GENDER-RELATED DIFFERENTIAL EXPRESSION OF MYOSIN HEAVY CHAIN ISOFORMS IN THE URETHRAL STRIATED MUSCLES OF RAT

Hypothesis / aims of study

Based upon morphology studies, female gender and elder age are risk factors for developing stress urinary incontinence, however, the mechanism is not entirely clear .The aim of this study was to investigate the myosin heavy chain isoforms(MHCIs) expression in the urethral striated muscles of rat to discuss the mechanism which may exist.

Study design, materials and methods

32 adult female nonporous Fisher rats at different ages were divided equally into 8 groups, group I (6 months, male),

group II (12 months, male), group III (24 months, male), group IV (32 months, male), group V(6 months, female),

group VI(12 months, female), group VI(24 months, female) and group VII(32 months, female). We had examined the fiber type distribution (by immunohistochemistry) as well as the expression of the corresponding MHCIs protein and messenger RNA (mRNA) (protein and mRNA expression, using Western blot or quantitative real-time polymerase chain reaction (RT-PCR)).

Results

The western blot shows that the expression of MHC- IIb and MHC- IIx increased from 12 months old, and they express more in male. The MHC- Ishows the opposite result. In the RT-PCR, we set the group I as the base, that means: group I (MHC-IIb=1.00 MHC-IIx=1.00). so, group II (MHC-IIb=0.74 MHC-IIx=8.22), group III (MHC-IIb=1.65 MHC-IIx=5.97), group IV (MHC-IIb=33.51 MHC-IIx=2179.83), group V(MHC-IIb=1234.75 MHC-IIx=6608.01), group VI(MHC-IIb=761.83 MHC-IIx=6295.04), group VI(MHC-IIb=3508.57 MHC-IIx=13216.02) and group VI(MHC-IIb=3019.30 MHC-IIx=12189.35).

Interpretation of results

The outcome revealed that the urethral striated muscles are consisted of four kind of isoforms: MHC- I,MHC- IIa, MHC- IIb and MHC- IIx. The expression of MHC- IIb and MHC- IIx increased from 12 months old, and they express more in male(p<0.01).

Concluding message

The change with age of rats urethral striated muscle MHC-II b and MHC-II x could be the molecular basis of dysfunction of urethral sphincter, while the sex may contribute a key role.

Disclosures

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