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Effects of Life Kinetic training on neuroplasticity-related neurotrophic factors: A controlled intervention study

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Abstract

The aim of this study is to investigate whether Life Kinetic training - an educational program that stimulates the neuronal learning process, restructures neural networks, reduces neural symptoms, and improves concentration and visual system performance - has an effect on the development of certain neurotrophic factors in basketball players. Additionally, the study aims to highlight the impact of Life Kinetic training as a tool for enhancing athletic performance across various sports disciplines. A total of 30 male athletes, aged between 15 and 18, voluntarily participated in the research, divided into an experimental group (n=15) and a control group (n=15). Participants' height, body weight, and body mass index (BMI) were measured. To identify neurotrophic factors, Brain Derived Neurotrophic Factor (BDNF), Nerve Growth Facor (NGF), Neurotrophin – 3 (NT-3), and Neurotrophin – 4 (NT-4) levels were analyzed. All tests were administered as pre-tests and post-tests. The data were statistically evaluated using SPSS 25.0 software. The Paired Sample T-test was utilized to determine intra-group changes between pre-test and post-test measurements, while the Independent Sample T-test was used to compare variables between the experimental and control groups. Results were evaluated at a 95% confidence interval, with $p < 0.05$ considered statistically significant. Statistical evaluation revealed a significant difference in BDNF and NGF parameters between the groups. Although no significant difference was found between groups for NT-3 and NT-4 parameters, the experimental group showed greater improvement in pre-test and post-test values compared to the control group. In conclusion, it can be stated that Life Kinetic training applied for duration of 2 months (8 weeks) has a positive effect on the development of neurotrophic factors in basketball players.

Introduction

In recent years, the relationship between physical activity and neurocognitive health has become a central focus in the fields of exercise science and neuroscience. Beyond its well-documented effects on cardiovascular, metabolic, and musculoskeletal systems, regular physical exercise is now recognized for its capacity to induce significant adaptations in the central nervous system. Among emerging neuromotor training modalities, Life Kinetic (LK) represents an innovative approach designed to simultaneously stimulate motor and cognitive functions. Unlike traditional coordinative training that focuses on the mastery and automation of movement, LK emphasizes the continuous introduction of novel and non-automated tasks, preventing neural adaptation and forcing the brain to constantly form new compensatory networks. Developed by German football coach Horst Lutz, LK is structured around motor tasks integrated with perceptual and cognitive challenges, aiming to activate cortical areas and enhance the efficiency of information processing (Duda, 2015).

The theoretical underpinning of LK training lies in the concept of neuroplasticity, defined as the brain's ability to reorganize its structure, function, and connections in response to internal and external stimuli. This plasticity is essential for learning, memory formation, motor skill acquisition, and adaptation to novel environmental demands (Nelson, 2000). This structural reorganization is mediated by a specific

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biochemical cascade where physical stimuli trigger the release of neurotrophic factors, which in turn modulate synaptic efficacy and enhance cognitive domains such as executive function and memory. Specifically, synaptic plasticity, a fundamental component of neuroplasticity, refers to activity-dependent changes in synaptic strength and density, which are vital for enhancing inter-neuronal communication. The motor-cognitive complexity imposed by LK training is hypothesized to act as a potent stimulus for promoting such plastic changes, particularly within regions such as the hippocampus and prefrontal cortex - areas critically involved in executive functioning and sensorimotor integration (Hyde et al., 2009).

A growing body of evidence suggests that interventions that challenge the brain through novel, coordinated, and cognitively demanding motor tasks, such as those involved in LK training, can lead to the formation of new synaptic pathways, thereby improving concentration, coordination, and psychomotor performance (Lutz, 2014). This is supported by findings indicating that increased synaptogenesis correlates with improved neural efficiency and cognitive performance. Moreover, it has been proposed that sustained exposure to such multimodal stimuli may facilitate functional and structural neuroplasticity, both of which are regarded as prerequisites for achieving elite-level athletic performance (Debarnot et al., 2014; Jacini et al., 2009).

