AB150. Dietary resistant maltodextrin ameliorates testicular function and spermatogenesis in streptozotocin-induced diabetic rats

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Objective: This study investigated the effect of resistant maltodextrin (RMD) on reproduction in streptozotocin (STZ)-induced type 2 diabetic male rats.
Methods: Forty male rats were induced with diabetes by a single intraperitoneal injection of STZ (50 mg/kg) and nicotinamide (100 mg/kg). Five groups were analyzed in total: normal, Diabetic rats without RMD, diabetic rats with RMD 1.2 g/100 g diet (1×), with RMD 2.4 g/100 g (2×), and with RMD 6.0 g/100 g (5×).
Results: The groups of diabetic rats with the RMD supplement, compared to those without supplement, showed improved plasma glucose control, attenuated insulin resistance and recovery of testosterone level & spermatogenesis stage. The STZ-induced diabetes mellitus (DM) caused a significant reduction in serum testosterone, testis androgen receptor (AR), steroidogenic acute regulatory protein (StAR) and 3β-hydroxysteroid dehydrogenase (3β-HSD) protein, but a statistical recovery in each of these was observed in the 5× group. TUNEL-positive cells were observed in the Diabetic without RMD group and RMD treatment reduced apoptotic germ cells. The expression of Bax/Bcl2 was induced in the diabetic group and also significantly reduced in the 5× group.
Conclusions: Dietary RMD may improve metabolic control in STZ-induced diabetic rats and attenuate hyperglycemia-related impaired male reproduction and testicular function.

Keywords: Diabetes mellitus (DM); resistant maltodextrin (RMD); hypogonadism; spermatogenesis

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AB151. Human tissue kallikrein 1 restores erectile function of streptozotocin-induced diabetic rats by activating PI3K/eNOS pathway and inhibiting oxidative stress

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Objective: Our previous study has proved the protective effect of human tissue kallikrein 1 (hKLK1) on erectile function of aged rats with the transgenic Sprague-Dawley (SD) rats harboring *hKLK1* gene (TGR), but its role on diabetic erectile dysfunction (ED) is still unknown.
Methods: An 8-week-old male wild type SD rats (WTR) and age matched TGR were randomly divided into five groups (n=8 in each group): (I) untreated WTR as the control; (II) untreated TGR; streptozotocin-induced diabetic (III) wild type rats (WTDM); and (IV) transgenic rats (TGDM); (V) TGDM with bradykinin receptor 2 inhibitor HOE140 (TGDMH). Erectile response, represented with the ratio of peak intracavernous pressure to systemic mean arterial pressure and area under the curve, were measured by cavernous nerve electrostimulation after 12 weeks. Penile corpus cavernosum (CC) tissues from all rats were harvested for *hKLK1* gene identification and mechanism exploration. The related genes expressions of PI3 kinase (PI3K)/endothelial nitric oxide synthase (eNOS) pathway in CC of each group were assayed to detect the effect of hKLK1 *in vivo*. CC smooth muscle