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Neurobiological Mechanisms Underlying the Therapeutic Effects of Physical Exercise in Autism Spectrum Disorder: A Narrative Review

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Abstract

Background. Autism Spectrum Disorder (ASD) is a heterogeneous neurodevelopmental condition characterized by impairments in social communication and the presence of restricted and repetitive behaviors. Increasing evidence suggests that structured physical exercise may serve as a supportive therapeutic approach. However, although exercise has been shown to improve both core symptoms and associated difficulties in ASD, the underlying biological mechanisms remain poorly understood.

Aim. This review aims to summarize current evidence on the neurobiological mechanisms that may underlie the beneficial effects of physical exercise in individuals with Autism Spectrum Disorder.

Material and methods. A narrative review of the literature was performed, focusing on studies exploring the interaction between physical exercise and neurobiological processes relevant to ASD. Studies were identified through databases such as PubMed and Scopus. Both clinical and preclinical studies were included to capture mechanistic insights related to brain function and structure.

Results. Physical exercise modulates ASD related mechanisms involving immune regulation, gut-brain communication, synaptic plasticity, and hormonal responses. Current evidence points to changes in microglial activation, cytokine profiles, and gut microbiota composition, although mechanistic data remain largely preclinical.

Conclusions. Structured physical exercise represents a promising complementary strategy in ASD, with potential benefits mediated through multiple neurobiological pathways. A better understanding of these mechanisms may support the development of targeted and individualized exercise based interventions tailored to the diverse needs of individuals with ASD.

Key words:
Autism Spectrum Disorder,
Exercise,
Physical Activity,
Neuroinflammation,
Microbiota,
Gut-brain Axis

1. Introduction

Autism Spectrum Disorder (ASD) is a heterogeneous neurodevelopmental condition affecting approximately 1% of the global population (Zeidan et al., 2022). It typically becomes apparent in early childhood and persists throughout life, although its presentation may change over time. According to the ICD-11, ASD is characterized by persistent difficulties in social communication and interaction, along with restricted, repetitive, and inflexible patterns of behavior, interests, or activities (Kamp-Becker, 2024). Cognitive abilities in individuals with ASD are highly variable, ranging from intellectual disability to above average intelligence. ASD is also often accompanied by co-occurring conditions, such as attention-deficit/hyperactivity disorder or anxiety disorders. Motor impairments are frequently observed and may include difficulties with coordination, balance, and fine motor skills (Castaño et al., 2024; Kamp-Becker, 2024).

The etiology of Autism Spectrum Disorder is complex and multifactorial, involving an interplay of genetic susceptibility and environmental influences. Identified risk factors include advanced parental age, prenatal exposure to infections or toxins, perinatal complications, and genetic variants that may disrupt early brain development and neural connectivity (Marco et al., 2011). The pathophysiology of Autism Spectrum Disorder is complex and involves alterations at both network and molecular levels. Neuroimaging and electrophysiological studies consistently demonstrate atypical patterns of brain connectivity. Prefrontal cortex, amygdala, and temporal regions show functional and structural differences that are thought to underlie social and communicative impairments (Vilela et al., 2024).

ASD is also associated with abnormalities at the microstructural and molecular levels. For example, dysregulated serotonergic and glutamatergic signaling has been reported, which may disrupt neurogenesis, synaptic development, and the balance between excitatory and inhibitory activity, ultimately contributing to altered neural connectivity and function (Wang et al., 2025). In parallel, changes in brain derived

neurotrophic factor (BDNF), which regulates neuronal survival and synaptic plasticity, may contribute to impaired neurodevelopment and synaptic maturation. Additionally, growing evidence indicates a role for neuroinflammation and immune dysregulation, with elevated pro inflammatory cytokines and activation of microglia and astrocytes (Pretorius et al., 2025). In addition, alterations in gut microbiota composition have been increasingly observed in Autism Spectrum Disorder, often characterized by reduced microbial diversity and an imbalance between beneficial and potentially harmful bacterial species. These changes may influence brain function through the gut brain axis by modulating immune responses, producing neuroactive metabolites, and affecting neurotransmitter systems (Wang et al., 2025).

