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1. Introduction

The human skin, encompassing over 30 m² of surface area including appendages and hair follicles, provides a complex and selective habitat for a diverse range of microorganisms—collectively termed the skin microbiome. Skin microbiome plays a critical role in the evolutionary history of human skin. The skin microbiome has evolved through genetic mutations to enhance its survival on the skin, aiming for perfect symbiosis with humans. This evolution allows the skin microbiome to adapt to environmental changes, thereby significantly influencing human skin health as a 'second skin' [1]. This includes bacteria, fungi, viruses, and microeukaryotes that together form a unique ecosystem distinct from those found in the gut, oral cavity, or vaginal tract [2,3]. The skin's environmental characteristics—such as acidic pH (4.5–5.5), cooler temperatures (29–34 °C), varying moisture levels, limited nutrients, and host-derived factors—shape its microbial composition. Core bacterial genera like *Staphylococcus*, *Corynebacterium*, and *Cutibacterium*, as well as fungi such as *Malassezia*, are differentially distributed across sebaceous, moist, and dry regions of the skin [4,5].

Far from being passive colonizers, these microbial communities contribute actively to skin health. The skin microbiome supports immune homeostasis, produces antimicrobial metabolites, and enhances barrier integrity. Commensals secrete bioactive compounds such as fatty acids, polyamines, bacteriocins, and indole derivatives that suppress pathogenic species, modulate inflammatory responses, and promote tissue repair [6,7]. These microbial functions are influenced by external factors such as environmental exposure, hygiene, cosmetics, and climate, as well as intrinsic host factors like inflammation and skin barrier status [2]. These microbial communities also serve as biomarkers of skin health: shifts in microbial diversity and structure are linked to acne, aging, and inflammatory conditions [8]. As such, skin microbiome profiling provides a framework for creating personalized skincare strategies tailored to an individual's unique biological and microbial profile [9,10], enhancing efficacy while minimizing irritation and imbalance [11].

Culture-independent methods such as high-throughput DNA sequencing have revolutionized our ability to map the microbiome in unprecedented detail. These tools have linked shifts in microbial communities with skin disorders including atopic dermatitis, psoriasis, acne, and diabetic wounds [12-14]. Moreover, they enable a more nuanced view of microbial contributions to skin physiology beyond what culture-based studies could reveal [15]. Despite these advances, there remains a substantial gap in our understanding of the healthy skin microbiome across populations, age groups, and skin types—an area with great potential to inform targeted and microbiome-informed interventions.

To explore this opportunity, we employed an unsupervised machine learning algorithm to cluster skin microbiome profiles into eight distinct groups. We focused on the largest cluster to demonstrate how microbial community structure can inform personalized skincare development. This data-driven approach supports the integration of microbiome-targeted ingredients, including postbiotics, into formulations tailored to specific microbial and physiolo-

gical skin characteristics. By aligning product design with individual microbiome profiles, this strategy paves the way for more effective, personalized, and microbiome-focused solutions.

2. Materials and Methods

2.1. Participant Recruitment and Data Collection

The study included participants who completed a skin microbiome sampling kit (n=862). Privacy Policy was followed throughout participant information processing. Analyses were performed using deidentified data. Microbiome samples were self-collected from the forehead area using cotton swabs. Prior to swabbing, the participants followed a 24-hour wash-out period, avoiding any facial products or washing with water. The swabbing protocol includes swabbing the forehead area between the eyebrows for 80 seconds. Swab samples were stored in liquid buffer at room temperature and shipped within four days of sampling. A questionnaire was completed by all the participants to report their demographic information (gender, ethnicity, location, age), skin profiles (skin type, conditions, issues), and general lifestyle habits (skin routines and goals). All personally identifiable information (PII) was removed before clustering and microbiome analyses, which were conducted on locally encrypted hardware storage using FileVault (Apple) and BitLocker (Microsoft). For server data transfers, we employed secure HTTPS connections with current SHA-256 and RSA encryption protocols. Access to critical team resources including GitHub, AWS, and OneDrive was protected by mandatory two-factor authentication throughout the study.

