Pharmacological Reports
2011, 63, 130–138
ISSN 1734-1140

Ejaculatory dysfunction in streptozotocin-induced diabetic rats: the role of testosterone

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Abstract:
Hyperglycemic and hypoinsulinemic states caused by diabetes mellitus are usually related to some type of sexual dysfunction, resulting in infertility in humans and experimental models, mostly due to their effects on ejaculatory function. This study aimed to evaluate the possible role of testosterone in the restoration of normal ejaculatory function in diabetic rats. Male Wistar rats were randomly allocated into 3 experimental groups: control, diabetic (streptozotocin), and diabetic with testosterone supplementation (streptozotocin plus testosterone). The following parameters were assessed at the end of the experiment: body weight, circulating testosterone levels, number of spermatozoa ejaculated in the uterus through natural mating, and weight and *in vitro* isometric contractions of the vas deferens. Diabetic rats showed reduced plasma testosterone levels and ejaculatory dysfunction as observed by a lack in the spermatozoa ejaculated into the uterus of receptive females. In these diabetic rats, no difference was observed in the sensitivity of the vas deferens to norepinephrine, with or without the presence of the cocktail (cocaine plus propranolol). In spite of this, an increased sensitivity to methoxamine through the α_1-adrenoceptor was observed. Testosterone supplementation did not restore these parameters to control values. We conclude that, in this experimental model, the lack of testosterone was not directly related to the diabetes-induced ejaculatory dysfunction.

Key words:
diabetes, ejaculation, testosterone, norepinephrine, infertility