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**Title:** EFFECTS OF TYPE II DIABETES MELLITUS ON CONTRACTILITY AND EXPRESSIONS OF NITRIC OXIDE SYNTHASE IN BLADDER

**Aims of Study :**

Bladder dysfunction is a common complication of diabetes mellitus(DM). This systemic disease is classified to insulin dependent form (Type I) and insulin independent form (Type II). There are numerous studies for diabetic cystopathy in Type I DM animal model but few published studies for diabetic cystopathy in Type II DM in spite of high prevalence. The objective of this study was to evaluate alteration of contractility and expression of nitric oxide synthase (NOS) that generate nitric oxide (NO) in detrusor muscle using Otsuka Long-Evans Tokushima Fatty (OLETF) rats known as spontaneous Type II DM animal model.

**Methods :**

OLETF (n=10) and LETO (Long-Evans Tokushima Otsuka) rats (n=10) as control were used. Oral glucose tolerance test was done on 20 weeks after birth in all animals and confirmed evolution of DM. Bladder were obtained on 40 weeks after birth. Bladder muscle strips were utilized to generate the response to electrical stimulation(ES, 2-32Hz) in Tyrode solutions. The contractile responses were monitored via an FT03 force transducer and recorded on a Grass 7D polygraph and expressed as the g tension per 100mg of tissue. And RT-PCR were performed to determine the alteration of neuronal NOS (nNOS), endothelial NOS (eNOS), inducible NOS (iNOS) and growth factors in detrusor muscle.

**Results :**

Contractile response to ES and bethanechol were significantly decreased in DM group, especially there are more decline of contractile response in the higher frequency stimulation. Expressions of nNOS and nerve growth factor were significantly increased in DM group but expressions of eNOS and iNOS were not changed.

**Conclusions :**

Our study shows that NO may play a roles in Type II diabetic bladder dysfunction. We think that increased NO through up-regulated nNOS in diabetic bladder can augment relaxability and affect sensory of bladder, cellular proliferation and viability of detrusor muscle. And we think these changes in diabetic bladder were mainly caused by change of nNOS expression.