2.2. Microbiome Sequencing and Cluster Analysis

Microbial DNA was extracted from the collected swabs, followed by amplification of targeted 16S ribosomal RNA (rRNA) using V1-V3 region for bacteria and Internal Transcribed Spacer 2 (ITS2) region for fungi and sequencing of the targeted region. Sequencing was performed by a third-party CLIA-CAP certified diagnostic lab. Contaminants were removed using a proprietary algorithm. Distinct clusters of skin microbial profiles were identified through unsupervised machine learning techniques. This study focuses on one of the largest clusters among the eight optimal clusters generated. The raw bacterial and fungal microbiome data for this cluster were processed using the phyloseq (v1.46.0) package in R (v4.3.3) as the local environment, enabling analysis and visualization of microbiome load, diversity, and taxonomic composition in conjunction with the associated metadata using ggplot2 (v3.5.1). Statistical analyses were performed using the Wilcoxon rank-sum test for pairwise comparisons and the Kruskal-Wallis test for multiple group comparisons. Benjamini-Hochberg correction was applied for multiple comparisons.

2.3. Microbiome Isolation and Culture

To develop skin-beneficial microbiome ingredients, two bacteria (*Streptococcus, Corynebacterium*) were isolated from facial skin samples. The collection of human facial skin samples in this study was approved by the Institutional Review Board (IRB Protocol Number: HBABN01-210217-HR-0181-01) of the H&BIO Corporation R&D CENTER. The isolated bacteria were stored at the COSMAX Microbiome Bank. A single colony of each bacterium was picked from the plate and grown overnight in a skin-mimetic broth and incubated at 30 °C on a shaker (160 rpm) for 48 h in the dark. After incubation, the culture was filtered using a syringe filter. All culture filtrates were prepared in triplicate [16].

2.4. Cell Culture and Treatment

The human dermal fibroblast cell line Hs68 was obtained from the American Type Culture Collection. Hs68 cells were seeded at a density of 3.5×10^5 in a 6-well plate and cultured in Dulbecco's modified Eagle's medium supplemented with 1% antibiotic antimycotic and 10% fetal bovine serum (FBS) for 24 h in an incubator at 37 °C and 5% CO_2. Immortalized human keratinocytes (HaCaT) were also obtained from the ATCC. The cells were cultured in DMEM media supplemented with 10% FBS and incubated for 24 h in an incubator at 37 °C and 5% CO_2.

2.5. Gene Expression Analysis

2.5.1. Barrier enhancement Effect

For collagen type I alpha 1 chain (COL1A1) and matrix metalloproteinase-1 (MMP-1) assay in UV-irradiated condition, the Hs68's medium was removed, Dulbecco's phosphate-buffered saline was added, and the cells were irradiated with or without 12 mJ/cm^2 of UVB. After UVB irradiation, DPBS was removed and replaced with FBS-free DMEM media. *Streptococcus* spp. postbiotics [1% (w/w)] were treated and further incubated for 24 h. A sample treated with UV light, but no treatment was used as the negative control [17-19]. The gene expression of COL1A1 and MMP-1 was analyzed using PCR.

2.5.2. Anti-inflammation effect

HaCaT cells were cultured in DMEM media supplemented with 10% FBS and incubated for 24 h in an incubator at 37 °C and 5% CO_2. The medium was changed every 3-4 days, and the cells were sub-cultured when they overgrew. Cells were seeded at a density of 5 × 105/well and washed with phosphate-buffered saline (PBS) after 24 h of incubation. The cell medium was replaced with FBS-free DMEM, and 10 ng/mL polyinosinic–polycytidylic acid (Poly(I:C)) and 10 ng/mL interleukin 4 (IL-4) were added to induce inflammation. *Corynebacterium* postbiotics [1% (w/w)] was added to the cells and incubated for 4 h. The gene expression of and interleukin-1β (IL-1β) was analyzed using PCR with the aforementioned conditions, except that the primers for IL-1β were used [20,21].