In the biological context, neuroplasticity involves a cascade of processes, including dendritic branching, axonal sprouting, and synapse remodeling. These neuroadaptive mechanisms are regulated in part by neurotrophic factors, which serve as essential biochemical mediators for neuronal growth, survival, and synaptic modulation. Among these, Brain-Derived Neurotrophic Factor (BDNF) plays a prominent role in modulating synaptic efficacy, long-term potentiation, and cognitive plasticity (Kazak & Yarım, 2014). Physical exercise has consistently been shown to enhance BDNF expression, linking motor activity with higher-order brain functions such as attention, problem-solving, and learning (Uzbay, 2004; Yau et al., 2016).

Exercise-induced increases in BDNF and other neurotrophins have also been implicated in the prevention of neurodegenerative diseases, including Alzheimer's and Parkinson's disease. Indeed, impaired neurotrophin synthesis is associated with cognitive decline and neuronal atrophy, whereas physical activity appears to counteract these effects by promoting neurogenesis and synaptic integrity (Manni et al., 2013; Yoo et al., 2003). The hippocampus, a brain region integral to memory and spatial navigation, is particularly responsive to such neurobiological adaptations, and volumetric increases in this region have been reported following both aerobic and coordinative training programs (Hötting & Röder, 2013).

Given the documented relationship between physical activity, neuroplasticity, and neurotrophic modulation, it is plausible to suggest that the unique motor-cognitive stimulation of LK could have a measurable effect on neurobiological markers. While the cognitive benefits of LK are increasingly documented, empirical evidence regarding its molecular impact remains scarce. Most research has utilized aerobic or resistance exercises, leaving a critical gap in understanding how integrative, non-automated motor-cognitive training specifically alters the concentration of neurotrophic biomarkers in athletic populations. (Özocak et al., 2019; Kolb et al., 2003).

The selection of basketball players for this study is predicated on the high neurocognitive and sensorimotor demands inherent in the sport. Basketball is characterized by rapid decision-making, spatial awareness, and the integration of complex motor patterns under high-pressure conditions, such as simultaneous dribbling, field scanning, and multi-directional movement. These sport-specific demands closely align with the core principles of Life Kinetic (LK) training, which utilizes non-automated motor tasks to challenge the central nervous system. By targeting the intersection of motor coordination and cognitive processing, it is hypothesized that LK training will be particularly effective for basketball players, whose performance relies heavily on neural efficiency and neuroplasticity facilitated by neurotrophic factors such as BDNF. In light of these considerations, the current study hypothesizes that an 8-week Life Kinetic training intervention will lead to a statistically significant increase in serum BDNF and NGF levels among adolescent basketball players compared to a control group, while also eliciting a positive directional trend in NT-3 and NT-4 concentrations as an indicator of broader neuro-molecular adaptation.

Methods

Participants

This study included 30 healthy, licensed male basketball players aged 15 to 18, who were actively training and registered in Hakkari Province. Participants were free

from any known medical conditions or injuries that might interfere with participation in physical activity. The sample size was determined based on a power analysis using G*Power (version 3.1.9.6, Germany); based on a predicted effect size of f = 0.40, an alpha level of 0.05, and a power of 0.80, a minimum total sample size of 24 participants was required. Accordingly, the 30 athletes were assigned to the experimental group (n = 15) or the control group (n = 15) using a computer-generated random sequence. While both groups continued their regular basketball training, the experimental group additionally participated in an 8-week Life Kinetic (LK) program, consisting of three 30-minute sessions per week following their routine practice.

All procedures were conducted at the indoor sports facility of the Hakkâri Provincial Directorate of Youth and Sports. Prior to testing, written informed consent was obtained from the participants' legal guardians, and ethical approval was granted by the Ethics Committee of Atatürk University Faculty of Sports Sciences (Ethics approval no.: E-70400699-000-2200099727). Medical clearance was obtained for all participants to confirm their eligibility to participate.

