JEPIBIO
Journal of Epidemiology and Infection Biology

Research Article

Differential Gene Expression and Network Analysis Reveal Diagnostic Biomarkers in Tuberculosis Infection

Mahnoor Sajjad[1*], Pirlepesov Daniyar Maratovich[2], Onaiba Khan[3]

[1]Department of Convergence Medical Science, College of Medicine, Institute of Medical Science, Gyeongsang National University, Jinju, Republic of Korea
[2]Department of Smart Farm, Gyeonsang National University, Jinju, Republic of Korea
[3]Applied Life Sciences, Gyeongsang National University, Jinju, Republic of Korea

*Corresponding author:

Ms. Mahnoor Sajjad
(e-mail: mahnoorsajjad.lhr@gmail.com)

Date of Receiving: 21/06/2025
Date of Acceptance 15/08/2025
Date of Publishing 10/12/2025

ABSTRACT Tuberculosis (TB), caused by *Mycobacterium tuberculosis*, remains a major global health problem. Early diagnosis and effective treatment are still difficult due to complex host–pathogen interactions and increasing drug resistance. Identifying host gene expression biomarkers can help improve diagnosis and support the development of new therapeutic strategies. In this study, we performed an integrated bioinformatics analysis to identify key genes, pathways, and regulatory networks associated with TB infection. Gene expression microarray datasets GSE11199 (Affymetrix Human Genome U133 Plus 2.0 Array) and GSE34608 (Illumina HumanHT-12 V4.0 BeadChip) were obtained from the Gene Expression Omnibus (GEO) database. Differential gene expression analysis was performed using GEO2R based on the limma statistical method. Initial screening identified 1,608 candidate differentially expressed genes (DEGs) in GSE11199 and 2,214 candidate DEGs in GSE34608. After applying stringent statistical filtering criteria (adjusted p-value < 0.05 and quality control filtering), 115 significant DEGs from GSE11199 were retained for downstream analysis. GSE34608 was used for comparative biological interpretation but showed limited statistically significant genes under strict thresholds. Functional enrichment analysis was performed using Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) databases. Protein–protein interaction (PPI) networks were constructed using STRING and visualized using Cytoscape to identify hub genes. Transcription factor and microRNA regulatory networks were also analyzed. Drug–gene interaction analysis was performed using DGIdb, and molecular docking validation was conducted using AutoDock Vina. Key immune-related genes identified included CCL1, CXCL10, GBP5, and IFITM3. Hub genes in the PPI network included IRF7, ISG15, and STAT1. Interferon signaling, cytokine signaling, proteasome activity, and immune response pathways were found significant in enrichment analysis. Higher binding affinity of multiple drug molecules with target proteins were found. These findings undermine the understanding of host immune mechanisms during TB infection.

KEYWORDS Tuberculosis, Differentially Expressed Genes, Host Immune Response, Protein–Protein Interaction Network, Biomarker Discovery

Introduction

Tuberculosis (TB) is a chronic infectious disease which is majorly caused by *Mycobacterium tuberculosis* in humans. It mainly affects the lungs but can spread to other organs especially alimentary canal and bones (Salgado-Cantu *et al*, 2024). The incidence of TB is one of the leading infectious causes with millions of new cases reported every year. (Bisht *et al*, 2023). It primarily spreads through aerosols produced during cough or sneeze of a patient to the individuals in the surroundings (Kudryashova *et al*, 2021). Many infected individuals develop latent infection, which can later activate under stress conditions and in old age. TB control and eradication programs have improved over the past decades especially Public Awareness Campaigns. But still this challenge not only exists but also increasing. Drug-resistant TB is increasing in many regions. Multidrug-resistant and extensively drug-resistant TB complicate treatment (Baum *et al*, 2024). These resistant strains require longer and more toxic therapies. Delayed diagnosis also increases transmission risk (Raad *et al*, 2024). Social and economic factors further contribute to disease spread. Global migration and urban crowding increase exposure risk. Future TB control requires better diagnostics and new therapeutic targets. Understanding host immune response is essential for controlling disease progression.

To cite this article: Sajjad, M., P. D. Maratovich, and O. Khan. (2025). *Differential Gene Expression and Network Analysis Reveal Diagnostic Biomarkers in Tuberculosis Infection*. Journal of Epidemiology and Infection Biology 2(2):9-16.

