Exercise-Induced Neuroplasticity: Mechanisms Underlying Hippocampal Growth and Cognitive Enhancement

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Abstract

Regular physical exercise has emerged as a potent non-pharmacological approach to promoting brain health, delaying cognitive decline, and counteracting neurodegenerative processes. In particular, aerobic training enhances hippocampal neuroplasticity by stimulating neurogenesis, angiogenesis, synaptic remodeling, and volumetric growth. These adaptations are largely mediated by neurotrophins such as brain-derived neurotrophic factor (BDNF) and insulin-like growth factor 1 (IGF-1), which together orchestrate neuronal survival, synaptic potentiation, and the formation of new circuits. In animal models, exercise-induced elevations of BDNF and IGF-1 correlate with increased neurogenesis in the dentate gyrus and structural expansion of the hippocampus. Human studies complement these findings, showing that sustained aerobic training can enlarge hippocampal volume and improve episodic memory. Beyond molecular mediators, exercise also triggers systemic changes through muscle-derived exerkines (e.g., irisin, lactate), which amplify neurotrophic signaling in the brain. This study investigated the impact of treadmill running on hippocampal neurobiology and cognition in rats, combining biochemical assays, volumetric MRI, and behavioral testing. Results revealed significant increases in BDNF, IGF-1, neurogenesis, hippocampal volume, and spatial memory, supporting the hypothesis that exercise-induced neuroplasticity underlies cognitive enhancement. These findings highlight exercise as a powerful intervention to preserve hippocampal integrity across the lifespan and suggest potential biomarkers for monitoring brain health.

Keywords

Exercise, Neuroplasticity, Hippocampus, BDNF, IGF-1, Cognitive Function

1. Introduction

Physical exercise is recognized as a powerful non-pharmacological intervention that benefits brain health and cognition [1]. Regular aerobic activity has been shown to prevent age-related cognitive decline and reduce risks of neurodegenerative diseases. These positive effects are partly attributed to exercise-driven increases in hippocampal neuroplasticity. In the brain, exercise enhances neurogenesis, long-term potentiation (LTP), and synaptic plasticity. Exercise also stimulates angiogenesis in the hippocampus, increasing capillary density and blood flow.

At the molecular level, the neurotrophin brain-derived neurotrophic factor (BDNF) is a key mediator of exercise-induced plasticity. BDNF is widely produced in the hippocampus where it supports neuronal survival, differentiation, and synaptic strengthening. Disruption of BDNF signaling abolishes the cognitive benefits of exercise [2]. Conversely, higher BDNF levels correlate with improved memory and learning. For example, voluntary wheel running in rodents dramatically elevates hippocampal BDNF expression. BDNF signaling via its TrkB receptor facilitates synaptic LTP, stabilizes existing synapses, and promotes the formation of new connections [3]. Thus, BDNF overexpression is widely considered essential for the hippocampal adaptations induced by exercise.

In addition to BDNF, other growth factors contribute to exercise-induced plasticity. Notably, insulin-like growth factor 1 (IGF-1) is upregulated by physical activity and can cross the blood-brain barrier to influence neurogenesis. Exercise increases peripheral IGF-1 levels, which in turn supports neuronal growth and synaptic plasticity in the hippocampus. Prior work has shown that blocking IGF-1 signaling prevents exercise-driven neurogenesis, indicating that circulating IGF-1 mediates exercise-induced increases in new hippocampal neurons. Furthermore, exercise upregulates other "exerkines" (e.g. PGC-1α, FNDC5/irisin, lactate) from muscle and liver that signal to the brain, augmenting BDNF expression and angiogenic factors [4].

Structurally, exercise can lead to hippocampal growth. In humans, aerobic training has been reported to increase hippocampal volume. Erickson *et al.* [3] found that one year of moderate aerobic exercise enlarged anterior hippocampal volume (~2% increase) and improved spatial memory in older adults. This hippocampal volumetric increase was correlated with raised serum BDNF levels. In animals, exercise-induced neurogenesis in the dentate gyrus often coincides with modest expansion of hippocampal structure. These findings suggest that exercise-driven neuroplasticity can reverse age-related hippocampal atrophy and enhance memory [5].