Given the complex nature of ASD, treatment strategies are typically multimodal and individualized. Management relies mainly on behavioral and educational interventions, supported when needed by pharmacological approaches that target associated symptoms and co-morbidities such as anxiety or attention deficit hyperactivity disorder (Wang et al., 2025). Physical activity is increasingly recognized as a valuable supportive intervention in ASD (Castaño et al., 2024). Regular exercise programs have been associated with improvements in motor skills, coordination, balance, and physical fitness, as well as benefits in attention, executive functioning, and social behavior. In addition, physical activity may help reduce stereotypical and aggressive behaviors, contributing to overall physical, cognitive, and psychosocial well being (Castaño et al., 2024; Foxall et al., 2025). This review aims to identify and summarize the neurobiological mechanisms underlying the beneficial effects of physical activity in Autism Spectrum Disorder, as understanding these processes may support the development of more targeted and effective exercise based interventions, optimize therapeutic outcomes, and contribute to personalized treatment strategies.

2. Materials and methods

This narrative review was conducted to synthesize current evidence on the neurobiological mechanisms underlying the effects of physical activity in Autism Spectrum Disorder. The analysis focused on neuroinflammation, neurotrophic factors such as brain derived neurotrophic factor, gut microbiota, hormonal responses including oxytocin and cortisol, and synaptogenesis. Literature search was performed using electronic databases, primarily PubMed and Scopus, to identify relevant peer reviewed studies. The search strategy combined terms related to autism, physical activity, and neurobiological mechanisms, including neuroinflammation, gut microbiota, and synaptogenesis. Studies were selected based on their relevance to the scope of the review, methodological rigor, and contribution to understanding exercise related neurobiological changes. Non English publications, conference abstracts without full text access, and studies not addressing biological mechanisms were excluded. Due to the narrative nature of the review, findings were analyzed qualitatively, with emphasis on identifying converging evidence across different biological pathways. Variability in study design, population characteristics, and types of exercise interventions was taken into account when interpreting the results.

3. Results

3.1. Exercise Mediated Modulation of Neuroinflammation

Neuroinflammation is increasingly recognized as an important component of Autism Spectrum Disorder pathophysiology. Evidence from clinical and postmortem studies indicates persistent inflammatory activity in multiple brain regions, reflected by increased levels of pro inflammatory cytokines in cerebrospinal fluid and brain tissue, as well as the presence of brain specific autoantibodies (Usui et al., 2023). Increased levels of inflammatory mediators, including interleukin 1 beta, interleukin 6, tumor necrosis factor alpha, and monocyte chemoattractant protein 1, have been reported in peripheral blood, cerebrospinal fluid, and brain tissue of individuals with ASD (Alzghoul et al., 2019; Shen et al., 2023). Altered proportions and functions of immune cells involved in innate and adaptive responses further indicate disrupted immune regulation, with possible autoimmune involvement (Hughes et al., 2023). In addition to classical pro-inflammatory cytokines, changes in factors such as transforming growth factor beta 1, hepatocyte growth factor, epidermal growth factor, and platelet derived growth factor have also been described (Usui et al., 2023). Notably, elevated neonatal levels of selected cytokines, including interleukin 1 beta and interleukin 4, have been associated with greater symptom severity in ASD (Alzghoul et al., 2019).

Increased immune activity in Autism Spectrum Disorder has also been associated with astrogliosis and microglial activation, suggesting that glial cells may participate in the altered neuroimmune environment observed in this condition (Liao et al., 2020). Microglia are essential for synaptic pruning, cytokine signaling, and the maturation of neuronal circuits. Therefore,

persistent microglial activation may disturb synaptic refinement and contribute to atypical patterns of brain connectivity. Astrocytes, in turn, regulate extracellular glutamate levels, support blood brain barrier function, and maintain synaptic homeostasis. Thus, astrocytic dysfunction or reactive astrogliosis may further affect neuronal excitability and plasticity. Together, these findings indicate that alterations in microglial and astrocytic function may represent one of the mechanisms linking immune dysregulation with abnormal neurodevelopment in Autism Spectrum Disorder (Liao et al., 2020).

Physical activity may reduce neuroinflammatory activity through several complementary mechanisms. Regular exercise has been shown to lower the production of pro inflammatory cytokines and regulate immune responses (Chen et al., 2025). In a chronic stress mouse model, four weeks of running exercise reduced depressive like behaviors, restored hippocampal microglial M1/M2 balance, and regulated inflammatory cytokine production, possibly through adiponectin receptor 1 and adenosine monophosphate activated protein kinase, nuclear factor kappa B, and signal transducer and activator of transcription 3 signaling (Liu et al., 2024). Similarly, in another experimental model of neurodegeneration, exercise reduced interleukin 1 beta, interleukin 6, and tumor necrosis factor alpha levels, while supporting neuronal survival and regeneration through hippocampal insulin like growth factor 1 receptor related mechanisms (Chen et al., 2025). More directly in the context of Autism Spectrum Disorder, a study using an ASD rat model showed that six weeks of voluntary running wheel exercise improved social interaction, social novelty preference, exploratory behavior, spontaneous activity, learning, and memory. These behavioral effects were accompanied by reduced ionized calcium binding adaptor molecule 1 positive microglial activation in the hippocampal CA1 and CA3 regions. Exercise also altered the hippocampal cytokine profile, decreasing interleukin 2 and interleukin 4 levels while increasing interleukin 7 and interleukin 10, suggesting a shift toward reduced neuroinflammatory activity (Lan et al., 2026). Together, these findings indicate that exercise may attenuate neuroinflammation in Autism Spectrum Disorder by modulating hippocampal microglial activation and cytokine signaling.

