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Transcriptome dissection reveals shared upregulated genes and enriched pathways involved in skin disease concurrence

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Abstract

Introduction: The coexistence of skin diseases is common. Although molecular studies have made significant efforts to understand each disease entity, the shared molecular basis explaining their concurrence remains largely unknown. This study aims to identify common upregulated genes in skin diseases that may serve as potential biomarkers.
Methods: Gene expression datasets were retrieved from online databases and compared to healthy controls, focusing on genes common between concurrent diseases. The pathways these molecules are involved in were then analyzed, and their protein–protein interactions were illustrated.
Results: The analysis revealed that *SLC44A5* is strongly upregulated in psoriasis and vitiligo; *ARNTL2* is upregulated in lichen planus and vitiligo; *HIST1H2BG* is upregulated in lichen planus and psoriasis; *S100A7A* is upregulated in psoriasis and alopecia areata; *CXCL9* is strongly upregulated in vitiligo and alopecia areata, as well as in lichen planus and alopecia areata, and cutaneous lupus erythematosus and lichen planus; and *SERPINB4* is upregulated in acne and rosacea.
Conclusions: This study enhances the understanding of skin disease concurrence and lays the groundwork for studying these diseases at a genomic level. It also helps identify common therapeutic targets that work for coexisting diseases.

Keywords: acne, alopecia areata, psoriasis, rosacea, vitiligo

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Introduction

Coexistence of skin diseases is not rare. Based on the latest study, patients with two or more skin diseases make up 1.2% of those treated at dermatology clinics (1). Common combinations of diseases include psoriasis and vitiligo in 52.8% of cases with concurrent disease, followed by lichen planus and vitiligo (17.0%), lichen planus and psoriasis (3.2%), psoriasis and alopecia areata (7.6%), vitiligo and alopecia areata (5.7%), and lichen planus and alopecia areata (3.8%) (1). An association of lichen planus and cutaneous lupus erythematosus to yield lichen planus / lupus erythematosus overlap syndrome is well documented (2). In addition, the association of rosacea and acne is also common, occurring in 25% (3) to 40% of rosacea subjects (4% of acne subjects) (4, 5). Patients with acne concurrent with rosacea tend to be females (over 80%) (3, 4) with a mean age of 23 years (5).

Despite colossal efforts in molecular studies to examine individual diseases, the general molecular background for the co-occurrence of skin diseases is not yet well understood. The focus of this study is to identify the upregulated genes and pathways shared by different skin diseases.

Materials and methods

This research was exempted from review by the Arabian Gulf University's Research and Ethics Committee (reference nos. E32-PI-6-22 and E34-PI-6-22) because this study used publicly anonymized information on individuals available in trusted sources of information such as surveillance databases, registries, and health insurance. Microarray data available on Gene Expression Omnibus (GEO) (Table 1) were analyzed using Transcriptome Analysis Console version 4.0.1.36 (Thermo Fisher Scientific, Waltham, Massachusetts, US) to identify the upregulated genes in each of the diseases compared to healthy controls with a fold change of ≥ 2 and *F*-test < 0.05. The study then identified overlapping genes between the co-occurring diseases.

For gene sets that have multiple probes, the upregulated differentially expressed genes were first identified. Duplicate genes were then discarded and probes unannotated for all diseases. Common genes were identified between diseases of interest by merging data frames using Python 3.12 (Python Software Foundation, Beaverton, OR, US). These genes were then analyzed using Reactome (https://reactome.org/; Ontario Institute for Cancer Research, New York University Langone Medical Center, European Bioinformatics Institute, and Oregon Health & Science University) and ShinyGO 0.82 (http://bioinformatics.sdstate.edu/go/; South Dakota State University). In addition, where relevant, the protein–protein interactions were illustrated using STRING 12.0 (https://string-db.org/; Novo Nordisk Foundation Center for Protein Research, European Molecular Biology Laboratory, Swiss Institute of Bioinformatics, University of Copenhagen, and University of Zurich).