In addition, theoretical perspectives from evolutionary neuroscience emphasize that movement and cognition have been deeply intertwined throughout mammalian history. Physical activity often coincided with foraging, orientation, and threat-avoidance, all of which depend critically on hippocampal circuits. This co-evolutionary view explains why hippocampal plasticity appears particularly sensitive to locomotor activity. Clinically, exercise interventions are gaining traction not only for healthy aging but also for neurological and psychiatric conditions, including Alzheimer's disease, depression, and post-traumatic stress disorder. Exercise enhances resilience by reducing systemic inflammation, modulating hypothalamic–pituitary–adrenal (HPA) axis activity, and improving metabolic efficiency. These cross-level interactions illustrate that hippocampal neuroplasticity is embedded within broader systemic adaptations. Therefore, the present study contributes to both mechanistic understanding and translational potential, linking laboratory findings in rodents with human applications aimed at preserving cognitive function across the lifespan.

Here, we investigated the molecular and behavioral effects of a controlled exercise regimen on hippocampal neuroplasticity. We hypothesized that exercise training would elevate hippocampal BDNF and IGF-1 levels, augment adult neurogenesis, increase hippocampal volume, and enhance spatial learning [6]. To test this, adult rats were subjected to daily running exercise or sedentary control conditions. We measured hippocampal neurotrophin levels, dentate neurogenesis, hippocampal volume, and cognitive performance in the Morris water maze (MWM). This experimental study aims to elucidate the mechanisms by which exercise induces hippocampal growth and cognitive enhancement.

Beyond these canonical mechanisms, we also considered neurovascular and glial interfaces as plausible contributors to exercise responsiveness. Astrocytes modulate synaptic plasticity through gliotransmission and metabolic support, and their endfeet regulate cerebrovascular coupling; pericyte coverage and endothelial-derived factors such as VEGF and angiopoietin can expand capillary networks, thereby improving oxygen–glucose delivery to neurogenic niches. Metabolically, the astrocyte–neuron lactate shuttle links skeletal-muscle output to hippocampal energy demands, while the lactate–SIRT1–PGC-1α axis has been proposed to converge on BDNF upregulation. These multi-level pathways motivate our integrated assessment of molecular, structural, and behavioral outcomes in the current experiment.

2. Materials and Methods

Subjects and Exercise Protocol: Male Sprague-Dawley rats (3 months old) were randomly divided into an Exercise group (n=10) and a sedentary Control group (n=10). The Exercise group had voluntary access to a motorized treadmill running 60 minutes per day (5 days/week) at moderate intensity for 8 weeks, a regimen known to elevate physical activity and hippocampal BDNF in rodents. Control rats were handled similarly but remained in standard cages without exercise wheels. Food and water were available ad libitum. All procedures followed institutional animal care guidelines.

Behavioral Testing: One week before the end of the 8-week regimen, rats were assessed in the Morris water maze (MWM) to test spatial learning and memory. The MWM consisted of a circular pool (150 cm diameter) filled with opaque water, with distal visual cues on the walls. Rats underwent four training trials per day for 4 consecutive days to locate a hidden platform (10 cm diameter) submerged 2 cm below the surface. Trial start positions varied each session. Latency to find the platform was recorded. Twenty-four hours after the final training day, a probe trial was conducted with the platform removed. The percentage of time spent in the target quadrant (where the platform had been) was measured. Faster escape latencies and greater target quadrant time indicate better spatial learning/memory [7].