2.5.2. Melanin Content Assay

To analyze the anti-melanogenic effects of *Corynebacterium* postbiotics, a melanin content assay was performed. Murine melanoma cells (B16F10) were obtained from ATCC. The cells were suspended in 2 mL of DMEM media supplemented with 10% FBS, inoculated into 6-well plates at a density of 1 × 10^6 cells/well, and incubated at 37 °C and 5% CO_2 for 24 h until 40-50% of the cells adhered to the bottom of the wells. Subsequently, they were treated with α-Melanocyte-stimulating hormone (α-MSH) at a concentration of 100 nM to induce melanogenesis. Subsequently, *Corynebacterium* ferment [1% (w/w)] was added to the cells and incubated for 3 days. The cells were then harvested and centrifuged at 1,000 rpm for 10 min to obtain the supernatant. The supernatant was evaluated for melanin secretion inhibition by measuring the absorbance at 490 nm in triplicate using a microplate reader (Victor 3, PerkinElmer). Percentage melanin secretion was calculated using the equation from Choi et al. [22], with arbutin (100 ppm, Sigma Aldrich) serving as a positive control.

2.6. RNA isolation and real-time PCR

Total RNA was isolated from cells using TRIzol reagent according to the manufacturer's instructions. cDNA was synthesized from 1 μg of total RNA using Reverse Transcription Premix under the following reaction conditions: cDNA synthesis at 45 °C for 45 min and heat inactivation at 95 °C for 5 min. Gene expression sig-nals were quantified with real-time PCR, and the data were analyzed using StepOne Plus™ system software. Real-time PCR ampli-fication reactions were performed using SYBR Green PCR Master Mix with premixed ROX. Total RNA was isolated from cells using TRIzol reagent according to the manufacturer's instructions. cDNA was synthesized from 1 μg of total RNA using Reverse Transcription Premix under the following reaction conditions: 45 °C for 45 min and 95 °C for 5 min. Gene expression sig-nals were quantified with real-time PCR, and the data were analyzed using StepOne Plus™ system software. Real-time PCR ampli-fication reactions were performed using SYBR Green PCR Master Mix with premixed ROX. The following primer pairs were used in the reactions performed in an ABI 7300 following the manufacturer's instructions: β-actin (F: 5'-GGCCATCTCTTGCTCGAAGT-3' and R: 5'-GACACCTTCAACACCCAGC-3'), IL-1β (F: 5'-GTCATTCGCTCCCACATTCT-3' and R: 5'-ACTTCTTGCCCCCTTTGAAT-3'), MMP-1 (F: 5'-CGAAATTTGCCGACAGAGATGA-3' and R: 5'-GTCCCTGAACAGCCCAGTACTT-3'), and COL1A1 (F: 5'-CTCGAGGTGGACACCACCCT-3' and R: 5'-CAGCTGGATGGCCACATCGG-3'). The reaction conditions were as follows: initiation at 50 °C for 2 min and 95 °C for 10 min, followed by cycling at 95 °C for 10 s and 60 °C for 1 min for 40 cycles. β-actin was used as an internal control.

