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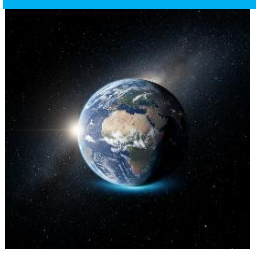

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Exercise-induced neuroplasticity: role of BDNF in cognitive function across healthy and clinical populations — a narrative review

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Abstract

Physical exercise has increasingly been recognized as an important modulator of neuroplasticity and cognitive functioning across the lifespan. Among proposed biological mediators, brain-derived neurotrophic factor (BDNF) has emerged as a key molecule linking physical activity with adaptive neural changes. This narrative review summarizes current evidence regarding exercise-induced BDNF signaling and its association with cognitive outcomes in both healthy individuals and clinical populations.

Available findings indicate that physical activity promotes neuroplastic processes including neurogenesis, synaptic remodeling, and enhanced neuronal survival, particularly within hippocampal networks involved in learning and memory. In healthy populations, exercise primarily supports cognitive maintenance, executive function enhancement, and age-related cognitive resilience. In contrast, in clinical populations—including individuals with Alzheimer's disease, Parkinson's disease,

depression, mild cognitive impairment, and stroke—exercise appears to contribute to cognitive recovery and functional adaptation through neurotrophic mechanisms.

Accumulating evidence suggests that exercise intensity represents an important factor influencing BDNF response; however, no single training modality has been consistently identified as superior across conditions. Considerable heterogeneity in study design, patient characteristics, and measurement methods limits direct comparison between studies.

Overall, BDNF-mediated neuroplasticity may represent a shared biological pathway underlying exercise-related cognitive benefits, although its relationship with functional outcomes remains complex. Future research should focus on individualized exercise prescriptions and improved translational understanding of neuroplastic mechanisms supporting cognitive health and rehabilitation.

Keywords

BDNF, neuroplasticity, physical exercise, cognition, neurological disorders, aerobic exercise

1. Introduction

In recent years, increasing attention has been directed toward physical exercise as a therapeutic strategy aimed at enhancing neuroplasticity and improving cognitive outcomes in both healthy and clinical populations [1]. Reported cognitive outcomes include memory, learning and executive functions modulation. A growing number of studies have suggested possible connection between exercise and cognition through shared biological mediator – Brain-derived Neurotrophic Factor (BDNF) [2,3]. Many of them also focused on finding the most beneficial training strategies in improving clinical outcomes for healthy individuals as well as patient groups. Different types of physical activity and their intensities

have been suggested with no clear consensus regarding optimal protocols [4,5]. The narrative review aims to summarize recent knowledge and discuss possible directions for future research.
Unlike previous condition-specific reviews, the present narrative review integrates evidence across healthy aging and multiple neurological and neuropsychiatric conditions to identify shared BDNF-mediated mechanisms underlying exercise-induced cognitive adaptation.

2. Literature search strategy

The literature for this narrative review was identified through searches conducted in PubMed and Google Scholar databases using combinations of keywords such as "exercise," "brain-derived neurotrophic factor," "neuroplasticity," "cognition," "Alzheimer's disease," "Parkinson's disease," "depression," and "stroke." Priority was given to recent systematic reviews, meta-analyses, and clinical trials investigating exercise-induced changes in BDNF and cognitive outcomes in both healthy individuals and clinical populations. Relevant background articles addressing neurobiological mechanisms were also included to provide conceptual context. Due to the narrative nature of this review, study selection was based on thematic relevance rather than strict methodological criteria.

3. Neurobiological Mechanisms and Cognitive Effects of Exercise-Induced Plasticity

Brain-derived Neurotrophic Factor (BDNF) plays a major role in neuroplasticity. It influences not only neurogenesis, but also gliogenesis and synaptogenesis, and is known to enhance synaptic signaling and neuronal survival. Moreover, it participates in the regulation of apoptosis and the elimination of improperly formed neural connections [2]. Particularly high concentrations of BDNF are observed in the hippocampus, a brain structure strongly associated with memory formation and learning processes. Through modulation of synaptic efficiency and structural plasticity, BDNF contributes to adaptive changes in neural networks underlying cognitive functioning [6].

