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In Silico Identification of Hub Genes and Molecular Mechanisms in Temporal Lobe Epilepsy via Weighted Gene Co-expression Network Analysis

Mustafa Makav [1], Hüseyin Avni Eroğlu [2], Cemre Aydeğer [3*], Barış Kaban [4], Murat Kayabaş [5] and Turgut Dolanbay [6]

[1]Kafkas University, Faculty of Veterinary Medicine, Department of Physiology, Kars, Türkiye; [2,3]Çanakkale Onsekiz Mart University Faculty of Medicine, Department of Physiology, Çanakkale, Türkiye; [4]Kafkas University, Faculty of Medicine, Department of Emergency Medicine, Kars, Türkiye; [5]Sincan Training and Research Hospital, Department of Neurosurgery, Ankara, Türkiye; [6]Malatya Turgut Özal University, Faculty of Medicine, Department of Emergency Medicine, Malatya, Türkiye
*Correspondence: cemre.ayd@hotmail.com

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ABSTRACT

Introduction: Temporal lobe epilepsy (TLE) that is often resistant to medication is the most prevalent type of focal epilepsy in adults. This study aimed to identify the genetic networks and fundamental molecular mechanisms involved in the pathogenesis of TLE using *in silico* methods, thereby defining potential target genes (hub genes) for novel therapeutic approaches. Methods: The GSE6947 dataset, obtained from the NCBI Gene Expression Omnibus (GEO) database, was used in this study. Weighted Gene Co-expression Network Analysis (WGCNA) was performed on a total of 129 samples and 5000 genes using a systems biology approach. Biological processes were identified through Gene Ontology (GO) enrichment analysis. To detect central genes via the Protein-Protein Interaction (PPI) network, Cytoscape software and the Maximal Clique Centrality (MCC) algorithm were employed. Furthermore, hub gene candidates were validated through Module Membership (MM) analysis. Results: WGCNA identified 18 co-expression modules, among which the Pink Module was directly associated with synaptic transmission and neurotransmitter release and demonstrated the strongest correlation with epilepsy. Based on the MCC scores, DCTN1, UBE2E1, UBE2E2, KLC1, UBE2E3, NEDD4L, STAT3, GABARAPL1, CYCS, and DCTN3 were identified as the top 10 hub genes. In the MM analysis, UBE2E2, RAB6B, PPP3CB, and CYFIP2 exhibited the highest values. Conclusion: The results of the analysis indicate that synaptic dysfunction, neuroinflammation (STAT3), mitochondrial stress (CYCS), and axonal transport mechanisms (DCTN1, DCTN3, KLC1) play critical roles in the pathophysiology of TLE. These identified hub genes are strong candidates for understanding the molecular mechanisms of TLE and for developing novel biomarkers and therapeutic strategies.

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INTRODUCTION

Epilepsy is a common and complex neurological disorder marked by repeated, spontaneous seizures caused by irregular, coordinated neuronal activity in the brain (Bandopadhyay et al., 2021; Kaya et al., 2025; Prasanna et al., 2021). Temporal lobe epilepsy (TLE) is the most common form of focal epilepsy in adults originating primarily from the temporal lobes, often involving mesial temporal structures, including the hippocampus. It accounts for approximately one third of all diagnosed epileptic patients and frequently presents as medically refractory, resistant to anti-epileptic drugs (Martinez and Peplow, 2023). The disorder is often chronic and can lead to significant cognitive impairments, particularly in temporal lobe-related functions; however, deficits in extratemporal functions and global intelligence may also be observed. These impairments may precede epilepsy

onset and worsen with disease progression, due to structural brain damage and the cumulative impact of seizures and other neurological insults (Helmstaedter and Kockelmann, 2006)

TLE seizures related to hippocampal sclerosis are induced following perinatal injury, febrile convulsions, or other brain insults. Diagnosing the condition generally involves a combination of clinical assessment, reviewing the patient's medical history, analyzing electroencephalogram results, and conducting brain imaging studies. On the other hand, MRI abnormalities like hippocampal atrophy may be absent at onset and develop over time (Berg, 2008; Martinez and Peplow, 2023). Clinically, patients with TLE experience complex partial seizures, often with impaired awareness. Moreover, psychiatric comorbidities, including depression, are commonly seen. (Garcia, 2011). The underlying neurobiology involves synaptic reorganization and neuronal loss in hippocampal circuits, leading to increased neuronal excitability and seizure generation (Ren and Curia, 2021; Sutula, 1990).