2.6. Formulation

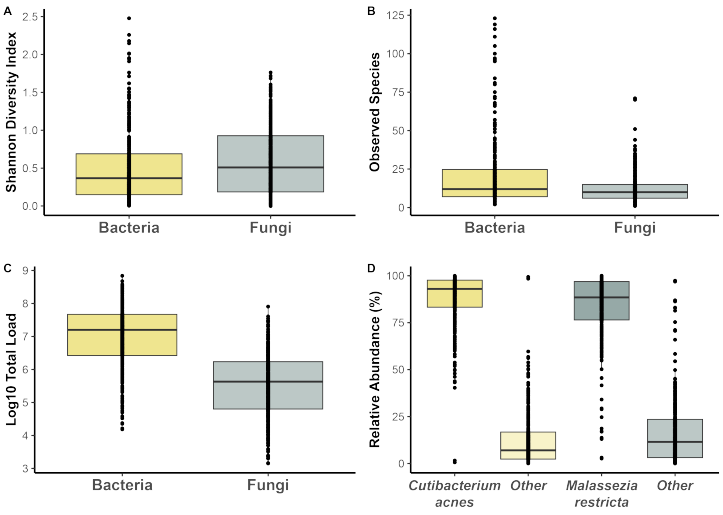
The serum was prepared by sequentially combining ingredients. Ingredients (water, panthenol, sodium phytate, sorbitol water, niacinamide, allantoin, acacia senegal gum xanthan gum) were mixed in the main vessel at 250 rpm for 5 minutes, followed by the addition of a premix of glycerin methylpropanediol and polyacrylate crosspolymer-6 under agitation at 300 rpm for 5 minutes. Polyglyceryl-3 distearate glyceryl stearate citrate was then slowly added with continuous mixing at 500 rpm for 15 minutes. The mixture was heated to 75–80°C. Separately, the oil phase (glyceryl stearate, squalane, caprylic/capric triglyceride) was prepared at 80°C and gradually added to the main vessel. Emulsification was performed using a homogenizer at 5000 rpm for 5 minutes. After emulsification, the batch was cooled to 40°C, and ingredients (ethylhexylglycerin tocopherol, phenoxyethanol, saccharomyces ferment filtrate sodium benzoate potassium sorbate, streptococcus spp. ferment extract filtrate, corynebacterium ferment, water, 1,2-hexanediol sodium hyaluronate crosspolymer butylene glycol xylose polyglutamic acid, tetrahexyldecyl ascorbate, tocopherol, butylene glycol water fomes officinalis (mushroom) extract, water salix alba (willow) bark extract) were added sequentially. A second homogenization was conducted at 5000 rpm for 3 minutes, followed by cooling to 30°C to complete the formulation.

2.6. Statistical Analysis

Statistical analysis for all experiments was conducted after repeating them at least three times, with each experiment being performed in triplicate. The results are presented as mean ± standard deviation. For *in vitro* cell assay analysis, statistical analysis was performed using the SPSS statistical package version 25.0 (SPSS Inc., Chicago, IL, USA). Statistical analyses were conducted with a two-tailed Student's t-test, and p-values of less than 0.05 and 0.01 were considered statistically significant.

3. Results

3.1. Metadata and Microbiome Results of Cluster 1



In our investigation, we identified eight optimal skin microbiome clusters using unsupervised machine learning based on facial microbiome profiles. Among these, we focus on one of the largest clusters, Cluster 1, which represents a substantial segment of the population. This cluster is characterized by a microbiome predominantly colonized by *Cutibacterium acnes* and *Malassezia restricta*, particularly in the forehead region **(Figure 1D)**. It also exhibits low diversity in both evenness and richness, driven by the high dominance of these lipophilic species **(Figure 1)**. Individuals within the cluster were subclustered into three groups: **healthy**, comprising those with no self-reported inflammatory skin conditions (e.g., acne, rosacea, eczema, or psoriasis); **inflamed**, consisting of individuals who reported having such conditions; and **other,** which included participants whose skin concerns were not clearly specified **(Table 1)**.

Figure 1. Microbiome Results of Cluster 1. (A) Shannon Diversity Index (B) Observed Species Index (C) Absolute Load (D) Relative Abundance Table of Dominant Species and Others

Demographic analysis revealed distinctive characteristics among the subgroups of Cluster 1. The predominant age demographic was individuals in their thirties, with the In-flamed group showing a higher representation of younger participants (19-30 years old) in the early reproductive stage. Skin sensitivity patterns demonstrated clear group differentiation, with 64% of participants in the Healthy cohort reporting non-sensitive skin. Conversely, the In-flamed and Other groups predominantly reported skin sensitivity (66.9% and 64.7%, respectively). Skin type distribution also varied notably between groups. The In-flamed group exhibited higher proportions of combination and oily skin types relative to the healthy group, while the Other group demonstrated the highest prevalence of dry skin type. In contrast, the Healthy cohort showed the highest percentage of balanced skin (39.5%), followed by combination-to-dry skin types **(Table 1)**.