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Table 1 | **List of Gene Expression Omnibus (GEO) accession numbers and number of healthy skin biopsies and lesional skin biopsies for each dataset analyzed per disease.**

Disease	GEO accession	Number of samples	
		Healthy control/ skin	Lesional skin
Acne	GSE53795	—	12
Alopecia areata	GSE45512	5	5
	GSE58573	3	2
	GSE74761	3	3
Cutaneous lupus	GSE184989	4	68
Lichen planus	GSE130403	12	38
Psoriasis	GSE14905	21	33
Rosacea	GSE65914	20	38
Vitiligo	GSE65127	10	10

Results and discussion

Nine data sets were analyzed, and biopsies were collected from 78 healthy subjects and 209 lesional skin subjects.

SLC44A5, *GDA*, and *RNASE7* are strongly upregulated in psoriasis and vitiligo

One hundred sixty-three genes were found to be upregulated in psoriasis and vitiligo. These represented the majority of genes involved in signal transduction, the immune response, and the cell cycle (Fig. S1a). The most significant enriched pathways pertained to *TNFR1*-mediated processes involving ceramide synthesis and proapoptotic signaling (Fig. S2a).

Among these genes, *PI3* (a potent antimicrobial peptide against bacteria and fungi [6], foldchange 44.71, *F*-test = 1.19×10^{-24}), *GDA* (a microtubule assembly protein [7], foldchange 28.63, *F*-test = 2.46×10^{-14}), and *LCE3D* (a keratinization gene [8], foldchange 25.98, *F*-test = 8.13×10^{-25}) are the top three upregulated genes in psoriasis. The three highest upregulated genes in vitiligo were found to be *YIF1B* (involved in endoplasmic reticulum to Golgi vesicle–mediated transport [9], foldchange 53.93, *F*-test = 5.59×10^{-9}), *RNASE7* (has antimicrobial action against bacteria and fungi [10], foldchange 13.83, *F*-test = 2.90×10^{-5}), and *ACPP* (an enzyme involved in substrate dephosphorylation [11], foldchange 11.81, *F*-test = 7.42×10^{-6}).

Based on the findings, *SLC44A5*, *GDA*, and *RNASE7* are greatly upregulated in both conditions. Figure S3a illustrates the protein–protein interaction network for the upregulated common genes of the two diseases.

ARNTL2 is differentially upregulated in lichen planus and vitiligo

A single gene, *ARNTL2*, was found to be overexpressed in lichen planus (foldchange 3.47, $p = 1.69 \times 10^{-13}$) and vitiligo (foldchange 3.27, $p = 0.0002$).

ARNTL2 regulates the circadian rhythm by influencing gene transcription in the nucleus (12) and protein translation in the cytosol (13). Therefore, *ARNTL2* plays roles in various cancers (14–17). The role of *ARNTL2* in inflammation has not been well described. The literature describes *ARNTL2* as a potential modulator of inflammation. For instance, *TNF* is an effective inducer of *ARNTL2* through the NF-κB pathway, promoting its nuclear localization (18). Moreover, *ARNTL2* has direct interaction with the *IL21* gene, which it binds to the promoter, inhibiting its expression (19), suggesting immunosuppressive activity (20). However, the specific relation of *ARNTL2* with other cytokines remains to be elucidated and requires further investigation. The protein–protein interaction network has also shown that *ARNTL2* interacts with many circadian rhythm–related genes, such as *CRY2*, *CSNK1*, *CRY1*, *CLOCK*, *SERPINE1*, *NPAS2*, *ARNTL*, *CIPC*, and *PER2.0*.

HIST1H2BG, *POSTN*, and *NIPBL* were greatly upregulated in psoriasis and lichen planus

Seventy-five upregulated genes were found to be common in lichen planus and psoriasis and involved in the immune system—including interferon signaling, signaling by interleukins (IL-2 family, IL-3, IL-5, granulocyte-macrophage colony-stimulating factor, IL-6 family, IL-7, IL-4 and IL-13, IL-12 family, and IL-37)—chromatin organization, DNA replication, cell cycle, reproduction, and RNA metabolism (Fig. S1b). The majority of the genes are involved in VEGFA/VEGFR2 signaling, transforming growth factor (TGF)-β signaling, oncostatin M signaling, leptin signaling, and feedback loops of STAT3 signaling (Fig. S2b).

