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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.

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

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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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Pangratz-Fuehrer and Hestrin, 2011; Yang JM et al., 2012). The long-range gamma synchronization of neuronal activity also depends on the development of supragranular cortical layers and horizontal intracortical connections that also show delayed development from the second postnatal week in rodents (Luhmann et al., 1986; Bureau et al., 2004; Borgdorff et al., 2007). Therefore, similar developmental profiles of gamma oscillations, perisomatic inhibition and intracortical connectivity fit the widely-accepted theory of gamma rhythmogenesis.

Recent studies have revealed that oscillations at gamma frequencies are transiently expressed in the rat barrel cortex during the first postnatal week (Yang et al., 2009; Minlebaev et al., 2011; Yang JW et al., 2012), well before the emergence of perisomatic inhibition and the development of supragranular layers. In the present review, we will describe the distinct features, generative mechanisms and potential physiological roles of these early gamma oscillations (EGOs) during cortical development.

EGOs: electrographic features

EGOs are short-lived oscillations lasting approximately 200 ms in the gamma frequency range (peak frequency about 55 Hz), reliably evoked through sensory stimulation (Minlebaev et al., 2011; Yang JW et al., 2012). EGOs can also occur spontaneously (Yang et al., 2009) in the rat barrel cortex during the first postnatal week. EGOs characterize the initial part of a complex sensory-evoked response, which also comprises spindle-burst (8–25 Hz) oscillations (Khazipov et al., 2004; Minlebaev et al., 2007; Yang JW et al., 2012). EGOs and spindle-bursts, in turn, are nested in a delta-wave lasting approximately 500 ms (Marcano-Reik and Blumberg, 2008; Minlebaev et al., 2009). EGOs are best observed in the layer 4 (L4) of a cortical barrel column after a brief deflection of a single principal whisker. However, EGOs are hard to detect in responses evoked through the stimulation of multiple whiskers, during which slower spindle-burst oscillations dominate the activity (Minlebaev et al., 2007, 2009; Colonnese et al., 2010).

Developmental profile of EGOs and relevance to premature human electroencephalograms (EEGs)

As a part of the immature sensory response, EGOs are expressed in the barrel cortex during a restricted developmental time window, i.e., the first postnatal week, where the cortical barrel map is formed, representing a critical period of sensory-dependent thalamocortical plasticity in the barrel cortex (Fig. 1) (Van der Loos and Woolsey, 1973; Fox, 1992, 2002; Erzurumlu and Gaspar, 2012). The close of the critical period in the L4 barrel cortex by the end of the first postnatal week coincides with an abrupt disappearance of EGOs and immature bursting (Colonnese et al., 2010; Minlebaev et al., 2011). This coincidence suggests an involvement of the immature activity patterns, including EGOs, in barrel map development during the critical period.

Although there is agreement on the developmental disappearance of EGOs in the barrel cortex after P7–8, the onset of these oscillations is somewhat controversial. Yang and colleagues have reported that EGOs are observed at birth (postnatal day 0 (P0)) (Yang JW et al., 2012), whereas Minlebaev and colleagues (Minlebaev et al., 2011) have observed only delta-waves, occasionally organized in groups of 2–3 delta-waves, without any significant patterning of activity in the gamma frequency at P0–1 (see an example response at P1 in Fig. 1B), and an emergence of gamma oscillations starting from postnatal day P2 (Minlebaev et al., 2011).

In human premature neonates aged <25 gestational weeks, corresponding to the earliest stages of thalamocortical development compared with the late embryonic/term rat (Higashi et al., 2002; Kostovic and Judas, 2010), intermittent ''smooth'' delta waves lacking rapid oscillatory components (''brushes'') dominate the electroencephalographic activity. These delta wave oscillations, reminiscent of the responses evoked through sensory stimuli in the P0–1 rat barrel cortex shown in a previous study (Minlebaev et al., 2011), become intermixed with rapid rhythms (''delta-brushes'') by the seventh month of gestation in humans. Delta-brushes constitute dominant activity patterns in all cortical areas and fade near term (Anderson et al., 1985; Stockard-Pope et al., 1992; Lamblin et al., 1999; Scher, 2006; Andre et al., 2010). Similarly in rats, delta-brushes are also reliably evoked through sensory stimuli in the somatosensory and visual cortex of premature neonates (Hrbek et al., 1973; Milh et al., 2007; Colonnese et al., 2010; Stjerna et al., 2012). Therefore, from the developmental standpoint, a similar developmental sequence of smooth delta waves followed by delta-brushes is also expected in the rat. Thus, it would be of interest to know whether delta waves without EGOs are present in fetuses and preterm neonates in rats, which display EGOs at birth, and mice, in which barrel map development is delayed 1–2 days compared with rats (Rhoades et al., 1990; Schlaggar et al., 1993; Rebsam et al., 2002).

The rapid rhythms of delta-brushes in premature human neonates typically occur within an 8–25 Hz frequency band, and the association of delta-brushes with an activity at gamma frequency has not been reported (Ellingson, 1958; Dreyfus-Brisac, 1962; Parmelee et al., 1969; Nolte et al., 1969; Goldie et al., 1971; Watanabe and Iwase, 1972; Hrbek et al., 1973; Engel, 1975; Vanhatalo et al., 2002, 2005; Milh et al., 2007; Colonnese et al., 2010; Dreyfus-Brisac, 1962). Therefore, it remains unknown whether EGOs are present in humans. Notably, in the rat, EGOs are restricted to the L4 of a single cortical column and are hardly detected, even during intracortical recordings near the cortical surface (Fig. 2C) (Minlebaev et al., 2011), suggesting that EGOs would also be hard to detect using conventional scalp EEG recordings from premature human babies. However, it might become