Life Kinetic Training Protocol

The experimental group received an 8-week LK exercise program conducted three times per week (Mondays, Wednesdays, and Fridays) immediately following their routine basketball practice. Each LK session lasted 30 minutes and was structured into three distinct phases: a 5-minute cognitive warm-up, 20 minutes of main training involving the specific tasks outlined in Table 1, and a 5-minute cool-down. The program emphasized sensorimotor coordination, cognitive processing, and perceptual-motor integration. To ensure a high level of neuroplastic stimulus, exercises were performed in 3 sets of 5–6 minutes with 1-minute rest intervals. The training followed the principle of "novelty over automation," where tasks were modified as soon as a participant achieved approximately 60% proficiency to prevent neural adaptation.

The intensity of the sessions was monitored using the Borg Rating of Perceived Exertion (RPE) scale, targeting a range of 5–7 (moderate to hard) to ensure sufficient cognitive demand without inducing excessive physical exhaustion that could impair learning.

Exercises were progressively intensified each week (Table 1) by increasing movement complexity, adding dual-task demands, and incorporating visual restrictions. Each session concluded with a standardized cool-down phase, including 10 minutes of jogging and 5 minutes of stretching.

Table 1
Weekly exercise protocol.

Week	Exercise Protocol
1	*Coordination and Basic Dual Tasks* • Throw and catch a ball with right/left hand • Single-leg balance – eyes closed for 10 seconds • Count backwards by 3s from 30 while dribbling a ball
2	*Opposite Movements and Memory Load* • Right hand up – left knee up • Perform the opposite direction of the coach's command • Bounce the ball off the wall – touch the object matching the color when a color is called out
3	*Rhythm, Balance, and Increased Cognitive Load* • Dribble with both hands at different speeds • While dribbling with the right hand, toss and catch a tennis ball with the left (and vice versa) • While dribbling with the right hand, toss and catch a tennis ball with the left (and vice versa)
4	*Advanced Dual Tasks* • Stop the coach when they make a mistake in a predetermined movement sequence • Dribble with one hand while drawing shapes with the other • While dribbling with the right hand, draw circles with a large ribbon using the left hand (and vice versa)
5	*Complex Tasks and Reaction Timing* • Perform paired movements based on different colored (catch ball) throws while dominant/non-dominant eye is covered • Solve a math problem (e.g., 2+2) with every jump • Opposite arm-leg movement
6	*Reaction + Decision-Making Speed* • Move towards the color opposite to the one that is called out • Stop dribbling when the coach says a multiple of 3 • Respond to sudden direction commands with appropriate movement
7	*Fast Thinking and Complex Combinations* • Perform synchronized movements with a partner • Practice shooting in basketball with dominant/non-dominant eye covered • Find objects based on given movement commands while blindfolded
8	*Maximum Confusion* • Throw Life Kinetic training balls straight and catch them crosswise • Catch a target ball using Life Kinetic training balls • Perform opposite movements on a balance board using a tennis ball

Anthropometric Measurements

Participants' age was recorded from national identity documentation. Body height was measured barefoot using a stadiometer with ±1 mm precision (Seca, Germany), and body mass was recorded using a

calibrated digital scale with ±0.1 kg sensitivity. Body Mass Index (BMI) was calculated using the formula: BMI = weight (kg) / height² (m²).

Blood Sampling and Biochemical Analysis

To minimize the influence of circadian rhythm and acute metabolic changes, peripheral venous blood samples were collected from all participants between 08:30 and 10:00 AM following an overnight fast of at least 10 hours. Samples were obtained from both the the experimental and control groups before and after the 8-week LK training program. Furthermore, participants were instructed to avoid any strenuous physical activity for 48 hours prior to blood collection to ensure that the measured neurotrophic factors reflected chronic adaptations rather than acute exercise effects. Samples were obtained by certified phlebotomists and handled under standardized conditions to prevent hemolysis. Serum samples were processed in 5 mL gel-separator tubes, allowed to clot for 10–20 minutes at room tempereture, and centrifuged at 3000 rpm for 20 minutes. The resultant serum was aliquoted into sterile tubes and stored at –80°C until biochemical analysis.

