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JOURNAL ARTICLE

Hypoprolactinemia does not prevent restoration of normal spermatogenesis in gonadotropin-suppressed, testosterone-replaced rats

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We have previously shown that suppression of gonadotropins and spermatogenesis can be produced in rats by immunization against gonadotropin releasing hormone (GnRH). Administration of testosterone (T) alone is effective in restoring complete spermatogenesis in these rats, although it is not effective in doing so in chronically treated hypophysectomized rats. This suggests that a pituitary factor(s) other than luteinizing hormone (LH) and follicle-stimulating hormone (FSH) is required to restore normal spermatogenesis. The studies described herein test the hypothesis that prolactin (PRL) is the additional requirement for complete restoration of spermatogenesis. Twenty rats were immunized against GnRH, and four groups of five each received either 1) 24-cm T-filled Silastic implant (TSI), 2) TSI plus bromocriptine pellet (B), 3) B plus empty Silastic implant (SI), or 4) SI alone. Five nonimmunized rats received SI alone and served as controls. All rats were sacrificed 2 months after treatment. GnRH immunization and B administration suppressed gonadotropins and PRL levels, respectively, and advanced spermatids were not detectable in these rats. Testis weight was suppressed to about 19% of controls. The number of advanced spermatids in control rats was 220 +/- 23 x 10(6). TSI administration restored advanced spermatids to numbers comparable to controls in GnRH-immunized rats whether the rat received B (191 +/- 17 x 106) or not (217 +/- 18 x 10(6)). Additionally, we determined mRNA levels for PRL and FSH beta subunit (FSH beta) in the pituitary by Northern blot and densitometric scanning. The mRNA levels of PRL mirrored serum PRL levels, and the same was true for FSH beta subunits and serum FSH levels. These data show that suppression of PRL has no effect on the ability of T to restore complete spermatogenesis in GnRH-immunized rats. This observation mitigates against the hypothesis that PRL is the pituitary factor required to allow complete restoration of spermatogenesis to occur.

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