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Urinary vanin-1 reflects an ameliorating effect for renal injury in Dahl-salt sensitive rats

Background In salt-sensitive hypertension, oxidative stress plays a major role in the progression of renal disease partly through the activation of mineralocorticoid receptor. Previously, we demonstrated that urinary vanin-1 is an early renal biomarker of oxidative tubular injury. However, it remains unknown that urinary vanin-1 could reflect the treatment effect. The objective of this study is to clarify the treatment effect for renal tubular damage in Dahl salt-sensitive rats <DS rats> on high-salt diet.

Methods Male DS rats were maintained on regular-salt diet or high-salt diet for 4 weeks. The high-salt diet group was subdivided into three groups to receive vehicle, tempol, or eplerenone during the period. After 4 weeks of the treatment, blood pressure was measured by tail-cuff method and kidney tissues were evaluated. Reactive oxygen species were assessed by the measurements of malondialdehyde and the by immunostaining of 4HNE.

Results A high-salt intake for 4 weeks caused histologically renal tubular damages in DS rats, which were suppressed by tempol and eplerenone. Although there were no significant differences in proteinuria and urinary NAG between rats treated with HS and those with high-salt diet and tempol, urinary excretions of vanin-1 showed significantly lower in rats treated with high-salt diet and tempol than those with high-salt diet.

Conclusion These results suggest that urinary vanin-1 is a potentially sensitive biomarker of ameliorating effect for histological renal tubular damage in salt-sensitive hypertension.