Serum concentrations of Brain-Derived Neurotrophic Factor (BDNF), Nerve Growth Factor (NGF), Neurotrophin-3 (NT-3), and Neurotrophin-4 (NT-4) were quantified using high- sensitivity enzyme-linked immunosorbent assay (ELISA) kits, following the manufacturers' instructions. The specific kit details and analytical sensitivities were as follows:

- BDNF was measured using the Human BDNF ELISA Kit (BT LAB, Cat. No: E1302Hu). Sensitivity: 0.023 ng/mL; intra-assay CV < 8%; inter-assay CV < 10%.
- NGF levels were determined using the Human NGF ELISA Kit (BT LAB, Cat. No: E2102Hu). Sensitivity: 3.48 pg/mL; intra-assay CV < 8%; inter-assay CV < 10%.
- NT-3 was measured using the Human NT-3 ELISA Kit (BT LAB, Cat. No: E1305Hu). Sensitivity: 15.12 ng/L; intra-assay CV < 8%; inter-assay CV < 10%.
- NT-4 was analyzed using the Human NT-4 ELISA Kit (USCN, Cat. No: SEA107Hu). Sensitivity: 6 pg/mL; intra-assay CV < 10%; inter-assay CV < 12%.

Absorbance readings were performed at 450 nm using a BioTek ELx800 Universal Microplate Reader, and all wash steps were conducted using the ELx50 Washer. Concentrations were calculated using a four-parameter logistic (4-PL) standard curve, and results were expressed in ng/mL or pg/mL depending on the target analyte.

Data Analyses

All statistical analyses were conducted using IBM SPSS Statistics version 25.0 (IBM Corp., Armonk, NY, USA). Normality of distribution was assessed using the Shapiro–Wilk test. Descriptive statistics were used to summarize participant characteristics (age, height, weight, and BMI). The normality of distribution was assessed using the Shapiro–Wilk test, and the homogeneity of variances was verified via Levene's test. Since the data met the assumptions for parametric testing, the Paired Sample *t*-test was employed to compare pre- and post-test values within each group. Between-group comparisons were analyzed using the Independent Samples *t*-test. Statistical significance was set at $p < 0.05$, with a 95% confidence interval reported for mean differences. Furthermore, effect sizes (Cohen's *d*) were calculated and interpreted as small (0.20), medium (0.50), or large (0.80) to support the practical significance of the findings.

Results

The demographic characteristics of the participants are presented in Table 2. Intra - group analysis revealed no statistically significant differences in any neurotrophic factors within the control group ($p > 0.05$). In contrast, a statistically significant increase was observed in BDNF ($p = .002$) and NGF ($p = .006$) levels within the experimental group. The effect sizes for these increases were calculated as d = 1.22 and d = 1.08, respectively. No significant differences were detected for NT-3 ($p = .891$, d = 0.05) and NT-4 ($p = .119$, d = 0.59) in the experimental group (Table 3).

Table 2
Demographic information of the participants.

	EG (n= 15)	CG (n= 15)	Minimum	Maximum
Age (years)	16.80 ± 1.14	16.46 ± 1.06	15	18
Height (m)	1.81 ± 0.07	1.76 ± 0.05	1.68	1.96
Body Weight (kg)	70.01 ± 11.29	67.84 ± 8.59	54.75	90
BMI (kg/m^2)	21.16 ± 2.31	21.82 ± 2.23	17.28	25.39

BMI: Body mass index; EG: Experimental group; CG: Control group.

Table 3
Statistical comparison of neurotrophic factor pre-test and post-test score.

