



Tumor Promoter Bioinformatics Analysis in Glioma

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BIOINFORMATICS

Gliomas are the most frequent kind of intracranial primary malignant tumour, accounting for 80% of all primary malignant tumours of the central nervous system. Currently, the only recommended therapies for glioma are surgical resection with adjuvant radiotherapy and temozolomide adjuvant chemotherapy combined with radiotherapy. However, patients' overall survival is barely 15 months. Glioma is resistant to radiotherapy and chemotherapy, and this malignant behaviour leads to a high recurrence rate. As a result, treatments are typically ineffectual. As a result, people with glioma do not gain considerably from traditional treatment (Kaper et al., 2004). As a result, there is an urgent need to develop novel diagnostic approaches and, in particular, more effective treatment options. The use of gene expression microarrays provides a realistic and effective method for studying gliomas. The current work aims to use bioinformatics tools to identify the major protein-coding genes of glioblastoma and hence hunt for novel biomarkers and therapeutic targets for the treatment of glioma (Aranda et al., 2004). First, mRNA microarray datasets from the Gene Expression Omnibus collection were used to identify differentially expressed genes (DEGs) between gliomas and normal tissues. Gene Ontology (GO), the Kyoto Encyclopedia of Genes and Genomes (KEGG), the Protein-Protein Interaction (PPI) network, and statistical analysis were used to identify DEGs. Following that reverse transcription-quantitative PCR (RT-qPCR) and western blot were employed to confirm the bioinformatics analysis results. Gliomas are very aggressive and deadly solid tumours of the primary central nervous system that arise from glial cells. The most prevalent type of primary intracranial malignant tumour is glioma, which accounts for 51.4% of all primary brain tumours. Furthermore, glioma is responsible for 80% of initial malignant tumours of the central nervous system (Ishii et al., 2007). The World Health Organization (WHO) divided glioma into grades I-IV

in 2016, based on malignancy, active mitosis, and necrosis. WHO grades I/II denote low-grade gliomas, whereas WHO grades III/IV denote high-grade gliomas, the aggressiveness of which is growing. Each grade has a relative specificity that can be used to guide subsequent clinical treatment. As computer technology becomes more common and advanced, the number of high-throughput platforms for gene expression analysis grows (Nweze 2009). Microarray analysis and next-generation sequencing technologies are becoming increasingly significant in oncology care and have been shown to have a wide range of clinical uses. Microarray analysis on high-throughput platforms is a powerful method for detecting virtual genetic or epigenetic changes in cancer progression. Microarray analysis is also frequently utilised in cancer diagnosis and prognosis. Large-scale gene expression profiles can be used to uncover transcriptional networks linked to up-regulated signalling pathways. This method also enables for the identification of previously unknown tumour subtypes with distinct genetic and/or clinical phenotypes or treatment responses. Gene expression profiles can also be used to identify specific mutations that are associated with distinct molecular features. Several tumor-related databases have previously been built (Albert et al., 2009). Over the last decade, the Gene Expression Omnibus (GEO) database has emerged as the principal public repository for high-throughput microarray and sequence-based functional genomic datasets. Furthermore, in addition to archiving, cross-linking, and making massive amounts of data available for free download, GEO provides various user-friendly web-based tools and policies to assist users in querying and analysing the data. Furthermore, the applications of bioinformatics analysis are constantly evolving. As a result, genetic molecular diagnosis and genetic analysis play an essential part in disease. Microarrays of gene expression enable to detect differentially expressed genes (DEGs) in tumour and normal tissues. These genes could be useful indicators for cancer detection and treatment. However, no significant glioma molecular targets have been discovered.

Bioinformatics analysis is a method of discovering potential biomarkers associated with diseases by using different bioinformatics analysis tools, such as gene ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) analysis. The current work used bioinformatics analysis of the GEO database to look for protein-coding genes that were differentially expressed between glioma and normal tissues. To identify the biological roles of the identified genes, bioinformatics analytical tools such as GO functional enrichment analysis were applied. To identify the hub genes, a protein-protein interaction (PPI) network of DEGs was built. Data from the GSE108474, GSE109857, and GSE116520 datasets were used in this investigation to screen 400 frequent DEGs in normal and glioma patient tissues. The functions and enrichment pathways of the DEGs were then investigated. Furthermore, DEG PPI networks were built to discover hub genes. To validate the results, the major genes identified were subjected to reverse transcription-quantitative PCR (RT-qPCR). A combination of temozolomide and radiation improves glioma prognosis; yet, few patients live longer than 5 years. There is thus an urgent need to discover a novel therapeutic strategy for the treatment of glioblastoma. The current study sought to

uncover accurate biomarkers in glioma in order to enhance patient diagnosis and predict survival, as well as to identify novel therapeutic targets.

REFERENCES

1. Kaper JB, Nataro JPT, Mobley HLT (2004). Pathogenic *Escherichia coli*. *Nat Rev Microbiol.* 2: 123-140.
2. Aranda KRS, Fagundes-Neto U, Scaletsky ICA (2004). Evaluation of multiplex PCRs for diagnosis of infection with diarrheagenic *Escherichia coli* and *Shigella* spp. *J Clin Microbiol.* 42: 5849-5853.
3. Ishii S, Meyer KP, Sadowsky MJ (2007). Relationship between phylogenetic groups, genotypic clusters, and virulence gene profiles of *Escherichia coli* strains from diverse human and animal sources. *Appl Environ Microbiol.* 73: 5703-5710.
4. Nweze EI (2009). Virulence Properties of Diarrheagenic *E. coli* and Etiology of Diarrhea in Infants, Young Children and Other Age Groups in Southeast, Nigeria. *Am-Eurasian J Sci Res.* 4: 173-179.
5. Albert MJ, Rotimi VO, Dhar R, Silpikurian S, Pacsa AS, et al (2009). Diarrhoeagenic *Escherichia coli* are not a significant cause of diarrhoea in hospitalised children in Kuwait. *BMC Microbiol.* 9: 62.