Betaendorphins: Immune-Stimulatory Activity

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Abstract

Endorphins are endogenous morphine produced in pituitary gland response to stress. There are three types of endorphins beta endorphins, enkephalins, and dynorphins binds with mu, kappa, and delta receptors situated on nervous system and immune cells. Betaendorphins are an abundant endorphins, has immune stimulatory activity by activation of immune cells and inhibition of chronic psychological stress induced NF-KB key transcription factor involved in immune modulation by release of pro-inflammatory cytokines. This article highlights about the basic research findings of betaendorphins and its immune-stimulatory activity.

Keywords: HPA-axis; Immune Modulation; Neuropeptides; Cortisol; NF-KB; IL-1; IL-6; COX-2; TNF-α

Introduction

Endorphins are endogenous morphine produced in pituitary gland response to stress and pain. There are three types of endorphins beta endorphins, enkephalins, and dynorphins binds with mu, kappa and delta receptors situated on nervous system and immune cells. Betaendorphins are an abundant endorphins, more potent than morphine, synthesized and stored in the anterior pituitary gland, precursor of POMC (Proopiomelanocortin). During stress the endorphin receptors are increased and bind abruptly with endorphins.

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Endorphin receptors are situated on most innate and adaptive immune cells. Binding of betaendorphins to the mu receptors situated on innate and adaptive immune cells such as neutrophils, macrophages, dendritic cells, natural killer cells, T cells, B cells, and mast cells, results in activation of immune cells (immune-stimulatory activity), produce opsonin, granzyme-B, IFN-γ and antibodies involved in anti-inflammatory activity, antiviral activity, antibacterial activity and antitumor activity [1-20].

Beta-endorphins inhibits chronic psychological stress induced activation of sympathetic nervous system activity, activation of parasympathetic nervous system activity of autonomic nervous system (ANS) results in activation of IL-2, IFN-Υ immunestimulatory cytokines.

Beta-endorphins inhibits chronic psychological stress induced activation of NF-KB a key transcription factor induce inflammatory mediators by inhibiting HPA-axis mediated release of cortisol, ACTH and noradrenaline neuropeptides through autonomic nervous system results in activation of inflammatory mediators such as IL-1β, IL-6, TNF-α and COX-2 which activates NF-KB a key transcription factor involved in chronic inflammation induced immune modulation by releasing pro-inflammatory cytokines such as IL-4, IL-5, IL-10, IL-13, IL-17 and TGF-β [22,24,26].

Beta-endorphins inhibits chronic psychological stress induced activation of NF-KB a key transcription factor involved in chronic inflammation and tumor progression, which antagonize P53 tumor suppressor gene, a guardian of the genome. mutated in more than 50% of all cancers involved in immune surveillance by inflammatory mediators such as NO (nitric oxide), ROS (Reactive oxygen species), and RNS (Reactive nitrogen species) free radicals, AID (Activation induced cytidine deaminase) enzyme expressed by NF-KB transcription factor [20-27].

Endorphins are produced during yoga, pranayama, mindful meditation, intense physical exercise, Love, Tender, care, acupuncture, music therapy, pranic healing, sympathy and empathy in caring the patient [5-10].

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Conclusion and Future Perspective

Beta-endorphins are an abundant endorphins synthesized and stored in the anterior pituitary gland. It has got immune-stimulatory activity and inhibits chronic psychological stress induced activation of NF-KB a key transcription factor mediated immune modulation. Beta endorphins can be used in holistic preventive and therapeutic applications in treatment of various diseases such as cancer and infectious diseases without adverse effects and inexpensive. Thorough understanding of beta-endorphins, mechanism of action and it’s immune-stimulatory activity, dose dependent duration of action, prognosis related to disease helpful for future therapeutic applications.

Bibliography


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