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 ORIGINAL ARTICLE

A Clinical Evaluation of Scalp Barrier Function, Ceramide Levels, and Microbiome in Diverse Dandruff Patients

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ABSTRACT

Dandruff is a common chronic scalp condition that affects approximately half the population irrespective of their origin. Dandruff scalps are characterized by flaking skin, pruritus, and minimal visible scalp inflammation. At the biological level, dandruff scalp presents a disruption of the barrier function supported by lower levels of ceramides in the stratum corneum and typically accompanied by altered microbiome diversity, including a higher abundance of *Malassezia* yeasts and exacerbated sebum peroxidation. This study evaluated the relationship between skin barrier integrity in association with epidermal ceramide profile, microbiome imbalance, and inflammatory markers in pathophysiology of dandruff in an ethnically diverse panel. Our results confirm a significant increase in TEWL and decrease in hydration along with an increase in erythema, dryness, flakiness, and itchiness in patients with dandruff vs normal scalps; and an elevation of IL1RA:IL1α ratio dependent on the severity of the dandruff, supporting the inflammatory association with dandruff. For the first time, a study shows that dandruff scalps have a significantly higher amount of short-chain ceramides and a significantly lower proportion of long-chain ceramides consistent with lower conformational ordering and, thus explaining a higher permeability of the skin contributing to barrier dysfunction. In addition, reduced phytosphingosine and dihydrosphingosine based ceramides (NP, AP, NDS) were also observed, supporting a weakened scalp barrier. In addition to an expected increase in *Malassezia*, especially *Malassezia restricta*, in dandruff scalp, an increase in *Staphylococcus aureus* and decrease in *Malassezia globosa* was also observed as compared to healthy scalp in the population analyzed.

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INTRODUCTION

Seborrheic dermatitis (SD) and dandruff are of a continuous spectrum of the same disease that affects the seborrheic areas of the body, with dandruff being milder and specifically localized to the scalp. It is characterized by a flaky, pruritic scalp and affects up to half the world's population post-puberty.[1,2] Compared with dandruff, SD can affect the scalp as well as other seborrheic areas and is more severe when pruritus, flaking, scaling, and skin inflammation are involved. Environmental and host factors may alter the sebaceous gland activity, sebum composition, epidermal barrier function, and scalp microbiome balance. Various environmental (eg, pollution; stress) and intrinsic (eg, puberty; individual susceptibility) factors may affect the sebaceous gland activity and thus the sebum composition, scalp surface microbiome, and skin barrier function. Interactions between these factors, all contribute to the pathogenesis of SD and dandruff.[3]

Although the exact pathophysiology of dandruff is still not completely decoded, current theories highlight the role of the microbiome on the skin surface in the pathogenesis. Several scalp microbiome studies from different populations have revealed the association of dandruff with bacterial and fungal dysbiosis.[4,5] Another study comparing the major bacterial-fungal populations colonizing dandruff scalps in China and France suggests that targeting one particular *Malassezia* species by antifungals instead of using broad-spectrum antifungals and rebalancing the dandruff scalp microbiota could be a common approach to improve dandruff condition.[6,7] Overall scalp microbiome composition significantly differed between normal and dandruff groups linked with hyperproliferation of lipophilic yeasts of the genus *Malassezia*[8] and *Staphylococcus spp*. Additional microbial markers such as *Aspergillus* and *Pseudomonas* have also been proposed.[5,9]

The role of *Malassezia* in the dandruff scalp barrier dysfunction has been related to various mechanisms. *Malassezia*, especially *M restricta*, is able to disrupt keratinocyte layers in vitro, suggesting an active role in accelerated flake formation.[10] *Malassezia* spp is able to metabolize and oxidize sebum-derived lipids such as triglycerides, squalene, and fatty acids into inflammatory compounds, thereby affecting the skin barrier.[11] *Malassezia* is also able to produce indole derivatives such as malassezin, which activity against aryl hydrocarbon receptors may impact skin inflammation.[12,13]

Despite the different studies, the relationship between molecular markers of the epidermal barrier, specifically the ceramide composition, and the scalp microbiome composition of healthy and dandruff scalp remains to be explored.

A healthy stratum corneum (SC) forms a protective barrier to prevent water loss and protect against external aggressors, pathogens, and allergens. Severe or chronic barrier disruption can impair proper hydration, leading to atypical epidermal proliferation, keratinocyte differentiation, and SC maturation, which may underlie the characteristic flaking and scaling of the scalp experienced by dandruff sufferers.[1]

Ceramides, the principal lipids in the SC, are found in the lipid lamellae that surrounds corneocytes and are a crucial component of the skin barrier function. Alterations in the SC ceramide class composition and reductions of the total amount of ceramides have been associated with several skin conditions, including atopic dermatitis and SD.[14-16] Biochemical analysis of dandruff-affected scalp revealed that dandruff was associated with a dramatic decrease in free lipid levels, with significant decreases in ceramides, fatty acids, and cholesterol.[17] The depleted and disorganized structural lipids of the dandruff SC are consistent with the weakened barrier indicated by elevated transepidermal water loss (TEWL).[1]

It is widely accepted that scalp dysbiosis triggers distress in keratinocytes, leading to barrier breakdown. Especially commensal bacteria that are commonly depleted in skin dysbiosis have been reported to play important roles in skin ceramides content, such as Cutibacterium acnes (*C acnes*)[18] that stimulate intercellular lipids synthesis and Staphylococcus epidermidis (*S epidermidis*)[19] that facilitate ceramides release through sphingomyelinase. In turn, the barrier leakage exacerbates the inflammatory response via inflammatory mediators. Traditionally, inflammatory cytokines are measured from biopsies or blood for the diagnosis of SD.[20-22] Very few studies have investigated the presence of localized biomarkers on the skin for dandruff.[21]

We performed the first study to assess inflammatory mediators on the SC in cohorts with different Investigator's Global Assessment (IGA) scores.