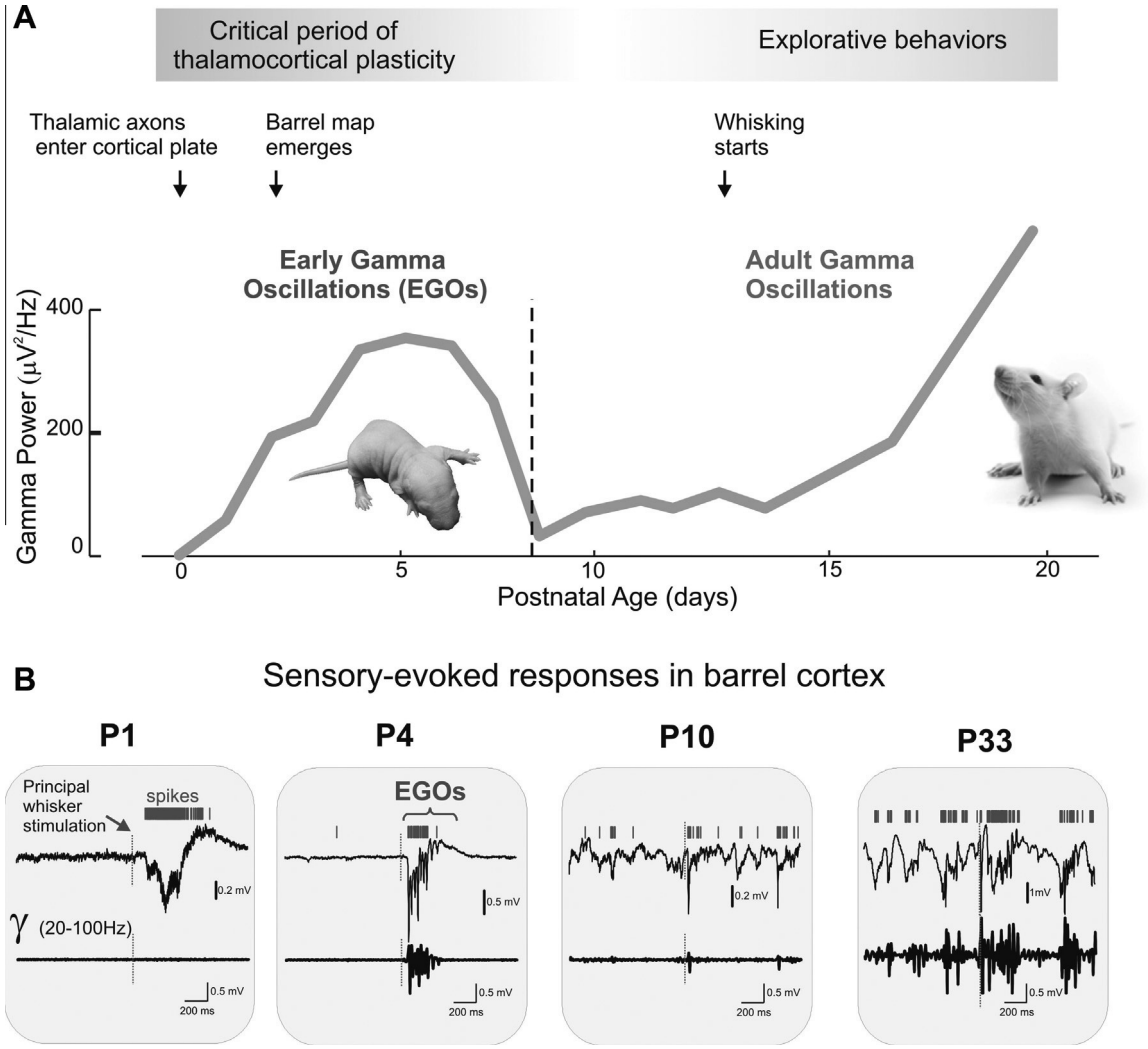

Fig. 1. Development of gamma oscillations in the rat somatosensory cortex. (A) Age-dependent power of sensory-evoked gamma oscillations in the L4 of the somatosensory barrel cortex. Early gamma oscillations (EGOs) are expressed during the critical period of thalamocortical plasticity in the whisker-barrel system and abruptly disappear at the end of the critical period. The emergence of adult gamma oscillations is associated with the onset of explorative behaviors and active whisking. (B) Sensory responses evoked through brief single whisker deflection in the L4 layer of the corresponding cortical column at different postnatal ages (at P1, recordings are from the dense cortical plate). Example traces show the local field potential (black traces) and multiple unit activity (spikes, red bars). The stimulus is indicated using vertical red lines. Below, gamma band (20–100 Hz)-filtered traces. Note the lack of gamma oscillations at P1, EGOs evoked in a P4 rat, disappearance of EGOs in a P10 rat and an UP state associated with gamma activity in a P33 rat. Note the developmental increase in pre-stimulus continuous baseline activity occurring in a counterpoint to the disappearance of early gamma oscillations. Adapted from Minlebaev et al. (2011) with permission.

feasible to detect EGOs through reducing the electrode size, increasing topographic stimulations and taking advantage of the stimulus-lock feature of EGOs, which facilitates the detection of these oscillations in an average response.

As scalp EEG recordings in humans only reveal a progressive increase of gamma activity during the postnatal and adolescent period (Uhlhaas et al., 2010), intracortical recordings from the cortical surface in the rat also show only a progressive developmental increase in gamma power from the second postnatal week onward, without revealing the transient expression of gamma activity, which is restricted to L4, during the first postnatal week (Minlebaev et al., 2011). These later emerging gamma oscillations likely correspond to classical inhibition-based gamma oscillations, and their development correlates with emerging explorative functions and, in the case of the barrel system, is also associated with an onset of active whisking (Landers and Philip, 2006; Colonnese et al., 2010).

Time lock to stimulus

In the adult brain, sensory-induced cortical gamma oscillations typically occur with variable delays from trial to trial after stimulus onset and therefore, these induced gamma oscillations are essentially lost during multiple response averaging (Tallon-Baudry and Bertrand, 1999). Thus, EGOs are different from the induced adult gamma oscillations, as their temporal relationship with the stimulus is preserved from trial to trial (Fig. 2). As a result, stimulus-locked EGOs are apparent in the stimulus-triggered average local field potentials (LFPs) and spike time histograms during the first 200 ms of the

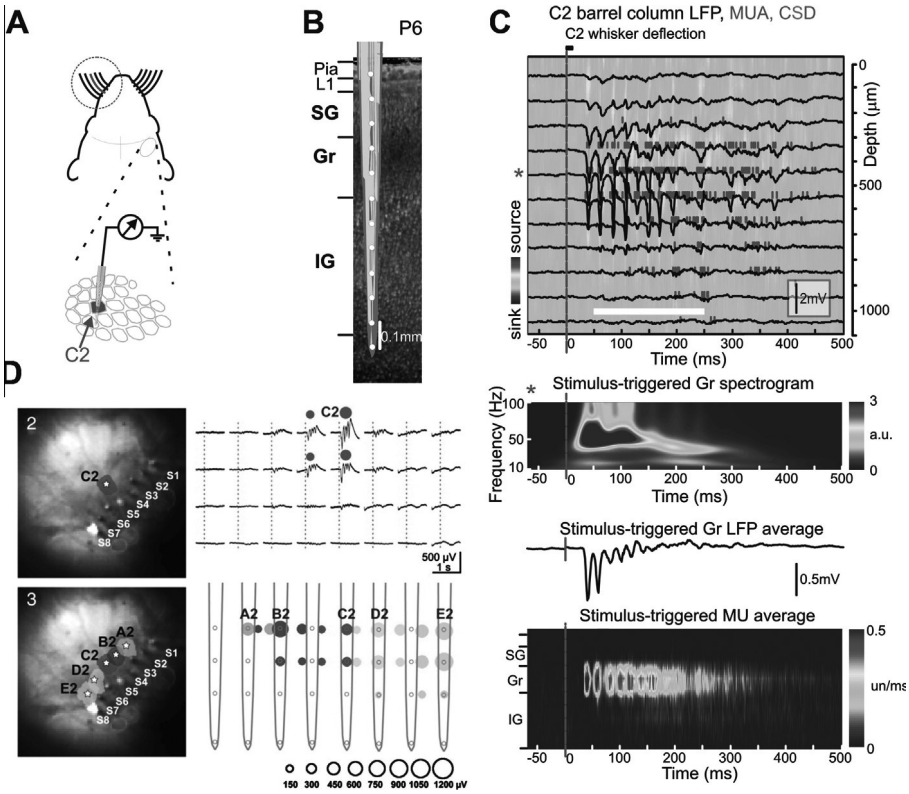

Fig. 2. Main features of the early gamma oscillations. (A) Scheme of the experimental setup for recordings of electrical activity from a cortical barrel column. (B) Recording sites of a multi-electrode array overlaid on a Ctip-2-stained coronal slice. (C) Sensory response evoked through C2 principal whisker (PW) stimulation at different depths of the C2 cortical barrel column (LFP-black traces, bandpass 5–100 Hz; MUA-red bars overlaid on a color-coded current source density plot (CSD)). Below: the stimulus-triggered averages (n = 100) for (L4) (red asterisk) wavelet spectrogram, average L4 layer LFP and MUA PSTHs across layers. Recordings from a P6 rat. (D) Simultaneous voltage-sensitive dye imaging (VSDI) and the multi-electrode recording of sensory-evoked cortical responses. Mechanical stimulation of the C2 whisker in a P1 rat elicits local VSDI (left) and local electrophysiological (right) responses in the C2 barrel (*D*2). Color-coded localization of the evoked cortical VSDI (left) and electrophysiological (right) responses to stimulation of single whiskers in arc 2 (*D*3). The first negative peak amplitude of the electrophysiological response corresponds to the size of the color-coded circles as shown below the graph. Adapted from Minlebaev et al. (2011) (A–C) and Yang JW et al. (2012) (D) with permission.

sensory-evoked response (Fig. 2C) (Minlebaev et al., 2011; Yang JW et al., 2012).

Although the stimulus-locked feature and dominant frequency of EGOs do not change through the first postnatal week, delays in EGOs from the stimulus are strongly age dependent, attaining values of approximately 50–60 ms at P2–3 and showing a nearly twofold reduction to 30 ms at P7. This reduction likely reflects an increase in the velocity of axonal spikes transmission along the three-synaptic somatosensory pathway. In this regard, it is surprising that the frequency of EGOs remains unchanged during this period. EGOs are primarily driven through the thalamic gamma oscillator (see below), suggesting a fundamental mechanism of thalamic gamma rhythmogenesis, which is transiently expressed and facilitates the maintenance of gamma activity at a fixed frequency during the first postnatal week; notably, this mechanism occurs independent of the axonal conduction velocity.

Spatial characteristics of EGOs

The spatial properties of EGOs were addressed using voltage-sensitive dye (VSD) imaging, electrophysiological recordings of the LFPs and multi-unit activity (MUA) in the barrel cortex using multishank electrode arrays (Fig. 2) (Minlebaev et al., 2011; Yang JW et al., 2012). Both approaches revealed the compartmentalization of EGOs within a cortical barrel receiving input from the corresponding whisker. In VSD recordings, cortical areas responding to the stimulation of a single whisker were 200–300 microns in diameter in P0–5 rats, which corresponds to the size of the barrel column, and these responses barely overlapped (Fig. 2D) (Yang JW et al., 2012). In older animals, the cortical areas activated through single whisker stimulation increased to involve adjacent columns (see also (Borgdorff et al., 2007)). Extracellular LFP and multiple unit recordings also showed segregated responses between neighboring barrel columns (Minlebaev et al., 2011; Yang JW et al., 2012).

The confinement of EGOs to a single cortical barrel suggests support through topographic synapses connecting neurons in corresponding thalamic barreloids and cortical barrels. These results also indicate that functional connectivity in the thalamocortical system is precise during the first postnatal week. However, non-topographic inputs from adjacent barreloids also exist

but remain weak and subthreshold, supported through whole-cell recordings of the responses evoked through adjacent whisker stimulation (Minlebaev and Khazipov, unpublished observation). These "latent" non-topographic synapses likely undergo pruning at a critical period similar to pruning of the aberrant connections described during the development of various circuits (Crepel et al., 1976; Rakic, 1977; Campbell and Shatz, 1992; Katz and Shatz, 1996; Lichtman and Colman, 2000; Chen and Regehr, 2000; Debski and Cline, 2002; Ruthazer et al., 2003). However, these subthreshold non-topographic inputs might also provide the basis for an expansion of synapses established through neurons in neighboring barreloids to sensory-deprived cortical territories (Van der Loos and Woolsey, 1973; Simons and Land, 1987; Fox, 1992, 2002).