Table 1 Metadata Overview of Cluster 1

	Healthy	Inflamed	Other	Total
	n=86	n=242	n=34	n=362
Age Range				
8-18 (Puberty)	0.0	1.2	0.0	0.8
19-30 (Early Reproductive)	17.4	24.8	14.7	22.1
31-40 (Mid Reproductive)	41.9	50.8	50.0	48.6
41-45 (Late Reproductive)	16.3	12.4	11.8	13.3
46-55 (Perimenopause)	11.6	7.9	14.7	9.4
>55 (Postmenopause)	12.8	2.9	8.8	5.8
Face Issues				
Acne/breakouts	0.0	39.5	15.7	31.1
Wrinkles	45.6	21.3	18.6	24.7
Hyperpigmentation	20.4	18.0	8.6	17.4
Rosacea	0.0	12.9	4.3	10.1
Eczema	0.0	7.2	2.9	5.7
Psoriasis	0.0	1.2	1.4	1.0
None	34.0	0.0	0.0	5.1
Other	0.0	0.0	48.6	5.0
Skin Sensitivity				
Yes	36.0	66.9	64.7	59.4
No	64.0	33.1	35.3	40.6
Skin Type				
Balanced	39.5	12.4	5.9	18.2
Combination	27.9	53.3	44.1	46.4
Dry	26.7	22.7	44.1	25.7
Oily	4.7	10.3	2.9	8.3
Unknown	1.2	1.2	2.9	1.4

3.2. Intra-cluster Stratification of Healthy vs. Inflamed Subgroups

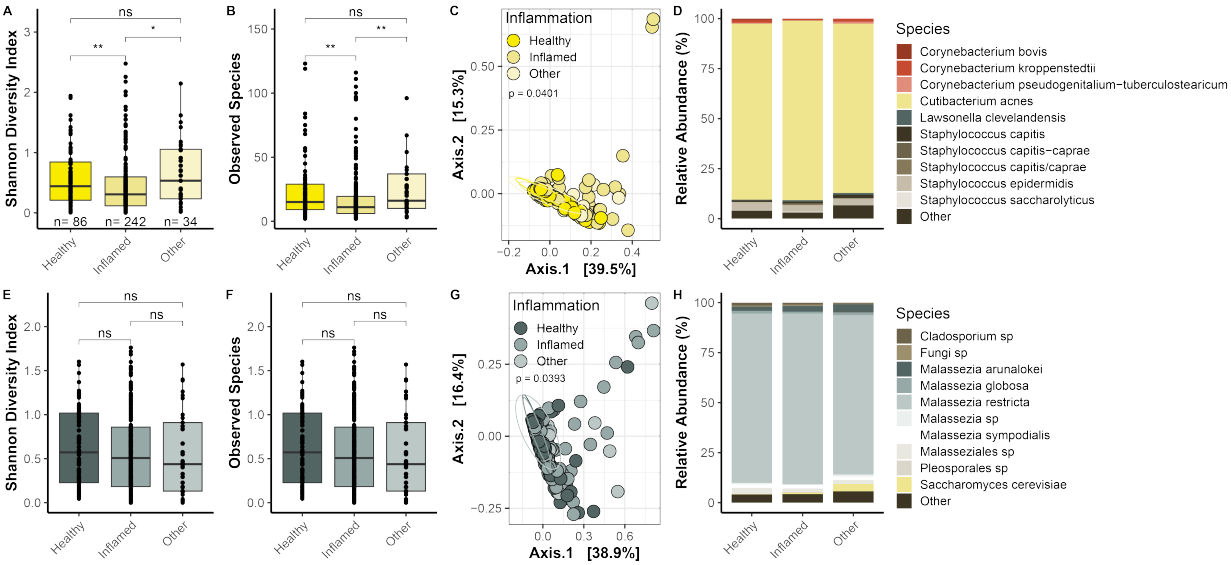
Alpha diversity analysis revealed that bacterial evenness **(Figure 2A)** and richness **(Figure 2B)** were significantly lower in the Inflamed group compared to both the Healthy and Other groups. In contrast, no significant differences in fungal alpha diversity were observed among the three groups **(Figure 2E, F)**. This demonstrates that skin inflammation is associated with a reduction in bacterial diversity, while fungal diversity remains conserved across skin conditions.

