Salt preference and taste response: modification by angiotensin II

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Angiotensin II is known as the main circulating hormone of the rennin-angiotensin-aldosteron system (RAAS) that is able to act on the central nervous system (CNS) to increase sympathetic outflow, arginine vasopressin release, water intake, and salt appetite. In rodents, it is reported that systemic administration with aldosteron or other mineral corticoids increases intake of hyperosmotic sodium salts, which otherwise are strongly avoided. Interestingly, this hormone also induce greater taste nerve responses to salts which might lead to stronger avoidance. The effect of aldosteron is known to start slowly and need more than an hour to reach its maximum. Therefore, there is a possibility that two different mechanisms may be involved in the modulation by RAAS on salt preference and taste response; at the earlier stage angiotensin II may directly influence taste responses at periphery and induce salt preference at CNS and at the later stage aldosteron may greatly contribute to increase amiloride-sensitive ENaC channel activities which may cause greater salt taste responses and sodium adsorption.

To investigate this possibility, we, as the first step, examined if the taste receptor cell would be a target for angiotensin II and taste responses would be modulated by the hormone by comparing mouse taste nerve responses before and after administration with angiotensin II. Expression of AT1 and AT2 which are receptors for angiotensin II in the taste cell was examined by using a RT-PCR analysis. The results suggest that the taste cell is a target for angiotensin II which expresses AT1 and AT2. Administration with angiotensin II produced small but significant inhibition of taste nerve responses to NaCl but clear enhancement of those to sucrose. The inhibition of salt responses by this hormone at periphery may be involved in factors which may lead to behavioral intake of hyperosmotic sodium salts occurred through its action on the CNS. The enhancement of sweet sensitivity may also help to increase water intake and salt appetite. The underlining mechanisms for the action of angiotensin II in the taste cell remained for future studies.