This study evaluated the cyclical and interdependent relationship between the microbiome, inflammation, epidermal lipids, and skin barrier function in the pathophysiology of dandruff.

We undertook a detailed analysis of the lipid and microbiome profile of normal vs dandruff scalp in a diverse population to understand the relationship between ceramides levels, microbiome, and barrier function in dandruff-affected and normal scalps.

METHODS

Clinical Study Design

The study was carried out at a clinical facility (SGS Stephens Inc.) in healthy (no history of dandruff within the last 3 years) and dandruff-affected male and female subjects of African American/Black, Caucasian/White, Asian, and Hispanic racial and ethnic groups aged 18 years and older. All subjects used a non-antidandruff shampoo for 14 days before the clinical visit. All clinical assessments were carried out at one-time point on the scalp after the washout.

Dandruff eligibility was assessed with a 5-point IGA (0: no flakes, to 4: severe, large, pronounced flakes). Subjects with mild to moderate scores of adherent flakes (small flakes ~1 mm to moderate flakes ~2 mm in size) were considered eligible. An expert grader assessed for signs of erythema, dryness, and flakiness (0: none, to 4: severe), and subjects reported the degree of itching and tightness for the global scalp (0: none, to 4: severe).

Instrumental Analysis

Assessments were performed along hair partings in designated zones representative of the global IGA on the scalp. The hair was parted to expose the scalp as much as possible for instrumental assessments to be collected on scalp skin.

Hydration (Corneometer)

The DermaLab (Cortex Technology) was used to measure skin hydration. A hand-held probe placed on the scalp measured skin conductance at a single frequency equal to 300 kHz, which can be related to the water content of the SC on an arbitrary scale. Three independent readings were taken side by side, and the result was considered valid if the difference between any 2 of the 3 readings was <5 au. The mean of the 3 readings was then calculated for each subject and used for data analysis.

TEWL

TEWL was assessed using Tewameter®. Tewameter TM Nano (Courage + Khazaka Electronic GmbH) to measure the passive transfer of water through the SC. The measurement of water evaporation is based on the diffusion principle in an open chamber, and the density gradient is measured indirectly by 2 pairs of sensors located inside the hollow cylindrical probe. Data are analyzed by a microprocessor and reported in g/m2/h. A decrease in TEWL values reflects an improvement in the barrier properties of the skin. The mean of the 3 readings was then calculated for each subject and used for data analysis.

Ceramide and Cytokine Collection

D100 D-Squame Standard Sampling discs (Clinical & Derm LLC) were used as a noninvasive technique to collect skin cells from the scalp for lipid and cytokine analysis. Six (6) sequential tapes were collected from two (2) adjacent but distinct zones, both in a lesional area of the scalp. One zone was used for ceramide analysis, and the second adjacent zone was used for cytokines analysis. Following application of each strip, a blunt instrument was used to apply rolling pressure along the scalp at the hair parting for 30 seconds to promote contact with the scalp.

Tape strips collected for ceramide analysis were stored on D120 D-Squame Standard Storage Cards –80°C and were analyzed for different types of ceramides following Metabolon, Inc. methods of mass spectrometry and comparative profiling as described above.[23] Wilcoxon rank-sum tests were used to identify biochemicals that differed significantly between experimental groups on quantitative data normalized to the extracted disks as well as quantitative data normalized to cholesterol. These are non-parametric tests that compare the distributions of the ranks between the 2 groups being compared. Kruskal-Wallis tests were also employed for multi-group comparisons. A summary of the numbers of biochemicals that achieved statistical significance ($P \leq 0.05$), as well as those approaching significance ($0.05 < P < 0.10$), is provided.

Tape strips for cytokine analysis were stored in 2 mL microcentrifuge tubes at –80°C.

Microbiome Analysis

Microbiome samples were collected from the subjects' scalp using technique described previously.[5] Samples were divided into 2 sets and processed either for quantitative polymerase chain reaction (qPCR) or whole genome sequencing (WGS) at CosmosID Inc. using established protocols. For qPCR, total *Malassezia* and *M restrica* DNA was quantified with CFX 96 Real-Time System (BioRad) using appropriate primer sets.

For WGS, DNA was extracted using Zymo MicroPrep Extraction kit following manufacturer's protocol. DNA libraries were prepared using the Nextera XT DNA Library Preparation Kit (Illumina) and TDT Unique Dual Index with total DNA input of and sequenced on an Illumina NovaSeq platform 2x150b at CosmosID.

Bioinformatics Analysis Methods

Analyses were performed by CosmosID using a proprietary algorithm under the CosmosID-HUB.

