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Cross-tissue transcriptome-wide association study reveals novel psoriasis susceptibility genes

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ABSTRACT

Background: Psoriasis is a chronic, immune-mediated inflammatory skin disorder with a strong genetic component. Although numerous GWAS have identified risk loci, many associated variants lie in non-coding regions, complicating functional interpretation.
Objective: This study aimed to identify novel psoriasis susceptibility genes by integrating large-scale GWAS and eQTL data using a cross-tissue TWAS approach.
Methods: We integrated psoriasis GWAS summary statistics from the FinnGen database with GTEx V8 eQTL data. A cross-tissue TWAS was performed using UTMOST, followed by validation with single-tissue TWAS via FUSION. Conditional and joint analyses were conducted to delineate independent genetic signals, and gene-based analysis was performed using MAGMA. Causal relationships were evaluated using Mendelian randomization (MR) and Bayesian colocalization analyses. Key SNPs were functionally characterized using CADD, GERP++, and RegulomeDB for pathogenicity prediction and regulatory potential assessment. Finally, functional network analysis was conducted using GeneMANIA.
Results: The cross-tissue TWAS identified 259 genes significantly associated with psoriasis ($p < 0.05$), with 12 remaining significant after FDR correction. Single-tissue TWAS validation revealed 655 significant genes, with an overlap of three protein-coding candidates: POLI, NFKB1, and ZFYVE28. Cross-validation with MAGMA refined the candidate set to NFKB1 and ZFYVE28. MR and colocalization analyses supported a causal relationship for NFKB1 in Skeletal Muscle, Transverse Colon, and Cultured Fibroblasts, and for ZFYVE28 in Subcutaneous Adipose Tissue and Esophageal Mucosa tissues. Functional annotation identified key SNPs including rs4235405, rs3774960, and rs1598856 for NFKB1, and rs1203786 for ZFYVE28, with varying degrees of pathogenicity and regulatory potential. GeneMANIA network analysis further implicated NFKB1 in NF-κB signaling and ZFYVE28 in vesicle-mediated transport.
Conclusion: Our integrative multi-omics approach identifies NFKB1 and ZFYVE28 as novel psoriasis susceptibility genes, providing potential biomarkers and therapeutic targets for this complex disease.

1. Introduction

Psoriasis is a chronic, immune-mediated inflammatory skin disorder that imposes a significant burden on global health. According to the 2019 Global Burden of Disease study, approximately 1–3 % of the world's population is affected by psoriasis [1,2], with an annual incidence ranging from 78.9 to 230 cases per 100,000 individuals [3]. Several large-scale studies have demonstrated a bimodal age distribution of disease onset, with the first peak occurring between 15 and 20 years of age and a second peak between 55 and 60 years [4–7]. The persistent and recurrent nature of psoriasis, along with associated pruritus and pain, severely impacts both the physical and mental well-being of patients and diminishes their quality of life [8].

Twin and familial studies have established a strong genetic component in psoriasis susceptibility. Large-scale twin studies estimate the heritability of psoriasis to be between 60 % and 75 %, with concordance rates of 35–72 % in monozygotic twins and 12–30 % in dizygotic twins [9–11]. Although some rare forms of psoriasis follow a Mendelian inheritance pattern, the majority of cases exhibit complex polygenic inheritance, wherein multiple genetic variants with modest individual

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effects collectively contribute to disease development [12].

Recent genome-wide association studies (GWAS) have identified over 63 risk loci associated with psoriasis susceptibility [13]. However, many of the disease-associated variants reside in non-coding regions, complicating the assessment of their functional significance [14]. Moreover, complex linkage disequilibrium (LD) patterns can obscure the identification of the causal variants driving these associations [15]. These limitations highlight the need for integrative approaches that bridge the gap between genetic associations and the underlying biological mechanisms.

Transcriptome-wide association studies (TWAS), which integrate expression quantitative trait loci (eQTL) data with GWAS summary statistics, have emerged as a powerful strategy to address these challenges [16]. Cross-tissue TWAS methods—particularly the Unified Test for MOlecular SignaTures (UTMOST)—enhance single-tissue approaches by leveraging shared eQTL effects across tissues while preserving tissue-specific signals [16]. This approach has successfully identified novel susceptibility genes for various complex diseases, including rheumatoid arthritis [17] and inflammatory bowel disease [18].

In the present study, we aimed to systematically evaluate the relationship between genetically predicted gene expression and psoriasis risk by integrating large-scale GWAS data with GTEx V8 eQTL information. Through comprehensive cross-tissue TWAS analysis, followed by validation using single-tissue TWAS and MAGMA, we sought to identify novel psoriasis susceptibility genes and provide new insights into the underlying pathogenic mechanisms.