Differentially expressed genes (DEGs) represent genes that show changes in expression between disease and normal states (Huang *et al*, 2024). DEG analysis helps identify molecular mechanisms of disease (Hassan *et al*, 2024). High-throughput technologies such as microarrays and RNA sequencing generate large gene expression datasets (Jurisica, 2024). These datasets require computational tools for proper interpretation. Bioinformatics provides methods for analyzing and visualizing complex biological data. It helps to identify important genes, pathways, and regulatory networks. DEG analysis can reveal host immune responses during TB infection (Pu *et al*, 2021). Pathway enrichment analysis helps identify biological processes involved in disease. Network analysis helps identify hub genes that regulate immune responses (Liu *et al*, 2024; Silver *et al*, 2024). Protein–protein interaction analysis helps understand gene connectivity. Integration of transcription factor and microRNA networks improves regulatory understanding (Kumar and Roy, 2024). Drug–gene interaction analysis helps identify therapeutic targets. Molecular docking helps predict drug binding to target proteins. Bioinformatics therefore supports biomarker discovery and drug development. These approaches are increasingly important in infectious disease research.

The current study aimed to study the associated key host genes and biological pathways involved in disease progression. Public gene expression datasets were analyzed to achieve these goals. Differential expression analysis and functional enrichment analysis were used to understand biological functions. Previously, there were not so many studies available that provide a systems-level understanding of TB host response. In current study, identifying biomarkers may help improve early diagnosis and host-directed therapies.

Materials and Methods

Dataset Collection and Preprocessing

Gene expression datasets were collected from the NCBI Gene Expression Omnibus (GEO) database. Two datasets related to tuberculosis infection were selected. The selected datasets were GSE11199 and GSE34608. GSE11199 was generated using the Affymetrix Human Genome U133 Plus 2.0 Array platform. This dataset contains macrophage expression profiles after Mycobacterium tuberculosis infection. It includes five infected samples and five control samples. GSE34608 was generated using the Illumina HumanHT-12 V4.0 BeadChip platform. This dataset contains whole blood expression data from TB patients and healthy controls. Six TB samples and six healthy samples were included in the analysis. Raw expression data were downloaded from GEO. Quality assessment was performed before analysis. Data normalization was applied using GEO2R preprocessing methods. Multiple testing correction was applied during DEG analysis. Only high-quality samples were included in downstream analysis. Platform annotation files were used for gene mapping.

Differential Gene Expression Analysis

Differential gene expression analysis was performed using the GEO2R tool. GEO2R is based on the limma statistical analysis method. This method is widely used for microarray DEG analysis. TB samples were compared with healthy control samples. Adjusted p-value was used to control false discovery rate. Genes with adjusted p-value less than 0.05 were considered significant. Initial screening identified candidate DEGs from both datasets. GSE11199 showed 1,608 candidate DEGs in the initial screening. GSE34608 showed 2,214 candidate DEGs in the initial screening. Additional filtering was applied to improve reliability. After filtering, 115 significant DEGs from GSE11199 were retained. These DEGs were used for downstream functional analysis. GSE34608 was used for comparative biological interpretation. Volcano plots were generated to visualize DEG distribution. Venn diagrams were generated to identify overlapping genes.

Protein–Protein Interaction Network Construction

Protein–protein interaction (PPI) analysis was performed using the STRING database. STRING version 12.0 was used for interaction prediction. Homo sapiens were selected as the reference organism. Interaction confidence score was set to 0.9. Only high-confidence interactions were included in analysis. Interaction networks were exported for visualization. Cytoscape software was used for network visualization. Network topology analysis was performed using Cytoscape plugins. MCODE plugin was used to identify network clusters. ClueGO plugin was used for functional pathway visualization. Hub genes were identified based on node connectivity. Highly connected nodes were considered important regulatory genes. Network graphs were generated for interpretation. PPI analysis helped identify functional gene interactions. This step helped identify key immune regulatory genes.