Among them, the three highest upregulated genes in psoriasis were *ANXA1* (inhibits neutrophil tissue accumulation [21], foldchange 10.96, $p = 1.61 \times 10^{-9}$), *HIST1H2BG* (a nuclear protein with antimicrobial function [22], foldchange 5.88, $p = 2.77 \times 10^{-8}$), and *MALAT1* (regulates inflammation through toll-like receptors, foldchange 5.8. $p = 6.48 \times 10^{-9}$) (23). However, for lichen planus, the top three upregulated genes were *HIST1H2BG* (foldchange 12.86, $p = 1.48 \times 10^{-8}$), *FGL2* (involved in immunomodulation, where its membrane-bound form mediates inflammation and soluble form induces immunosuppression [24], foldchange 1.3, $p = 1.41 \times 10^{-13}$), and *B2M* (involved in inflammation [25], foldchange 6.48, $p = 1.72 \times 10^{-17}$).

The results indicate that *HIST1H2BG*, *POSTN* (involved in inflammation through the NF-κB pathway) (26), and *NIPBL* were significantly upregulated in both diseases. The protein–protein interaction network is shown in Figure S3b.

S100A7A, *GDA*, and *SLC44A5* are significantly upregulated in psoriasis and alopecia areata

One hundred sixty-nine genes were found to be upregulated in common between psoriasis and alopecia areata, and the majority of them involve the immune system, including innate immunity, cytokine signaling, neutrophil degranulation, and antimicrobial peptides (Fig. S1c). The most enriched pathways are also immune-related pathways like those involved in alternative complement activation, including C3 and C5 activation (Fig. S2c).

The top three genes most upregulated in psoriasis are *SERPINB4* (a chronic inflammation-inducing autoantigen (27), foldchange 582.51, *F*-test = 4.93×10^{-30}), *DEFB4* (microbicidal and cytotoxic neutrophil peptide (28), foldchange 196.82, *F*-test = 1.49×10^{-25}), and *S100A7A* (involved in inflammatory responses (29), foldchange 160.28, *F*-test = 8.97×10^{-22}). On the other hand, the three most upregulated genes in alopecia areata are *ACPP* (foldchange 20.53, *F*-test = 1.16×10^{-5}), *SLC44A5* (a choline transporter (30), foldchange 19.11, *F*-test = 1.91×10^{-7}), and *VNN1* (with roles in oxidative-stress responses (31), foldchange 14.06, *F*-test = 1.16×10^{-7}).

S100A7A, *GDA*, and *SLC44A5* are strongly upregulated in both. The protein–protein interaction network is shown in Figure S3c.

CXCL9 is strongly upregulated in both alopecia areata and vitiligo

Both vitiligo and alopecia areata had nine upregulated genes in common. They participate in signal transduction and IL-10 signaling (Fig. S1d). The majority of these genes are chemokine receptors recruiting chemokines (Figs. S2d and S3d). *CXCL9* was found to be significantly upregulated in both diseases.

CXCL9, *CXCL10*, and *LYZ* are highly upregulated in alopecia areata and lichen planus

Thirty-three genes were found to be upregulated in lichen planus and alopecia areata, with the majority playing a role in the immune system such as in the adaptive immune system (T-cell receptor [TCR] signaling and major histocompatibility complex [MHC] class II presentation) and cytokine signaling (interferon signaling, IL-10 signaling, and IL-12 family signaling), Figures S1e and S2e.

Of these genes, the top three upregulated genes in alopecia areata were *CXCL9* (involved in immunoregulatory and inflammatory processes [32], foldchange 12.16, $p = 1.49 \times 10^{-6}$), *CXCL10* (elicits the migration of monocytes, natural killer cells, and T-cell and controls adhesion molecule expression [33], foldchange 7.83, $p = 3.46 \times 10^{-5}$), and *GZMA* (a protease with proinflammatory properties [34], foldchange 4.61, $p = 8.91 \times 10^{-9}$). However, the three top upregulated genes in lichen planus were *CXCL9* (foldchange 157.14, $p = 4.68 \times 10^{-26}$), *CXCL10* (foldchange 45.72, $p = 1.24 \times 10^{-18}$), and *LYZ* (amplifies inflammation [35], foldchange 15.05, $p = 5.35 \times 10^{-20}$).

CXCL9, *CXCL10*, and *LYZ* were significantly upregulated in both diseases. The protein–protein interaction network is shown in Figure S3e.

