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Cardiovascular Endocrinology

ENDOCRINE HYPERTENSION AND ALDOSTERONE EXCESS II

Impact of the Gut Microbiome and Renin-Angiotensin-Aldosterone System in Hypertensive Patients With Low-Salt Intake

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Abstract: Salt intake is one of most important environmental factors responsible for triggering the onset of hypertension. Renin-angiotensin-aldosterone system (RAAS) plays a key role in adjusting sodium homeostasis and blood pressure. Recently, the potential role of the gut microbiome (GM) in altering the health of the host has drawn considerable attention. We investigated the impact of intestinal microflora and RAAS in hypertensive patients with low-salt or high-salt intake using an observational study.
A total of 239 participants were enrolled and their GMs and clinical backgrounds examined, including the renin-angiotensin-aldosterone system and inflammatory cytokine levels. On the basis of enterotypes—determined by cluster analysis—and salt intake, the participants were classified into four groups, low salt/GM enterotype 1, low salt/GM enterotype 2, high salt/GM enterotype 1, and high salt/GM enterotype 2.
The prevalence of hypertension was significantly lower in the low-salt intake (low salt/GM enterotype 1 = 47% vs low salt/GM enterotype 2 = 27%, $p = 0.04$) groups. No significant difference in the prevalence of hypertension was observed for the two GM enterotype groups with high-salt intake (GM enterotype 1 = 50%, GM enterotype 2 = 47%; $p = 0.83$). Plasma aldosterone concentration was significantly different among the four groups ($p < 0.01$). Furthermore, the relative abundance of *Blautia*, *Bifidobacterium*, *Escherichia-Shigella*, *Lachnoclostridium*, and *Clostridium sensu stricto* was also significantly different among these enterotypes. This suggested in certain individuals (with specific gut bacteria composition) changing dietary habits—to low salt—would be
ineffective for regulating hypertension through RAAS. Our findings provide a new strategy for controlling blood pressure and preventing the development of hypertension through restoring GM homeostasis.

Diabetes Mellitus and Glucose Metabolism

DIABETES TECHNOLOGY

Characterization and Functional Rescue of a Nephrogenic Diabetes Insipidus Causing S127F Substitution in V2 Vasopressin Receptor

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