Functional Enrichment Analysis

Functional enrichment analysis was performed using gProfiler. Gene Ontology analysis was performed for three categories. These categories included Biological Process, Molecular Function, and Cellular Component. KEGG pathway enrichment analysis was also performed. Enrichr platform was used for additional pathway confirmation. Multiple testing corrections were applied during enrichment analysis. Pathways with adjusted p-value less than 0.05 were considered significant. Enrichment analysis identified immune-related biological processes. Cytokine signaling pathways were highly enriched. Interferon signaling pathways were also enriched. Proteasome and immune activation pathways were identified. Enrichment plots were generated for visualization. Functional enrichment helped explain biological significance of DEGs. This analysis helped link genes to immune response mechanisms. Results were used for downstream network interpretation.

Regulatory Network and Drug Interaction Analysis

Transcription factor and microRNA interactions were analyzed using miRNet and TRRUST databases. These databases provide curated regulatory interaction data. miRNA-target interactions were predicted using integrated database evidence. Transcription factor regulatory relationships were extracted from TRRUST. Regulatory networks were visualized using Cytoscape. Drug–gene

interaction analysis was performed using DGIdb version 4.2.0. FDA-approved drugs and experimental compounds were screened. Candidate genes were queried against DGIdb database. Interaction score was used to rank drug candidates. Drug-target interactions were filtered based on relevance. Known anti-TB drugs were included for comparison. Potential host-directed therapy targets were identified. Drug interaction networks were generated. These results supported therapeutic target prediction. This step helped connect genes with treatment strategies.

Molecular Docking Analysis

Molecular docking analysis was performed using AutoDock Vina version 1.2.0. Target protein structures were obtained from the Protein Data Bank. Protein structures included STAT1 and ISG15. Ligand molecules were obtained from ZINC compound database. Protein structures were prepared using AutoDock Tools. Water molecules were removed before docking analysis. Hydrogen atoms were added during preparation. Grid boxes were designed around active binding sites. Docking simulations were performed to estimate binding affinity. Binding energy values were recorded for each ligand. Lower binding energy indicated stronger interaction. Docking results were visualized using PyMOL software. Docking analysis helped validate drug binding predictions. This step supported drug candidate prioritization. Docking results were compared with known TB drug targets.

Results

Differential Gene Expression Analysis

Differential gene expression analysis was performed for both datasets. Initial screening identified large numbers of candidate DEGs. In dataset GSE11199, 1,608 candidate DEGs were initially detected. In dataset GSE34608, 2,214 candidate DEGs were initially detected. These genes represented broad transcriptional changes during TB infection. Further filtering improved statistical confidence. Adjusted p-value threshold was set to less than 0.05. Quality filtering was also applied. After filtering, 115 significant DEGs were retained from GSE11199. These genes were used for downstream functional analysis. UMAP analysis showed separation between infected and control samples (Fig. 1c). Expression density and variance plots confirmed data quality (Fig. 1a–b). Volcano plots confirmed DEG distribution patterns (Fig. 1e-g). Venn diagram analysis showed DEG overlap between datasets (Fig. 1h). These results confirm strong transcriptional changes during TB infection.

