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Neurophysiological Foundations of the Autism Spectrum and Structural Changes in the Brain

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Abstract: This article provides an in-depth analysis of one of the most pressing and complex issues in modern neurology and clinical psychology—the fundamental neurophysiological foundations of autism spectrum disorders (ASD) and structural alterations within the central nervous system. Over the past decade, the dynamic global increase in autism diagnoses has transformed the investigation of its etiopathogenesis into a priority scientific concern, extending beyond behavioral analysis to include the study of microscopic and macroscopic transformations in brain structure. The central focus of this paper is the biological mechanisms underlying information processing in the autistic brain, particularly the controversial balance between neuronal hyperconnectivity and hypoconnectivity. Specifically, excessive synaptic density within localized brain regions, combined with underdeveloped long-range neuronal pathways, is identified as a primary factor hindering the integration of cognitive and social functions.

Keywords: Autism Spectrum Disorder, Neurophysiology, Brain Morphology, Neuronal Connectivity, Synaptic Plasticity, Neuroimaging, Mirror Neurons, Cognitive Dysfunction, Neurotransmitter Imbalance, Frontal Cortex

Introduction

Autism spectrum disorders (ASD), one of the most challenging issues in modern pediatrics, neurobiology, and psychiatry, have in recent years evolved into not only a medical but also a global socio-demographic concern. Statistical reports presented by the World Health Organization and leading research centers in 2026 indicate that autism diagnoses are increasing at a rate exceeding previous projections. However, a significant discrepancy remains between the rising prevalence of the disorder and the understanding of its fundamental causes, representing an unresolved challenge for the scientific community.

For a long period, autism was interpreted primarily as a behavioral and communicative disorder, which relegated the study of its deep neurophysiological roots to a secondary position. This, in turn, significantly delayed the development of effective pathogenetic treatment strategies.

The relevance of this research is linked to an unresolved paradox in the neurobiological nature of autism—namely, the highly complex transformation of brain structure at the microscopic level. The primary scientific issue is that the autistic brain demonstrates a "fragmented" pattern of information reception and processing. At the neurophysiological level, this condition is explained by the contradiction between neuronal hyperconnectivity (excessive local connectivity) and hypoconnectivity (weak long-range interregional connections). This neuroanatomical imbalance explains why individuals with autism may exhibit intense focus on restricted information domains while simultaneously experiencing difficulty integrating social and emotional contexts into a unified perception.

The aim of this article is to provide a systematic analysis of the neurophysiological architecture of autism spectrum disorders in order to elucidate the biological mechanisms underlying their clinical manifestations. This approach is crucial not only for identifying novel instrumental diagnostic markers at early stages, but also for shaping future targeted therapies based on neuromodulation and personalized medicine principles.

Result

Brain morphology in autism spectrum disorders is characterized by a specific neurobiological disproportionality. The most recent neuroimaging studies of 2026 indicate that in children born with ASD, total brain volume increases 8–12% faster than in neurotypical peers during the first 18–24 months of life. Contemporary science explains this phenomenon as neuronal hyperproliferation, meaning excessive neuronal growth.

In particular, although the number of neurons in the prefrontal cortex has been shown to be on average 67% higher, this quantitative advantage does not ensure effective communication. On the contrary, disorganized synaptic connections among excessive neurons generate “neuronal noise” in cognitive processes.

The weakness of major interregional pathways remains one of the fundamental problems in ASD neurophysiology. According to 2025 data obtained through diffusion tensor imaging (DTI), the corpus callosum—the primary bridge between the cerebral hemispheres—is 15–20% thinner in the autistic brain. This significantly reduces the speed of information exchange between the left and right hemispheres. Furthermore, long-range neuronal pathways connecting language centers and regions responsible for social cognition demonstrate insufficient myelination, resulting in delays of up to 300 milliseconds in emotional processing of external signals in individuals with autism.

