Role and functions of neuro development in spinal cord injury.

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Abstract

The spinal cord plays a role in the transmission pathway information transmission between the brain and the peripheral nervous system. It is the complex arrangement of patterns and connections makes visualization difficult. From its small cross-sectional area and location body. However, significant advances in imaging technology. It has arisen over the past decade and has improved resolution, allowing better assessment of this more detailed anatomy.

Keywords: Cellular transplantation, Electrophysiology.

Introduction

Precise control of neural progenitor cell proliferation and differentiation is critical for central nervous system development. Fusion in Sarcoma (FS) is an RNA-binding protein pathogenically associated with Amyotrophic Lateral Sclerosis (ALS) and Frontal Temporal Oar Degeneration (FTOD) but the role of FUS in neurodevelopment is yet to be defined. Here we report a pivotal role for FUS in regulating human cortical brain and spinal cord development via human derived organics. We found that CRISPR/CAS9-mediated FUS depletion leads to enhanced neuronal proliferation and differentiation in cortical brain organics [1].

Interestingly however these phenotypic impairments in spinal cord organics. In addition FUS binds to [Trk] Tyrosine Kinase receptor mRNA for neurotropic (Ntrk3) and regulates the expression of different is forms of Ntrk3 in a tissue-specific manner. Finally RNA-mediated reduction of NTRK3 levels rescued the effects of her FUS on the development of brain and spinal cord organics suggesting that Ntrk3 is involved in developmental changes in her FUS-regulated organic. Our results revealed a role in neurodevelopment of the human central nervous system. Spinal Cord Injury (SCI) is a debilitating injury of the central nervous system with complex pathological mechanisms leading to sensory and motor dysfunction. Current treatments for spinal cord injury are aimed at symptomatic relief rather than pathological causes. Several studies have reported that signalling pathways play an important role in the pathological process of his SCI and neuronal recovery mechanisms. The PI3K/Akt signalling pathway is a key pathway closely related to the pathological process of SCI, and activation of this pathway slows the inflammatory response, prevents glial scar formation, and promotes recovery of neuronal function [2].

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According to the latest World Health Organization (WHO) report approximately million people worldwide suffer from SCI, and the global incidence of SCI is estimated at per year Due to the reduced ability of autonomic nerve repair after spinal cord injury and the rapid onset of various pathological processes, the injured spinal cord cannot easily undergo tissue repair and reconstitution of function. Therefore, neurological recovery after SCI is a difficult issue in the medical community. Although many studies have been conducted on the molecular and cellular mechanisms of SCI the exact pathophysiological mechanisms of SCI are still unknown due to the lack of a comprehensive understanding of the pathological processes and mechanisms associated with SCI. is However, current

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SCI treatment focuses primarily on symptom palliation rather than pathophysiological interventions.Therefore, improving our understanding of its pathogenesis and identifying key molecular therapeutic targets are essential for effective treatment of SCI [4,5].

Conclusion

In the acute phase spinal cord ischemia, edema, inflammation, and free radical-mediated peroxidation mainly occur In the sub-acute phase, vascular system destruction and neuronal apoptosis mainly occur The main characteristics of chronic SCI are axon tip blight cystic cavity, and glial scarring, which inhibit axonal growth and regeneration. The pathophysiology of SCI involves a series of interrelated events, such as vascular ischemia, hypovolemic, excitotoxicity, cytotoxicity, cacogenic edema, ion homeostasis imbalance, and mitochondrial dysfunction.

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