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Inhibition of Spermatogenesis and Steroidogenesis During Long-Term Treatment with hCG in the Rat

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The effects of chronic treatment with hCG (100 I.U.s.c. every second day) on testicular morphology, LH receptor levels and concentration of steroid intermediates of the Δ_4 and Δ_5 biosynthetic pathways were studied in adult rats for periods extending from one to 12 weeks. Treatment with hCG causes a decrease in testis weight, a maximal inhibitory effect being observed after two and four weeks of treatment. At these time intervals, the loss of testis weight is accompanied by degenerative changes in most seminiferous tubules and hypertrophy of Leydig cells. Administration of hCG for one week leads also to an almost complete loss of LH binding sites and to a marked stimulation of the levels of testicular steroids of the Δ_4 and Δ_5 pathways, as well as to an increase in weights of secondary reproductive organs. The initial increment of testicular steroid levels is followed after two weeks of hCG administration by an apparent decrease of 17,20-desmolase activity suggested by a reduction in the levels of androst-5-ene-3β,17β-diol, androstenedione, testosterone, and 5α-dihydrotestosterone and an increase in the concentrations of pregnenolone, 17-OH-pregnenolone, progesterone and 17-OH-progesterone. Plasma and pituitary LH levels are maximally reduced at one and four weeks of treatment, respectively, while plasma and pituitary FSH levels are only slightly reduced after four weeks of hCG administration. The effects of hCG on all of the above-mentioned parameters, except for testicular morphology and testis weight, are completely reversible at the eight and 12 week intervals. This transiency in the effects of hCG is accompanied by a gradual increase in plasma levels of hCG antibodies. The present data show that chronic treatment of adult rats with hCG induces a marked degeneration of the seminiferous tubules and an inhibition of spermatogenesis that accompanies the well-known loss of testicular LH receptors and inhibition of the steroidogenic pathway.

Key words: hCG, spermatogenesis, steroidogenesis

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Testosterone mediates expression of the selenoprotein PHGPx by induction of spermatogenesis and not by direct transcriptional gene activation

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