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].

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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.