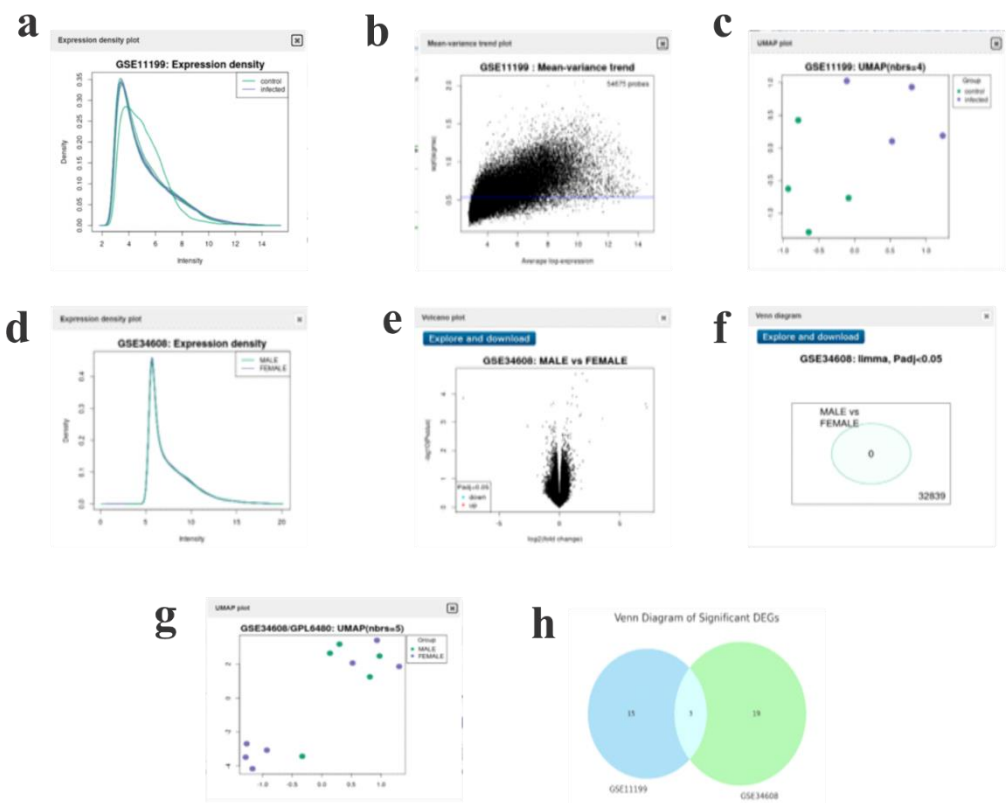

Fig. 1. Gene expression quality and clustering analysis. (a) Expression distribution of control and infected samples. (b) Mean variance plot showing expression variability. (c) UMAP plot showing separation between TB infected and control samples. (d) Volcano plot showing upregulated and downregulated genes. (e) Expression density plot showing normalization distribution. (f) Venn diagram showing DEG overlap. (g) UMAP validation plot. (h) Venn diagram showing shared and unique DEGs between GSE11199 and GSE34608 datasets.

Protein–Protein Interaction Network Analysis

Protein interaction networks were constructed using significant DEGs. STRING database was used to predict protein interactions. High-confidence interaction score threshold was applied. Interaction data were imported into Cytoscape software. The PPI network showed strong connectivity among immune genes (Fig. 2). Several gene clusters were identified in network analysis. Hub genes were identified based on node connectivity. Key hub genes included IRF7, ISG15, and STAT1. These genes are known immune regulatory genes. These genes are involved in interferon signaling pathways. Network structure suggested strong immune activation. Chemokine signaling genes were also connected. Network visualization showed central immune signaling clusters. These findings suggest coordinated immune gene regulation during TB infection. These hub genes may act as potential biomarkers.