Ageing and various health conditions have been linked to reduced BDNF expression, which may negatively affect neuronal resilience and plasticity [2,7]. Understanding these biological mechanisms provides a framework for interpreting how physical activity may influence cognitive outcomes through exercise-induced neuroplastic adaptations described in subsequent sections.

4. Evidence from Healthy Populations

Exercise-related cognitive improvements can be observed in healthy populations even after a few sessions of exercise. A follow-up increase in BDNF positively impacts neurotransmission through enhancing neuronal survival and neurogenesis. All of these mechanisms contribute to neuroplasticity

which enables the brain to adapt to environmental demands. This phenomenon is present in different age groups and either alters cognition or prevents its loss. An improvement in activities dependent on memory and learning have also been documented. They include both memory formation and its consolidation. Efficient neurotransmission is crucial in keeping cortical excitability. This outlines why engaging in physical activity can largely improve brain function responsible for cognition [1].

Given the role of BDNF in exercise-induced plasticity, it raises a question about what kind of exercise is optimal for meaningful increase in BDNF levels. While every exercise generates an increase in BDNF levels, some have shown better results than others. Recent reviews state that aerobic training is the one to focus on in the first place [3,8]. In addition to this, intensity, duration and frequency of the exercise need to be taken into consideration while analyzing BDNF levels. In order to observe BDNF response, exercise must be above aerobic threshold. This observation was one of the supporting points that indicated intensity of the exercise as one of the most important factors in BDNF release. This mostly applied to high intensity exercise which, in most cases, caused larger increase in BDNF [3]. The other review also stated that intensity changed BDNF levels and that high intensity aerobic exercise had more beneficial effect on it. However, it also indicated that high intensity interval training could have even more beneficial effect on BDNF levels and neuroplasticity [8]. For instance, a clinical trial that studied exercise intensity on BDNF focused on moderate-intensity continuous training (MICT), vigorous-intensity continuous training (VICT) and sprint interval training (SIT). The results suggested that SIT caused higher increase in BDNF in plasma that MICT and VICT. Although, VICT was more effective than MICT, which proves a point of intensity-dependent outcome. [9] Another trial also probed into the matter of intensity in training in healthy adults. It demonstrated that high intensity interval exercise stimulated BDNF secretion more effectively in comparison to moderate intensity continuous exercise. Moreover, it acted stronger in enhancing synaptic plasticity in the cerebral cortex [10]. Taken together, current evidence suggests an intensity-dependent pattern of exercise-induced BDNF response, with higher intensity protocols appearing to elicit more pronounced neuroplastic adaptations in healthy individuals.

Exercise-induced brain health is also a case in adolescents and not only adults. Higher BDNF levels promote cognitive functioning which might result in better educational performance due to enhancement in memory and learning. Improving neuronal signaling and BDNF release from early years might result in maintaining stronger cognition until later age. As presented in a recent systematic review and meta-analysis on exercise-induced BDNF in adolescents, the study confirmed that both moderate and high intensity exercise seems to increase BDNF levels in adolescent population [11].

Aging is a natural process associated with gradual changes in brain function, particularly within cognitive and executive domains. With advancing age, neuroplasticity and neuromodulatory capacity may become less efficient, which contributes to progressive cognitive decline. Reduced levels of BDNF

are often observed alongside other age-related alterations, suggesting a potential role of neurotrophic support in maintaining neuronal resilience. Therefore, strategies aimed at enhancing BDNF availability may be relevant for preserving cognitive performance in older adults [12].

Physical activity has been proposed as one of the non-pharmacological approaches capable of modulating these processes. Positive effects of exercise on cognition in older populations have been described in a recent systematic review, where activity-induced increases in BDNF were associated with improvements in memory and learning performance, accompanied by structural changes in the hippocampus [7]. Furthermore, a recent randomized controlled trial investigating high-intensity interval training in healthy older individuals demonstrated improvements in hippocampal-dependent learning, highlighting the relevance of exercise intensity and the central role of hippocampal plasticity in ageing-related cognitive outcomes [12].

Altogether, findings from healthy adults, adolescents and ageing populations indicate that physical activity may contribute to maintaining cognitive resilience across the lifespan, largely through mechanisms related to BDNF-mediated neuroplasticity.

