

Letter to the Editor

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Letter by Zhou and Zhou Regarding Article, “Hypertension: Renin–Angiotensin–Aldosterone System Alterations”

To the Editor:

Activation of the renin–angiotensin–aldosterone system (RAAS) is believed to play an important role in hypertension. However, although having a higher risk of hypertension,¹ blacks exhibit lower plasma renin levels compared with whites.² Moreover, black hypertensives show an accentuated responses to diuretics but blunted responses to angiotensin-converting enzyme inhibitors as monotherapy.³ How RAAS activity is altered in hypertension is still not completely understood.

Recently, Te Riet et al⁴ reviewed RAAS alterations in hypertension and emphasized the role of tissue angiotensin in hypertension. In their review, they have not considered the possibility that the RAAS alterations may be secondary to excretory organ insufficiency, which is implicated in hypertension.⁵ Here, we use the functional sweat gland insufficiency in blacks as an example to explain this issue.

The sweat glands excrete water, salts, and numerous other endogenous and exogenous chemical substances, including aldosterone⁶ and angiotensin.⁷ The sweat glands and the kidneys cooperate in the maintenance of water–salt homeostasis under the regulation of the neuroendocrine system, including the RAAS. Sweat excretion is determined by many factors, including race, perinatal development, environmental temperature, physical activity, and skin diseases.⁵ Low temperature inhibits sweat excretion, for example, a decrease in environmental temperature from 30°C to 22°C may decrease daily insensible perspiration from 695 to 381 mL in a resting adult.⁸ Human evolution to environmental temperature has led to racial differences in sweat excretion. Compared with whites, blacks exhibit a thrifty sweating pattern of tropical adaptation, characterized by lower sweat rates, both the insensible (lower skin conductance,⁹ an indicator of sweat-electrolyte excretion) and sensible perspiration, with a higher heat evaporation efficiency.¹⁰ Obviously, the thrifty sweating pattern increases the risk of chemical accumulation (eg, sodium retention) and renal overload and subsequent renal disease, especially when blacks live in low-temperature high-latitude regions, which further exacerbate their functional sweat gland insufficiency. This could explain the observed sodium retention and salt sensitivity in American blacks,² as well as their larger glomerular volume than that of not only whites but also Senegal blacks with the similar genetic background.¹¹ Excess sodium is known to inhibit renin release from the juxtaglomerular cells,¹² accounting for the low plasma renin in blacks.² Stimulating renal excretion can compensate for decreased sweat excretion, explaining the higher sensitivity of black hypertensives to diuretics.³

Hypertension is closely associated with innate and acquired excretory organ insufficiency, including renal disease, low nephron number, decreased sweat rates and, in women, ceased menstrual excretion (amenorrhea/menopause).⁵ Excretory organ insufficiency can lead to abnormal water–salt metabolism, which may, in turn,

alter RAAS activity through feedback mechanisms. Given that (1) excess sodium inhibits renin release and (2) excretory organ insufficiency may reduce aldosterone removal, it is not surprising that hypertension is associated with increased aldosterone:renin ratio.

In summary, it seems that RAAS alteration is not the cause of essential hypertension but rather a consequence of excretory organ insufficiency.

Disclosures

None.

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