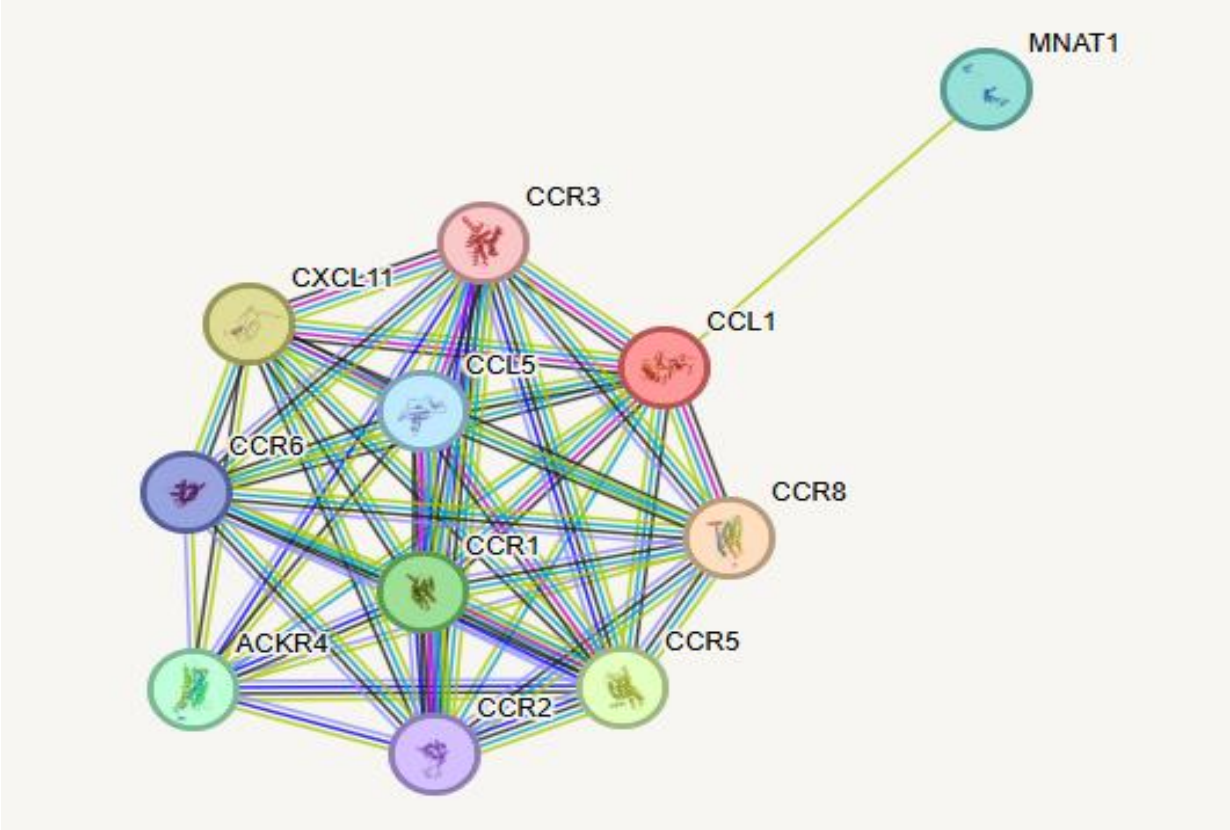

Fig. 2. Protein–protein interaction network generated using STRING and visualized in Cytoscape. Nodes represent proteins. Edges represent predicted interactions. Hub genes show high connectivity within immune signaling clusters.

Functional Enrichment and Pathway Analysis

Functional enrichment analysis was performed using GO and KEGG databases. Biological process analysis showed strong immune response enrichment. Cytokine-mediated signaling pathways were significantly enriched. Interferon signaling pathways were highly enriched. Proteasome activity pathways were also enriched. Immune activation pathways were strongly detected. Molecular function analysis showed cytokine receptor binding enrichment. Cellular component analysis showed immune complex localization. KEGG pathway analysis confirmed immune signaling pathway enrichment (Fig. 3). Viral interaction with cytokine pathways was also enriched. JAK-STAT signaling pathway was identified. Pathway enrichment supported immune activation results. Enrichment results confirmed DEG biological relevance. These pathways are known to play key roles in TB infection. These results support host immune activation during disease progression.

Drug–Gene Interaction Analysis

Drug–gene interaction analysis was performed using DGIdb database. Several drug-target interactions were identified. Known anti-TB drugs showed host interaction links. Rifampin showed the highest number of interaction links. Isoniazid and pyrazinamide also showed multiple interactions. Interaction scores helped rank drug candidates (Fig. 4). Most interactions were classified as inhibitors. Very few agonist interactions were detected. No antibody interaction was observed. These results suggest inhibition-based therapeutic mechanisms (Table 1). Several host immune genes showed drug interaction potential. These results suggest possible host-directed therapy targets. These findings connect immune genes with treatment strategies. These results support therapeutic target discovery.

