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The contribution of brain angiotensin II to the baroreflex regulation of renal sympathetic nerve activity in conscious normotensive and hypertensive rats.

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Abstract

Angiotensin II receptor density in the brain is elevated when dietary salt intake is raised or in the state of hypertension. The aim of this study was to evaluate whether the angiotensin II modulation of the baroreceptor control of renal sympathetic nerve activity was altered under these conditions. Wistar rats, fed either a regular (0.25% w/w sodium) or high-salt diet (3.1% w/w sodium), or stroke-prone spontaneously hypertensive rats (SHRSPs) were implanted with cannulae in the carotid artery, jugular vein and the cerebroventricle and with recording electrodes on the renal sympathetic nerves. Three days later, baroreceptor gain curves were generated for renal sympathetic nerve activity and heart rate before and following intracerebroventricular (i.c.v.) administration of losartan (15 mug) to block angiotensin AT1 receptors. The rats fed a regular diet had a mean blood pressure of 116 +/- 3 mmHg and heart rate of 467 +/- 25 beats min(-1), which remained unchanged after the i.c.v. administration of losartan. The sensitivity or curvature coefficient of the baroreceptor curve for renal sympathetic nerve activity was increased by 36% ($P < 0.05$) following losartan. In the rats fed a high-salt diet, all cardiovascular variables and the losartan-induced increase in the baroreceptor curvature coefficient for renal sympathetic nerve activity (29%) were similar to values in rats on the regular sodium diet. The heart rate baroreceptor curvature coefficient was not altered in either the rats fed a regular or a high-salt diet. The slope of the renal sympathetic nerve activity baroreflex gain curve in the SHRSPs was less and the increase following administration of losartan (54%) was greater than in the Wistar rats. These data indicate that in the conscious state, the tonic inhibitory action of brain angiotensin II on the baroreflex regulation of renal sympathetic nerve activity was unaffected by raised dietary sodium, but its role was enhanced in the SHRSPs.

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