

Martire et al.

Supplemental Figures

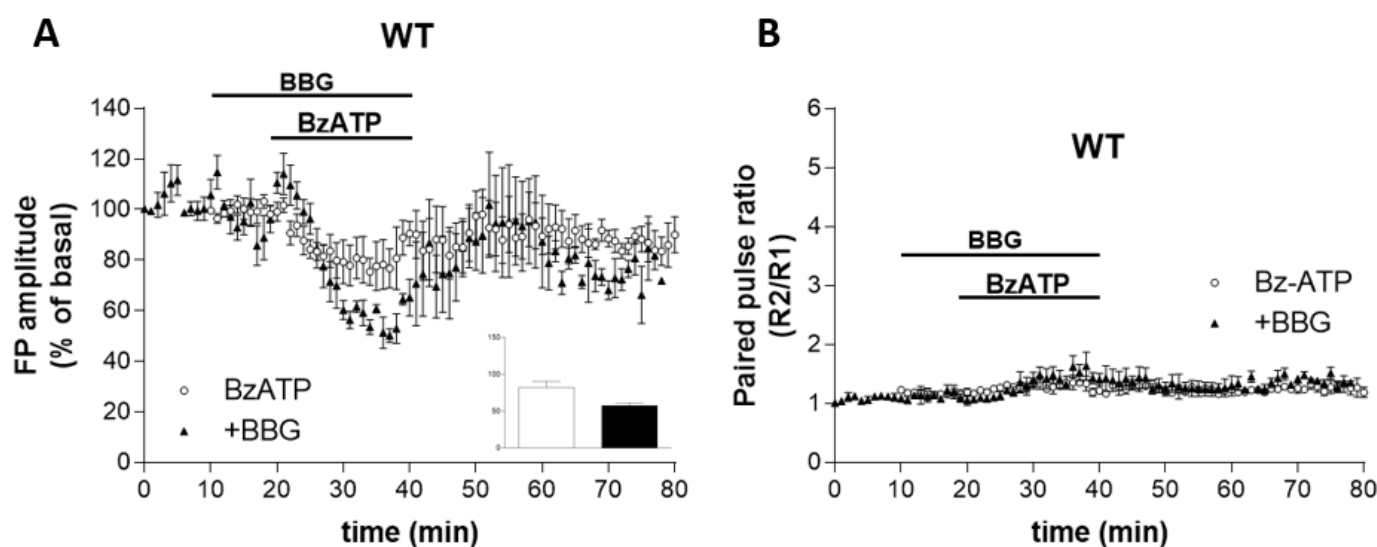


Figure S1.

BzATP-induced depression of corticostriatal synaptic transmission in WT mice is not reversed by the P2X7R antagonist Coomassie Brilliant Blue G (BBG)

In WT mice, the inhibitory effect of BzATP (50 μ M for 20 min) on FP ($82.08 \pm 8.12\%$ of basal, $n = 4$) was not prevented by the P2X7R antagonist BBG (1 μ M, for 30 min) ($57.42 \pm 3.43\%$, of basal $n = 3$, panel A). On the contrary, a slight and non-significant worsening was induced. The trend of the mean PPR resulted superimposable with or without BBG (panel B), indicating a substantial absence of any effect. According to Jiang et al. (2000), BBG 1 μ M is below the IC(50) values for inhibition of the other P2XRs, thus suggesting P2X7R selectivity. BBG (Sigma-Aldrich) was dissolved in distilled water.

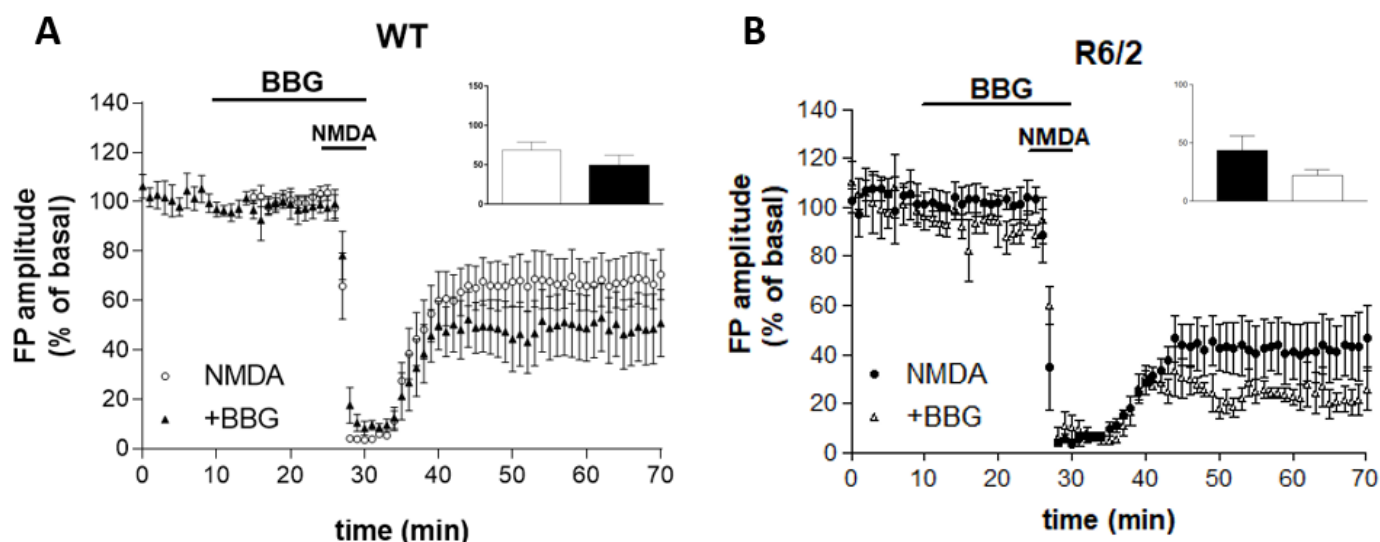


Figure S2.

P2X7R antagonist BBG did not reduce NMDA toxicity in corticostriatal slices from R6/2 and WT mice

The NMDA experimental paradigm has been already used by our group to assess the role of A_{2A} (Martire et al., 2007) and A₁ (Ferrante et al., 2014) receptors in excitotoxicity in corticostriatal slices from WT and R6/2 mice. NMDA (75 μ M) is applied to the slices over 5 min, inducing a reduction of FP amplitude which results increased in R6/2 mice. In WT mice (panel A) the recovery of the FP after NMDA application ($68.31 \pm 10.44\%$ of basal after 40 min of washout, $n = 7$) was not significantly affected by the BBG treatment (1 μ M, for 20 min) ($48.32 \pm 13.80\%$ of basal after 40 min of washout, $n = 7$). In R6/2 mice (panel B) NMDA toxicity was higher ($43.20 \pm 12.68\%$ of basal after 40 min of washout, $n = 3$) than in WT mice, but also in this case BBG did not exert a protective action; on the contrary, a worsening, even if non-significant, was induced ($21.69 \pm 5.36\%$ of basal after 40 min of washout, $n = 4$). NMDA (Sigma-Aldrich) was dissolved in NaOH 0.1 N.

References

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