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NETWORK BASED FUNCTIONAL AND PATHWAY ANALYSIS OF PSORIASIS ASSOCIATED GENES USING BIOINFORMATICS APPROACHES

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Abstract:

Psoriasis is a chronic immune-mediated inflammatory skin disorder characterized by dysregulated keratinocyte proliferation, immune cell infiltration, and complex genetic susceptibility. Increasing evidence suggests that cytokine signaling pathways and immune regulatory genes play a pivotal role in disease pathogenesis. The present study employed an integrative bioinformatics approach to investigate functional interactions and molecular pathways associated with psoriasis-related genes. A curated set of thirty psoriasis-associated genes, including IL17A, TNF, IL23A, IL12B, STAT3, TYK2, and TRAF3IP2, was analyzed using multiple computational platforms. Protein–protein interaction (PPI) networks were constructed using STRING and GeneMania to identify functional gene interactions and regulatory hubs. Functional enrichment and pathway analyses were performed using Enrichr, DAVID, Reactome, and Metascape to determine significantly enriched biological processes, molecular functions, and signaling pathways.The analysis revealed a highly interconnected gene network predominantly enriched in immune response regulation, cytokine-mediated signaling, leukocyte activation, and inflammatory pathways. Hub gene identification highlighted key regulatory molecules, including IL17A, TNF, IL23A, IL6, and STAT3, which demonstrated strong interaction connectivity and central roles in inflammatory signaling cascades. Pathway enrichment consistently identified IL-17 signaling, TNF signaling, JAK–STAT signaling, and NF-κB signaling pathways as major contributors to psoriasis pathogenesis. The findings provide comprehensive insights into the molecular mechanisms underlying psoriasis and highlight potential therapeutic targets.

Keywords: *Psoriasis; Cytokine Signaling; IL-17 Signaling Pathway; TNF Signaling Pathway; JAK–STAT Pathway; Protein–Protein Interaction Network; Bioinformatics Analysis.*

Introduction

Psoriasis is a chronic, immune-mediated inflammatory skin disorder characterized by abnormal keratinocyte proliferation, epidermal hyperplasia, and infiltration of immune cells, particularly T lymphocytes and dendritic cells. The disease affects approximately 2–3% of the global population and is considered a multifactorial disorder influenced by genetic susceptibility, environmental triggers, and immune dysregulation. Clinically, psoriasis presents as erythematous, scaly plaques that commonly affect the scalp, elbows, knees, and lower back, significantly impacting patient quality of life and increasing the risk of associated comorbidities such as psoriatic arthritis, metabolic syndrome, and cardiovascular disease (3).

The pathogenesis of psoriasis is primarily driven by dysregulated interactions between innate and adaptive immune responses. Among the various immunological pathways involved, the interleukin-23/interleukin-17 (IL-23/IL-17) axis has been recognized as a central mechanism in disease development. Activation of dendritic cells stimulates the production of IL-23, which promotes differentiation and expansion of T helper 17 (Th17) cells. These Th17 cells subsequently produce pro-inflammatory cytokines such as IL-17A, IL-17F, and IL-22, which stimulate keratinocyte proliferation and amplify inflammatory signaling cascades. Additionally, tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) further contribute to immune activation and inflammatory amplification, highlighting the complex cytokine network involved in psoriasis progression (4).

Genetic studies have identified multiple susceptibility loci associated with psoriasis, demonstrating a strong hereditary component in disease development. Genome-wide association studies (GWAS) have revealed several genes involved in immune signaling, cytokine regulation, and epidermal differentiation, including IL17A, IL23A, IL12B, TYK2, CARD14, STAT3, and TRAF3IP2. These genes regulate critical signaling pathways such as JAK–STAT signaling, NF-κB activation, and cytokine-mediated immune responses, which collectively influence inflammatory gene expression and immune cell activation (8). The identification of these susceptibility genes has significantly improved understanding of psoriasis molecular mechanisms and has facilitated the development of targeted biologic therapies.