3.2. Impact of Physical Exercise on Gut Microbiota and Gut Brain Axis

In Autism Spectrum Disorder, the gut brain axis has gained increasing attention as a potential link between gastrointestinal disturbances, immune dysregulation, and altered neurodevelopment (Fattorusso et al., 2019). This axis refers to bidirectional communication between the central nervous system, the enteric nervous system, and the gut microbiota, allowing intestinal and neural signals to influence each other. Brain derived signals can affect gut motility, intestinal barrier function, and microbial composition, whereas gut microbiota may modulate brain function through immune activation, vagal pathways, microbial metabolites, peptides, and neurotransmitter related signaling (Xue et al., 2022).

Both clinical studies and animal models have shown changes in gut microbiota composition in ASD, including differences in microbial diversity, bacterial abundance, and microbial metabolites (Xue et al., 2022). Several studies have reported reduced microbial diversity, together with changes in major bacterial groups, such as lower abundance of Firmicutes and relatively higher abundance of Bacteroidetes. Increased levels of specific bacteria, including Sutterella, have also been found in duodenal biopsies and stool samples from individuals with ASD and gastrointestinal symptoms, suggesting altered mucosal microbial balance (Fattorusso et al., 2019; Xue et al., 2022). Experimental studies further support a possible functional role of the gut microbiota, as transplantation of microbiota from individuals with ASD into germ free mice induced autism like behaviors (Sharon et al., 2019). In contrast, supplementation with selected beneficial bacteria, such as Lactobacillus reuteri, improved social deficits in some mouse models (Sgritta et al., 2019). However, evidence supporting more invasive microbiota targeted interventions, such as fecal microbiota transplantation or antibiotic therapy, remains limited and requires further controlled clinical studies to confirm their safety, efficacy, and long term effects in ASD.

In this context, physical activity may represent a safer and more accessible strategy, as regular exercise has been shown to influence gut microbiota composition, microbial diversity, and gut brain axis signaling (Xue et al., 2022). Regular physical activity has been associated with shifts in Firmicutes and Bacteroidetes, as well as enrichment of beneficial bacteria such as Bifidobacterium, Lactobacillus, Akkermansia muciniphila. These changes may increase the production of short chain fatty acids, including butyrate, acetate, and propionate, which support intestinal barrier integrity, mucus production, immune regulation, and anti inflammatory signaling. Through these effects, exercise may improve gut homeostasis and influence gut brain axis communication (Min et al., 2024; Wegierska et al., 2022). Overall, gut microbiota

alterations may contribute to ASD through effects on intestinal inflammation, microbial metabolites, immune regulation, and gut brain axis signaling. Physical activity may represent a safe and accessible strategy to modulate microbiota composition, support gut barrier function, and indirectly influence neuroimmune and behavioral outcomes in ASD (Xue et al., 2022).

3.3. Exercise as a Modulator of Synaptic Plasticity

Neuroplasticity represents another important biological process through which physical activity may influence autism related symptoms. Proper brain development depends on the continuous remodeling of synapses, including the strengthening and weakening of synaptic connections through long term potentiation (LTP) and long term depression (LTD) (Cassilhas et al., 2016). These mechanisms are essential for learning, memory, sensory adaptation, and social behavior. In ASD, synaptic plasticity appears to be dysregulated, with reported alterations in excitatory and inhibitory signaling, glutamatergic transmission, dendritic spine maturation, and synaptic pruning (Bozdagi et al., 2010; Li et al., 2025; Tang et al., 2014).

