УЧЕНЫЕ ЗАПИСКИ КАЗАНСКОГО УНИВЕРСИТЕТА.
СЕРИЯ ЕСТЕСТВЕННЫЕ НАУКИ

2018, Т. 160, кн. 4
С. 677–685

ISSN 2542-064X (Print)
ISSN 2500-218X (Online)

UDC 591.18

EARLY ACTIVITY PATTERNS AND THALAMOCORTICAL SYNAPTIC PLASTICITY DURING THE "BRAIN SPURT" PERIOD

A.R. Nasretdinov [a], *R.N. Khazipov* [a,b]

[a] *Kazan Federal University*, *Kazan*, *420008 Russia*
[b] *INMED*, *Aix-Marseille University*, *INSERM*, *Marseille*, *13273 France*

Abstract

Development of thalamocortical sensory maps during the "brain spurt" period, which spans through the second half of gestation in human and neonatal period in rodents, is characterized by particular immature patterns, which are thought to be implicated in the activity-dependent formation of the topographic thalamocortical circuits. Here, we discuss how these early activity patterns can support plasticity at the developing thalamocortical synapses and contribute to the refinement of the initially crude protomaps through the competition between sensory inputs for the cortical territories.

Keywords: neonate, somatosensory cortex, electroencephalography, neuronal networks, development

Introduction

Just like an inflation phase in the evolution of the Universe, the "brain spurt" period during mammalian brain development is characterized by the explosive rates of neuronal growth/differentiation and the formation of synaptic connections between neurons. The brain growth spurt takes place throughout the second half of gestation in humans and after birth, in the neonatal period, in altricious animals. During this time, the most important connections form within the brain to support basic functions, and this process largely relies on the innate mechanisms involving guidance molecules and trophic factors, as well as the internally generated neuronal network activities. Following the "brain spurt" period, further development of the neuronal networks proceeds more gradually through an increase in the circuit complexity and elimination of some transient circuits to enable acquisition of the fine skills, and this process largely depends on the interactions with the environment. Here, we discuss the activity-dependent mechanisms implicated in the early development of the thalamocortical sensory maps during the "brain spurt" period in the context of the early synchronized neuronal activity patterns expressed in the developing brain. Indeed, while the general connectivity protomaps are to a large extent encoded in our genes to enable certain level of topographic organization, wealth of evidence indicates on the important roles of the activity-dependent mechanisms in the early neuronal wiring in the thalamocortical system to support its refinement through the competition between thalamic inputs

for their cortical targets, which may involve Hebbian mechanisms of synaptic plasticity. Several age-specific neuronal network activity patterns have now been identified in the thalamocortical circuits during the "brain spurt" period, both in the neonatal rodents and preterm human neonates, revealing striking similarities in the early brain function across species. Some of these patterns, such as early gamma oscillations, were shown to enable a high degree of synchronization between the topographic thalamic and cortical neurons and support spike-time dependent plasticity in these connections. Some previous reviews addressed various aspects of the early activities expressed in the thalamocortical networks during the "brain spurt" period: their electrographic features and generative mechanisms, their roles in synaptic plasticity and neuronal network formation, and control of the developmental neuroapoptosis [1–21]. We focus our review on the roles of the early activity patterns in the activity-dependent formation of the topographic thalamocortical maps during the "brain spurt" period.

Sensory Maps

Sensory maps in the primary sensory cortex are organized in cortical columns, each being finely tuned to specific input from the sensory space. For example, in the somatosensory barrel cortex of rodents each cortical barrel column receives sensory information mainly from the corresponding (so-called principal) whisker at the animal snout [22, 23]. Precise topography in this map is ensured by the segregation of the thalamic axon terminals arising from the neurons in the corresponding barreloid in the relay thalamus and compact anatomical organization of the dendrites of thalamorecipient neurons within the cortical barrels. Also, recurrent excitatory connectivity between the cortical barrel neurons, which amplifies the incoming thalamic input, is essentially restricted to one barrel, whereas lateral inhibition efficiently contrasts sensory inputs from different whiskers. A similar topographic organization of the thalamocortical maps is also observed in other regions of the somatosensory cortex, as well as in a form of retinotopic and tonotopic thalamocortical maps in visual and auditory cortices, respectively.