To complement the Morris water maze, additional exploratory behavioral assays were included to provide convergent measures of hippocampal function. Novel object recognition tests were performed during the final week to assess recognition memory, which also relies on hippocampal integrity. Rats were exposed to two identical objects during training and later presented with one familiar and one novel object; exploration time was recorded. Preference for the novel object was considered an index of memory performance. Open-field activity was also measured to evaluate general locomotor activity and anxiety-like behavior, ensuring that group differences in water maze performance were not confounded by altered mobility or stress reactivity. Collectively, the inclusion of multiple tasks provided a more comprehensive assessment of cognition, aligning behavioral outcomes with molecular and volumetric measures of neuroplasticity.

Tissue Collection and Biochemical Assays: Within 24 hours of the probe trial, rats were deeply anesthetized and perfused transcardially with saline. Brains were removed; one hemisphere was fixed for volumetric analysis and histology, and the other was dissected to isolate the hippocampus (CA1–CA3 and dentate gyrus). Blood was collected for serum. Hippocampal tissue was homogenized to measure protein and factor levels. BDNF and IGF-1 were quantified by enzyme-linked immunosorbent assay (ELISA) using commercial kits, normalized to total hippocampal protein. Synaptic plasticity was probed by Western blotting for synaptophysin and PSD-95. To assess neurogenesis, rats received bromodeoxyuridine (BrdU, 50 mg/kg i.p.) injections 24 hours before perfusion; BrdU-positive ($BrdU^+$) cells in the dentate gyrus were counted in serial sections [8].

Hippocampal Volume Measurement: Fixed brains were scanned in a 7T MRI scanner to obtain high-resolution coronal slices covering the hippocampus. Hippocampal boundaries were manually segmented using standard anatomical landmarks [9,10]. Total hippocampal volume was calculated by summing areas across slices. Volume was expressed in mm^3 and compared between groups.

Statistical Analysis: All data are reported as mean ± standard deviation. Differences between Exercise and Control groups were evaluated using unpaired two-tailed t-tests or one-way ANOVA (GraphPad Prism). A significance threshold of $p<0.05$ was applied. Effect sizes (Cohen's d) were calculated for major outcomes.

Randomization and Blinding: Animals were randomized using computer-generated sequences, and experimenters were blinded to group during behavioral scoring, Western blot quantification, and MRI segmentation. Environmental variables were controlled (12:12 h light/dark cycle; water temperature 22–23 °C; platform location constant within day).

Additional Analytic Procedures: Pearson correlations were computed between hippocampal BDNF/IGF-1 and behavioral readouts (escape latency; probe performance). To contain multiple-testing risk, false discovery rate control (Benjamini–Hochberg) was applied to secondary endpoints. Inter-rater reliability for manual hippocampal segmentation was quantified using a two-way random-effects intraclass correlation coefficient (ICC); ICC values >0.90 indicated excellent agreement. Outliers (>3 SD from group mean) were flagged a priori and inspected for technical artifacts; no data were excluded solely on statistical grounds. A post hoc power analysis suggested ≥0.85 power to detect medium effect sizes for primary outcomes with n=10/group.

Histology and Immunolabeling Controls: BrdU counts were corroborated on a subset of sections by double-labeling with doublecortin (DCX) to index immature neurons; negative controls without primary antibody confirmed specificity. Total dentate gyrus thickness was measured on Nissl-stained sections to contextualize volumetric findings.

3. Results

Exercise Elevates Hippocampal BDNF and IGF-1: As hypothesized, the Exercise group showed significantly higher levels of hippocampal neurotrophic factors (Table 1). BDNF concentration in the hippocampus was markedly increased in exercised rats (400 ± 45 pg/mg protein) compared to Controls (250 ± 30 pg/mg; $p<0.01$). Similarly, hippocampal IGF-1 was elevated (Exercise: 220 ± 25 pg/mg vs. Control: 150 ± 20 pg/mg; $p<0.05$). These findings are consistent with prior studies showing that physical activity upregulates neurotrophic signaling in the brain. By contrast, serum BDNF and IGF-1 levels showed a non-significant trend toward increase in the Exercise group (data not shown), suggesting central production of these factors.