Parameter	Group	Pre-Test (Mean ± SD)	Post-Test (Mean ± SD)	t	p	Cohen's d
BDNF (ng/mL)	EG	2.61 ± 1.68	4.48 ± 1.36	3.333	.002*	1.22
	CG	2.04 ± 1.24	2.03 ± 1.34	0.143	.887	-0.01
NGF (pg/mL)	EG	278.64 ± 150.52	440.19 ± 149.56	2.949	.006*	1.08
	CG	253.52 ± 246.95	250.36 ± 99.309	0.023	.981	-0.02
NT – 3 (ng/L)	EG	1959.81 ± 1318.41	2030.80 ± 1485.77	0.138	.891	0.05
	CG	1666.64 ± 1040.39	1610.05 ± 538.32	0.187	.853	-0.07
NT – 4 (pg/mL)	EG	27.09 ± 15.47	36.78 ± 17.43	1.610	.119	0.59
	CG	26.99 ± 18.67	25.65 ± 16.19	0.209	.835	-0.08

BMI: EG: Experimental Group; CG: Control Group; SD: Standard Deviation; BDNF: Brain-Derived Neurotrophic Factor; NGF: Nerve Growth Factor; NT-3: Neurotrophin-3; NT-4: Neurotrophin-4.

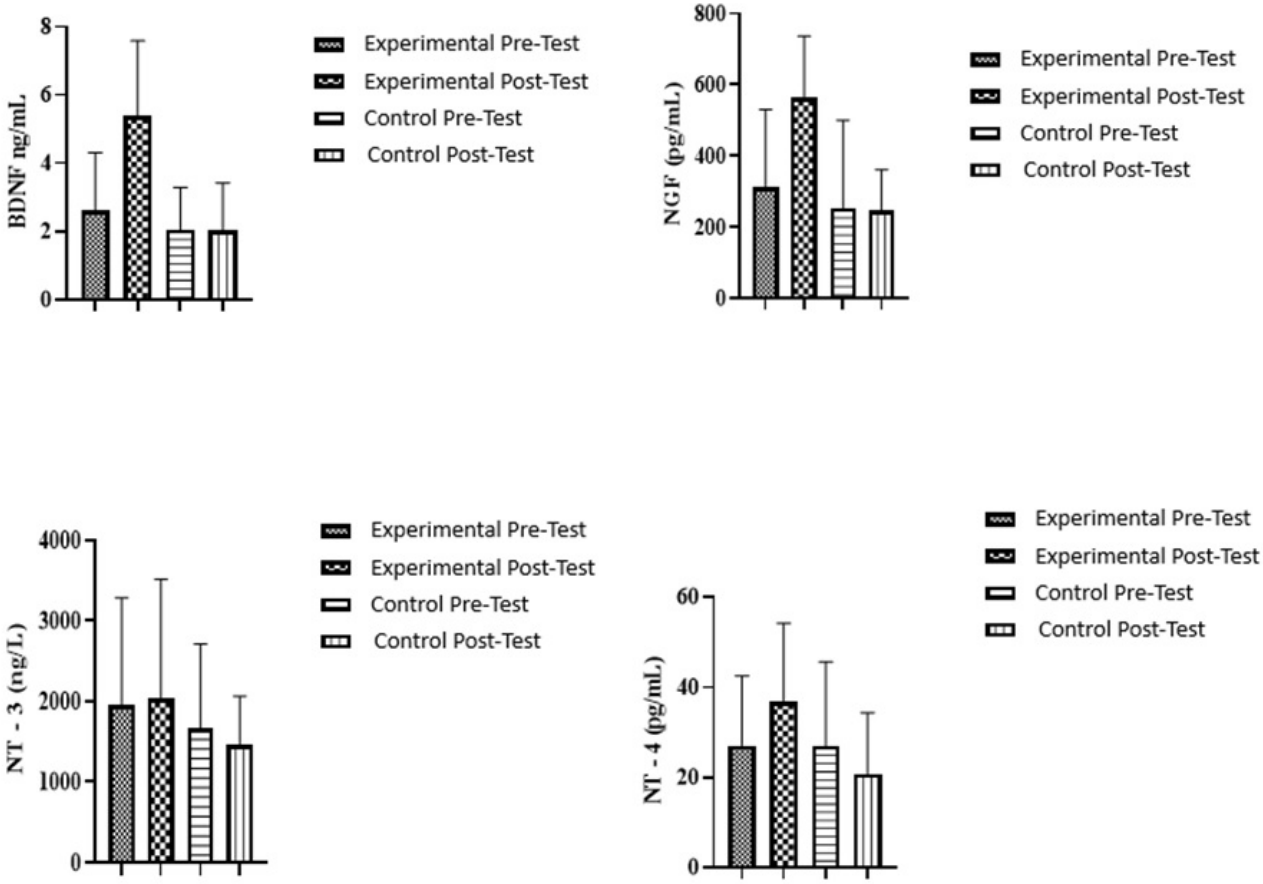

Figure 1. Graphical Representation of Neurotrophic Factor Pre-Test and Post-Test Scores.