TLE causes significant molecular changes in brain tissue. Previous studies have shown that several proteins associated with neuroinflammation, intracellular signaling, lipid metabolism, and oxidative stress are differentially expressed in patients with TLE. Moreover, TLE has different non-coding RNA profiles and GABA receptor levels (Brooks-Kayal et al., 1998; Glazyrin et al., 2024; Huang et al., 2015; Manna et al., 2022; Martinez & Peplow, 2023; Neumann & Britsch, 2024).These complex molecular changes contribute to seizure generation, hippocampal sclerosis, and the chronic nature of TLE, and determining these protein alterations may serve as both biomarkers and potential therapeutic targets.

In light of this information, the present study aimed to investigate the genes affected in TLE through silico analysis and to identify hub genes that could potentially be targeted for new approaches.

MATERIALS AND METHODS

Data Set Selection

The study was conducted using GSE6947, a dataset of temporal lobe epilepsy gene expression data obtained from the NCBI Gene Expression Omnibus (GEO). The dataset consists of gene expression profiles obtained from the GPL6947 Illumina platform.

Weighted Gene Co-expression Network Analysis (WGCNA)

Gene co-expression network analysis (WGCNA) was used to evaluate gene expression profiles according to system biology. R software (R Foundation for Statistical Computing, Vienna, Austria) was used for WGCNA (R Core Team, 2024). Before the analysis, the raw data were filtered with low variance, and the analysis matrix included 129 samples and 5000 genes.

The soft-threshold value that is appropriate for the independent structure of the network from the scale determined for the scale-free topology analysis was observed. The soft-threshold power was elevated, and the average linkage was lowered (Figure 1). Therefore, the power value was set to seven, and the scale-free topology fit index (R^2) was above 0.85.

Pearson correlation coefficients were calculated, and the correlation matrix was converted into an adjacency matrix via soft threshold power. Subsequently, the topological overlap matrix (TOM) was calculated for the topological similarities among the genes. The genes were grouped using the TOM matrix and a hierarchical clustering method (Figure 2). A dynamic tree cut was applied to the co-expression modules, and resembling modules were determined using the module eigengene correlation. The modules transformed gene symbols via the Genotype-Phenotype Lattice 6947 platform.