Methodology

A bioinformatics-based analytical approach was adopted to identify disease-associated genes and molecular pathways involved in psoriasis. The methodology involved identification of psoriasis-related genes, interaction network construction, functional enrichment analysis, and pathway mapping using various web-based databases and bioinformatics tools. This integrated workflow helps in understanding immune dysregulation, inflammatory signaling, and keratinocyte proliferation associated with psoriasis pathogenesis.

The bioinformatics tools used in this study are summarized in Table 1.

Table 1: Bioinformatics Tools and Databases used

Sr. No.	Tool Name	Description	Purpose in Study	Website
1.	**DisGeNET**	A comprehensive platform integrating information on gene–disease associations from curated databases and literature	Identification of psoriasis-associated genes	**https://www.disgenet.org**
2.	**STRING**	A database providing known and predicted protein–protein interactions	Construction of protein–protein interaction networks	**https://string-db.org**

3.	**GeneMANIA**	A gene interaction prediction tool based on co-expression, pathways, and genetic interactions	Exploration of gene interactions and co-expression patterns	**https://genemania.org**
4.	**Enrichr**	An interactive enrichment analysis tool integrating multiple gene libraries	Functional enrichment analysis and hub gene identification	**https://maayanlab.cloud/Enrichr**
5.	**Reactome**	A curated database of biological pathways and reactions	Identification of biological pathways involved in psoriasis	**https://reactome.org**
6.	**DAVID**	Database for Annotation, Visualization and Integrated Discovery	Functional annotation and enrichment of GO terms and KEGG pathways	**https://davidbioinformatics.nih.gov/**
7.	**Metascape**	An integrated platform for gene list annotation **and analysis**	Express analysis, PPI network construction, GO and KEGG enrichment	**https://metascape.org**

Results

1. DisgeneNet

A total of 30 genes associated with psoriasis were selected based on literature evidence and public databases, including IL17A, TNF, IL23A, IL12B, TYK2, CARD14, STAT3, IL13, NOS2, IL6, VEGFA, IL4, APOE, CRP, IL10, TNFAIP3, ERAP1, TRAF3IP2, MKI67, TNIP1, ICAM1, NFKBIA, CAT, CYP1A1, LTA, IL1B, TGFA, IFIH1, and IFNLR1.

Gene	Gene Full Name	N diseases$_g$	N variants$_g$	Score$_{gda}$	N PMIDs	N Chemicals	N PMIDs Chemicals
IL23R	interleukin 23 receptor	34	45	1	4		
IL12B	interleukin 12B	29	115	1	3		
IL23A	interleukin 23 subunit alpha	3	0	1	3		
TNF	tumor necrosis factor	312	9	1	2		
IL13	interleukin 13	44	10	1	2		
CARD14	caspase recruitment domain family ...	9	570	1	2		
TYK2	tyrosine kinase 2	21	451	1	1		
STAT3	signal transducer and activator of tr...	98	316	1	1		
NOS2	nitric oxide synthase 2	87	19	1	1		
IL17A	interleukin 17A	36	0	1	0		
IL4	interleukin 4	56	2	0.95	1		
VEGFA	vascular endothelial growth factor A	114	9	0.95	1		
APOE	apolipoprotein E	105	46	0.95	1		
IL6	interleukin 6	214	9	0.95	1		

Figure 1: Gene list from the tool DisGeNET

ERAP1	endoplasmic reticulum aminopeptid...	6	48	0.9	4		
TRAF3IP2	TRAF3 interacting protein 2	8	143	0.9	3		
TNFAIP3	TNF alpha induced protein 3	28	64	0.9	2		
TNIP1	TNFAIP3 interacting protein 1	8	20	0.9	2		
CRP	C-reactive protein	67	1	0.9	1		
CAT	catalase	97	6	0.9	1		
NFKBIA	NFKB inhibitor alpha	41	125	0.9	1		
MKI67	marker of proliferation Ki-67	25	1	0.9	1		
LTA	lymphotoxin alpha	48	20	0.9	0		
IL10	interleukin 10	128	51	0.9	0		
CYP1A1	cytochrome P450 family 1 subfamily ...	96	4	0.9	0		
ICAM1	intercellular adhesion molecule 1	61	7	0.9	0		
IFIH1	interferon induced with helicase C do...	33	682	0.85	1		
IFNLR1	interferon lambda receptor 1	1	2	0.85	1		
IL1B	interleukin 1 beta	166	5	0.85	1		
TGFA	transforming growth factor alpha	22	15	0.85	1		

Figure 2: Gene list from tool DisGeNET

DisGeNET provided a list of psoriasis-associated genes, which was used as the input gene set for subsequent bioinformatics analyses.

