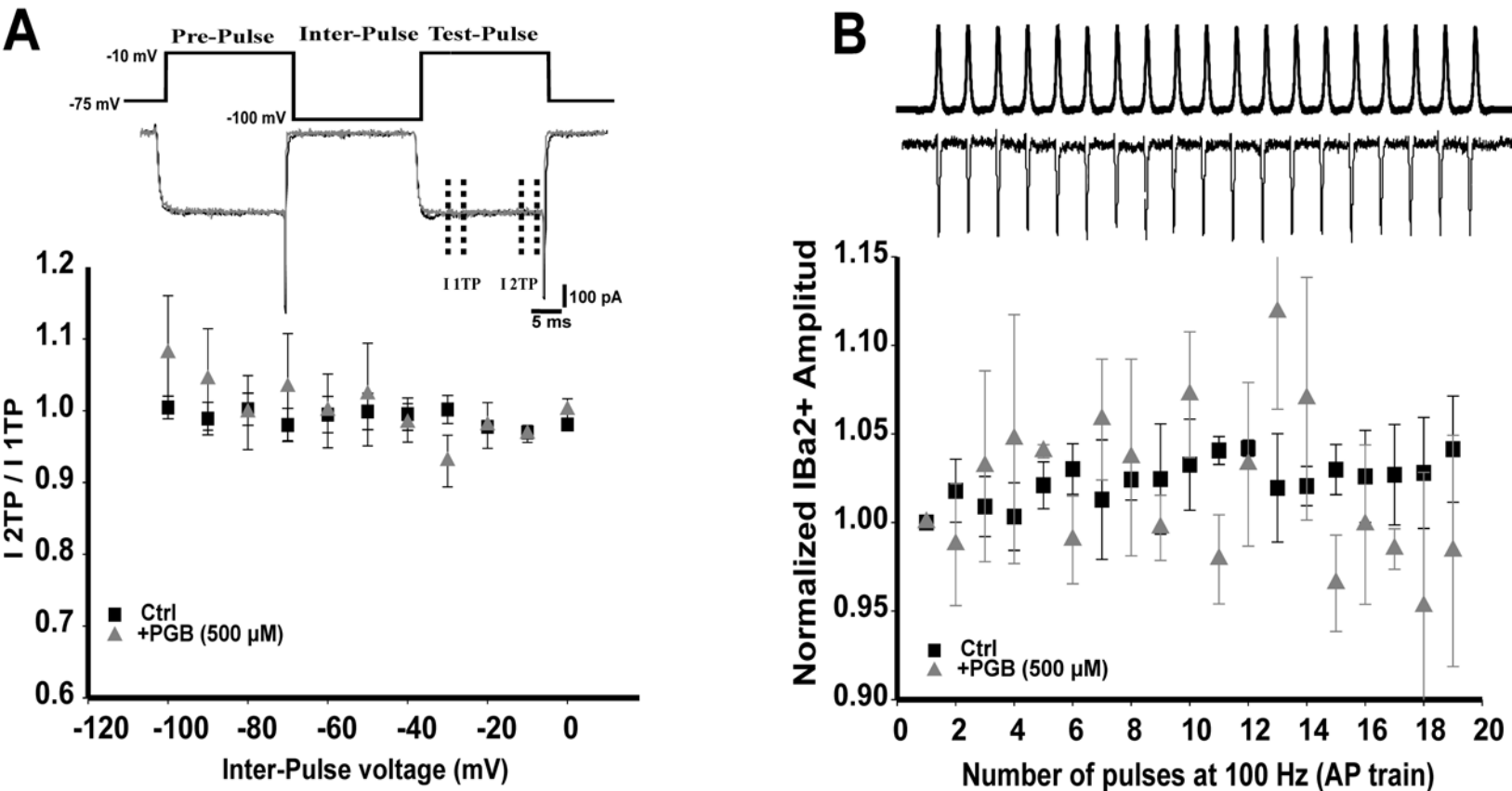


# Pregabalin modulation of neurotransmitter release is mediated by change in intrinsic activation/inactivation properties of CaV2.1 calcium channels.

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## Supplementary Figure 2



**Supplementary Figure 2.** Calcium-dependent processes are modified in presence of barium (Ba<sup>2+</sup>) as the charge carrier.

Ca<sup>2+</sup> was replaced by Ba<sup>2+</sup> (2 mM) as the charge carrier in the extracellular solution.

A. Inactivation protocol consisting of paired square pulses to -15 mV (pre-pulse, PP, and test pulse, TP) separated by depolarizing voltage steps (inter-pulse voltage VIP from -75 mV to -10 mV, 10 mV increments) together with representative calcium currents for an interpulse at -100 mV. are shown for control and +PGB 500  $\mu$ M condition. I2TP/I1TP ratio at Test-Pulse (TP) versus VIP is plotted (bottom). No significant differences were found between Ctrl and +PGB (500  $\mu$ M). B. Normalized current amplitudes during 100 Hz. train of APs. Ba<sup>++</sup> current facilitation observed in the absence of PGB (maximum of 104 $\pm$ 3%, n=3) was similar than in the presence of 500  $\mu$ M PGB (106 $\pm$ 6%, n = 3).