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Temporal coding at the immature depolarizing gabaergic synapse

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Abstract

In the developing hippocampus, GABA exerts depolarizing and excitatory actions and contributes to the generation of neuronal network driven giant depolarizing potentials (GDPs). Here, we studied spike time coding at immature GABAergic synapses and its impact on synchronization of the neuronal network during GDPs in the neonatal (postnatal days P2-6) rat hippocampal slices. Using extracellular recordings, we found that the delays of action potentials (APs) evoked by synaptic activation of GABA(A) receptors are long (mean, 65 ms) and variable (within a time window of 10-200 ms). During patch-clamp recordings, depolarizing GABAergic responses were mainly subthreshold and their amplification by persistent sodium conductance was required to trigger APs. AP delays at GABAergic synapses shortened and their variability reduced with an increase in intracellular chloride concentration during whole-cell recordings. Negative shift of the GABA reversal potential (EGABA) with low concentrations of bumetanide, or potentiation of GABA(A) receptors with diazepam reduced GDPs amplitude, desynchronized neuronal firing during GDPs and slowed down GDPs propagation. Partial blockade of GABA(A) receptors with bicuculline increased neuronal synchronization and accelerated GDPs propagation. We propose that spike timing at depolarizing GABA synapses is determined by intracellular chloride concentration. At physiological levels of intracellular chloride GABAergic depolarization does not reach the action potential threshold and amplification of GABAergic responses by non-inactivating sodium conductance is required for postsynaptic AP initiation. Slow and variable excitation at GABAergic synapse determines the level of neuronal synchrony and the rate of GDPs propagation in the developing hippocampus.

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Keywords

Development, Gamma aminobutyric acid, Hippocampus, Neonatal