Mechanism of SEMA3B gene silencing and clinical significance in glioma

C.H. Pang, W. Du, J. Long and L.J. Song

Department of Neurosurgery, The First Affiliated Hospital, Zhengzhou University, Zhengzhou, China

Corresponding author: L.J. Song
E-mail: songlaijun99@163.com

Received September 17, 2015
Accepted January 4, 2016
Published March 18, 2016
DOI http://dx.doi.org/10.4238/gmr.15017664

ABSTRACT. The aim of the current study was to explore mechanisms of SEMA3B gene expression and its clinical significance in glioma, and provide a theoretical foundation for investigating individualized treatment in glioma. Paraffin-embedded tissues from 43 patients with a confirmed clinical diagnosis of glioma following neurosurgery at the First Affiliated Hospital of Zhengzhou University from December 2013 to April 2014 were selected randomly. An additional three normal brain tissues were obtained following encephalic decompression excision due to acute craniocerebral injury in the same period, which were used as the control group. Immunohistochemical staining for vascular endothelial growth factor was performed on the glioma tissues from the 43 patients. Genomic DNA was extracted for bisulfate conversion and sequencing. SEMA3B was fully expressed in the three normal brain tissues, and incompletely expressed in the 43 glioma tissues, with a lack of expression in 48.8% (21/43) of samples. Moreover, 58% of high-grade gliomas (grade III and IV) lacked SEMA3B expression, which was significantly more than those that lacked expression (20%) in low-grade gliomas (grade I and II), indicating that, as the clinical pathological grade increased, SEMA3B expression decreased. The occurrence and development of malignant tumors is a product of multiple genes and other factors. Here, we provide theoretical basis for
glioma development and prognosis involving DNA-methylation driven silencing of SEMA3B, and thus, SEMA3B is a potential target for directed treatments against glioma.

**Key words:** SEMA3B; Glioma; Immunohistochemistry; DNA methylation