Early Activity Patterns

Topographic thalamocortical maps develop during the "brain spurt" period. In the rat whisker-barrel system, this process starts from the invasion of the growing thalamic afferents into the dense cortical plate at birth, formation and elaboration of synaptic connections, segregation of the receptive fields of the cortical neurons and projection fields of the sensory inputs so that functional topographic maps are formed during the first postnatal week [3]. This period is also known as a critical period for barrel map plasticity, when alterations in sensory input or central activity may dramatically change whisker-barrel map [3, 24–27], and is also characterized by extremely high levels of the activity-dependent plasticity (both potentiation and depression) at thalamocortical synapses [28–31]. During this stage, age-specific patterns of spontaneous and sensory-evoked synchronized neuronal activity are expressed in the barrel cortex. They include delta-waves that are expressed during the two first postnatal days and spindle-bursts (SBs) and early gamma oscillations (EGOs) that are expressed through the postnatal days (P) 2–7 [10, 32–36]. Interestingly, both SBs and

EGOs disappear after P7 coinciding with a closure of the critical period of the barrel map plasticity. These early bursting activity patterns are also expressed in other regions of somatosensory cortex [37], as well as in the visual cortex [38–41] of neonatal rats during comparable phase of the retinotopic thalamocortical maps formation. Together, these findings suggest that the early activity patterns are instrumental for the activity-dependent development of the topographic thalamocortical maps. However, the exact mechanisms underlying the roles of the early activity patterns in developmental plasticity at thalamocortical synapses still remain largely hypothetical.

Thalamocortical Synaptic Plasticity

One hypothesis assumes that the early activity patterns support developmental plasticities through the Hebbian mechanisms of the spike-time dependent plasticity [42, 43]. In this model, the strength of thalamocortical synaptic connections is potentiated if presynaptic thalamic neurons fire shortly before the postsynaptic cortical neurons. In contrast, the synapse will be depressed if the postsynaptic neuron fires before the presynaptic one. From this viewpoint, EGOs enable multiple coherent firing of the topographically linked thalamic and cortical neurons with ~7 msec lag and, therefore, create conditions for the long-term potentiation at the topographic thalamocortical synapses [33, 35]. The latter has also been confirmed in the experiments using thalamocortical slices in vitro, where the pairing of the thalamic input with spikes in the barrel neurons as it occurs during natural EGOs resulted in long-term potentiation at the thalamocortical synapse [35]. Interestingly, mimicking spindle-bursts in the same experimental conditions resulted in slight but significant long-term depression. Because EGOs specifically support synchronization at the topographic synapses, whereas spindle-bursts synchronize larger cortical territories involving several neighbor barrels, it could be suggested that these two patterns support oppositely directed forms of plasticity – potentiation (with future synapse stabilization) in the case of EGOs and depression (with future synapse elimination) in the case of spindle-bursts [32, 44]. There are several important questions that should be addressed to verify this hypothesis, however. Why does coherent activation of thalamocortical synapses and cortical neurons at gamma and spindle-burst frequencies causes opposite forms of plasticity? One mechanism may involve different activation of NMDA receptors, which are involved in the induction of the LTP by oscillations at different frequencies. Evidently, the activity at gamma frequency should enable more efficient activation of NMDA receptors as the duration of each EGO cycle (~20 msec) approximates the peak of NMDA receptor-mediated conductance (~ 10–15 msec) [45, 46]. Therefore, from the spike-time dependent plasticity viewpoint, depolarization and firing of the postsynaptic cell during the forthcoming gamma cycle should efficiently alleviate magnesium block and enable currents through NMDA receptors which were activated during previous gamma cycle. Spindle-burst oscillations occur at lower frequency (from 5 to 25 Hz) and depolarization and firing of the postsynaptic cells during the next cycle should occur at much longer delay (40–200 msec) that is at the decay of NMDA receptor-mediated conductance activated during the previous cycle of the oscillation. On the other hand, NMDA receptors are activated and efficiently summate during the repetitive firing of thalamic affer-

ents during spindle-bursts to generate nearly half of the delta-envelope of the transmembrane currents during spindle-bursts [45]. This is likely due to a large contribution of NMDA receptors to synaptic transmission in the immature neurons, their longer kinetics, high cell membrane resistance, and extremely long delays of the feedforward inhibition during thalamocortical stimulation [35, 47, 48]. This raises the question of how spike timing in thalamocortical transmission impacts the induction of synaptic plasticity during the immature bursting activity [49]? This question remains open for future investigations.