Exercise is a well recognized modulator of neuroplasticity. Regular physical activity can increase brain derived neurotrophic factor, support neurogenesis, promote synaptic remodeling, and improve activity dependent communication between neuronal circuits (Cassilhas et al., 2016). In experimental models of ASD, exercise has been shown to improve behavioral outcomes and influence synaptic structure and function in regions such as the hippocampus and medial prefrontal cortex. For example, exercise training in valproic acid induced ASD models has been associated with improved long term potentiation, changes in synaptic density and morphology, and modulation of synapse related molecular pathways (King et al., 2024; Mohammadkhani et al., 2024). In another study, exercise training in valproic acid induced ASD rats partially reversed abnormal synaptic organization in the medial prefrontal cortex and normalized several synapse related phosphoproteins (Tu et al., 2023). Together, these animal studies suggest that exercise may influence neuroplasticity related pathways in ASD models by modulating neurotrophic signaling, synaptic remodeling, and activity dependent plasticity. However, these findings should be interpreted cautiously, as evidence from human studies remains limited and it is not yet clear whether similar mechanisms occur in individuals with ASD.

3.4. Hormonal Responses to Physical Exercise in Autism Spectrum Disorder

Hormonal regulation may represent an additional pathway through which physical activity could influence functioning in ASD. Oxytocin is of particular interest because of its role in social cognition and socioemotional behavior. A recent cross sectional study found no overall difference in hypothalamic gray matter volume or peripheral oxytocin levels between autistic and non autistic adults, but reported a positive association between peripheral oxytocin levels and hypothalamic gray matter volume within the ASD group, suggesting that individual differences in the oxytocin system may be relevant to ASD neurobiology (Haaf et al., 2024). In a related study assessing hormonal responses to rapid cycling exercise, basal oxytocin and cortisol concentrations did not differ between adults with ASD and controls, and post exercise levels were also not significantly different between groups. However, plasma oxytocin increased after exercise in autistic adults with a low cortisol response, indicating that physical activity may engage oxytocin related pathways in a subgroup specific manner (Albantakis et al., 2021). Overall, these findings suggest a potential interaction between exercise, stress regulation, and oxytocin signaling in ASD, although the evidence remains preliminary and requires confirmation in larger studies.

4. Discussion

The evidence summarized in this review suggests that physical activity may influence ASD through several interacting biological pathways rather than through a single isolated mechanism. This is particularly relevant given the clinical and biological heterogeneity of ASD, in which different individuals may present with distinct patterns of immune dysregulation, altered gut brain axis signaling, impaired synaptic plasticity, and stress related hormonal responses. From this perspective, exercise should not be viewed solely as a behavioral or motor intervention, but also as a potential modulator of neurobiological processes involved in neurodevelopment and adaptive functioning.

Among the mechanisms discussed, the strongest preclinical evidence concerns neuroinflammation and synaptic plasticity. Animal studies indicate that exercise may reduce microglial activation, regulate cytokine production, and improve activity dependent synaptic plasticity in brain regions involved in cognition, memory, and social behavior. However, this evidence should be interpreted with caution, as most mechanistic studies have been conducted in

experimental models, particularly valproic acid induced rodents, which only partially reproduce the complexity and heterogeneity of ASD in humans.

The gut brain axis and hormonal regulation represent additional promising but less established mechanisms. Exercise induced changes in gut microbiota may affect intestinal barrier integrity, microbial metabolite production, immune signaling, and communication between the gut and the central nervous system. Nevertheless, direct evidence linking exercise driven microbiota changes with behavioral improvement in ASD remains limited. Similarly, available human studies suggest that oxytocin and cortisol responses to physical exercise may vary between individuals, indicating that neuroendocrine effects of exercise may depend on baseline biological characteristics and stress reactivity.

Overall, current findings support the potential value of structured physical activity as a complementary strategy in ASD. Its accessibility, safety, and adaptability make it particularly attractive for individualized intervention programs. However, future studies should include larger clinical cohorts, standardized exercise protocols, longitudinal follow up, and integrated biomarker assessment. Such approaches may help determine which forms of exercise are most effective, which biological pathways are most responsive, and which subgroups of individuals with ASD are most likely to benefit.

5. Conclusions

Structured physical exercise represents a promising complementary approach in Autism Spectrum Disorder. Its potential benefits may be mediated through several biological mechanisms, including attenuation of neuroinflammation, modulation of gut microbiota and gut brain axis signaling, enhancement of neuroplasticity, and regulation of hormonal responses related to stress and social behavior. Despite these promising findings, the mechanistic evidence remains incomplete, particularly in human studies. Further well designed clinical research is needed to establish optimal exercise type, intensity, frequency, and duration, as well as to identify biological markers that may predict response. Given the heterogeneity of ASD, future interventions should be individualized according to motor abilities, behavioral profile, and co occurring conditions. Physical activity may therefore serve as a safe and accessible supportive strategy within broader multidisciplinary care for individuals with ASD.

Declarations

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