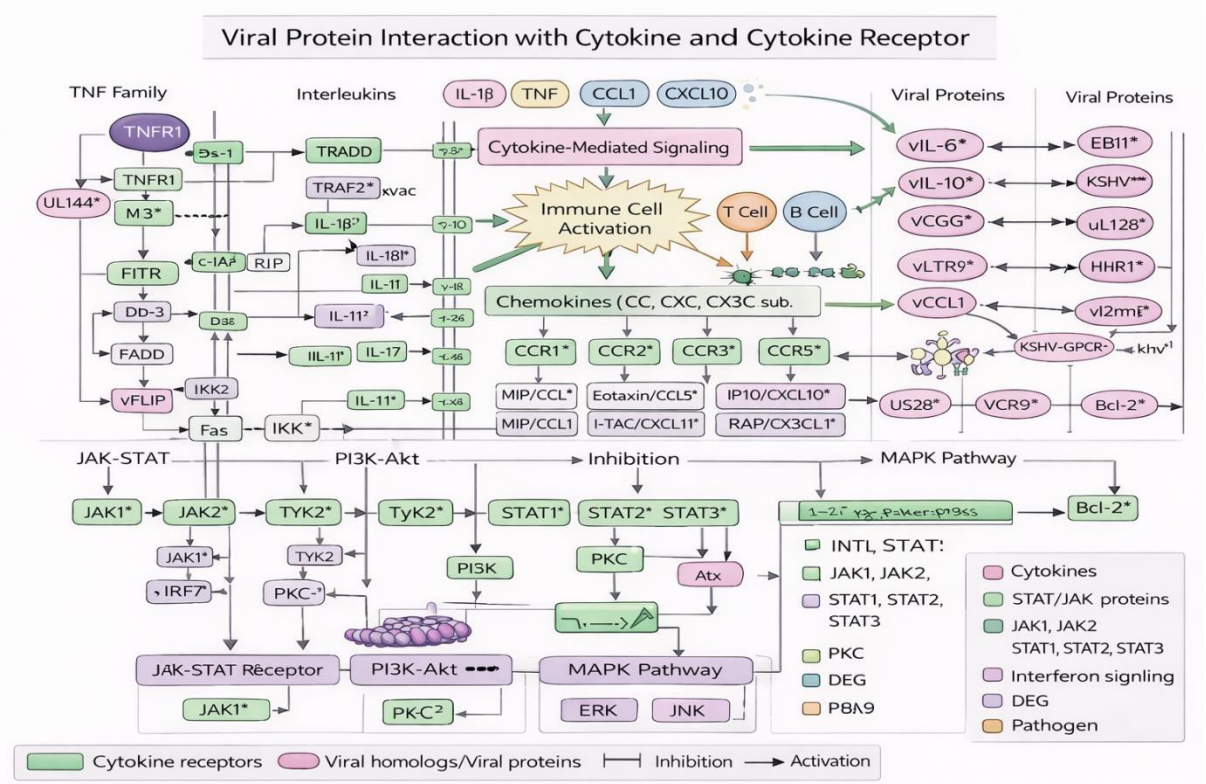

Fig. 3. Hypothetical KEGG pathway enrichment showing cytokine signaling and viral interaction pathways. Colored nodes represent enriched pathway genes. Arrows represent signaling interactions.