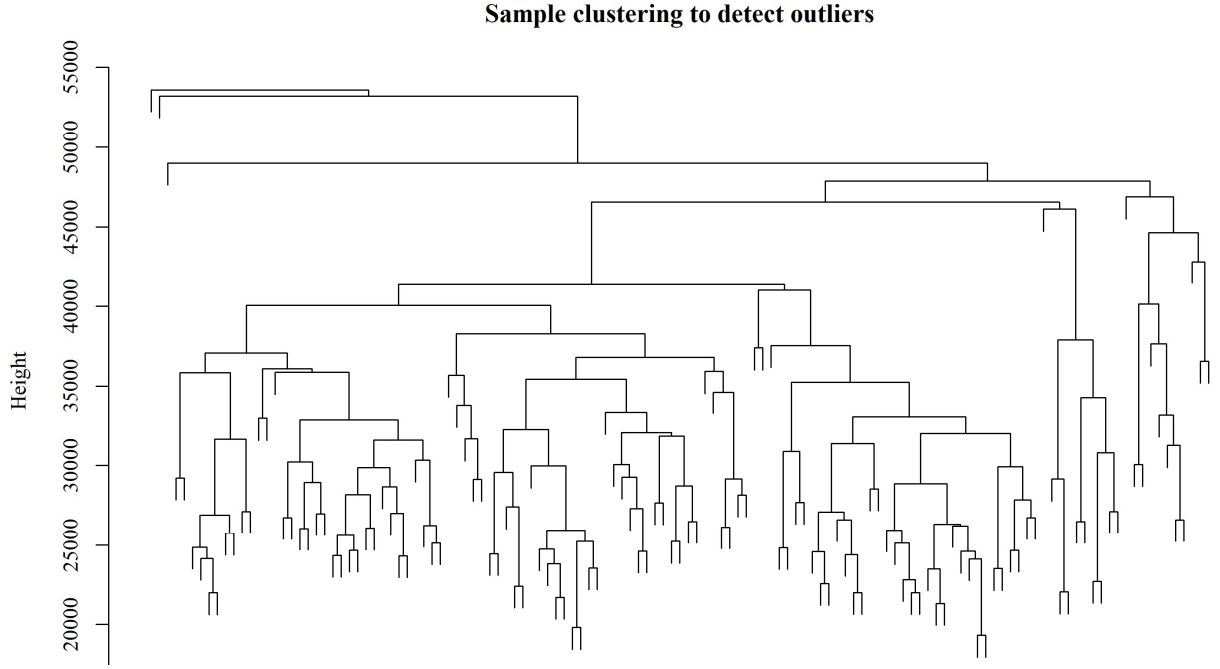

Fig. 1: Sample clustering dendrogram used to detect potential outlier samples.

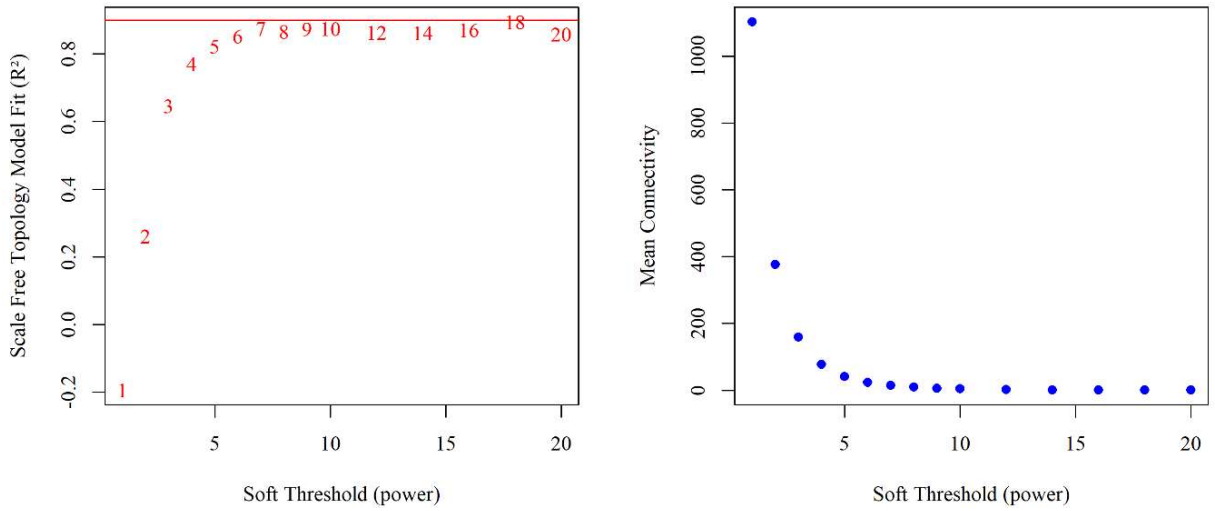

Fig. 2: Determination of soft-threshold power for WGCNA. (A) Scale-free topology fit index for various soft-thresholding powers. (B) Mean connectivity for different soft-thresholding powers. A soft-thresholding power of **7** was selected to construct the weighted gene co-expression network.

Functional Enrichment Analysis

Gene Ontology (GO) Biological Process enrichment was applied to each module for determination of biological gene function of the genes by cluster Profiler R package (Davison and Davenport, 2002). The gene symbols were transformed into the ENTREZ ID format, and GO enrichment analysis was performed with the following parameters:

- Ontology: Biological Process (BP)
- Multiple comparison correction: Benjamini-Hochberg (BH)
- Significance threshold: adjusted P<0.05

Protein-Protein Interactions (PPIs) and Hub Gene Analysis

Protein–protein interactions (PPIs) between module genes were analyzed using the STRING database (Search Tool for the Retrieval of Interacting Genes/Proteins; https://string-db.org) (Szklarczyk et al., 2023).

