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JOURNAL ARTICLE

Pituitary control of proliferation and differentiation of Leydig cells and their putative precursors in immature hypophysectomized rat testis

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The objective of this study was to determine the effects of pituitary hormones (luteinizing hormone [LH], follicle-stimulating hormone [FSH], growth hormone [GH], and prolactin [PRL]) on interstitial cell proliferation and differentiation in the testis of immature hypophysectomized rats. Macrophages, Leydig cells, precursor mesenchymal cells, endothelial lymphatic cells, and myoid cells were studied. Our experimental approach was aimed at determining whether changes in a cellular subpopulation observed after pituitary hormone treatments were the result of division of existing cells in the population, of differentiation of interstitial precursor cells, or both. In this context, it must be stressed that our data reflected the effects of hormones to prevent the decline of cells due to hypophysectomy rather than their recovery. Macrophage proliferation was taken into account because macrophages closely resemble Leydig cells and are known to proliferate after hormonal treatment. A double-labeling procedure (acid phosphatase and anti-bromodeoxyuridine [anti-BUdR]) revealed that LH, FSH, and PRL increased the number of testicular macrophages 105-, 104-, and 103-fold, respectively, in hypophysectomized rats compared to hypophysectomized control animals. BUdR incorporation in testicular macrophages was greater after PRL treatment than after LH and FSH supplementation. In contrast, we were unable to demonstrate any effect of rat GH on the macrophage population. Light microscopic analysis of plastic embedded sections of treated rat testis revealed that LH increased the numbers of Leydig, precursor mesenchymal, and myoid cells 6-, 4-, and 1.3-fold, respectively. LH also stimulated BUdR incorporation into all interstitial cell types. PRL administration increased both the number of Leydig and precursor mesenchymal cells (each 3-fold) but decreased the number of endothelial lymphatic cells (1.5-fold) when compared to the control animals. In contrast, FSH did not increase the number and proliferation of Leydig cells but exerted a slight proliferative effect on the other interstitial cell populations. In GH-treated rats, the number of precursor mesenchymal cells increased two fold above the control rats. GH also exerted slight proliferative effects on both precursor mesenchymal and myoid cells. Immunohistochemical studies of steroidogenic enzymes in the testicular interstitium of treated rats demonstrated the presence of steroidogenic enzymes, not only in Leydig and precursor mesenchymal cells, but also in some (1%-2%) endothelial lymphatic cells and myoid cells. This may indicate that both of these cell types are also constitutively equipped to perform steroidogenesis or that they are precursor cells undergoing

differentiation. Taken together, changes in the number of Leydig cells in our animal model appeared more likely to be dependent on the transformation of precursor cells than on division of preexisting mature Leydig cells.

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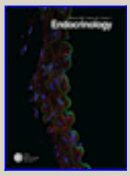

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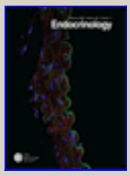

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