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Gene Expression Changes in Scalp Psoriasis Lesions Treated with Biologic Agents

Steenhuis Cipriano*

Department of Regenerative Cosmetology and Trichological Science, Kyoto University, Kyoto, Japan

Introduction

Psoriasis is a chronic, immune-mediated skin disease that affects approximately 2–3% of the global population. Characterized by well-demarcated, erythematous, and scaly plaques, psoriasis results from a dysregulated immune response involving both the innate and adaptive arms. Among its various clinical forms, scalp psoriasis is particularly challenging to treat due to its frequent recurrence, distinctive hair-bearing anatomical site, and impact on psychosocial well-being. More than 50% of psoriatic patients present with scalp involvement at some point during their disease course, often with debilitating pruritus, bleeding, and physical discomfort. In recent years, the advent of biologic agents-targeted therapies designed to modulate specific components of the immune system-has revolutionized the treatment of moderate-to-severe psoriasis, including scalp psoriasis. Biologics such as Tumor Necrosis Factor-alpha (TNF-α) inhibitors, interleukin (IL)-17 inhibitors, and IL-23 inhibitors have shown remarkable clinical efficacy, offering rapid clearance and prolonged remission [1-3].

While clinical improvements in plaque morphology are well-documented, there is growing interest in understanding how these treatments affect molecular and genomic pathways underlying the disease. Studies of gene expression profiling in psoriatic lesions have uncovered a "psoriatic transcriptome" composed of genes that drive inflammation, keratinocyte hyperproliferation, angiogenesis, and immune activation. Investigating how this gene signature changes in response to biologics can provide deeper insights into treatment mechanisms, biomarkers of response, and the potential for disease modification.

Description

Scalp psoriasis, while clinically distinct, shares the core pathogenic mechanisms with plaque psoriasis. At the heart of its development lies a genetic predisposition combined with environmental and immune triggers. Genome-Wide Association Studies (GWAS) have identified over 60 susceptibility loci, many of which regulate T-cell activation, cytokine signaling, and antigen presentation. In the psoriatic scalp lesion, several gene families are consistently overexpressed. These genes collectively sustain a self-amplifying inflammatory loop, where activated dendritic cells and keratinocytes produce IL-23 and TNF-α, fueling the expansion of pathogenic Th17 and Th1 cells, which in turn secrete IL-17 and IFN-γ to drive keratinocyte proliferation and barrier dysfunction. Importantly, scalp-specific features-such as the presence of terminal hair follicles, sebaceous glands, and a unique microbiome-may modify this transcriptomic profile, creating a subset of scalp-specific biomarkers and therapeutic targets.

Studies on etanercept and adalimumab reveal a rapid reduction in the expression of inflammatory cytokines (IL-1β, IL-6), chemokines (CXCL1, CXCL10), and keratinocyte activation markers (KRT6A, S100A7). Within 4 weeks, many genes return to non-lesional levels. However, some genes-such as IL-17A and IL-22-remain resistant to suppression, suggesting incomplete normalization. Ustekinumab downregulates IL-23A, IL-12B, and Th1/Th17-associated transcripts. Gene expression changes correlate strongly with histological improvement. Notably, ustekinumab reduces expression of IFN-γ-related genes, such as STAT1 and CXCL9/10, highlighting its unique effect on Th1 polarization. IL-17 blockers like secukinumab and ixekizumab result in dramatic and swift suppression of the "IL-17 transcriptome," including DEFB4A, CCL20, IL-36G, and S100A8/A9. These agents also reduce epidermal hyperplasia and neutrophil-associated gene expression. By week 12, gene expression in treated lesions closely resembles non-lesional skin, achieving near-complete molecular remission. Guselkumab and risankizumab produce slower but more sustained gene normalization. These agents reduce IL-23-dependent genes like IL-17A/F, IL-22, and RORC. Importantly, they also normalize keratinocyte-derived genes such as KRT17, indicating reversal of the hyperproliferative state. Long-term data show persistence of transcriptomic normalization even after discontinuation [4,5].

The concept of molecular remission-the reversion of lesional gene expression to a non-lesional or normal state-has emerged as a crucial metric for biologic efficacy. Studies show that early molecular responders often achieve better long-term clinical remission, lower relapse rates, and improved quality of life. Furthermore, baseline gene expression profiles may predict therapeutic response. Such molecular stratification paves the way for precision medicine in psoriasis, tailoring therapy to individual gene signatures and optimizing outcomes. Most studies are performed on trunk or limb lesions. Scalp psoriasis may have distinct transcriptomic signatures influenced by hair follicles, sebum, and microbial flora. Most gene expression studies are conducted in adults aged 18–65. Pediatric and geriatric scalp psoriasis may involve different immune and genomic mechanisms. RNA-seq and transcriptomics are expensive and not routinely available in clinical practice.

Conclusion

Biologic agents not only offer profound clinical benefits in scalp psoriasis but also reshape the underlying pathogenic gene expression profile, driving lesions toward a state of molecular remission. Agents targeting IL-17 and IL-23 demonstrate the most consistent transcriptomic normalization, highlighting their central role in disease pathogenesis. Understanding these molecular changes offers a dual advantage: it elucidates the mechanism of action of biologics and provides a framework for personalized therapy. The ability to predict response based on gene signatures and monitor remission through molecular profiling may soon transform scalp psoriasis management from empiric treatment to precision medicine. As scalp psoriasis remains a stigmatizing and difficult-to-treat condition, integrating genomic insights with clinical practice will be key to achieving lasting remission, improving patient quality of life, and advancing the future of dermatologic care.

***Address for Correspondence:** Steenhuis Cipriano, Department of Regenerative Cosmetology and Trichological Science, Kyoto University, Kyoto, Japan; E-mail: steenhuipriano@ao.jp*

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Conflict of Interest

None.

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