Researcher finds link between brain signaling and renal function

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Boston University School of Medicine (BUSM) researchers recently uncovered a brain signaling pathway responsible for regulating the renal excretion of sodium. The findings appear in the *Journal of the Federation of American Societies for Experimental Biology*.

Hypertension, or chronic high blood pressure, affects one-third of adults, significantly increasing cardiovascular risk and mortality. Approximately 50 percent of hypertensive patients are salt-sensitive and exhibit an increase in blood pressure following salt-intake.

According to the researchers, little is known about the mechanisms acting in the brain to control the removal of dietary salt from the body through the kidneys. "Our data shows that changes in dietary sodium intake evoked natural site-specific changes in brain hypothalamic paraventricular nucleus (PVN) Gαi2 protein levels," said Senior Author Richard Wainford, PhD, assistant professor of pharmacology and experimental therapeutics.

Wainford and his team explored the role of Gαi2 signal transduction proteins in the brain pathways activated to regulate salt and water excretion and subsequently blood pressure. They identified a previously unknown role of PVN Gαi2-subunit proteins as a central mechanism mediating the suppression of renal sympathetic nerve traffic to the kidneys and the renal excretion of sodium.

These data provide a target for new therapies that may improve

cardiovascular and renal excretory function. This may help treat multiple disease states, such as salt-sensitive hypertension and congestive heart failure caused by elevated sodium intake.

Provided by Boston University Medical Center