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Staphylococcus aureus aggravates psoriasis in human keratinocytes with the assistance of IL-17A and TNF- α, along with a contribution from genes associated with skin cutaneous melanoma

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ABSTRACT

Psoriasis patients were 4- 5 times more likely to have S. aureus colonize their skin. Psoriasis is caused by staphylococcal infection-induced keratinocyte death, which is maintained by elevated cytokine production of TNF-α and IFN-γ. The study sought to determine the association between the influence of gene expression of certain genes in psoriasis infections in the presence of S. aureus and their effect in Skin Cutaneous Melanoma infections. GEO with accession number (GSE207390) was used to acquire the data. The microarray test was used to perform this investigation. Six sets of keratinocytes, IL-17A, and TNF-α were cultivated together. Several kinds of bioinformatics tools were employed to fulfill the study's objective. Gene hub analysis of 34284 genes was scanned. Six genes demonstrated high expression rates were expressed during the metastatic stage: *CCL27*, *IL19*, *PAPPA2*, *UHRF1*, *IFNAR2*, and *GLS2*. *IFNAR2* and *UHRF1* had the highest levels of expression. The expression of the *COL4A4* gene rose in response to sun exposure, but the expression of the other genes remained same. The existence of cytokine groups with *S. aureus* in keratinocytes influences the expression difference compared to the other groups. This work adds to our understanding of the molecular alterations that occur in the epidermis of psoriasis patients, as well as their relationship to comorbidities.

Keywords: Psoriasis; *Staphylococcus aureus*; Keratinocytes; Interleukin-17; Tumor Necrosis.

INTRODUCTION

One of the most prevalent inflammatory skin diseases, psoriasis is thought to have several etiological variables, such as trauma, alcohol, medications, infections, excessive topical corticosteroid usage, and endocrine abnormalities. Certain kinds of bacteria, including staphylococci and streptococci, are known to be linked to the initiation and aggravation of psoriasis [1]. Patients with psoriasis had a 4- 5 fold increased risk of *S. aureus* colonizing their skin. The staphylococcal enterotoxins found in high concentrations in *S. aureus* isolates from psoriasis patients suggest that the presence of both agr-positive genes and toxins may be essential for disease activity in psoriasis patients. In contrast to those suffering from lichen planus or atopic dermatitis (AD) [2, 3].

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Superantigens and proteolytic toxins may be able to prolong skin inflammation by directly disrupting the normal skin barrier and activating skin-targeted T-cells. Super-antigens could only be crucial in a small percentage of instances, despite the fact that they are probably one component that can regulate how a disease progresses. Few research has found that psoriasis patients have genes that encode S. aureus toxins. This finding might lend credence to the idea that super-antigens contribute to the aggravation of psoriasis [4, 5].

According to a previous study, staphylococcal infection causes keratinocyte destruction in psoriasis, which is sustained by increased production of the cytokines TNF-α and IFN-γ. Staphylococcal peptidoglycan can also encourage keratinocyte growth, and staphylococcal enterotoxins can stimulate T-cells, resulting in a more systemic immunological response [6, 7].

Antibiotics frequently have broad-spectrum action, which can cause skin damage. Additionally, the maintenance treatment of inflammatory skin disorders is under strain due to the rise in antibiotic resistance. Chronic inflammation may be sustained by the overabundance of S. aureus in the microbiome of psoriasis patients [8, 9].

Although it has previously been demonstrated that *S. aureus* can exacerbate psoriatic inflammation and that IL-17A/TNF-A regulate the transcriptional programs in human keratinocytes, the interplay between S. aureus and IL-17A or TNF-A on the expression of human keratinocyte genes has not been profiled systematically through reproducible microarray pipelines. Besides, the applicability of these keratinocyte signatures in skin cutaneous melanoma (SKCM) has not been explored in a translational manner.

MATERIALS AND METHODS

Data collection: The data was collected from GEO with accession number (GSE207390). Raw CEL files (GSE207390_RAW.tar) and the Series Matrix file (GSE207390_series_ matrix.txt.gz) were downloaded from NCBI GEO (accession: GSE207390) on 2025-03-15. Data were obtained from https://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE207390. The background correction, normalization, and summarization of probes were done using raw CEL files to facilitate standardized analysis of the probes. The cross-checking of sample annotations was done by the Series Matrix.

