The sweat glands excrete water, salts, and numerous other endogenous and exogenous chemical substances, including aldosterone and angiotensin. The sweat glands and the kidneys cooperate in the maintenance of water–salt homeostasis under the regulation of the renin–angiotensin–aldosterone system, including the RAAS. Sweat excretion is determined by many factors, including race, perinatal development, environmental temperature, physical activity, and skin diseases. 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. 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, an indicator of sweat-electrolyte excretion) and sensible perspiration, with a higher heat evaporation efficiency. How RAAS activity is altered in hypertension is still not completely understood.

Recently, Te Riet et al10 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. Here, we use the functional sweat gland insufficiency in blacks as an example to explain this issue.

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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/amenopause). 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.

References
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