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Endocannabinoid and Nitric Oxide-Dependent IGF-I-Mediated Synaptic Plasticity at Mice Barrel Cortex

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Supplementary Materials:

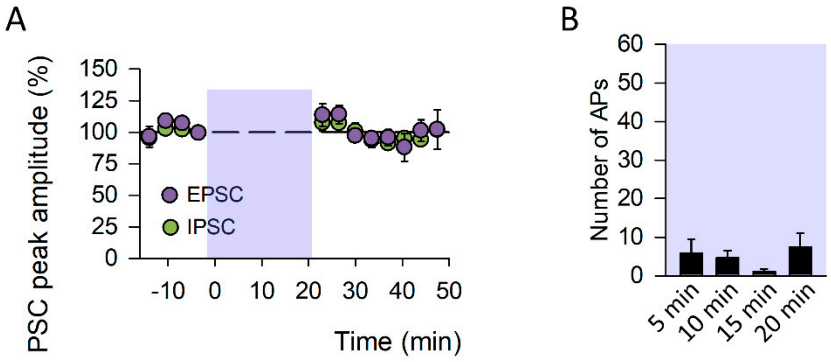

Figure S1. The protocol of stimulation does not induce plasticity. **A**) Plot showing a long-lasting stable recording of EPSCs (blue circles; n=5, p=0.83) and IPSCs (green circles; n=5, p=0.23) using the same protocol as in figure 1a, but in the absence of IGF-I. **B**) Number of action potentials recorded at different time points (5,10,15 and 20 min), showing the lack of effect of the protocol used (n=5, p=0.502).

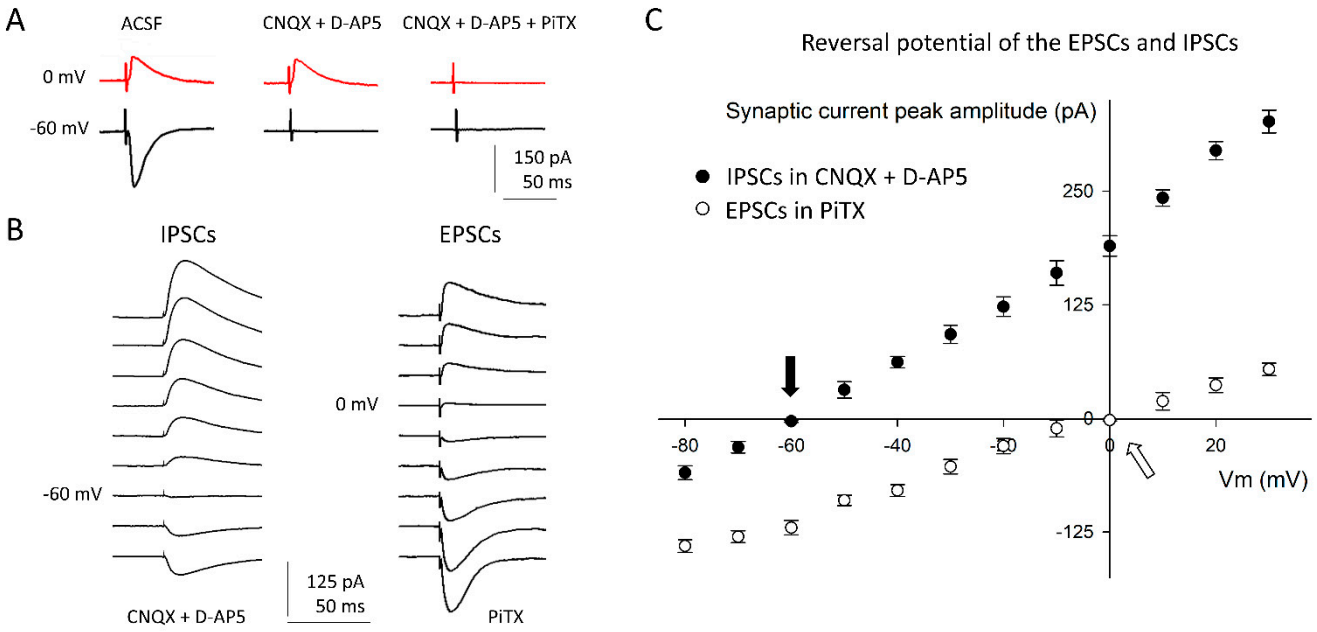

Figure S2. Reversal potential of the EPSCs and IPSCs. **A**) Representative synaptic currents recorded at 0 mV (top red recordings) and at −60 mV (bottom black recordings) in ASCF (left), in CNQX + D-AP5 (middle) and in CNQX + D-AP5 + PiTX (right). Note that the synaptic currents recorded at −60 mV are totally abolished under CNQX + D-AP5 indicating that are glutamatergic, being unaffected these recorded at 0 mV that are blocked with PiTX suggesting that are GABAergic ones. **B**) Representative IPSCs and EPSCs recorded in CNQX + D-AP5 (left) and in PiTX (right), respectively, while holding the membrane potential in steps of 10 mV. Note that −60 mV is the IPSC reversal potential whereas the EPSC reversal potential is 0 mV. C, Plot of the synaptic current peak amplitude measured at 10 mV steps from −80 mV to +30 mV in CNQX + D-AP5 (IPSCs, black circles) and in PiTX (EPSCs, white circles). Black arrow point to the IPSC reversal potential (−61.6 ± 1.7 mV, n=6) and white arrow point to the EPSC reversal potential (0 ± 2.6 mV, n=6).