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SHORT COMMUNICATION

A STUDY ON THE INTERACTION OF ACTH AND PROSTAGLANDIN $F_{2\alpha}$

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The discovery of antagonism between prostaglandins (PG) of the E and F type in several tissues was followed by the postulation of an antagonism between PGE-mediated effects of trophic hormones and $PGF_{2\alpha}$ in endocrine tissues [1]. In relation to steroid producing cells such an antagonism was observed in human granulosa cells [2]. Few observations have been reported on the adrenocortical effects of $PGF_{2\alpha}$ which exhibited a moderate steroid stimulating effect in the rat's decapsulated adrenal [3] but failed to affect steroid production by bovine adrenocortical slices [4]. ACTH-stimulated release of $PGF_{2\alpha}$ by isolated feline adrenocortical cells has also been reported [5]. The possibility of an antagonism between ACTH and $PGF_{2\alpha}$ was indicated by the enhancement of ACTH-stimulated corticosterone production of the decapsulated rat adrenal gland after treatment with PG synthetase inhibitors [6]. The present experiments were designed to study the interaction of ACTH and $PGF_{2\alpha}$ in isolated adrenocortical zones.

Male Sprague–Dawley (CFY) rats, weighing 130–155 g, were kept on a semisynthetic diet (Na^+:165, K^+:145 meqv./kg). The animals were transauricularly hypophysectomized 16–20 h before decapitation. The adrenals were separated into capsular (fibrous capsule + zona glomerulosa) and decapsulated tissue (z. fasciculata reticularis + medulla) according to Giroud *et al.* [7]. The technique of incubation as well as the details of aldosterone radioimmunoassay and corticosterone competitive protein binding assay have been previously described [6]. Synthetic $PGF_{2\alpha}$ (Enzaprost F, Chinoin) was added to the medium both in the 30-min preincubation and the 60-min incubation periods, ACTH (Synacthen, Ciba) was added in the 60-min incubation period only.

Maximal production rates of aldosterone and corticosterone by the capsular gland were achieved by 500 ng/ml ACTH ($= 1.7 \times 10^{-7}$M). $PGF_{2\alpha}$ in the range of $10^{-7} - 10^{-5}$M failed to exert any influence on steroid production rate. The extreme concentration of 10^{-4}M evoked a further increase in aldosterone but had no effect on capsular corticosterone production rate (Fig. 1).

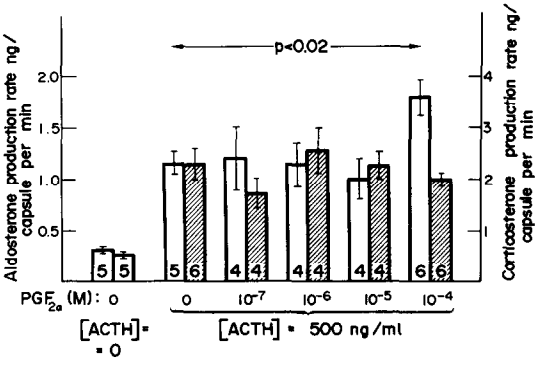

Fig. 1. Effect of ACTH and $PGF_{2\alpha}$ on capsular aldosterone (empty bars) and corticosterone (hatched bars) production rate (ng per capsule per min.). The figures in the columns indicate the number of experiments.

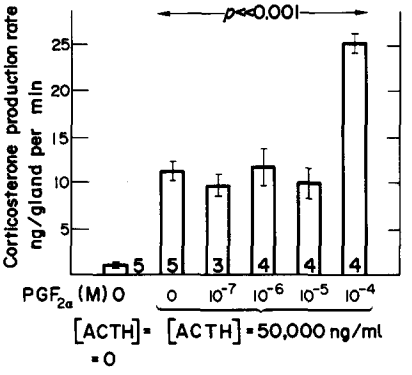

Fig. 2. Effect of ACTH and $PGF_{2\alpha}$ on corticosterone production by the decapsulated gland (ng per gland per min).

Production rate of corticosterone by the decapsulated gland was examined in the presence of 5,000 ng/ml ACTH which evoked half the stimulation achieved by 50,000 ng/ml, the highest concentration applied by us on this preparation. $PGF_{2\alpha}$ did not influence corticosterone output in the range of $10^{-7} - 10^{-5}$M while 10^{-4}M increased it by a factor of two (Fig. 2).

The present results are not compatible with the assumption of an antagonism between ACTH and $PGF_{2\alpha}$ in the adrenal cortex. Further experiments are required to ascertain whether stimulation of steroid production rate by the extreme concentration (10^{-4}M) of $PGF_{2\alpha}$ has any physiological meaning.

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