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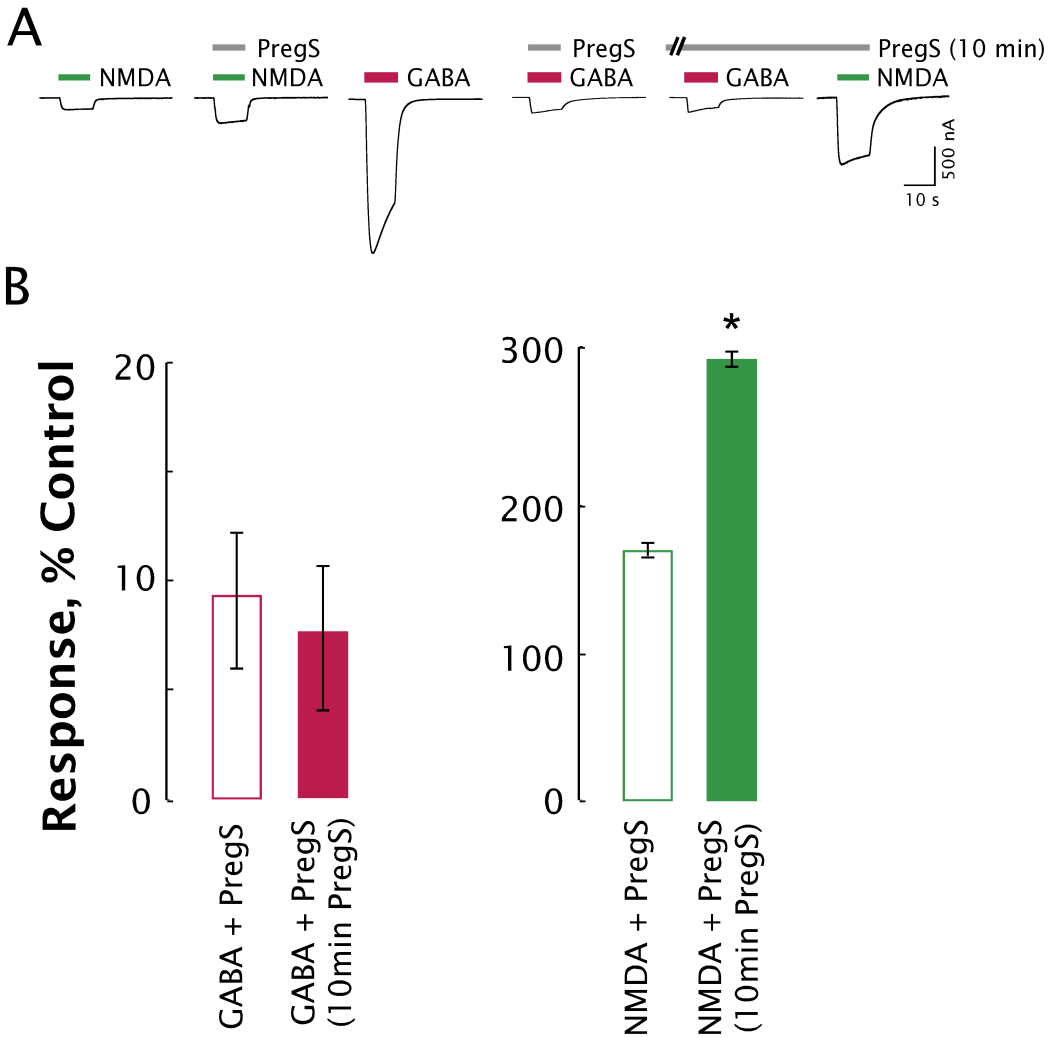
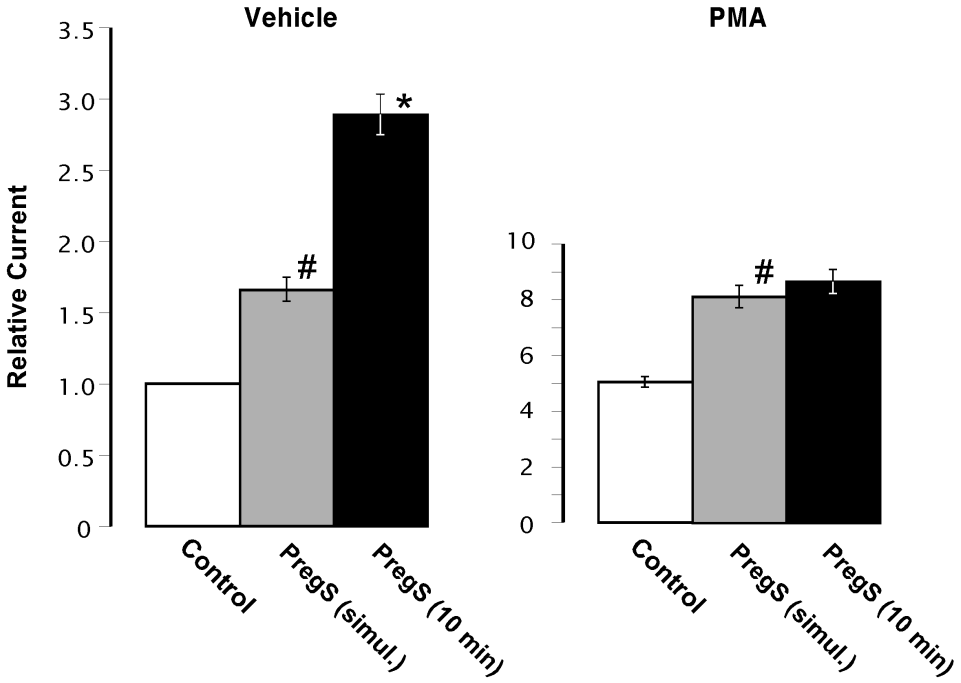
The Neuroactive Steroid Pregnenolone Sulfate Stimulates Trafficking of Functional NMDA Receptors to the Cell Surface via a Non-Canonical G-Protein and Ca^{++} Dependent Mechanism

Molecular Pharmacology



Supplemental Figure 1. Delayed potentiation is NMDAR specific. (A) Representative traces obtained from a *Xenopus* oocyte co-expressing NR1-1a/NR2A and α1β2γ2s receptors show typical responses to control applications of NMDA (300 μM) or GABA (500 μM), and applications of NMDA + PS and GABA + PS with or without a prolonged (10 min) pre-exposure to PS (100 μM). (B) Averaged peak current responses to NMDA + PS without (open bars) or after (solid bars) 10 min pre-exposure to PS. V_h = -70 mV. Responses are normalized to the response in the absence of PS (note that PS allosterically inhibits GABA responses but enhances NMDA responses). Statistical significance was assessed by 2-tail paired t-test. Error bars represent SEM.



Supplemental Figure. 2. Delayed potentiation is occluded by pretreatment with PMA. Response of oocytes expressing NR1-1a/NR2A receptors to 300 μM NMDA is potentiated by treatment with PMA (2 μM, 10 min). PMA-treated oocytes exhibit rapid potentiation by PregS, but delayed potentiation is absent. Left, vehicle treated oocytes, right, PMA treated (note difference in scale). Mean peak NMDA induced currents are expressed relative to the NMDA response prior to treatment with PMA and/or PregS. Error bars indicate s.e.m. Number of oocytes tested was 7 (vehicle) and 8 (PMA). # Significantly different from control.
* Significantly different from simultaneous addition of NMDA and PregS ($p < 0.001$, paired 2-tail t-test).