Peculiarity of Thalamocortical Connectivity

One of the difficulties in studying the developmental plasticity at thalamocortical synapses involves high levels of divergence and convergence in the thalamocortical connectivity. Dual recordings from the thalamic relay neurons in VPM barreloids and L4 neurons in cortical barrels revealed a very high connection probability, yet with low strength, at each thalamocortical synapse [50]. Therefore, the developmental plasticity in this system may essentially involve an increase in the number of topographic connections with pruning of the non-topographic ones. This is different from some other systems where the target cell receives only one or few very powerful "bone" inputs, and their development involves initial formation of the overnumerous and relatively weak synapses, competition between these inputs with the activity-dependent selection and strengthening of some that will become the "bone" connections and elimination of others. This paradigm operates, for example, in the development of neuromuscular connections, where each muscle fiber is initially connected by multiple motor neurons and only one or few of them are selected and strengthened through the competition during development [51, 52]. Similarly, thalamic relay neurons in visual and somatosensory thalamus are initially innervated by tens of weak bottom-up connections, and only one or two are selected through the competition and pruning [53, 54]. While the developmental wiring in the thalamocortical system may use some elements of this paradigm, there are some critical issues that should be verified. Firstly, is there a phenomenon of the initial synapse overproduction and consequent pruning in the thalamocortical connectivity? A recent study using thalamocortical slices of neonatal mice revealed that L4 neurons are initially connected by only a few thalamic neurons and that developmental increase in thalamocortical connectivity during the critical period occurs through a progressive experience-dependent increase in the number of L4 cells connected by single thalamic axon without a change in the efficacy at the individual synapses [55]. While these findings go against the "overproduction–pruning" paradigm, they do not rule out that some of the early established synapses are non-topographic (i.e., erroneously connecting thalamic neuron with L4 cells in a wrong barrel) and undergo pruning during the critical period. Another related issue is how equal are in strength thalamocortical synapses made by the highly converging thalamic neurons on L4 cells? What if among these mainly weak connections there are one or few of the "bone" megasynapses that are overseen during random sampling? Such rare megasynapses with an order higher amplitude and low variability are actually detected during large scale sampling of the intracortical connections [56] and if also present in the thalamocortical system, they could well be a substrate for the developmental selection through competition.

Acknowledgments. This work was supported by INSERM (LIA), the subsidy allocated to Kazan Federal University for the state assignment no. 6.2313.2017/4.6 in the sphere of scientific activities, and performed in the framework of the Program of Competitive Growth of Kazan Federal University.