According to the Independent Samples t-test analysis of neurotrophic factor levels, a statistically significant difference was found between the experimental and control groups in the post-test values of BDNF and NGF ($p < 0.05$).

Discussion

The primary aim of this study was to examine the effects of an eight-week LK training program on selected neurotrophic factors (BDNF, NGF, NT-3, and NT-4) in adolescent basketball players aged 15 to 18. Using a controlled pretest-posttest design, significant findings were observed, particularly in BDNF and NGF levels, in favor of the experimental group, while NT-3 and NT-4 showed positive trends despite lacking statistical significance. While these results provide encouraging evidence, they should be interpreted with caution considering the specific sample size and the eight-week intervention period.

Table 4
Comparison of neurotrophic factor values between the experimental and control groups.

Parameter	Groups	Pre-Test			Post-Test		
		Mean ± SD	t	p	Mean ± SD	t	p
BDNF (ng/mL)	EG	2.61 ± 1.68	1.054	0.301	4.48 ± 1.36	4.790	<.001**
	CG	2.04 ± 1.24			2.03 ± 1.34		
NGF (pg/mL)	EG	278.64 ± 150.52	0.336	0.739	440.19 ± 149.56	3.860	<.001*
	CG	253.52 ± 246.95			250.36 ± 99.39		
NT – 3 (ng/L)	EG	1959.81 ± 1318.41	0.676	0.504	2030.80 ± 1485.77	1.031	0.311
	CG	1666.64 ± 1040.39			1610.05 ± 538.32		
NT – 4 (pg/mL)	EG	27.09 ± 15.47	0.016	0.987	36.78 ± 17.43	1.812	0.080
	CG	26.99 ± 18.67			25.65 ± 16.19		

EG: Experimental Group; CG: Control Group; SD: Standard Deviation; BDNF: Brain-Derived Neurotrophic Factor; NGF: Nerve Growth Factor; NT-3: Neurotrophin-3; NT-4: Neurotrophin-4.

One of the most notable outcomes of this study was the significant increase in Brain-Derived Neurotrophic Factor (BDNF) levels. The experimental group demonstrated a marked rise in BDNF levels from 2.61 ± 1.68 ng/mL to 4.48 ± 1.36 ng/mL, whereas no significant change was observed in the control group. This discrepancy between groups suggests that the observed elevation is not merely a byproduct of regular basketball training, but likely a response to the specific motor-cognitive stimulus of LK**.** These results support the idea that exercise, particularly motor-cognitive integrated training such as LK, can promote neuroplasticity through upregulation of neurotrophic factors. Previous studies by Cotman and Engesser (2002) and Vega et al. (2003) have also highlighted the role of physical activity in preserving neuronal integrity and enhancing brain function. Considering the cognitively demanding nature of basketball, the ability of LK to enhance attention, reaction time, and motor planning may partly explain its effect on BDNF modulation.

