Angiotensin

Angiotensin is a peptide hormone that causes vasoconstriction and an increase in blood pressure. It is part of the renin-angiotensin system, which regulates blood pressure. Angiotensin also stimulates the release of aldosterone from the adrenal cortex to promote sodium retention by the kidneys.

An oligopeptide, angiotensin is a hormone and a dipsogen. It is derived from the precursor molecule angiotensinogen, a serum globulin produced in the liver. Angiotensin was isolated in the late 1930s (first named 'angiotonin' or 'hypertensin') and subsequently characterized and synthesized by groups at the Cleveland Clinic and Ciba laboratories.

The other main brain entry of the information related to fluid and cardiovascular balance are the lamina terminalis (LT) and one of the sensory circumventricular organs (CVOs), the area postrema (AP). The LT, consisting of the median preoptic nucleus (MnPO) and the other two sensory CVOs—i.e., subfornical organ (SFO) and organum vasculosum of the lamina terminalis (OVLT) —is recognized as a site in the brain that is crucial for the physiological regulation of hydroelectrolyte balance. The SFO and OVLT lack a blood–brain barrier and contain cells that are sensitive to humoral signals, such as changes in plasma and cerebrospinal fluid sodium concentration (Vivas et al. 1990), osmolality (Sladek and Johnson 1983), and angiotensin II(ANG II) levels (Ferguson and Bains 1997; Simpson et al. 1978). Such unique features make the SFO and OVLT key brain regions for sensing the status of the body fluids and electrolytes. Humoral and neural signals that arrive to the two main brain entries—that is, the CVOs of the LT and within the hindbrain the AP-NTS—activate a central circuit that includes integrative areas such as the MnPO, the paraventricular (PVN), the supraoptic (SON), lateral parabrachial nucleus (LPBN), dorsal raphe nucleus (DRN), and neurochemical systems such as the angiotensinergic, vasopressinergic, oxytocinergic (OT), and serotonergic (5-HT) systems.

Once these signals act on the above-mentioned neurochemical networks, they trigger appropriate sympathetic, endocrine, and behavioral responses. Therefore, after a body fluid deficit, water and sodium intake and excretion need to be controlled to minimize disturbances of hydromineral homeostasis. In this context, hypovolemia and hyponatremia induced by body fluid depletion stimulate central and peripheral osmo–sodium receptors, taste receptors, volume and arterial/cardiopulmonary baroreceptors, and the renin–angiotensin system (RAS). This latter system, for example, acts mainly through the sensory CVOs and/or the AP to activate brain neural pathways that elevate BP, release vasopressin and aldosterone (ALDO), increase renal sympathetic nerve activity, and increase the ingestion of water and sodium [1)].

Angiotensin-converting enzyme

Angiotensin-converting enzyme.

Angiotensin-converting enzyme inhibitor

see Angiotensin-converting enzyme inhibitor.

1)
Vivas L, Godino A, Dalmasso C, Caeiro XE, Macchione AF, Cambiasso MJ. Neurochemical Circuits Subserving Fluid Balance and Baroreflex: A Role for Serotonin, Oxytocin, and Gonadal Steroids. In: De Luca LA Jr, Menani JV, Johnson AK, editors. Neurobiology of Body Fluid Homeostasis: Transduction and Integration. Boca Raton (FL): CRC Press/Taylor & Francis; 2014. Chapter 9. PubMed PMID: 24829993.