

In a study, Wu et al. used the transient [middle cerebral artery occlusion](#) (tMCAO) [mice model](#) to investigate the role of circCCDC9 in [stroke pathogenesis](#). They found that the expression of [circCCDC9](#) was significantly decreased in the brains of tMCAO mice. The [Evans blue](#) and brain water content were significantly higher in the Pre-IR and Pre-IR+Vector mice, while these patterns were partially reversed by overexpression of circCCDC9. The [nitrite](#) content and eNOS expression were decreased in the Pre-IR and Pre-IR+Vector groups, which was restored by circCCDC9 overexpression. Overexpression of circCCDC9 also inhibited the expression of [Caspase 3](#), [Bax/Bcl-2](#) ratio and the expression of [Notch1](#), [NICD](#) and [Hes1](#) in tMCAO mice. [Knockdown](#) of circCCDC9 increased the expression of [Caspase-3](#), [Bax/Bcl-2](#) ratio, and the expression of [Notch1](#), [NICD](#), and [Hes1](#). In summary, overexpression of circCCDC9 protected the [blood-brain barrier](#) and inhibited [apoptosis](#) by suppressing the [Notch1](#) signaling pathway, while knockdown of circCCDC9 had the opposite effects. The findings showed that circCCDC9 is a potential novel therapeutic target for cerebrovascular protection in [acute ischemic stroke](#) ¹⁾.

¹⁾

Wu L, Xu H, Zhang W, Chen Z, Li W, Ke W. Circular RNA circCCDC9 alleviates ischaemic stroke ischaemia/reperfusion injury via the Notch pathway. *J Cell Mol Med*. 2020 Oct 29. doi: 10.1111/jcmm.16025. Epub ahead of print. PMID: 33124180.

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