



Epinephrine also acts on beta cells and insulin secretion

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Abstract

In a recent review examining neurotransmitter modulation of insulin secretion, the significant impact of epinephrine was not addressed. Its primary action involves inhibiting insulin release *via* alpha-adrenergic receptors, thereby reducing the response to insulin secretion stimulators, through the activation of K⁺ channels and resulting in membrane hyperpolarization in beta cells.

Key Words: Epinephrine; Insulin; Islets; Glucose; Human

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Core Tip: Among the neurotransmitters influencing insulin secretion, the role of epinephrine (EPI) might be underestimated. EPI mainly inhibits insulin release through alpha-adrenergic receptors, thereby attenuating the response to insulin secretion stimulators.

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TO THE EDITOR

We have reviewed with interest the concise examination by Kong *et al*[1] of neurotransmitter influence on insulin secretion. While the authors extensively cover norepinephrine (NEPI), the role of epinephrine (EPI) is overlooked. Both EPI and NEPI, acting as neurotransmitters and hormones, are synthesized and released in the central

and peripheral nervous systems and the adrenal medulla[2]. Despite NEPI's primary role as a neurotransmitter, the significance of EPI, which also functions as a hormone, should not be disregarded for its neurotransmitter functions. Hence, EPI's impact closely parallels that of NEPI, though with more pronounced peripheral effects[2].

EPI can prompt insulin release *via* beta-adrenergic receptor activation, involving adenylate cyclase, cAMP generation, and the cAMP Response Element-Binding Protein pathway[3]. However, its primary effect, mediated by alpha-adrenergic receptor activation, inhibits insulin secretion through the Protein kinase A pathway. This inhibition significantly moderates the response to insulin's strongest stimulants[4]. EPI achieves this by activating K⁺ channels, leading to hyperpolarization of pancreatic beta cell membranes[5,6].

The above concise overview of EPI's impact on insulin secretion complements the excellent and comprehensive review of neurotransmitter effects on insulin secretion[1].

FOOTNOTES

Author contributions: Zabuliene L and Ilias I researched for this work; Zabuliene L and Ilias I wrote the manuscript. Both authors agree to this publication.

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