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# Effect of Angiotensin II on Synaptic Transmission in the Rat Subfonical Organ

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Circulating substances can directly access the neurons in circumventricular organ (CVO) located on the border of cerebral ventricles. The neurons in CVO are considered to play a pivotal role in blood-brain communication. In subfonical organ (SFO), a CVO in forebrain, angiotensin II (AII) is known to reduce A type $K^+$ current and input resistance, but enhance firing rate. However, little is known on the effect of AII on the synaptic transmission in SFO.

Whole cell currents were recorded from SFO neurons of forebrain slices under an upright microscope equipped with DIC system. The slices were perfused with recording solution containing (mM): NaCl 126, KCl 5, $NaH_2PO_4 \cdot H_2O$ 1.2, $CaCl_2 \cdot 2H_2O$ 2.4, $MgCl_2$ 1.2, $NaHCO_3$ 26, glucose 10. The pipette solution contained (mM): KCl 140, $CaCl_2$ 0.5, EGTA 5, HEPES 20, MgATP 5 and biocytin (0.1%) (pH 7.2).

All the neurons recorded (n=12) showed spontaneous postsynaptic currents that were blocked by bicuculline (20 $\mu$M), a $GABA_A$ receptor antagonist, but not by CNQX (10 $\mu$M), excitatory amino acid glutamate receptor antagonist or tetrodotoxin (TTX, 1 $\mu$M), suggesting that they are spontaneous inhibitory postsynaptic currents (sIPSC). The amplitude, frequncy and decay time of sIPSCs ranged between 50 to 140 pA (83.4$\pm$7.33 pA), 1 to 19 Hz (6.22$\pm$1.94 Hz) and 10 to 30 ms (18.1$\pm$1.5 ms), respectively. AII reduced the mean amplitude and frequency of sIPSCs to 86% and 60% of the controls, respectively ( $p < 0.05$). In 5 neurons, AII produced inward current (15-105 pA). In contrast, the decay time constant of sIPSCs were not affected by AII ($10^{-7}$ M).

Our results suggest that circulating AII inhibits the release of GABA from GABAergic terminals in SFO.