Beta diversity analysis assessed using Bray-Curtis dissimilarity and PERMANOVA indicated significant differences in overall microbial community structure for both bacteria **(Figure 2C)** and fungi **(Figure 2G)** across the Healthy, Inflamed, and other groups. This illustrates that both bacterial and fungal communities undergo significant structural alterations in inflammatory skin conditions. While fungal diversity remains stable, community composition shifts significantly, parallel to the compositional changes observed in the bacterial community.

At the taxonomic level, several shifts were observed across bacterial and fungal communities. In the bacterial community, *Staphylococcus epidermidis* showed a decreasing trend in the Inflamed group compared to the Healthy group, while *Cutibacterium acnes* exhibited a tendency toward higher abundance in the Inflamed group relative to both Healthy and Other groups. *Cutibacterium bovis* was not detected in the Other group **(Figure 2D)**. In addition, *Corynebacterium pseudogenitalium* was significantly reduced in the Inflamed group compared to both the Healthy (adjusted p = 0.0001) and Other (adjusted p = 0.001) groups.

Among fungal taxa, *Cladosporium* was lower in abundance in both the Inflamed and Other groups compared to Healthy, while *Saccharomyces cerevisiae* was more abundant in

the Inflamed group **(Figure 2H)**, although the difference was not significant. *Malassezia globosa* was significantly less abundant in the Inflamed group compared to the Healthy group (adjusted p = 0.045).



ogether, our findings demonstrate a higher prevalence and diversity of

Figure 2 Microbiome Results of Target Cluster. (A) Bacterial Shannon Diversity (B) Bacterial Observed Species (C) Bacterial Relative Abundance (D) Bacterial Bray-Curtis Dissimilarity PCoA (E) Fungal Shannon Diversity (F) Fungal Observed Species (G) Fungal Relative Abundance **(H)** Fungal Bray-Curtis Dissimilarity PCoA. *p<0.05, **p<0.01

Corynebacterium species within the healthy cohort compared to the inflamed group. Notably, the inflamed group also exhibited a higher percentage of self-reported skin sensitivity, a condition often associated with impaired barrier function. Based on these insights, we screened five candidate postbiotic ferments with anti-inflammatory, barrier-reinforcing, and collagen-supporting activities. Out of these, two candidates were selected that best matched the specific needs of the Cluster 1 skin profile:

- A *Corynebacterium* postbiotics with demonstrated anti-inflammatory and anti-melanogenic properties, aimed at restoring skin barrier health and improving uneven skin tone.
- A *Streptococcus* postbiotics with proven collagen-boosting activity, supporting dermal structure and resilience.

3.3. Ingredient Isolation and Efficacy Data for Corynebacterium *and* Streptococcus *spp. postbiotics*

We evaluated the effects of Streptococc-

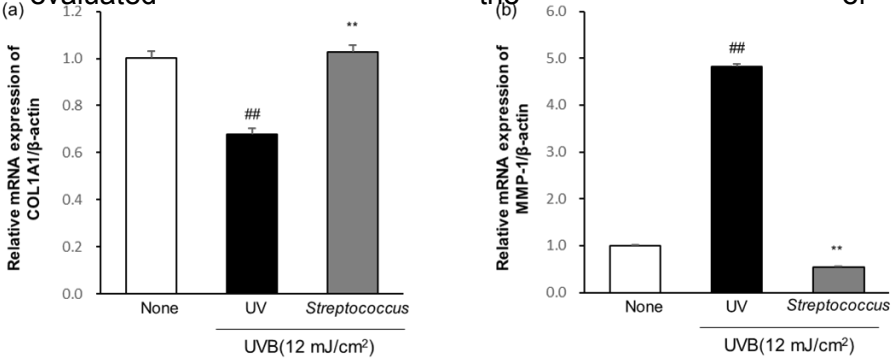


Figure 3 Effect of Streptococcus spp. postbiotics on the mRNA expression levels of COL1A1 and MMP-1 in Hs68 cells. #p<0.05 vs. None, ##p<0.01 vs. None, ###p<0.005 vs. None, *p < 0.05 vs. UV, **p < 0.01 vs. UV.