2. STRING analysis

STRING was used to construct a protein–protein interaction network for psoriasis-associated genes.

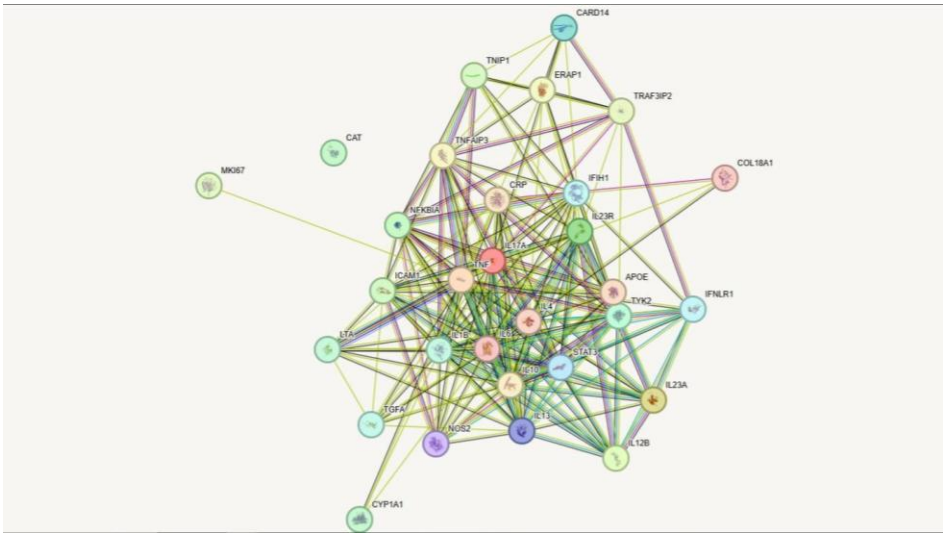
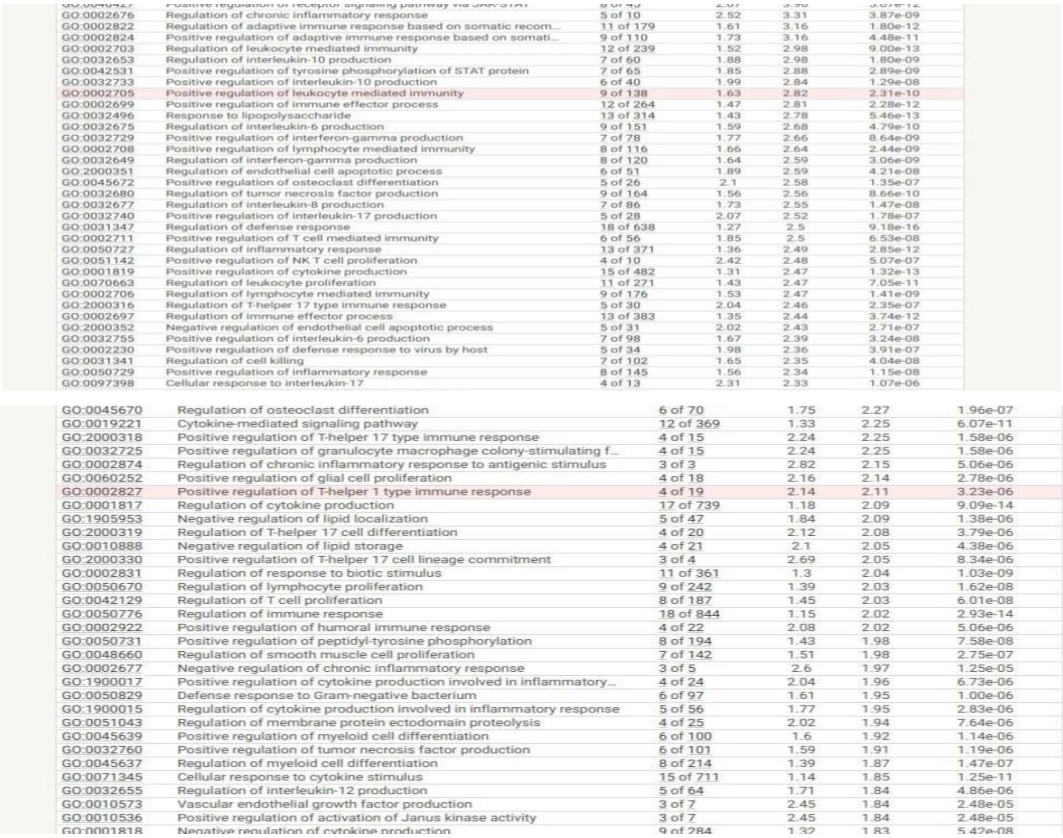