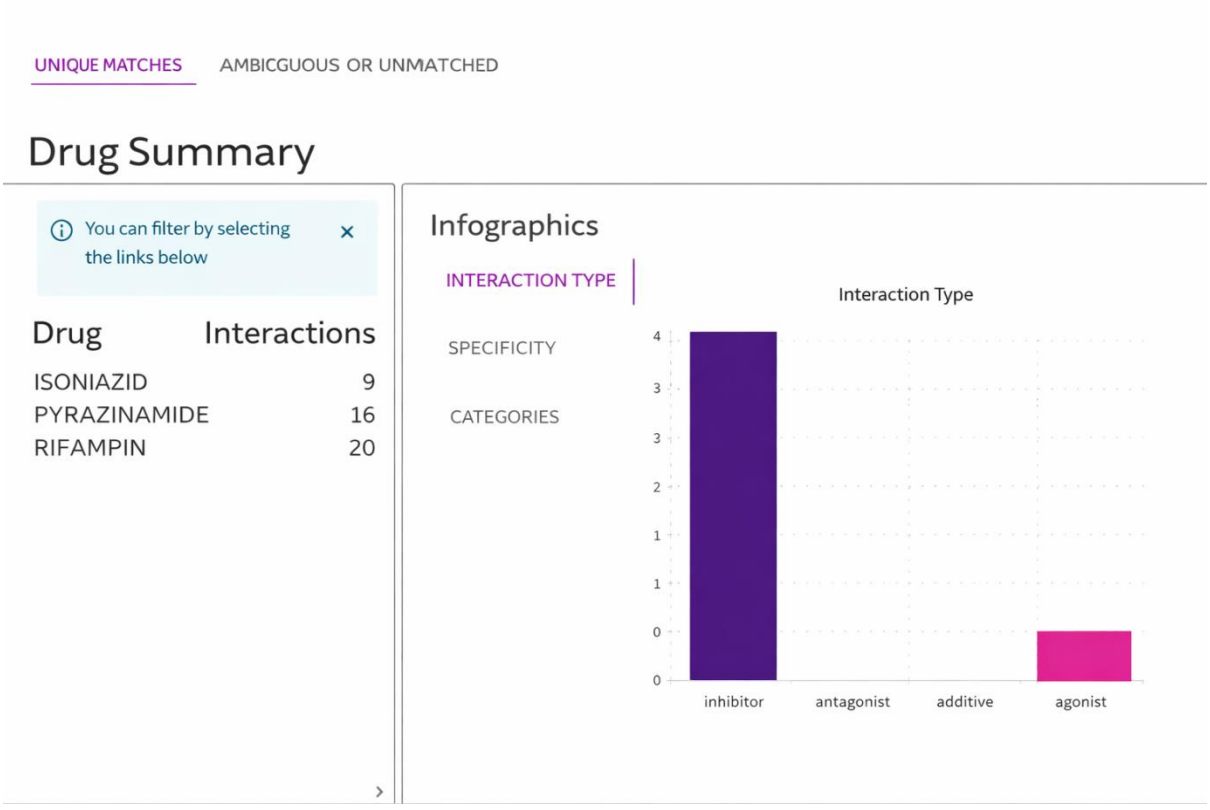

Fig. 4. Drug–gene interaction summary. Bar graph shows number of interactions per drug. Interaction types include inhibitor, agonist, antibody, and antagonist.

Table 1. Drug–gene interaction results showing drug name, target gene, approval status, and interaction score.

Drug	Gene	Approval	Interaction score
Isoniazid	ASTN2	Approved	5.80
Rifampin	RIPOR2	Approved	5.59
Isoniazid	DUX1	Approved	3.87
Rifampin	MAFK	Approved	3.73
Rifampin	CUX2	Approved	3.73
Isoniazid	INHA	Approved	1.93
Rifampin	AGBL4	Approved	1.86
Rifampin	GRIK1-AS2	Approved	1.24
Rifampin	DUX1	Approved	1.24
Rifampin	XPO1	Approved	0.62

Molecular Docking Analysis

Molecular docking analysis was performed to validate drug binding potential. Target proteins included STAT1 and ISG15. Candidate ligands were obtained from compound databases. Docking simulations were performed using AutoDock Vina. Binding affinity values were calculated for each compound. Several compounds showed strong binding affinity. The strongest binding affinity reached −9.5 kcal/mol. Multiple compounds showed binding below −7 kcal/mol. These values indicate stable protein–ligand interactions. Docking poses showed stable binding site interactions. Hydrogen bonding interactions were observed in docking models. Docking visualization confirmed binding site compatibility. Docking results supported drug-target predictions (Table 2). These findings support potential therapeutic candidate identification.

Table 2. Molecular docking results showing compound ID and binding affinity values against target proteins.

Sr #	Drugs	Binding affinity
1	ZINC000000002005	-8.3
2	ZINC000003784397	-4.9
3	ZINC000003818808	-5.9
4	ZINC000004974291	-9.5
5	ZINC000022443609	-6.9
6	ZINC000169295247	-7.1

Discussion

This study investigated the presence of psoriasis type and Tuberculosis remains a major global infectious disease challenge. Understanding host molecular responses is essential for disease control. This study used bioinformatics approaches to analyze host gene expression changes. Differential gene expression analysis showed clear transcriptional differences between TB and control samples. UMAP analysis confirmed strong separation between infected and control groups (Fig. 1). Expression density and variance plots confirmed dataset quality. Volcano plots showed clear DEG distribution patterns. Initial DEG screening showed large transcriptional gene pools. After statistical filtering, 115 significant DEGs were retained from GSE11199. These DEGs represent high-confidence infection-associated genes. These findings support strong host immune activation during TB infection. Previous studies have also reported immune-related DEG signatures in TB. Interferon-regulated genes are commonly reported in TB transcriptomics studies (Yang *et al*, 2024b; Vijayaraghavan *et al*, 2025). Our results support these previously reported immune activation patterns. These findings confirm the reliability of DEG-based TB transcriptomic analysis.