5. Evidence from Clinical Populations

Neurological disorders are commonly characterized by dysregulation of neuronal pathways associated with processes such as inflammation, protein misfolding, and changes in cortical metabolism. Many of these mechanisms converge on impaired neuroplasticity, which is considered a shared feature across various neurological and neuropsychiatric conditions. Exercise has been proposed as a potential non-pharmacological intervention capable of modulating these mechanisms and supporting adaptive neural responses. Through its influence on neurotrophic signaling, particularly BDNF-dependent pathways, physical activity may contribute to restoring functional balance within disrupted neural systems [13].

Based on a recent systematic review with meta-analysis, physical exercise is associated with an increase in circulating BDNF in different types of neurological disorders, suggesting a potential role in neuroprotection of the hippocampus and preserving cognitive function in people affected by neurological conditions [4]. Consistently, another systematic review also indicated that adults with neurocognitive disorders benefited from exercise training. Physical activity may reduce risk of neurodegeneration and strengthen memory and executive functioning. For that reason, exercise training has been suggested as a part of rehabilitation strategies in neurological patients. However, the effect of physical activity varies among different conditions and patients and needs to be studied individually with consideration of disease characteristics and individual factors [14].

One of the clinical areas where exercise-related neuroplasticity has been most widely investigated is Alzheimer's disease (AD) and conditions associated with mild cognitive impairment (MCI).

One of the mechanisms responsible for memory loss and cognitive decline in AD may involve reduced BDNF levels in specific regions of the brain, particularly the hippocampus and amygdala. BDNF is also

regulated by other proteins present in AD causing the negative symptoms of the disease. Due to complexity of underlying mechanisms and relations between them and the molecules, non-pharmacological ways of treating the disorder might be the main focus right now. Different studies suggest that exercise may be beneficial in non-progression of cognitive dysfunction. Despite variability across studies, a large number of them showed increase in BDNF levels, enhanced neural function in hippocampus and amygdala. Moreover, exercise-induced increase in vascularity of the brain, improvement in mitochondrial function, immunomodulation and anti-inflammatory function may also act in favor of AD patients. Aerobic exercise, in particular, has been highlighted as potentially beneficial in AD because of its possible best effect on cognitive impairment, especially memory [15,16]. Considering that Alzheimer's disease develops gradually, similar exercise-related mechanisms have also been investigated in earlier stages of cognitive decline, particularly in individuals with mild cognitive impairment. The results of systematic review and meta-analysis reported potential association between exercise and reducing the risk of progression from MCI to AD or other dementias. This highlights the potential importance of early intervention in such diseases [17]. For instance, a recent clinical study demonstrated that active lifestyle may contribute to preventing or delaying onset of different neurological diseases by enhancing molecular pathways such as BDNF-dependent pathway and improved cognitive outcomes [18].

Together with Alzheimer's Disease, Parkinson's Disease (PD) is one of the most prevalent neurodegenerative disorders in the world. It affects not only motor function but also many non-motor features including cognitive impairment, particularly associated with learning and memory. Although the mechanisms in AD and Parkinson's disease may differ from each other, they share one common phenomenon which is decline in BDNF levels. This may be one of the components responsible for some of the symptoms in Parkinson's Disease. Despite existing pharmacological treatment in this condition, it doesn't fully meet therapeutic expectations and has many unfavorable side effects. Given these limitations, increasing attention has been directed toward non-pharmacological strategies, particularly physical exercise, as a potential modulator of neuroplasticity and BDNF signaling in Parkinson's disease. Despite certain methodological limitations, exercise has been associated with increased BDNF levels in PD. What remains unclear is the association of BDNF with motor and non-motor improvements in patients. As indicated in a recent systematic review and meta-analysis, exercise may contribute to improvements in overcoming motor deficits in PD patients. There has also been evidence that physical activity may induce neuronal changes in dopamine-related regions of the brain. As dopamine pathways are mostly disrupted in PD, this may have a significant impact on the course of the disease [19].

As research on exercise in Parkinson's disease expands, increasing attention has been directed toward understanding how specific training characteristics influence clinical outcomes. Currently, no single exercise modality has been identified as clearly superior in PD management. Nevertheless, various

forms of training appear to be beneficial for patients, with some evidence suggesting greater benefits associated with higher-intensity training [20]. Moreover, evidence from long-term cohort studies suggests that sustained physical activity may contribute to slower progression of motor impairment and preservation of functional abilities in early Parkinson's disease. In addition, different forms of physical activity appeared to influence distinct symptom domains, indicating that exercise-related benefits may extend beyond general neuroplastic effects [21].

