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Effects of short-term stimulation of serotoninergic pathways on the pulsatile secretion of luteinizing hormone in the absence and presence of acute opiate-receptor blockage

R. J. Urban and J. D. Veldhuis
Department of Internal Medicine, University of Virginia School of Medicine, Charlottesville 22908.

To investigate the role of the serotoninergic system in regulating pulsatile gonadotropin secretion in man, we tested the influences of a novel selective serotonin re-uptake inhibitor (fluoxetine HCl) on episodic LH release in men. Spontaneous LH pulsatility was assessed by computerized analysis of serial LH concentrations measured in blood samples withdrawn at 10 min intervals for 24 h. Possible alterations in pituitary responsiveness were tested by administering three consecutive two-hourly intravenous pulses of GnRH (10 micrograms, 10 micrograms, and 100 micrograms). The effects of fluoxetine (20 mg orally three times daily for one wk) were assessed in a double-blind, placebo-controlled design. Compared with the placebo, fluoxetine elicited no changes in 24 h mean serum LH concentrations, LH pulse characteristics (Cluster analysis), or LH secretion and clearance parameters assessed in response to exogenous GnRH administration (deconvolution analysis) in the presence of normal opiatergic tone (nine healthy young men), and during acute blockade of the opiatergic system (seven young men treated with the mu-opiate receptor antagonist, naltrexone). In summary, a selective enhancer of serotoninergic activity (fluoxetine HCl) does not affect pulsatile LH release basally or in the presence of acute inhibitory opiatergic tone. Since this probe does modify prolactin secretion in man, we conclude that stimulation of the serotoninergic system by this selective neuroendocrine probe shows no demonstrable coupling between the serotoninergic and the opiatergic pathways that modulate pulsatile LH release in man.

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