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Literature Review

Exercise and Neuroplasticity: Mechanisms and Implications

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ABSTRACT

Physical exercise may support brain health through neuroplastic adaptations. However, molecular and cellular findings from animal studies require careful interpretation when considered alongside human neuroimaging and cognitive outcomes. This literature review aims to synthesize human and animal evidence on the mechanisms of exercise-related neuroplasticity and discuss their implications for learning, memory, and brain health. This literature review used an initial Google Scholar search conducted in August–September 2025 with the terms neuroplasticity", "neuroplasticity" AND "history", "exercise" AND "BDNF", "neuroplasticity and exercise", and "exercise" AND "learning" AND "memory". The literature was updated in September 2026, with a priority on publications from 2016 to 2026. Human and animal studies and reviews were selected for relevance and synthesized narratively. Animal studies indicate that exercise can influence synaptic plasticity, neuronal morphology, glial responses, and neurotrophic signaling involving brain-derived neurotrophic factor, insulin-like growth factor-1, and vascular endothelial growth factor. Human studies report variable effects on brain volume, functional connectivity, and cognitive performance. Structural benefits may reflect preservation rather than consistent increases in brain volume. Exercise modality, intensity, duration, and frequency may influence these responses, but available evidence does not establish a universally optimal prescription. Peripheral biomarkers and neuroimaging changes do not directly demonstrate neurogenesis or necessarily indicate cognitive improvement. Exercise represents a promising strategy for supporting neuroplasticity and selected cognitive functions. Translation into practice requires attention to population characteristics, exercise protocols, and measured outcomes. Longer-term studies integrating mechanistic and functional assessments are needed to establish the persistence and clinical relevance of these benefits.

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INTRODUCTION

Neuroplasticity refers to the structural and functional changes in the brain that enable it to adapt to intrinsic and extrinsic stimuli by reorganizing its architecture, function, and neural connectivity, ultimately leading to physiological and morphological modification[1–3]. The

understanding of neuroplasticity evolved through successive theoretical and experimental advances. In the 1890s, Ramón y Cajal proposed that mental activity could modify neuronal connections, introducing the concept of "cerebral gymnastics" and anticipating an experience-dependent view of brain organization[4]. In 1949, Hebb proposed that repeated neuronal activation could strengthen synaptic connections and facilitate the formation of cell assemblies, providing a theoretical framework for learning and memory[5]. During the 1960s, experiments in Aplysia linked changes in synaptic transmission with behavioral modifications, shifting plasticity research toward experimentally measurable mechanisms[6]. The landmark reports of hippocampal long-term potentiation in 1973 subsequently demonstrated a persistent activity-dependent enhancement of synaptic efficacy, although this finding alone did not establish how memories are stored[4,5]. By the 1990s, studies of spike-timing-dependent plasticity, metaplasticity, and homeostatic plasticity had expanded the concept beyond synaptic strengthening to encompass timing-dependent modification and regulation of neuronal activity[6]. Together, these advances established neuroplasticity as a set of dynamic and regulated processes, providing a foundation for investigating how experience and interventions such as physical exercise influence brain function[7].

In the adult brain, neuroplasticity is continuously regulated, although its magnitude varies according to physiological and pathological conditions. This indicates that neuroplasticity is a dynamic process that can be modulated by multiple factors, including age, experience, and environmental influences[8]. As an adaptive mechanism, neuroplasticity reflects the brain's capacity to reorganize itself in response to environmental interactions, thereby supporting learning, memory formation, and functional recovery following injury. The remarkable complexity of brain structure and function has made neuroplasticity a central focus of contemporary neuroscience research[9]. Nevertheless, physiological aging and neurodegenerative disorders can trigger neuronal cell death through several pathological mechanisms, including excitotoxicity, oxidative stress, and the release of senescence-associated secretory phenotype (SASP) factors. Conversely, various lifestyle-related factors are known to activate endogenous protective mechanisms that counteract these pathological processes. Among these, physical exercise has consistently emerged as one of the most effective interventions[10].

