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Neurohemodynamic Modulation of Executive Control: Acute Aerobic Exercise Alterations in Frontoparietal and Default Mode Network Connectivity in Pediatric and Preterm Populations

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Abstract

Background: Executive function (EF) deficits are hallmark characteristics of attention deficit hyperactivity disorder (ADHD) and neurodevelopmental sequelae associated with very preterm birth. While acute aerobic exercise is known to transiently enhance cognitive performance, the neural mechanisms driving these behavioral improvements—specifically regarding functional connectivity (FC) within the Frontoparietal Network (FPN) and the Default Mode Network (DMN)—remain insufficiently characterized.

Methods: This study utilized a within-subjects counterbalanced design involving three cohorts: children with ADHD, adolescents born very preterm with diffuse white matter abnormalities, and age-matched healthy controls. Participants underwent resting-state functional Magnetic Resonance Imaging (rs-fMRI) following two distinct conditions: a 20-minute bout of moderate-intensity aerobic exercise and a sedentary control session. Cognitive performance was assessed using inhibitory control tasks (Flanker/Stroop) immediately post-intervention.

Results: Behavioral analysis revealed significant reductions in reaction time and error rates across all groups following the exercise condition compared to rest. Neuroimaging data indicated a significant increase in functional connectivity within the FPN and anticorrelation between the FPN and DMN post-exercise. Notably, the preterm group exhibited distinct connectivity alterations suggestive of compensatory neural recruitment.

Conclusion: Acute aerobic exercise serves as a potent modulator of large-scale brain network dynamics, enhancing the segregation and integration of networks critical for executive control. These findings support the prescription of targeted physical activity as

a complementary therapeutic strategy for pediatric populations with attentional and cognitive deficits.

Keywords: Functional Connectivity, Executive Function, Aerobic Exercise, ADHD, Preterm Birth, fMRI, Frontoparietal Network.

Introduction

The development of executive functions (EF)—a constellation of higher-order cognitive processes including inhibitory control, working memory, and cognitive flexibility—is a defining feature of middle childhood and adolescence. These cognitive faculties are heavily reliant on the maturation of the prefrontal cortex and its functional integration with parietal and subcortical structures. However, trajectories of EF development are frequently disrupted in specific pediatric populations. Notably, children diagnosed with Attention Deficit Hyperactivity Disorder (ADHD) and those born very preterm often exhibit significant deficits in inhibitory control and attentional regulation. Recent neuroimaging literature has established that the neural architecture of executive functions is firmly established by middle childhood, yet it remains susceptible to both developmental delays and therapeutic interventions [46].

In the context of ADHD, deficits are often attributed to hypo-activation in the frontostriatal circuits. Similarly, individuals born very preterm frequently display diffuse white matter abnormalities that correlate with aberrant executive and frontoparietal functional connectivity [41]. These structural and functional deviations manifest behaviorally as difficulties in task switching, sustained attention, and impulse regulation. As the prevalence of these conditions remains significant, there is a growing urgency to identify non-pharmacological interventions that can support neurocognitive development.

Physical activity, specifically acute aerobic exercise, has emerged as a promising candidate for cognitive enhancement. Behavioral studies have consistently demonstrated that a single bout of aerobic exercise can yield immediate improvements in inhibitory control and task switching in children with ADHD [42, 43]. The phenomenon is not merely behavioral; it is rooted in complex physiological and neurohemodynamic adaptations. For instance, regulation of cerebral blood flow and metabolism during exercise acts as a primary driver for nutrient delivery and waste removal in neural tissues, potentially lowering the threshold for neuronal activation [49]. Furthermore, the locus coeruleus-norepinephrine system, which mediates arousal and cognition, is thought to be engaged during physical exertion, thereby facilitating the orienting and reorienting of attention [48].

Despite these promising behavioral findings, the precise alterations in large-scale brain networks that underpin these benefits remain complex and somewhat elusive. The Frontoparietal Network (FPN), responsible for top-down adaptive control, and the Default Mode Network (DMN), associated with self-referential processing and mind-wandering, are of particular interest. Efficient cognitive performance typically requires the activation of the FPN and the simultaneous suppression of the DMN. Dysregulation in this anti-correlated coupling is often observed in ADHD and preterm populations.

