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P1462 / #3365

Topic: AS09 Motor and Sensory Systems

SENSORIMOTOR RESTRICTION DURING DEVELOPMENT INDUCES MITOCHONDRIAL ALTERATION WITHIN MUSCLE AND BRAIN STRUCTURES

Mélanie Van Gaever¹, Frédéric Daussin¹,
Orlane Dupuis¹, Olivier Dupuy²,
Jacques-Olivier Coq^{3,4}, Erwan Dupont¹,
Marie-Hélène Canu¹

¹ Univ. Lille, Univ. Artois, Univ. Littoral Côte d'Opale,
Ulr 7369 - Urepss - Unité De Recherche

Pluridisciplinaire Sport Santé Société, Loos, France

² Faculty of Sport Sciences (STAPS), University of
Poitiers, Laboratory Move (ur 20296), Poitiers, France

³ Centre National de la Recherche Scientifique (CNRS),
UMR7287 Institut des Sciences du Mouvement (ISM),
Bouches-du-rhône, Marseille, France

⁴ Centre National de la Recherche Scientifique (CNRS),
Umr7287 Institut Des Sciences Du Mouvement (ism),
Marseille, France

Physical inactivity and sedentary lifestyle during childhood can lead to musculoskeletal and motor deficits but also cognitive and behavioral disorders. Most children with developmental coordination disorders or autism spectrum disorders show sensorimotor impairments, reduced physical activity, altered interactions with their environment and atypical motor development. Since ATP produced by mitochondria is essential for muscle contraction, neurotransmission, and brain plasticity processes, emerging hypotheses suggest that functional alterations could be related to energy metabolism. Understanding the mechanisms behind these neurodevelopmental deficits is a prerequisite for developing remediation strategies, and the later are crucial to promote self-reliance and independence, to improve future quality of life and to decrease the risk of neurodegenerative diseases during ageing. In order to better understand the emergence of neuromuscular or cognitive disorders during childhood, a model of early sensorimotor restriction was developed in rats. It consists in casting pups' hindlimbs from birth to postnatal day 28 (P28). These animals display a prominent motor phenotype that includes muscle weakness, locomotor disturbances and altered cognitive and executive functions. Our aim was to determine whether early sensorimotor restriction alters mitochondrial metabolism in rat muscles and brain structures. Enzyme activities of citrate synthase and respiratory chain complexes I, II, and IV were measured using a spectrophotometric technique in two hind limb muscles (soleus and extensor digitorum longus) and several brain structures (hippocampus, prefrontal cortex, striatum, cerebellum and sensorimotor cortex) in control rats and rats experiencing early sensorimotor restriction at P15 and P28. Our results show a decrease in mitochondrial enzyme activities in the soleus, in the extensor digitorum longus, and in the hippocampus, while no effect was observed in other brain structures. To sum up, the effect of early sensorimotor restriction on mitochondrial metabolism is structure-dependent, structures involved in cognitive functions being more affected than those involved in motor processes.

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THALAMIC NEURAL ACTIVITY IS ESSENTIAL FOR TRIGGERING PATTERNS OF SPONTANEOUS ACTIVITY IN THE DEVELOPING SENSORY CORTICES

Dorien Vandael¹, Teresa Guillamón Vivancos²,
Daniel Torres Romero¹, Mar Anibal¹,
Lorenzo Puche-Aroca¹, Miguel Valdeolmillos¹,
Francisco Martini¹, Guillermina Lopez-Bendito¹

¹ Instituto de neurociencias CSIC-UMH, Developmental
Neurobiology, Alicante, Spain

² Instituto de neurociencias CSIC-UMH, Developmental
Neurobiology, alicante, Spain

During postnatal development, the mammalian cerebral cortex exhibits patterns of synchronized neuronal activity that are known to play a key role in the refinement of cortical circuits before sensory perception. However, the origin and function of these spontaneous activity patterns in the early establishment of cortical sensory areas and circuits remain largely unknown. Recent work from our laboratory has demonstrated that prenatal spontaneous activity in the thalamus is crucial for the development of sensory cortical maps (Anton-Bolaños et al., 2019). This work suggests that patterns of spontaneous activity observed in the cortex may also contain crucial information about the construction of emerging circuits, but their origin and contribution to the specification of sensory cortical territories remain elusive. By performing *in vivo* mesoscale calcium imaging, we reveal specific patterns of spontaneous activity as cortical sensory territories emerge. Interestingly, altering the patterned spontaneous activity in the embryonic thalamus directly disrupt these specific sensory cortical activity patterns. Furthermore, as already known for somatosensory circuits (Anton-Bolaños et al 2019), we found that hampering thalamic activity also caused changes in the retinotopic map as evidenced by the disorganisation of the thalamocortical visual projections. Thus, our results show that thalamic spontaneous activity is crucial for the proper emergence and refinement of both somatosensory and visual sensory circuits. Finally, to determine in a cell-type specific manner the cortical genes and gene networks that are regulated by the patterned thalamic activity, we performed single-nuclei RNA-seq experiments in cortical sensory areas during development in mice with no cortical activity in sensory areas. Our data will increase our understanding on how thalamic activity influences sensory cortical cell type composition, activity patterns and gene expression. Altogether, this project will shed light on the contribution and origin of early spontaneous activity patterns in the early establishment of sensory cortical territories and circuits.

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