Physical activity and exercise represent low-cost, non-pharmacological interventions that are effective for both the primary and secondary prevention of numerous chronic diseases, including diabetes mellitus, cancer, cardiovascular disease, and sarcopenia. Beyond their systemic health benefits, physical exercise exerts profound effects on brain health and has been associated with a reduced risk of neurodegenerative disorders, including dementia[11]. Exercise creates a biological environment that promotes neuroplasticity by enhancing neuronal development, survival, and activity, particularly within the hippocampus. These effects are mediated, in part,

through increased synthesis and secretion of brain-derived neurotrophic factor (BDNF), a key neurotrophic factor involved in neuronal survival, synaptic plasticity, and cognitive function[1]. Consequently, neuroplasticity has become a fundamental mechanism underlying recovery from brain injury and neurodegenerative diseases by facilitating the formation of new neural pathways and compensatory neuronal networks[12].

This literature review aims to synthesize evidence from human and animal studies on the molecular, cellular, structural, and functional mechanisms underlying exercise-related neuroplasticity and to discuss their implications for learning, memory, and brain health during aging. It also examines how exercise characteristics may influence these outcomes and considers the limitations of translating mechanistic findings into practical exercise interventions.

LITERATURE REVIEW

This article was prepared as a literature review. The initial literature search was conducted using Google Scholar in August–September 2025 with the search terms “neuroplasticity”, "neuroplasticity" AND "history", "exercise" AND "BDNF", “neuroplasticity and exercise”, and "exercise" AND "learning" AND "memory". The literature was subsequently updated in September 2026 during manuscript revision to incorporate recent evidence and replace older references, prioritizing publications from 2016 to 2026. Articles were selected according to their relevance to the concepts and mechanisms of neuroplasticity, exercise-related molecular and cellular adaptations, structural and functional brain changes, and implications for learning, memory, and brain health. The included literature comprised original human and animal studies, literature reviews, systematic reviews, and meta-analyses. Evidence was synthesized narratively, with distinctions made between human and animal findings and between molecular, neuroimaging, electrophysiological, and cognitive outcomes.

Basic Concepts of Neuroplasticity

The mammalian central nervous system (CNS) was once considered structurally stable after birth, with only limited potential for change throughout life. However, advances in neuroscience have demonstrated that the brain retains a remarkable capacity for lifelong plasticity. Neural plasticity enables the nervous system to modify synaptic connections in response to experience or injury, thereby supporting learning, memory formation, and functional recovery[12,13].

Neuroplasticity is broadly defined as the brain's ability to modify, reorganize, and adapt both its structural and functional properties throughout life in response to experience[3]. Rather than representing a single biological process, neuroplasticity encompasses multiple levels of adaptation. At the highest level are adaptive behaviors, such as learning and memory, which reflect

the integration of structural remodeling and functional changes within the brain. These adaptations are rooted in molecular and cellular mechanisms involving synapses, neuronal circuits, and complex patterns of neural connectivity. As the primary sites of neuronal communication, synapses serve as the central substrates of neuroplasticity, undergoing continuous structural and functional remodeling in response to neuronal activity. Neuronal plasticity is mediated through multiple mechanisms, including neurogenesis, apoptosis, neural network reorganization, and experience-dependent synaptic plasticity[10,13].

Repeated synaptic activation induces long-lasting changes in synaptic efficacy through Hebbian plasticity, including long-term potentiation (LTP), long-term depression (LTD), and spike-timing-dependent plasticity (STDP)[10,12,13]. Hebbian plasticity describes the strengthening of synaptic connections following repeated, correlated activity of presynaptic and postsynaptic neurons; in some forms of associative learning, neuromodulatory signaling also contributes to memory formation[5]. This process primarily involves excitatory glutamatergic synapses, as well as certain inhibitory GABAergic synapses. These activity-dependent changes constitute the core mechanism of neuroplasticity and provide the foundation for the formation of engrams, the physical representations of experience encoded within synaptic networks[10,12,13]. Hebbian plasticity is widely regarded as a fundamental mechanism underlying information encoding and storage in the brain. By strengthening neural connections between coactive neurons, it enables the brain to associate events that occur simultaneously, thereby providing a fundamental basis for learning and memory[14].

Exercise-Induced Mechanisms of Neuroplasticity

Aerobic exercise, as part of rehabilitation strategies, has been shown to enhance neuroplasticity at multiple levels, including molecular, cellular, and functional reorganization of the nervous system[15]. Exercise-induced structural and functional adaptations of the brain have been consistently demonstrated in both human and animal studies. A growing body of evidence indicates that physical exercise plays a pivotal role in enhancing brain function throughout the lifespan[7].