CXCL9 and *S100A9* are significantly upregulated in cutaneous lupus erythematosus and lichen planus

A total of 1,697 genes were found to be upregulated in lichen planus and cutaneous lupus erythematosus, and the majority of them are related to the immune system (innate immune system, adaptive immune system, and cytokine signaling), signal transduction, protein metabolism, cell cycle, programmed cell death, and DNA replication (Figs. S1f and S2f).

The top three upregulated genes in lichen planus were *CXCL9* (foldchange 138.19, *F*-test = 2.84×10^{-29}), *S100A9* (released from cells upon inflammation, infection, or cell damage [36], foldchange 102.03, *F*-test = 2.72×10^{-29}), and *PI3* (foldchange 59.97, *F*-test = 1.21×10^{-17}), whereas the top three upregulated genes in cutaneous lupus erythematosus were *CXCL10* (foldchange 80.21, *F*-test = 1.11×10^{-19}), *S100A9* (foldchange 72.76, *F*-test = 2.72×10^{-29}), and *CXCL9* (foldchange 65.12, *F*-test = 2.84×10^{-29}).

Based on this observation, *CXCL9* and *S100A9* are strongly upregulated in both conditions.

SERPINB4, *S100A7A*, and *RPS4Y1* are extremely upregulated in acne and rosacea

Six hundred thirty-seven genes were differentially upregulated in rosacea and acne, with the majority of them being immune related such as those involved in cytokine signaling (IL-4 and IL-13 family, IL-10, and IL-20 family), the adaptive immune system (B-cell receptor signaling, MHC class II antigen presentation, and TCR signaling), and the innate immune system (neutrophil degranulation, Fc-gamma receptor dependent phagocytosis, and Fc-epsilon signaling), Figure S1g. The top enriched pathways were alpha-linolenic omega-3 and linoleic omega-6 acid metabolism and alpha-linolenic acid metabolism (Fig. 2g). Of these genes, the most significantly upregulated genes among acne were *SERPINB4* (a serine protease inhibitor involved in inflammation [37], foldchange 384.53, *F*-test = 3.39×10^{-30}), *S100A7A* (an antimicrobial peptide [38], foldchange 209.48, *F*-test = 8.36×10^{-25}), and *CXCL8* (involved in inflammation through neutrophil recruitment [39], foldchange 166.67, *F*-test = 2.01×10^{-15}). In rosacea, the most upregulated genes were *RPS4Y1* (induces inflammation through p38 MAPK activation pathway [40], foldchange 99.05, *F*-test = 3.41×10^{-05}) and *SERPINB4* (foldchange 146.24, *F*-test = 3.39×10^{-30}).

SERPINB4, *S100A7A*, and *RPS4Y1* were significantly upregulated in both conditions.

Conclusions

This study has outlined the overexpressed genes and pathways shared among eight of the most common concomitant cutaneous diseases: psoriasis and vitiligo, lichen planus and vitiligo, lichen planus and psoriasis, psoriasis and alopecia areata, vitiligo and alopecia areata, lichen planus and alopecia areata, cutaneous lupus erythematosus and lichen planus, and rosacea and acne. This enhances the understanding of skin disease concurrence and lays the groundwork for studying them at a genomic level. It also helps identify common therapeutic targets that work for coexisting diseases.