Existing literature largely supports our findings. For instance, Schor et al. (2019) observed significant post-exercise increases in BDNF following a maximal ramp test in judo athletes. Correa et al. (2016) reported higher BDNF concentrations in international-level sprinters compared to national-level and sedentary controls. Similarly, Zoladz et al. (2008) demonstrated significant increases in plasma BDNF following a five-week endurance training intervention. However, not all studies are consistent; William and Ferris (2012) reported no significant changes in BDNF after aerobic training, while Kim (2016) observed lower baseline BDNF levels in long-term table tennis athletes compared to sedentary controls. These inconsistencies suggest that BDNF responses may vary based on training modality, intensity, duration, and participant characteristics. From an analytical perspective, the efficacy of LK training in our study likely stems from the "non-automation" principle, which requires continuous neural adaptation and high cognitive load. Unlike repetitive aerobic or highly practiced motor tasks where neural efficiency may eventually lead to a plateau in neurotrophic release, the novelty and complexity inherent in LK may prevent this adaptation, thereby sustaining the stimulus for BDNF secretion. In our study, the observed increase in BDNF aligns with the hypothesis that complex motor-cognitive challenges inherent to LK training stimulate synaptogenesis and plasticity (Lutz, 2014).

Similarly, a significant increase in Nerve Growth Factor (NGF) levels was found in the experimental group (from 278.64 ± 150.52 pg/mL to 440.19 ± 149.56 pg/mL), whereas no meaningful changes were observed in the control group. These findings are in line with previous reports by Pan et al. (2017) and Hatem et al. (2016), which emphasize the neuroprotective effects of exercise in mitigating memory loss, neurological disorders, and motor impairments. Supporting evidence also comes from Sari and Vakili (2022) and Bamaç et al. (2011), who observed similar outcomes in athletic populations. While these findings indicate a strong association between LK training and NGF elevation, it is essential to distinguish this correlation from direct causality. The observed increase suggests that the unique sensory-motor integration required by LK may trigger pathways involved in neuronal survival and synaptic remodeling. NGF is known to play a vital role in neuronal survival and synaptic remodeling, indicating that the LK program may serve as a promising non-pharmacological strategy to promote neurocognitive resilience.

Although no statistically significant difference was found in Neurotrophin-3 (NT-3) levels, the experimental group showed a 3.62% increase, whereas the control group experienced a 3.4% decrease. While

these directional trends might suggest a subtle biological response, they should be interpreted with extreme caution due to the lack of statistical significance. Dominguez Sanchez et al. (2018) and Chang et al. (2019) reported that resistance and swimming training could enhance NT-3 levels, although studies involving human participants remain scarce. The absence of a significant effect in our study may be attributed to the relatively small sample size or the 8-week duration, which might have been insufficient to elicit systemic changes in NT-3 levels compared to BDNF or NGF. Furthermore, considering that NT-3 is involved in spinal cord and muscle regeneration (Li et al., 2012), and is typically modulated by high-intensity or long-term aerobic stimuli (Sharma et al., 2010), it is possible that the coordinative and cognitive focus of LK training does not target the specific physiological pathways required for robust NT-3 upregulation. Consequently, further research with larger cohorts and longer intervention periods is required to clarify the responsiveness of NT-3 to integrated cognitive-motor models.

Similarly, no statistically significant difference was observed in Neurotrophin-4 (NT-4) levels; however, the experimental group exhibited a 36.78% increase in mean NT-4 levels, while the control group showed a 4.96% decrease. Despite the substantial numerical increase in the experimental group, the lack of statistical significance suggests that these results should be considered preliminary and interpreted with caution, potentially reflecting high individual variability within the sample. These findings echo results from Dominguez Sanchez et al. (2018) and Maejima et al. (2018), who demonstrated NT-4 increases following exercise interventions in rodent models.

From an analytical perspective, the disparity between the large percentage increase and the lack of statistical significance may be due to the small sample size (n=15 per group) or the inherent fluctuations in systemic neurotrophin levels. While NT-4 plays a critical role in synaptic transmission and neurogenesis (Bonanni et al., 2022), its specific regulatory pathways in response to cognitive-motor training like LK are not yet fully established. Therefore, these findings represent a correlational trend rather than a definitive causal effect, highlighting the need for future studies with larger cohorts to determine whether such trends reach statistical power over longer durations.

In conclusion, the LK training program administered over an 8-week period elicited significant enhancements in key neurotrophic markers, particularly BDNF and NGF,in adolescent basketball players. These findings suggest that integrated motor-cognitive training may serve as a potent stimulus for neurotrophic upregulation, potentially enhancing neural adaptability in cognitively demanding athletic environments. While NT-3 and NT-4 did not exhibit statistically significant changes, the positive directional trends observed in the experimental group warrant further investigation to determine if longer intervention periods or larger sample sizes would yield more robust effects.

