Bioinformatics Analysis of Potential Key Genes and Pathways in Pediatric Patients with Langerhans Cell Histiocytosis

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Abstract

Background: Langerhans cell histiocytosis (LCH) is the most common benign tumor in children, however its etiology remains incompletely understood. This aim of this study was to employ bioinformatics methods to identify differentially expressed genes (DEGs) in LCH patients. The identification of these DEGs can offer novel insights and research directions for the prevention, early diagnosis, and treatment of LCH.

Methods: Gene expression data from GSE122476 was retrieved from the Gene Expression Omnibus (GEO) database. Using the R language, differentially expressed genes (DEGs) were identified between normal blood samples and bone tissue samples from LCH patients. The DEGs were subjected to functional and pathway analysis utilizing the DAVID database. The functional analysis, pathway enrichment, and protein-protein interactions of these genes were further examined using DAVID and STRING. To identify core network genes and significant protein interaction modules, Cytoscape software was employed.

Results: A total of 229 DEGs were identified, comprising 107 up-regulated genes and 122 down-regulated genes. GO and KEGG enrichment analyses showed that the up-regulated DEGs were involved in processes such as immune response, hypoxia response, cell apoptosis, lipid antigen binding, and membrane composition. On the other hand, down-regulated DEGs were associated with inflammation, positive regulation of MAP kinase activity, and serine-type endopeptidase inhibitor activity. The KEGG enrichment analysis showed that the up-regulated DEGs exhibited significant enrichment in hematopoietic cell lineage, the interaction between viral proteins and cytokines and their receptors, TNF signaling pathway, cell apoptosis, and Th1, Th2, and Th17 cell differentiation. Conversely, the down-regulated DEGs were significantly enriched in cytokine receptor interaction, lysosome, and the PI3K-AKT signaling pathway. Further analysis using the STRING database resulted in the identification of 10 key genes within the protein network interaction map. These genes included TNF, CD69, IL7R, CCL4, IL3RA, CD1C, TNFRSF9, HAVCR2, IL2RG, and GZMA.

Conclusion: In this study, 10 DEGs were identified as potential candidate diagnostic biomarkers for LCH patients. While these DEGs hold promise for their potential utility in LCH diagnosis, it is important to note that further experiments are required to validate their functional pathways and elucidate their roles as hub genes associated with LCH.

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