Angiotensin II

Angiotensin I is converted to angiotensin II (AII) through the removal of two C-terminal residues by the enzyme angiotensin-converting enzyme (ACE), primarily through ACE within the lung (but also present in endothelial cells, kidney epithelial cells, and the brain). Angiotensin II acts on the central nervous system to increase vasopressin production, and also acts on venous and arterial smooth muscle to cause vasoconstriction. Angiotensin II also increases aldosterone secretion; it, therefore, acts as an endocrine, autocrine/paracrine, and intracrine hormone.

Iron overload plays a key role in secondary bleeding after ICH in Angiotensin II-induced hypertensive mice. Iron chelation during the process of Ang II-induced hypertension suppresses secondary bleeding after ICH [1)].

Angiotensin II receptor

Angiotensin II receptor

1)
Wang J, Tang XQ, Xia M, Li CC, Guo C, Ge HF, Yin Y, Wang B, Chen WX, Feng H. Iron chelation suppresses secondary bleeding after intracerebral hemorrhage in angiotensin II-infused mice. CNS Neurosci Ther. 2021 Aug 4. doi: 10.1111/cns.13706. Epub ahead of print. PMID: 34346561.