reactivity across all three prepulse levels. QUIN at all three doses significantly increased prepulse-induced startle reactivity compared to vehicle treatment. The drug did not significantly affect reactivity to pulse-alone stimuli (data not shown). $***P < 0.001$, based on Fisher's LSD post-hoc group comparisons following presence of a significant [$F(3,28) = 11.89$, $P < 0.001$] main effect of treatment in the initial 4 × 3 (treatment × prepulse level) ANOVA. All values in (A) and (B) are means±S.E.M. N=8 in each drug condition.