In adults, gamma oscillations facilitate the synchronization of activity in adjacent cortical columns (Engel et al., 1990), which primarily occurs at the level of supragranular cortical layers 2/3 (L2/3), displaying the highest power of gamma activity (Minlebaev et al., 2011). However, during the first postnatal week, L2/3 are weakly activated during sensory responses (Armstrong-James, 1975; Stern et al., 2001; Bureau et al., 2004; Minlebaev et al., 2011). The development of L4 → L2/3 synapses rapidly occurs during the second postnatal week (Micheva and Beaulieu, 1996; Stern et al., 2001; Bender et al., 2003; Maravall et al., 2004; Bureau et al., 2004), also referred to as a critical period for these connections (Stern et al., 2001; Feldman and Brecht, 2005; Feldman, 2009). L2/3 → L2/3 synapses are also initially sparse and start to support the horizontal spread of activity from the end of the first postnatal week (Borgdorff et al., 2007; Gireesh and Plenz, 2008; Yang JW et al., 2012). Thus, the confinement of EGOs to a single column likely reflects the immaturity of the supragranular layers.

Synaptic correlates of EGOs

Whole cell recordings of synaptic currents from L4 neurons and pharmacological analyses revealed several features of EGOs that are remarkably distinct from adult gamma oscillations (Figs. 3 and 4) (Minlebaev et al., 2011): (i) EGOs are primarily generated through gamma-rhythmic glutamatergic excitatory postsynaptic currents (EPSCs), which generate active sinks of field EGOs in L4 (Fig. 4F); in contrast, adult gamma oscillations are primarily generated through gamma-rhythmic inhibitory postsynaptic currents (IPSCs) that generate active sources of field gamma oscillations (Fig. 3C, D) (Penttonen et al., 1998; Csicsvari et al., 2003; Mann et al., 2005; Hasenstaub et al., 2005; Oren et al., 2010); (ii) The engagement of IPSCs to EGOs is age-dependent: before P5, only few neurons display any IPSCs, and if present, these responses occur at the end of EGOs; by P5–7, IPSCs show gamma-rhythmicity during EGOs. However, the relative conductance and gamma-power of IPSCs are inferior to EPSCs during this period, in contrast to adult gamma oscillations, characterized by the domination of IPSCs (Fig. 3E) (Mann et al., 2005; Hasenstaub et al., 2005); (iii) The blockade of cortical inhibition does not modify EGOs at P2–4, but strongly reduces these oscillations at P6 (Fig. 3A). Thus, EGOs primarily result from a gamma-rhythmic excitatory input to L4 neurons, constituting the only drive for EGOs during the first postnatal days. Local inhibitory circuits, central for gamma rhythmogenesis in adult networks, are progressively recruited and support EGOs only by the end of the first postnatal week.

Thus, what is the source of the gamma-rhythmic glutamatergic EPSCs that drive EGOs in L4? In adults, L4 neurons receive excitatory inputs from the thalamus (approximately 15% of the total number of inputs (Benshalom and White, 1986)), and the remaining inputs are generated from the adjacent excitatory neurons of the home barrel (Lefort et al., 2009; Feldmeyer, 2012). Both types of synapses, thalamic and local, generate active sinks in L4. Although the involvement of thalamic inputs is clear, as the troughs of gamma oscillations and associated firing of cortical neurons are tightly locked, and these inputs occur at standard delays after the activation of thalamic VPM units (Minlebaev et al., 2011; Yang JW et al., 2012), little is known about L4–L4 connectivity during the first postnatal week and the participation of these synapses in the generation of EGOs. If present, these connections would cooperate with the thalamic input, and given the intrabarrel connectivity pattern, these connections would contrast the topographic organization of the cortical activity (Douglas et al., 1995).

Electrical synapses between excitatory L4 neurons present another mechanism that could potentially support neuronal synchronization during EGOs. The level of connectivity between cortical neurons via gap junctions attains nearly 40–70% during the first postnatal week (Connors et al., 1983; Yu et al., 2012). Interestingly, lineage-dependent transient electrical coupling between the pyramidal cortical cells guides the formation of excitatory synapses in ontogenic columns (Yu et al., 2012). Several correlated activity patterns, synchronized through gap junctions, have been described in slices of the developing cortex *in vitro*, including correlated calcium waves, neuronal domains, spontaneous plateau assemblies, and carbachol-induced beta oscillations (Yuste et al., 1992, 1995; Kandler and Katz, 1998; Peinado, 2000, 2001; Dupont et al., 2006; Crepel et al., 2007). Gap junctions between excitatory neurons are eliminated after the first postnatal week (Connors et al., 1983; Yu et al., 2012) in a counterpoint to the emergence of the electrical and chemical synapses between the fast-spiking interneurons (instrumental for adult gamma rhythmogenesis) during the second postnatal week (Yang JM et al., 2012). Thus, the participation of electrical synapses in the synchronization of EGOs is a plausible mechanism. However, the existence of electrical synapses between L4 stellate cells during the first postnatal week and the roles of these cells in neuronal synchronization during EGOs also remain unknown.

