SYNAPTIC PRUNING DEFICITS AS A NEUROLOGICAL BASIS FOR SENSORY OVERLOAD IN YOUNG CHILDREN WITH AUTISM

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Annotation. *This study examines the neurological mechanisms underlying sensory overload in young children with Autism Spectrum Disorder (ASD), focusing specifically on deficits in synaptic pruning. Synaptic pruning is a critical neurodevelopmental process in which redundant or weak synaptic connections are eliminated to optimize neural network efficiency. In children with autism, insufficient synaptic pruning leads to an excess of neural connections, causing hyperresponsiveness to sensory stimuli such as sound, light, and touch.*

The paper discusses the molecular, genetic, and neurophysiological aspects of synaptic pruning deficits, their impact on sensory processing, and the clinical implications for diagnosis, intervention, and therapeutic strategies. Understanding these mechanisms is essential for developing personalized medical approaches to improve quality of life and functional outcomes in children with ASD.

Keywords: *Autism Spectrum Disorder, Synaptic Pruning, Sensory Overload, Neurodevelopment, Pediatric Neurology.*

Introduction

In young children with Autism Spectrum Disorder (ASD), the sensory system is often hyperresponsive, which significantly affects their daily functioning and may lead to emotional and behavioral difficulties.

Research indicates that one of the primary neurological mechanisms underlying this phenomenon is a deficit in synaptic pruning. Synaptic pruning is a critical neurodevelopmental process during childhood, in which redundant or weak synaptic connections are eliminated to optimize neural network efficiency and enhance information processing speed.

In children with autism, this process is frequently insufficient, resulting in an excess of synaptic connections. Consequently, sensory signals from the environment, including auditory, visual, and tactile inputs, are excessively amplified, leading to sensory overload. This paper aims to analyze the neurological basis of sensory overload in young children with autism from a medical perspective, focusing specifically on synaptic pruning deficits and their clinical implications.

Main part

Synaptic pruning is one of the fundamental neurodevelopmental processes, occurring from early childhood through adolescence, in which unnecessary or underused synaptic connections are selectively eliminated. Synapses serve as communication points between neurons, transmitting electrical and chemical signals that underpin all brain functions.

During normal neurodevelopment, neuronal activity is assessed, and weak or unused synapses are removed, whereas active connections are reinforced. This process ensures efficient neural communication, rapid information transmission, and proper cognitive development. A deficit in synaptic pruning leads to excessive synaptic connections, resulting in disorganized neural networks and abnormal signal processing. Clinically, such deficits are associated with autism, hyperactivity, attention difficulties, and sensory processing disorders.

Molecular studies indicate that synaptic pruning involves key neurotransmitters, including glutamate and gamma-aminobutyric acid (GABA), as well as the activity of microglial cells, which are the brain's immune cells. Genetic investigations have further demonstrated that mutations or dysregulation in genes controlling synaptic pruning are prevalent in individuals with ASD. Therefore, synaptic pruning is essential for normal brain development, and its impairment constitutes a neurological basis for sensory hyperreactivity in children with autism.

Children with ASD frequently exhibit symptoms of sensory overload, characterized by heightened responsiveness to environmental stimuli, including sounds, light, colors, tactile sensations, and movement. Even ordinary environmental inputs, such as background noise or light changes, can trigger stress or behavioral dysregulation.

From a medical standpoint, these symptoms are linked to deficits in synaptic pruning: excessive synapses prevent the brain from properly filtering and modulating sensory inputs, leading to hyperstimulation of the central nervous system. Neurophysiological studies indicate that children with autism often display increased connectivity between the cerebral cortex and the thalamus, intensifying sensory signal processing. Furthermore, electroencephalography (EEG) studies reveal heightened high-frequency activity in these children, which corresponds with overactive sensory processing. Consequently, sensory overload is a major clinical feature of ASD, affecting daily functioning, learning, and social adaptation, and its understanding is essential for effective therapeutic interventions.

Recent studies on synaptic pruning deficits and sensory overload in children with autism have provided significant insights into the neurobiological mechanisms of ASD. Molecular and genetic research has identified disrupted microglial activity and mutations in key genes responsible for synaptic pruning as contributing factors. Clinical studies demonstrate that sensory overload not only causes emotional distress but also contributes to sleep disturbances, attention deficits, and social adaptation challenges in children with ASD.

From a medical perspective, various intervention strategies have been developed, including sensory integration therapy, environmental modifications, pharmacological approaches, and family support programs. Understanding the synaptic pruning process is also critical for future development of molecular or gene-targeted therapies. Pediatricians and neurologists can monitor and optimize environmental stimuli to support adaptive neural development, improving both functional outcomes and quality of life for children with ASD.

Conclusion

In conclusion, the primary neurological basis of sensory overload in young children with autism is the deficit in synaptic pruning. Excessive synaptic connections result in hyperstimulation of the central nervous system, amplifying sensory inputs and complicating daily functioning.

From a medical perspective, understanding the mechanisms of synaptic pruning deficits is essential for designing targeted therapies, creating optimal environments, and developing potential genetic or molecular interventions to improve outcomes in children with ASD.

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