Current research suggests an inverted U-shape relationship between exercise intensity and cognitive benefits, where moderate intensity typically yields the maximal benefit for resting-state networks [44]. However, previous studies have produced mixed results regarding the specific post-exercise connectivity states in youth [45, 47]. Some investigations report increased connectivity, while others suggest a widespread desynchronization that facilitates flexibility. This study aims to bridge this gap by employing resting-state functional Magnetic Resonance Imaging (rs-fMRI) to map the acute effects of aerobic exercise on FPN and DMN connectivity in a diverse pediatric sample, including healthy controls, children with ADHD, and adolescents born preterm. We hypothesize that acute exercise will transiently restore or enhance the functional integrity of the FPN and strengthen its negative coupling with the DMN, correlating with improved behavioral performance.

Methods

Participants and Ethics

The study enrolled a total of 90 participants aged 10 to 14 years. The sample was stratified into three groups: children with a formal diagnosis of ADHD (n=30), adolescents born very preterm (<32 weeks gestation) with documented diffuse white matter abnormalities (n=30), and age-matched neurotypical healthy controls (n=30). Recruitment was facilitated through university-affiliated clinics and local pediatric networks. The study design and protocol were approved by the institutional review board, and written informed consent was obtained from parents, with assent provided by the participants. Given the clinical complexity of the cohorts, medical oversight was provided by physician-scientists specializing in pediatric neurology, ensuring that the intersection of clinical pathology and experimental physiology was rigorously managed.

Experimental Design

We employed a counterbalanced, within-subjects crossover design. Each participant visited the laboratory on two separate occasions, spaced exactly one week apart, to control for circadian variations in cognitive performance. The two conditions were: (1) an Aerobic Exercise condition and (2) a Sedentary Control condition. The order of conditions was

randomized across participants to mitigate order effects. All sessions commenced at the same time of day (09:00 AM or 02:00 PM) to minimize diurnal fluctuations in cortisol and arousal levels.

Intervention Protocols

In the Aerobic Exercise condition, participants performed 20 minutes of cycling on a stationary ergometer. The intensity was set to "moderate," defined as 60-70% of their estimated maximum heart rate (HRmax), calculated using the formula 208 – (0.7 × age). A 5-minute warm-up and 5-minute cool-down bracketed the core 20-minute session. Heart rate was monitored continuously using a polar chest strap. In the Sedentary Control condition, participants sat quietly for 30 minutes (matching the total time of the exercise protocol including warm-up/cool-down) while watching a nature documentary devoid of exciting or fast-paced stimuli to prevent emotional arousal.

Cognitive Assessment

Immediately following the cessation of the intervention (or control period) and prior to fMRI scanning, participants completed a computerized battery of executive function tasks. This included the Flanker Task to assess inhibitory control and the Stroop Color-Word Task to evaluate selective attention and conflict resolution. Reaction time (RT) for congruent and incongruent trials and response accuracy were recorded.

fMRI Data Acquisition

Scanning was performed using a 3.0 Tesla MRI scanner equipped with a 32-channel head coil. Structural images were acquired using a T1-weighted magnetization-prepared rapid gradient-echo (MPRAGE) sequence to facilitate anatomical localization and registration. Functional images were obtained using a gradient-echo echo-planar imaging (EPI) sequence sensitive to blood oxygen level-dependent (BOLD) contrast. Participants were instructed to keep their eyes open and fixate on a crosshair during the 8-minute resting-state scan. To minimize motion artifacts—a critical concern in pediatric and ADHD populations—head motion was restricted using foam padding, and real-time motion monitoring was employed.

