Altered Renal Tight Junction Protein Expression and Pressure Natriuresis in Dahl Salt-Sensitive Rats

Gheun-Ho Kim, Chor Ho Jo, Sua Kim, Il Hwan Oh, Joon-Sung Park

Department of Internal Medicine, Hanyang University College of Medicine, Seoul, Korea

Background

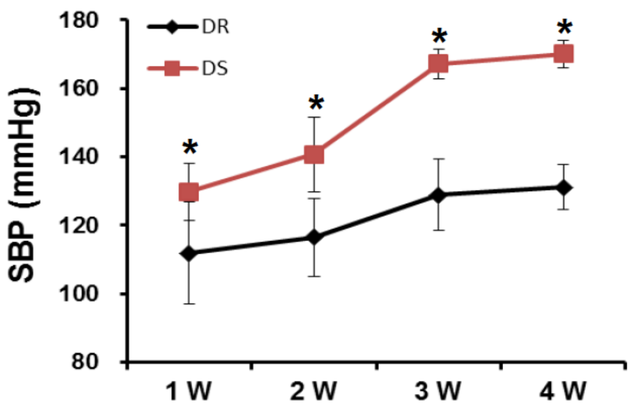
Altered pressure natriuresis is an important mechanism of hypertension, but it remains elusive at a molecular level. We hypothesized that tight junction (TJ) may have a role in pressure natriuresis because paracelullar NaCl transport affects renal interstitial hydrostatic pressure in the kidney.

Methods

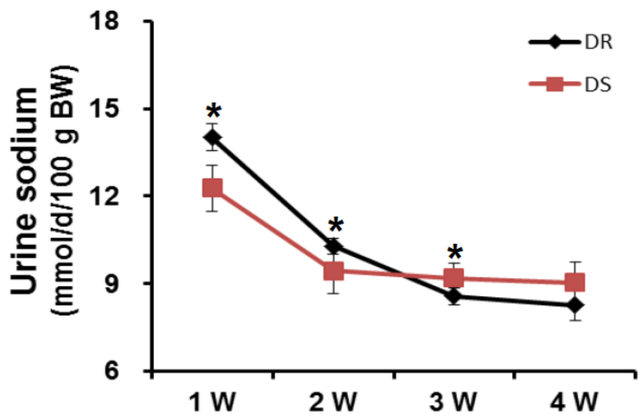
To assess the association of salt-sensitive hypertension with altered renal TJ protein expression, Dahl salt-sensitive (DS) and Dahl salt-resistant (DR) rats were put on an 8% NaCl-containing rodent diet for 4 weeks. Systolic blood pressure (SBP) and urine sodium excretion were weekly measured, and kidneys were harvested for immunoblotting and quantitative PCR analysis at the end of animal experiment.