cus spp. postbiotics on key skin-related gene expression in UV-damaged human dermal fibroblasts. Treatment with 1% Streptococcus spp. postbiotics significantly upregulated the mRNA expression of COL1A1, showing a 1.51-fold increase compared to the UV-treated control group ($p < 0.05$; **Figure 3**). This suggests that Streptococcus spp. postbiotics effectively restored COL1A1 expression levels close to those observed in healthy, undamaged cells. In contrast, the expression of MMP-1, a collagen-degrading enzyme, was significantly downregulated following Streptococcus spp. postbiotics treatment, showing a 0.54-fold decrease relative to UV-damaged cells ($p < 0.05$), indicating its potential anti-aging and collagen-protective effect. We further evaluated the anti-inflammatory effects of Corynebacterium postbiotics by analyzing the gene expression of IL-1β, a known pro-inflammatory factor, in cells treated with Poly(I:C) 10 μg/ml plus IL-4 10 ng/ml ($p < 0.05$; **Figure 4A**). Treatment with 1% Corynebacterium postbiotics significantly reduced IL-1β gene expression from 1.87-fold to 1.38-fold relative to the control conditions.

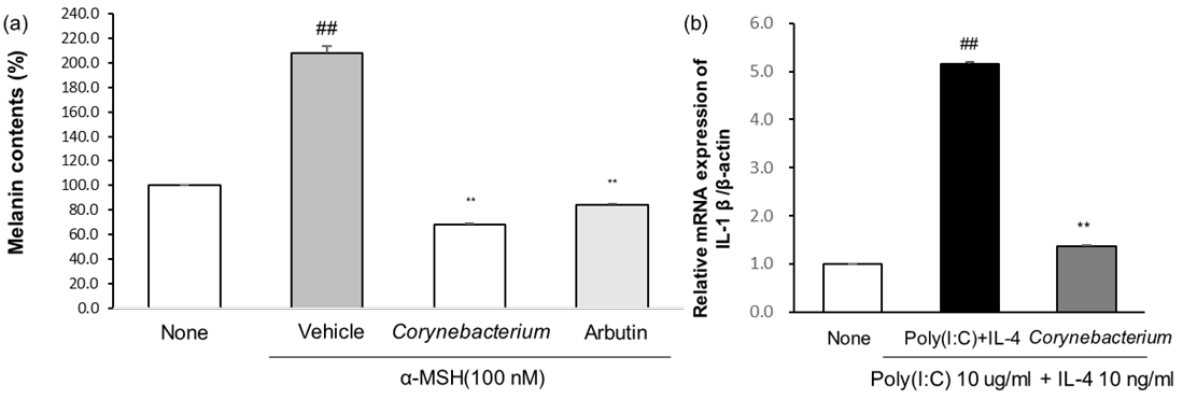


Figure 4 Effect of Corynebacterium postbiotics on the mRNA expression levels of the IL-1β in HaCaT cells and melanin content in B16F10 cells. #p<0.05 vs. None, ##p<0.01 vs. None, *p < 0.05 vs. Inducer or Vehicle, **p < 0.01 vs. Inducer or Vehicle.

also assessed the anti-melanogenic activity of Corynebacterium postbiotics using a melanin content assay in α-MSH-stimulated melanocytes (**Figure 4B**). Treatment with 10% Corynebacterium postbiotics significantly reduced melanin levels to 0.684-fold relative to the untreated baseline ($p < 0.05$). In comparison, α-MSH stimulation alone increased melanin content to 2.08-fold, while arbutin at the same concentration reduced it to 0.845-fold. These results indicate that Corynebacterium postbiotics more effectively suppressed melanin production than arbutin under identical conditions ($p < 0.05$). *3.4. Cluster 1-Specific Formulation Approach*

Cluster 1 exhibits a fair to medium skin tone, with a predominance of combination to dry skin with sensitivity and a notable proportion of oily skin. This complex skin profile involves both sebum excess and barrier weakness, requiring a multi-faceted care approach. To address these needs, we developed a targeted serum formulation incorporating the postbiotic complex *Streptococcus spp.* postbiotics for barrier support and soothing of sensitive, dry-prone areas **(Figure 3)**. In addition, *Corynebacterium postbiotics*, combined with niacinamide and vitamin C, was included to promote skin brightening and reduce post-acne hyperpigmentation **(Figure 4)**. This ingredient strategy was designed to provide comprehensive care for Cluster 1 skin, addressing sensitivity, dryness, oiliness, and uneven tone simultaneously.

