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JOURNAL ARTICLE

Effects of hypophysectomy and alterations in spermatogenic function on Leydig cell volume, number, and proliferation in adult rats

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The two major objectives of this study were to determine (i) whether the pituitary is required to maintain Leydig cell number per testis, and (ii) whether alterations in spermatogenic function in the absence of LH can affect Leydig cell volume, number, and 3H-thymidine incorporation in vivo. Four experimental treatments tested the combinations of two factors: (i) the intact pituitary vs hypophysectomy (Hypox); and (ii) arrested vs active spermatogenesis. Subdermal Silastic capsules were used to deliver a low dosage of estradiol in addition to a low dosage of testosterone (TE), which arrested spermatogenesis, or a high dosage of testosterone (HTE) which maintained active spermatogenesis. All four treatments (TE, HTE, Hypox and Hypox-HTE) inhibited LH secretion for 16 weeks. Control rats were sham hypophysectomized. Leydig cell volume per testis and the volume of an average Leydig cell decreased 70-85% (P less than 0.01 vs controls) in all treated rats, whether deprived of LH (TE) or of all pituitary secretions (Hypox), and whether spermatogenesis was arrested (TE, Hypox) or maintained by exogenous testosterone (HTE, Hypox-HTE). This result suggested that LH was the only factor required to maintain Leydig cell volume, since the absence of other pituitary factors or alterations in spermatogenic function could not override or modify the effect of LH deprivation. No significant differences were found in Leydig cell number per testis or the proportion of Leydig cells labeled with 3H-thymidine among control and experimentally treated rats. In contrast to Leydig cell volume, which depended on LH, Leydig cell number and Leydig cell division were maintained at control values in the absence of pituitary factors and spermatogenic function for 16 weeks.

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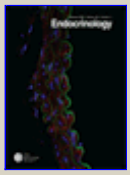

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