

# Nur77

Mitochondrial fragmentation drastically regulates mitochondrial homeostasis in brain illness. However, the role of mitochondrial fragmentation in cerebral ischemia reperfusion injury remains unclear. Nur77, a regulator of mitochondrial homeostasis, is associated with heart and liver IR injury, but its effects on mitochondrial function in cerebral IR injury has not been studied intensively.

The aim of a study of Zhao et al. from the Department of Neurosurgery, People's Liberation Army General Hospital, No. 5 Nanmencang Hutong, Dongcheng District, Beijing, was to explore whether cerebral IR injury is modulated by Nur77 via modification of mitochondrial homeostasis. Our results indicated that Nur77 was upregulated in reperfused brain tissues. Genetic ablation of Nur77 reduced infarction area and promoted neuron survival under IR burden. Biochemical analysis demonstrated that Nur77 deletion protected mitochondrial function, attenuated mitochondrial oxidative stress, preserved mitochondrial potential, and blocked mitochondria-related cell apoptosis. In addition, we illustrated that Nur77 mediated mitochondrial damage via evoking mitochondrial fragmentation that occurred through increased mitochondrial fission and decreased fusion. Besides, this results also demonstrated that Nur77 controlled mitochondrial fragmentation via upregulating INF2 in a manner dependent on the Wnt/ $\beta$ -catenin pathway; inhibition of the Wnt pathway abrogated the protective effect of Nur77 deletion on reperfused-mediated neurons. Altogether, this study highlights that the pathogenesis of cerebral IR injury is associated with Nur77 activation followed by augmented mitochondrial fragmentation via an abnormal Wnt/ $\beta$ -catenin/INF2 pathway. Accordingly, Nur77-dependent mitochondrial fragmentation and the Wnt/ $\beta$ -catenin/INF2 axis may represent novel therapeutic targets to reduce cerebral IR injury <sup>1)</sup>.

<sup>1)</sup>  
Zhao H, Pan W, Chen L, Luo Y, Xu R. Nur77 promotes cerebral ischemia-reperfusion injury via activating INF2-mediated mitochondrial fragmentation. J Mol Histol. 2018 Oct 8. doi: 10.1007/s10735-018-9798-8. [Epub ahead of print] PubMed PMID: 30298449.

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