Protein-protein interactions (PPIs) between genes in modules were analyzed using the STRING database (Search Tool for the Retrieval of Interacting Genes/Proteins; https://string-db.org). The obtained PPIs network was downloaded TSV format for visualization and topographical analysis in Cytoscape software (v3.10.4) (Shannon et al., 2003).

Hub genes were identified using the Cytoscape cytoHubba plugin. The analysis was performed using the maximum clique centrality (MCC) algorithm, and the top 10 genes with the highest connectivity were designated as hub genes in each module.

Hub Gene Analysis Based on Module Membership

Module membership (MM) analysis was performed to identify the central genes within the modules obtained from the WGCNA results. MM values were obtained by calculating the Pearson correlation between the gene expression matrix and the module eigengenes. Genes within the pink module were selected, and the MM value was calculated for each gene, which were ranked according to their absolute MM values. Genes with the highest MM values were considered the most central genes within the module. Based on the absolute MM values, the top 20 genes were identified as hub gene candidates for the pink module. The resulting probe IDs were converted into gene symbols using a platform annotation file and saved for further analysis.

RESULTS

WGCNA Results

In order to determine the appropriate soft-threshold value in WGCNA analysis, the scale-free topology fit index and average connectivity were analyzed. It was observed that as the soft-threshold value increased, the scale-free topology fit index also increased. When the power value was 7, the scale-free topology fit index exceeded 0.85, indicating that the network approached a scale-free structure. At this power value, the average connectivity of the network remained at an acceptable level. Therefore, a soft-threshold value of 7 was chosen for constructing the gene co-expression network. Using this parameter, correlations between genes were calculated based on the constructed network structure, and TOM was obtained. Subsequently, a hierarchical clustering method was applied, and genes were grouped into co-expression modules.

The WGCNA analysis identified a total of 18 co-expression modules. These modules are represented by different colors (Figure 3). Among them, the five largest modules were determined and contained within these modules are given in Table 1. These modules represent gene networks associated with epilepsy.

Table 1: Identified modules and number of genes in each module.

Module	Gene Number
Pink	1647
Blue	481
Brown	475
Red	358
Green	370

Functional Enrichment Results

The five main modules named pink, blue, brown, red, and green were evaluated, and their results were visualized using dot-plot graphs. It was determined that the pink module was associated with synaptic transmission and neurotransmitter release (Figure 4A). The blue module was found to be related to protein synthesis and ribosomal biogenesis processes (Figure 4B). The brown module was found to be particularly associated with cellular structure organization and metabolic processes (Figure 4C). The red module was associated with RNA processing and post-transcriptional regulation (Figure 4D). The green module was shown to be related to glial cell biology and myelin formation (Figure 4E).

PPIs Results

Among all identified modules, the pink module was strongly correlated with epilepsy; therefore, this module was selected for further functional enrichment and protein-protein interaction (PPI) network analyses. DCTN1, UBE2E1, UBE2E2, KLC1, UBE2E3, NEDD4L, STAT3, GABARAPL1, CYCS, and DCTN3 had the highest MCC scores in the pink module. These were accepted as the top 10 hub genes (Figure 5).

