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제 목 INFLUENCE OF GLUCOCORTICOIDS ON NICOTINIC AND MUSCARINIC STIMULATION-INDUCED CATECHOL-AMINE SECRETION FROM THE RAT ADRENAL GLAND

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내 용

The influence of glucocorticoids on the secretory responses of catecholamines (CA) evoked by acetylcholine (ACh), DMPP, McN-A-343, excess K^+ and Bay-K-8644 from the isolated perfused rat adrenal gland and to clarify the mechanism of its action.

The perfusion of the synthetic glucocorticoid dexamethasone (10-100 uM) into an adrenal vein for 20min produced relatively a dose-dependent inhibition in CA secretion evoked by ACh (5.32mM), excess K^+ (56mM), DMPP (a selective nicotinic receptor agonist, 100uM for 2min), McN-A-343 (a muscarinic receptor agonist, 100uM for 4min), Bay-K-8644 (a calcium channel activator, 10 uM for 4min) and cyclopiazonic acid (a releaser of intracellular Ca^{2+}, 10uM for 4min).

Similarly, the preperfusion of hydrocortisone (30uM) for 20min also depressed significantly the secretory responses of CA evoked by nicotinic and muscarinic receptor stimulation as well as membrane-depolarization, Ca^{2+} channel activation and the release of intracellular Ca^{2+}. Furthermore, even in the presence of betamethasone (30uM), CA secretion evoked by ACh, excess K^+, DMPP and McN-A-343 was also markedly inhibited.

These experimental results taken together suggest that glucocorticoids cause the marked inhibition of CA secretion evoked by both cholinergic nicotinic and muscarinic receptor stimulation from the perfused rat adrenal gland, indicating strongly that this inhibitory effect may be mediated by inhibiting influx of extracellular calcium as well as the release of intracellular calcium in the rat adrenomedullary chromaffin cells.