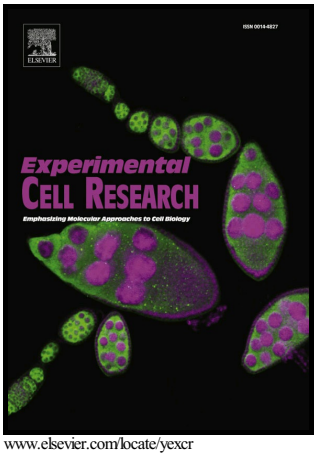
Author's Accepted Manuscript



www.elsevier.com/locate/yexcr

MiR-138/SIRT1 axis is implicated in impaired learning and memory abilities of cerebral ischemia/reperfusion injured rats

Feng Tian, Chao Yuan, Hongmei Yue

PII: S0014-4827(18)30200-3
DOI: https://doi.org/10.1016/j.yexcr.2018.03.042
Reference: YEXCR10992

To appear in: *Experimental Cell Research*

Received date: 8 January 2018
Revised date: 22 March 2018
Accepted date: 30 March 2018

Cite this article as: Feng Tian, Chao Yuan and Hongmei Yue, MiR-138/SIRT1 axis is implicated in impaired learning and memory abilities of cerebral ischemia/reperfusion injured rats, *Experimental Cell Research,* https://doi.org/10.1016/j.yexcr.2018.03.042

MiR-138/SIRT1 axis is implicated in impaired learning and memory abilities of cerebral ischemia/reperfusion injured rats

Feng Tian[1], Chao Yuan[1], Hongmei Yue[*]

Department of Neurology, Jining No.1 People's Hospital

[*]Correspondence to: Department of Neurology, Jining No.1 People's Hospital, No.6 Jiankang Road，Jining City，Shandong Province, China.
yhmthsd@163.com.

Abstract

The present study aimed to explore whether deregulated miR-138 is implicated in cerebral I/R injury-impaired learning and memory abilities. Rats were subjected to bilateral common carotid occlusion followed by reperfusion to induce cerebral I/R injury. A model of oxygen-glucose deprivation and reperfusion (OGD/R) was conducted to mimic cerebral I/R conditions in vitro. MiR-138 expression levels were reduced in the hippocampus of cerebral I/R injured rats. Inhibition of miR-138 ameliorated the impaired learning and memory abilities of rats, and promoted autophagy and thus attenuated apoptosis in the OGD/R-treated hippocampal neurons. Moreover, miR-138 targets the 3'-UTR of SIRT1 and repressed its expression. These results showed that miR-138 could improve the learning and memory abilities via promoting autophagy under cerebral I/R injured conditions.

Keywords: cerebral ischemia/reperfusion injury; bilateral common carotid occlusion; hippocampus; learning; memory; autophagy; SIRT1

[1]These authors contributed equally to this work and should be considered as co-first authors.