2. Materials and methods

2.1. Study design

We employed a multi-step strategy to identify psoriasis susceptibility genes. First, we integrated psoriasis GWAS data from the Finnish database with eQTL data from GTEx V8 and performed a cross-tissue TWAS analysis using UTMOST. Candidate genes identified at the discovery stage were then validated through single-tissue TWAS analysis with FUSION and gene-based analysis using MAGMA. Independent signals were delineated by conditional and joint analysis, and causal relationships were evaluated via Mendelian randomization (MR) and colocalization analyses. Finally, we performed functional network analysis using GeneMANIA. The overall study design is illustrated in Fig. 1.

2.2. Psoriasis GWAS data Source

The FinnGen project combines genotype data from Finnish biobanks with health outcome data from national health registers, using the International Classification of Diseases, 10th Revision (ICD-10) for

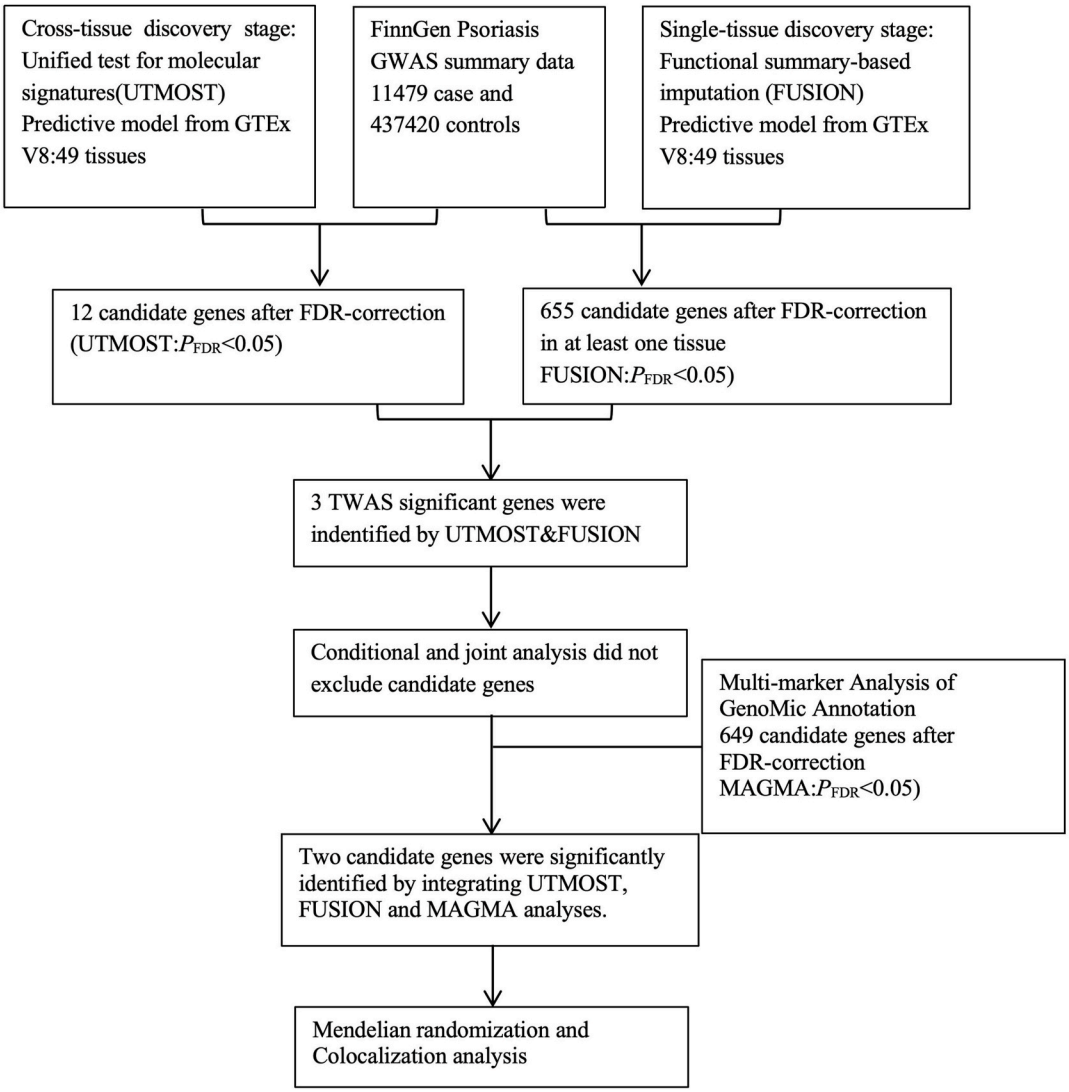

Fig. 1. Workflow of the study design.

diagnostic criteria. This study utilized psoriasis GWAS summary statistics from the FinnGen database. The dataset comprised 11,479 psoriasis cases and 437,420 controls, covering 19,345,588 variants.

