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EARLY GAMMA OSCILLATIONS

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Abstract—Gamma oscillations have long been considered to emerge late in development. However, recent studies have revealed that gamma oscillations are transiently expressed in the rat barrel cortex during the first postnatal week, a ''critical'' period of sensory-dependent barrel map formation. The mechanisms underlying the generation and physiological roles of early gamma oscillations (EGOs) in the development of thalamocortical circuits will be discussed in this review. In contrast to adult gamma oscillations, synchronized through gamma-rhythmic perisomatic inhibition, EGOs are primarily driven through feedforward gamma-rhythmic excitatory input from the thalamus. The recruitment of cortical interneurons to EGOs and the emergence of feedforward inhibition are observed by the end of the first postnatal week. EGOs facilitate the precise synchronization of topographically aligned thalamic and cortical neurons. The multiple replay of sensory input during EGOs supports long-term potentiation at thalamocortical synapses. We suggest that this early form of gamma oscillations, which is mechanistically different from adult gamma oscillations, guides barrel map formation during the critical developmental period. **© 2013 IBRO. Published by Elsevier Ltd. All rights reserved.**

Key words: gamma oscillations, neonate, cortex, thalamus, electroencephalogram.

Contents

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Abbreviations: CSD, current source density; dLGN, dorsal lateral geniculate nucleus; EEG, electroencephalograms; EGO, early gamma oscillation; EPSC, excitatory postsynaptic current; IPSC, inhibitory postsynaptic current; L2/3, layer 2/3; L4, layer 4; LFP, local field potential; MUA, multi-unit activity; NMDA, N-methyl-D-aspartic acid; P0, postnatal day 0; PSTH, peristimulus time histogram; PTX, picrotoxin; STDP, spike timing-dependent plasticity; VPM, ventral posteromedial nucleus of the thalamus; VSD(I), voltage-sensitive dye (imaging).

INTRODUCTION

Neuronal synchronization in gamma (30–90 Hz) oscillations is fundamental for cortical functions. In the adult brain, gamma oscillations have been hypothesized to subserve perceptual binding and facilitate the transient formation of functional assemblies through the synchronization of neuronal firing, thereby supporting synaptic plasticity. A number of excellent reviews have addressed the physiological mechanisms underlying the generation and role of gamma oscillations in cognitive functions in the adult brain (Gray and Singer, 1989; Singer and Gray, 1995; Fries et al., 2001; Buzsaki and Draguhn, 2004; Fries, 2009; Wang, 2010; Whittington et al., 2011; Buzsaki and Wang, 2012). Considerable evidence indicates that the synchronization of neurons in gamma oscillation is based on synchronous inhibition through fast-spiking perisomatic parvalbumin-containing basket interneurons (Bartos et al., 2007; Whittington et al., 2011; Buzsaki and Wang, 2012). Gamma oscillations have long been considered to emerge relatively late in development. In humans, gamma oscillations emerge several months after birth and show a developmental increase until adulthood (for review, (Uhlhaas et al., 2010). Similarly, in rodents, gamma oscillations emerged during the second postnatal week (Leinekugel et al., 2002; Lahtinen et al., 2002; Doischer et al., 2008). This delayed development of gamma oscillations likely reflects the delayed maturation of the perisomatic inhibition. Indeed, in rodents, basket cells develop fast-spiking features, forming synapses with excitatory cells and establishing chemical and electrical synapses with other basket cells from the end of the first postnatal week, and the maturation of these cells proceeds through the first postnatal month (Du et al., 1996; Chattopadhyaya et al., 2004; Daw et al., 2007; Huang et al., 2007; Doischer et al., 2008; Okaty et al., 2009; Wang and Gao, 2010; Goldberg et al., 2011;

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