Figure 3: Protein-protein interaction network of 30 psoriasis-associated genes using STRING

The network consisted of 30 interacting genes, indicating strong functional connectivity related to psoriasis.The generated interaction network demonstrated strong connectivity among the proteins, suggesting coordinated molecular activity. Several genes showed multiple interactions, indicating their potential role as key regulatory or hub genes involved in psoriasis.

Gene ontology analysis was performed to identify enriched biological processes associated with the gene set.



GO:0002676	Regulation of chronic inflammatory response	5 of 10	2.52	3.31	3.87e-09
GO:0002822	Regulation of adaptive immune response based on somatic recom...	11 of 179	1.61	3.16	1.80e-12
GO:0002824	Positive regulation of adaptive immune response based on somati...	9 of 110	1.73	3.16	4.48e-11
GO:0002703	Regulation of leukocyte mediated immunity	12 of 239	1.52	2.98	9.00e-13
GO:0032653	Regulation of interleukin-10 production	7 of 60	1.88	2.98	1.80e-09
GO:0042531	Positive regulation of tyrosine phosphorylation of STAT protein	7 of 65	1.85	2.88	2.89e-09
GO:0032733	Positive regulation of interleukin-10 production	6 of 40	1.99	2.84	1.29e-08
GO:0002705	Positive regulation of leukocyte mediated immunity	9 of 138	1.63	2.82	2.31e-10
GO:0002699	Positive regulation of immune effector process	12 of 264	1.47	2.81	2.28e-12
GO:0032496	Response to lipopolysaccharide	13 of 314	1.43	2.78	5.46e-13
GO:0032675	Regulation of interleukin-6 production	9 of 151	1.59	2.68	4.79e-10
GO:0032729	Positive regulation of interferon-gamma production	7 of 78	1.77	2.66	8.64e-09
GO:0002708	Positive regulation of lymphocyte mediated immunity	8 of 116	1.66	2.64	2.44e-09
GO:0032649	Regulation of interferon-gamma production	8 of 120	1.64	2.59	3.06e-09
GO:2000351	Regulation of endothelial cell apoptotic process	6 of 51	1.89	2.59	4.21e-08
GO:0045672	Positive regulation of osteoclast differentiation	5 of 26	2.1	2.58	1.35e-07
GO:0032680	Regulation of tumor necrosis factor production	9 of 164	1.56	2.56	8.66e-10
GO:0032677	Regulation of interleukin-8 production	7 of 86	1.73	2.55	1.47e-08
GO:0032740	Positive regulation of interleukin-17 production	5 of 28	2.07	2.52	1.78e-07
GO:0031347	Regulation of defense response	18 of 638	1.27	2.5	9.18e-16
GO:0002711	Positive regulation of T cell mediated immunity	6 of 56	1.85	2.5	6.53e-08
GO:0050727	Regulation of inflammatory response	13 of 371	1.36	2.49	2.85e-12
GO:0051142	Positive regulation of NK T cell proliferation	4 of 10	2.42	2.48	5.07e-07
GO:0001819	Positive regulation of cytokine production	15 of 482	1.31	2.47	1.32e-13
GO:0070663	Regulation of leukocyte proliferation	11 of 271	1.43	2.47	7.05e-11
GO:0002706	Regulation of lymphocyte mediated immunity	9 of 176	1.53	2.47	1.41e-09
GO:2000316	Regulation of T-helper 17 type immune response	5 of 30	2.04	2.46	2.35e-07
GO:0002697	Regulation of immune effector process	13 of 383	1.35	2.44	3.74e-12
GO:2000352	Negative regulation of endothelial cell apoptotic process	5 of 31	2.02	2.43	2.71e-07
GO:0032755	Positive regulation of interleukin-6 production	7 of 98	1.67	2.39	3.24e-08