Apart from neurodegenerative disorders, the therapeutic role of exercise has also been explored in neuropsychiatric disorders, particularly depression. As one of the most prevalent mental health disorders worldwide, it represents a major global health concern affecting not only mood but also memory, attention, perception and learning processes. While pharmacotherapy remains a primary treatment approach for depression, growing evidence suggests that physical exercise may provide comparable benefits in certain populations. Exercise has also shown potential in enhancing pharmacological or psychotherapeutic interventions. Despite growing evidence, the role of physical activity is undervalued. Moreover, various forms of exercise appear to be associated with improvements in both mood and cognitive functioning [22].

Among the proposed mechanisms underlying depression, a decline in BDNF levels has been proposed as one of the components. Emerging evidence suggests that disrupted neuroplasticity and reduced BDNF signaling may contribute to depressive symptoms, while both acute and long-term physical exercise appear to promote neurotrophic responses associated with improvements in mood and cognitive functioning [23,24]. In light of these neuroplastic mechanisms, recent meta-analytical research has attempted to determine which exercise modalities and training doses may produce the most pronounced neurotrophic and clinical effects in individuals with depression. Findings indicated that combined training protocols, particularly those integrating aerobic and resistance exercise, were associated with the most consistent increases in BDNF levels. The analysis suggested a nonlinear relationship between training volume and neurotrophic response, indicating that moderate-to-high exercise doses may provide greater benefits compared with lower-intensity approaches. Interestingly, while several forms of physical activity appeared beneficial, no single modality was universally superior, highlighting the importance of individualized exercise prescription [5].

Beyond chronic neurodegenerative and neuropsychiatric conditions, exercise-induced neuroplasticity has also been investigated in acute neurological injuries such as stroke, where recovery of cognitive and functional abilities remains a major therapeutic challenge. Stroke represents a major global health burden resulting in motor, executive and cognitive impairments that often demand intensive rehabilitation and lead to dependency on others. Similarly to other diseases described in this review, post-stroke BDNF levels appear to be reduced compared to healthy adults. Lower BDNF concentrations may be associated with negative post-stroke effects on different functions, especially cognition. Recent

systematic review and meta-analysis on this topic suggested possible role of measuring BDNF levels during the rehabiliation process in order to associate them with potential improvement. Furthermore, it may also show potential as a prognostic factor in acute stroke [25]. The importance of preserving cognition in post-stroke population has been highlighted concerning how largely it could improve life quality. Strategies aimed at maintaining executive function, attention, memory, and learning ability are widely discussed in the rehabilitation literature. Aerobic exercise is commonly researched as a rehabilitation strategy. Its potential benefits include enhanced cerebral perfusion, BDNF-dependent neuroplasticity and reducing neuroinflammation. Given that neuroplasticity is probably a determining factor in regaining cognition and other features after a stroke, aerobic exercise might be a crucial part of post-stroke recovery [26]. However, recent systematic review suggested that high intensity interval training might be particularly beneficial in improving neuroplasticity after stroke [27]. Similarly, a randomized clinical trial indicated moderate-intensity continuous training to be specifically effective in the regenerative-compensatory period of stroke rehabilitation [28]. Collectively, further clarification of individualized exercise strategies remains an important research direction. Nevertheless, exercise as treatment and recovery strategy has considerable therapeutic potential and remains an important area for future investigation.

Overall, findings across neurodegenerative, neuropsychiatric, and post-injury conditions suggest that exercise-induced neuroplasticity may represent a shared mechanism supporting cognitive recovery, although variability in study design and clinical outcomes highlights the need for further translational research.

6. Discussion

This narrative review aimed to demonstrate a common pattern in exercise-induced BDNF signaling in both healthy and clinical populations with particular focus on different neurological disorders. The findings suggest beneficial effects of exercise on cognition alongside other brain functions. Exercise intensity is suggested to be one of the factors modulating the impact of physical activity on BDNF circulation and, consequently, cognitive functions [5,10]. Nevertheless, several methodological limitations should be acknowledged due to inconsistent study results regarding the most effective exercise modalities and training parameters [4,5,19].

