Segmental Renal Sodium Handling in Relation to the Human SAH Gene

To the Editor:

The expression of SAH (spontaneously hypertensive rat-clone A-hypertension–associated) gene is markedly higher in the kidneys of spontaneously hypertensive rats compared with normotensive Wistar-Kyoto control rats.1 In rats, SAH is expressed mainly in proximal renal tubules.2 The human SAH gene is located on chromosome 16p13.11 and is also expressed in the kidney.3 A large number of previous studies suggested association of the human SAH gene with hypertension and related metabolic phenotypes, such as obesity, hypercholesterolemia, and hypertriglyceridemia. However, we recently analyzed blood pressure, measures of obesity, serum lipids, and blood glucose as continuous phenotypes in 2603 relatives from 560 families and 31 unrelated subjects (mean age: 41.3 years). From this sample, we had excluded all of the women taking contraceptive pills or hormonal replacement therapy. Considering single nucleotide polymorphisms, as well as haplotypes, none of the phenotype-genotype associations reached statistical significance.4

In this journal, we reported recently on the genetic association of segmental renal sodium handling with DRD1 and GRK4.5 In a family based random sample of 611 untreated white subjects, we measured the endogenous lithium clearance to assess fractional sodium excretion (FE_{Na}) and proximal and distal tubular sodium reabsorption. Because the human SAH gene is expressed in the kidney,1–3 we used the same sample and same methods as published previously5 to explore the possible association of renal sodium handling with the aforementioned variation in human SAH. Because of this, all of the participants were in balance with regard to sodium intake and excretion, our family based findings on FE_{Na} suggest that genetic variability in human SAH might influence salt intake. However, the more likely explanation is that the borderline significance for the family based association between FE_{Na} and variability in SAH might have arisen by chance. We conclude that, in spite of its renal expression,1–3 the human SAH gene is unlikely to have an important role in renal sodium handling.

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Disclosures

None.

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