A recent systematic review by Herrera and Leon-Rojas (2024) reported that aerobic exercise, particularly at high intensity, enhances cortical excitability and improves cognitive function. In addition, exercise induces structural and functional adaptations in the cerebral cortex and other brain regions. These beneficial effects have been consistently observed across different age groups, including both healthy individuals and patients with neurological disorders[16].

Another systematic review by Fernandes et al. (2020) demonstrated that exercise promotes neuroplasticity by increasing the production of neurotrophic factors, including brain-derived neurotrophic factor (BDNF), glial cell line-derived neurotrophic factor (GDNF), and nerve

growth factor (NGF), as well as by upregulating the expression of their corresponding receptors, tropomyosin receptor kinase B (TrkB) and p75 neurotrophin receptor (p75 NTR). Activation of these signaling pathways contributes to improvements in cognitive performance, particularly in learning and memory, as demonstrated in both human and animal studies [2].

The effects of exercise on neuroplasticity may vary according to exercise modality, intensity, session duration, weekly frequency, and overall intervention length. These relationships should be interpreted in relation to the population studied and the outcome assessed. In an experimental study involving 20 healthy adults, Andrews et al. (2020) compared 20 minutes of high-intensity interval cycling, moderate-intensity continuous cycling, and rest. The interval protocol alternated three minutes at 50% heart rate reserve with two minutes at up to 90% heart rate reserve, whereas continuous cycling was performed at 50% heart rate reserve. High-intensity interval exercise enhanced the motor cortical response to intermittent theta-burst stimulation compared with rest, while moderate-intensity exercise produced intermediate responses. These findings indicate that exercise intensity can influence acute, long-term potentiation-like cortical plasticity, although this assessment does not directly measure hippocampal synaptic potentiation or establish sustained cognitive benefits[1].

Gholami et al. (2025) synthesized 35 randomized controlled trials in older adults and found that aerobic, resistance, and combined exercise increased resting peripheral brain-derived neurotrophic factor (BDNF) concentration for repeated training. Resistance training showed the largest estimated effect among the modality subgroups. In separate subgroup analyses, the largest effects were reported for 12-week programs, moderate-to-vigorous or vigorous exercise, and frequencies of three to four sessions per week. However, these comparisons across studies do not establish the superiority of resistance training or demonstrate that combining these characteristics produces an optimal prescription. Furthermore, increased circulating BDNF represents a neurotrophic response rather than direct evidence of brain neurogenesis or improved cognition[17].

Evidence from longer interventions also identifies protocols associated with structural and functional benefits. Blackmore et al. (2025) compared low-intensity exercise, moderate-intensity continuous training, and high-intensity interval training in healthy adults aged 65–85 years. Participants trained three times per week for six months. The high-intensity protocol consisted of four four-minute treadmill intervals at 85–95% of peak heart rate, separated by three-minute active recovery periods, with a total session duration of 40 minutes including warm-up and cool-down. This group showed greater improvement in hippocampal-dependent paired associative learning than the other groups, alongside preservation of hippocampal volume and changes in functional connectivity. Thus, high-intensity interval training has demonstrated promising effects on selected cortical and hippocampal outcomes, whereas several exercise

modalities can enhance peripheral BDNF. Nevertheless, the available evidence does not establish a universally optimal exercise type or dose for neuroplasticity across populations and outcomes[18].

Macroscopic Structural and Functional Changes

Human neuroimaging studies have investigated exercise-related changes in brain structure and function. Structural magnetic resonance imaging (MRI) is used to estimate brain volume and cortical thickness before and after exercise interventions. In a meta-analysis of randomized trials involving humans, Li et al. (2026) found a small relative benefit of exercise on brain volume, suggesting preservation rather than a consistent absolute increase[19]. Tarumi et al. (2025) reviewed aerobic training studies and reported variable findings for hippocampal volume and prefrontal cortical thickness[20]. To assess functional connectivity, Jung et al. (2026) obtained resting-state functional MRI (fMRI) scans before and after a six-month aerobic exercise intervention in older adults. Functional connectivity was quantified using pairwise correlations between preprocessed blood oxygen level-dependent (BOLD) time series from brain regions within predefined networks. Comparing these correlations before and after training allowed the researchers to evaluate changes in the temporal coordination of activity within brain networks. BOLD signals provide an indirect measure of neural activity rather than a direct recording of synaptic transmission. Compared with light-intensity training, moderate-to-vigorous training was associated with greater dorsal attention network connectivity within a limited age range. In selected age groups, changes in default mode and frontoparietal control network connectivity were associated with improvements in cognitive performance[21].

