Mechanisms Underlying Exercise-Induced Neuroplasticity: Exerkines and Long-Term Synaptic Potentiation

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Received date: 05-June-2022, Manuscript No: jmso-22-70820; **Editor assigned:** 07-June-2022, PreQC No. jmso-22-70820(PQ); **Reviewed**: 20-June-2022, QC No. jmso-22-70820 (Q); **Revised Date:** 23-June-2022, Manuscript No: jmso-22-70820(R); **Published date:** 30-June-2022, doi: 10.35248/2376-0389.22.9.6.449

Abstract

By altering cellular and molecular processes in the brain, exercise may enhance cognitive function. We contend that a key component of exercise's beneficial effects on the brain is the facilitation of Long-term synaptic Potentiation (LTP)-related pathways by exerkines, which are substances produced as a result of physical activity. This review lists the synaptic pathways that cytokines activate and may enhance LTP. For a total of 16 exerkines, we showed how blood and brain exerkine levels are affected by the type of exercise (e.g., aerobic or resistance exercise) and whether it is repeated several times (i.e., chronic exercise) (i.e., chronic exercise). The creation of customised physical activity routines may be made possible by this information. Last but not least, this study may help to focus future research on basic gaps in our understanding of the biophysical connections between muscle activity and the brain at both the cellular and system levels.

Keywords: Exercise • Myokine • Long-term potentiation • Synaptic plasticity • Inflammation • Intracellular signaling peptides and proteins • Cytokines • Neurotrophic factor • Brain • Biomarkers

Introduction

In the 1930s, the positive impact of physical activity on cognition first appeared in literature [1]. A search in PubMed using the terms "exercise" AND "cognition" reveals the explosive growth of this field in recent years, with over 100 publications per year in 1998 and over 3000 in 2020. The complexity of the neurophysiological pathways that mediate the advantageous effects of physical exercise on the brain, however, shows that the underlying mechanisms of exercise-induced cognitive gains are still not entirely known. Neuroplasticity is crucially important in this regard. The term "neuroplasticity" describes the brain's capacity for structural and functional changes in response to internal or external stimuli from the environment or bodily organs [2].

There are currently a tonne of studies demonstrating that physical exercise exposure, whether acute (defined as a single bout) or chronic (defined as a programme of repeated bouts), may very likely cause neuroplasticity.

In human acute exercise research, Proton Magnetic Resonance Spectroscopy (1H-MRS) has been used to evaluate transient changes in neurotransmitter levels, such as glutamate and Gamma-Aminobutyric Acid (GABA), shortly after physical exercise [3]. In the mammalian brain, glutamate and GABA play key roles as neurotransmitters that mediate Long-Term Synaptic Potentiation (LTP) and Long-Term Synaptic Depression (LTD) via glutamatergic and GABAergic pathways, respectively. The neuroplastic processes LTP and LTD lead the brain's excitatory synaptic connections to either get stronger or get weaker.

Early LTP and LTD both cause changes in the synapse that involve a quick, temporary change in the function of synaptic proteins that are already present and a slower, more significant change in the availability of synaptic proteins that involves targeting cell DNA and inducing the transcription of new proteins (late LTP or LTD) [4].

Many brain regions exhibit "LTP-like" processes that increase the effectiveness of synaptic neurotransmission through neural networks. These processes are essential for numerous domains of cognitive function. For instance, it has been proposed that disruption of LTP-like processes in the hippocampus, prefrontal, visual, auditory, and motor cortex will affect executive function, working memory, and episodic memory, as well as visual, auditory, and motor processing. Aging, Alzheimer's disease, major depressive disorder, and other mental and neurological conditions can all cause these abnormalities. While invasive in vivo or in vitro electrophysiological experiments are required for the direct detection of LTP, non-invasive methods can also be used to measure LTP-like activities [5]. Transcranial Magnetic Stimulation (TMS)., for instance, can be used to evaluate LTP-like processes in the human motor cortex. Additionally, Visually Evoked Potentials (VEP) and Auditory Evoked Potentials (AEP) measurements on Electroencephalography (EEG) can reveal LTP-like processes in the visual cortex or auditory cortex, respectively [6].

Exercise has the potential to alter the brain's neuroplasticity in both short-term and long-term ways. Early LTP is thought to be responsible for the short-lived functional changes in the brain that take place during and/or right after intense exercise. TMS, EEG, Functional Near-infrared Spectroscopy (fNIRS), and Functional Magnetic Resonance Imaging can all be used to identify these Functional Brain Alterations (fMRI). Furthermore, although meaningful structural alterations have only been noted after chronic exercise, late LTP mechanisms are likely triggered during and/or immediately after acute exercise [7].

