

is complicated by the fact that prostaglandins also act directly at the end organ level, *e.g.* adrenal or corpus luteum *in vitro*. In conclusion, prostaglan-

dins act at the hypothalamic, pituitary and end organ level. A primary or predominant site of action cannot be defined at present.

### Cellular Regulation of the Adenohypophyseal Gonadotropic Function

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The data now available on the cellular mechanisms by which the secretion of FSH and LH is regulated have been obtained in part using partially purified preparations of hypothalamic origin and in part by means of the synthetic decapeptide, LH-RH, described by Schally *et al.* (1971). LH-RH acts on the release of both hormones, LH and FSH. Many experimental results, but not all, are in favour of the hypothesis that cyclic AMP is an intermediate in the action of hypothalamic hormone(s) regulating the secretion of LH and FSH. This would imply first the binding of the hormone to a specific mem-

brane cell receptor with the subsequent activation of adenylyl cyclase. Some data have been obtained on the physico-chemical aspects of the binding of LH-RH to the anterior pituitary cells or cell membranes, but although it has been stated that LH-RH increases the content of cAMP in the tissue, no one has been able to demonstrate the activation of adenylyl cyclase in this system. How cAMP then promotes release of gonadotropins is still unclear. cAMP activates a protein kinase which participates in the phosphorylation processes. Phosphorylation of microtubules is possibly an important event in the release mechanism. It is also postulated that cAMP acts either by altering the permeability of the cellular membranes to  $\text{Ca}^{2+}$  or by affecting the binding of  $\text{Ca}^{2+}$  to membrane proteins.  $\text{Ca}^{2+}$  intervenes in many intracellular mechanisms and is essential for the release process.

### Hypothalamo-Pituitary-Testicular Feedback Mechanism During Mammalian Sexual Maturation

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The pubertal changes in the mammalian life are brought about by hormones, either secreted for the first time or secreted in much greater quantities than previously. The attainment of sexual maturation is however a complex process which requires maturation and interaction not only of gonads and the reproductive tract, but also of the pituitary and importantly of the neuro-endocrine mechanisms which ultimately control gonadotropin secretion. Presumably there is a marked change in the sensitivity of the hypothalamic-pituitary negative feedback centres to gonadal steroids during sexual maturation. With the shifting of the sensitivity set-points, pubertal developments may be viewed as a

continuum lasting several days in the rats or several years in man, secondary sexual characteristics beginning only when a critical level of steadily increasing gonadotropin-releasing hormone is attained. Apparently the mechanism for hypothalamic regulation of pituitary gonadotropic activity and release of the releasing hormones are relatively inactive or inhibited during immature stages. A certain degree of physiological maturation of the central nervous system is evidently required before the pre-pubertal inhibition is released and the hypothalamo-pituitary mechanism becomes active.

In the current study the sensitivity of the pituitary-gonadal responses to exogenous synthetic LH-RH was evaluated in sexually immature and mature male rats. The conditioning influence of prior treatment of gonadotropins and sex steroid hormones on the feedback relationship in the pituitary-gonadal axis was also examined.

The decapeptide was administered i.v. to the animals by infusion for a 4 h period and immediately after blood was collected. LH, FSH, testosterone and 5 $\alpha$ -dihydrotestosterone were estimated by radioimmunoassay techniques. Infusion of the decapeptide induced a considerable rise in serum LH and FSH in both mature and immature animals.



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