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Epinephrine also acts on beta cells and insulin secretion

Epinephrine and insulin

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Abstract

In a recent review on neurotransmitter modulation of insulin secretion, the impact of epinephrine was omitted, despite its significant influence. Epinephrine primarily inhibits insulin release *via* alpha-adrenergic receptors, attenuating the response to insulin secretion stimulators. This effect is mediated through the activation of K+ channels, inducing membrane hyperpolarization in beta cells.

Key Words: Epinephrine; insulin; islets; glucose; human

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Core Tip: Among the neurotransmitters acting on the secretion of insulin, the role of epinephrine may be overlooked. Epinephrine primarily inhibits insulin release through alpha-adrenergic receptors, dampening the response to insulin secretion stimulators.

TO THE EDITOR

We have read with interest the concise review by Kong *et al* [1] on the regulation of insulin secretion by neurotransmitters. While the authors extensively cover the role of norepinephrine (NEPI), they omit discussion on epinephrine (EPI). Both EPI and NEPI, classified as neurotransmitters and hormones, undergo synthesis and release in the central and peripheral nervous system and the adrenal medulla [2]. Although NEPI primarily serves as a neurotransmitter, EPI, while acting as a hormone, should not be disregarded for its neurotransmitter functions. Thus, the impact of EPI closely parallels that of NEPI, albeit with heightened peripheral effects [2].

EPI may stimulate insulin release through beta-adrenergic receptor activation, involving adenylate cyclase (AC), cyclic adenosine monophosphate (cAMP) generation, and the CREB pathway [3]. However, its predominant impact, mediated by alpha-adrenergic receptor activation, is to inhibit insulin secretion through the protein kinase A (PKA)

signaling pathway. This inhibitory effect is substantial, effectively tempering the secretory response to insulin's most potent stimulators [4]. EPI achieves this inhibition by activating K+ channels, leading to subsequent hyperpolarization of pancreatic beta cells' membrane [5, 6].

This brief exposition on the action of EPI on insulin secretion complements the excellent review of neurotransmitters' effects on insulin secretion.

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