

Experimental spinal cord injury treatment

Cui et al. from the Department of Neurosurgery Department of Pathology, Baoding First Central Hospital, [Baoding](#), Hebei Province, China evaluated the neuroprotective [efficacy](#) of [fisetin](#) against the [experimental model of spinal cord injury](#) (SCI).

SCI was induced in male [Sprague Dawley rats](#) by placing an aneurysm [clip extradural](#). [Rats](#) were treated either with vehicle or fisetin for 28 days after SCI.

Treatment with fisetin significantly attenuated SCI-induced alterations in mechano-tactile and thermal allodynia, hyperalgesia, and nerve conduction velocities. SCI-induced upregulated [TNF \$\alpha\$](#) , [interleukins](#), inducible [nitric oxide synthase](#), [cox-2](#), [Bcl-2](#)-associated X protein and [caspase 3](#) mRNA expressions in the spinal cord and these were markedly reduced by fisetin. Spinal [nuclear factor kappa B](#) and nuclear factor of kappa light polypeptide gene enhancer in [B-cells](#) inhibitor-alpha protein levels were also significantly downregulated by fisetin. Hematoxylin and eosin staining of the [spinal cord](#) suggested that fisetin significantly ameliorated histological aberrations such as neuronal degeneration, necrosis, and inflammatory infiltration induced in it.

Fisetin exerts [neuroprotection](#) via modulation of nuclear factor kappa B/nuclear factor of kappa light polypeptide gene enhancer in B-cells inhibitor-alpha pathway by inhibiting release of inflammatory mediators (inducible nitric oxide synthase and cyclooxygenase-II), proinflammatory cytokines (tumor necrosis factor-alpha and interleukins), apoptotic mediators ([Bcl-2](#)-associated X protein and caspase-3) ¹⁾.

¹⁾

Cui J, Fan J, Li H, Zhang J, Tong J. Neuroprotective potential of fisetin in an experimental model of spinal cord injury: via modulation of NF- κ B/I κ B α pathway. *Neuroreport*. 2021 Jan 18. doi: 10.1097/WNR.0000000000001596. Epub ahead of print. PMID: 33470764.

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