GO:0002230	Positive regulation of defense response to virus by host	5 of 34	1.98	2.36	3.91e-07
GO:0031341	Regulation of cell killing	7 of 102	1.65	2.35	4.04e-08
GO:0050729	Positive regulation of inflammatory response	8 of 145	1.56	2.34	1.15e-08
GO:0097398	Cellular response to interleukin-17	4 of 13	2.31	2.33	1.07e-06
GO:0045670	Regulation of osteoclast differentiation	6 of 70	1.75	2.27	1.96e-07
GO:0019221	Cytokine-mediated signaling pathway	12 of 369	1.33	2.25	6.07e-11
GO:2000318	Positive regulation of T-helper 17 type immune response	4 of 15	2.24	2.25	1.58e-06
GO:0032725	Positive regulation of granulocyte macrophage colony-stimulating f...	4 of 15	2.24	2.25	1.58e-06
GO:0002874	Regulation of chronic inflammatory response to antigenic stimulus	3 of 3	2.82	2.15	5.06e-06
GO:0060252	Positive regulation of glial cell proliferation	4 of 18	2.16	2.14	2.78e-06
GO:0002827	Positive regulation of T-helper 1 type immune response	4 of 19	2.14	2.11	3.23e-06
GO:0001817	Regulation of cytokine production	17 of 739	1.18	2.09	9.09e-14
GO:1905953	Negative regulation of lipid localization	5 of 47	1.84	2.09	1.38e-06
GO:2000319	Regulation of T-helper 17 cell differentiation	4 of 20	2.12	2.08	3.79e-06
GO:0010888	Negative regulation of lipid storage	4 of 21	2.1	2.05	4.38e-06
GO:2000330	Positive regulation of T-helper 17 cell lineage commitment	3 of 4	2.69	2.05	8.34e-06
GO:0002831	Regulation of response to biotic stimulus	11 of 361	1.3	2.04	1.03e-09
GO:0050670	Regulation of lymphocyte proliferation	9 of 242	1.39	2.03	1.62e-08
GO:0042129	Regulation of T cell proliferation	8 of 187	1.45	2.03	6.01e-08
GO:0050776	Regulation of immune response	18 of 844	1.15	2.02	2.93e-14
GO:0002922	Positive regulation of humoral immune response	4 of 22	2.08	2.02	5.06e-06
GO:0050731	Positive regulation of peptidyl-tyrosine phosphorylation	8 of 194	1.43	1.98	7.58e-08
GO:0048660	Regulation of smooth muscle cell proliferation	7 of 142	1.51	1.98	2.75e-07
GO:0002677	Negative regulation of chronic inflammatory response	3 of 5	2.6	1.97	1.25e-05
GO:1900017	Positive regulation of cytokine production involved in inflammatory...	4 of 24	2.04	1.96	6.73e-06
GO:0050829	Defense response to Gram-negative bacterium	6 of 97	1.61	1.95	1.00e-06
GO:1900015	Regulation of cytokine production involved in inflammatory response	5 of 56	1.77	1.95	2.83e-06
GO:0051043	Regulation of membrane protein ectodomain proteolysis	4 of 25	2.02	1.94	7.64e-06
GO:0045639	Positive regulation of myeloid cell differentiation	6 of 100	1.6	1.92	1.14e-06
GO:0032760	Positive regulation of tumor necrosis factor production	6 of 101	1.59	1.91	1.19e-06
GO:0045637	Regulation of myeloid cell differentiation	8 of 214	1.39	1.87	1.47e-07
GO:0071345	Cellular response to cytokine stimulus	15 of 711	1.14	1.85	1.25e-11
GO:0032655	Regulation of interleukin-12 production	5 of 64	1.71	1.84	4.86e-06
GO:0010573	Vascular endothelial growth factor production	3 of 7	2.45	1.84	2.48e-05
GO:0010536	Positive regulation of activation of Janus kinase activity	3 of 7	2.45	1.84	2.48e-05
GO:0001818	Negative regulation of cytokine production	9 of 284	1.32	1.83	5.42e-08