2.3. eQTL data Source

We used expression quantitative trait loci (eQTL) data from the GTEx V8 dataset [19]. This dataset includes gene expression profiles across 49 distinct tissues from 838 post-mortem donors, with sample sizes ranging from 73 in kidney cortex to 706 in skeletal muscle. Quality control criteria for the data were as follows: RNA Integrity Number (RIN) > 6.0, a minimum sequencing read length of 50 bp, and a sequencing depth exceeding 20 million alignable reads. At the gene expression level, transcripts were required to have a transcript per million (TPM) > 0.1 and to be present in at least 10 samples for a given tissue.

2.4. Cross-tissue TWAS analysis

We conducted a cross-tissue analysis using the UTMOST tool, which quantifies gene–trait associations at the tissue level to assess the genetic risk of psoriasis. This method balances tissue-specific and shared effects, thereby improving both the efficiency and accuracy of gene discovery [17,20]. In this analysis, we employed the generalized Berk–Jones (GBJ) test to integrate statistical signals across tissues [20,21] and applied false discovery rate (FDR) correction for multiple testing, setting the significance threshold at FDR <0.05.

2.5. Single-tissue TWAS analysis

We integrated psoriasis GWAS data with eQTL data from 49 GTEx V8 tissues using the FUSION tool [22]. The analysis workflow was as follows: First, linkage disequilibrium (LD) between the prediction models and GWAS variants was estimated based on European samples from the 1000 Genomes Project. Next, we evaluated the impact of SNPs on gene expression using multiple prediction models (BLUP, BSLMM, LASSO, Elastic Net, and top 1) [23]. The model with the best predictive performance was selected to determine gene weights, which were then combined with the GWAS Z-scores for psoriasis in the TWAS analysis. Genes were considered significant if they met the following criteria [1]: an FDR <0.05 in the cross-tissue TWAS analysis, and [2] an FDR <0.05 in at least one single-tissue TWAS analysis.

2.6. Conditional and joint analysis

To identify independent genetic signals, we performed conditional and joint (COJO) analysis within FUSION [22]. By accounting for linkage disequilibrium among markers, this analysis provides a more comprehensive understanding of the genetic architecture [24]. Genes that remained significant after conditioning were defined as jointly significant, whereas those that lost significance were regarded as marginally significant.

2.7. Gene analysis

We conducted gene-based analysis using MAGMA software (v1.08), which aggregates SNP-level association statistics into gene scores to quantify the association between each gene and the psoriasis phenotype [25,26]. Detailed parameter settings and methodological specifics are available in the original MAGMA documentation [27].

2.8. MR analysis and Bayesian colocalization analysis

We performed MR analysis using the TwoSampleMR package [28], treating cis-eQTL SNPs as instrumental variables, gene expression as the exposure, and the psoriasis GWAS as the outcome. Genome-wide significant SNPs ($P < 5 \times 10^{-8}$) were selected and subjected to LD pruning ($r^2 < 0.001$). Given that only a single independent instrumental variable was available, the MR effect was estimated using the Wald ratio method, with significance set at $P < 0.05$.

Subsequently, we conducted Bayesian colocalization analysis using the coloc package [29,30] to assess whether GWAS and eQTL signals share a common causal variant. The analysis focused on the posterior probabilities (PP) for five hypotheses, with PP.H4 > 0.75 considered indicative of a shared causal variant between the GWAS and eQTL signals [29].

2.9. SNP functional annotation and pathogenicity prediction

For the key SNPs identified through TWAS, MR, and colocalization analyses, we performed comprehensive functional annotation and pathogenicity prediction. We obtained the precise genomic coordinates and gene structure information for candidate genes from the NCBI Gene database and Ensembl (GRCh38/hg38). The Combined Annotation Dependent Depletion (CADD) tool was used to evaluate the pathogenicity potential of variants, with higher scores indicating greater deleteriousness. We assessed evolutionary conservation using Genomic Evolutionary Rate Profiling (GERP++), where higher scores represent more conserved sites. Regulatory potential was evaluated using RegulomeDB, which integrates information from ENCODE including DNase hypersensitivity, transcription factor binding, and promoter/enhancer marks. For disease association analysis, we queried the GWAS Catalog to identify reported associations between these SNPs and other diseases, exploring potential shared pathogenic mechanisms. Expression quantitative trait loci (eQTL) effects were extracted from the GTEx V8 dataset across multiple tissues, focusing on effect sizes (β), directions, and significance levels.

2.10. Functional network analysis

We utilized the GeneMANIA platform to integrate datasets of genetic interactions, pathways, and co-expression, along with other gene functional relationships, to gain a deeper understanding of the potential biological functions of the target genes [31,32].