Results

Systolic blood pressure

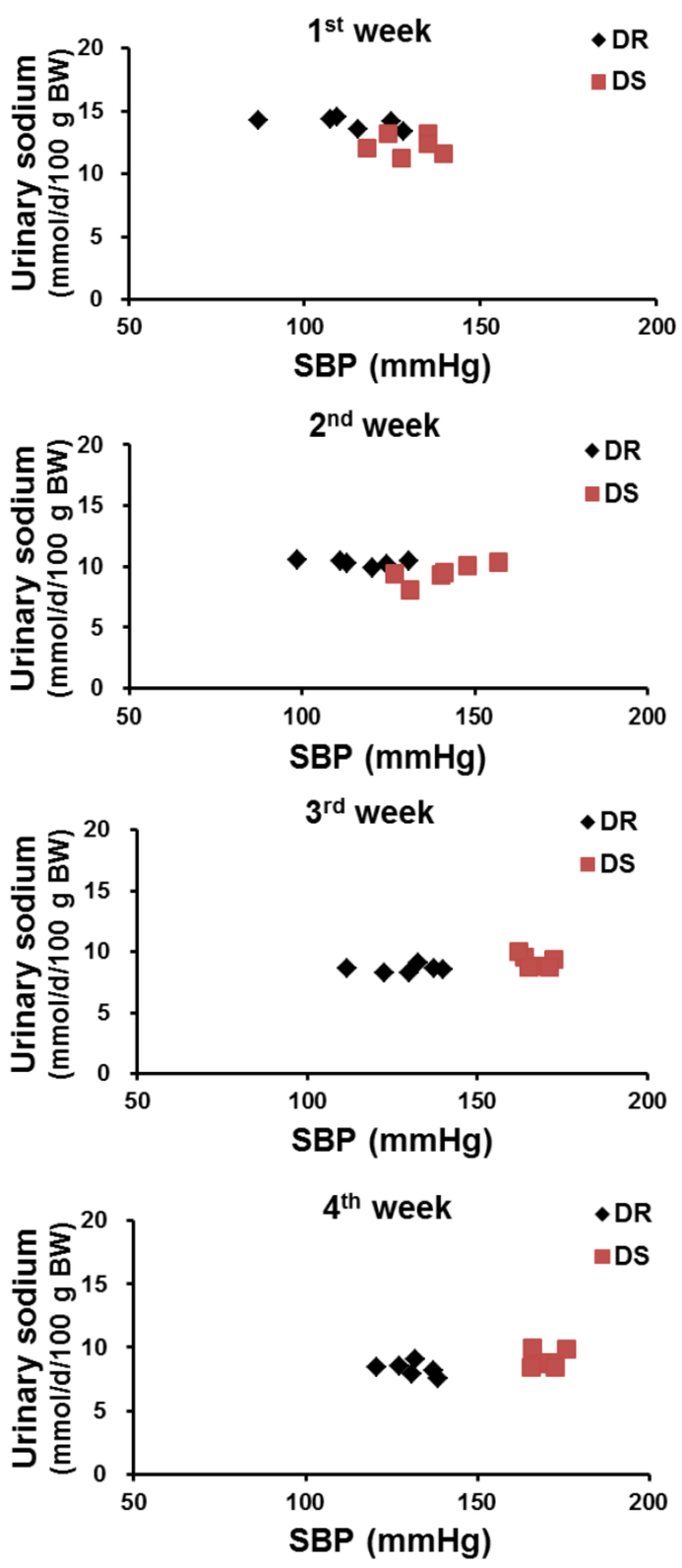


Urinary sodium excretion

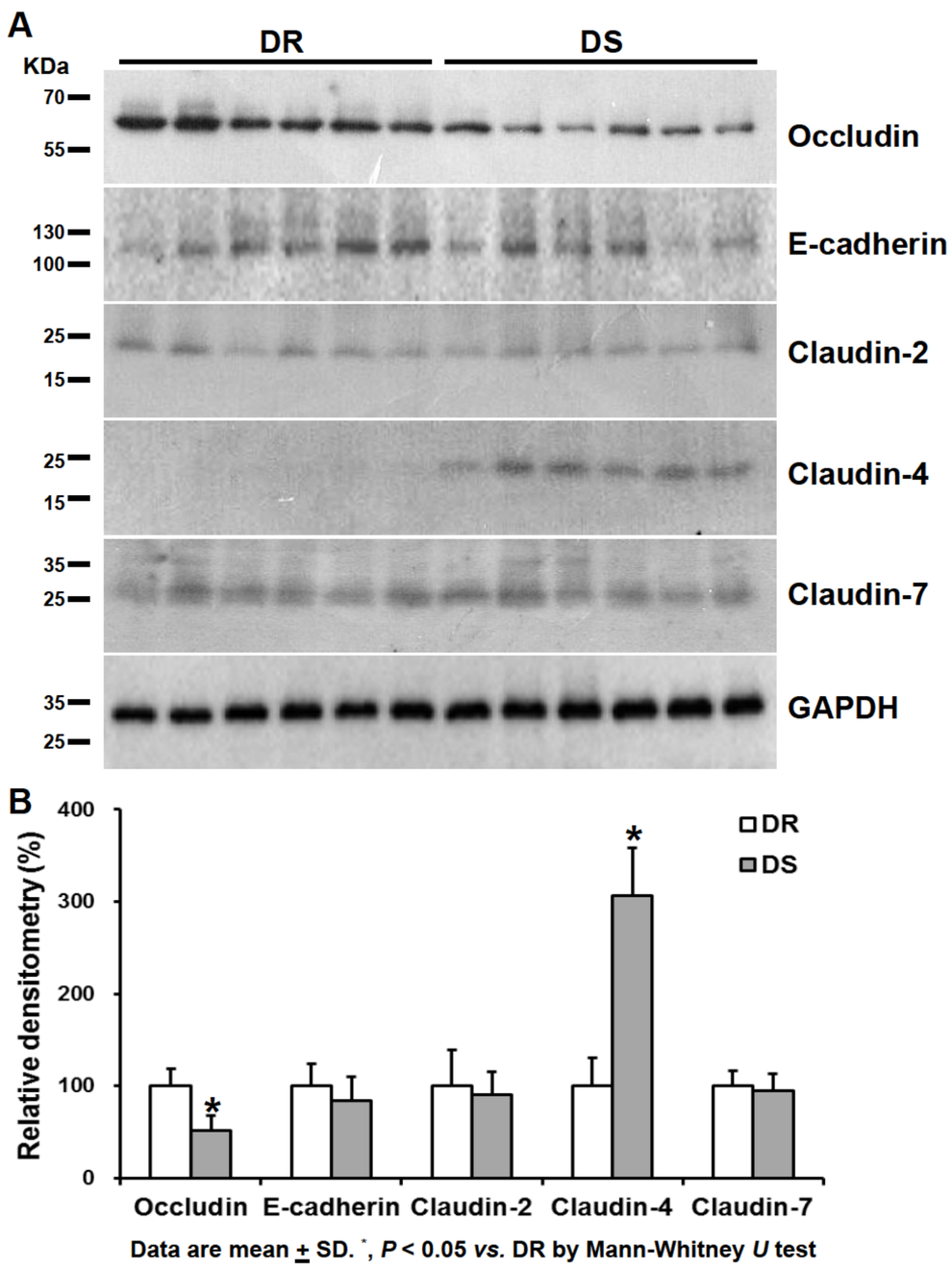


Data are means ± SD. *, $P < 0.05$ *vs.* DR by Mann-Whitney *U* test

Pressure-natriuresis relationship

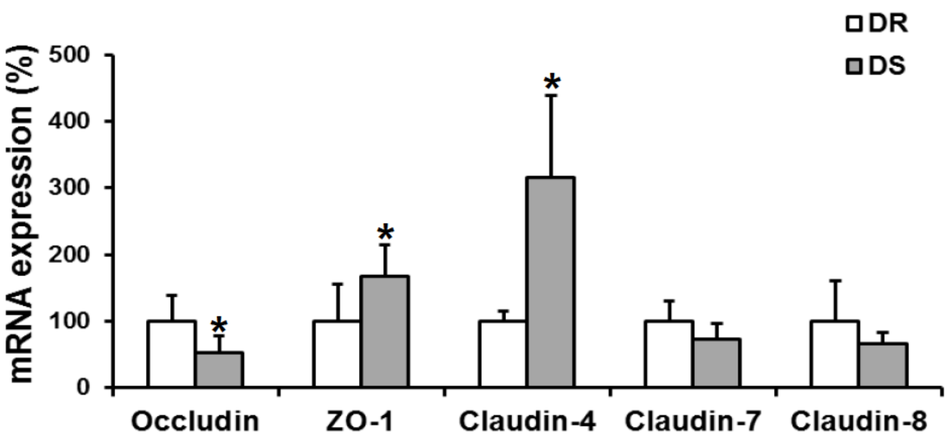


Immunoblot analysis



Data are mean ± SD. *, $P < 0.05$ *vs.* DR by Mann-Whitney *U* test

qPCR analysis



Data are mean ± SD. *, $P < 0.05$ *vs.* DR by Mann-Whitney *U* test

Conclusion

In DS rats, salt-sensitive hypertension was associated with differential changes in renal TJ protein expression. Both upregulation of claudin-4 and downregulation of occludin might increase paracellular NaCl transport in the kidney, resulting in impaired pressure natriuresis in DS rats.

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