Shannon alpha diversity metrics were calculated. Wilcoxon Rank-Sum tests were performed between groups.

Skin Biomarker Analysis

Luminex Assays for the Evaluation of Scalp Surface Biomarkers

Scalp surface biomarker analysis was performed on a subgroup of both healthy and dandruff populations. The protocol for the extraction of the protein samples from the D-Squame discs was described elsewhere.[24] Briefly, the samples were extracted with phosphate-buffered saline (PBS) containing an additional 0.25M NaCl and protease

inhibitor for 30 min with sonication on ice. The extracts were centrifuged for 5 min at 2100 xg to remove debris from the skin. Aliquots of the samples were kept and quantified with a BCA Protein Assay Kit. The samples were supplemented with 2% BSA and frozen in –80°C until cytokine Luminex analysis (ProcartaPlex Human Inflammation Panel 20plex and interleukin 1 receptor antagonist [IL1RA]). Extractions from 6 discs collected from the same location were pooled together.

An estimate of the false discovery rate (q-value) is calculated to consider the multiple comparisons that normally occur in metabolomic-based studies. For example, when analyzing 200 compounds, we would expect to see about 10 compounds meeting the $P \leq 0.05$ cut-off by random chance. The q-value describes the false discovery rate; a q-value below 0.10 is an indication of high confidence in a result. While a higher q-value indicates diminished confidence, it does not necessarily rule out the significance of a result. Other lines of evidence may be taken into consideration when determining whether a result merits further scrutiny. Such evidence may include a) significance in another dimension of the study, b) inclusion in a common pathway with a highly significant compound, or c) residing in a similar functional biochemical family with other significant compounds.

GraphPad Prism was used for statistical analyses. Paired t-test was used to analyze the cytokine secretion between the donor groups. One-way ANOVA was used to compute the level of significance between donors with different IGA scores.

RESULTS

Clinical Parameters

Demographics

A total of 212 subjects were recruited for the study. Gender, ethnicity, race, and Fitzpatrick skin type distribution were roughly equal for subjects in the 2 groups. A small panel of normal and dandruff subjects also participated in a scalp surface biomarker subgroup. The details of the ethnic distribution of all subjects are given in Table 1 below.

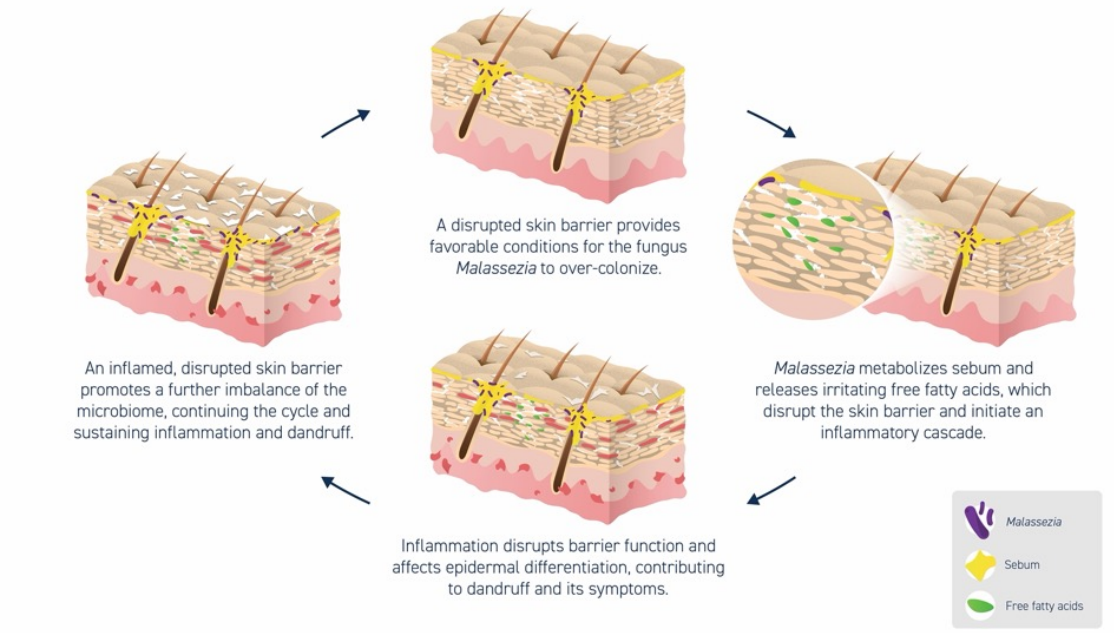
The average IGA for all the subjects in Group 2 was 2.29, indicating a mild dandruff population.

TABLE 1.

Demographic Information for the Dandruff, Healthy, and Scalp Surface Biomarker Subgroup

	Healthy Scalp		Dandruff Scalp	
	Total Panel N=106	Biomarker Subgroup N=13	Total Panel N=107	Biomarker Subgroup N=13
Age	39±9	42±10	40±10	42±7
Gender				
Female	68	11	75	9
Male	38	2	31	4
Fitzpatrick Type				
I - III	67	10	76	9
IV - VI	39	3	30	4
Race/ Ethnicity				
Asian	22	4	22	3
Black	27	3	23	3
Hispanic	29	3	28	4
White	28	3	32	3
IGA				
2	NA	NA	62	4
2.5	NA	NA	27	7
3	NA	NA	17	2

FIGURE 1. The cycle of dandruff.