3. Results

3.1. TWAS analyses in cross-tissue and single-tissue

The cross-tissue TWAS analysis identified 259 genes significantly associated with psoriasis ($p < 0.05$; see Table S1), among which 12 genes remained statistically significant after multiple testing correction (PFDR <0.05; see Table S1). Further validation using single-tissue TWAS analysis revealed that 655 genes achieved significance (FDR <0.05) in at least one tissue (see Table S2). Notably, three genes overlapped between the 12 significant genes from the cross-tissue analysis and the single-tissue results (FDR <0.05). These three candidate protein-coding genes POLI, NFKB1, and ZFYVE28 are detailed in Table 1.

3.2. COJO analysis

COJO analysis of POLI, NFKB1, and ZFYVE28 did not reveal any significant tissue-dependent conditional effects. Our results did not uncover notable interactions or condition-specific expression differences among these genes across various tissues, suggesting that they likely exhibit relatively independent regulatory patterns.

3.3. Gene analysis by MAGMA

Gene-level testing using MAGMA identified 649 genes significantly associated with psoriasis (FDR <0.05; see Table S3). To ensure the robustness of our findings, we applied a cross-validation strategy by integrating the three candidate genes from the initial screening with the

Table 1
The significant genes for psoriasis risk in cross-tissue UTMOST analysis.

Gene symbole	CHR	Ensemeble ID	Location (hg38)	UTMOST_P_{FDR}
POLI	18	ENSG00000101751	54269517–54321266	0.001
NFKB1	4	ENSG00000109320	102501330-102617302	0.003
ZFYVE28	4	ENSG00000159733	2269582-2418651	0.034

MAGMA-significant gene set. This approach ultimately highlighted two promising candidate genes: NFKB1 and ZFYVE28 (Fig. 2A).

3.4. MR and colocalization results

The NFKB1 gene, located on chromosome 4q24, was significantly associated with psoriasis in several tissues based on FUSION analysis: Skeletal Muscle (TWAS.Z = 4.079, TWAS.P.fdr = 0.010), Transverse Colon (TWAS.Z = 3.720, TWAS.P.fdr = 0.026), and Cultured Fibroblasts (TWAS.Z = 4.079, TWAS.P.fdr = 0.031). MR analysis confirmed a causal relationship between NFKB1 and psoriasis ($p < 0.05$). In the Cultured Fibroblasts group, the odds ratio (OR) was 1.326 (95 % CI: 1.143–1.538; see Fig. 3). However, suitable instrumental variables could not be identified for MR analysis in the Skeletal Muscle and Transverse Colon tissues. Colocalization analysis further supported these findings, with the posterior probability for hypothesis 4 (PP.H4) being 0.821 in Skeletal Muscle (Fig. 2B), 0.818 in Transverse Colon (Fig. 2C), and 0.815 in Cultured Fibroblasts (Fig. 2D).

The ZFYVE28 gene, located on chromosome 4q32.3, was significantly associated with psoriasis in the Subcutaneous Adipose Tissue (TWAS.Z = 3.572, TWAS.P.fdr = 0.038) and Esophageal Mucosa (TWAS.Z = 3.575, TWAS.P.fdr = 0.036) tissues according to FUSION analysis. MR analysis further confirmed a causal relationship between ZFYVE28 and psoriasis ($p < 0.05$). In the Subcutaneous Adipose Tissue group, the OR was 1.178 (95 % CI: 1.077–1.289), while in the Esophageal Mucosa group, the OR was 1.211 (95 % CI: 1.079–1.338). Colocalization analysis supported these associations, with PP.H4 values of 0.751 in Subcutaneous Adipose Tissue (Fig. 4A) and 0.945 in Esophageal Mucosa (Fig. 4B).

