Menopause reduces the levels of sexual hormones. Urolological complications are commonly observed in the postmenopausal women (1). The urinary symptoms associated with late postmenopause include frequency, nocturia, urgency and urge incontinence. Testosterone replacement therapy has favorable effects on the lack of sexual desire, bone loss and decreased muscle mass (2, 3). However, little is known about the effects of testosterone replacement in the urinary symptoms in postmenopause. Therefore, in the present study, we used a rat model of ovariecotmy to evaluate the effects of testosterone in voiding dysfunction and in vitro bladder alterations.

**HYPOTHESIS / AIM OF STUDY**

- Two-month old female Sprague–Dawley rats (250-280 g) were anesthetized (ketamine/xylazine, 60:6 mg/kg, IP), and submitted to bilateral ovariectomy, whereas sham-operated rats were manipulated but ovaries were left intact. Rats were divided into the following groups: Sham, OVX and OVX plus testosterone undecanoate treatment (IM).
- After 30 days of treatment, the general characteristics of ovariecotmy model (body, perigonadal fat mass, uterus, bladder and urethra weights), as well as the arterial pressure by tail-cuff technique were evaluated. Blood samples were collected for testosterone serum quantification by chemiluminescence.
- Urodynamic studies were realized in anesthetized animals (urethane 1.8 g/kg) by cystometry.
- A separate group had the bladder and urethra removed. In vitro functional assays were evaluated in isolated bladder strips using a 4 mm myograph system and isolated urethra ring using 5 mL myograph system, each system containing Krebs solution (pH 7.4, 37 °C, O2–CO2 (95%: 5%)). In the bladder, concentration-response curves for the muscarinic agonist carbachol (CCl) and the β3-selective agonist mirabegron were conducted. Electrical Field Stimulation (EFS) was applied in bladder tissue placed between two platinum electrodes connected to a Grass S88 stimulator at 80 V, 1 ms pulse width and trains of stimuli lasting 10 s at varying frequencies (1-32 Hz). In the urethra, concentration-response curves for angiotensin II (Ang II) were conducted.

**STUDY DESING, MATERIALS AND METHODS**

**RESULTS**

Bilateral ovariectomy significantly increased body weight, blood pressure and perigonadal fat mass, reduced uterus weight and diminished the hormonal levels of testosterone compared with Sham group.

The voiding dysfunctions in OVX rats were characterized by increases of baseline pressure, threshold pressure, voiding frequency and post-micturition pressure.

**INTERPRETATION OF RESULTS**

According to our study, supraphysiological dose of testosterone (10 mg/kg) improves the voiding dysfunctions and the in vitro alterations of bladder and urethra smooth muscle. It is likely that testosterone acts by activating the androgen receptor in the lower urinary tract, triggering genomic and non-genomic signaling pathways. However, additional studies are required to elucidate these mechanisms.

**CONCLUSION**

Testosterone replacement at a physiologival dose (5 mg/kg) does not improve the bladder dysfunctions by ovariecotmy; however, at a supraphysiological dose (10 mg/kg), testosterone replacement normalizes all the functional changes in lower urinary tract smooth muscle. Our findings suggest that androgen therapy may have therapeutic benefits for urologic complications associated with postmenopause.

**REFERENCES**