References

1. Katz L.C., Shatz C.J. Synaptic activity and the construction of cortical circuits. *Science*, 1996, vol. 274, no. 5290, pp. 1133–1138. doi: 10.1126/science.274.5290.1133.
2. Vitali I., Jabaudon D. Synaptic biology of barrel cortex circuit assembly. *Semin. Cell Dev. Biol.*, 2014, vol. 35, pp. 156–164. doi: 10.1016/j.semcdb.2014.07.009.
3. Erzurumlu R.S., Gaspar P. Development and critical period plasticity of the barrel cortex. *Eur. J. Neurosci.*, 2012, vol. 35, no. 10, pp. 1540–1553. doi: 10.1111/j.1460-9568.2012.08075.x.
4. Leighton A.H., Lohmann C. The wiring of developing sensory circuits – from patterned spontaneous activity to synaptic plasticity mechanisms. *Front. Neural Circuits*, 2016, vol. 10, art. 71, pp. 1–13. doi: 10.3389/fncir.2016.00071.
5. Tiriac A., Blumberg M.S. The case of the disappearing spindle burst. *Neural Plast.*, 2016, vol. 2016, art. 8037321, pp. 1–7. doi: 10.1155/2016/8037321.
6. Luhmann H.J., Sinning A., Yang J.W., Reyes-Puerta V., Stuttgen M.C., Kirischuk S., Kilb W. Spontaneous neuronal activity in developing neocortical networks: From single cells to large-scale interactions. *Front. Neural Circuits*, 2016, vol. 10, art. 40, pp. 1–14. doi: 10.3389/fncir.2016.00040.
7. Lindemann C., Ahlbeck J., Bitzenhofer S.H., Hanganu-Opatz I.L. Spindle activity orchestrates plasticity during development and sleep. *Neural Plast.*, 2016, vol. 2016, pp. 5787423.
8. Yang J.W., Reyes-Puerta V., Kilb W., Luhmann H.J. Spindle bursts in neonatal rat cerebral cortex. *Neural Plast.*, 2016, vol. 2016, art. 3467832, pp. 1–11. doi: 10.1155/2016/3467832.
9. Egorov A.V., Draguhn A. Development of coherent neuronal activity patterns in mammalian cortical networks: Common principles and local hetereogeneity. *Mech. Dev.*, 2013, vol. 130, nos. 6–8, pp. 412–423. doi: 10.1016/j.mod.2012.09.006.
10. Minlebaev M., Ben-Ari Y., Khazipov R. Network mechanisms of spindle-burst oscillations in the neonatal rat barrel cortex in vivo. *J. Neurophysiol.*, 2007, vol. 97, no. 1, pp. 692–700. doi: 10.1152/jn.00759.2006.
11. Colonnese M., Khazipov R. Spontaneous activity in developing sensory circuits: Implications for resting state fMRI. *Neuroimage*, 2012, vol. 62, no. 4, pp. 2212–2221. doi: 10.1016/j.neuroimage.2012.02.046.
12. Khazipov R., Minlebaev M., Valeeva G. Early gamma oscillations. *Neuroscience*, 2013, vol. 250, pp. 240–252. doi: 10.1016/j.neuroscience.2013.07.019.
13. Marshall P.J., Meltzoff A.N. Body maps in the infant brain. *Trends Cognit. Sci.*, 2015, vol. 19, no. 9, pp. 499–505. doi: 10.1016/j.tics.2015.06.012.
14. Cirelli C., Tononi G. Cortical development, electroencephalogram rhythms, and the sleep/wake cycle. *Biol. Psychiatry*, 2015, vol. 77, no. 12, pp. 1071–1078. doi: 10.1016/j.biopsych.2014.12.017.
15. Khazipov R., Luhmann H.J. Early patterns of electrical activity in the developing cerebral cortex of humans and rodents. *Trends Neurosci.*, 2006, vol. 29, no. 7, pp. 414–418. doi: 10.1016/j.tins.2006.05.007.