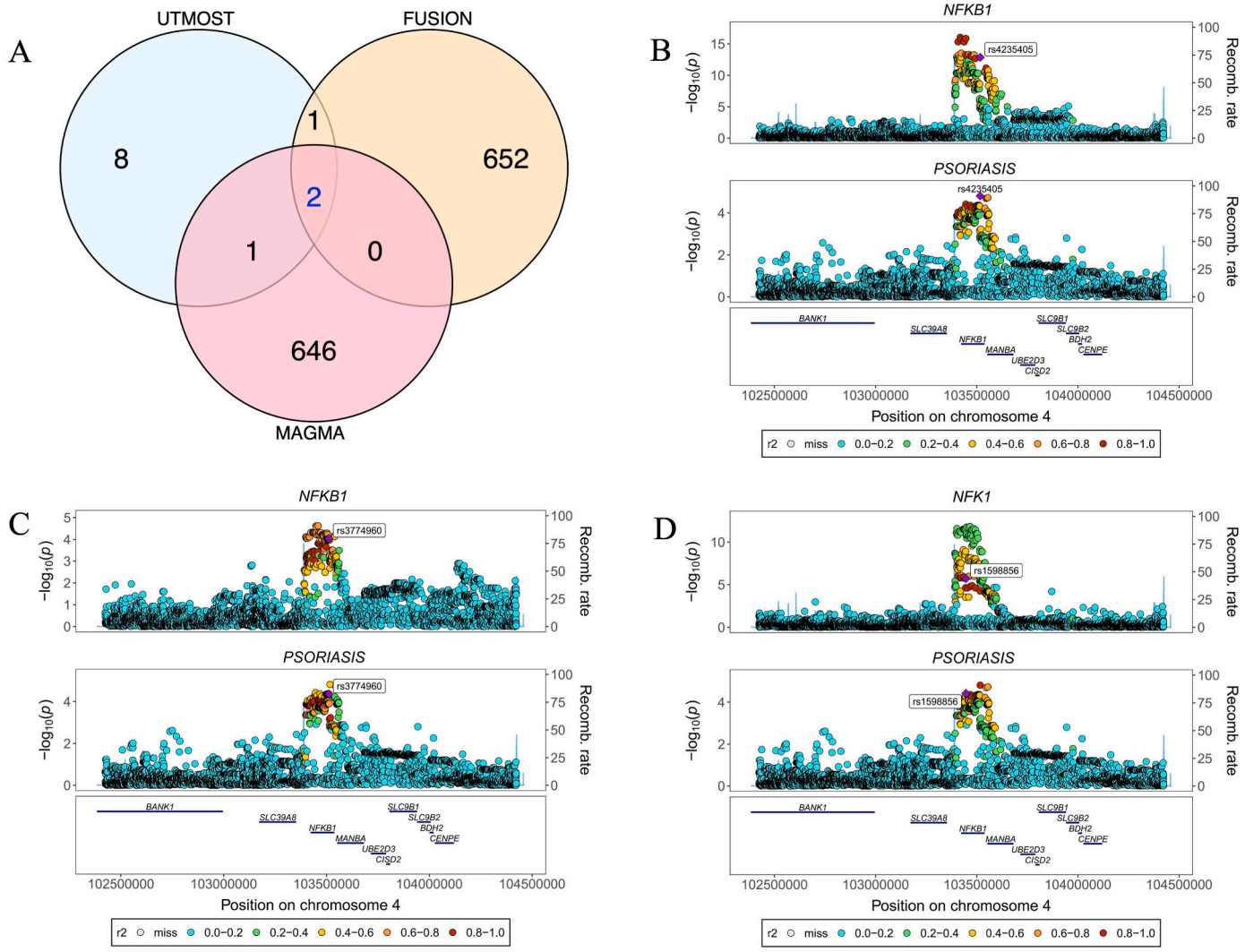

Fig. 2. A. Validation of positive results using UTMOST, FUSION, and MAGMA methods. B. Colocalization results for the NFKB1 gene in Muscle_Skeletal tissue. C. Colocalization results for the NFKB1 gene in Colon_Transverse tissue. D. Colocalization results for the NFKB1 gene in Cells_Cultured_fibroblasts.

Gene	Panel	Method	beta	OR(95%CI)	pval
ZFYVE28	Adipose_Subcutaneous	Wald ratio	0.16	1.17(1.07−1.28)	3.14e−04
ZFYVE28	Esophagus_Mucosa	Wald ratio	0.18	1.21(1.07−1.33)	8.05e−04
NFKB1	Cells_Cultured_fibroblasts	Wald ratio	0.28	1.32(1.14−1.53)	1.91e−04

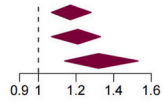

Fig. 3. Forest plot of Mendelian randomization (MR) analysis results.