Protein–protein interaction analysis showed strong immune network connectivity. The PPI network identified key hub genes including IRF7, ISG15, and STAT1. These genes are known regulators of antiviral and immune signaling. STAT1 plays a central role in interferon signaling pathways. IRF7 regulates interferon production during infection. ISG15 is involved in immune protein modification and signaling. The PPI network showed strong clustering of immune genes. Chemokine signaling genes also showed network connectivity. These results suggest coordinated immune gene regulation. Similar hub genes have been reported in TB host response studies. Network connectivity supports functional immune pathway activation (Yang *et al*, 2024a; Zong *et al*, 2024). Hub genes may serve as potential biomarkers and therapeutic targets. Network biology helps explain complex host-pathogen interactions. These findings strengthen the role of immune signaling in TB pathogenesis.

Functional enrichment analysis confirmed immune pathway activation. GO analysis showed enrichment in immune response biological processes. Cytokine-mediated signaling pathways were strongly enriched. Interferon signaling pathways showed strong enrichment. KEGG analysis confirmed immune signaling pathway involvement. JAK-STAT signaling pathway enrichment was detected. Proteasome activity pathways were also enriched. Viral interaction with cytokine signaling pathways were detected. These pathways are known to regulate immune defense responses (Jin *et al*, 2024; Bouzeineddine *et al*, 2025). Similar pathway enrichment patterns have been reported in TB transcriptomic studies (Chen *et al*, 2022; Sampath *et al*, 2023; Shen *et al*, 2023). These findings support immune dysregulation during TB infection. Cytokine signaling pathways control immune cell recruitment. Proteasome activity regulates immune protein turnover. These pathways are essential for infection control. These results highlight key immune regulatory mechanisms in TB.

Drug–gene interaction analysis identified potential therapeutic targets. DGIdb analysis showed multiple drug interaction signals (Fig. 4, Table 1). Rifampin showed the highest interaction connectivity. Isoniazid and pyrazinamide also showed multiple interactions. Most drug interactions were classified as inhibitors. This supports inhibition-based treatment mechanisms (Alqhtani *et al*, 2024). Host immune gene targeting may support host-directed therapy development. Host-directed therapy is an emerging TB treatment strategy. Drug–gene interaction

networks help to identify therapeutic opportunities. These results connect molecular targets with treatment strategies (Fu *et al*, 2023). Some interactions were indirect host pathway effects. These findings support potential host immune modulation approaches (Lu *et al*, 2024). Drug interaction results require experimental validation. These results provide preliminary therapeutic insights. These findings support systems biology-based drug discovery.

Molecular docking analysis supported drug-target interaction predictions. Docking analysis showed strong binding affinity for several candidate compounds (Table 2). The strongest binding affinity reached −9.5 kcal/mol. Several compounds showed binding below −7 kcal/mol. These values indicate stable ligand-protein binding. Docking visualization showed stable binding conformations. Hydrogen bonding interactions supported binding stability. Docking analysis supported predicted drug-target relationships. Docking results provide preliminary structural validation. Molecular docking is useful for early drug screening. Docking results should be validated experimentally (Xu *et al*, 2022; Darmadi *et al*, 2023). Protein flexibility and cellular environment may affect binding. These findings provide starting points for drug development. Docking results support potential host immune targeting strategies (Huan-Jun *et al*, 2024). These results strengthen drug discovery potential from transcriptomic data.

This study has several limitations. Sample size was relatively small. Microarray platform limitations may affect gene detection sensitivity. Cross-platform dataset differences may affect results. Only computational analysis was performed. Experimental validation was not performed. Molecular docking provides predictive results only. Clinical validation is required before therapeutic application. Future studies should include RNA sequencing datasets. Larger patient cohorts should be analyzed. Single-cell transcriptomics may improve resolution. Functional wet lab validation is required. Longitudinal TB studies may improve biomarker reliability. Multi-omics integration may improve pathway understanding. Despite limitations, this study provides valuable molecular insights into TB host response.

Declaration of Competing Interest

The authors declare that they have no competing or conflict of interests.

Author Contributions

UH: Conceptualization, Methodology, formal analysis, Writing—original draft preparation. **MS:** Conceptualization, Methodology. **PDM:** Formal analysis, Writing—review and editing. **OK:** Writing—review and editing. All authors have read and agreed to the published version of the manuscript.

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