Figure 4: Gene ontology biological processes lists

Table 2: GO Biological Process and Molecular Function Enrichment results from STRING

Theme no.	Biological Themes	Representative Enriched GO Biological Process Terms
Theme1	Immune & Inflammatory Response (Core Psoriasis Pathology)	• Regulation of inflammatory response. • Positive regulation of the immune effector process. • Regulation of defense response. • Immune response.
Theme2	Cytokine Production & Cytokine-Mediated Signaling	• Regulation of cytokine production. • Regulation of interleukin-6 production. • Regulation of tumor necrosis factor production.
Theme3	T-Cell, Th17 & Adaptive Immune Activation	• Regulation of T cell mediated immunity. • Regulation of T-helper 17 type immune response. • Positive regulation of lymphocyte mediated immunity.
Theme4	Leukocyte Activation, Migration & Cell Adhesion	• Regulation of leukocyte activation. • Positive regulation of leukocyte cell-cell adhesion. • Regulation of leukocyte mediated immunity.
Theme5	Response to External Stimuli & Host Defense	• Response to lipopolysaccharide. • Defense response to bacterium. • Response to other organisms.

The enriched biological processes were mainly related to immune response and inflammatory regulation.The enriched biological processes were primarily related to immune response, inflammatory regulation, and cellular signaling, highlighting their importance in psoriasis pathogenesis.

3. GeneMania network analysis

GeneMANIA analysis was used to explore gene interactions and co-expression patterns.

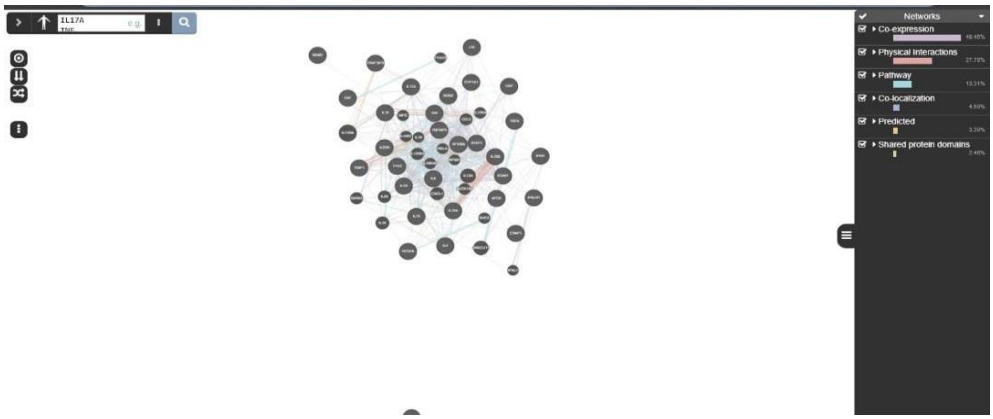


Figure 5: GeneMANIA functional association network highlighting query genes and predicted related genes.