Sample annotation & group mapping: The distribution of the A6 study groups was in the following manner; control (keratinocytes only), keratinocytes and S. aureus, keratinocytes and IL-17A, keratinocytes and TNF-alpha, keratinocyte and (*S. aureus* and IL-17A), and keratinocyte and (*S. aureus* and TNF-alpha).

Experimental groups and sample annotation: Annotation of experimental samples and data Raw data of keratinocyte transcriptomes were retrieved at NCBI GEO (GSE207390). The dataset is featured with 27 samples and 18 of them were chosen to be analyzed. The GSM IDs and group mapping are presented in Supplementary Table S1. The samples were stratified into six experimental groups based on the treatment conditions (see Supplementary Table S1): Control group (Ctrl): keratinocytes in culture without stimulation (GSM6285367 - GSM6285369). IL-17A condition: keratinocytes stimulated by IL-17A in solitude (GSM6285 370GSM6285372). TNF- alpha group: TNF- alpha alone (GSM6285373 -GSM6285375) stimulated keratinocytes. *S. aureus* cluster: keratinocytes incubated with heat-killed Staphylococcus aureus isolated (GSM62853767SM62853760). *S. aureus* + IL-17A group keratinocytes co-stimulated with S. aureus and IL-17A (GSM6285382GSM6285384). *S. aureus* + TNF-alpha group: keratinocytes that were co-stimulated with S. aureus and TNF-alpha (GSM6285385–GSM6285387).

Three biological replicates were present in each group. The metadata in the form of GSM accession numbers, names of the samples, and names of groups can be found in Supplementary Table S1 to guarantee the ability to reproduce and be transparent about the group assignment.

Supplementary Table S1 contains sample-group mapping, metadata (GSM IDs, group labels, replicates, and batch). Sample to group mapping is provided in Supplementary Table S1.

The data was analyzed using R 4.3.1. The packages and version were the following: affy v1.74, limma v3.56, hgu133plus2.db v3.13, ggplot2 v3.4, sva v3.48. They were imported to affy::ReadAffy(raw CEL files). RMA (affy::rma) was used to perform background correction, normalization and summarization. The intensities at the probe-level were aggregated to probesets, which result in log2 expression values.

GEO2R, STRING, Meatscape, R package, iDEP 1.1, ShinyGO v0.741, and UAMP were used to test of more than 34284 genes under the study. Data results of 250 genes were extracted.

A 9 out of 250 genes were selected for more investigation included applied on the ULCAN database for determining their expressions in the metastasis stages. Bulk tissue gene expression was used to visualize the expression in genders in different tissues using GTEx portal Analysis.

RESULTS AND DISCUSSION

Gene hub analysis of 34284 genes was scanned. Plots of quality control were created prior to and following normalization. Boxplots ensured that the medians were aligned after the RMA, which was a good sign of normalized data. PCA plots indicated the obvious segregation of the biological groups with less batch effect. Volcano plots were used to identify any notable differentially expressed genes (adj.$p \leq 0.05$, and log2FC ≥ 1; Fig. 1).

