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Excitatory actions of GABA in the intact neonatal rodent hippocampus in vitro

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Abstract

The excitatory action of GABA is considered to be a hallmark of the developing nervous system. However, in immature brain slices, excitatory GABA actions may be secondary to neuronal injury during slice preparation. Here, we explored GABA actions in the rodent intact hippocampal preparations and at different depths of hippocampal slices during the early postnatal period (post-natal days [P] 1- 7). We found that in the intact hippocampus at P1-3: (i) GABA exerts depolarizing action as seen in cell-attached single GABA(A) channel recordings; (ii) GABA(A) receptor (GABA(A)-R) agonist isoguvacine and synaptic activation of the GABA(A)- Rs increase the frequency of multiple unit activity (MUA) and the frequency of the network-driven Giant Depolarizing Potentials (GDPs); and that (iii) NKCC1 antagonist bumetanide suppresses GDPs and the excitatory actions of isoguvacine. In the hippocampal slices at P2-5, isoguvacine and synaptic activation of GABA(A)- Rs evoked excitatory responses at all slice depths, including surface and core. Thus, GABA exerts excitatory actions in the intact hippocampus (P1-3) and at all depths of hippocampal slices (P2-5). Therefore, the excitatory actions of GABA in hippocampal slices during the first postnatal days are not due to neuronal injury during slice preparation, and the trauma-related excitatory GABA actions at the slice surface are a fundamentally different phenomenon observed during the second postnatal week. © 2013 Valeeva, Valiullina and Khazipov.

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Keywords

Development, GABA, Giant depolarizing potentials, Hippocampus, Inhibitory postsynaptic potentials, Intracellular chloride, NKCC1