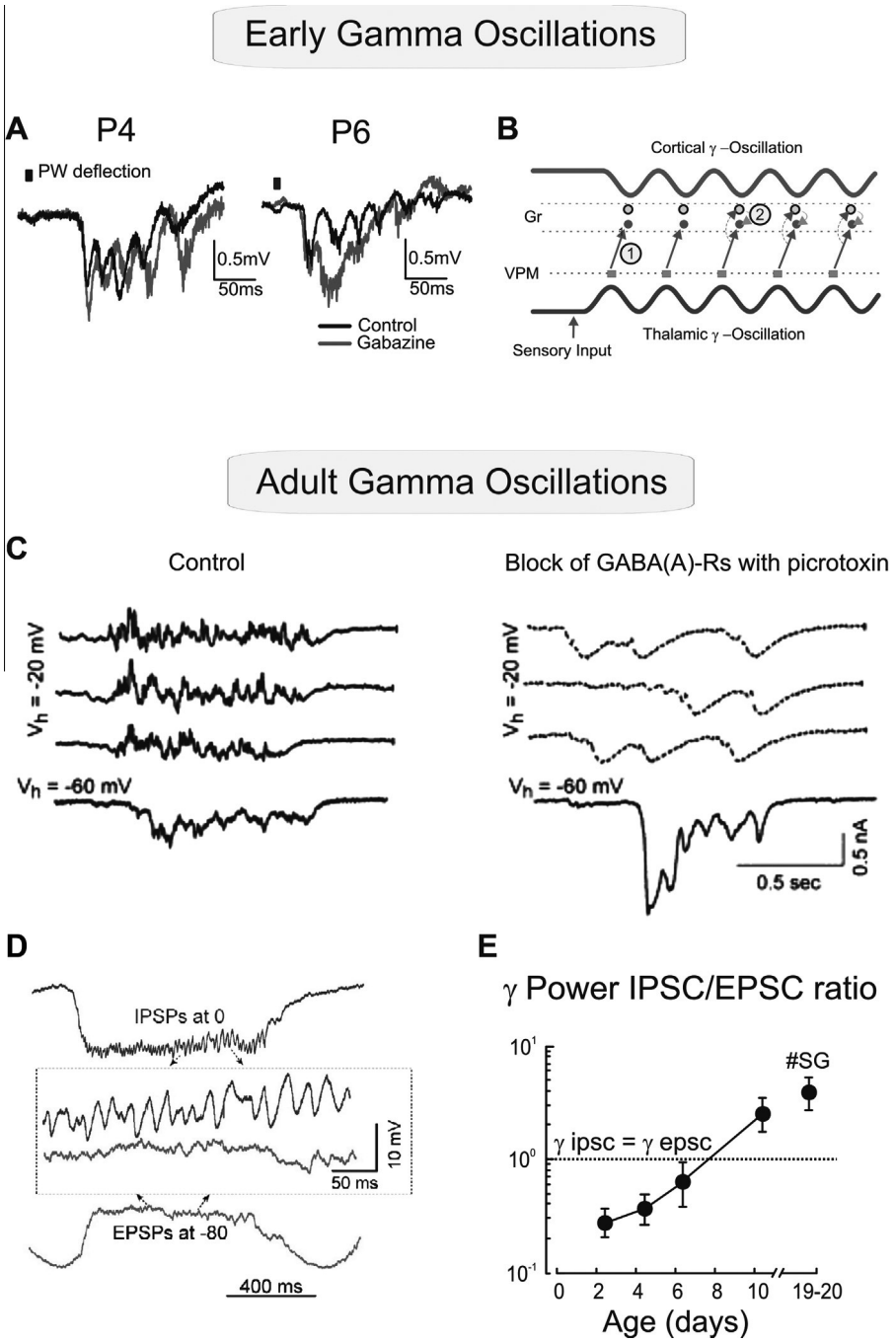

Fig. 3. Developmental recruitment of GABAergic inhibition in the synchronization of gamma oscillations. (A) Extracellular EGOs evoked through principal whisker deflection in L4 in P4 and P6 rats before (black traces) and after (red traces) the epipial application of the GABA(A) receptor blocker gabazine. Note that the blockade of inhibition does not affect EGOs at P4 but reduces these oscillations at P6. (B) Proposed network EGOs model. Sensory input from a whisker activates the gamma oscillator in the thalamic barreloid, which imposes topographic feedforward synchronization in the corresponding cortical barrel [1]. Cortical interneurons become involved in EGOs in an age-dependent manner starting from ~P5 [2]. (C) Three consecutive examples of synaptic currents arriving in a layer 5 pyramidal cell *in vitro* while voltage is clamped near the reversal potential of the UP state (−20 mV in this cell) and one trace at −60 mV. The local application of picrotoxin (right traces) results in a marked decrease in higher-frequency components of the synaptic currents. (D) Intracellular recordings in the ferret prefrontal cortex illustrating the amplitude and time course of excitation-dominated synaptic barrages recorded at −80 mV (red) and inhibition-dominated barrages recorded at 0 mV (blue), for two representative UP states. Membrane potentials are expanded further for illustration (inset). (E) Age-dependence of the IPSC/EPSC gamma power ratio. Adapted from Minlebaev et al. (2011) (A, B, E) and Hasenstaub et al. (2005) (C, D) with permission.

Thalamic origin of EGOs

Sensory input to the cortex is relayed via the thalamus (Castro-Alamancos, 2004; Jones, 2009). Several lines of evidence obtained using simultaneous recordings from the topographically aligned neurons in the thalamus and cortex indicate that gamma-rhythmic excitation is relayed to L4 neurons during cortical EGOs

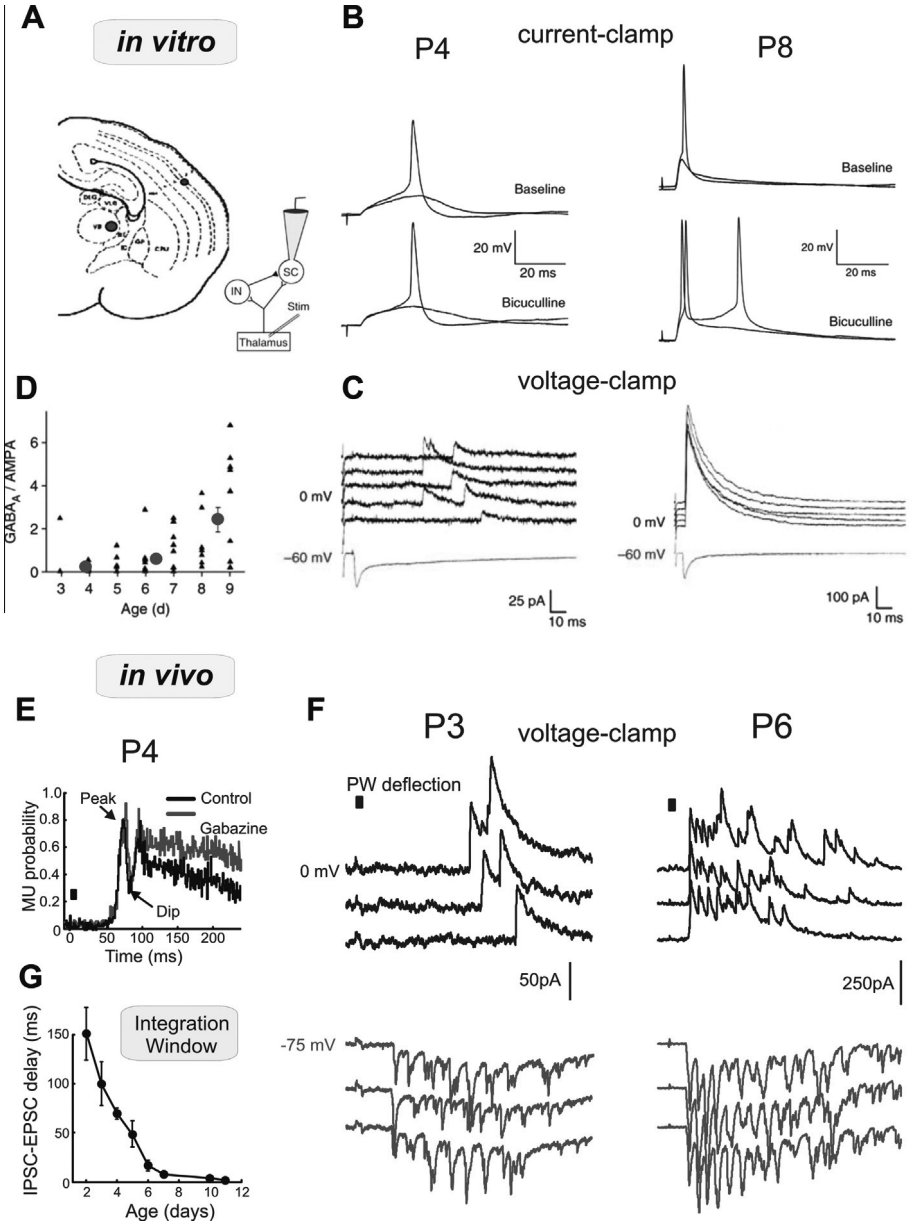

Fig. 4. Development of the feedforward inhibition. (A) Drawing of a thalamocortical slice. Responses evoked through the stimulation of the thalamus are recorded in a L4 stellate cell. (B) In current-clamp mode, the blockade of GABA(A) receptor-mediated transmission with bicuculline affects thalamocortical-evoked responses in stellate cells at P8 but not at P4. (C) In voltage clamp mode, IPSCs are only weakly and unreliably activated through thalamocortical input in the neonate, but strongly activated by P7–9. (D) Age-dependence of the GABA(A) receptor-mediated IPSC ratio to EPSC. (E) In a P4 rat *in vivo*, the blockade of cortical GABA(A) receptors with gabazine does not affect the inhibition of spikes (dip) after the sensory-evoked potential. (F) Whole-cell responses evoked through principal whisker stimulation in L4 neurons at P3 and P6 recorded in voltage clamp to separate IPSCs (blue top traces at 0 mV) and EPSCs (red bottom traces at −75 mV). (G) Age dependence of the integration window, defined as the difference between EPSCs and IPSCs onset delays. Adapted from Daw et al. (2007) (A–D) and Minlebaev et al. (2011) (E–G) with permission.

through thalamic inputs (Minlebaev et al., 2011; Yang JW et al., 2012). First, whisker stimulation evokes gamma rhythmic MUA in the corresponding barreloid in the ventral posterior medial (VPM) thalamic relay nucleus; this thalamic gamma activity is highly coherent with cortical EGOs such that thalamic units fire approximately 7 ms ahead of cortical L4 neurons (Fig. 5B). This thalamocortical binding is maintained for eight EGO cycles, indicating a multiple replay of a sensory input in topographic thalamocortical microcircuits (Minlebaev et al., 2011). Second, the electrical microstimulation of a barreloid reliably evokes cortical EGOs, whereas barreloid lesions completely eliminate both sensory-evoked and spontaneous EGOs (Fig. 5C) (Yang JW et al., 2012). Finally, none of the patterns of correlated activity in the isolated cortical network *in vitro* correspond to EGOs observed *in vivo* (Khazipov and Luhmann, 2006; Allene and Cossart, 2010). Thus, thalamic gamma oscillator is instrumental for the generation of cortical EGOs. However, how

