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Control of Secretion of Hypothalamic Hormones

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Data will be presented which indicate that the median eminence region of the hypothalamus is not directly responsible for the synthesis of the gonadotrophin releasing factors. It will be shown that the paraventricular region is specifically involved in the synthesis of FSH-RF, while the suprachiasmatic and the arcuate-ventromedial zones are specifically devoted to the synthesis of LH-RF.

Three experimental conditions will be discussed in which the intrahypothalamic stores of one gonadotrophin releasing factor have been modified without changing those of the other. These observations are not compatible with the hypothesis that one single hypothalamic factor controls the release of both LH and FSH as recently suggested.

In a series of *in vitro* experiments it has been shown that acetylcholine is able to liberate FSH from the anterior pituitary only if fragments of the basal part of the hypothalamus are present in the incubation media. These data have been taken as indicating that acetylcholine stimulates the release of FSH-RF from the incubated hypothalami, and that FSH-RF released under the influence of acetylcholine in turn enhances the secretion of FSH from the incubated pituitaries. It will be suggested that acetylcholine may play a major role in transferring extrahypothalamic influences to the neurons which synthesize the gonadotrophin releasing factors.

The synthetic decapeptide synthesized by Schally and his co-workers is able to release LH and FSH when injected into the carotid artery of the rat. The kinetics of the release of the two hormones under the influence of the decapeptide are quite different, LH being released more promptly than FSH. The activity of the decapeptide may be modulated by changing the levels of sex steroids in the general circulation. Apparently estrogens facilitate the release of LH, while androgens enhance the release of FSH.

Modulating Effects of Prostaglandins on the Release of Hypothalamic Hormones

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The prostaglandins are discussed as mediators of biological events in the hypothalamo-hypophyseal system. This phenomenon was first shown with respect to ACTH-release. A direct effect of PGE_1 was demonstrated by implantation into the medial basal hypothalamus. The effect of TRH on release of TSH *in vitro* from rat hemipituitaries is influenced by PGE_1. PGE_1 stimulates GH-release from bovine anterior pituitary tissue, PGE_1 and PGE_2 increase incorporation of labelled leucine into GH and prolactin. Zor *et al.* have studied cyclic AMP levels in anterior pituitary tissue following incubation with several prostaglandins, in order of potency being $PGE_1 > A_1 > B_1 > F_{1\alpha}$. None of them released LH in the system. Caldwell *et al.* have observed LH release by PGE_2 in a pituitary superfusion system. Both PGE_2 and $F_{2\alpha}$ stimulate LH release. Harms *et al.* have shown a neurotransmitter-like effect of PGE_2 after injection into the third ventricle PGE_2 increases plasma LH, while PGE_1 elevates prolactin. The stimulatory effects of prostagladins are antagonized or blocked by inhibitors of prostaglandin synthesis, *e. g.* 7-oxa-13-prostynoic acid. The *in vitro* effect of LH-RH on gonadotrophin release is modulated by prostaglandins. The evaluation of prostaglandin effects on hormone release