

Introduction
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It has long been discovered that gonadal steroid synthesis and release are controlled by the pituitary gonadotropins, LH and FSH (Davis et al., 1978). Testosterone secretion in males is mainly controlled by LH, while FSH has a minor role in this respect (Theodore et al., 1979).

Gonadotropins secretion by the anterior pituitary is stimulated by the hypothalamic GnRH (Silverman et al., 1979). On the other hand, gonadal steroids cause feedback inhibition of gonadotropine secretion by acting both directly on the pituitary (Gual et al., 1975) and indirectly on the hypothalamus inhibiting the secretion of GnRH (Rudenstein et al., 1979; Wieland, et al., 1986).

Many studies suggested that opioid peptides play an important role in the control of gonadotropine secretion (Theodore et al., 1979; Ellingboe et al., 1982; Spencer & Whitehead, 1986). Bhanot & Wilkinson (1984) suggested that opioid peptides mediate the inhibitory effect of gonadal steroids on the hypothalamic secretion of GnRH, but do not affect the pituitary response to GnRH or the

inhibitory effect of testosterone on the anterior pituitary. The inhibitory role of opioid peptides in the control of gonadotropine secretion is evidenced by three lines of evidences : First, exogenous opiate, such as morphine, inhibit LH secretion, and this effect is reversed by concurrent administration of the specific opiate antagonist, naloxone (Bruni et al., 1977; Cicero et al., 1979). Secondly, naloxone given alone increases LH secretion (Blank et al., 1980). Thirdly, endogenous opioid peptides such as met-enkephalin and β -endorphin inhibit LH secretion (Burni et al., 1977; Blank et al., 1980).

The presence of gonadal steroids is important for the opioid peptides to act on the hypothalamus. This is proved by acute reduction of the inhibitory effects of both endogenous and exogenous opiates on LH and FSH secretion following gonadectomy (Petraglia et AL., 1984), also gonadectomy results in reduction of the ability of naloxone to stimulate serum LH and FSH (Bhanot & Wilkinson, 1984).

However, reported results are not always consistant with this simple concept of endogenous opioids mediating

negative feedback effects of gonadal steroids (Spencer & Whitehead, 1986). For example, opiates, have been shown to modify LH secretion in adult castrated rats without testosterone replacement (Cicero et al., 1980; Motta & Martini, 1982). Also, in intact female or steroid-treated ovariectomized rats, naloxone can stimulate LH secretion when steroids are exerting positive feedback effects on gonadotropine secretion (Gabriel et al., 1983).

The present work aims at studying the effects of stimulation and blockage of opioid receptors on the feedback control of gonadotropin secretion by testosterone in male rats.

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