These imaging findings describe changes at the macroscopic level. At the microscopic level, experimental evidence suggests that exercise-related neuroplasticity may involve neurogenesis, changes in synaptic structure and function, angiogenesis, and glial responses, with neurotrophic and growth factors contributing to these processes[7,15].

Microscopic Structural Changes

A fundamental principle of neurobiology states that "neurons that fire together, wire together." In other words, the simultaneous activation of two neurons strengthens the synaptic connections between them. This principle forms the basis of synaptic plasticity, which underlies the brain's capacity for learning and adaptation. Physical exercise has been shown to enhance synaptic function by modulating glutamatergic neurotransmission, particularly through the α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) and N-methyl-D-aspartate (NMDA) receptors[22].

Dendrites and axons are the principal structural components of neurons and play critical roles in exercise-induced neuroplasticity[22]. The effects of exercise on neuronal morphology have

been extensively investigated across multiple brain regions[7]. In animal models, exercise has been shown to mitigate disease- or stress-related alterations in dendritic spines across different brain regions. Treadmill exercise reversed the reduction in dendritic spine density in the hippocampal CA3 region of rats exposed to chronic unpredictable mild stress[23], while voluntary running before the induction of vascular dementia attenuated the loss of dendritic spine density in the rat striatum[24].

Exercise-related cellular and structural adaptations may contribute to changes in synaptic function. Long-term potentiation (LTP), a persistent enhancement of synaptic transmission following patterned stimulation, provides an electrophysiological measure of synaptic plasticity[7]. In male Wistar rats, Radahmadi et al. (2016) assessed dentate gyrus responses by stimulating the medial perforant pathway and recording field excitatory postsynaptic potentials (fEPSPs) and population spikes. The fEPSP slope and population spike amplitude were evaluated to characterize synaptic transmission and neuronal responsiveness, while LTP measurements assessed sustained potentiation following induction. Treadmill exercise for 21 or 42 days enhanced dentate gyrus responsiveness and LTP. These findings provide animal evidence of exercise-related hippocampal functional plasticity, but electrophysiological potentiation alone does not establish improved learning or memory[25].

In addition to its effects on neurons, exercise also modulates the structure and function of glial cells. Astrocytes, the predominant glial cell type in the brain, play a crucial role in regulating metabolic and energetic support from the cerebral vasculature to neurons. Because optimal astrocytic function is essential for maintaining neuronal activity, exercise-induced improvements in neuronal function are thought to be mediated, at least in part, by adaptive changes in astrocyte physiology[26]. In mice exposed to chronic alcohol, treadmill exercise attenuated astrocyte reactivity in the primary motor cortex, normalizing astrocytic soma size and partially reducing the excessive growth of astrocytic processes[27]. In aged female mice, voluntary wheel running shifted hippocampal microglial gene expression toward a younger profile and increased the relative proportion of homeostatic microglia compared with sedentary aged mice[28].

Neurotropic Factors and Neuroplasticity

The mechanisms underlying exercise-induced neuroplasticity involve the coordinated actions of several neurotrophic and growth factors[7]. At the molecular level, chronic aerobic exercise alters the peripheral concentrations of brain-derived neurotrophic factor (BDNF), insulin-like growth factor-1 (IGF-1), and vascular endothelial growth factor (VEGF). Chronic exercise has also been shown to enhance the transport and uptake of BDNF into the central nervous system (CNS)[16].

Brain-derived neurotrophic factor (BDNF) is a neurotrophin involved in neuronal growth, survival, and synaptic plasticity. Although glutamatergic neurons are an established source of BDNF, BDNF expression has also been detected in GABAergic interneurons[7,29]. Beyond neurons, BDNF is also synthesized and released by microglia, T and B lymphocytes, monocytes, and skeletal muscle cells, highlighting its broad physiological role in neural and systemic adaptation[11].

Brain-derived neurotrophic factor (BDNF) is generated through the post-translational processing of its glycosylated precursor protein, pro-brain-derived neurotropic factor (proBDNF), into its mature form, mature brain-derived neurotropic factor (mBDNF)[29]. Mature BDNF selectively binds to tropomyosin receptor kinase B (TrkB), whereas proBDNF specifically binds to the p75 neurotrophin receptor (p75 NTR) on the membrane of target cells[30].

