sequentially maintained on 3 days baseline investigate, a low-salt diet for 7 days (3 g/day, NaCl), then a high-salt diet for 7 days (18 g/day). Salt-sensitivity was diagnosed in 10 subjects who exhibited a response of the increase in mean BP by ≥10% from low-salt period to high-salt period. Total adiponectin was determined using a validated sandwich ELISA employing an adiponectin-specific antibody.

Results There was no difference of plasma adiponectin between normotensive salt sensitive subjects and normotensive salt resistant subjects during any salt intake. However, plasma adiponectin higher significantly in high salt intake in normotensive salt resistant subjects than low salt diet (6.1±1.3 vs 7.1±1.7 μg/ml, p=0.047). However, high salt intake could not affect adiponection in normotensive salt sensitive subjects (6.4±2 vs 5.9±2.1 μg/ml, p=0.481).

Conclusions Our data indicates that the disturbance of adiponectin exists in normotensive salt sensitive subjects during high salt diet, which may be a novel underlying mechanism of salt sensitivity.

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HIGH SALT INTAKE FAIL TO ENHANCE PLASMA ADIPONECTIN IN NORMOTENSIVE SALT-SENSITIVE SUBJECTS

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Objectives Evidences show that salt could modulate adiponectin and inflammation level in normal individuals. Therefore, we hypothesised that abnormalities of adiponectin and inflammation may be the potential mechanism of salt sensitivity. Aims of the study were to investigate whether different alteration of adiponectin and inflammation level in response of high salt were exhibited between normotensive salt sensitive and salt resistant subjects.

Methods 30 normotensive subjects (aged 25–50 years) were selected from a rural community of Northern China. All of the people were