Modeling of perisomatic GABA-A mediated short-term synaptic plasticity on the hippocampal pyramidal neurons

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GABA mediated synaptic transmission is important for population oscillations and network plasticity. Perisomatic GABA-A synaptic transmission shares the dogma of short-term synaptic plasticity, but also has unique properties. Based on our patch clamp recordings from the pyramidal soma in CA1 region of mouse hippocampus, we expanded an existing synaptic phenomenological model. Our GABA-A data allows fitting of some of the model parameters during pair-pulse depression, post-tetanic depression and recovery of post-tetanic depression. We found our data did not support the hypothesis that there is an intermediate readily releasable pool inside the GABA presynaptic terminal. We also simulated data from CHL1 mutant mice, which had abnormal GABA-A synaptic transmission, and found the shift of TauR could significantly affect the short-term plasticity in the mutants. Our model could also explain how a change of intrinsic excitability of pyramidal neurons, through slow recovery of post-tetanic depression of GABA-A synapses, could facilitate CA1 long-term potentiation in the hippocampus. Thus, we conclude that disinhibition of pyramidal neurons by synaptic depression of GABA-A is an important factor in plasticity of neural network.

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References