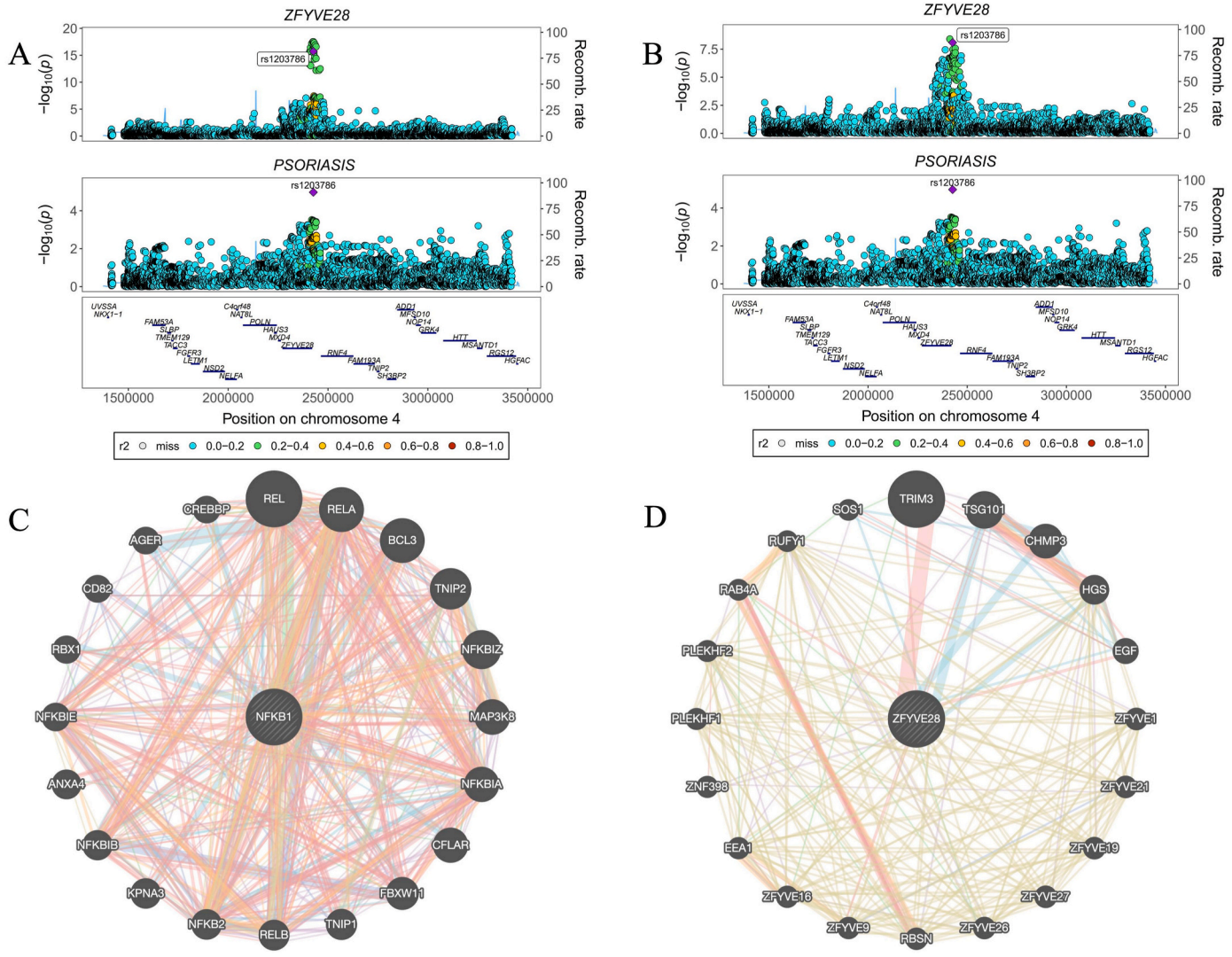

Fig. 4. A. Colocalization results of the ZFYVE28 gene with Adipose_Subcutaneous tissue. BColocalization results of the ZFYVE28 gene with Esophagus_Mucosa tissue. C. GeneMANIA analysis results for the NFKB1 gene. D. GeneMANIA analysis results for the ZFYVE28 gene.

3.5. *Functional annotation and pathogenicity analysis of key SNPs*

To gain deeper insights into the mechanisms by which NFKB1 and ZFYVE28 contribute to psoriasis susceptibility, we performed comprehensive characterization of key SNPs within these gene regions. The NFKB1 gene is located on chromosome 4q24 (precise coordinates chr4:103,422,486–103,538,459 in GRCh38/hg38), spanning approximately 116 kb and containing 24 exons. Within this gene region, we identified three SNPs closely associated with psoriasis (Table S4). rs4235405 (chr4:103,498,421, A/G) functions as a significant eQTL in skeletal muscle tissue ($\beta = 0.28$, $P = 3.9 \times 10^{-5}$); rs3774960 (chr4:103,505,201, C/T) acts as a negative regulator of NFKB1 in transverse colon tissue ($\beta = -0.31$, $P = 2.6 \times 10^{-5}$); and rs1598856 (chr4:103,506,245, A/G) significantly increases NFKB1 expression in cultured fibroblasts ($\beta = 0.33$, $P = 1.8 \times 10^{-6}$). All three SNPs demonstrated high posterior probabilities in colocalization analysis (PP.H4>0.8), supporting the sharing of causal variants between GWAS and eQTL signals.

