

Role of Kallidin in the Bioactive Peptides in Physiological Processes

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INTRODUCTION

Kallidin is a bioactive peptide that belongs to the kinin family of peptides. It is generated through the enzymatic cleavage of high-molecular-weight kininogen (HMWK) by the enzyme kallikrein. Kallidin, along with its derivative bradykinin, plays a significant role in various physiological processes. In this article, we delve into the functions and implications of kallidin, highlighting its importance in vascular regulation, inflammation, and potential therapeutic applications.

DESCRIPTION

Kallidin is formed by the cleavage of HMWK by kallikrein, an enzyme present in various tissues and body fluids. This cleavage generates a 10-amino acid peptide known as Lys-bradykinin or kallidin. Kallidin is then rapidly metabolized by the enzyme kininase I, also known as angiotensin-converting enzyme, into bradykinin, a closely related peptide with similar biological functions. Kallidin and bradykinin are potent vasodilators, meaning they cause the relaxation and widening of blood vessels. They achieve this by binding to specific receptors, called bradykinin B1 and B2 receptors, present on the smooth muscle cells of blood vessels. Activation of these receptors leads to the production of nitric oxide and other vasodilatory substances, resulting in increased blood flow and decreased vascular resistance. This vasodilatory effect of kallidin and bradykinin contributes to the regulation of blood pressure and the maintenance of cardiovascular homeostasis. Kallidin and bradykinin are involved in the inflammatory response and pain sensation. They promote the release of pro-inflammatory molecules, such as cytokines and chemokines, from immune cells, thereby initiating and amplifying the inflammatory cascade. Additionally, they enhance the permeability of blood vessels, allowing immune cells to migrate to the site of inflammation. This increased vascular permeability and recruitment of immune cells contribute to the characteristic signs of inflammation, including redness, swelling, and pain.

The biological activities of kallidin and bradykinin have sparked interest in their therapeutic potential. Here are a few areas where kallidin-related peptides have been investigated. Hypertension and Cardiovascular Disorders: The vasodilatory properties of kallidin and bradykinin make them potential targets for the treatment of hypertension and other cardiovascular disorders. Research is focused on developing agonists or modulators of bradykinin receptors to enhance vasodilation and reduce blood pressure. Pain Management, Kallidin and bradykinin are implicated in pain sensation due to their ability to stimulate nociceptors (pain receptors). Targeting the bradykinin receptors or enzymes involved in the metabolism of these peptides may offer new avenues for pain management.

Inflammatory Disorders, Modulating the effects of kallidin and bradykinin in inflammatory processes holds promise for the treatment of inflammatory disorders, such as rheumatoid arthritis and inflammatory bowel disease. Developing selective agonists or antagonists for bradykinin receptors may help control inflammation and limit tissue damage. Wound Healing and Tissue Regeneration, Kallidin and bradykinin are involved in tissue repair and wound healing processes. Their ability to promote vasodilation and enhance angiogenesis (formation of new blood vessels) may have applications in regenerative medicine and tissue engineering [1-4].

CONCLUSION

Overall, kallidin is a crucial player in the regulation of various physiological processes, including blood pressure, inflammation, pain sensation, smooth muscle activity, and hormone secretion. Dysregulation of the kallidin system has been implicated in various diseases, including hypertension, inflammatory conditions, and pain disorders. Therefore, understanding the role of kallidin in these processes is essential for developing targeted therapeutic interventions.

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CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

REFERENCES

- Proud D, Kaplan AP (1988) Kinin formation: Mechanisms and role in inflammatory disorders. Annu Rev Immunol. 6(1): 49-83.
- 2. Reynolds CJ, Togias A, Proud D (1999) Airway neural re-

sponses to kinins tachyphylaxis and role of receptor subtypes. Am J Respir Crit Care Med. 159(2): 431-438.

- 3. Davis AJ, Perkins MN (1994) Induction of BI receptors In vivo in a model of persistent inflammatory mechanical hyperalgesia in the rat. Neuropharmacol. 33(1): 127-133.
- 4. Piek T, Gobbo M, Mantel P, Rocchi R, Kramer JV (2009) B2-kininergic action of linear and cyclic tryptophan6-and tyrosine6-kallidin. Life Sci. 59(25-26): PL391-PL397.