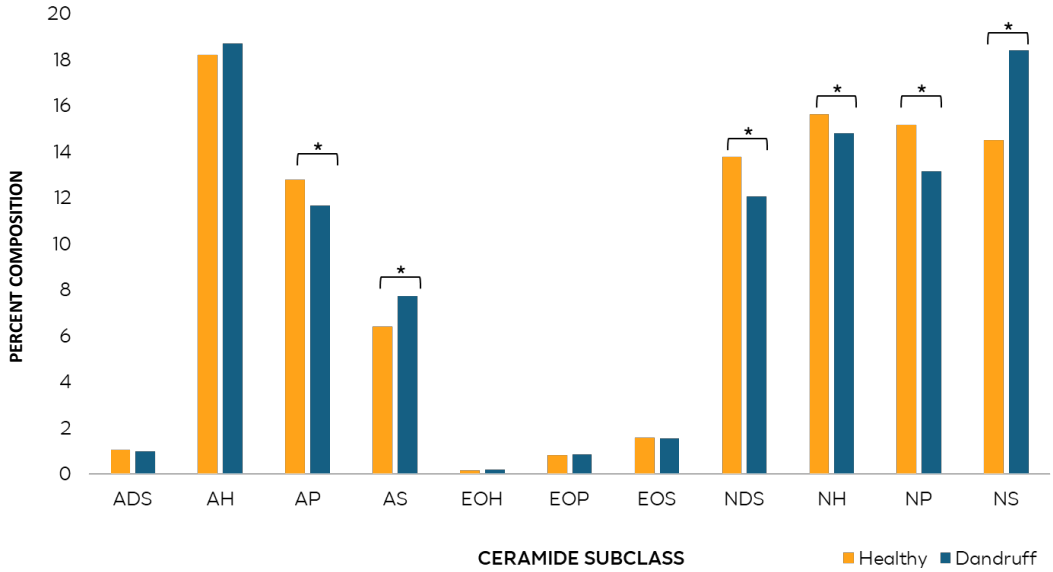
Clinical Scores

All the clinical parameters evaluated using 5-point IGA scale demonstrated significant elevation in dandruff group compared to the normal subjects. Significant increases in the erythema (0.02 vs 0.18), dryness (0.08 vs 0.7), flakiness (0 vs 1.2), and itchiness (0.2 vs 0.65) were observed for dandruff scalps vs normal scalps at $P<.05$.

TEWL and Hydration

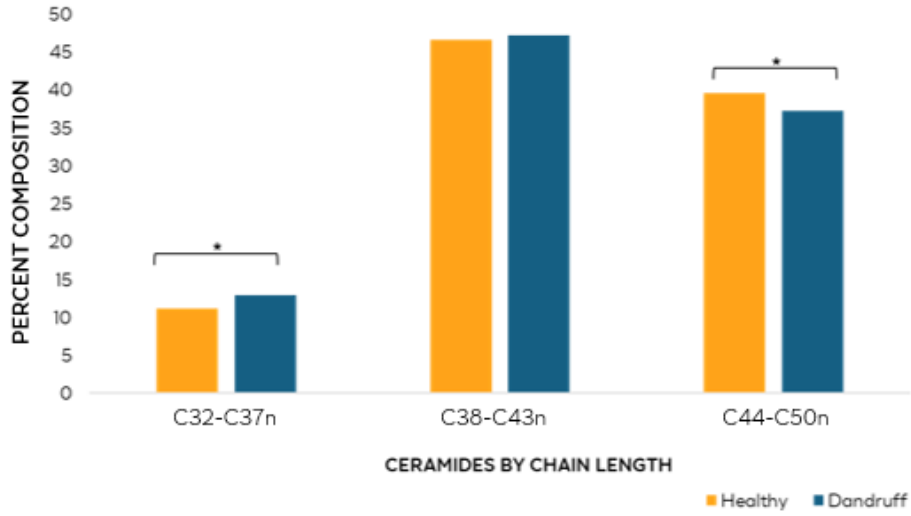
There was a significant increase in TEWL for dandruff (24 g/m^{2}h) vs normal (21 g/m^{2}h) scalps ($P<0.05$). A significant decrease in hydration (corneometer) for dandruff vs normal scalps (28 au vs 44 au, respectively; $P<0.05$) was observed. These results are consistent with previous literature that dandruff scalp has a significant damage in skin barrier function as well as increased dryness (decreased hydration).

FIGURE 2. Percent composition of ceramide subclasses in dandruff affected vs healthy scalps.



*$P\leq0.05$.

FIGURE 3. Percent composition of longer chain (C44-C50), medium chain (C38-43) and shorter chain (C32-37) ceramides in healthy vs dandruff scalp.



*$P \leq 0.05$.