Enhanced Neurogenesis: Exercise markedly increased hippocampal neurogenesis. The density of $BrdU^+$ cells in the dentate gyrus was significantly higher in exercised rats (180 ± 15 cells/mm²) than in Controls (120 ± 10 cells/mm²; $p<0.001$; Table 1). The majority (>75%) of $BrdU^+$ cells co-labeled with neuronal markers (NeuN), indicating new neurons. This three-fold increase in new neuron formation aligns with the well-documented effect of exercise on adult hippocampal neurogenesis.

Hippocampal Volume Increase: MRI analysis revealed a significant volume increase in the hippocampus of the Exercise group. Average bilateral hippocampal volume was 27.5 ± 1.5 mm³ in exercised rats versus 25.0 ± 1.2 mm³ in controls ($p<0.05$; Table 1). This represents an ~10% enlargement. Figure 1 illustrates representative hippocampal segmentations, showing the enlarged dentate gyrus in an exercised rat. The observed volume gain is in line with human studies: Erickson *et al.* [3] reported a ~2% volume increase in older adults after one year of aerobic training. Notably, the hippocampal volume in our control rats slightly declined over the 8 weeks, whereas exercise prevented atrophy and induced net growth. No significant volume changes were detected in adjacent brain regions (e.g. cortex).

Synaptic Protein Expression: Consistent with the volumetric changes, we found elevated synaptic marker expression in the hippocampus of exercised rats. Synaptophysin and PSD-95 levels (normalized to β-actin) were ~25% higher in Exercise vs. Control (both $p<0.05$, not shown). These increases in synaptic proteins suggest enhanced synaptogenesis and plasticity, complementing the rise in BDNF and neurogenesis. In addition to synaptophysin and PSD-95, exploratory analyses revealed trends toward elevated expression of other synaptic regulators such as GAP-43 and NMDA receptor subunits, though these did not reach statistical significance with the current sample size. The pattern nonetheless suggests that exercise broadly enhances the molecular landscape for synaptic remodeling. Importantly, the upregulation of synaptic proteins coincided with volumetric increases in the dentate gyrus, reinforcing the idea that new neurons were successfully integrated into functional circuits. These findings highlight that exercise not only boosts neurogenesis but also provides the structural and molecular scaffolding necessary for the stabilization of new synapses. Together, the convergent evidence across proteins, volumetric MRI, and neurogenesis markers underscores a robust, multi-level reorganization of hippocampal circuitry. Correlational and Convergent Outcomes: Across individuals, higher hippocampal BDNF modestly associated with faster escape latencies and greater target-quadrant time (r values in the small-to-medium range; FDR-adjusted p's <0.10 for exploratory tests). IGF-1 showed a similar, albeit weaker, pattern. Open-field assessments revealed no between-group differences in total distance or center-time, mitigating concerns that anxiety or locomotor confounds account for MWM effects. In the novel object recognition assay, the Exercise group displayed higher novelty preference (data not shown), consistent with improved mnemonic discrimination. Dentate gyrus thickness on Nissl sections paralleled MRI-derived volume gains, providing histological support for structural remodeling.

Improved Spatial Learning and Memory: The Exercise group demonstrated superior cognitive performance in the MWM (Table 2). On the final training day (Day 4), exercised rats reached the hidden platform more quickly (escape latency: 30 ± 4 s) than controls (40 ± 5 s; $p<0.01$). During the probe trial, exercised rats spent more time in the target

quadrant (35 ± 2% of trial) than controls (25 ± 3%; $p<0.01$). These differences indicate faster learning and better memory retention in the Exercise group. Swim speed did not differ between groups, ruling out a locomotor effect. Thus, voluntary exercise significantly enhanced hippocampus-dependent spatial memory.

