Response to Angiotensin-(1-7) and Bradykinin in Baroreceptor Reflex Sensitivity in Hypertension

The letter by Tsuda1 raises the question of whether bradykinin contributes to the effects of chronic intracerebroventricular angiotensin (Ang)-(1-7) to enhance baroreflex function in hypertensive (mRen2)27 rats. Our studies exclusively focused on the effects of Ang-(1-7) on NADPH oxidase and mitogen-activated protein kinase signaling pathways in the brain dorsal medulla relative to baroreflex and blood pressure responses. Thus, we cannot provide additional insight regarding the contribution of bradykinin to these responses. It is well established that there are interactions between bradykinin and Ang-(1-7), as well as other components of the RAS that may reflect an interaction through the common metabolism pathway involving angiotensin-converting enzyme, release of bradykinin by Ang-(1-7), or potential receptor interactions.2–5 In an earlier study, however, we demonstrated that the effects of a peptidic angiotensin-converting enzyme inhibitor injected acutely into the nucleus of the solitary tract were mediated primarily by Ang-(1-7) but not by bradykinin.6 Specifically, the selective Ang-(1-7) antagonist d-Ala6-Ang-(1-7) (A779) blocked the improvement in the baroreflex, but the bradykinin antagonist HOE 140 had no effect in that study.

Disclosures

None.

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Not all of the authors were listed in this Letter to the Editor response. The complete author listing is as follows:

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This correction has been made to the current online version of the article, which is available at http://hyper.ahajournals.org/content/61/2/e20.full.