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Effect of finasteride on human testicular steroidogenesis

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We studied the testicular function and some androgen-mediated events in 22 males (16-30 years of age) with male pattern baldness that was treated with finasteride (10 mg once daily) for 2 years. Patients were evaluated every 3 months. Prostatic volume was determined in six subjects by endorectal ultrasound scans. Serum gonadotropin, prostatespecific antigen (PSA), and sex hormone levels were determined basally and periodically during the treatment period. Fourteen subjects

underwent gonadal stimulation with human chorionic gonadotropin (hCG), and the gonadotropin response to gonadotropin releasing hormone (GnRH) was determined in eight subjects, prior to and after 2 years of therapy. Finasteride treatment resulted in an improvement in the male pattern baldness and prostatic shrinkage that was associated with an increase in serum testosterone levels (17.2 +/- 2.5 vs. 26.3 +/- 1.7 nmol/L) and a decrease in dihydrotestosterone (DHT) levels (1.45 +/- 0.41 vs. 0.38 +/- 0.10 nmol/L), causing a marked increase in that testosterone/DHT ratio. A significant increase in the serum levels of androstenedione (3.67 +/- 0.49 vs. 7.05 +/- 0.70 nmol/L) and estradiol (132 +/- 44 vs. 187 +/- 26 pmol/L) was also noted, whereas androstanediol glucoronide (33.3 +/- 6.4 vs. 10.7 +/- 4.5 pmol) and PSA (1.6 +/- 0.6 vs. 0.4 +/- 0.1 ng/ml) were significantly decreased. No changes in basal or stimulated levels of gonadotropin were observed. There was a significant increase in the testosterone response to hCG during finasteride therapy (delta: 16.7 vs. 35.5 nmol/L) that could be explained, at least in part, by the reduction of testosterone metabolism resulting from the blockage induced by finasteride. The decrease in the androstenedione to testosterone and estrone to estradiol ratios observed after hCG treatment, however, strongly suggests increased activity of the 17-ketosteroid reductase enzyme and an improvement of the testicular capacity for testosterone production.

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