The ZFYVE28 gene is located on chromosome 4q32.3 (precise coordinates chr4:168,070,071–168,339,852 in GRCh38/hg38), spanning approximately 270 kb and containing 18 exons. Within this gene region, rs1203786 (chr4:168,258,453, C/T) functions as a multi-tissue eQTL, significantly affecting ZFYVE28 expression in both subcutaneous adipose tissue ($\beta = 0.29$, $P = 2.8 \times 10^{-5}$) and esophageal mucosa ($\beta = 0.32$, $P = 1.5 \times 10^{-6}$) (Table S4).

Functional prediction analysis (Table S4) revealed that although

these SNPs are located in non-coding regions, they all reside within functional regulatory elements. Within the NFKB1 gene region, rs1598856 exhibited the highest pathogenicity potential (CADD = 8.241, GERP++ = 4.37), with a RegulomeDB score of 3a, and is located in a strong enhancer region; rs3774960 (CADD = 5.872, RegulomeDB = 4) is positioned near a transcription factor binding site; and rs4235405 (CADD = 4.213, RegulomeDB = 5) is situated in an active enhancer region. Within the ZFYVE28 gene region, rs1203786 (CADD = 6.842, GERP++ = 3.65, RegulomeDB = 4) is located in a regulatory region, supporting its potential as a functional variant.

Beyond psoriasis, these SNPs are associated with various other diseases. SNPs in the NFKB1 gene region are linked to multiple autoimmune disorders: rs4235405 with inflammatory bowel disease, rs3774960 with systemic lupus erythematosus, and rs1598856 with rheumatoid arthritis. The rs1203786 SNP in the ZFYVE28 gene region is associated with type 2 diabetes and other skin disorders, providing new perspectives on the comorbidity between psoriasis and metabolic dysregulation.

3.6. GeneMANIA analysis

The gene interaction network centered on NFKB1 is presented in Fig. 4C. This network is significantly enriched for functional pathways, including the NF-κB signaling pathway, regulation of inflammatory responses, and immune system processes. Similarly, the network built around ZFYVE28 (Fig. 4D) is notably enriched for pathways involved in vesicle-mediated transport, protein transport, and endosomal sorting processes.

4. Discussion

This study employed an integrative multi-omics approach to systematically evaluate the relationship between genetically predicted gene expression and psoriasis risk. Through a cross-tissue TWAS analysis combined with a multi-tier validation strategy, we identified and confirmed two novel psoriasis susceptibility genes, NFKB1 and ZFYVE28. This approach not only overcomes the limitations of functional annotation in traditional GWAS but also provides robust evidence for the causal association between these genes and psoriasis through MR analyses and colocalization analyses.

Furthermore, we performed comprehensive functional characterization of key SNPs within these gene regions, revealing their tissue-specific regulatory effects, pathogenicity potential, and cross-disease associations. This detailed SNP-level annotation enhances our understanding of the molecular mechanisms by which NFKB1 and ZFYVE28 may contribute to psoriasis pathogenesis and highlights potential shared genetic pathways with other autoimmune and metabolic disorders.

As a core member of the NF-κB transcription factor family, the NFKB1 gene plays a crucial role in the complex pathogenesis of psoriasis [10,33] [34]. It encodes the p50 protein, which is essential for binding to consensus sequences within the promoter regions of multiple genes. Studies have shown that a four-nucleotide deletion at the rs28362491 locus in the promoter can markedly reduce promoter activity, thereby affecting protein expression levels and regulatory networks [35].

GWAS have revealed that genes involved in the NF-κB signaling pathway participate in three fundamental networks: (i) those that maintain skin barrier function, (ii) those that regulate the innate immune response mediated by NF-κB and interferon signaling, and (iii) those that govern the adaptive immune response involving $CD8^+$ lymphocytes and Th17 signaling [10]. Against this backdrop, variations in NFKB1 are particularly significant.

At the genetic level, studies in Han Chinese populations have found that the rs1020760 variant is closely associated with both the onset and familial inheritance of psoriasis [36]. Another study indicated that additional variants in the same chromosomal region, such as rs1609798, might also be linked to psoriasis risk [37]. However, the existing findings remain somewhat uncertain and heterogeneous, reflecting the complex genetic architecture of psoriasis.

Variations in NFKB1 may contribute to psoriasis pathogenesis through multiple molecular mechanisms. First, they may modulate gene transcription activity, thereby affecting the expression of inflammation-related genes. Second, they might alter the protein levels of the NF-κB transcription complex, which in turn impacts immune regulation and inflammatory responses [33,34]. Activation of this pathway leads to the transcription of various inflammatory mediators—including pro-inflammatory cytokines, chemokines, and growth factors—that are closely associated with the initiation and persistence of psoriasis [34, 38].

