

As highly conserved endogenous non-coding RNAs, miRNAs are highly conserved endogenous non-coding RNAs, which mainly regulate the expression of protein-coding genes [12]. Neuron necrosis is the a main feature of acute ischemic stroke. Neuron necrosis and mainly occurs in the central area of cerebral infarction, but although it can also be observed exists in the ischemic penumbra around the central area of cerebral infarction. In ischemic penumbra, cCells are in a semi-dormant state and can be transformed into normal brain tissues. Therefore, rescuing the rescue of penumbra has become a hot spot of the current research [13,14]. In this study, rats were divided into three groups. The results showed that compared with the rats in the Sham group, the content of CSF miR-151a-3p in CSF was increased in the rats of the I/R group and ASO-miR-151a-3p treatment groups compared with Sham group, but the content of CSF miR-151a-3p in the of ASO-miR-151a-3p treatment group was decreased lower than that in compared with the I/R group, with statistical significance ($P < 0.05$). The results of neurological grading score, cerebral infarction area observation, brain edema and apoptosis analysis analysis of the three groups of rats showed that although the content of CSF miR-151a-3p in CSF of the I/R model group was significantly higher than that of in the Sham group, the content of CSF miR-151a-3p in CSF of the ASO-miR-151a-3p treatment group was lower than that of in the I/R group, with statistical significance ($P < 0.05$). Therefore, it can be inferred that blocking the silencing of miR-151a-3p can protect prevent the nerve injury caused by cerebral ischemia reperfusion I/R.

Recent studies It has been have shown that miRNAs also play a very important role in the pathogenesis of inflammation. For example, miR-155 and miR-146a are highly expressed in macrophages stimulated by LPS, while miR-155 can activate IKK (I κ B kinase) and NF- κ B, which in turn lead to the release of downstream inflammatory factors and play a role [15,16]. In addition, studies have shown that, in addition, miR-34a, miR1224 and miR-151a have been proved shown to play an important regulatory role in inflammatory response [17,18]. In this study,