References

1. Hamdi D, Alhamdi K, Kadhim T. Alternating and coexisting skin diseases: are they real concepts? J Pakistan Assoc Dermatologists. 2023;33:525–31.
2. Jicha KI, Wang DM, Miedema JR, Diaz LA. Cutaneous lupus erythematosus / lichen planus overlap syndrome. JAAD Case Rep. 2021;17:130–51.
3. Mekokishvili L. Coexistence of acne and rosacea: results of a single center retrospective study. Paper presented at 24th World Congress of Dermatology; 2019 Jun 10–15; Milan.
4. Yoshizawa Y. Rosacea in adolescents and young adults: high prevalence concurrent with acne vulgaris treated with benzoyl peroxide and/or adapalene. J Clin Cases Rep. 2023;2023:265–76.
5. Chen H, Lai W, Zheng Y. Rosacea in acne vulgaris patients: subtype distribution and triggers assessment—a cross-sectional study. J Cosmet Dermatol. 2021;20:1889–96.
6. Deng J, Leijten E, Zhu Y, Nordkamp MO, Ye S, Pouw J, et al. Multi-omics approach identifies PI3 as a biomarker for disease severity and hyper-keratinization in psoriasis. J Dermatol Sci. 2023;111:101–8.
7. Fernandez JR, Sweet ES, Welsh WJ, Firestein BL. Identification of small molecule compounds with higher binding affinity to guanine deaminase (cypin) than guanine. Bioorg Med Chem. 2010;18:6748–55.
8. LCE3D late cornified envelope 3D [Homo sapiens (human)]: National Library of Medicine; 2025 [updated 2025 Jan 4, cited 2025 Jul 29]. Available from: https://www.ncbi.nlm.nih.gov/gene/84648.
9. Alterio J, Masson J, Diaz J, Chachlaki K, Salman H, Areias J, et al. Yif1B is involved in the anterograde traffic pathway and the Golgi architecture. Traffic. 2015;16:978–93.

