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Ontogeny of kainate-induced gamma oscillations in the rat CA3 hippocampus in vitro

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Abstract

© 2015 Tsintsadze, Minlebaev, Suchkov. GABAergic inhibition, which is instrumental in the generation of hippocampal gamma oscillations, undergoes significant changes during development. However, the development of hippocampal gamma oscillations remains largely unknown. Here, we explored the developmental features of kainate-induced oscillations (KA-Os) in CA3 region of rat hippocampal slices. Up to postnatal day P5, the bath application of kainate failed to evoke any detectable oscillations. KA-Os emerged by the end of the first postnatal week; these were initially weak, slow (20-25 Hz, beta range) and were poorly synchronized with CA3 units and synaptic currents. Local field potential (LFP) power, synchronization of units and frequency of KA-Os increased during the second postnatal week to attain gamma (30-40 Hz) frequency by P15-21. Both beta and gamma KA- Os are characterized by alternating sinks and sources in the pyramidal cell layer, likely generated by summation of the action potential—associated currents and GABAergic synaptic currents, respectively. Blockade of GABA(A) receptors with gabazine completely suppressed KA-Os at all ages indicating that GABAergic mechanisms are instrumental in their generation. Bumetanide, a NKCC1 chloride co-transporter antagonist which renders GABAergic responses inhibitory in the immature hippocampal neurons, failed to induce KA-Os at P2-4 indicating that the absence of KA-Os in neonates is not due to depolarizing actions of GABA. The linear developmental profile, electrographic features and pharmacological properties indicate that CA3 hippocampal beta and gamma KA- Os are fundamentally similar in their generative mechanisms and their delayed onset and developmental changes likely reflect the development of perisomatic GABAergic inhibition.

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Keywords

Beta, CA3 region, Gaba receptors, Gamma, Hippocampal, Kainate, NKCC1, Oscillations