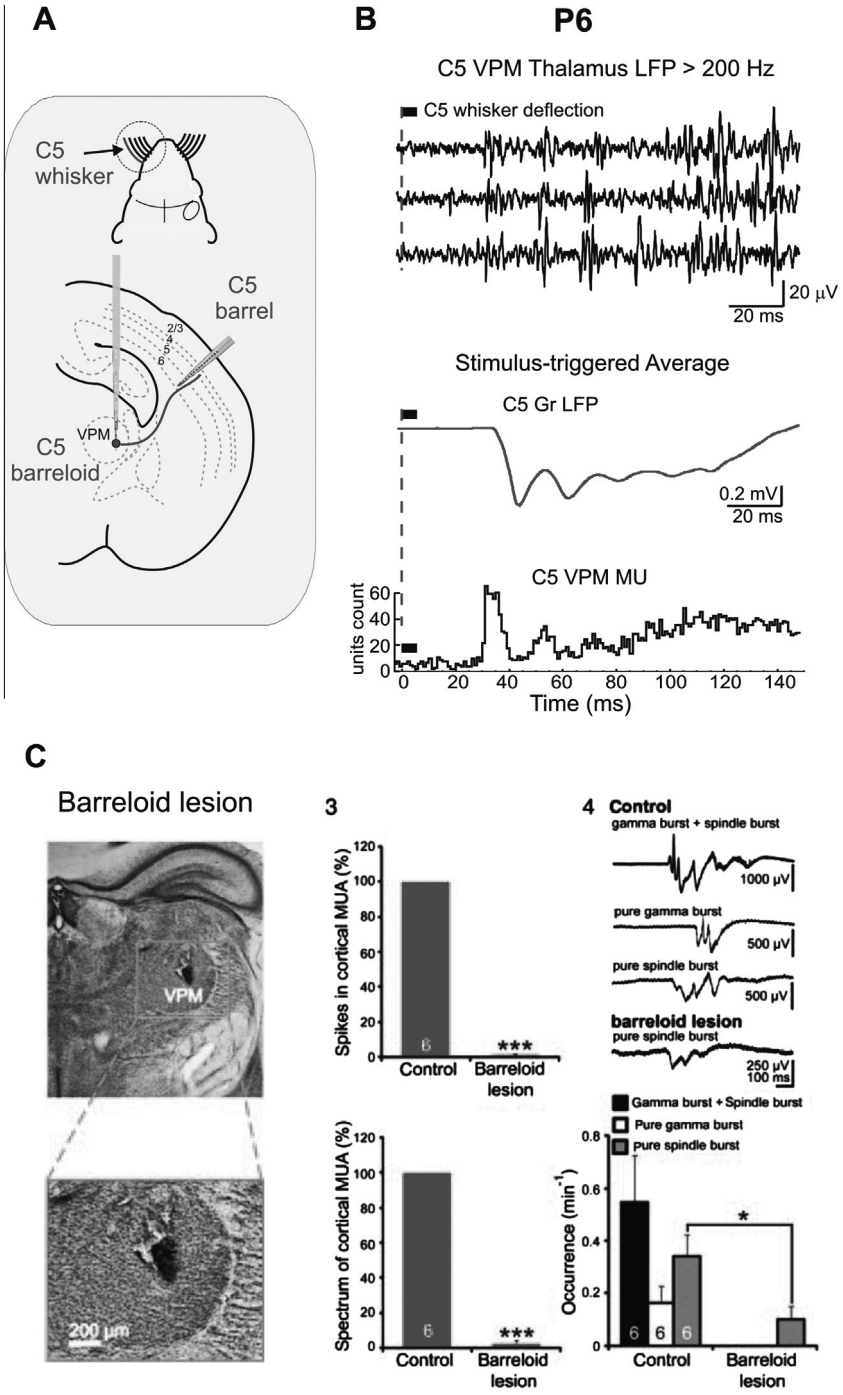

Fig. 5. Thalamic origin of the early gamma oscillations. (A) Experimental setup for simultaneous recordings of single whisker-evoked responses in the corresponding VPM barreloid and cortical barrel column in a P6 rat *in vivo*. (B) Three sequential responses to C5 whisker deflections in the C5 barreloid of VPM thalamus (top) and average L4 LFP (*red*) and a histogram of spikes in C5 barreloid (*black histogram*) from 100 deflections. (C) Local lesion of the thalamus (left panels) blocks spontaneously occurring cortical gamma burst activity. The pooled data from P1 rats illustrating the relative number of MUA spikes and the power of the MUA spectrum in the 30–60 Hz range (panel 3). An example of spontaneous gamma and spindle bursts before and after barreloid lesioning (traces in top panel 4). After barreloid lesioning, the gamma-containing spindle and pure gamma bursts were completely blocked, and only pure spindle bursts were observed (lower panel 4). Adapted from Minlebaev et al. (2011) (A–B) and Yang JW et al. (2012) (C) with permission. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

gamma oscillations are generated in the neonatal thalamus remains elusive. In adults, thalamic neurons in both relay and reticular nuclei maintain 40-Hz firing during depolarization beyond −45 mV (Pinault and Deschenes, 1992; Pedroarena and Llinas, 1997). Supported through corticothalamic feedback, these intrinsic oscillations might provide the basis for the gamma range resonant activity in the cortico-thalamo-cortical circuit (Steriade et al., 1996; Llinas et al., 1998; Jones, 2009). Whether such mechanisms support

gamma rhythmogenesis during the neonatal period is unknown. Whole-cell recordings from neonatal mice thalamocortical slices revealed depolarized values (−50/−55 mV) of the resting membrane potential in thalamic VP and reticular neurons (Warren and Jones, 1997). Similar results have also been obtained in ferret dorsal lateral geniculate nucleus (dLGN) during the early postnatal period (Ramoa and McCormick, 1994). Both studies have demonstrated that neonatal thalamic neurons are more sensitive to depolarizing currents, resulting from higher input resistance, and show rhythmic firing with little adaptation. Synapses between the relay and reticular thalamic nuclei and thalamocortical and corticothalamic synapses are already observed in P1 mice, and these synapses show an abrupt developmental change in properties at the end of the first postnatal week (Evrard and Ropert, 2009). Reticular neurons are also interconnected through depolarizing GABAergic synapses and generate network-driven giant depolarizing potentials (Pangratz-Fuehrer et al., 2007). Therefore, during the first postnatal week, the thalamic network possesses key elements that could generate synchronized activities. However, none of the developmental studies *in vitro* have reported spontaneous or lemniscal-evoked forms of thalamic activity organized in gamma oscillations corresponding to the *in vivo* pattern of EGOs. Further studies are required to elucidate the mechanisms of neonatal thalamic gamma rhythmogenesis, which is central to the understanding of the origins of EGOs.

Thalamic gamma oscillations might also rely on feedforward excitation from the periphery. In the adult cat visual system, simultaneous multiple unit recordings showed strong synchronization of oscillatory responses between retina, LGN and cortex, indicating that cortical neurons can be synchronized through oscillatory activity relayed from the retina through the LGN (Castelo-Branco et al., 1998; Neuenschwander et al., 2002). This feedforward synchronization mechanism, operating in the 60 to 120-Hz frequency range, was primarily observed for static stimuli. In contrast, in response to moving stimuli, cortical synchronization occurred independent of oscillatory inputs from the LGN, with oscillation frequency ranging from 30 to 60 Hz. Whether the generation of thalamic EGOs relies on the gamma rhythmic input from the sensory periphery or relay brainstem nuclei remains unknown. However, the single microstimulation of a thalamic barreloid efficiently triggers cortical EGOs (Yang JW et al., 2012), suggesting that gamma patterns derived from the sensory periphery or signals relayed from the brainstem nuclei are not critical for gamma rhythmogenesis in the neonatal VPM thalamus.

Perisomatic inhibition

The synchronization of adult gamma oscillations is primarily based on synchronous inhibition through fast-spiking perisomatic-projecting basket cells (Bartos et al., 2007; Whittington et al., 2011; Buzsaki and Wang, 2012). The limited participation of GABAergic inhibition in the generation of EGOs likely reflects the immaturity of perisomatic inhibition during the first postnatal week. Indeed, basket cells develop fast-spiking phenotypes, incorporate into the network and form electrical and chemical synapses from the end of the first postnatal week, and the development 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; Pangratz-Fuehrer and Hestrin, 2011; Yang JM et al., 2012; Le and Monyer, 2013). In addition to gamma-rhythmogenesis, perisomatic basket cells also play a pivotal role in feedforward inhibition, which sets the time window for the integration of excitatory synaptic inputs (Pouille and Scanziani, 2001; Wehr and Zador, 2003; Gabernet et al., 2005; Higley and Contreras, 2006). However, studies in neonatal mice thalamocortical slices *in vitro* revealed that feedforward inhibition plays no detectable role in regulating L4 circuit function and is not activated through thalamocortical input during the first postnatal week (Daw et al., 2007) (Fig. 4A–D), reflecting a lack of functional connectivity between the interneurons in the circuit, including weak subthreshold thalamic inputs and low fast-spiking interneurons to stellate cell connection probability and strength. At P6–7, there is a coordinated increase in the thalamocortical input to fast-spiking interneurons and in fast-spiking interneurons to stellate cell functional connectivity, associated with a depolarizing-to-hyperpolarizing switch in the polarity of GABAergic responses, leading to the developmental recruitment of feedforward inhibition. The delayed development of the feedforward inhibition has been also confirmed *in vivo* (Minlebaev et al., 2011). Indeed, the onset of sensory-evoked IPSCs was delayed for > 100 ms from the onset of EPSCs at P2 to P3 (Fig. 4F). This temporal integration window rapidly shortened during the first postnatal week (Fig. 4G), indicating the developmental recruitment of feedforward inhibition through the end of the first postnatal week, consistent with the observations obtained *in vitro*. Accordingly, the suppression of units after the first peak in stimulus-triggered unit histogram (dip) was unchanged at P2–4, but was strongly reduced at P6 after the blockade of cortical inhibition (Fig. 4E). Taken together with the delayed recruitment of inhibition to EGOs, these results indicate that the two facets of perisomatic inhibition, i.e., feedforward inhibition and gamma synchronization, show remarkably similar developmental profiles during the first postnatal week.

EGOs, synaptic plasticity and critical period

EGOs are expressed in the barrel cortex during a restricted developmental time window, i.e., the first postnatal week, a critical period for thalamocortical barrel map development and refinement. The formation of this map likely involves two processes: (i) the strengthening and stabilization of synapses, connecting neurons between topographically aligned thalamic barreloids and cortical barrels and (ii) the depression/elimination of the non-topographic synapses connecting neurons in non-corresponding barreloids and barrels.