10. Rademacher F, Dreyer S, Kopfnagel V, Glaser R, Werfel T, Harder J. The antimicrobial and immunomodulatory function of RNase 7 in skin. Front Immunol. 2019; 10:2553.
11. ACP3 acid phosphatase 3 [Homo sapiens (human)]: National Library of Medicine; 2025 [cited 2025 Jul 29]. Available from: https://www.ncbi.nlm.nih.gov/gene/55.
12. Xiong W, Li J, Zhang E, Huang H. BMAL1 regulates transcription initiation and activates circadian clock gene expression in mammals. Biochem Biophys Res Commun. 2016;473:1019–25.
13. Lipton JO, Yuan ED, Boyle LM, Ebrahimi-Fakhari D, Kwiatkowski E, Nathan A, et al. The circadian protein BMAL1 regulates translation in response to S6K1-mediated phosphorylation. Cell. 2015;161:1138–51.
14. Wang S, Ma X, Ying Y, Sun J, Yang Z, Li J, et al. Upregulation of ARNTL2 is associated with poor survival and immune infiltration in clear cell renal cell carcinoma. Cancer Cell Int. 2021;21:341.
15. Wang T, Wang K, Zhu X, Chen N. ARNTL2 upregulation of ACOT7 promotes NSCLC cell proliferation through inhibition of apoptosis and ferroptosis. BMC Mol Cell Biol. 2023;24:14.
16. Zhang H, Shan G, Jin X, Yu X, Bi G, Feng M, et al. ARNTL2 is an indicator of poor prognosis, promotes epithelial-to-mesenchymal transition and inhibits ferroptosis in lung adenocarcinoma. Transl Oncol. 2022;26:101562.
17. Wang Z, Liu T, Xue W, Fang Y, Chen X, Xu L, et al. ARNTL2 promotes pancreatic ductal adenocarcinoma progression through TGF/BETA pathway and is regulated by miR-26a-5p. Cell Death Dis. 2020;11:692.
18. Olkkonen J, Kouri VP, Kuusela E, Ainola M, Nordstrom D, Eklund KK, et al. DEC2 blocks the effect of the ARNTL2/NPAS2 dimer on the expression of PER3 and DBP. J Circadian Rhythms. 2017;15:6.
19. Lebailly B, He C, Rogner UC. Linking the circadian rhythm gene Arntl2 to interleukin 21 expression in type 1 diabetes. Diabetes. 2014;63:2148–57.
20. Wu G, Ren H, Hu Q, Ma H, Chen H, Zhou L, et al. The circadian rhythm key gene ARNTL2: a novel prognostic biomarker for immunosuppressive tumor microenvironment identification and immunotherapy outcome prediction in human cancers. Front Immunol. 2023;14:1115809.
21. Sugimoto MA, Vago JP, Teixeira MM, Sousa LP. Annexin A1 and the resolution of inflammation: modulation of neutrophil recruitment, apoptosis, and clearance. J Immunol Res. 2016;2016:8239258.
22. Agak GW, Mouton A, Teles RM, Weston T, Morselli M, Andrade PR, et al. Extracellular traps released by antimicrobial TH17 cells contribute to host defense. J Clin Invest. 2021;131.
23. Zhao G, Su Z, Song D, Mao Y, Mao X. The long noncoding RNA MALAT1 regulates the lipopolysaccharide-induced inflammatory response through its interaction with NF-kappaB. FEBS Lett. 2016;590:2884–95.
24. Fu L, Liu Z, Liu Y. Fibrinogen-like protein 2 in inflammatory diseases: a future therapeutic target. Int Immunopharmacol. 2023;116:109799.
25. Muneshige K, Uchida K, Aikawa J, Iwase D, Takano S, Miyagi M, et al. beta2-microglobulin alters the profiles of inflammatory cytokines and of matrix metalloprotease in macrophages derived from the osteoarthritic synovium. Cent Eur J Immunol. 2022;47:332–8.
26. Muratsu J, Sanada F, Koibuchi N, Shibata K, Katsuragi N, Ikebe S, et al. Blocking periostin prevented development of inflammation in rhabdomyolysis-induced acute kidney injury mice model. Cells. 2022;11:3388.
27. Iversen OJ, Lysvand H, Slupphaug G. Pso p27, a SERPINB3/B4-derived protein, is most likely a common autoantigen in chronic inflammatory diseases. Clin Immunol. 2017;174:10–7.
28. Suarez-Carmona M, Hubert P, Delvenne P, Herfs M. Defensins: "Simple" antimicrobial peptides or broad-spectrum molecules? Cytokine Growth Factor Rev. 2015;26:361–70.
29. Liang H, Li J, Zhang K. Pathogenic role of S100 proteins in psoriasis. Front Immunol. 2023;14:1191645.
30. Hedtke V, Bakovic M. Choline transport for phospholipid synthesis: an emerging role of choline transporter-like protein 1. Exp Biol Med (Maywood). 2019;244: 655–62.
31. Yu H, Cui Y, Guo F, Zhu Y, Zhang X, Shang D, et al. Vanin1 (VNN1) in chronic diseases: future directions for targeted therapy. Eur J Pharmacol. 2024;962:176220.
32. Yin X, Wang Z, Wu T, Ma M, Zhang Z, Chu Z, et al. The combination of CXCL9, CXCL10 and CXCL11 levels during primary HIV infection predicts HIV disease progression. J Transl Med. 2019;17:417.
33. Gudowska-Sawczuk M, Mroczko B. What is currently known about the role of CXCL10 in SARS-CoV-2 infection? Int J Mol Sci. 2022;23:3673.
34. Metkar SS, Menaa C, Pardo J, Wang B, Wallich R, Freudenberg M, et al. Human and mouse granzyme A induce a proinflammatory cytokine response. Immunity. 2008;29:720–33.
35. Xi Z, Shu L, Xiao L, Fang X, Dai M, Wang J, et al. Macrophage NLRP3 inflammasome mediates the effects of sympathetic nerve on cardiac remodeling in obese rats. Circ Res. 2024;135:596–613.
36. Nasser MW, Elbaz M, Ahirwar DK, Ganju RK, Conditioning solid tumor microenvironment through inflammatory chemokines and S100 family proteins. Cancer Letters. 2015; 365:11–22.
37. Xiao W, Sha K, Wang M, Tan Z, Wang Y, Xu S, et al., SERPINB3/B4 Is Increased in Psoriasis and Rosacea Lesions and Has Proinflammatory Effects in Mouse Models of these Diseases. Journal of Investigative Dermatology. 2024; 144:2706–18 e6.
38. Son ED, Kim HJ, Kim KH, Bin BH, Bae IH, Lim KM, et al., S100A7 (psoriasin) inhibits human epidermal differentiation by enhanced IL-6 secretion through IkappaB/NF-kappaB signalling. Experimental Dermatology. 2016; 25:636–41.
39. Alsabbagh MM, Cytokines in psoriasis: From pathogenesis to targeted therapy. Human Immunology. 2024; 85:110814.
40. Chen Y, Chen Y, Tang C, Zhao Q, Xu T, Kang Q, et al., RPS4Y1 Promotes high glucose-induced endothelial cell apoptosis and inflammation by activation of the p38 MAPK signaling. Diabetes, Metabolic Syndrome Obesity. 2021; 14:4523–34.