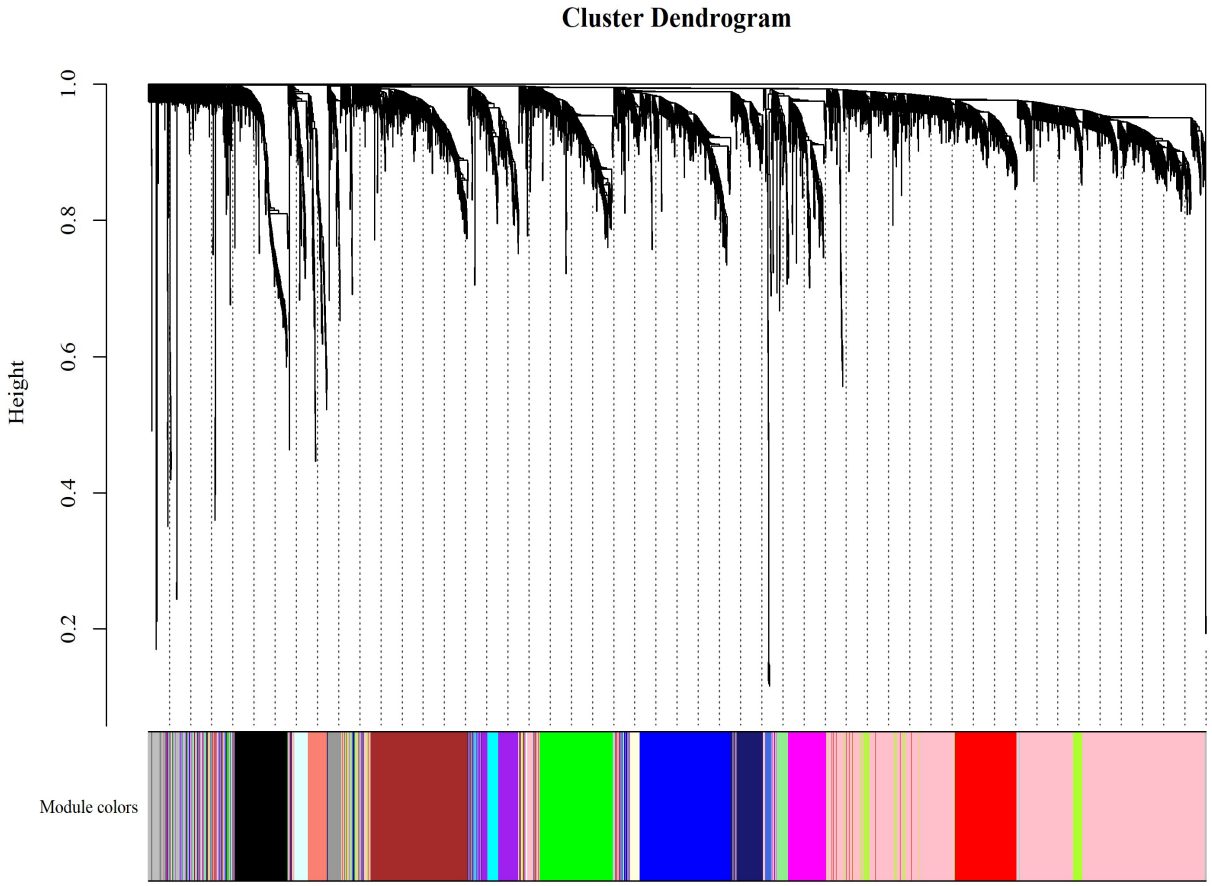

Fig. 3: Gene dendrogram and module colors.

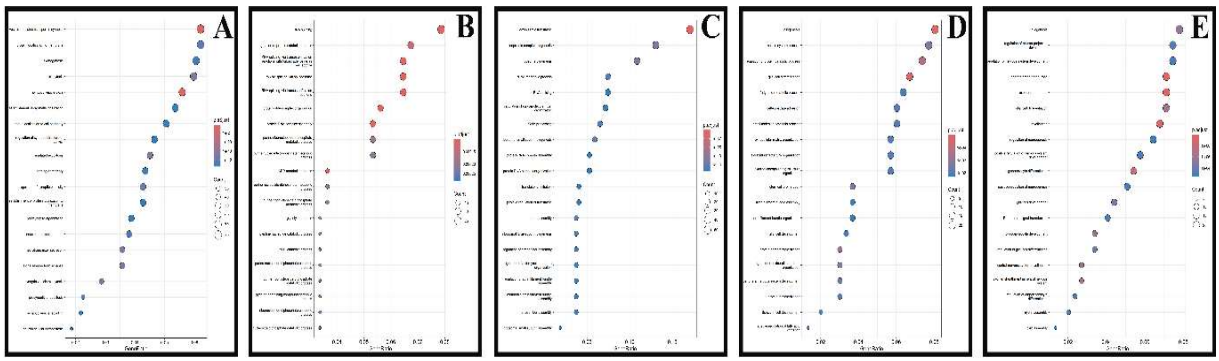

Fig. 4: Gene Ontology (GO) biological process enrichment analysis of genes in the identified WGCNA modules. Panels A–E correspond to the pink, blue, brown, red, and green modules, respectively. The x-axis represents the GeneRatio. Dot size indicates the number of genes enriched in each GO term, and color indicates the adjusted p-value. Only significantly enriched GO terms (adjusted P< 0.05) are shown.

