

Zinc deficiency may play a role in high blood pressure

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Lower-than-normal zinc levels may contribute to high blood pressure (hypertension) by altering the way the kidneys handle sodium. The study is published ahead of print in the *American Journal of Physiology—Renal Physiology*.

Zinc deficiency is common in people with [chronic illnesses](#) such as type 2 diabetes and [chronic kidney disease](#). People with low zinc levels are also at a higher risk for hypertension. The way in which the kidneys either excrete sodium into the urine or reabsorb it into the body—specifically through a pathway called the sodium chloride cotransporter (NCC)—also plays a role in blood pressure control. Less sodium in the urine typically corresponds with higher blood pressure. Recent research has suggested that zinc may help regulate proteins that in turn regulate the NCC, but a direct link between [zinc-deficiency](#)-induced hypertension has not been examined.

Researchers compared male mice with zinc deficiency to healthy controls with normal zinc levels. The zinc-deficient mice developed [high blood pressure](#) and a corresponding decrease in

urinary sodium excretion. The [control group](#) did not experience the same changes. A small group of the zinc-deficient mice were fed a zinc-rich diet partway through the study. Once the animals' zinc reached adequate levels, blood pressure began to drop and urinary sodium levels increased. "These significant findings demonstrate that enhanced renal [sodium] reabsorption plays a critical role in [zinc-deficiency]-induced hypertension," the research team wrote.

"Understanding the specific mechanisms by which [zinc deficiency] contributes to [blood pressure] dysregulation may have an important effect on the treatment of hypertension in chronic disease settings," the researchers added.

More information: Clintoria R. Williams et al, Zinc Deficiency Induces Hypertension by Promoting Renal Sodium Reabsorption, *American Journal of Physiology-Renal Physiology* (2019). [DOI: 10.1152/ajprenal.00487.2018](#)

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