16. Luhmann H.J., Khazipov R. Neuronal activity patterns in the developing barrel cortex. *Neuroscience*, 2018, vol. 368, pp. 256–267. doi: 10.1016/j.neuroscience.2017.05.025.
17. Blumberg M.S., Sokoloff G., Tiriac A., Del Rio-Bermudez C. A valuable and promising method for recording brain activity in behaving newborn rodents. *Dev. Psychobiol.*, 2015, vol. 57, no. 4, pp. 506–517. doi: 10.1002/dev.21305.
18. Tiriac A., Sokoloff G., Blumberg M.S. Myoclonic twitching and sleep-dependent plasticity in the developing sensorimotor system. *Curr. Sleep Med. Rep.*, 2015, vol. 1, no. 1, pp. 74–79. doi: 10.1007/s40675-015-0009-9.
19. Luhmann H.J. Review of imaging network activities in developing rodent cerebral cortex *in vivo*. *Neurophotonics*, 2017, vol. 4, no. 3, art. 031202, pp. 1–8. doi: 10.1117/1.NPh.4.3.031202.
20. Luhmann H.J., Khazipov R. Neuronal activity patterns in the developing barrel cortex. *Neuroscience*, 2018, vol. 368, pp. 256–267. doi: 10.1016/j.neuroscience.2017.05.025.
21. Lotfullina N., Khazipov R. Ethanol and the developing brain: Inhibition of neuronal activity and neuroapoptosis. *Neuroscientist*, 2018, vol. 24, no. 2, pp. 130–141. doi: 10.1177/1073858417712667.
22. Petersen C.C.H. The functional organization of the barrel cortex. *Neuron*, 2007, vol. 56, no. 2, pp. 339–355. doi: 10.1016/j.neuron.2007.09.017.
23. Feldmeyer D., Brecht M., Helmchen F., Petersen C.C.H., Poulet J.F.A., Staiger J.F., Luhmann H.J., Schwarz C. Barrel cortex function. *Prog. Neurobiol.*, 2013, vol. 103, pp. 3–27. doi: 10.1016/j.pneurobio.2012.11.002.
24. O'Leary D.D.M., Ruff N.L., Dyck R.H. Development, critical period plasticity, and adult reorganizations of mammalian somatosensory systems. *Curr. Opin. Neurobiol.*, 1994, vol. 4, no. 4, pp. 535–544. doi: 10.1016/0959-4388(94)90054-X.
25. Feldman D.E., Brecht M. Map plasticity in somatosensory cortex. *Science*, 2005, vol. 310, no. 5749, pp. 810–815. doi: 10.1126/science.1115807.
26. Feldman D.E. Synaptic mechanisms for plasticity in neocortex. *Annu. Rev. Neurosci.*, 2009, vol. 32, pp. 33–55. doi: 10.1146/annurev.neuro.051508.135516.
27. López-Bendito G., Molnár Z. Thalamocortical development: How are we going to get there? *Nat. Rev. Neurosci.*, 2003, vol. 4, no. 4, pp. 276–289. doi: 10.1038/nrn1075.
28. Feldman D.E., Knudsen E.I. Experience-dependent plasticity and the maturation of glutamatergic synapses. *Neuron*, 1998, vol. 20, no. 6, pp. 1067–1071. doi: 10.1016/s0896-6273(00)80488-2.
29. Isaac J.T.R., Crair M.C., Nicoll R.A., Malenka R.C. Silent synapses during development of thalamocortical inputs. *Neuron*, 1997, vol. 18, no. 2, pp. 269–280. doi: 10.1016/s0896-6273(00)80267-6.
30. Crair M.C., Malenka R.C. A critical period for long-term potentiation at thalamocortical synapses. *Nature*, 1995, vol. 375, no. 6529, pp. 325–328. doi: 10.1038/375325a0.
31. An S., Yang J.W., Sun H., Kilb W., Luhmann H.J. Long-term potentiation in the neonatal rat barrel cortex *in vivo*. *J. Neurosci.*, 2012, vol. 32, no. 28, pp. 9511–9516. doi: 10.1523/JNEUROSCI.1212-12.2012.
32. Yang J.W., Hanganu-Opatz I.L., Sun J.J., Luhmann H.J. Three patterns of oscillatory activity differentially synchronize developing neocortical networks *in vivo*. *J. Neurosci.*, 2009, vol. 29, no. 28, pp. 9011–9025. doi: 10.1523/JNEUROSCI.5646-08.2009.
33. Yang J.W., An S., Sun J.J., Reyes-Puerta V., Kindler J., Berger T., Kilb W., Luhmann H.J. Thalamic network oscillations synchronize ontogenetic columns in the newborn rat barrel cortex. *Cereb. Cortex*, 2013, vol. 23, no. 6, pp. 1299–1316. doi: 10.1093/cercor/bhs103.

34. Mitrukhina O., Suchkov D., Khazipov R., Minlebaev M. Imprecise whisker map in the neonatal rat barrel cortex. *Cereb. Cortex*, 2015, vol. 25, no. 10, pp. 3458–3467. doi: 10.1093/cercor/bhu169.

35. Minlebaev M., Colonnese M., Tsintsadze T., Sirota A., Khazipov R. Early gamma oscillations synchronize developing thalamus and cortex. *Science*, 2011, vol. 334, no. 6053, pp. 226–229. doi: 10.1126/science.1210574.

36. Akhmetshina D., Nasretdinov A., Zakharov A., Valeeva G., Khazipov R. The nature of the sensory input to the neonatal rat barrel cortex. *J. Neurosci.*, 2016, vol. 36, no. 38, pp. 9922–9932. doi: 10.1523/JNEUROSCI.1781-16.2016.

37. Khazipov R., Sirota A., Leinekugel X., Holmes G.L., Ben Ari Y., Buzsaki G. Early motor activity drives spindle bursts in the developing somatosensory cortex. *Nature*, 2004, vol. 432, no. 7018, pp. 758–761. doi: 10.1038/nature03132.

38. Hanganu I.L., Ben Ari Y., Khazipov R. Retinal waves trigger spindle bursts in the neonatal rat visual cortex. *J. Neurosci.*, 2006, vol. 26, no. 25, pp. 6728–6736. doi: 10.1523/JNEUROSCI.0752-06.2006.

