



Increased Expression Of Toll-Like Receptor 2 Mrna Following Permanent Middle Cerebral Artery Occlusion In Rat: Role Of TRPV1 Receptors

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Background: Stroke is a major cause of mortality and long term disability in adults. TRPV1 has a pivotal role in neuroinflammation. Among TLRs, TLR2 significantly participate in induction of inflammation in brain. In this study, the effect of TRPV1 receptor agonist and antagonist on outcome and gene expression of TLR2 in a rat model of permanent middle cerebral artery occlusion (MCAO) was investigated.

Methods: Forty male rats were assigned to the following groups: sham, vehicle (stroke), AMG9810 (selective TRPV1 antagonist, 0.5 mg/kg; 3 h after stroke), and capsaicin (1 mg/kg; 3 h after stroke). Stroke was induced by permanent middle cerebral artery occlusion and behavioral functions were assessed 1, 3, and 7 days after stroke. Infarct volume, brain edema and mRNA expression of TLR2 were also evaluated at the end of the study.

Results: While stroke animals showed infarctions and behavioral functions, we did not observe any cerebral infarction and behavioral functions in sham-operated animals. AMG9810 decreased neurological deficits 7 days after cerebral ischemia ($P < 0.01$). In the ledged beam-walking test, the slip ratio was increased following ischemia ($*P < 0.05$). AMG9810 improved this index in animals undergone stroke. However, capsaicin enhanced the slip ratio 3 and 7 days after cerebral ischemia ($\#P < 0.05$). TLR2 $P < 0.05$ (mRNA expression was elevated in ischemic rats).

Conclusion: Our data indicate that pharmacological blockade of TRPV1 by AMG9810 attenuates behavioral function and mRNA expression of TLR2. Therefore, it might be useful as a potential target for the treatment of ischemic stroke.

Key words: Cerebral ischemia, TLR2, TRPV1, Inflammation



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