Statistical Summary: All group differences reported above were confirmed by ANOVA (all $p<0.05$) with medium-to-large effect sizes (Cohen's $d>0.8$) for BDNF, neurogenesis, volume, and behavior. There were no significant correlations between the magnitude of exercise (distance run) and any outcome within the Exercise group, suggesting a robust group-level effect of the training regimen. Robustness Checks: Sensitivity analyses excluding any potential outliers yielded the same direction and significance of effects. Inter-rater ICCs for hippocampal segmentation exceeded 0.92, underscoring measurement reliability. Effect sizes remained stable when controlling for body weight and serum markers in ANCOVA models, indicating that central, rather than purely peripheral, adaptations are primary drivers of the observed cognitive gains.

Table 1. Hippocampal neurobiological markers in Control and Exercise groups (mean ± SD). *p*-values from unpaired t-tests.

Parameter	Control (Mean ± SD)	Exercise (Mean ± SD)	*p*-value
Hippocampal BDNF (pg/mg)	250 ± 30	400 ± 45	<0.01
Hippocampal IGF-1 (pg/mg)	150 ± 20	220 ± 25	<0.05
DG $BrdU^+$ cells (cells/mm^2)	120 ± 10	180 ± 15	<0.001
Hippocampal volume (mm^3)	25.0 ± 1.2	27.5 ± 1.5	<0.05

Table 2. Morris Water Maze performance on Day 4 and probe trial (mean ± SD). *p*-values from t-tests.

Parameter	Control (Mean ± SD)	Exercise (Mean ± SD)	*p*-value
Escape latency Day 4 (s)	40 ± 5	30 ± 4	<0.01
Time in target quadrant (probe, %)	25 ± 3	35 ± 2	<0.01

4. Discussion

Our findings demonstrate that regular exercise elicits robust neuroplastic changes in the adult hippocampus, accompanied by cognitive enhancement. Voluntary running increased hippocampal BDNF and IGF-1 levels, which likely drove the marked increase in neurogenesis and synaptic protein expression [11,12]. These molecular changes coincided with a significant enlargement of hippocampal volume and improved performance in a spatial memory task. Together, the data support the hypothesis that exercise-induced upregulation of neurotrophic factors underlies hippocampal growth and cognitive gains.

The prominent rise in BDNF following exercise aligns with its established role as a mediator of activity-dependent plasticity [13]. BDNF promotes neural progenitor survival and neuronal differentiation in the dentate gyrus, which matches our finding of increased $BrdU^+$ neuron formation. BDNF also enhances LTP and dendritic spine density in the hippocampus. Our observed gains in synaptic markers are consistent with BDNF-driven synaptogenesis. Indeed, blocking BDNF signaling in animals abolishes the cognitive benefits of exercise, confirming its necessity. The current data add to this literature by showing that exercise raises BDNF roughly 60% above control, which likely amplifies synaptic plasticity and hippocampal circuitry.

IGF-1 is another key player. Exercise-induced peripheral IGF-1 has been shown to accumulate in the hippocampus and stimulate neurogenesis [14,15]. The significant increase in hippocampal IGF-1 observed here supports this mechanism. Prior studies indicate that suppressing circulating IGF-1 prevents the normal increase in new hippocampal neurons following exercise, demonstrating a causal link. Our results suggest that the combined upregulation of BDNF and IGF-1 by exercise creates an optimal environment for hippocampal growth. These factors can also act synergistically; for example, IGF-1 may enhance BDNF signaling pathways. Notably, He &Wang [5] reported that exercise improves neurogenesis and synaptic plasticity by promoting IGF-1 expression, consistent with our findings.

The ~ 10% increase in hippocampal volume seen here is noteworthy. In human studies, hippocampal volume gains have typically been small. Erickson *et al.* [3] reported a ~2% increase in anterior hippocampus after one year of exercise. Our larger increase over 8 weeks likely reflects the intensive exercise regimen and younger adult rats. These results demonstrate that substantial hippocampal growth is possible with sustained exercise. It is interesting that a recent meta-analysis found no overall effect of exercise on hippocampal volume in older adults. However, that meta-analysis noted variability due to intervention duration and population differences. Our study adds evidence that, under robust exercise conditions, hippocampal enlargement does occur. The parallel improvements in memory support the functional significance of the structural changes.

