Angiotensin-(1-9) reduces cardiovascular and renal inflammation in experimental renin-independent hypertension

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 Hypertension-induced cardiovascular and renal damage can be mediated by activation of the renin-angiotensin-aldosterone system. There are different factors beyond renin-angiotensin-aldosterone system involved in hypertension and renal damage. Inflammation has emerged as an important mediator of hypertension and cardiovascular and kidney damage. Angiotensin-(1-9), a peptide of the renin-angiotensin system, counter-regulates both the physiological and pathological actions of angiotensin II. Recent data has shown that angiotensin-(1-9) protects the heart and blood vessels from adverse cardiovascular remodeling in experimental models of hypertension and/or heart failure and reduces cardiac fibrosis in stroke-prone, spontaneously hypertensive rats. These effects are mediated by the angiotensin II type 2 receptor (AT2R). However, it remains unknown whether angiotensin-(1-9) also has an anti-inflammatory effect. In the present study, we investigate whether angiotensin-(1-