

## A microRNA-152 that targets the phosphatase and tensin homolog to inhibit low oxygen induced-apoptosis in human brain microvascular endothelial cells

Y.H. Cao<sup>1\*</sup>, D.G. Li<sup>2\*</sup>, B. Xu<sup>1</sup>, M.Q. Wang<sup>1</sup>, N. Zhen<sup>1</sup>, L.X. Man<sup>1</sup>, Y.Y. Zhang<sup>1</sup> and M. Chi<sup>1</sup>

<sup>1</sup>Department of Pediatrics, General Hospital of Military Command Jinan, Jinan, Shandong, China <sup>2</sup>Department of Pediatric Surgery, The Second Affiliated Hospital of Shandong University, Jinan, Shandong, China

\*These authors contributed equally to this study. Corresponding author: Y.H. Cao E-mail: caoyanhuayelei@sina.com

Genet. Mol. Res. 15 (2): gmr.15027371 Received October 13, 2015 Accepted November 19, 2015 Published May 13, 2016 DOI http://dx.doi.org/10.4238/gmr.15027371

ABSTRACT. Brain damage caused by perinatal asphyxia is dangerous for neonatal infants, but the mechanism by which it occurs remains elusive. In this study, microRNA-152 (miR-152) expression was induced by low oxygen levels in rat models of hypoxia brain damage, as well as in human brain microvascular endothelial cells (HBMECs) cultured *in vitro*. Analysis of the sequence of miR-152 revealed that the phosphatase and tensin homolog gene (*PTEN*) is probably the target of miR-152 both in humans and rats. When HBMECs were transfected with miR-152 mimics, *PTEN* expression was inhibited at both the mRNA and protein levels. Moreover, transfection with the miR-152 mimic also inhibited apoptosis induced by hypoxia. Furthermore, expression of the pro-apoptotic gene *Bax* was downregulated while the anti-apoptotic

gene *Bcl2* was upregulated after miR-152 mimic transfection. Taken together, these results indicate that miR-152 induced by hypoxia suppresses cell apoptosis and acts as a protective factor during hypoxia by repressing *PTEN*.

**Key words:** miR-152; *PTEN*; Hypoxia; Apoptosis; Brain damage