The development of this map is critically dependent on sensory-driven activity and likely involves N-methyl-D-aspartic acid (NMDA) receptor-dependent long-term potentiation and depression, the two forms of synaptic plasticity regarded as functional precursors of further synapse stabilization and elimination, respectively (Van der Loos and Woolsey, 1973; Fox, 1992, 2002; Feldman et al., 1999; Feldman and Brecht, 2005; Feldman, 2009); see for the most recent update (Erzurumlu and Gaspar, 2012). Consistent with this hypothesis, the levels of NMDA-receptor-dependent forms of synaptic plasticity (both long-term potentiation and depression) are particularly high during the critical period (Feldman et al., 1999).

Although it has not yet been explicitly demonstrated for thalamocortical synapses during the first postnatal week, considerable evidence indicates that developmental synaptic plasticity follows the Hebbian principle, suggesting that "neurons that fire together wire together" (Hebb, 1949), and the rules of spike time-dependent plasticity (STDP) (Debanne et al., 1996; Markram et al., 1997; Song et al., 2000; Dan and Poo, 2006), suggesting that that synaptic connection between two neurons will be potentiated if the presynaptic neuron fires just before the postsynaptic neuron (causal condition) and depressed if the postsynaptic neuron fires ahead of the presynaptic neuron (acausal condition).

From the plasticity standpoint, EGOs provide excellent conditions for potentiation at topographic thalamocortical synapses. First, the lack of the feedforward inhibition creates a wide window for the powerful summation of thalamocortical inputs during EGOs and the depolarization of stellate cells. During each EGO cycle, the firing of postsynaptic stellate cells occurs milliseconds after the firing of presynaptic thalamic neurons, creating conditions for the potentiation of the topographic thalamocortical synapses. The plasticity impact should be multiplied by a replay of this sequential thalamocortical firing for up to eight consecutive EGO cycles, during which the STDP causality rule is preserved, with postsynaptic stellate cell firing, preceded by presynaptic thalamic neuron firing. In addition, the temporal summation of EPSPs during sensory-driven responses results in the depolarization of stellate cells and facilitates the activation of NMDA receptors; precisely, half of the current underlying delta component of sensory response is mediated through NMDA receptors (Minlebaev et al., 2009). Therefore, we suggest that EGOs are instrumental for the long-term potentiation of topographic thalamocortical synapses with further enforcement and stabilization during the critical period. However, the activation of non-topographic synapses, which remain largely subthreshold during sensory responses evoked through neighbor whisker stimulation, creates acausal STDP conditions to support long-term depression with further elimination of these non-topographic synapses. Although this hypothesis requires verification, experiments performed in thalamocortical slices *in vitro* provide support for this idea. Indeed, mimicking EGOs in slices through paired subthreshold gamma-rhythmic thalamic inputs with action potentials in L4 neurons resulted in the long-term potentiation of thalamocortical EPSPs (Minlebaev et al., 2011).

Summary points

1. Gamma oscillations are transiently expressed in L4 of the barrel cortex during the critical period of barrel map formation (first postnatal week). These EGOs are mechanistically different from adult gamma oscillations (Table 1).
2. Feedforward gamma-rhythmic input from thalamus is essential for EGOs.
3. Perisomatic interneurons are recruited to EGOs by the end of the first postnatal week, along with an emergence of the feedforward inhibition. The lack of the feedforward inhibition creates a wide integration window for excitatory inputs during EGOs.
4. EGOs synchronize topographically aligned thalamic and cortical neurons, facilitate the multiple replay of sensory input and create conditions for the STDP at thalamocortical synapses.
5. Inhibition-based "adult" gamma oscillations that enable horizontal synchronization emerge during the second postnatal week as a result of delayed development of perisomatic inhibition and associative layers.

Table 1. Distinct features of the early and adult gamma oscillations

Feature	Early gamma oscillations	Adult gamma oscillations
Age	P 0 (2)–P7	> P13
Lock to stimulus	Stimulus-locked, can be observed in average responses	Non-locked to stimulus (induced), are lost in average responses
Spatial organization	Restricted to a single cortical column	Local and long-range synchronization
CSD profile	Active sinks (maximal in L4)	Multiple active sinks and active sources (maximal in L2/3)
Cellular Correlates	L4 cells fire at high probability (20–50% during each gamma cycle)	Cells fire at < 5% probability during each gamma cycle
Synaptic correlates	Purely Glutamate before P5, glutamate > GABA at P5–7	GABA > glutamate
Synchronization mechanism	< P5: Feedforward (thalamic) gamma-excitation P5–7: Feedforward (thalamic) gamma-excitation + local Inhibition	Synchronization through inhibition
Functions	Synchronization of the topographically aligned thalamic and cortical neurons; multiple replay of sensory input and LTP at thalamocortical synapses	Local and long-range binding of cortical neurons in temporal assemblies, STDP

CONFLICT OF INTEREST STATEMENT

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as potential conflicts of interest.

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REFERENCES

Allene C, Cossart R (2010) Early NMDA receptor-driven waves of activity in the developing neocortex: physiological or pathological network oscillations? J Physiol 588:83–91.

Anderson CM, Torres F, Faoro A (1985) The EEG of the early premature. Electroencephalogr Clin Neurophysiol 60:95–105.

Andre M, Lamblin MD, d'Allest AM, Curzi-Dascalova L, Moussalli-Salefranque F, The SN, Vecchierini-Blineau MF, Wallois F, Walls-Esquivel E, Plouin P (2010) Electroencephalography in premature and full-term infants. Developmental features and glossary. Neurophysiol Clin 40:59–124.

Armstrong-James M (1975) The functional status and columnar organization of single cells responding to cutaneous stimulation in neonatal rat somatosensory cortex S1. J Physiol 246:501–538.

Bartos M, Vida I, Jonas P (2007) Synaptic mechanisms of synchronized gamma oscillations in inhibitory interneuron networks. Nat Rev Neurosci 8:45–56.

Bender KJ, Rangel J, Feldman DE (2003) Development of columnar topography in the excitatory layer 4 to layer 2/3 projection in rat barrel cortex. J Neurosci 23:8759–8770.

Benshalom G, White EL (1986) Quantification of thalamocortical synapses with spiny stellate neurons in layer IV of mouse somatosensory cortex. J Comp Neurol 253:303–314.

Borgdorff AJ, Poulet JF, Petersen CC (2007) Facilitating sensory responses in developing mouse somatosensory barrel cortex. J Neurophysiol 97:2992–3003.

Bureau I, Shepherd GM, Svoboda K (2004) Precise development of functional and anatomical columns in the neocortex. Neuron 42:789–801.

Buzsaki G, Draguhn A (2004) Neuronal oscillations in cortical networks. Science 304:1926–1929.

Buzsaki G, Wang XJ (2012) Mechanisms of gamma oscillations. Annu Rev Neurosci 35:203–225.

Campbell G, Shatz CJ (1992) Synapses formed by identified retinogeniculate axons during the segregation of eye input. J Neurosci 12:1847–1858.

Castelo-Branco M, Neuenschwander S, Singer W (1998) Synchronization of visual responses between the cortex, lateral geniculate nucleus, and retina in the anesthetized cat. J Neurosci 18:6395–6410.

Castro-Alamancos MA (2004) Dynamics of sensory thalamocortical synaptic networks during information processing states. Prog Neurobiol 74:213–247.

Chattopadhyaya B, Di Cristo G, Higashiyama H, Knott GW, Kuhlman SJ, Welker E, Huang ZJ (2004) Experience and activity-dependent maturation of perisomatic GABAergic innervation in primary visual cortex during a postnatal critical period. J Neurosci 24:9598–9611.

Chen C, Regehr WG (2000) Developmental remodeling of the retinogeniculate synapse. Neuron 28:955–966.

Colonnese MT, Kaminska A, Minlebaev M, Milh M, Bloem B, Lescure S, Moriette G, Chiron C, Ben-Ari Y, Khazipov R (2010) A conserved switch in sensory processing prepares developing neocortex for vision. Neuron 67:480–498.

Connors BW, Benardo LS, Prince DA (1983) Coupling between neurons of the developing rat neocortex. J Neurochem 3:773–782.

Crepel F, Mariani J, Delhaye-Bouchaud N (1976) Evidence for a multiple innervation of Purkinje cells by climbing fibres in the immature rat cerebellum. J Neurobiol 7:567–578.

Crepel V, Aronov D, Jorquera I, Represa A, Ben Ari Y, Cossart R (2007) A parturition-associated nonsynaptic coherent activity pattern in the developing hippocampus. Neuron 54:105–120.

Csicsvari J, Jamieson B, Wise KD, Buzsaki G (2003) Mechanisms of gamma oscillations in the hippocampus of the behaving rat. Neuron 37:311–322.

Dan Y, Poo MM (2006) Spike timing-dependent plasticity: from synapse to perception. Physiol Rev 86:1033–1048.