3.4. Serum HRIPT Results

The HRIPT confirmed that serum is non-irritant and non-sensitizing, making it suitable for cosmetic use. Based on the test population of 112 subjects, with 56 subjects with self-perceived sensitive skin, and under the conditions of this study, the test material identified as

our facial serum did not demonstrate a potential for eliciting dermal irritation or inducing sensitization.

4. Discussion

In this study, we employed an unsupervised machine learning approach to classify facial skin microbiome profiles into eight distinct clusters, focusing on Cluster 1 for targeted skincare formulation. Cluster 1 was dominated by *Cutibacterium acnes* and *Malassezia restricta*, showed reduced microbial diversity, and had a high prevalence of self-reported sensitivity—often corresponding to younger individuals with oily or combination skin. While most individuals in this cluster exhibited inflammation-prone microbiome signatures, a subset presented with non-inflamed, healthy skin despite similar microbial dominance. This intra-cluster variability allowed further stratification to explore microbial and host factors contributing to dysbiosis. Within the inflamed subgroup, reduced abundance of *Staphylococcus epidermidis* and *Corynebacterium* species—particularly *C. pseudogenitalium*—was observed, alongside a non-significant increase in *C. acnes*, suggesting their roles in preserving skin health.

Staphylococcus epidermidis is known to contribute to barrier integrity, immune modulation, and antimicrobial defense [23-24], whereas *C. acnes* plays a dual role depending on strain type. While it aids in lipid metabolism and pathogen inhibition via propionic acid production [25,26], certain phylogroups such as IA1 and IA2 are linked to inflammation and acne through biofilm formation and immune activation [14, 27-28]. Although strain-level resolution of C. acnes was beyond the scope of this study, the observed increase highlights a valuable direction for future investigation into its strain-specific roles. Additionally, *C. pseudogenitalium* was significantly depleted in inflamed individuals, supporting previous studies that suggest a potential immunomodulatory role for non-diphtherial *Corynebacterium* species in maintaining microbial homeostasis and barrier health [29-30].

To address the unique features of Cluster 1 skin, we formulated a microbiome-informed serum combining *Streptococcus* spp. and *Corynebacterium* postbiotics with niacinamide and vitamin C. The *Streptococcus* postbiotic enhanced collagen synthesis and barrier repair by upregulating COL1A1 and downregulating MMP-1 in UV-damaged fibroblasts, while the *Corynebacterium* postbiotic reduced IL-1β and melanin levels more effectively than arbutin in inflammatory models. These ingredients were selected to address the sensitivity, dryness, and post-inflammatory hyperpigmentation commonly seen in this cluster.

Safety and efficacy were confirmed via HRIPT and in vitro assays, demonstrating the non-irritating, non-sensitizing nature of the final serum, even among sensitive users. This study illustrates the power of microbiome segmentation in guiding personalized formulation design. By matching ingredient function with specific microbial patterns and host phenotypes, microbiome-informed skincare offers a promising avenue for more effective, targeted, and well-tolerated products. A follow-up clinical trial is underway to assess real-world outcomes in individuals identified as Cluster 1, validating improvements in barrier function, inflammation, and skin tone.

5. Conclusion

In conclusion, this study successfully characterized the skin microbiome, reinforcing its critical role in maintaining skin health and resilience, and underscoring its potential as a target for innovation in next-generation skincare. By leveraging microbiome cluster analysis, we demonstrate the feasibility of a data-driven, personalized formulation approach that aligns ingredient selection with the specific biological and microbial characteristics of each skin segment. This microbiome-informed strategy offers a promising path toward enhancing skincare efficacy while minimizing adverse effects such as irritation or imbalance. Further clinical evaluation will provide direct evidence of the formulation’s impact on improving skin barrier function, reducing sensitivity, and evening skin tone in individuals exhibiting the Cluster 1 microbiome profile, ultimately validating the application of microbiome-personalized skincare in the development of future cosmetic innovations.

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