It is important to emphasize that while the current results are promising, they represent an initial exploration within a specific demographic. Therefore, caution should be exercised when generalizing these findings to other populations or clinical contexts. Furthermore, while the role of neurotrophic factors in neuroprotection is well-documented in literature, the extent to which LK training can serve as a preventative intervention for neurodegenerative disorders remains a hypothesis that requires rigorous longitudinal and clinical validation. Future studies involving diverse age groups, extended training durations, and various sporting contexts are essential to fully elucidate the underlying mechanisms and confirm the generalizability of this neurobiological model.

Conclusion

This study provides experimental evidence that an 8-week LK training program positively influences specific neurobiological markers in adolescent basketball players. Significant increases in BDNF and NGF concentrations were observed in the experimental group, suggesting that coordinative-cognitive training protocols such as LK can enhance neuroplasticity and promote neurotrophic support mechanisms within this specific demographic. Although NT-3 and NT-4 levels did not exhibit statistically significant changes, these positive trends suggest that the 8-week timeframe may represent an early stage of neurobiological response, indicating that extended intervention protocols are required to fully capture the potential impact of LK on these specific neurotrophins.

Given the complex demands of basketball, which integrates rapid motor execution with real-time decision-making, LK training appears to support the biochemical dimensions of athletic performance. The original contribution of this research lies in demonstrating that the integration of non-automated coordination and cognitive load can trigger neurotrophic responses even in high-performance athletes who are already subject to regular physical

stimuli. While the findings underscore the potential utility of such training, conclusions regarding its role in preventing neurodegenerative conditions remain hypothetical and require rigorous clinical validation before they can be generalized beyond athletic populations. From a practical perspective, coaches and sports scientists may consider LK as a non-invasive, cognitively-driven adjunctive tool to enhance neural adaptability without increasing physical fatigue.

Limitations and Directions for Future Research

While the findings of this study provide valuable insights, several limitations must be acknowledged to contextualize the results. First, the sample consisted exclusively of male basketball players aged 15 to 18, limiting the generalizability of the results to other age groups, female athletes, and individuals involved in different sports disciplines. Second, neurotrophic factor levels were measured at only two time points. This design does not allow for a detailed understanding of the time-course dynamics; therefore, the results should be viewed as a snapshot of an 8-week adaptation rather than a definitive long-term trend. Third, although LK training is inherently based on motor-cognitive integration, this study did not assess any cognitive performance parameters such as reaction time, attention, memory, or executive functions. Empirically testing the correlation between these biochemical increases and functional cognitive gains remains a critical next step. Lastly, the molecular mechanisms underlying the observed changes were not examined.

To enhance the scientific depth of future research, it is specifically recommended to:

- Utilize randomized cross-over designs with larger cohorts to minimize individual biological variability.
- Incorporate a "dose-response" analysis by comparing various weekly frequencies of LK training to identify the optimal threshold for neurotrophic upregulation.
- Integrate neuroimaging techniques (e.g., fMRI or EEG) alongside biochemical analyses to map the systemic changes in neurotrophins to specific brain regions involved in motor-cognitive tasks.
- Extend the follow-up period to 3–6 months to assess the "washout effect" and determine how long these neurobiological adaptations persist after training cessation.

Aknowledgement

This study is summarised from Umut Yılmaz's doctoral thesis (Thesis no: 761077).

Authors' Contribution

Study Design: UY, MG, AK, HP; Data Collection: UY; Statistical Analysis: UY; Manuscript Preparation: UY.

Ethical Approval

The study was approved by Ataturk University Faculty of Sports Sciences(Ethics approval no.: E-70400699-000-2200099727) and it was carried out in accordance with the Code of Ethics of the World Medical Association also known as a declaration of Helsinki.

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Conflict of Interest

The authors hereby declare that there was no conflict of interest in conducting this research.

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