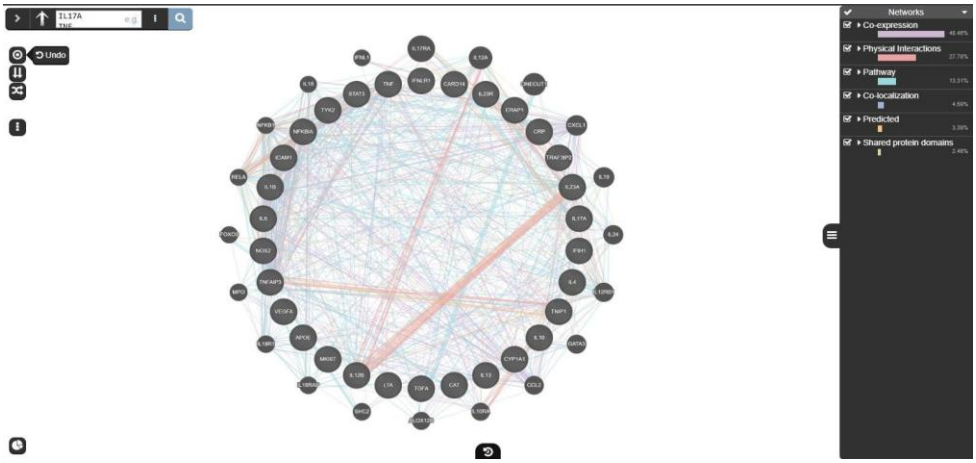


Figure6: GeneMANIA network displaying different interaction types such as co-expression, physical interactions, and predicted associations

The interaction network revealed functional associations and co-expression among psoriasis-related genes. The interaction network revealed functional associations and co-expression among genes, indicating coordinated regulation of immune-related and inflammatory pathways.

4. Enrichr functional enrichment

Enrichr was used to identify functional enrichment across multiple gene libraries.

The Enrichr analysis provided an overview of functional enrichment across multiple gene set

Table 3: Presence of psoriasis-associated genes in Enrichr libraries

30 genes	KEGG 2021 human	Reactome pathway 2024	Wikipathway 2024 human	DisgeneNet	OMIM disease	Jensen disease	CHEA 2022	ENCODE	ARCHS4 human tissues	Allen brain atlas	Count
IL17A	YES	YES	YES	YES		YES	YES		YES	YES	8
TNF	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	10
IL23A	YES	YES	YES	YES		YES	YES	YES	YES		8
IL12B	YES	YES	YES	YES	YES	YES			YES		7
IL23R	YES	YES	YES	YES	YES	YES		YES	YES		8
TYK2	YES	YES	YES			YES	YES	YES			6
CARD14						YES	YES				2
STAT3	YES	YES	YES	YES		YES	YES	YES		YES	8
IL13	YES	YES	YES	YES		YES	YES	YES	YES		8
NOSZ	YES	YES		YES		YES	YES	YES		YES	7
IL6	YES	YES	YES	YES	YES	YES	YES	YES	YES	YES	10
VEGFA		YES	YES	YES		YES	YES	YES		YES	7
IL4	YES	YES	YES	YES		YES	YES	YES	YES	YES	9
APOE				YES	YES		YES				3
CRP				YES		YES	YES				3
IL1O	YES	YES	YES	YES	YES	YES	YES		YES	YES	9
TNFAIP3	YES	YES		YES		YES	YES	YES	YES	YES	8
ERAP1				YES		YES		YES	YES		4
TRAF3IP2	YES			YES		YES	YES	YES		YES	6
MKI67										YES	1
TNIP1				YES			YES	YES	YES		4
ICAM1	YES	YES	YES		YES	YES	YES	YES	YES	YES	9
NFKBIA	YES	YES	YES		YES	YES	YES	YES	YES		8
CAT				YES				YES		YES	3
CYP1A1									YES		1
LTA	YES	YES		YES	YES	YES	YES	YES	YES		8
IL1B	YES	YES	YES	YES	YES	YES	YES		YES	YES	9
TGFA							YES		YES		2
IFIH1	YES	YES						YES	YES		4
IFNLR1	YES	YES						YES	YES		4

Based on their repeated presence across enriched libraries, suggesting their central role in disease-associated molecular mechanisms. Based on the enrichment results across multiple gene libraries, Hub genes were identified by selecting genes that appeared repeatedly in significantly enriched libraries. These genes showed consistent involvement in immune response, inflammatory regulation, and signaling pathways relevant to psoriasis.

