

Neuropathic pain is one of the most debilitating disorders. Currently available treatments for neuropathic pain are still unsatisfactory as they have only limited treatment effect and patients may suffer from unwanted side effects. Mechanism-based approaches to neuropathic pain treatment are considered to be more effective. Therefore multiple studies are dedicated to study the pathophysiological mechanisms of neuropathic pain. One of the possible underlying mechanism that causes neuropathic pain is neuroinflammation. Recent studies suggested that angiotensin II (main effector molecule of the renin-angiotensin system) via its receptors in the central nervous system may be involved in the neuroinflammatory processes. The aim of this study was to investigate the role of angiotensin receptor type 1 in the development and maintenance of neuropathic pain induced in animal model. Spinal nerve ligation (L5) was used as a model of peripheral neuropathy. Our results showed that treatment with AT₁R blocker losartan markedly reduced thermal hyperalgesia and reduced increased sensitivity to mechanical stimuli in the SNL-operated rats. This indicates a possibly significant role of AT₁ receptors in the development of neuropathic pain, probably due to reduction of neuroinflammation in the nervous system. These findings and further study of the mechanisms by which AT₁R modulate neuroinflammation during peripheral neuropathy may bring new therapeutic approaches for neuropathic pain treatment.