

제 목 INFLUENCE OF 17- $\alpha$ -ESTRADIOL ON CATECHOLAMINE  
SECRETION FROM THE PERFUSED RAT ADRENAL GLAND

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내 용

It has been known that adrenal corticosteroids influence the expression of adrenomedullary catecholamine-synthetizing enzymes and also suppress the emission of axonal-like processes in cultured chromaffin cells. In the present study, it was attempted to investigate the effect of 17 $\alpha$ -estradiol on catecholamine(CA) secretion evoked by acetylcholine(ACh), DMPP, McN-A-343, excess K<sup>+</sup> and Bay-K-8644 from the isolated perfused rat adrenal gland.

The perfusion of 17 $\alpha$ -estradiol (10<sup>-6</sup>-10<sup>-4</sup>M) into an adrenal vein for 20min produced relatively dose-dependent inhibition in CA secretion evoked by ACh (5.5 x 10<sup>-3</sup>M), DMPP (10<sup>-4</sup>M for 2min), McN-A-343 (10<sup>-4</sup>M for 4min) and Bay-K-8644 (10<sup>-7</sup>M for 4min), while did not affect the CA secretory effect of high K<sup>+</sup> (5.6 x 10<sup>-2</sup>M). Also, in the presence of 17 $\beta$ -estradiol, CA secretion of ACh, DMPP and McN-A-343 without any effect on excess K<sup>+</sup>-evoked CA secretion. However, in adrenal glands preloaded with 17 $\alpha$ -estradiol (10<sup>-5</sup>M) plus tamoxifen (10<sup>-5</sup>M), which is known to be a selective antagonist of estrogen receptors (for 20min), CA secretory responses evoked by ACh, DMPP and McN-A-343 were considerably recovered as compared to that of 17 $\alpha$ -estradiol only, but excess K<sup>+</sup>-induced CA secretion was not affected.

These results suggest that 17 $\alpha$ -estradiol causes the marked inhibition of CA secretion evoked by cholinergic receptor stimulation, but not that by excess K<sup>+</sup>, indicating strongly that this effect may be mediated by inhibiting influx of extracellular calcium into the rat adrenomedullary chromaffin cells through the activation of inhibitory estrogen receptors, and it also plays a modulatory role in regulating CA secretion.