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INCREASED URETHRAL EXPRESSION OF TRANSIENT RECEPTOR POTENTIAL VANILLOID 1 AND 4 IN CYCLOPHOSPHAMIDE-INDUCED CYSTITIS

Hypothesis / aims of study
To investigate the effect of cyclophosphamide (CYP)-induced cystitis on the expression of transient receptor potential vanilloid 1 and 4 (TRPV 1 and 4) in rat urethra and to determine their role in inflammation-induced dysfunction of rat urinary bladder.

Study design, materials and methods
Female Sprague-Dawley rats were assigned to control (n=30) and experimental (n=30) groups. In the experimental group, cystitis was induced by intraperitoneal injection of CYP (200 mg/kg), while the control group received an intraperitoneal injection of saline. After 3 days, urodynamic studies were conducted to measure the inter-contraction interval and contraction pressure. The expression and cellular localization of rat urethral TRPV1 and TRPV4 were determined by western blot and immunofluorescence analyses, respectively.

Results
In cystometrograms, the inter-contraction interval (min) was significantly lower in the experimental group (14.7 ± 0.8) than in the control group (5.9 ± 1.2) ($p<0.05$). The average contraction pressure (mmHg) was significantly higher in the experimental group (16.3 ± 0.9) than in the control group (10.1 ± 1.3) ($p<0.05$). In the urethral mucosa, TRPV1 was mainly expressed in the cytoplasm, whereas TRPV4 was predominantly detected in the cell membrane. In the urethral smooth muscle, TRPV1 was detected in the muscle cells, whereas TRPV4 was detected in the tissues surrounding the muscle bundles. Furthermore, the urethral levels of TRPV1 and TRPV4 were significantly higher in experimental group than in the control group ($p<0.05$).

Interpretation of results
Inflammatory changes in the urinary bladder might significantly alter the urethral expression of TRPV1 and TRPV4, resulting in distinctive expression patterns in different urethral tissues.

Concluding message
This finding suggests that TRPV1 and TRPV4 might play a role in the urethral functional impairment observed in bladder dysfunction.

References
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Disclosures
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