Exercise induced neuroplasticity appears to influence cognitive outcomes among both healthy and clinical populations. However, the functional implications may differ between specific groups. In healthy individuals, exercise-related effects were mainly observed in executive functions improvement including working memory, cognitive flexibility, organization, focusing and emotional regulation [1].

In this group, physical activity primarily serves prevention and adaptability rather than rehabilitation and regaining functionality as in clinical populations. Exercise-induced neurotrophic factors such as BDNF could also be used in monitoring and evaluating the effects of non-pharmacological therapies. The variability across patient groups suggests that while BDNF signaling may represent a common biological pathway, its relationship with functional improvement is not always linear [4,5,19]. These differences highlight the importance of individualized exercise protocols regarding disease context and baseline cognitive status.

The growing body of evidence presented across this narrative review indicates that BDNF may represent a shared biological link between exercise and neuroplasticity in not only healthy but also clinical populations. Several studies discussed BDNF as a possible marker of rehabilitation response and prognosis assesment regarding cognition, yet needs to be interpreted carefully [4,25]. Due to substantial variability in applied training protocols, different patients' characteristics and changeability in measurement methods acoss different, consistent conclusions cannot always be drawn [4,5,19]. This may prove that neuroplasticity is based on complex mechanisms and is unlikely to rely on a single neurological factor. Consequently, further research is needed in order to clarify an optimal exercise strategies aimed at improving cognitive function through neuroplastic adaptation.

7. Conclusions

This narrative review highlights exercise-induced neuroplasticity as a shared mechanism supporting cognitive function across both healthy and clinical populations, with BDNF emerging as a potential biological mediator of these effects. While physical activity appears to enhance executive functions, memory, and adaptive brain processes, the magnitude of benefits varies depending on disease context, training parameters, and baseline cognitive status. Current evidence does not support a single optimal exercise modality, emphasizing the importance of individualized intervention strategies. Future research should focus on refining exercise prescriptions and improving translational understanding of neuroplastic mechanisms to optimize cognitive outcomes.

Author Contribution

Conceptualization, Magdalena Majkowska; methodology, Magdalena Majkowska, Maria Fengier, Oliwia Bolek, Piotr Szczepański, Justyna Kącikowska, Jakub Trzaskowski, Katarzyna Sordyl, Krzysztof Rogulski, Weronika Kuśmierczyk, Oliwia Grzelak; validation, Magdalena Majkowska, Maria Fengier, Piotr Szczepański, Oliwia Bolek, Oliwia Grzelak; formal analysis, Magdalena Majkowska, Krzysztof Rogulski, Weronika Kuśmierczyk; investigation, Magdalena Majkowska, Justyna Kącikowska,

Krzysztof Rogulski; resources, Magdalena Majkowska, Katarzyna Sordyl, Jakub Trzaskowski; writing—original draft preparation, Magdalena Majkowska; writing—review and editing, Magdalena Majkowska, Maria Fengier, Oliwia Bolek; visualization, Magdalena Majkowska, Oliwia Grzelak, Piotr Szczepański; supervision, Magdalena Majkowska, Piotr Szczepański, Maria Fengier; project administration, Magdalena Majkowska. All authors have read and agreed to the published version of the manuscript.

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References

[1] Revelo Herrera SG, Leon-Rojas JE. The Effect of Aerobic Exercise in Neuroplasticity, Learning, and Cognition: A Systematic Review. Cureus. 2024 Feb 11;16(2):e54021. doi: 10.7759/cureus.54021.

[2] BDNF: a key factor with multipotent impact on brain signaling and synaptic plasticity. Kowiański P, Lietzau G, Czuba E, Waśkow M, Steliga A, Moryś J. Cell Mol Neurobiol. 2018;38:579–593. doi: 10.1007/s10571-017-0510-4

[3] De Assis GG, Gasanov EV, de Sousa MBC, Kozacz A, Murawska-Cialowicz E. Brain derived neutrophic factor, a link of aerobic metabolism to neuroplasticity. J Physiol Pharmacol. 2018 Jun;69(3). doi: 10.26402/jpp.2018.3.12. Epub 2018 Oct 18.