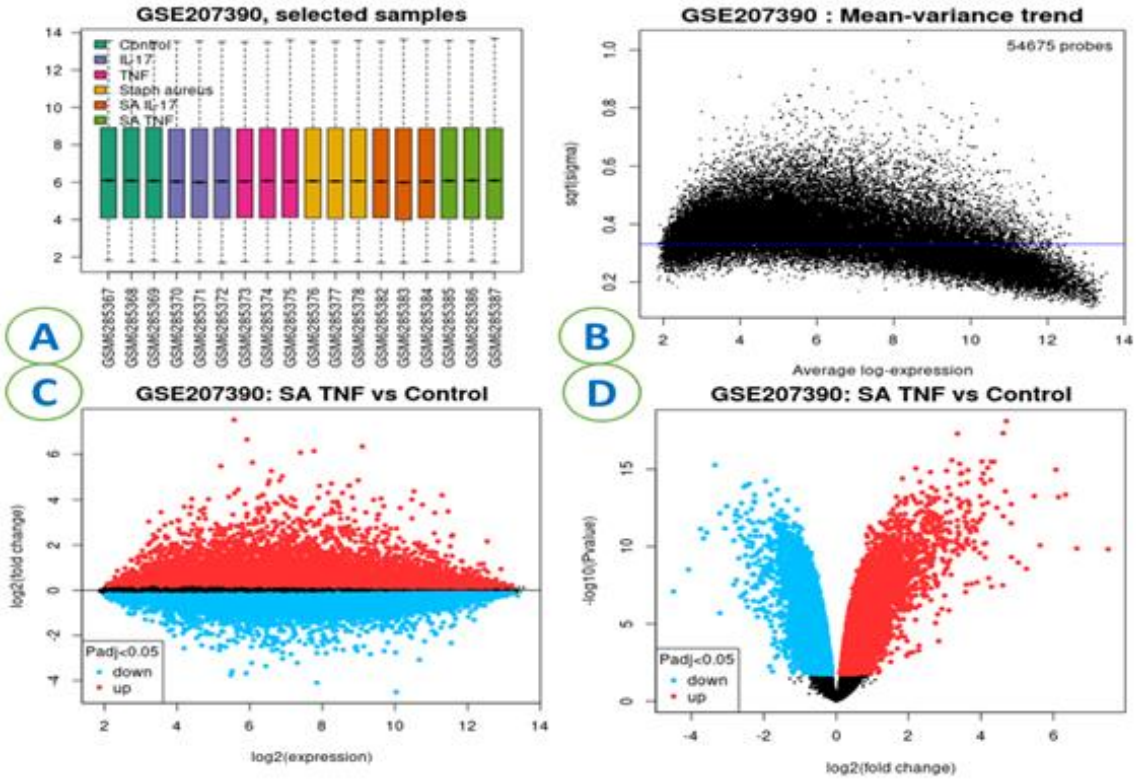

Figure 1: A: The box plot of the keratinocyte profiles displays the standardized and mutually comparable between the six research groups. B: The log expression mean-variance trend for 34284 probes. It facilitates comprehension of the relationship between the variation in gene expression and the mean expression levels. C: The gene predominance between the (*S. aureus* + TNF-α) and control groups with log2. D: The distribution, at log10 expression levels, of highly variable genes between the first and sixth groups.

Following log transformation and normalization, the distribution data of all genes according to the six groups under study are displayed in Figure 2A, which represents the next set of plots.

This graph probably sheds light on the variations and patterns of expression among the many groups that are being studied. The limma graph used to calculate the gene-to-gene correlation is displayed in Figure 2B. 3792 genes were found to be shared across the strains of the first and sixth groups and the second and fourth groups, suggesting a possible link or similarity in the expression of genes between the four sets.

Figure 2C depicts volcano plots to show the distribution of fold-change and statistical significance which indicate subsets of strong differentially regulated genes under SA+TNF-a relative to control. There was no indication of asymmetric distribution of log ratios in MA plots (Fig. 2B), which was expected to validate the strength of normalization. For every gene group, the level of gene expression drastically decreased. The distribution of all samples is shown in Figure 2D, where the dots are best aligned in a straight line. This alignment suggests that the results for the test's moderated t-statistic fit the distribution that was predicted theoretically at $p < 0.05$.