5. Reactome pathway analysis

The results highlighted immune-related and inflammatory pathways involved in psoriasis pathogenesis. The analysis highlighted immune-related and inflammatory signaling pathways, emphasizing their contribution to the molecular pathogenesis of psoriasis.

Figure 7: Reactome pathways highlighting the involvement of selected psoriasis related genes

6. DAVID functional annotation

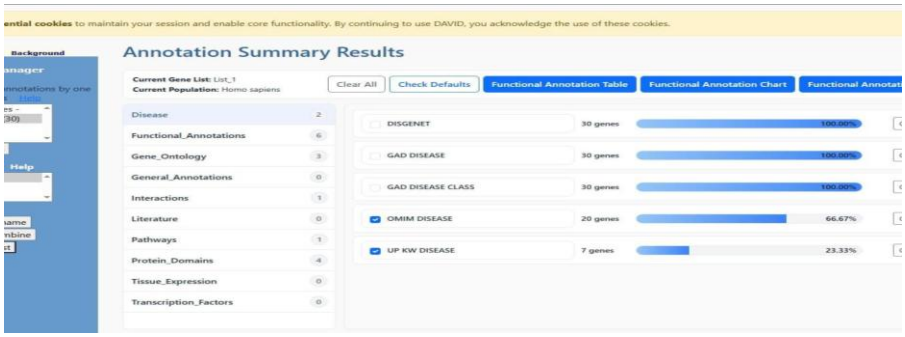


Figure 8: DAVID functional annotation summary.

The enriched gene ontology terms show these genes play key roles in immune system functions. KEGG pathways reveal their involvement in inflammation and immune signaling.

7. Metascape analysis

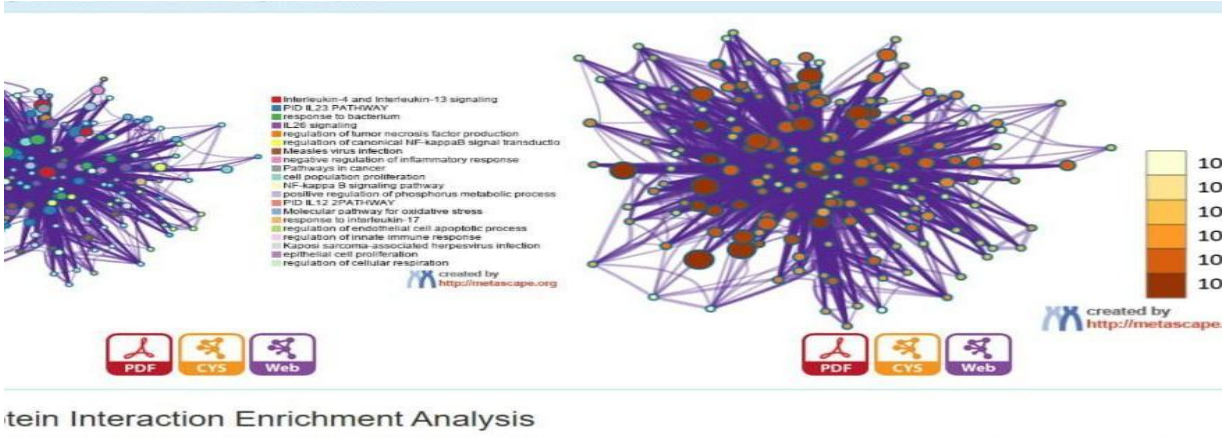


Figure 9: Protein-protein interaction network generated using Metascape.

The analysis generated protein–protein interaction networks along with GO and KEGG enrichment results, identifying functional clusters related to immune response and inflammatory signaling in psoriasis.

Conclusion

This study used bioinformatics to analyze psoriasis-associated genes, revealing key genes, interaction networks, and immune-related pathways. Functional enrichment highlighted inflammatory and immune regulatory

mechanisms in disease pathogenesis. These findings improve understanding of psoriasis molecular mechanisms and may guide biomarker discovery and therapeutic development.

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