Ceramide Levels

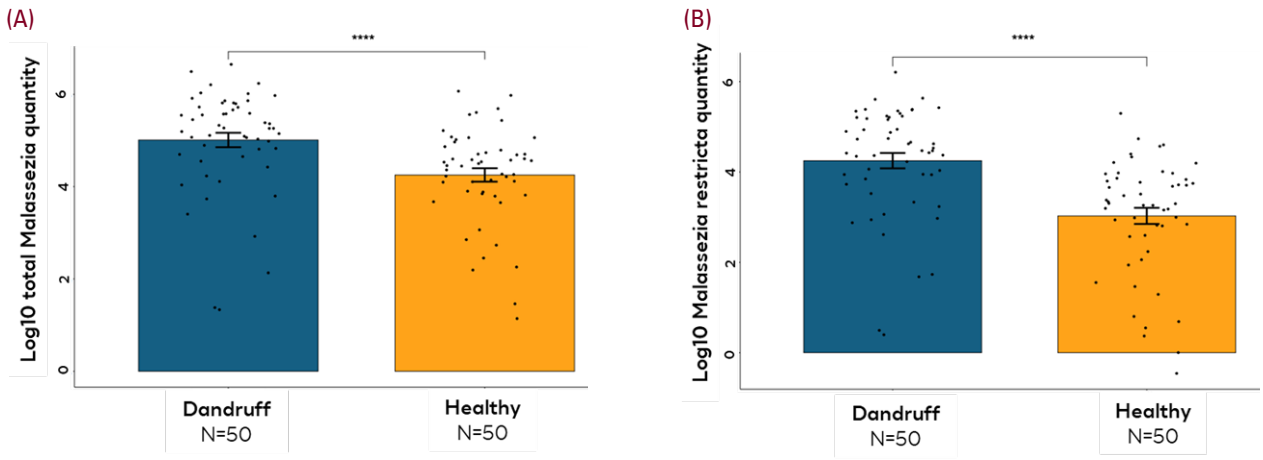
There was a significant change in specific subclass of ceramides in dandruff scalp as compared to normal scalp (Figure 2).

Ceramides AP, NDS, NH, and NP decreased in dandruff scalps vs normal scalps. In addition, there was a significant increase in ceramide NS and AS in dandruff scalps.[25,26] The significance of these changes in individual ceramides are not clear at present. However, published literature suggests that an increase in Ceramide NS is a hallmark of inflammatory conditions, such as dandruff.[16]

An increase in free fatty acids (FFAs) was also observed in dandruff scalps vs normal scalps. Previous work suggests that increased FFAs contribute to skin barrier dysfunction and upregulated ceramidase activity. However, additional research needs to be conducted to understand this further in the context of scalp.[27]

Dandruff scalps have a significantly higher proportions of short-chain ceramides (C32-C37; $P<0.05$) and a significantly lower proportion of long-chain ceramides (C44-C50; $P<0.05$) (Figure 3). This data is consistent with the finding that lower chain length ceramides provide a lower conformational ordering of the intercellular lipids and, thus, a higher permeability of the skin contributing to barrier dysfunction.[26,28]

FIGURE 4. Quantification of (A) Malassezia and (B) M restricta in dandruff and healthy scalp by qPCR.



****$P \leq 0.0001$

Microbiome Profiles

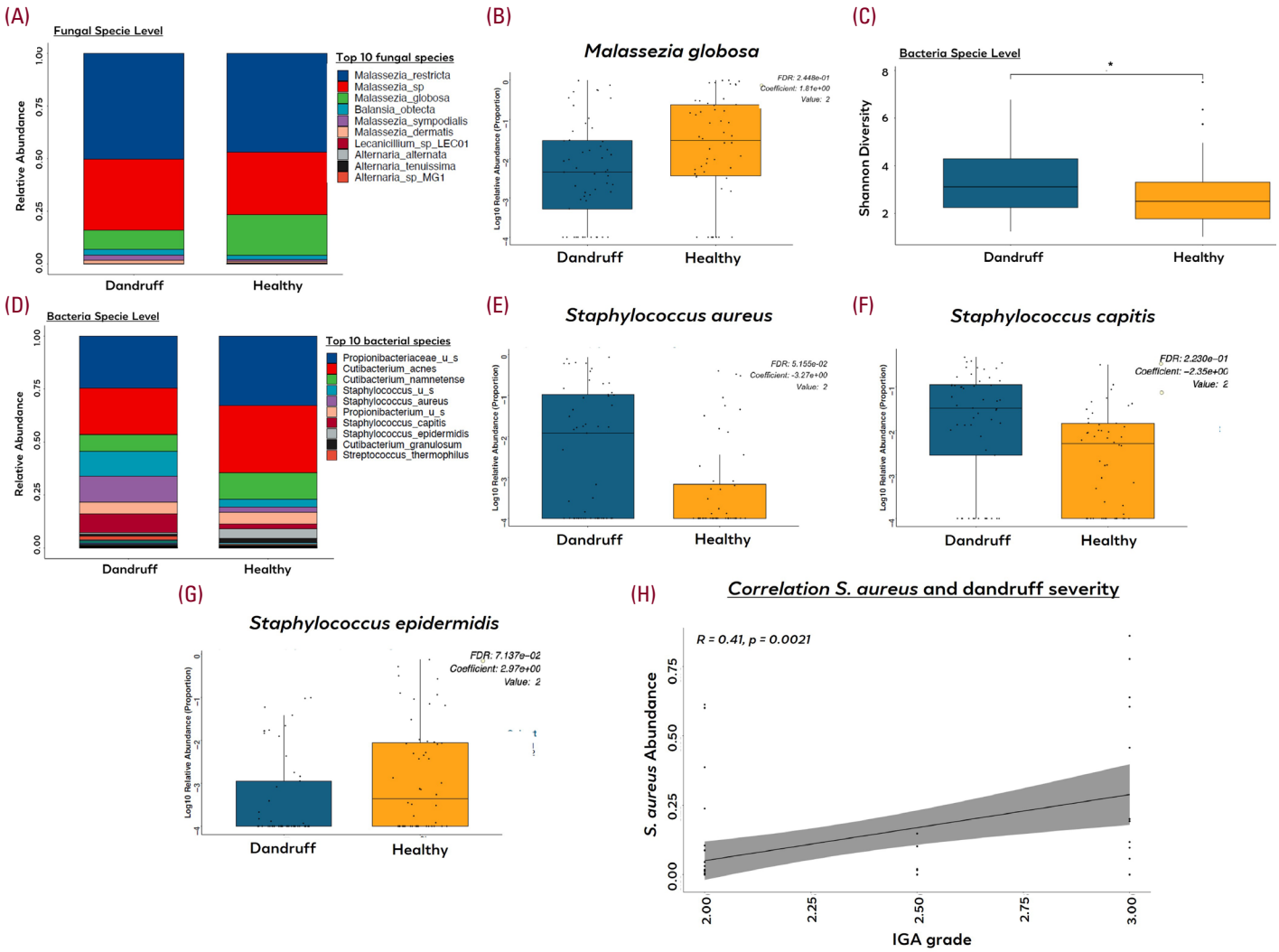
Quantitative analysis confirmed that *Malassezia* and *M restricta* loads were higher in dandruff scalp (Figure 4).