Daw MI, Ashby MC, Isaac JT (2007) Coordinated developmental recruitment of latent fast spiking interneurons in layer IV barrel cortex. Nat Neurosci 10:453–461.

Debanne D, Gähwiler BH, Thompson SM (1996) Cooperative interactions in the induction of long-term potentiation and depression of synaptic excitation between hipppocampal CA3-CA1 cell pairs *in vitro*. Proc Natl Acad Sci U S A 93:11225–11230.

Debski EA, Cline HT (2002) Activity-dependent mapping in the retinotectal projection. Curr Opin Neurobiol 12:93–99.

Doischer D, Hosp JA, Yanagawa Y, Obata K, Jonas P, Vida I, Bartos M (2008) Postnatal differentiation of basket cells from slow to fast signaling devices. J Neurosci 28:12956–12968.

Douglas RJ, Koch C, Mahowald M, Martin KA, Suarez HH (1995) Recurrent excitation in neocortical circuits. Science 269:981–985.

Dreyfus-Brisac C (1962) The electroencephalogram of the premature infant. World Neurol 3:5–15.

Du J, Zhang L, Weiser M, Rudy B, McBain CJ (1996) Developmental expression and functional characterization of the potassium-channel subunit Kv3.1b in parvalbumin-containing interneurons of the rat hippocampus. J Neurosci 16:506–518.

Dupont E, Hanganu IL, Kilb W, Hirsch S, Luhmann HJ (2006) Rapid developmental switch in the mechanisms driving early cortical columnar networks. Nature 439:79–83.

Ellingson RJ (1958) Electroencephalograms of normal, full-term newborns immediately after birth with observations on arousal and visual evoked responses. Electroencephalogr Clin Neurophysiol Suppl 10:31–50.

Engel AK, Konig P, Gray CM, Singer W (1990) Stimulus-dependent neuronal oscillations in cat visual cortex: inter-columnar interaction as determined by cross-correlation analysis. Eur J Neurosci 2:588–606.

Engel R (1975) Abnormal electroencephalograms in the neonatal period. Springfield, Illinois: Charles C. Thomas.

Erzurumlu RS, Gaspar P (2012) Development and critical period plasticity of the barrel cortex. Eur J Neurosci 35:1540–1553.

Evrard A, Ropert N (2009) Early development of the thalamic inhibitory feedback loop in the primary somatosensory system of the newborn mice. J Neurosci 29:9930–9940.

Feldman DE (2009) Synaptic mechanisms for plasticity in neocortex. Annu Rev Neurosci 32:33–55.

Feldman DE, Brecht M (2005) Map plasticity in somatosensory cortex. Science 310:810–815.

Feldman DE, Nicoll RA, Malenka RC (1999) Synaptic plasticity at thalamocortical synapses in developing rat somatosensory cortex: LTP, LTD., and silent synapses. J Neurobiol 41:92–101.

Feldmeyer D (2012) Excitatory neuronal connectivity in the barrel cortex. Front Neuroanat 6:24.

Fox K (1992) A critical period for experience-dependent synaptic plasticity in rat barrel cortex. J Neurosci 12:1826–1838.

Fox K (2002) Anatomical pathways and molecular mechanisms for plasticity in the barrel cortex. Neuroscience 111:799–814.

Fries P (2009) Neuronal gamma-band synchronization as a fundamental process in cortical computation. Annu Rev Neurosci 32:209–224.

Fries P, Neuenschwander S, Engel AK, Goebel R, Singer W (2001) Rapid feature selective neuronal synchronization through correlated latency shifting. Nat Neurosci 4:194–200.

Gabernet L, Jadhav SP, Feldman DE, Carandini M, Scanziani M (2005) Somatosensory integration controlled by dynamic thalamocortical feed-forward inhibition. Neuron 48:315–327.
Gireesh ED, Plenz D (2008) Neuronal avalanches organize as nested theta- and beta/gamma-oscillations during development of cortical layer 2/3. Proc Natl Acad Sci U S A 105:7576–7581.
Goldberg EM, Jeong HY, Kruglikov I, Tremblay R, Lazarenko RM, Rudy B (2011) Rapid developmental maturation of neocortical FS Cell intrinsic excitability. Cereb Cortex 21:666–682.
Goldie L, Svedsen-Rhodes U, Easton J, Roberton NR (1971) The development of innate sleep rhythms in short gestation infants. Dev Med Child Neurol 13:40–50.
Gray CM, Singer W (1989) Stimulus-specific neuronal oscillations in orientation columns of cat visual cortex. Proc Natl Acad Sci U S A 86:1698–1702.
Hasenstaub A, Shu Y, Haider B, Kraushaar U, Duque A, McCormick DA (2005) Inhibitory postsynaptic potentials carry synchronized frequency information in active cortical networks. Neuron 47:423–435.
Hebb DO (1949) The organization of behaviour. New York: John Wiley and Sons.
Higashi S, Molnar Z, Kurotani T, Toyama K (2002) Prenatal development of neural excitation in rat thalamocortical projections studied by optical recording. Neuroscience 115:1231–1246.
Higley MJ, Contreras D (2006) Balanced excitation and inhibition determine spike timing during frequency adaptation. J Neurosci 26:448–457.
Hrbek A, Karlberg P, Olsson T (1973) Development of visual and somatosensory evoked responses in pre-term newborn infants. Electroencephalogr Clin Neurophysiol 34:225–232.
Huang ZJ, Di Cristo G, Ango F (2007) Development of GABA innervation in the cerebral and cerebellar cortices. Nat Rev Neurosci 8:673–686.
Jones EG (2009) Synchrony in the interconnected circuitry of the thalamus and cerebral cortex. Ann N Y Acad Sci 1157:10–23.
Kandler K, Katz LC (1998) Coordination of neuronal activity in developing visual cortex by gap junction-mediated biochemical communication. J Neurosci 18:1419–1427.
Katz LC, Shatz CJ (1996) Synaptic activity and the construction of cortical circuits. Science 274:1133–1138.
Khazipov R, Luhmann HJ (2006) Early patterns of electrical activity in the developing cerebral cortex of humans and rodents. Trends Neurosci 29:414–418.
Khazipov R, Sirota A, Leinekugel X, Holmes GL, Ben Ari Y, Buzsaki G (2004) Early motor activity drives spindle bursts in the developing somatosensory cortex. Nature 432:758–761.
Kostovic I, Judas M (2010) The development of the subplate and thalamocortical connections in the human foetal brain. Acta Paediatr 99:1119–1127.
Lahtinen H, Palva JM, Sumanen S, Voipio J, Kaila K, Taira T (2002) Postnatal development of rat hippocampal gamma rhythm in vivo. J Neurophysiol 88:1469–1474.
Lamblin MD, Andre M, Challamel MJ, Curzi-Dascalova L, d'Allest AM, De Giovanni E, Moussalli-Salefranque F, Navelet Y, Plouin P, Radvanyi-Bouvet MF, Samson-Dollfus D, Vecchierini-Blineau MF (1999) Electroencephalography of the premature and term newborn. Maturational aspects and glossary. Neurophysiol Clin 29:123–219.
Landers M, Philip ZH (2006) Development of rodent whisking: trigeminal input and central pattern generation. Somatosens Mot Res 23:1–10.
Le MC, Monyer H (2013) GABAergic interneurons shape the functional maturation of the cortex. Neuron 77:388–405.
Lefort S, Tomm C, Floyd Sarria JC, Petersen CC (2009) The excitatory neuronal network of the C2 barrel column in mouse primary somatosensory cortex. Neuron 61:301–316.
Leinekugel X, Khazipov R, Cannon R, Hirase H, Ben Ari Y, Buzsaki G (2002) Correlated bursts of activity in the neonatal hippocampus in vivo. Science 296:2049–2052.
Lichtman JW, Colman H (2000) Synapse elimination and indelible memory. Neuron 25:269–278.
Llinas R, Ribary U, Contreras D, Pedroarena C (1998) The neuronal basis for consciousness. Philos Trans R Soc Lond B Biol Sci 353:1841–1849.
Luhmann HJ, Martinez Millan L, Singer W (1986) Development of horizontal intrinsic connections in cat striate cortex. Exp Brain Res 63:443–448.
Mann EO, Suckling JM, Hajos N, Greenfield SA, Paulsen O (2005) Perisomatic feedback inhibition underlies cholinergically induced fast network oscillations in the rat hippocampus in vitro. Neuron 45:105–117.
Maravall M, Stern EA, Svoboda K (2004) Development of intrinsic properties and excitability of layer 2/3 pyramidal neurons during a critical period for sensory maps in rat barrel cortex. J Neurophysiol 92:144–156.
Marcano-Reik AJ, Blumberg MS (2008) The corpus callosum modulates spindle-burst activity within homotopic regions of somatosensory cortex in newborn rats. Eur J Neurosci 28:1457–1466.
Markram H, Lubke J, Frotscher M, Sakmann B (1997) Regulation of synaptic efficacy by coincidence of postsynaptic APs and EPSPs. Science 275:213–215.
Micheva KD, Beaulieu C (1996) Quantitative aspects of synaptogenesis in the rat barrel field cortex with special reference to GABA circuitry. J Comp Neurol 373:340–354.
Milh M, Kaminska A, Huon C, Lapillonne A, Ben Ari Y, Khazipov R (2007) Rapid cortical oscillations and early motor activity in premature human neonate. Cereb Cortex 17:1582–1594.
Minlebaev M, Ben Ari Y, Khazipov R (2009) NMDA receptors pattern early activity in the developing barrel cortex in vivo. Cereb Cortex 19:688–696.
Minlebaev M, Ben-Ari Y, Khazipov R (2007) Network mechanisms of spindle-burst oscillations in the neonatal rat barrel cortex in vivo. J Neurophysiol 97:692–700.
Minlebaev M, Colonnese M, Tsintsadze T, Sirota A, Khazipov R (2011) Early gamma oscillations synchronize developing thalamus and cortex. Science 334:226–229.
Neuenschwander S, Castelo-Branco M, Baron J, Singer W (2002) Feed-forward synchronization: propagation of temporal patterns along the retinothalamocortical pathway. Philos Trans R Soc Lond B Biol Sci 357:1869–1876.
Nolte R, Schulte FJ, Michaelis R, Weisse U, Gruson R (1969) Bioelectric brain maturation in small-for-dates infants. Dev Med Child Neurol 11:83–93.
Okaty BW, Miller MN, Sugino K, Hempel CM, Nelson SB (2009) Transcriptional and electrophysiological maturation of neocortical fast-spiking GABAergic interneurons. J Neurosci 29: 7040–7052.
Oren I, Hajos N, Paulsen O (2010) Identification of the current generator underlying cholinergically induced gamma frequency field potential oscillations in the hippocampal CA3 region. J Physiol 588:785–797.
Pangratz-Fuehrer S, Hestrin S (2011) Synaptogenesis of electrical and GABAergic synapses of fast-spiking inhibitory neurons in the neocortex. J Neurosci 31:10767–10775.
Pangratz-Fuehrer S, Rudolph U, Huguenard JR (2007) Giant spontaneous depolarizing potentials in the developing thalamic reticular nucleus. J Neurophysiol 97:2364–2372.
Parmelee AH, Akiyama Y, Stern E, Harris MA (1969) A periodic cerebral rhythm in newborn infants. Exp Neurol 25:575–584.
Pedroarena C, Llinas R (1997) Dendritic calcium conductances generate high-frequency oscillation in thalamocortical neurons. Proc Natl Acad Sci U S A 94:724–728.
Peinado A (2000) Traveling slow waves of neural activity: a novel form of network activity in developing neocortex. J Neurosci 20:RC54.
Peinado A (2001) Immature neocortical neurons exist as extensive syncitial networks linked by dendrodendritic electrical connections. J Neurophysiol 85:620–629.