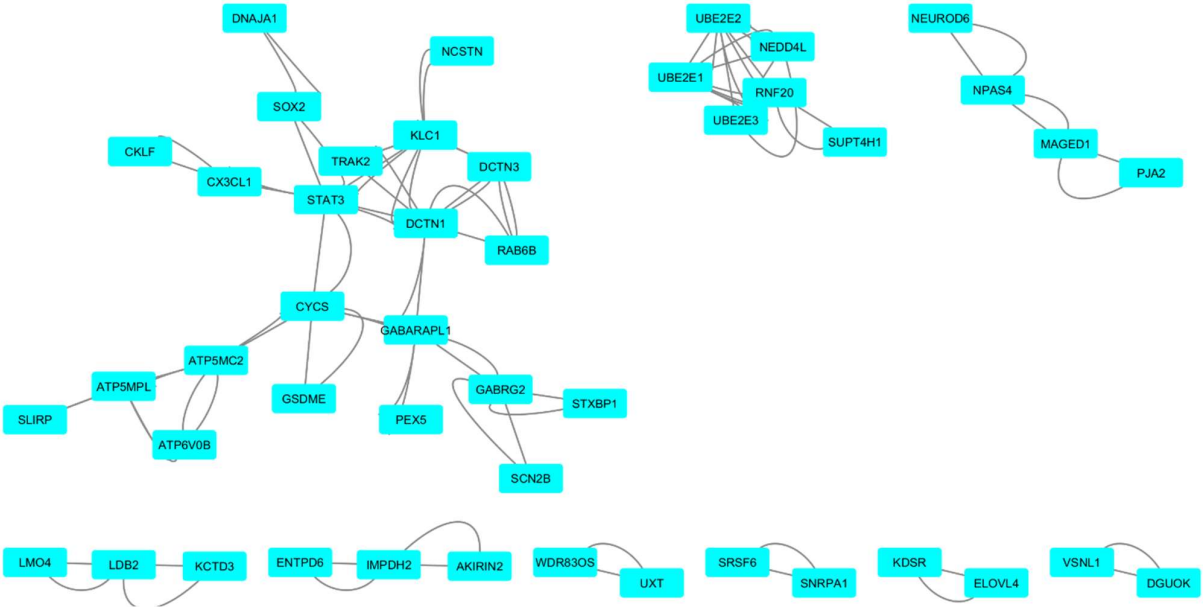

Fig. 5: Protein–protein interaction (PPI) network constructed for genes in the selected module using the STRING database and visualized in Cytoscape. Nodes represent proteins encoded by the genes and edges represent protein–protein interactions.

MM Results

The absolute MM values, that rank by the genes in the pink module exhibited that UBE2E2, RAB6B, PPP3CB, CYFIP2, CALM3, CHCHD6, NSF, KIF3C, MYT1L, PDXP, CAMKV, ATP6V1H, PNCK, CHN1, RUNDC3A, LY6H, CADPS, SCN3B, and CDK5. The genes and their details are given Figure 6.

DISCUSSION

TLE is the most common cause of drug-resistant epilepsy, and remains a challenging disorder to manage, especially in patients who do not respond adequately to antiepileptic drugs (Cao et al., 2023). Therefore, the development of new strategies based on the molecular alteration of brain tissue is crucial. This study focused on data belonging to patients with TLE, and it is relevant that synaptic functions, neurotransmitter release, RNA processing, protein synthesis, and glial cell development have played a pivotal role in TLE. Moreover, it was determined that the UBE2E2, RAB6B, PPP3CB, and CYFIP2 genes had the highest MM in the main module.

Neuroinflammation has been increasingly emphasized in recent years. Microglia and astrocytes that can be activated the JAK/STAT signal pathway are the main cell in neuroinflammation (Sun et al., 2023). In this study, it was indicated that STAT3, a central component of the JAK/STAT pathway, is one of the hub genes in TLE. Similarly, previous experimental studies show that inhibition of STAT3 is increased both epileptic activity and neuroinflammation (Grabenstatter et al., 2014). Therefore, STAT3 may be considered a potential target for the treatment of TLE.

