Sodium transporter appears likely target for treating salt-sensitive hypertension

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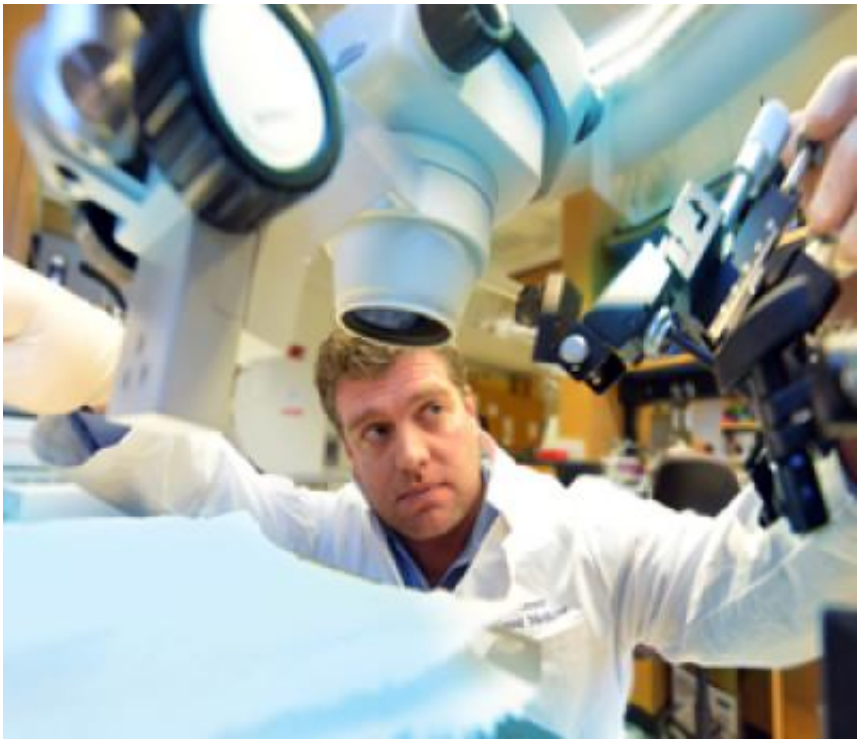

Sodium transporter appears a likely target for treating salt-sensitive hypertension. Credit: Phil Jones, GRU Photographer

Genetics and demographics likely put you at risk for salt-sensitive hypertension, and scientists are looking for a way to protect you.

The concept is that free radicals in the kidney prompt the organ to hold onto sodium rather than eliminate excess through urination. Excess

sodium, in turn, increases free radical production, said Dr. Paul O'Connor, hypertension researcher in the Section of Experimental Medicine at the Medical College of Georgia at Georgia Regents University.

Blocking the hydrogen transporter HVI – best known for its role in helping immune cells produce large amounts of free radicals to kill bacteria – may stop the unhealthy, vicious cycle.

"We think HVI overexpression in the kidney fuels the production of too many free radicals, which can activate the sodium transporters to hold onto more sodium, leading to hypertension and kidney damage," O'Connor said.

With the help of a New Investigator Award from the American Heart Association and a HV1 mutant rat, he plans to find out.

Sodium helps the kidneys regulate fluid volume and blood pressure. However being older, black and obese are risk factors for holding onto too much sodium. Nearly 40 percent of blacks and 30 percent of whites with healthy blood pressure have a tendency to hold onto salt and so-called salt sensitivity occurs in nearly 75 percent of blacks and more than half of whites with hypertension.

O'Connor has found HVI in the nephrons of the kidneys where decisions are made about how much sodium to retain. And, when looking at sodium transport inside renal tubule cells, he found a drug he was using inhibited free radical production and started putting the pieces together.

O'Connor and his collaborators developed the HV1 mutant of the Dahl salt-sensitive rat, which becomes hypertensive on a high-salt diet, to test the emerging hypothesis.

"We think if HV1 is not present, you will have reduced salt sensitivity, reduced oxidative stress and reduced renal injury when you feed these animals a high-salt diet," O'Connor said.

If he's correct, targeted treatment for salt-sensitive hypertension may emerge. "We think it might be an interesting target to help inhibit oxidative-stress related cardiovascular disease," O'Connor said.

Although free radicals are a known contributor to cardiovascular disease, antioxidants haven't proven effective therapy, said O'Connor, who would like to directly inhibit the source of free radicals rather than scavenge for them once they are formed. An estimated 1-in-50 patients in the United States develop treatment-resistant hypertension, which increases the risk of heart attack, stroke and other complications, according to studies published in the American Heart Association journal *Circulation*.

HV1 was discovered in 2006 in immune cells where is helps ensure the proper activity of the enzyme NADPH oxidase, which actually produces free radicals. It's now been found in a variety of cell types, and scientists are exploring its potential role in maladies such as ischemic stroke and cancer. O'Connor notes that his HIV mutant rats do not appear to have a compromised immune system.

O'Connor joined the MCG faculty in 2011.

Provided by Medical College of Georgia at Georgia Regents University

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