Penttonen M, Kamondi A, Acsady L, Buzsaki G (1998) Gamma frequency oscillation in the hippocampus of the rat: intracellular analysis in vivo. Eur J Neurosci 10:718–728.

Pinault D, Deschenes M (1992) Voltage-dependent 40-Hz oscillations in rat reticular thalamic neurons in vivo. Neuroscience 51:245–258.

Pouille F, Scanziani M (2001) Enforcement of temporal fidelity in pyramidal cells by somatic feed-forward inhibition. Science 293:1159–1163.

Rakic P (1977) Prenatal development of the visual system in rhesus monkey. Philos Trans R Soc Lond B Biol Sci 278:245–260.

Ramoa AS, McCormick DA (1994) Developmental changes in electrophysiological properties of LGNd neurons during reorganization of retinogeniculate connections. J Neurosci 14:2089–2097.

Rebsam A, Seif I, Gaspar P (2002) Refinement of thalamocortical arbors and emergence of barrel domains in the primary somatosensory cortex: a study of normal and monoamine oxidase a knock-out mice. J Neurosci 22:8541–8552.

Rhoades RW, Nett-Clarke CA, Chiaia NL, White FA, Macdonald GJ, Haring JH, Jacquin MF (1990) Development and lesion induced reorganization of the cortical representation of the rat's body surface as revealed by immunocytochemistry for serotonin. J Comp Neurol 293:190–207.

Ruthazer ES, Akerman CJ, Cline HT (2003) Control of axon branch dynamics by correlated activity in vivo. Science 301:66–70.

Scher MS (2006) Electroencephalography of the newborn: normal features. In: Holmes GL, Moshe S, Jones RH, editors. Clinical neurophysiology of infancy, childhood and adolescence. USA: Elsevier. p. 46–69.

Schlaggar BL, De Carlos JA, O'Leary DD (1993) Acetylcholinesterase as an early marker of the differentiation of dorsal thalamus in embryonic rats. Brain Res Dev Brain Res 75:19–30.

Simons DJ, Land PW (1987) Early experience of tactile stimulation influences organization of somatic sensory cortex. Nature 326:694–697.

Singer W, Gray CM (1995) Visual feature integration and the temporal correlation hypothesis. Annu Rev Neurosci 18:555–586.

Song S, Miller KD, Abbott LF (2000) Competitive Hebbian learning through spike-timing-dependent synaptic plasticity. Nat Neurosci 3:919–926.

Steriade M, Contreras D, Amzica F, Timofeev I (1996) Synchronization of fast (30–40 Hz) spontaneous oscillations in intrathalamic and thalamocortical networks. J Neurosci 16:2788–2808.

Stern EA, Maravall M, Svoboda K (2001) Rapid development and plasticity of layer 2/3 maps in rat barrel cortex in vivo. Neuron 31:305–315.

Stjerna S, Voipio J, Metsaranta M, Kaila K, Vanhatalo S (2012) Preterm EEG: a multimodal neurophysiological protocol. J Vis Exp 60:3774.

Stockard-Pope JE, Werner SS, Bickford RG (1992) Atlas of neonatal electroencephalography. 2nd ed. Raven Press.

Tallon-Baudry C, Bertrand O (1999) Oscillatory gamma activity in humans and its role in object representation. Trends Cogn Sci 3:151–162.

Uhlhaas PJ, Roux F, Rodriguez E, Rotarska-Jagiela A, Singer W (2010) Neural synchrony and the development of cortical networks. Trends Cogn Sci 14:72–80.

Van der Loos H, Woolsey TA (1973) Somatosensory cortex: structural alterations following early injury to sense organs. Science 179:395–398.

Vanhatalo S, Palva JM, Andersson S, Rivera C, Voipio J, Kaila K (2005) Slow endogenous activity transients and developmental expression of K+–Cl- cotransporter 2 in the immature human cortex. Eur J Neurosci 22:2799–2804.

Vanhatalo S, Tallgren P, Andersson S, Sainio K, Voipio J, Kaila K (2002) DC-EEG discloses prominent, very slow activity patterns during sleep in preterm infants. Clin Neurophysiol 113:1822–1825.

Wang HX, Gao WJ (2010) Development of calcium-permeable AMPA receptors and their correlation with NMDA receptors in fast-spiking interneurons of rat prefrontal cortex. J Physiol 588:2823–2838.

Wang XJ (2010) Neurophysiological and computational principles of cortical rhythms in cognition. Physiol Rev 90:1195–1268.

Warren RA, Jones EG (1997) Maturation of neuronal form and function in a mouse thalamo-cortical circuit. J Neurosci 17:277–295.

Watanabe K, Iwase K (1972) Spindle-like fast rhythms in the EEGs of low-birth weight infants. Dev Med Child Neurol 14:373–381.

Wehr M, Zador AM (2003) Balanced inhibition underlies tuning and sharpens spike timing in auditory cortex. Nature 426:442–446.

Whittington MA, Cunningham MO, LeBeau FE, Racca C, Traub RD (2011) Multiple origins of the cortical gamma rhythm. Dev Neurobiol 71:92–106.

Yang JM, Zhang J, Yu YQ, Duan S, Li XM (2012) Postnatal development of 2 microcircuits involving fast-spiking interneurons in the mouse prefrontal cortex. Cereb Cortex [Epub ahead of print].

Yang JW, An S, Sun JJ, Reyes-Puerta V, Kindler J, Berger T, Kilb W, Luhmann HJ (2012) Thalamic network oscillations synchronize ontogenetic columns in the newborn rat barrel cortex. Cereb Cortex [Epub ahead of print].

Yang JW, Hanganu-Opatz IL, Sun JJ, Luhmann HJ (2009) Three patterns of oscillatory activity differentially synchronize developing neocortical networks in vivo. J Neurosci 29:9011–9025.

Yu YC, He S, Chen S, Fu Y, Brown KN, Yao XH, Ma J, Gao KP, Sosinsky GE, Huang K, Shi SH (2012) Preferential electrical coupling regulates neocortical lineage-dependent microcircuit assembly. Nature 486:113–117.

Yuste R, Nelson DA, Rubin WW, Katz LC (1995) Neuronal domains in developing neocortex: mechanisms of coactivation. Neuron 14:7–17.

Yuste R, Peinado A, Katz LC (1992) Neuronal domains in developing neocortex. Science 257:665–669.

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