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Insulin-Dependent Diabetes Affects Testicular Function by FSH- and LH-Linked Mechanisms

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A study was conducted to form a unified hypothesis regarding the gonadotropin-related mechanisms that underlie alterations in the male reproductive system in individuals with diabetes. Streptozotocin-induced diabetes resulted in reduced fertility, prolificacy, and libido. Testes showed a marked decrease in the number and function of Leydig cells, the latter manifested as changes in the expression of biochemical markers, including the GLUT-3 hexose transporter, c-kit, insulin-like growth factor I (IGF-I), androgen receptors, and overall tyrosine phosphorylation, as assessed by Western blot and immunocytochemical analyses. The expression of c-kit, IGF-I, insulin, and follicle-stimulating hormone (FSH) receptors in the seminiferous tubules was also affected. Serum levels of luteinizing hormone (LH), FSH, and testosterone significantly decreased. There was a significant ($P < .05$) correlation between the serum levels of insulin and FSH. No significant correlation was found between the serum levels of insulin or glucose and LH. On the basis of our results, we conclude that, in insulin-dependent diabetes, 1) Leydig cell function and testosterone production decrease because of the absence of the stimulatory effect of insulin on these cells and an insulin-dependent decrease in FSH, which, in turn, reduces LH levels; and 2) sperm output and fertility are reduced because of a decrease in FSH caused by a reduction in insulin.

Key words: Testes, follicle-stimulating hormone, luteinizing hormone

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