[4] Ruiz-González D, Hernández-Martínez A, Valenzuela PL, Morales JS, Soriano-Maldonado A. Effects of physical exercise on plasma brain-derived neurotrophic factor in neurodegenerative disorders: A systematic review and meta-analysis of randomized controlled trials. Neurosci Biobehav Rev. 2021 Sep;128:394-405. doi: 10.1016/j.neubiorev.2021.05.025. Epub 2021 Jun 1.

[5] Yuping Z, Tianbi L, Wentao S, Yun L, Guodong Z. The Optimal Type and Dose of Exercise for Elevating Brain-Derived Neurotrophic Factor Levels in Patients With Depression: A Systematic Review

With Pairwise, Network, and Dose-Response Meta-Analyses. Depress Anxiety. 2024 Dec 21;2024:5716755. doi: 10.1155/da/5716755.

[6] Exercise training increases size of hippocampus and improves memory. Erickson KI, Voss MW, Prakash RS, et al. Proc Natl Acad Sci U S A. 2011;108:3017–3022. doi: 10.1073/pnas.1015950108

[7] de Assis GG, de Almondes KM. Exercise-dependent BDNF as a Modulatory Factor for the Executive Processing of Individuals in Course of Cognitive Decline. A Systematic Review. Front Psychol. 2017 Apr 19;8:584. doi: 10.3389/fpsyg.2017.00584.

[8] Ben-Zeev T, Shoenfeld Y, Hoffman JR. The Effect of Exercise on Neurogenesis in the Brain. Isr Med Assoc J. 2022 Aug;24(8):533-538.

[9] Reycraft JT, Islam H, Townsend LK, Hayward GC, Hazell TJ, Macpherson REK. Exercise Intensity and Recovery on Circulating Brain-derived Neurotrophic Factor. Med Sci Sports Exerc. 2020 May;52(5):1210-1217. doi: 10.1249/MSS.0000000000002242.

[10] Andrews SC, Curtin D, Hawi Z, Wongtrakun J, Stout JC, Coxon JP. Intensity Matters: High-intensity Interval Exercise Enhances Motor Cortex Plasticity More Than Moderate Exercise. Cereb Cortex. 2020 Jan 10;30(1):101-112. doi: 10.1093/cercor/bhz075.

[11] Azevedo KPM, de Oliveira VH, Medeiros GCBS, et al. The Effects of Exercise on BDNF Levels in Adolescents: A Systematic Review with Meta-Analysis. International Journal of Environmental Research and Public Health. 2020 Aug;17(17):E6056.

[12] Blackmore DG, Schaumberg MA, Ziaei M, Belford S, To XV, O'Keeffe I, Bernard A, Mitchell J, Hume E, Rose GL, Shaw T, York A, Barth M, Cooper EJ, Skinner TL, Nasrallah F, Riek S, Bartlett PF. Long-Term Improvement in Hippocampal-Dependent Learning Ability in Healthy, Aged Individuals Following High Intensity Interval Training. Aging Dis. 2024 Jun 27;16(3):1732-1754. doi: 10.14336/AD.2024.0642.

[13] Guo Y, Wang S, Chao X, Li D, Wang Y, Guo Q, Chen T. Multi-omics studies reveal ameliorating effects of physical exercise on neurodegenerative diseases. Front Aging Neurosci. 2022 Oct 31;14:1026688. doi: 10.3389/fnagi.2022.1026688.

[14] Théodore Decaix, Claire Bonnin, Karl Götze, Véronique François, Camille Petit, Clémentine Rivière, Sandrine Greffard, Emmanuel Cognat, Jacques Hugon, Claire Paquet, Louise Sindzingre, Matthieu Lilamand, Benefits of physical activity on cognitive function in patients with neurocognitive disorders: A systematic review, The Journal of Frailty & Aging, Volume 14, Issue 5, 2025, 100069, ISSN 2260-1341, https://doi.org/10.1016/j.tjfa.2025.100069.

[15] Jaberi S, Fahnestock M. Mechanisms of the Beneficial Effects of Exercise on Brain-Derived Neurotrophic Factor Expression in Alzheimer's Disease. Biomolecules. 2023 Oct 26;13(11):1577. doi: 10.3390/biom13111577.