Through TrkB, BDNF regulates dendritic plasticity by being selectively targeted to active synapses, thereby strengthening synaptic transmission. The interaction between BDNF and TrkB induces the phosphorylation of tyrosine residues within the receptor's tyrosine kinase domain, facilitating the recruitment of adaptor proteins. These adaptor proteins subsequently activate the Ras–mitogen-activated protein kinase (Ras–MAPK) signaling pathway, thereby promoting neuronal differentiation and proliferation[12,13].

TrkB activation also engages the phosphoinositide 3-kinase (PI3K)–Akt pathway, which supports neuronal survival. In parallel, TrkB-mediated activation of phospholipase C gamma (PLCγ) promotes the production of inositol trisphosphate (IP_3), leading to intracellular calcium release and activation of calcium/calmodulin-dependent kinases. These kinases contribute to the activation of cyclic AMP response element-binding protein (CREB), which is involved in long-term potentiation and other forms of synaptic plasticity[13].

In contrast, the binding of proBDNF, the immature form of BDNF, to its receptor, the p75NTR, triggers signaling cascades that lead to caspase-3-mediated apoptosis. ProBDNF/p75NTR-induced cell death occurs when the BDNF/TrkB signaling pathway is inhibited or absent[13]. Because proBDNF and mBDNF exert opposing effects, activation of their respective signaling pathways produces distinct cellular responses. ProBDNF signaling through p75NTR mediates processes such as apoptosis, dendritic spine retraction, and long-term depression (LTD), whereas mBDNF signaling through TrkB promotes neuronal survival, synapse formation, dendritic spine growth, and long-term potentiation (LTP). Therefore, the relative proportion of proBDNF and mBDNF release is a critical determinant of the direction of neuroplastic changes that occur in target neurons[11]. These findings indicate that the balance between proBDNF and mBDNF is not static but can be influenced by various environmental factors and therapeutic interventions. One intervention that has been shown to modulate this balance is physical exercise. Four weeks of aerobic exercise increased the mBDNF/proBDNF ratio in the hippocampus of rats with post-stroke hippocampal ischemia[30].

Brain-derived neurotrophic factor (BDNF) is capable of crossing the blood–brain barrier (BBB), and approximately 75% of the BDNF present in peripheral blood plasma is derived from the brain. Therefore, BDNF concentrations in plasma or serum are often regarded as indirect indicators of BDNF released within the brain and can be used to evaluate the effects of lifestyle interventions, such as physical exercise or caloric restriction, on BDNF-mediated neuroplasticity[11,15]. Brain-derived neurotrophic factor mediates neuroplasticity through multiple mechanisms operating over different time scales. BDNF/TrkB signaling can induce long-term potentiation (LTP) at glutamatergic synapses and promote the formation of new dendritic spines within seconds to minutes, thereby enabling the establishment of new memory traces at existing synapses. Over the course of several hours, BDNF contributes to protein synthesis-dependent long-term LTP consolidation and memory formation. Over longer time scales, BDNF also participates in the formation of memory engrams (long-term memory traces) by incorporating newly generated neurons into neural circuits through enhanced neurogenesis and synaptogenesis. Thus, BDNF signaling plays a central role in the cellular mechanisms of neuronal plasticity underlying memory formation, strengthening, and retrieval[11].

In addition to BDNF, insulin-like growth factor-1 (IGF-1) is another important modulator of neuronal function in the central nervous system (CNS). IGF-1 plays essential roles in synaptic plasticity, synaptic density, neurotransmitter transmission, neurogenesis, and neuronal differentiation. The enhancement of adult hippocampal neurogenesis, as well as the improvements in learning and memory induced by chronic exercise, has been largely attributed to the activation of the IGF-1 signaling pathway in the hippocampus[7].

Another important factor is vascular endothelial growth factor (VEGF), an angiogenic protein that also possesses neuroprotective and neurotrophic properties. VEGF promotes the proliferation of neuronal precursor cells and creates a vascular environment that supports neuronal growth. VEGF can be synthesized by peripheral vascular endothelial cells as well as by several cell types in the brain, including astrocytes, ependymal cells, and neural stem cells. Voluntary exercise has been shown to increase the expression of the VEGF receptors VEGFR1 (Flt-1) and VEGFR2 (Flk-1) in the hippocampus. Activation of the intracellular tyrosine kinase domains of these receptors initiates multiple signaling pathways that ultimately enhance the proliferation of neuronal precursor cells[7,15].