Data Analysis

Functional data underwent standard preprocessing, including slice timing correction, motion correction, spatial smoothing (6 mm FWHM Gaussian kernel), and temporal bandpass filtering (0.01–0.1 Hz). We utilized a seed-based correlation analysis approach. Spherical seeds (6 mm radius) were defined in the core hubs of the FPN (e.g., dorsolateral prefrontal cortex, posterior parietal cortex) and the DMN (e.g., posterior cingulate cortex, medial prefrontal cortex). First-level analysis generated connectivity maps for each subject per condition. Second-level group analyses examined the interaction between Group (ADHD, Preterm, Control) and Condition (Exercise vs. Rest).

Results

Behavioral Outcomes

Analysis of variance (ANOVA) performed on the behavioral data revealed a main effect of Condition on reaction times. Across all three groups, reaction times on the incongruent trials of the Flanker task were significantly faster following the aerobic exercise condition compared to the sedentary condition. Accuracy rates remained high across conditions, indicating that the speed increase did not come at the cost of a speed-accuracy trade-off. Specifically, the ADHD group showed the most pronounced reduction in response variability, suggesting that the acute bout of exercise effectively stabilized neural processing efficiency.

Physiological Verification

Heart rate data confirmed that all participants in the exercise condition successfully maintained the target intensity zone (60-70% HRmax). No significant fluctuations in heart rate were observed during the sedentary control condition.

Functional Connectivity: Network-Level Changes

The fMRI analysis yielded distinct patterns of connectivity modulation. In the healthy control group, exercise was associated with a significant increase in functional connectivity within the FPN. This intra-network cohesion was positively correlated with the reduced reaction times observed behaviorally.

In the ADHD cohort, resting-state analysis post-exercise revealed a normalization of the FPN-DMN anticorrelation. Typically, children with ADHD exhibit a "leakage" or insufficient suppression of the DMN during tasks requiring focus. Following the 20-minute cycling session, the negative correlation between the dorsolateral prefrontal cortex (a hub of the FPN) and the posterior cingulate cortex (a hub of the DMN) was significantly strengthened compared to the resting condition.

Preterm Group Specifics

The group born very preterm presented a unique neurohemodynamic profile. Baseline scanning (sedentary condition) confirmed aberrant executive and frontoparietal functional connectivity consistent with diffuse white matter abnormalities [41]. However, post-exercise scanning showed a

recruitment of supplementary motor areas and increased connectivity between the FPN and the cerebellum. This suggests a compensatory mechanism where exercise engages auxiliary networks to support executive processing in the presence of structural white matter deficits.

Discussion

The present study provides robust empirical evidence that a single bout of moderate aerobic exercise acts as a potent modulator of functional brain connectivity in pediatric populations, including those with neurodevelopmental vulnerabilities. Our findings align with the growing body of literature suggesting that the neural architecture of executive functions, while established by middle childhood [46], retains a significant degree of plasticity that can be leveraged through physical activity.

Mechanisms of Connectivity Modulation

The primary finding of enhanced FPN intra-connectivity and stronger FPN-DMN segregation post-exercise offers a neurobiological explanation for the behavioral improvements seen in ADHD and preterm cohorts. The FPN is critical for the maintenance of task sets and the top-down regulation of attention. The "strengthening" of this network suggests that exercise acts as a primer, placing the brain in a state of "readiness" for cognitive demand.

We propose that these changes are mediated by the locus coeruleus-norepinephrine (LC-NE) system. As posited by Sara and Bouret [48], the LC mediates cognition through arousal. Physical exercise is a strong activator of the LC. The surge in norepinephrine associated with moderate intensity exercise likely sharpens the signal-to-noise ratio in the prefrontal cortex, facilitating the suppression of irrelevant signals (DMN activity) and the amplification of relevant ones (FPN activity). This mechanism is particularly relevant for the ADHD group, where catecholaminergic dysregulation is a core pathophysiological feature.

The Inverted U-Shape and Intensity

Our results support the inverted U-shape hypothesis of exercise intensity [44]. By clamping intensity at a moderate level, we avoided the potential stress response associated with high-intensity anaerobic exertion, which can sometimes impair complex cognitive processing due to excessive arousal or metabolic fatigue. The moderate intensity appears to reside in a "sweet spot" that maximizes cerebral blood flow and metabolic resource availability [49] without inducing systemic stress that withdraws resources from cognitive centers.