Consistent with the qPCR results and the published literature, overall, scalp microbiome was dominated by *Malassezia spp* and the abundance of *M globosa* was significantly higher in healthy scalp compared to dandruff scalp (Figure 5A and 5B). By contrast, *M restricta* appears to be increased in dandruff scalp.

In addition to fungal profile, this study also evaluated bacterial diversity and it was found to be significantly higher in dandruff vs healthy group (Figure 5C), confirming published literature data.[29]

Common healthy scalp microbiome profile was confirmed with dominance of *Cutibacterium* (formerly named *Propionibacterium*) and *Staphylococcus spp*. Common trend was observed in dandruff group with lower abundance of *Cutibacterium spp* and increase of *Staphylococcus spp.* Our observation of lower abundance of *S epidermidis*, and

FIGURE 5. Distinct bacterial and fungal composition in dandruff and healthy scalp. (A) Fungal species composition, in dandruff and healthy scalp, (B) Key fungal species associated with healthy scalp, (C) Shannon diversity index for bacteria species in dandruff and healthy scalp, (D) Bacterial species composition in dandruff and healthy scalp, (E,F) Key bacterial species significantly more abundant in dandruff scalp. (H) Bacterial species significantly correlated with dandruff severity (IGA grade).



*$P \leq 0.05$

higher abundance of *S aureus* and *S capitis* altogether in dandruff scalp is novel and extends the previous findings[7] into a global and diverse population. Importantly, we also demonstrated significant correlation between *S aureus* abundance and dandruff as well as dandruff severity (IGA).

