

# Ischemia reperfusion injury

see [Cerebral ischemia-reperfusion injury](#).

see [Spinal cord ischemia reperfusion injury](#).

Previous studies have shown that Ischemia [reperfusion injury](#) (IRI) contributes to the injury process in the central nervous system (CNS), through the activation of the [immune system](#) <sup>1)</sup> <sup>2)</sup> <sup>3)</sup> <sup>4)</sup> <sup>5)</sup>.

<sup>1)</sup>

Gökce EC, et al. Neuroprotective effects of [thymoquinone](#) against spinal cord ischemia-reperfusion injury by attenuation of inflammation, oxidative stress, and apoptosis. *J Neurosurg Spine*. 2016;24(6):949–959. doi: 10.3171/2015.10.SPINE15612.

<sup>2)</sup>

Dimitrijevic OB, Stamatovic SM, Keep RF, Andjelkovic AV. Absence of the chemokine receptor CCR2 protects against cerebral ischemia/reperfusion injury in mice. *Stroke*. 2007;38(4):1345–1353. doi: 10.1161/01.STR.0000259709.16654.8f.

<sup>3)</sup>

Chen Y, et al. Overexpression of monocyte chemoattractant protein 1 in the brain exacerbates ischemic brain injury and is associated with recruitment of inflammatory cells. *J Cereb Blood Flow Metab*. 2003;23(6):748–755.

<sup>4)</sup>

Smith PD, et al. The evolution of chemokine release supports a bimodal mechanism of spinal cord ischemia and reperfusion injury. *Circulation*. 2012;126(11 Suppl 1):S110–S117.

<sup>5)</sup>

Strecker JK, Minnerup J, Gess B, Ringelstein EB, Schäbitz WR, Schilling M. Monocyte chemoattractant protein-1-deficiency impairs the expression of IL-6, IL-1 $\beta$  and G-CSF after transient focal ischemia in mice. *PLoS One*. 2011;6(10):e25863 doi: 10.1371/journal.pone.0025863.

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