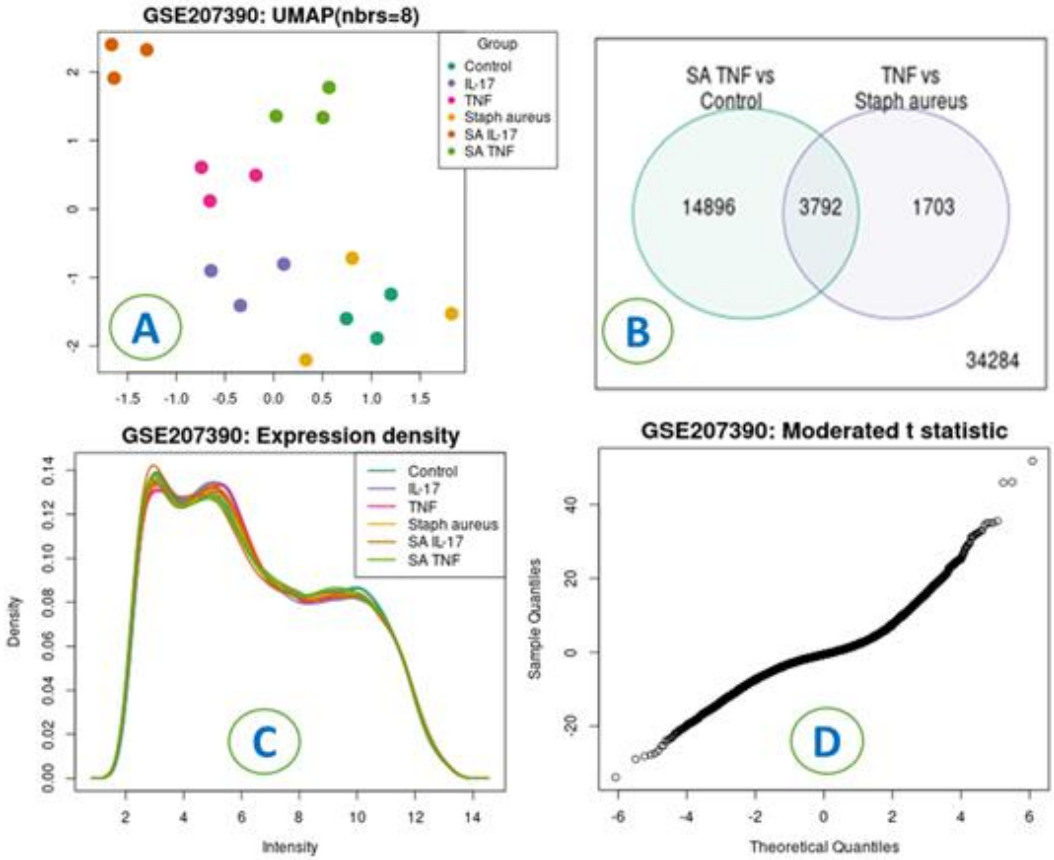

Figure 2: A: The UMAP figure illustrates the distribution of the six groupings. Each group is represented by a separate hue, which allows for a better comprehension of their distribution patterns in fewer dimensions. **B:** The limma graph shows 3792 related genes that are shared by the four groups divided into two sets. **C:** The expression density of each group is displayed on the curve, which offers information about the distribution of gene expression levels. **D:** The theoretical quantiles for each group derived from the t-statistical analysis, which aids in evaluating the degree of agreement between the observed data and the expected distribution.

Out of the 250 important genes, nine of them were explored in the further context (adjusted *p*-value ≤ 0.05), magnitude of fold-changes and biological relationship to psoriasis and melanoma pathways (*IL1F10*, *CCL27*, *IL19*, *PAPPA2*, *UHRF1*, *IFNAR2*, *GLS2*, *COL4A4*, *KRT78*). Table 1 depicts the frequency, *p*-value, and adj.*p*.value of chosen genes.

Table 1: Identification of selected ten genes, frequency, *p*-value, and adj. *p*-value

Gene symbol	Gene name	Frequency	*p*.value	adj.*p*.val
IL1F10	Interleukin 1 family member 10	1047	3.95E-26	1.01E-21
CCL27	C-C motif chemokine ligand 27	1014	5.57E-26	1.01E-21
IL19	Interleukin 19	575	2.46E-23	2.24E-19
PAPPA2	Pappalysin 2	389	1.61E-21	5.51E-18
UHRF1	Ubiquitin like with PHD and ring finger domains 1	308	1.97E-20	3.58E-17
IFNAR2	Interferon alpha and beta receptor subunit 2	173	8.76E-18	3.74E-15
GLS2	Glutaminase 2	155	2.93E-17	8.90E-15
COL4A4	Collagen type IV alpha 4 chain	136	1.10E-16	2.62E-14
KRT78	Keratin 78	279	1.23E-14	1.32E-11

Figure 3 presents the relative expression levels of each gene across the main tumor based on data from The Cancer Genome Atlas (TCGA) in order to illustrate the expression of a selected gene in skin cutaneous melanoma (SKCM). When compared to healthy individuals, it is evident that each of the chosen genes affects the likelihood of cancer developing in its primary stages. When expressed during the metastatic stage, six genes: *CCL27*, *IL19*, *PAPPA2*, *UHRF1*, *IFNAR2*, and *GLS2* showed high expression rates. The two with the greatest expressions were *IFNAR2* and *UHRF1*.