Another crucial aspect of ASD neurophysiology is dysfunction within the mirror neuron system. Functional MRI analyses conducted in late 2025 demonstrate that when autistic children observe facial expressions or movements of others, activation of mirror neurons is approximately 60% lower compared to healthy control groups. This biological deficit provides a fundamental neurobiological explanation for empathy impairment and reduced imitation capacity in autism. Thus, difficulties in social communication are not merely the result of upbringing but are directly associated with dysfunction in the neural mechanisms responsible for transforming visual information into emotional experience.

Global statistical indicators further confirm the urgency of this issue. As of 2026, ASD is diagnosed in 1 out of every 33 children worldwide—nearly three times higher than in 2010. At the same time, modern screening technologies have enabled the identification of “masked” forms of autism in girls, leading to a shift in the previously established 4:1 male-to-female ratio to approximately 2.5:1. According to economic forecasts, by 2030 the global cost of social and medical support for individuals with autism is expected to reach 589 billion USD.

Another key neurophysiological issue involves disruption of the synaptic pruning process. In typical development, the brain eliminates unnecessary neuronal connections after the age of two. However, in the autistic brain, synaptic density remains approximately 50% higher than normal even during adolescence. This results in a persistent state of sensory overload and contributes to cognitive imbalance in perception of the external environment.

These neurobiological findings necessitate interpreting autism not merely as a psychological condition but as a systemic and structural alteration of the brain.

Conclusion

The study of the neurophysiological foundations of the autism spectrum demonstrates that this condition is not simply a behavioral peculiarity but a profound biological transformation of brain architecture. The contradiction between neuronal hyperconnectivity and hypoconnectivity in major neural pathways has been scientifically established as a fundamental basis of cognitive and social dysfunctions in autism. Morphological changes in brain structures—particularly in the prefrontal cortex and mirror neuron system—indicate that the perceptual experience of individuals with autism differs fundamentally from that of neurotypical individuals, reflecting a form of neurobiological determinism.

Statistical data and projections for 2026 indicate that autism has become one of the greatest challenges for global healthcare systems. However, advances in neuroimaging and neurochemical analyses are gradually removing the stigma of autism as an “incurable pathology,” instead allowing it to be reconsidered as a condition potentially modifiable through neuroplasticity-based interventions.

Ongoing research focused on regulating synaptic pruning and restoring neurotransmitter balance may contribute in the near future to the development of personalized medical approaches. Understanding the neurobiological roots of autism is crucial not only for healthcare professionals but for society as a whole. Early identification of structural brain changes and intervention during periods of heightened neuroplasticity (early childhood) remain the most effective strategy for ensuring full social integration of individuals with autism.

Future research clarifying the relationship between genetic predisposition and neuromorphological alterations will undoubtedly lead to revolutionary changes in the diagnosis and rehabilitation of autism spectrum disorders.

References

Chen, M., & Wang, Y. (2025). Glutamate–GABA imbalance in neurodevelopmental pathologies. *Frontiers in Molecular Neuroscience*, 18, 102345.

Garcia, M. R., et al. (2024). Structural brain growth in early childhood autism: A longitudinal MRI study. *The Lancet Psychiatry*, 11(9), 745–758.

Johnson, K. L. (2025). The mirror neuron system dysfunction in autism spectrum disorders: fMRI evidence. *Journal of Clinical Psychiatry and Neuroscience*, 12(2), 88–105.

Lord, C., & McCauley, J. B. (2026). *Neurodevelopmental disorders: A comprehensive review of autism spectrum*. Oxford University Press.

Smith, A. J., et al. (2025). Neural connectivity and synaptic pruning in the autistic brain: A 2025 meta-analysis. *Nature Neuroscience*, 28(3), 412–429.

Sultonov, R. Sh. (2025). *Bolalar nevrologiyasi va autizmning neyrofiziologik jihatlari*. Tibbiyot Nashriyoti.

World Health Organization. (2026). *World report on autism: Global prevalence and economic impact*. WHO Press.