UBE2E2 is a key component of the ubiquitin-proteasome system that plays a critical role in processes such as synaptic protein regulation, receptor turnover, and synaptic plasticity in neuronal cells. It may contribute to the formation of epileptic circuits by altering protein turnover mechanisms and being subject to dynamic remodeling of neuronal networks. Furthermore, recent studies have shown that the ubiquitin-proteasome system influences synaptic plasticity by regulating synaptic protein stability and the surface expression of neurotransmitter receptors (Jarome and Devulapalli, 2018; Jarome and Helmstetter, 2013). Our results show that UBE2E2 is a hub gene. Moreover, the MCC value of UBE2E2 has red with that of UBE2E1. It has also been reported that regulation of distributed ubiquitin proteosome system decreases seizure severity (Jing et al., 2025). Therefore, focused UBE2E2 and UBE2E1 could support TLE treatment.

Excessive neuronal activity caused by epileptic seizures can lead to oxidative stress and mitochondrial dysfunction. In the circumstances, cytochrome C released from the mitochondria into the cytosol. Following, it activates caspase cascade so neuronal apoptosis initiates (Kovac et al., 2017; Waldbaum and Patel, 2010). In the present study, it was shown that the CYCS gene, which encodes cytochrome c, is a hub gene in TLE. Thus, alteration of CYCS may protect neurons in TLE. Various research indicated that agents including mitochondrial antioxidants, coenzyme Q10, and N-acetylcysteine have been reported to exhibit neuroprotective effects in experimental epilepsy models, is similar with the results (Pearson-Smith and Patel, 2017; Yang et al., 2020).

The transport of synaptic vesicles and organelles thought the axon is critical for maintaining synaptic function in the neurons. Microtubule-based axonal transport mechanism impairs neurological diseases including epilepsy (Hirokawa et al., 2009; Liu et al., 2025). In epileptic states, microtubules are implicated in altered neuronal development, eventually leading to neuronal loss, whereas in normal neurons microtubules contribute to proper neurite growth and axonal division (Gambino et al., 2020). The study noteworthy show that hub genes such as DCTN1, DCTN3, and KLC1 are associated with

intracellular axonal transport processes. Due to this reason, the inclusion of genes associated with axonal transport within the modules related to epilepsy suggests that intracellular transport mechanisms may also play an important role in epileptic network reorganization.

Astrocytes that originated from neuroepithelia contribute to homeostasis in the Central nervous system, so they regulate neuronal excitability via reducing glutamate level TLE. The dysfunction of astrocytes results in perturbations within neuronal networks, which subsequently lead to seizures and neuronal cell loss (Vezzani et al., 2022). Therefore, the identification of genes associated with glial functions supports the importance of neuron-glia interactions in the pathophysiology of epilepsy. Our PPIs indicate that processes related to cell biology and myelin formation are linked to neuronal but also glial mechanisms in TLE.

This study had some limitations. First, the analyses were performed using only in silico gene expression data, and the results must be validated through experimental studies. Furthermore, the analysis was conducted on a single GEO dataset, and validation analyses with different datasets would increase the reliability of the findings. In addition, it should be considered that changes observed at the transcriptome level may not always directly reflect protein expression or functional activity. Future studies should validate the identified hub genes using animal models or patient tissues and investigate their functional roles in epileptogenesis.

Conclusion

In conclusion, this study used a systems biology approach to identify gene co-expression networks associated with temporal lobe epilepsy and revealed potential hub genes associated with synaptic functions, neuroinflammation, calcium signaling, and mitochondrial stress. These genes are considered potential biomarkers and therapeutic targets that could contribute to understanding the molecular mechanisms underlying epilepsy.

DECLARATIONS

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Conflict of Interest: The authors declare no conflict of interest with anyone.

Data Availability: All the data is included in this study and can be obtained from corresponding author.

Ethics Statement: All the research procedures were conducted according to guidelines regarding use of animal for research purpose.

Author's Contribution: MM and HAE: Idea and design; CA and BK: Data collection and writing draft; MK and TD: reviewing and editing of the manuscript.

Generative AI Statement: The authors confirm that no AI-contributed technologies were utilized when preparing this manuscript.

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