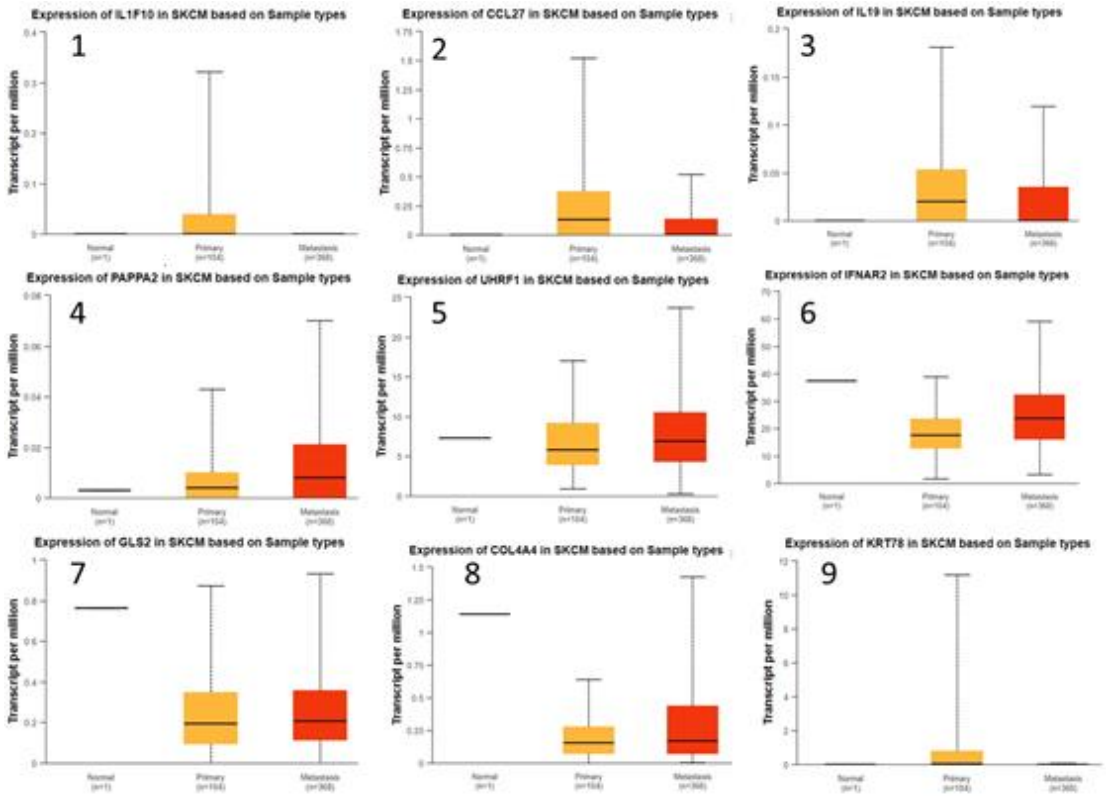

Figure 3: Box plots of selected expression genes in patients with skin cutaneous melanoma (SKCM) in primary and metastasis stages compared to the normal patients. 1-*IL 1F10*, 2- *CCL27*, 3- *IL19*, 4- *PAPPA2*, 5- *UHRF1*, 6- *IFNAR2*, 7- *GLS2*, 8- *COL4A4*, 9- *KRT78*.

The STRING tool was used to detect the protein-protein interaction. Analyses of P-P interactions showed that only two of the nine identified genes (*IL19* and *IFNAR2*) exhibited a binding relationship. The aluvial plot of ceRNA of chosen genes revealed distinct correlations based on their frequency (Fig. 4).

The current investigation found that there is no significant difference in gene expression in skin tissue before and after exposure to sunlight. A *COL4A4* gene expression increase was identified when the skin was exposed to sunlight as an exception to this rule. The statistical

analysis also revealed that there were no significant correlations in gene expression between males and females for all investigated genes.