[16] Pahlavani HA. Exercise therapy to prevent and treat Alzheimer's disease. Front Aging Neurosci. 2023 Aug 4;15:1243869. doi: 10.3389/fnagi.2023.1243869.
[17] Biazus-Sehn LF, Schuch FB, Firth J, Stigger FS. Effects of physical exercise on cognitive function of older adults with mild cognitive impairment: A systematic review and meta-analysis. Arch Gerontol Geriatr. 2020 Jul-Aug;89:104048. doi: 10.1016/j.archger.2020.104048. Epub 2020 May 12.
[18] Gaitán JM, Moon HY, Stremlau M, Dubal DB, Cook DB, Okonkwo OC, van Praag H. Effects of Aerobic Exercise Training on Systemic Biomarkers and Cognition in Late Middle-Aged Adults at Risk for Alzheimer's Disease. Front Endocrinol (Lausanne). 2021 May 20;12:660181. doi: 10.3389/fendo.2021.660181.
[19] Kaagman DGM, van Wegen EEH, Cignetti N, Rothermel E, Vanbellingen T, Hirsch MA. Effects and Mechanisms of Exercise on Brain-Derived Neurotrophic Factor (BDNF) Levels and Clinical Outcomes in People with Parkinson's Disease: A Systematic Review and Meta-Analysis. Brain Sci. 2024 Feb 21;14(3):194. doi: 10.3390/brainsci14030194.
[20] Paterno A, Polsinelli G, Federico B. Changes of brain-derived neurotrophic factor (BDNF) levels after different exercise protocols: a systematic review of clinical studies in Parkinson's disease. Front Physiol. 2024 Feb 20;15:1352305. doi: 10.3389/fphys.2024.1352305.
[21] Tsukita K, Sakamaki-Tsukita H, Takahashi R. Long-term effect of regular physical activity and exercise habits in patients with early Parkinson disease. *Neurology*. 2022;98:e859-e871. doi:10.1212/WNL.0000000000013218.
[22] Ross RE, VanDerwerker CJ, Saladin ME, Gregory CM. The role of exercise in the treatment of depression: biological underpinnings and clinical outcomes. Mol Psychiatry. 2023 Jan;28(1):298-328. doi: 10.1038/s41380-022-01819-w. Epub 2022 Oct 17.
[23] Murawska-Ciałowicz E, Wiatr M, Ciałowicz M, Gomes de Assis G, Borowicz W, Rocha-Rodrigues S, Paprocka-Borowicz M, Marques A. BDNF Impact on Biological Markers of Depression-Role of Physical Exercise and Training. Int J Environ Res Public Health. 2021 Jul 15;18(14):7553. doi: 10.3390/ijerph18147553.
[24] Jemni M, Zaman R, Carrick FR, Clarke ND, Marina M, Bottoms L, Matharoo JS, Ramsbottom R, Hoffman N, Groves SJ, Gu Y, Konukman F. Exercise improves depression through positive modulation of brain-derived neurotrophic factor (BDNF). A review based on 100 manuscripts over 20 years. Front Physiol. 2023 Mar 8;14:1102526. doi: 10.3389/fphys.2023.1102526.
[25] Mojtabavi H, Shaka Z, Momtazmanesh S, Ajdari A, Rezaei N. Circulating brain-derived neurotrophic factor as a potential biomarker in stroke: a systematic review and meta-analysis. J Transl Med. 2022 Mar 14;20(1):126. doi: 10.1186/s12967-022-03312-y.
[26] Zhu M, Chen W, Zhang J. Aerobic exercise, an effective intervention for cognitive impairment after ischemic stroke. Front Aging Neurosci. 2025 Apr 4;17:1514271. doi: 10.3389/fnagi.2025.1514271.

[27] Montero-Almagro G, Bernal-Utrera C, Geribaldi-Doldán N, Nunez-Abades P, Castro C, Rodriguez-Blanco C. Influence of High-Intensity Interval Training on Neuroplasticity Markers in Post-Stroke Patients: Systematic Review. J Clin Med. 2024 Mar 29;13(7):1985. doi: 10.3390/jcm13071985.
[28] Górna S, Podgórski T, Kleka P, Domaszewska K. Effects of Different Intensities of Endurance Training on Neurotrophin Levels and Functional and Cognitive Outcomes in Post-Ischaemic Stroke Adults: A Randomised Clinical Trial. Int J Mol Sci. 2025 Mar 20;26(6):2810. doi: 10.3390/ijms26062810.