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RESEARCH ARTICLE

Study of *TNIP1*, *CSK*, and *BANK1* gene expression in systematic lupus erythematosus in Iraqi children

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Abstract

SLE, or systemic lupus erythematosus in children, is a very severe long-term autoimmune disease where the body's own tissues are attacked by the immune system, and can lead to lupus nephritis, cardiovascular problems, and neurological complications. The study aims to determine the changes in gene expression of *TNIP1*, *CSK* and *BANK1* in children with SLE and to compare them with healthy controls, thereby contributing to the understanding of lupus pathogenesis. The total number of samples is 75 serum samples, of which 50 were collected from children attending hospitals who had clinical signs of lupus. In addition, 25 samples were collected from healthy children. The samples were kept in TRIzol to preserve the mRNA. The mRNA was transcribed into cDNA for the determination of gene expression. The results showed that the distribution of SLE in females (66%) was higher than the distribution of SLE in males (34%). The gene expression of *TNIP1* and *CSK* was significantly decreased in SLE children compared to healthy children. At the same time, our work demonstrated that gene expression of the *BANK1* gene was significant increased compared to that of healthy children. In conclusion, the gene expression of *TNIP1*, *CSK*, and *BANK1* differs in SLE children compared to healthy children, which may assist researchers and clinicians in using these genes as potential biomarkers of SLE detection. The findings are useful for understanding the molecular aspects of the disease and may in the future contribute to improved diagnostic and treatment approaches targeting these genes.

Keywords: *BANK1*, children, *CSK*, lupus, *TNIP1*

INTRODUCTION

Systemic lupus erythematosus (SLE) is a multifaceted autoimmune disorder, which is typified by dysregulated immune reaction and consequently causes chronic inflammation and damage to most organs (Charras et al., 2021). Compared to adult-onset disease, childhood-onset SLE is usually accompanied by more severe clinical manifestations and a higher risk of organ involvement, making it crucial to comprehend the molecular pathways underlying disease pathogenesis in this group of individuals (Sandling et al., 2018; Nelson et al., 2022). Genetic predisposition is a major cause of SLE (Smith et al., 2023). Numerous genes affect immunological control and inflammatory cascade

TNIP1 (Tumor necrosis factor-α-induced protein 3-interacting protein 1) is a gene that encodes the *ABIN-1* protein (A20 binding inhibitor of nuclear factor -1) and mediates the NF-kB (nuclear factor kappa B NF-kB-1) pathway, which is vital in immune and inflammatory reactions. *TNIP1* hypomorphic variants have been found to lower its expression, leading to sustained NF-kB activation and increased inflammation in SLE (Liu et al., 2018). *BANK1* (B Cell Scaffold Protein with Ankyrin Repeats 1) is a scaffold protein that regulates B cell receptor signalling. *BANK1* has also been linked with disrupted B cell signalling dynamics, causing autoantibody dysregulation in lupus (Dam et al., 2016). *CSK* (C-Terminal Src Kinase) may affect the regulation of Src (non-receptor tyrosine kinases)

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