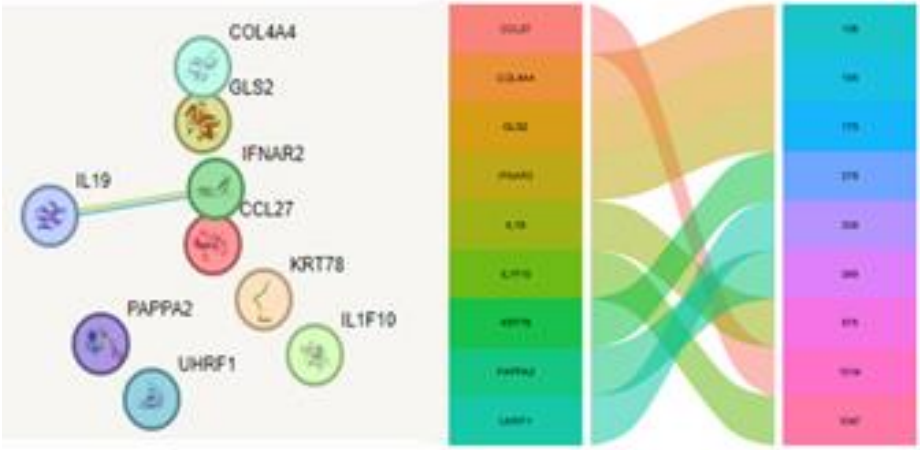

Figure 4: On the left: P-P interaction between 9 genes shows only IL 19 and IFNAR2 have been correlated using STRING tool. On the right: Different correlations between 9 gene using aluvial plot ceRNA tool.

We demonstrated that we agree with the existing literature that *S. aureus* abundance is high in psoriatic epidermis. Notably, *S. aureus*, acting in combination with IL-17A or TNF-a, caused synergistic transcriptional modifications in keratinocytes and thus it serves as a co-stimulator of psoriatic inflammation [10-12]. Several studies have found that *S. aureus* infection after brodalumab treatment aggravated psoriasis or that *S. aureus* infected Keratinocytes and skin express IL-17C [13, 14]. The considerable synergistic effect of heat-killed S. aureus with (IL-17A and TNF-α) on keratinocytes was investigated in this work.

Psoriasis is a chronic illness induced by Th1 cells, in which PGN from *S. aureus* induces the development of human cathepsin LL37 and VEGF in keratinocytes, as well as the expression of IL-13. *S. aureus* stimulates IFN-expression in keratinocytes via IL-12-mediated differentiation. *S. aureus* infection can also be mediated by necrotizing keratinocyte apoptosis mediated by RIPK1/RIPK3/MLKL, and RIPK1 causes keratinocyte death via TNF [15].

In line with the previous reports, there was additive and synergistic effects of IL-17A and TNF-alpha. Among the genes that were specifically enhanced by SA+TNF-alpha in our dataset were CCL27, UHRF1, and IFNAR2, which help to support the hypothesis that bacterial stimuli enhance cytokine-induced transcriptional programs. According to a recent study, 60% of the top 20 regulated genes displayed additive or synergistic responses to IL-17 and TNF, showing the relevance of these cytokines in developing the elevated molecular profile of Psoriasis. 20% of the entire psoriatic transcriptome relates to genetic alterations caused by particular cytokines in cultured keratinocytes. Furthermore, this gene cluster most likely represents the direct influence of inflammatory cytokines on disease phenotype [16, 17].

Gene-set enrichment analysis results for gene expression profiling showed similarities between the current study and previous research. They discovered several upregulated genes in signaling pathways, including the TNF, IL-17, and IFN-λ signaling pathways, which are assumed to play a significant role in the pathogenesis of psoriasis. The TNF signaling pathways OASL, CXCL1, STAT-1, AKR1B10, IL1F9, and CXCL9, as well as the IL-17 signaling system's *CCL20* and *CXCL8* are among the specific upregulated genes [18, 19].

Psoriatic skin lesions have been shown to upregulate the expression of the IL-17, IL-22, and IL-23 genes, which has prompted scientists to investigate the potential involvement of T-helper cells in psoriasis. The lists of keratinocyte genes activated by cytokines showed little overlap. This demonstrates the many functions that T-helper cells and these cytokines play in the pathophysiology of psoriasis. Through the downregulation of genes that control terminal differentiation, Th17, IL-22, causes abnormal proliferation of keratinocytes. In psoriasis, IL-17

and IL-22 can both increase the expression of keratinocyte antimicrobial defenses [20-21]. These research back up our findings.