It's significant to note that the pathways engaged during late LTP also promote the production of growth and survival-promoting proteins, including Brain-Derived Neurotrophic Factor (BDNF). BDNF transcription was seen both after brief bouts of intense exercise and long-term exercise. As a result of the increased BDNF availability, the dentate gyrus of the hippocampus may contain more neurons due to an upregulation of neurogenesis pathways. It was noted that LTP processes were more easily activated in these newly generated neurons. These new neurons do not survive for more than three weeks without active learning and, consequently, without the activation of LTP [8]. This may suggest that in order to mature further and connect to functional networks, newly created neurons need the survival-promoting chemicals that are released during LTP. The biochemical and structural changes to the brain observed in chronic exercise studies, such as increases in N-acetyl aspartate, a neurometabolic marker of neuronal integrity measured with 1H-MRS, and increases in grey matter volume and white matter microstructural organisation detected with Magnetic Resonance Imaging (MRI), could be explained by a successful process of neurogenesis. These are intriguing results because larger brain size and higher N-acetyl aspartate levels have been linked to improved cognitive function in older persons.

A large body of research indicates that both acute and chronic exercise have positive effects on the biological mechanisms that mediate neuroplasticity. This may be due to an enhanced response to LTP induction brought on by physical exercise, which in turn causes structural and functional changes in the brain that enhance cognitive function [9]. However, it is still unclear exactly how muscle action leads to LTP facilitation. The exerkine hypothesis is a widely accepted explanation for the process underlying the improvement in cognitive function following physical exercise. All peptides, metabolites, and nucleic acids that are released into the bloodstream during and after physical activity are referred to as exerkines. They are known as myokines, adipokines, or hepatokines, depending on the organ from which they are released. These terms relate to substances that are released as a result of physical activity from muscles, adipose tissue, or the liver.

The blood-brain barrier may be crossed by some of these cytokines [10]. Exerkines that have crossed the blood-brain barrier may be able to assist the signalling pathways that control LTP development.

In this narrative review, we explain how physical exercise-induced enhancement of the LTP process by the release of exerkines may contribute to bettering brain functions while demonstrating how it aligns with the widely held belief that exerkines are involved in a number of signalling pathways that facilitate neuroplasticity. Our overarching goal is to create a framework that organises all pertinent knowledge about eicosanoids that may be regulating early and late phases of LTP in the human brain. Only those growth factors, myokines, cytokines, metabolites, hormones, and neuropeptides that are known to be released or generated during physical activity and appear to have direct or indirect applications for the enhancement of early and/or late LTP were included in our review of the literature on these topics. We purposely avoided reviewing all exerkines that might pass the blood-brain barrier because there isn't enough data, it's unclear, or there is conflicting data to determine which exerkines' cellular pathways might facilitate LTP. We will describe each exerkine, discuss whether an effect is anticipated after acute or chronic exercise, and make a distinction between cardiovascular and resistance exercise for each one. In Section two, the LTP process will be briefly defined with an emphasis on the routes that will be discussed in the remaining sections of the study. Sixteen interesting eicosanoids will be discussed individually in section three. Growth factors, such as Brain-Derived Neurotrophic Factor (BDNF), Insulin-Like Growth Factor-1 (IGF-1), and Growth Hormone (GH), are discussed first. Pro- and anti-inflammatory biomarkers, such as cytokines and kynurenine, of which some are myokines, are then discussed. Then, we talk about additional myokines and metabolites, such as lactate and hydroxybutyrate. These include irisin, cathepsin-B, apelin, and adiponectin.

Conclusions

Exerkines, which are circulating substances brought on by exercise, were discussed along with their impact on pathways involved in LTP. We evaluated the physical exercise and subject factors for each of these inflammatory peptides that affect the changes in exerkine levels in the blood and the brain after short-term or long-term cardiovascular or resistance exercise.

This review outlines what is now properly understood and where research is lacking by merging and organising data from a sizable and quickly growing body of study. This information may help researchers develop a customised physical training programme to enhance cognitive health. One of the many benefits of physical activity for people of all ages, especially those with cognitive loss, is its positive impact on cognitive performance. In a society where the possibility of an ageing population looms large, this is crucial.

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Cite This Article: Smith T. Mechanisms Underlying Exercise-Induced Neuroplasticity: Exerkines and Long-Term Synaptic Potentiation. J Mult Scler. 2022, 09(6),449.