

HYPERTENSION INDUCED BY SALT LOADING IS RELATED TO STORAGE DYSFUNCTION IN RATS

Hypothesis / aims of study

Clinical observations indicate that many non-urological diseases seem to be associated with lower urinary tract symptoms (LUTS). According to a population-based study LUTS is an important part of the symptomatology of hypertension (HT), DM, and neurological disease. HT is one of the risk factors for worsening LUTS and decreasing the improvement of storage symptoms by α 1-blocker (Int J Urol 2003, 10:569). Sympathetic nerve activity also influences HT and LUTS (J Urol 2005, 174:1327). The spontaneous hypertensive rat is considered a valuable tool for exploring the pathogenesis of detrusor overactivity, but pathological changes in the bladder are thought to be irreversible. In salt-sensitive HT, increased oxidative stress elevates arterial pressure through central sympathoexcitation (Hypertension 2007,50:360). However, it has not yet been determined whether salt-sensitive HT contributes to LUTS and influences urine production rhythm. Thus, we examined whether salt-sensitive HT influences LUTS and urine production.

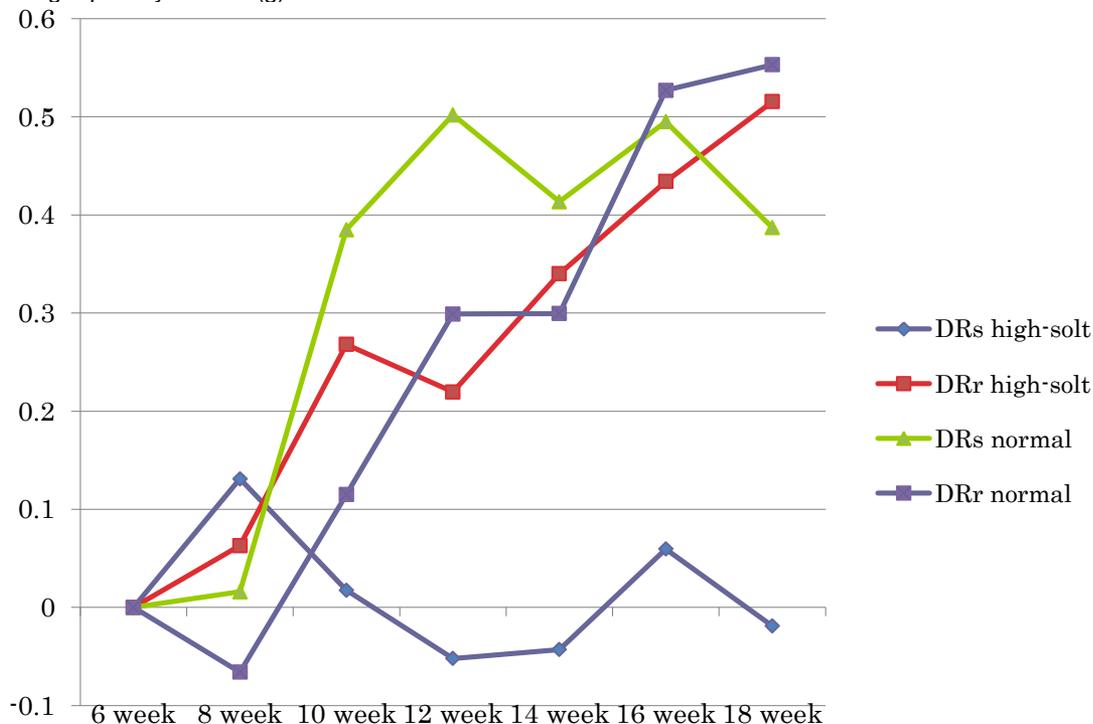
Study design, materials and methods

The sympathetic nerve activity is augmented with salt loading in Dahl salt-sensitive rats (DSs), but not in Dahl salt-resistant rats (DRr). Six-week-old male DSs and DRr were fed with a normal (0.252%) or high-salt (8%) diet and 40 ml water/day for 12 weeks. Blood pressure was measured through the tail artery in a non-anesthetized state. Urine volume and frequency were recorded all day long in a metabolic cage. We calculated the inactive period (sleeping) urine volume index (IUi), which was defined as urine volume during inactive period divided by 24-h urine output.

Results

A 20- to 30-mmHg increase in systolic and diastolic blood pressure was noted in DSs after 2-week salt loading. Salt loading increased urine production during the active (awake) period of the day both in DSs and DRr. In the inactive period, the mean voided volume gradually increased in DRs fed a normal or high-salt diet, while it did not change in DSr fed a high-salt diet. There were significant differences in mean voided volume after 10-week salt loading between DRs and DSr fed a high-salt diet. In DSs fed a high-salt diet, IUi increased 2 weeks after salt loading, and decreases furthermore 2 weeks later

Change quantity of IUi (g)



Interpretation of results

These results indicate that salt loading induces HT and decreases the voided volume, also changing the rhythm of urine production throughout the day. This model will be useful in defining the mechanisms governing the induction of storage dysfunction in patients with HT.

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Concluding message

Whole bladders were excised from the rats and made to expand with Krebs solution. We measured adenosine triphosphate and prostaglandin E2 from the collected solution.

Keywords: hypertension, salt loading, storage dysfunction, urine productio

Disclosures

Funding: none **Clinical Trial:** No **Subjects:** ANIMAL **Species:** Rat **Ethics Committee:** University of Fukui