According to our data, the (*S. aureus* + IL-17A) group's gene expression clearly had an impact since it was higher than that of the other groups for *IL1F10*, *CCL27*, *IL19*, and *KRT78*. In comparison to the other groups, the (*S. aureus* + TNF-α) group exhibits higher expression levels for *CCL27*, *UHRF1*, *IFNAR2*, and *KRT78*. Conversely, the group (*S. aureus* + TNF-α) showed lower gene expression for *PAPPA2* and *GLS2* when compared to the control group and the remaining groups. We can clearly see that the presence of cytokine groups with *S. aureus* in comparison to the other groups influences the difference in expression. Therefore, there is a substantial variation in the expression of genes when cytokines are present in conjunction with bacteria. This suggests that either bacterial genes directly influence cytokine genes, or that bacterial genes may clearly regulate cytokine genes, or vice versa.

According to a research, *S. aureus* acts on TLRs and TNFR1, interacts with NOD2, which activates NF-kB, and drives gene transcription, which results in the generation of Pro-IL-1β. These two signaling pathways are what S. aureus uses to activate inflammasomes in Alzheimer's disease. Additionally, it triggers purine receptor activation, which in turn triggers proinflammatory IL-1β and IL-18 and induces laryngitis when combined with inflammatory chemicals [22, 23].

A study was done on bacterial inoculation of persistent IL2 release during *S. aureus* arthritis, which resulted in a decrease in the systemic inflammatory response. The researchers expected that better immune control would minimize severe and permanent joint injury. When rAAV-IL2-treated mice were compared to the control group, both synovitis and bone erosion were reduced [24].

According to the results of the present study, the illness alters the expression of a certain set of genes. For instance, certain genes were chosen because they altered their expression in response to psoriasis and also had an impact on individuals with skin-colored cutaneous melanoma (SKCM). These genes that expressed similarly were *GLS2*, *PAPPA2*, *UHRF1*, *IFNAR2*, *IL19*, and *CCL27*. Therefore, based on the degree of expression, it may be concluded that these genes are illness markers. However, there is no difference in the expression of any gene between samples obtained from those exposed to sunshine and those who were not. *COL4A4* is an exception to this rule since it showed a high expression and a larger proportion of individuals in the group exposed to sunshine.

It is possible to argue that the significance of gene expression stems from its ability to quantify the extent of the illness and the proportion of the population at risk. It is important to acknowledge the significant influence of environmental, physiological, and genetic variables on the type of gene expression.

Based on the fact that *IFNAR2* and *UHRF1* were significantly expressed in both psoriatic keratinocytes and metastatic SKCM, there is a molecular overlap between chronic inflammation and oncogenic progression. This points to the possibility of a psoriasis-associated gene signature to be a melanoma risk biomarker. The utilization of large patient cohorts to construct disease profiles from various matrices will continue to contribute significantly to our understanding of disease causation and associated comorbidities.

The main findings that are highlighted in this piece of work are that a reproducible GEO-based pipeline has been discovered, synergistic gene signatures under the SA+IL-17A and SA+TNF-alpha have been identified and a new connection between psoriasis-related keratinocyte genes and melanoma progression has been established. These results enlarge the molecular psoriasis landscape and have a translational implication of comorbidities.

The weaknesses of the study are the use of microarray data that might be insensitive to the control of isoforms, and the relative small size of the sample. Causality needs to be proven by functional validation in independent cohorts and in vivo models. Moreover, the findings can have an impact on generalizability due to the presence of batch effects and population variability.

Conclusions

The existence of cytokines in the presence of *S. aureus* in keratinocytes changes gene expression in comparison to the six research groups. IL-17A and TNF- gene expression in the presence of S. Certain genes in *S. aureus* increase. In other words, bacterial genes can either directly or indirectly affect cytokine genes. One may argue that the significance of gene expression arises from its capacity to predict illness severity and the fraction of persons who are vulnerable to infection. This study added to our understanding of the molecular alterations that occur in the genes of people with psoriasis and its related disorders.

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Conflict of Interest: The authors declare no conflict of interest in this work.

Authors' Contribution: The study was conceived and designed by DAA, the bioinformatics analysis and writing of the manuscript were done by him. ZMS helped in data interpretation, literature review and editing of the manuscript. The final version of the manuscript was reviewed and approved by all the authors.

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