Role of Angiotensinogen and Relative Aldosterone Excess in Salt-Sensitive Hypertension

To the Editor:

We read with great interest the article by Michel et al.1 They demonstrated that circulating angiotensinogen concentration (AGT) was positively associated with aldosterone level and blood pressure in subjects of African ancestry with high urinary Na/K ratio. Their findings may elucidate one of the key mechanisms of salt-sensitive hypertension. We have several comments regarding their work.

Compared with aldosterone level alone, aldosterone/renin ratio (ARR) is more reproducible and could be an index for inappropriate aldosterone activity and salt sensitivity. In a recent publication from their group, urinary Na/K ratio was positively associated with blood pressure levels in subjects of African ancestry with high ARR.2 We also reported that high ARR but not aldosterone level was significantly associated with hypertension diagnosed based on home blood pressure measurement in a Japanese general population with high Na intake.3 These results2,3 suggest that high ARR, that is, relative aldosterone excess, may be attributable to salt-sensitive hypertension in subjects of Asian and African ancestry. In relative aldosterone excess, aldosterone does not fully decrease, although renin activity is suppressed by sodium-volume overload; this mechanism is supposed to be responsible for salt-sensitive hypertension caused by inappropriate sodium and fluid retention. Michel et al1 noted that AGT is an important determinant of renin-angiotensin-aldosterone system activity under the condition of renin suppression. Thus, we presume that the association of high ARR with salt-sensitive hypertension may partly be mediated by AGT. However, the detailed relationships between ARR and blood pressure or AGT were not described in their study.1

In patients with salt-sensitive hypertension, it is reported that nocturnal blood pressure decline is diminished, which is generally referred to as a “nondipping” pattern.4 We reported previously that high ARR was related to a nondipping pattern in individuals with high Na excretion, supporting the hypothesis that relative aldosterone excess may cause salt-sensitive hypertension.5

We, therefore, would like to know the relationship between AGT and ARR among their subjects of African ancestry with high urinary Na/K ratio. For a more detailed understanding, we would also like to know whether the association between ARR and blood pressure differs before and after adjustment for AGT. Furthermore, if they have ambulatory blood pressure monitoring data, it would be very interesting to see the association between AGT and the nondipping pattern, as well as with blood pressure level. This information could also help clarify the role of AGT in the renin-angiotensin system and in salt-sensitive hypertension.

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