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Puberty Is Delayed in Male Growth Hormone Receptor Gene—Disrupted Mice

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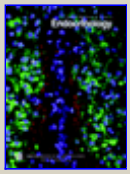
The role of insulin-like growth factor-I (IGF-I) in the initiation of puberty and testicular function is poorly understood. Growth hormone (GH) receptor (R) gene—disrupted mice or GHR gene "knockouts" (GHR-KO) are GH resistant and IGF-I deficient. To assess whether the age of sexual maturation is affected by the absence of IGF-I, various parameters of sexual development including testicular and accessory reproductive organ weights, balanopreputial separation, germ cell development, and intratesticular testosterone levels were determined in normal and GHR-KO mice between the ages of 25 and 60 days. In addition, at 36 days of age, the testosterone response to luteinizing hormone (LH) treatment was assessed in these mice. The results indicate that the balanopreputial separation was delayed 5 days, and a significant increase in the weights of the seminal vesicles (SV) occurred later in GHR-KO mice than in normal animals (between 30 and 35 days and between 35 and 40 days, respectively). Also, the weights of testes and epididymii were significantly reduced in GHR-KO mice. The intratesticular testosterone levels and the testosterone response to LH treatment were attenuated in GHR gene-disrupted mice. Furthermore, elongated spermatids appeared later in the testes of GHR-KO mice than in the testes of normal mice. These results suggest that the absence of IGF-I secretion delays the normal course of sexual maturation in male GHR-KO mice, indicating that IGF-I plays an important role in the initiation of puberty in male mice.

Key words: Sexual development, balanopreputial separation, seminal vesicles, insulin-like growth factor-I, testosterone



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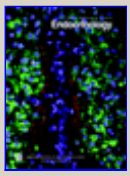

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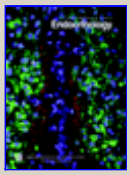

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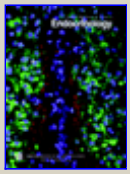

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