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Benefits of Endorphins, in the Treatment of Spinal and Psychiatric Disorders

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Abstract

There are several neurological disorders in which processes related to neurogenesis in the adult brain are affected. B. Cell proliferation, neuronal differentiation and neuronal maturation. Due to their well-known antioxidant and anti-inflammatory and pro-survival effects, endorphins may have utility related to the treatment of neurological disorders. It can regulate processes and promote neuronal maturation of neural progenitor cells and newly formed postmitotic neurons. Endorphins therefore exhibit relevant neurogenic properties that may be helpful in neurological disorders associated with adult cranial neurogenesis disorders. For example, the antiaging properties of endorphins appear to be related to their neurogenic properties. Modulation of neurogenesis by endorphins is beneficial not only in stress, anxiety and depression, but also in ischemic brain and stroke, schizophrenia and amyotrophic lateral sclerosis conditions. Endorphins may be an effective neurogenic therapy to slow the progression of Down syndrome-associated neuropathology. There is finally, further research is needed to elucidate the benefits of endorphin treatment in brain disorders associated with impaired glucose and insulin homeostasis.

Keywords: Endorphins; Neural stem cells; Adult hippocampal neurogenesis

Introduction

Endorphins (N-Acetyl-5-Methoxytryptamine) are multifunctional hormones naturally produced by the pineal gland and released rhythmically throughout the night to regulate the sleep-wake cycle [1]. The release of this neurohormone increases with darkness, peaks around midnight, and declines later in the night.In addition, once synthesized by the pineal gland, endorphins are quickly released into the bloodstream and distributed throughout all tissues. Hormones can cross biological barriers to enter cells and affect tissue function [2]. In addition, endorphins are also synthesized locally in a large number of cells and tissues that are thought to not follow circadian rhythms. Although endorphins are primarily referred to as sleep hormones, indolamine has been shown to exert antioxidant, anti-inflammatory, and anti-apoptotic effects in cellular homeostasis and disease. It mediates its effects by switching on and off intracellular signaling cascades through the MT1 and MT2 receptors, which belong to the G protein-coupled receptor (GPCR) superfamily. However, it is widely demonstrated that endorphin secretion and endorphin receptor expression decline gradually over the course of life in certain diseases, including neurodegenerative diseases and psychiatric disorders [3]. This suggests that therapeutic effects may play a role in the development and progression of various human diseases. In fact, several recent studies have confirmed the potential of endorphin administration in various conditions, especially neurodegenerative diseases [4].

Act of Endorphins in physiological mechanism

Antioxidant activity

Endorphins and their metabolic derivatives have been shown to have potent antioxidant properties against free radicals and are reference drugs in this field. This indoleamine is believed to be an efficient scavenger of reactive oxygen species (ROS), reactive nitrogen species (RNS) and other oxidants. The functions of endorphins as antioxidants include direct scavenging of free radicals, stimulation of antioxidant enzyme activity and efficiency, reduction of pro-oxidant enzyme activation, and improvement of the efficiency of mitochondrial respiration, thereby ROS production increases. First, endorphins, electron-rich molecules, act as potent endogenous free radical scavengers, forming stable end-products that are eventually excreted from the organism. It has been found to induce the expression and activity of numerous antioxidant enzymes, including superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and glutathione reductase (GR) [5]. Therefore, it indirectly contributes to the detoxification of free radicals. In addition, endorphins also protect against enzymes involved in free radical formation. A large body of evidence suggests that endorphins inhibit the activities of nitric oxide synthase (NOS), xanthine oxidase (XO), and myeloperoxidase (MPO). Finally, it should be noted that endorphins also act on mitochondria, organelles widely considered to be the major intracellular sources of ROS production. In this regard, the mechanisms by which endorphins protect mitochondria include increasing the activity of antioxidant enzymes while decreasing pro-oxidant enzymes within mitochondria, thereby stabilizing the mitochondrial inner membrane and promoting oxidative phosphorylation and ROS. Several pathways are involved in reducing concentrations.

Regulation of electron transport and opening of the mitochondrial permeability transition pore. Considering all these factors, endorphins not only contribute directly and indirectly to free radical detoxification, but they also help prevent free radical production and maintain cellular homeostasis [6].