39. Hanganu I.L., Staiger J.F., Ben-Ari Y., Khazipov R. Cholinergic modulation of spindle bursts in the neonatal rat visual cortex *in vivo*. *J. Neurosci.*, 2007, vol. 27, no. 21, pp. 5694–5705. doi: 10.1523/JNEUROSCI.5233-06.2007.

40. Colonnese M.T., Khazipov R. "Slow activity transients" in infant rat visual cortex: A spreading synchronous oscillation patterned by retinal waves. *J. Neurosci.*, 2010, vol. 30, no. 12, pp. 4325–4337. doi: 10.1523/JNEUROSCI.4995-09.2010.

41. Colonnese M.T., Kaminska A., Minlebaev M., Milh M., Bloem B., Lescure S., Moriette G., Chiron C., Ben-Ari Y., Khazipov R. A conserved switch in sensory processing prepares developing neocortex for vision. *Neuron*, 2010, vol. 67, no. 3, pp. 480–498. doi: 10.1016/j.neuron.2010.07.015.

42. Hebb D.O. *The Organization of Behaviour*. New York, John Wiley and Sons, 1949. 335 p.

43. Debanne D., Gahwiler B.H., Thompson S.M. Long-term synaptic plasticity between pairs of individual CA3 pyramidal cells in rat hippocampal slice cultures. *J. Physiol.*, 1998, vol. 507, no. 1, pp. 237–247. doi: 10.1111/j.1469-7793.1998.237bu.x.

44. Suchkov D., Sharipzyanova L., Minlebaev M. Horizontal synchronization of neuronal activity in the barrel cortex of the neonatal rat by spindle-burst oscillations. *Front. Cell. Neurosci.*, 2018, vol. 12, art. 5, pp. 1–12. doi: 10.3389/fncel.2018.00005.

45. Minlebaev M., Ben Ari Y., Khazipov R. NMDA Receptors pattern early activity in the developing barrel cortex in vivo. *Cereb. Cortex*, 2009, vol. 19, no. 3, pp. 688–696. doi: 10.1093/cercor/bhn115.

46. Khazipov R., Ragozzino D., Bregestovski P. Kinetics and Mg^{2+} block of *N*-methyl-D-aspartate receptor channels during postnatal development of hippocampal CA3 pyramidal neurons. *Neuroscience*, 1995, vol. 69, no. 4, pp. 1057–1065. doi: 10.1016/0306-4522(95)00337-I.

47. Daw M.I., Ashby M.C., Isaac J.T. Coordinated developmental recruitment of latent fast spiking interneurons in layer IV barrel cortex. *Nat. Neurosci.*, 2007, vol. 10, no. 4, pp. 453–461. doi: 10.1038/nn1866.

48. Valiullina F., Akhmetshina D., Nasretdinov A., Mukhtarov M., Valeeva G., Khazipov R., Rozov A. Developmental changes in electrophysiological properties and a transition from electrical to chemical coupling between excitatory layer 4 neurons in the rat barrel cortex. *Front. Neural Circuits*, 2016, vol. 10, art. 1, pp. 1–13. doi: 10.3389/fncir.2016.00001.

49. Butts D.A., Kanold P.O. The applicability of spike time dependent plasticity to development. *Front. Synaptic Neurosci.*, 2010, vol. 2, art. 30, pp. 1–9. doi: 10.3389/fnsyn.2010.00030.

50. Bruno R.M., Sakmann B. Cortex is driven by weak but synchronously active thalamocortical synapses. *Science*, 2006, vol. 312, no. 5780, pp. 1622–1627. doi: 10.1126/science.1124593.

51. Sanes J.R., Lichtman J.W. Development of the vertebrate neuromuscular junction. *Annu. Rev. Neurosci.*, 1999, vol. 22, pp. 389–442. doi: 10.1146/annurev.neuro.22.1.389.

52. Lichtman J.W., Colman H. Synapse elimination and indelible memory. *Neuron*, 2000, vol. 25, no. 2, pp. 269–278. doi: 10.1016/S0896-6273(00)80893-4.

53. Chen C., Regehr W.G. Developmental remodeling of the retinogeniculate synapse. *Neuron*, 2000, vol. 28, no. 3, pp. 955–966. doi: 10.1016/s0896-6273(00)00166-5.

