Response to Angiotensin-(1–7) and Kidney Disease: Friend or Foe

We appreciate Velkoska et al for their interests in our recent publication concerning the effects of angiotensin (Ang)-(1–7) in the rostral ventrolateral medulla (RVLM) on enhanced cardiac sympathetic afferent reflex (CSAR) and sympathetic activation in 2-kidney, 1-clip renovascular hypertensive rats.1

Indeed, as Velkoska et al have indicated the effects of Ang-(1–7) in patients or experimental models of kidney diseases are controversial.2 Some studies indicate that, as a counter regulator of the classic Ang II/Ang II type 1 (AT1) receptor axis–mediated effects, Ang-(1–7)/Mas receptor axis is expected to play a potential therapeutic role in kidney diseases. However, previous studies in our laboratory have indicated that Ang-(1–7) in the paraventricular nucleus is as effective as Ang II in enhancing the CSAR and increasing sympathetic outflow in 2-kidney, 1-clip rats.3 The increased Ang-(1–7) and Mas receptor activity in RVLM contributes to the enhanced CSAR and excessive sympathetic activation, which could propel the pathogenesis of hypertension and progression of organ damage in renovascular hypertension.1 Velkoska et al also have shown that Ang-(1–7) infusion has major adverse effects of increasing blood pressure and accelerating cardiac hypertrophy and fibrosis in rats with subtotal nephrectomy.4 In addition, Ang-(1–7) causes transition of tubuloepithelial cells into myofibroblasts, which contributes to renal fibrosis.5 Therefore, this leads to the question: is Ang-(1–7) a friend or foe in kidney diseases? This leads to interesting points for discussion that need to be further studied in the future.

Our present work focuses on determining the roles of Ang-(1–7) in modulating the CSAR, sympathetic activity, and blood pressure in specific central nuclei, RVLM.1 It should be noted that we did not investigate the effects of Ang-(1–7) in periphery. The examination of the changes of Ang-(1–7) level in the RVLM after peripheral injection of Ang-(1–7), which was suggested by Velkoska et al might be helpful in determining whether peripheral Ang-(1–7) could cross the blood–brain barrier via the circumventricular organs in the 2-kidney, 1-clip renovascular hypertension model, which requires further study. However, there are not enough evidences to show that Ang-(1–7) in the RVLM is correlated to the circulating Ang-(1–7) level till now.

In summary, we are honored in the interest Velkoska et al have shown in our work concerning the effects of Ang-(1–7) in the RVLM on the CSAR and sympathetic activity in renovascular hypertension. The specific roles of Ang-(1–7) in kidney diseases are controversial, so there is more work needed to be done to further understand it.

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