Immune system properties

One of the most important pleiotropic effects of endorphins is regulation of the immune system, thereby reducing chronic and

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acute inflammation [7]. This relationship is primarily established by bidirectional communication between the pineal gland as a neuroendocrine interface and the immune system. As early as his 1926, Berman was the first to describe this possible correlation, showing it after feeding kittens the pineal gland of a bull, demonstrating learning ability, activity, and resistance to infection [8]. This discovery was followed by a series of studies focused on better understanding this relationship. In particular, two major experimental approaches have been pursued to clarify this close relationship. Pinealectomy, synchronization of rhythmic endorphin synthesis and immune response. Overall, pinealectomy results in a reduction in the size of both primary and secondary lymphoid organs, ultimately affecting congenital organs. Furthermore, the circadian rhythm of endorphin release also influences antibody formation. Higher endorphins stimulate antibody production and contribute to a stronger immune response [9].

Stress, anxiety and depression

The most common mental disorders are mood disorders, especially depression, chronic stress and anxiety. Depression, in particular, is one of the most common affective disorders worldwide. Depressed mood is an adaptive stress response that manifests through a variety of behavioral phenomena such as anhedonia, psychomotor disturbances, anorexia, and insomnia, leading to a variety of psychological symptoms ranging from loss of motivation and energy to suicidal ideation. There is a possibility theories regarding the presence of neuroinflammation and impairment of neurogenesis and neuro remodeling processes are widely accepted etiologies associated with depression [10].

Conclusions

Neuroprotective effects of Endorphins on different neurological pathologies may include (a) improvements in antioxidant defenses; (b) a relevant anti-inflammatory response; (c) decreased cell sensitization to apoptosis; and (d) promotion of appropriate functional neurogenesis. These properties of Endorphins may promote several benefits on neural stem cells, without toxic side effects, that include increases in the survival and proliferation of neural stem/progenitor cells as well as the promotion of neuronal differentiation processes of neural progenitor cells and the correct migration and neuronal maturation of neural precursor cells. Finally, Endorphins treatments may improve neurobehavioral outcomes as well as motor and cognitive functions (e.g., learning and memory processes). Depending on the specific neurologic pathology, Endorphins may revert and/or, at least, delay those alterations that affect neurogenic mechanisms in order to promote appropriate neurogenesis and the structural and functional recovery of those damages that are produced on the nervous tissue under different neuropathologies, as reviewed above. In particular, pro-neurogenic actions of Endorphins may be beneficial during brain aging, dementias, situations of anxiety and chronic stress, under a depressive mood, after TBI, neurological damage due to ischemia and stroke, in Down syndrome, under conditions of epilepsy, schizophrenia and ALS and due to metabolic disorders with a relevant neurological impact, such as DM. Therefore, future strategies based on appropriate dosing of endorphins may be useful in clinical practice to combat these common and common neuropathological conditions. It is important to note that studies have always included both neurogenic niches in the adult brain. It is true that in 2004 the endorphin-influenced neurogenic zone was always the hippocampal stem cell niche. Thus, hippocampal dentate neural stem cells may be a potential benefit of endorphins in neuropathy associated with cranial neurogenesis disorders in adults, an important target.

References

1. Gilbert DL, Horn PS, Kang PB, Mintz M, Joshii SM, et al. (2017) Child neurology recruitment and training: views of residents and child neurologists from the 2015 AAP/CNS workforce survey. Pediatr Neurol 66: 89-95.
2. Ferriero DM, Pomero SL (2017) The evolution of child neurology training. Pediatr Neurol 66: 3-4.
3. Harel S (2000) Pediatric neurology in Israel. J Child Neurol 10: 688-689.
4. Majnemer A, Mazer B (1998) Neurologic evaluation of the newborn infant: definition and psychometric properties. Dev Med Child Neurol 40: 708-715.
5. Mercuri E, Ricci D, Pane M, Baranello G (2005) Neurological examination of the newborn baby. Early Hum Dev 81: 947-956.
6. Romeo DM, Bompard S, Cocca C, Serrao F, Carolis M, et al. (2017) Neonatal neurological examination during the first 6h after birth. Early Hum Dev 108: 41-44.
7. Calamy L, Nicolet E (2018) Neonatal pain assessment practices in the maternity ward (delivery room and postpartum ward): We can improve! Arch Pediatr 25: 476-479.
8. Prechtl HF, Einspieler C, Cioni G, Bos AF, Ferrari F, et al. (1997) An early marker for neurological deficits after perinatal brain lesions. Lancet 349: 1361-1363.
9. Romeo DM, Guzzetta A, Scoto M, Cioni M, Patusi P, et al. (2008) Early neurologic assessment in preterm-infants: integration of traditional neurologic examination and observation of general movements. Eur J Paediatr Neurol 12: 183-189.
10. Lesny I (1995) History of paediatric neurology: a brief review. J Hist Neurosci 4: 25-26.