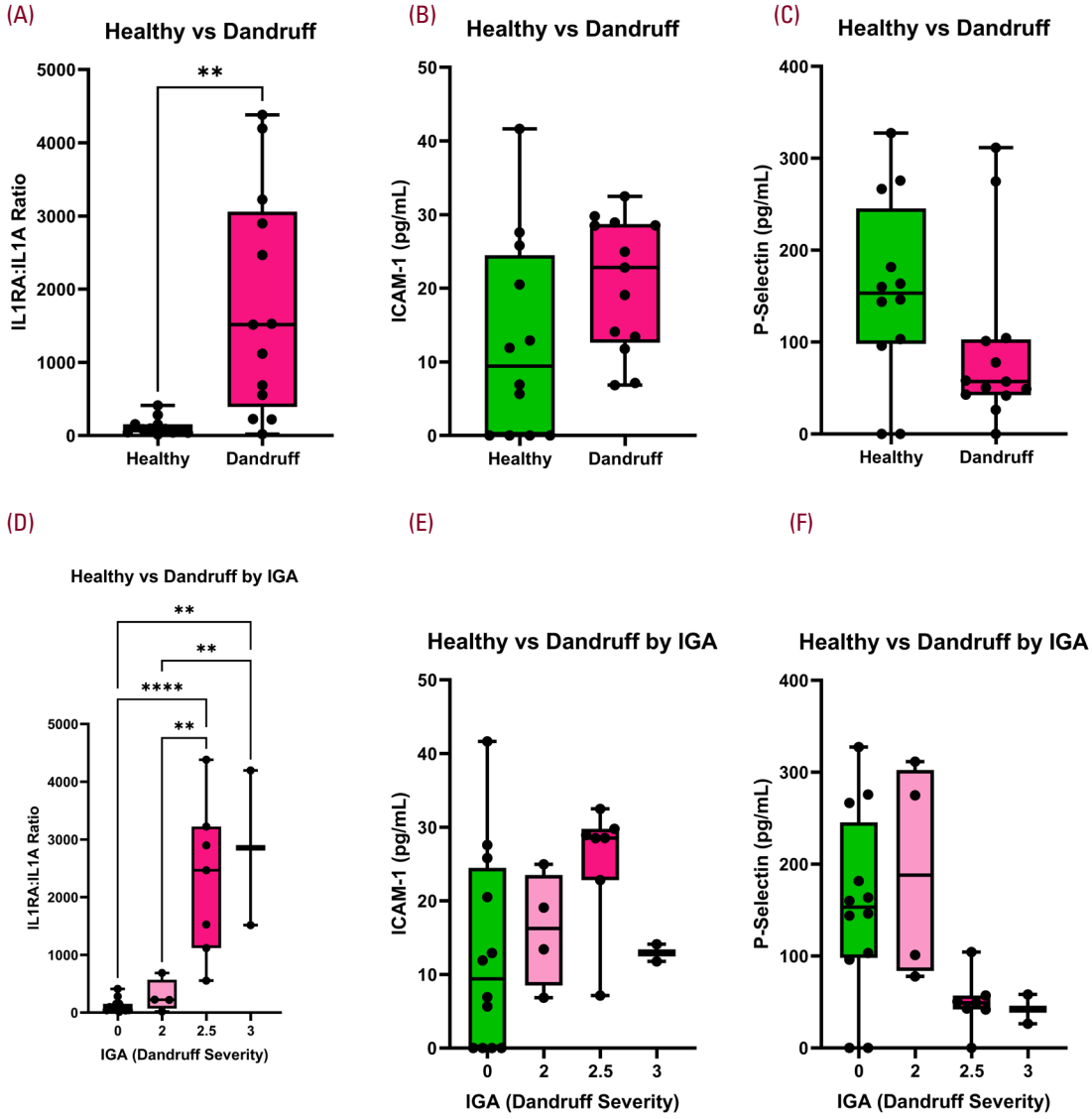
Dandruff is a prevalent chronic inflammatory skin condition of the scalp that has been associated with *Malassezia* yeasts. However, the microbiome role has not been fully elucidated yet, and the etiology of the disorder remains poorly understood. Although clinical symptoms of dandruff manifest locally, microbial dysbiosis beyond clinically affected skin sites such as on forehead of dandruff subjects[29] suggests that subjects undergo systemic alterations, which could be considered for redefining therapeutic approaches.

Cytokines and Proteomics

Dandruff scalp displayed the hallmark elevation of IL1RA:IL1a protein ratio dependent on the severity.

The development of dandruff is characterized by an inflammatory signature.[23] The overproduction of sebum on the scalp contribute to the over-colonization of *Malassezia*,

FIGURE 6. Cytokine secreted in subjects with and without dandruff and at different severities. There was an increased IL1RA:IL1A ratio in dandruff subjects and with increasing severity (A, B). Similarly, dandruff affected subjects had higher level of ICAM-1 (C, D) while a decreasing level of P-selectin (E, F).



**P<0.001, ****P<0.0001. Healthy (IGA 0) n=12; IGA 2 n=4, IGA 2.5 n=7, IGA 3 n=2.

which produce inflammatory metabolites. It has been previously shown that dandruff-bearing scalp is associated with higher inflammatory cytokine production, specifically, a higher IL1RA: IL1a ratio.[23] In our study, we have found that the elevation of IL1RA:IL1a ratio is also dependent on the severity of the dandruff (Figures 6A and 6B). We saw a significantly higher ratio in subjects with a dandruff grading (IGA grading) of 2.5 (n=7) and 3 (n=2), compared with healthy control (n=12, one sample not analyzed due to low quality) as well as IGA 2 (n=4).

As seen from the figure, dandruff scalp displayed significantly higher production of cell adhesion molecules. In addition, the study also demonstrated an increase of intercellular adhesion molecule 1 (ICAM-1) and decrease of P-selectin in dandruff scalp ($.05 < P<0.01$). Both adhesion molecules are typically increased for the recruitment of inflammatory cells to local areas. Comparing between IGA groups, ICAM-1 displayed increasing trend as the severity increased except at IGA 3P-selectin level had an inverse relationship with the severity of dandruff.

DISCUSSION

The role of the microbiome and barrier function in dandruff involves several key factors. The scalp's barrier function, much like the skin on other parts of the body, is crucial for maintaining hydration and protecting against environmental stressors. When the barrier function is compromised, it can lead to increased susceptibility to pro-inflammatory components such as dust, allergens, and transient microbes. In the case of inflammatory skin conditions, a compromised barrier is associated with microbial imbalance, which potentially contribute to the skin or scalp disorder. The scalp has its own microbial community, including bacteria and fungi.[5] Dandruff has been associated with an imbalance in this microbiome, particularly an overgrowth of certain fungi, notably *Malassezia spp*. The presence of *Malassezia spp*. can trigger immune responses either directly through TLR2 receptors[10] or indirectly by its metabolites,[12] leading to inflammation, barrier disruption, and subsequent flaking of the scalp skin. A healthy scalp barrier, maintained by ceramides, helps to prevent water loss and maintain hydration. If the barrier is compromised due to a lack of ceramides or imbalance of ceramide subclasses, the scalp can become dry and more prone to irritation due to the penetration of external stressors and opportunist pathogen invasion, potentially exacerbating dandruff.