Implications for Cognitive Function and Brain Health

Exercise-related changes in synaptic plasticity and hippocampal function may support improvements in learning and memory. Evidence from animal studies links exercise-induced synaptic remodeling and neurotrophic signaling with improved performance on learning and memory tasks[7]. Human studies also demonstrate functional benefits. Suwabe et al. (2018) found

that a single 10-minute bout of very light exercise improved mnemonic discrimination—the ability to distinguish similar visual stimuli—in healthy young adults, alongside increased functional connectivity between hippocampal and cortical regions. These findings link exercise-related brain responses with measurable memory performance, although an acute improvement in a specific task does not establish sustained enhancement of general learning ability[31].

During aging, exercise may help preserve brain structure and support neural networks involved in cognitive performance. In a randomized trial involving healthy adults aged 65–85 years, six months of high-intensity interval training (HIIT) improved hippocampal-dependent learning, assessed using a paired associative learning task, and was accompanied by preservation of hippocampal volume and increased functional connectivity between selected brain networks[18]. However, cognitive benefits are not consistently observed across trials. Tarumi et al. (2025) reviewed studies in which aerobic training did not produce significantly greater cognitive improvements than active control interventions. These findings support the potential of exercise to maintain cognitive health while highlighting variability in its effects. Improvements in cognitive test performance or preservation of brain structure should therefore be distinguished from demonstrated prevention of dementia[20].

These findings have implications for the design of exercise interventions. Exercise modality, intensity, session duration, weekly frequency, and intervention length should be explicitly defined when evaluating cognitive outcomes. For example, Blackmore et al. (2025) reported greater improvements in paired associative learning following supervised treadmill HIIT performed three times weekly for six months than following low- or moderate-intensity training. The HIIT protocol included four 4-minute intervals at 85–95% of peak heart rate, separated by 3-minute active recovery periods[18]. Nevertheless, its effectiveness for this outcome in healthy older adults does not establish its superiority for other cognitive domains or populations. Intervention design should account for participants' health status, physical capacity, adherence, and feasibility. The variability of findings across studies supports further comparative research rather than a universally optimal exercise prescription for neuroplasticity or cognition[7,20].

Translation into clinical practice requires careful interpretation of differences between animal and human evidence. Animal studies permit direct assessment and experimental manipulation of cellular and molecular processes, whereas human studies generally assess exercise responses using circulating biomarkers, neuroimaging, and cognitive tests[7]. These measures provide complementary information but do not independently establish the mechanisms underlying functional benefits. For example, circulating BDNF does not directly demonstrate hippocampal neurogenesis, and changes in brain volume or connectivity do not necessarily indicate cognitive improvement. In aged female mice, microglial depletion reduced exercise-induced hippocampal neurogenesis without eliminating the cognitive benefits of

exercise, illustrating a partial dissociation between cellular and behavioral outcomes[28]. Future studies should assess mechanistic indicators and functional outcomes together, clearly characterize exercise exposure, and include sufficient follow-up to determine the persistence and clinical relevance of cognitive benefits.

CONCLUSION

Physical exercise can influence neuroplasticity through molecular, cellular, structural, and functional adaptations. Animal studies provide evidence of changes in neurotrophic signaling, synaptic plasticity, neuronal morphology, and glial responses, whereas human studies demonstrate variable effects on brain structure, functional connectivity, and cognitive performance. Structural benefits may involve preservation of brain volume rather than consistent enlargement, and changes in biological markers or neuroimaging measures do not necessarily coincide with cognitive improvement.

These findings support the potential of exercise to promote selected aspects of learning, memory, and brain health during aging. However, differences in populations, exercise modalities, training doses, and assessment methods limit the identification of a universally optimal exercise prescription. Translation into practice should therefore consider individual characteristics and distinguish mechanistic evidence from demonstrated functional benefits. Further research integrating mechanistic assessments, cognitive outcomes, and longer-term follow-up is needed to clarify the persistence and clinical relevance of exercise-related adaptations.

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AUTHOR CONTRIBUTIONS

All authors contributed to the conceptualization of the review, literature search and selection, critical analysis and synthesis of the literature, and the drafting and revision of the manuscript. All authors reviewed and approved the final version of the manuscript.

CONFLICT OF INTEREST

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HUMAN AND ANIMAL RIGHTS

This review did not involve human participants or animals.

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