15

Effects of ACTH and other POMC-derived peptides on steroid secretion by isolated human adrenal cells. Eggens, U., Bähr, V., Li, C.H.*, Oelkers, W. Dept. of Internal Medicine, Klinikum Steglitz, Freie Universität Berlin and Hormone Research Laboratories, University of California, San Francisco*

Matsuoka et al. (J.Clin.Invest. 68, 752, 1981) showed an aldosterone-stimulating effect of ß-LPH in rat adrenal cells. We tested the effect of ACTH, extracted and synthetic ß-LPH, synthetic γ2-MSH and ß-endorphin on steroid production by isolated human adrenal cells (n= 10, cadaver kidney donors) and aldosteronoma cells (n=3). 10^{-9} M ACTH stimulated cortisol 6-fold, aldosterone (A) and 18-OH-B 2-3-fold (threshold: 10^{-11} M). 10^{-7} M extracted ß-LPH stimulated the three steroids to the same extent (threshold: 10^{-9} M). Up to 10^{-7} M of synthetic ß-LPH was without any effect. ß-endorphin and γ2-MSH had minor effects on cortisol and A at 10^{-6} M. ACTH stimulated A in 2/3 aldosteronoma preparations at 10^{-9} M, while γ2-MSH and ß-endorphin had minor effects between 10^{-8} and 10^{-6} M.
Conclusions: Stimulation of steroids by extracted ß-LPH seems to be due to traces of ACTH. ß-LPH itself has no effect. γ2-MSH and ß-endorphin are weak stimulators of A in normal and aldosteronoma cells at high (unphysiological) concentrations.

16

Effect of Atrial Natriuretic Factor (ANF) on aldosterone secretion: in vitro and in vivo studies

F. Mantero, M. Boscaro, S. Rocco, F. Fallo, D. D'Agostino
Istituto di Semeiotica Medica Università, Via Ospedale, 105 -35100 PADOVA-

It has recently been demonstrated that atrial natriuretic factor (ANF) is a potent inhibitor of aldosterone secretion. To further characterize this aspect, we have studied the effect of a synthetic analogue, rat Atriopeptin II (AP II) in vitro, both in a perfusion and in a static system of isolated rat glomerulosa cells. The in vivo effect of α-h ANF has also been investigated in 4 normal volunteers. In vitro: in the perfusion system cells were stimulated by K^+ (5 mEq/l), ACTH (26 pg/ml), Angiotensin II (10^{-7}M), Serotonin (10^{-7}M) and Ca ionophore A23187 ($2.5x10^{-6}$M); the modifications of the aldosterone response after adding AP II(10^{-7}M) to the system were then studied. Similar experiments were performed in the static incubation system, where the effect of scalar amounts of secretagogues or AP II on aldosterone release were measured. The results showed that AP II was able of significantly inhibiting either baseline and stimulated aldosterone secretion in both systems. In vivo: the bolus i.v. administration of 50-100 µg of α-h ANF decreased very slightly and not significantly both plasma aldosterone and renin activity; minimal changes on blood pressure, diuresis and natriuresis were recorded. Conclusions: this study confirms the potent in vitro inhibitory properties of ANF on aldosterone secretion, and adds serotonin and Ca^{++}ionophore to the list of secretagogues affected by ANF. This property is not easily demonstrable in vivo, possibly due to low doses, short-term administration or the intervention of compensatory mechanisms.