The unique lipid composition of the epidermal barrier is essential for its function, and recent studies have elucidated our understanding of the role of skin microbes in epidermal ceramide production and regulation of skin functions.[30] In this study, we have generated data to suggest the altered ceramide composition in the dandruff scalp may be associated with the altered microbes in dandruff scalp. It has been reported that ceramides NP and AP are decreased in other inflammatory skin conditions. In addition, EOP and AP are decreased in atopic dermatitis, and NP and AP are decreased in xerosis. Dandruff scalps have a significantly higher amount of short-chain ceramides and a significantly lower proportion of long-chain ceramides. These data are consistent with the finding that lower chain length ceramides provide a lower conformational ordering and, thus, a higher permeability of the skin contributing to barrier dysfunction.[16,28] In addition, our results also indicate a reduced phytosphingosine-based ceramides AP and NP, associated with a weakened barrier. In addition to their contribution in barrier function, phytosphingosine is reported to inhibit the growth of opportunistic fungal pathogens such as *Candida albicans*, suggesting a weakened antimicrobial barrier too.[31] A higher number of hydroxyl groups in the sphingoid base of ceramides may provide for the orthorhombic configuration by hydrogen bonds, which is important for optimal skin barrier function and water retention. Whether the changes in ceramide composition are a result of barrier disruption in dandruff or a cause of barrier disruption is not clear.

Reflective of this alteration to ceramide composition, subjects with dandruff had a significant increase in TEWL and a significant decrease in hydration vs normal scalps. It has been previously shown that dandruff-bearing scalp is associated with higher inflammatory cytokine production, specifically, a higher IL1RA:IL1a ratio.[24] In our study, we have also found elevated levels of IL1RA:IL1a ratio which is also dependent on the severity of the dandruff. These results are consistent with published literature that dandruff scalp has a significant damage in skin barrier function as well as increased inflammation.[1,32]

The changes in cell adhesion molecules have not been reported previously in dandruff. However, given the inflammatory nature of the condition, it is not surprising to see changes in expression of these cell adhesion molecules. This study also demonstrates that the changes in expression of these important mediators of inflammation can be detected via a non-invasive method with skin tape strips. This finding also reveals the potential biochemical pathway on the pathogenesis of dandruff. In healthy scalp, the levels of ICAM-1 and P-selectin keep the right balance of immune response against pathogens without inflaming

the skin. In studies with P-selectin knockout, there was an increase of infection and abnormal immune tolerance.[33-35] During the development of dandruff, a dysregulation of such defense occurred. A high level of ICAM-1 recruited more inflammatory cells, causing irritation. P-selectin is reduced, potentially causing additional unwanted opportunistic infections, exacerbating the inflammation. The sequence of the event is not known at this point. However, certain populations may have a genetically lower level of P-selectin, resulting in higher susceptibility to external triggers that creates a vicious cycle of inflammation.

We observe that scalp microbiome is dominated by *Malassezia spp*, consistent with literature.[5,8,32] Our findings also suggest an increased *Malassezia*, especially *M restricta* in dandruff scalp. By contrast, higher abundance of *M globosa* was shown in healthy scalp compared to dandruff scalp, as previously reported.[5]

Most *Malassezia spp* show a unique lipid dependency and require external lipids for their growth. Genome mining of the incomplete *M restricta* genome led to the identification of 8 lipase sequences. This report supports the hypothesis that the Malassezia lipase family is responsible for the hydrolysis of triacylglycerols into irritating FFA, the main component of human sebum.[8] Additionally, a recent study[11] showed that *M restricta* leads to peroxidation of sebum lipids, which can result in barrier damage. This is consistent with our hypothesis that scalp barrier disruption by various internal/external factors favor the penetration of irritating *Malassezia*-derived metabolites which in turn leads to inflammatory cascade and results in worsening of the dandruff condition.

The current study also implicates an important role of bacterial profile imbalance in dandruff pathology. Significant increase in *S aureus* in dandruff scalp was observed, which was strongly correlated with the condition severity and might be linked to the barrier disruption as shown for skin.[36] Additionally, since *S epidermidis* was shown to be involved in ceramide production in the skin, lower level of this species observed in dandruff scalp, might further contribute to barrier disruption.[19] The decrease in *C acnes* could also contribute to the defect in ceramides since it stimulates intercellular lipids.[18] In addition to ceramide synthesis, *S epidermidis* from healthy skin supports skin barrier function by their capacity to produce metabolites, which could activate Aryl Hydrocarbon receptor pathway.[37] All these studies point to the need for protection of the scalp barrier with the proper composition of ceramides (higher chain length and higher content of phytosphingosine-based ceramides) may help disrupt this inflammatory cascade and alleviate the etiology of dandruff. This offers a new innovative approach to managing dandruff through reducing inflammation by maintaining the protective barrier of the scalp with the topical application of optimized mixture of ceramides and along with treatment to address both fungal and bacterial imbalance in dandruff scalp.

DISCLOSURES

All of the authors are employees of L'Oreal.

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