ISDN2014_0113

Stromal vascular fraction in peripheral nerve regeneration

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Peripherial nerve injuries are very common (3–10%) in orthopaedic practice. Also treatment of them is continuous (Sunderland reports that satisfactory reinnervation of human muscle can occur after denervation of 12 months, sensory recovery occurs earlier), mostly patients are young, able-bodied. So the problem is actual for nowdays.

There are three suturing techniques: neurorrhaphy, interfascicular nerve grafting (ING) and nerve grafting with tubes.

We proposed a hypothesis that using ING with autological cells of stromal vascular fraction (SVF) can improve the results of treatment. In experiment on group of 22 Wistar rats (ING in sciatic nerve with gap 1 cm with transplantation SVF and group without it) we showed difference in appearing of sensitivity, electromyographic, histological, sciatic function index, laser Doppler investigations.

We invented method of substituting peripheral nerve defect using cells of SVF on clinical trial. Method: abdominal liposuction, enzyme treatment of adipose tissue (which contains mesenchymal stem cells, endothelial precusors, M2 polarizing macrophages and T-lymphocytes), received cells of the SVF transplantated into peripheral, central nerve segments and in autonervegraft between them, the end of nerve paste covered with fibrin glue.

Clinical example: Patient X. 25 years with diagnosis: «the consequences of the ulnar nerve damage in the upper third of the left shoulder». Defect ulnar nerve amounted to 5 cm. 25.08.11 operation: liposuction, receiving SVF. ING of ulnar nerve with the use of a calf nerve. Transplantation of SVF into peripheral, central nerve segments and in autonervegraft, covered with fibrin glue. 6 weeks after surgery the patient revealed painful sensibility in the autonomous area of innervation of the ulnar nerve, while the standard plastic appearance of pain sensitivity is celebrated on the timing is not less than 8–10 months after the operation.

For this moment operated 10 patients, whom now examining on different terms.

http://dx.doi.org/10.1016/j.ijdevneu.2015.04.092

ISDN2014_0114

Decreased mTOR signaling via p70S6K/eIF4B is associated with loss of the excitatory postsynaptic marker PSD-95 in autism

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Introduction: Autism, a common neurodevelopmental disorder, is believed to arise from defects in the establishment and maintenance of functional neuronal networks. However, the molecular mechanisms underlying the abnormal cortical circuitry seen in autistic brains remain to be elucidated. We previously found imbalances in BDNF and TrkB isoforms and decreased upstream components of the mTOR signaling pathway in postmortem brains of autism versus control subjects. Two signaling pathways downstream of mTOR, p70S6K/eIF4B and 4E-BP1/eIF4E, regulate protein synthesis at dendritic spines, which form excitatory postsynapses. In this study we examined whether these mTOR-mediated signaling pathways are disrupted in autism and whether their disruption is associated with changes in PSD-95, a marker of excitatory synapses.

Methods: Phospho-mTOR, mTOR, p70S6K, eIF4B, 4E-BP1, eIF4E and PSD-95 were measured by Western blotting in postmortem fusiform gyrus of 11 autism and 13 control subjects.

Results: Significantly decreased phospho-mTOR, mTOR, p70S6K, eIF4B and PSD-95 protein levels were observed in autism versus control fusiform gyrus. In contrast, no significant differences in 4E-BP1 and eIF4E protein expression were found.

Discussion: Our findings show decreased mTOR expression and activation and down-regulation of mTOR downstream pathway p70S6K/eIF4B in autism. This pathway is responsible for expression of RNAs encoding components of the translational machinery, and its down-regulation likely results in reduced protein translation at spines. The 4E-BP1/eIF4E pathway, which is responsible for translation of capped mRNAs, is unaffected in our autism cohort, supporting the specificity of the p70S6K/eIF4B pathway deficit in idiopathic autism. Finally, spine protein translation deficits are likely to adversely affect spine density, as suggested by our finding of decreased PSD-95 in autistic fusiform gyrus. Decreased spine density might perturb cortical circuitry and thus contribute to autism's cognitive and behavioural deficits.

http://dx.doi.org/10.1016/j.ijdevneu.2015.04.093

ISDN2014_0115

Born too soon? Cognitive and electrophysiological evaluation of atypical language processing in the prematurely born baby

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The last months of pregnancy are particularly important for the development of the central nervous system and many intrauterine events occurring between 24 and 40 weeks of gestation are crucial for fetal development. Thus, many children born prematurely will show language and/or cognitive disorders at school age. This study aims to establish a protocol for screening for these disorders in premature and full term born infants using the Mismatch Negativity (MMN) and auditory evoked potentials (AEPs), associated with auditory preattentional processing. Using an oddball paradigm during EEG recording, 40 healthy participants (3–32 years of age) were first evaluated to document the normal maturational changes of the MMN in the verbal and non-verbal modalities. Eighty-two children (3, 12 and 36 months of age), born at term or prematurely, were subsequently evaluated using EEG and a neurodevelopmental assessment (Bayley-III) to identify electrophysiological predictors of cognitive and language impairments. Based on MMN and AEPs characteristics, results revealed a distinct development of verbal and nonverbal preattentional processes in healthy children. Age-related differences for non-speech discrimination occurred