Our functional annotation and pathogenicity prediction analyses provide further evidence supporting the role of NFKB1 in psoriasis pathogenesis. The key SNPs we identified (rs4235405, rs3774960, and rs1598856) are located in regulatory regions with varying degrees of evolutionary conservation and functional significance. Notably, rs1598856 exhibited the highest pathogenicity potential (CADD = 8.241) and is situated in a highly conserved enhancer region with multiple transcription factor binding sites, suggesting its substantial regulatory impact on NFKB1 expression, particularly in fibroblasts. The tissue-specific eQTL effects of these SNPs align with the complex, multi-tissue nature of psoriasis pathology, where altered NFKB1 expression in skeletal muscle, colon, and fibroblasts may contribute to both local and systemic inflammatory processes. Furthermore, the associations of these SNPs with other autoimmune diseases, including inflammatory bowel disease, systemic lupus erythematosus, and rheumatoid arthritis, highlight potential shared genetic mechanisms underlying various immune-mediated disorders. This cross-disease relevance underscores the central position of NFKB1 in immune regulation and supports the concept of common pathways in autoimmune pathogenesis.

It is noteworthy that although current research has elucidated some mechanisms by which NFKB1 influences psoriasis, nearly 50 % of its genetic specificity remains unexplained [10]. Future studies should focus on integrative multi-omics approaches, large sample sizes, and functional validations to more comprehensively unravel the complex regulatory network of NFKB1 in psoriasis, ultimately providing a more precise genetic basis for personalized medicine.

The functional characteristics of the ZFYVE28 gene are also noteworthy. Recent studies have demonstrated that ZFYVE28 is highly expressed in renal tubular epithelial cells and plays an important role in kidney development and function. Its role in regulating the EGF signaling pathway is particularly prominent and may be relevant to the pathogenesis of psoriasis. Given that psoriasis is closely associated with the proliferation and migration of epidermal cells, the enhancement of EGF signaling by ZFYVE28 could be critical in this process [39]. Furthermore, evidence linking ZFYVE28 expression to skin inflammation suggests its potential involvement in psoriasis [40]. Although direct evidence connecting ZFYVE28 to psoriasis is currently lacking, its role in other skin disorders provides valuable insights for future research. Further investigation into the function of ZFYVE28 in psoriasis may reveal novel biomarkers and therapeutic targets.

The functional characterization of rs1203786 in the ZFYVE28 gene region provides additional insights into its potential role in psoriasis. This SNP demonstrates significant eQTL effects in both subcutaneous adipose tissue and esophageal mucosa, tissues that are not traditionally associated with psoriasis but may contribute to the systemic nature of the disease. With a moderate pathogenicity score (CADD = 6.842) and location in a weakly active enhancer region, rs1203786 likely influences ZFYVE28 expression through subtle regulatory mechanisms. Its association with type 2 diabetes is particularly intriguing, given the established comorbidity between psoriasis and metabolic disorders. This connection suggests that ZFYVE28 might function at the intersection of metabolic regulation and immune function, potentially explaining part of the complex relationship between psoriasis and metabolic syndrome. While the exact mechanisms remain to be elucidated, our findings

suggest that altered ZFYVE28 expression, influenced by genetic variants such as rs1203786, may affect cellular proliferation and endothelial function, contributing to both the cutaneous and systemic manifestations of psoriasis.

This study has several limitations. First, although we employed multiple validation strategies, our findings have not yet been replicated in an independent cohort. Second, due to the absence of functional experimental data, the precise roles of these genes in psoriasis pathogenesis remain poorly understood.

Future research should validate the functions of these genes through both in vitro and in vivo experiments. In particular, it is essential to elucidate the regulatory mechanisms governing the expression of NFKB1 and ZFYVE28 in psoriasis-relevant tissues, as well as their interactions with known pathogenic pathways. Such in-depth studies will contribute to the development of novel therapeutic strategies.

5. Conclusion

Using a cross-tissue TWAS analysis combined with a multi-tier validation strategy, we demonstrated that the expression of NFKB1 and ZFYVE28 is associated with psoriasis risk, providing new insights into the genetic architecture of the disease. Nevertheless, further in vitro and in vivo experiments are needed to validate the functions of these genes and to elucidate the regulatory mechanisms of their expression in psoriasis-relevant tissues.

CRediT authorship contribution statement

Fei Yan: Writing – original draft, Formal analysis, Data curation, Conceptualization. **Jing Tao:** Writing – original draft, Visualization, Validation, Supervision. **Jie Liu:** Writing – original draft, Project administration, Methodology, Investigation, Funding acquisition. **Yongliang Chen:** Writing – review & editing, Conceptualization. **Zongju Huang:** Writing – review & editing, Resources, Project administration, Methodology, Investigation, Funding acquisition.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jtauto.2025.100286.

Data availability

Data will be made available on request.

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