Genetics and Molecular Research

Dysregulation of TFDP1 and of the cell cycle pathway in high-grade glioblastoma multiforme: a bioinformatic analysis

X. Lu[1], X.D. Lv[1], Y.H. Ren[1], W.D. Yang[1], Z.B. Li[1], L. Zhang[1] and X.F. Bai[2]

[1]Department of Neurosurgery, Mudanjiang City Second People's Hospital, Mudanjiang, China
[2]Research Service Office, Mudanjiang City Second People's Hospital, Mudanjiang, China

Corresponding author: X.F. Bai
E-mail: xuefengb2y@sina.com

Genet. Mol. Res. 15 (2): gmr.15027646
Received September 16, 2015
Accepted December 9, 2015
Published June 10, 2016
DOI http://dx.doi.org/10.4238/gmr.15027646

ABSTRACT. Despite extensive research, the prognosis of high-grade glioblastoma multiforme (GBM) has improved only slightly because of the limited response to standard treatments. Recent advances (discoveries of molecular biomarkers) provide new opportunities for the treatment of GBM. The aim of the present study was to identify diagnostic biomarkers of high-grade GBM. First, we combined 3 microarray expression datasets to screen them for genes differentially expressed in patients with high-grade GBM relative to healthy subjects. Next, the target network was constructed via the empirical Bayesian coexpression approach, and centrality analysis and a molecular complex detection (MCODE) algorithm were performed to explore hub genes and functional modules. Finally, a validation test was conducted to verify the bioinformatic results. A total of 277 differentially expressed genes were identified according to the criteria $P < 0.05$ and $|\log_2(\text{fold change})| \geq 1.5$. These genes were most

significantly enriched in the cell cycle pathway. Centrality analysis uncovered 9 hub genes; among them, *TFDP1* showed the highest degree of connectivity (43) and is a known participant in the cell cycle pathway; this finding pointed to the important role of *TFDP1* in the progression of high-grade GBM. Experimental validation mostly supported the bioinformatic results. According to our study results, the gene *TFDP1* and the cell cycle pathway are strongly associated with high-grade GBM; this result may provide new insights into the pathogenesis of GBM.

Key words: Glioblastoma multiforme; Bioinformatics; Centrality analysis; Coexpression network

