Many asymptomatic patients with a small abdominal aortic aneurysm (AAA) are diagnosed incidentally during routine abdominal screening. However, there is no effective treatment option for small AAA despite their gradual expansion. Angiotensin II is thought to induce and perpetuate inflammation in the aortic wall, and previous studies reported the inhibitory effects of angiotensin II receptor blockers and angiotensin-converting enzyme inhibitors in experimental studies. However, there is no evidence for a beneficial effect of these medicines on AAA progression in clinical trials. Therefore, we focused on direct renin inhibition, because inhibition of multiple steps of the renin–angiotensin system might be required for treating AAA. The present study reported the inhibitory effect of direct renin inhibition on experimental AAA expansion associated with a significant reduction of excess renin–angiotensin system activation in the aneurysm wall. In addition, significant inhibition of activation of NF-kB (nuclear factor-kB), AP-1 (activator protein-1), and CREB (cAMP response element–binding protein) was also observed after treatment with aliskiren. These transcription factors are thought to cooperatively regulate the inflammatory gene expression profile associated with AAA progression. Therefore, treatment with aliskiren inhibited AAA progression through inhibition of inflammatory response and prorenin receptor elevation in the aneurysm wall, even in the absence of a blood pressure–lowering effect. Direct inhibition of renin activity using aliskiren could become a potent therapeutic option for treating hypertensive patients with small AAA.

High salt intake is a major risk factor for hypertension and cardiovascular events. Although salt reduction strategies have been proposed for years, their long-term efficiency and adherence are not optimal. Salt consumption is related to altered salty taste sensitivity and the development of a demand for salted food that involves the brain reward pathways. Capsaicin, a major pungent ingredient in chili pepper, has been shown to influence salty taste sensitivity in humans. Therefore, we hypothesize that the spicy flavor produced by capsaicin could modify the salty taste, which might result in salt reduction through impacting neural salty taste. In this issue of Hypertension, we show that salt intake and salty preference are related to the regional metabolic activity in the insula and orbitofrontal cortex of individuals. Furthermore, capsaicin administration enhances individual insula and orbitofrontal cortex metabolism in response to high-salt stimuli. In addition, the enjoyment of a spicy diet significantly reduces salt preference, daily salt intake, and blood pressure in the participants through modifying the neural processing of salty taste. This study provides insights for the enjoyment of spicy flavor as a promising behavioral intervention for reducing high salt intake and blood pressure. Like the well-known DASH (Dietary Approaches to Stop Hypertension) and Mediterranean diets, a spicy diet could benefit cardiovascular health.

High blood pressure (BP) results in cognitive loss late in life. Typically, high BP itself is viewed as causing brain dysfunction, but this presumption is challenged by evidence from animal models of early neural involvement in the development of hypertension. In a group of 154 midlife adults with untreated prehypertension, we investigated relationships between brain function and the progression of BP over a 2-year period. We tested both cognitive function and regional cerebral brain flow responses to challenging cognitive tests. As shown above, greater regional cerebral brain flow responses in frontostriatal areas predicted the progression of BP. Poorer performance on working memory tests also predicted progression of BP. In contrast, BP changes over the 2 years failed to predict follow-up cognitive function or regional cerebral brain flow. Analyses controlled for age, sex, race, and education, as well as known risk factors for BP progression. These results suggest that brain function is altered early in the course of hypertensive disease. Therefore, we see a need for additional research focused on identifying the brain mechanisms involved in human hypertension and their possible role in cognitive loss via, or independent of, high BP. In the meantime, our attempts to prevent hypertension and its cognitive sequelae can reasonably emphasize changing risk factors with known relationships to both BP and brain function. These include stress-reducing interventions, regular physical activity, and improved sleep hygiene.
Clinical Implications

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