

PHYSICAL EXERCISE AND INCREASED BDNF EXPRESSION: A PHYSIOLOGICAL APPROACH TO PRESERVE MEMORY

João Vitor Dana Schutz

Gabriel Ferreira Nunes

Giorgia Dallagnol Teixeira de Freitas

Luise Schramm Brill

Nicole Augusto

Ricardo Corrêa da Cunha

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INTRODUCTION

Brain-Derived Neurotrophic Factor (BDNF) is a relevant protein in neurophysiology, playing an important role in maintaining synaptic plasticity, especially in the hippocampus, a brain region responsible for memory consolidation. For its effects to manifest, this factor needs to bind to the tyrosine kinase B (TrkB) receptor, triggering a complex intracellular signaling cascade that regulates cognitive processes, mainly the preservation of memory. In this context, several physiological mechanisms can influence the increase in BDNF expression, promoting an increase in connectivity between neurons, a phenomenon known as connectome. Of these, the practice of physical exercise (PE) stands out, as it promotes an increase in Irisin, a myokine intrinsically associated with the increase in BDNF, triggering several physiological processes and thus contributing significantly to the preservation of memory.

OBJECTIVES

Explore the role of BDNF in memory preservation, highlighting the physiological mechanisms that influence its expression and the contribution of EF to neuronal connectivity.

METHODOLOGY

109 filtered articles were identified in the period between 2018 and 2023, through the SciElo, PubMed and Science Direct databases, using the BDNF descriptors; Physical exercises; Memory. From reading titles and abstracts, 56 of the articles found were submitted for literary review. As a result, 24 articles were included in the analysis, which had relevance to the study topic as inclusion criteria, while data duplication and insufficiency were exclusion criteria.

RESULTS

Among the hypotheses about the influence of the relationship between PE and memory, the most current is that the practice promotes the release of a series of proteins into the bloodstream, including PGC1- α in its activated form. This polymer is a transcription factor, which promotes the regulation of the expression of the FndC5 gene in both skeletal muscle and the hippocampus, leading to the production and release of the irisin protein into the blood. The increase in serum levels of this myokine that crosses the blood-brain barrier induces greater expression of the BDNF gene. This increase consequently generates greater translation of the BDNF protein, which plays a fundamental role in the process of modulating synaptic plasticity, necessary for long-term memory consolidation and effective information retrieval. BDNF is essential in this process by signaling through interaction with TrkB. The BDNF-TrkB system, therefore, induces a greater long-lasting potential (LTP), which quickly triggers actin polymerization in dendritic spines in the hippocampus, resulting in an increase in neuronal spread, reinforcing the study on the human connectome. This strengthening of synaptic plasticity contributes to long-term memory consolidation, facilitating information retrieval and improving memory efficiency by weakening irrelevant connections.

CONCLUSION

In conclusion, the interaction between BDNF, EF and the irisin protein is fundamental in understanding the mechanisms underlying the preservation of memory and brain plasticity. However, a limitation of this study is the complexity of the signaling pathways, which still require additional clarification.