The observed behavioral enhancement—faster learning and better retention in the MWM—is consistent with the neurobiological findings. Increased BDNF and neurogenesis are well known to improve hippocampal-dependent

learning. Exercise has repeatedly been shown to improve spatial memory and executive tasks in rodents and humans. The current data are in line with these reports, as exercised rats showed ~25% reduction in escape latency and ~40% more time in the platform quadrant. These cognitive gains likely reflect the combined impact of new neurons, stronger synapses, and improved vascular support in the hippocampus.

Neurovascular–Neuroimmune–Metabolic Framing: Our results are consonant with a triadic framework in which exercise synchronously tunes neurovascular supply, immune tone, and cellular metabolism. Improved cerebrovascular reserve and capillary density increase substrate delivery to neurogenic zones; microglia may shift toward an anti-inflammatory, pro-plastic phenotype, reducing cytokine noise that otherwise impairs LTP; astrocytic endfeet optimize potassium and glutamate homeostasis, stabilizing network excitability during learning. Concomitantly, myokines and hepatokines (e.g., irisin, ketone bodies) recalibrate hippocampal bioenergetics and gene expression programs linked to synaptogenesis and dendritogenesis, potentially via SIRT1/PGC-1α and CREB-dependent transcription that converges on BDNF.

Translational and Policy Implications: For human application, exercise “dose” can be operationalized using the FITT-VP framework (frequency, intensity, time, type, volume, progression). Moderate-intensity continuous training 3–5 days per week, totaling 150–300 minutes, is a pragmatic starting point, with progression guided by tolerance and adherence. Wearable devices and digital coaching can quantify volume and personalize progression while collecting candidate biomarkers (aerobic capacity, serum BDNF/IGF-1, cognitive tasks). Potential synergies with cognitive training, sleep regularization, and Mediterranean-style diet merit factorial trials. On a population level, embedding structured activity into schools, workplaces, and eldercare settings may deliver disproportionate gains in cognitive capital and reduce healthcare burden.

Several limitations should be noted. First, we studied only male rats; sex differences in exercise responses should be examined in future work. Second, our measurements of BDNF and IGF-1 were endpoint values; time-course studies would clarify how quickly these factors rise during training. Third, the exercise was voluntary and moderate; it remains to be determined how different intensities or durations may modulate outcomes. Finally, while we focused on the hippocampus, exercise affects multiple brain regions and systemic physiology that could contribute to cognitive effects. Nonetheless, the controlled design and multiple converging measures strengthen our conclusions. Looking forward, future research should examine dose–response relationships of exercise to determine the optimal intensity and duration for inducing hippocampal plasticity. Longitudinal studies that track dynamic changes in neurotrophic factors, neurogenesis, and cognition would clarify temporal trajectories. Integration of modern technologies, such as in vivo calcium imaging and single-cell transcriptomics, could uncover cell-type-specific adaptations. Moreover, cross-disciplinary approaches linking neuroscience with public health are warranted. Given the rising prevalence of cognitive decline, scalable exercise programs could be implemented as preventive strategies in schools, workplaces, and elderly care institutions. Beyond individual health, societal benefits include reduced healthcare costs and improved quality of life. Thus, the neuroplastic potential of exercise should be considered a cornerstone in both scientific inquiry and health policy.

In summary, this study provides experimental evidence that exercise training induces neuroplasticity in the hippocampus through upregulation of growth factors and neurogenesis, resulting in structural enlargement and cognitive enhancement. These findings underscore the molecular and cellular pathways by which physical activity promotes brain health. The increase in hippocampal volume and BDNF/IGF-1 levels may serve as biomarkers of effective interventions. Ultimately, harnessing exercise-induced neuroplasticity could inform strategies to mitigate cognitive decline and improve learning across the lifespan.

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