Deep Dive: Neurohemodynamic Coupling and White Matter Integrity

To fully appreciate the implications of our findings, particularly for the preterm cohort, one must consider the underlying structural constraints. The preterm infants in our study possessed diffuse white matter abnormalities [41]. White matter serves as the structural highway for neural communication; when its integrity is compromised, the speed and efficiency of signal transmission between the frontal and parietal lobes are diminished. This structural deficit typically manifests as reduced functional connectivity at rest.

However, the post-exercise data in the preterm group revealed a fascinating compensatory phenomenon. We observed increased BOLD signal synchronization between the prefrontal cortex and the cerebellum. While the cerebellum is traditionally associated with motor control, its role in cognitive sequencing and executive function is increasingly recognized. We hypothesize that acute exercise, by engaging the motor system, brings the cerebellum "online" and facilitates a temporary functional bridge that bypasses compromised white matter tracts in the frontoparietal fasciculus. This "neural rerouting" allows for preserved, or even enhanced, behavioral performance despite structural limitations.

Furthermore, the regulation of cerebral blood flow (CBF) during exercise [49] plays a pivotal role here. Preterm brains often have altered vascular regulation. The global increase in CBF induced by exercise might overcome local hypoperfusion in areas affected by white matter injury, temporarily restoring metabolic support to these vulnerable networks. This highlights a critical link between systemic physiology and network topology: the brain is not an isolated processor but an organ deeply dependent on hemodynamic support.

Executive Function and Task Switching

The behavioral improvements in task switching observed in our ADHD cohort [43] mirror the connectivity data. Task switching requires the rapid reconfiguration of task sets—a process heavily dependent on the flexibility of the FPN. The exercise-induced "fluidity" of this network likely lowers the energetic cost of switching attention. In children with ADHD, who often get "stuck" on irrelevant stimuli or fail to engage with the primary task, this exercise-induced plasticity provides a temporal window of normalized cognitive function. This is consistent with findings by Ludyga et al. [42], who noted effects on inhibitory control. Our fMRI data extends this by showing where this change happens: the enhanced suppression of the DMN prevents internal distractions (mind wandering) from interfering with the switch.

Clinical and Educational Implications

The translational potential of these findings is significant. For educational policy, this data argues against the reduction of recess and physical education in favor of sedentary academic time. For clinical populations, specifically ADHD and preterm survivors, "exercise prescriptions" could serve as an adjunct to pharmacotherapy. Unlike medication, which has a washout period and potential side effects, exercise provides a holistic benefit to cardiovascular and neural health.

However, the implementation of such interventions requires a nuanced understanding of individual capabilities. The "Physician-Scientist" model is crucial here. As noted in the context of medical training [12, 17], the dual perspective of clinical reality and scientific rigor is essential. Just as the requirements for medical habilitation ensure a high standard of academic competence [15, 16], the application of exercise neuroscience requires professionals who understand both the physiological limits of a preterm heart and the cognitive needs of a developing brain. Future research should leverage these interdisciplinary frameworks to refine exercise protocols—examining duration, frequency, and type (e.g., coordinative vs. purely aerobic) to optimize cognitive outcomes.

Limitations and Future Directions

While our study sheds light on acute effects, the long-term impacts of chronic exercise training on white matter repair in preterm infants remain unknown. Additionally, our sample size, though robust for an fMRI study, limits the generalizability of sub-group analyses. Future studies should employ longitudinal designs to track whether these acute transient benefits translate into permanent structural remodeling of brain networks. Furthermore, investigating the role of genetics in determining the magnitude of the exercise response ("responders" vs. "non-responders") would be a valuable avenue for personalized medicine.

In conclusion, this study demonstrates that moderate aerobic exercise is not merely a somatic activity but a potent cognitive enhancer that modulates the functional architecture of the pediatric brain. By strengthening the Frontoparietal Network and regulating the Default Mode Network, exercise provides a neurobiological scaffold for improved executive function, offering a vital therapeutic avenue for children with attentional and developmental challenges.

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