54. Arsenault D., Zhang Z.W. Developmental remodelling of the lemniscal synapse in the ventral basal thalamus of the mouse. *J. Physiol.*, 2006, vol. 573, pt. 1, pp. 121–132. doi: 10.1113/jphysiol.2006.106542.

55. Crocker-Buque A., Brown S.M., Kind P.C., Isaac J.T., Daw M.I. Experience-dependent, layer-specific development of divergent thalamocortical connectivity. *Cereb. Cortex*, 2015, vol. 25, no. 8, pp. 2255–2266. doi: 10.1093/cercor/bhu031.

56. Lefort S., Tomm C., Floyd Sarria J.C., Petersen C.C. The excitatory neuronal network of the C2 barrel column in mouse primary somatosensory cortex. *Neuron*, 2009, vol. 61, no. 2, pp. 301–316. doi: 10.1016/j.neuron.2008.12.020.

Recieved
June 25, 2018

Nasretdinov Azat Rafailovich, Junior Research Fellow

Kazan Federal University
ul. Kremlevskaya, 18, Kazan, 420008 Russia
E-mail: *AzRNasretdinov@kpfu.ru*

Khazipov Rustem Narimanovich, Doctor of Medical Sciences, Leading Research Fellow

Kazan Federal University
ul. Kremlevskaya, 18, Kazan, 420008 Russia

INMED, Aix-Marseille University, INSERM
163 Avenue de Luminy BP13, Marseille, 13273, France
E-mail: *roustem.khazipov@inserm.fr*

УДК 591.18

Паттерны ранней активности и синаптическая пластичность соматосенсорных таламокортикальных карт во время критического периода развития

А.Р. Насретдинов[1], *Р.Н. Хазипов*[1,2]

[1]*Казанский (Приволжский) федеральный университет, г. Казань, 420008, Россия*
[2] *Университет Экс-Марсель, г. Марсель, 13273, Франция*

Аннотация

В статье представлен обзор актуальных исследований, посвященных развитию таламокортикальных сетей во время критического периода. Описаны характерные ранние паттерны нейрональной активности в соматосенсорной коре и высказано предположение, что они являются инструментом для развития топографичных таламокортикальных карт. Отметим, что на данный момент нет полноценного понимания точных механизмов, формирующих влияние ранних паттернов активности на пластичность таламокортикальных синапсов. В поддержку гипотезы, рассматриваемой

в статье, проанализированы модель пластичности на основе правила Хэбба и результаты экспериментов на таламокортикальных срезах, где наблюдались разные эффекты пластичности в зависимости от частотных характеристик ранних паттернов активности. Сделан вывод, что такое различие связано с большим вкладом NMDA-рецепторов в синаптическую передачу в незрелых нейронах. Кроме того, в статье особое внимание уделено особенностям таламокортикальных связей и подчеркнуто, что пластичность в этой системе, вероятнее всего, определяется увеличением количества топографичных и удалением нетопографичных связей.

Ключевые слова: новорожденные, соматосенсорная кора, электроэнцефалография, нейронные сети, развитие

Поступила в редакцию
25.06.18

Насретдинов Азат Рафаилович, младший научный сотрудник

Казанский (Приволжский) федеральный университет
ул. Кремлевская, д. 18, г. Казань, 420008, Россия
E-mail: *AzRNasretdinov@kpfu.ru*

Хазипов Рустем Наримановичч, доктор медицинских наук, ведущий научный сотрудник

Казанский (Приволжский) федеральный университет
ул. Кремлевская, д. 18, г. Казань, 420008, Россия

Средиземноморский институт нейробиологии (INMED), Университет Экс-Марсель, Национальный институт здравоохранения и медицинских исследований Франции (INSERM)
163 Авеню де Люмини BP13, г. Марсель, 13273, Франция
E-mail: *roustem.khazipov@inserm.fr*

***For citation*:** Nasretdinov A.R., Khazipov R.N. Early activity patterns and thalamocortical synaptic plasticity during the “brain spurt” period. *Uchenye Zapiski Kazanskogo Universiteta. Seriya Estestvennye Nauki*, 2018, vol. 160, no. 4, pp. 677–685.

***Для цитирования*:** *Nasretdinov A.R., Khazipov R.N.* Early activity patterns and thalamocortical synaptic plasticity during the “brain spurt” period // Учен. зап. Казан. ун-та. Сер. Естеств. науки. – 2018. – Т. 160, кн. 4. – С. 677–685.