

Article

Heat-Treated *Lacticaseibacillus rhamnosus* Skinbac™ SB06 Modulates Axillary Malodor-Associated Bacteria In Vitro and Demonstrates Antiperspirant and Deodorant Efficacy In Vivo

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Abstract

Background: The axillary microbiome is a major contributor to body malodor generation through bacterial metabolism of apocrine and eccrine secretions. Dysbiosis of this microbial community, particularly through overgrowth of odorigenic species such as *Staphylococcus aureus*, is associated with increased volatile compound production and local skin inflammation. Heat-treated postbiotics represent a promising class of cosmetic ingredients combining microbiological safety with retained bioactive properties. **Objective:** We aimed to evaluate the in vitro safety, molecular mechanisms, antipathogen and anti-inflammatory properties of heat-treated *Lacticaseibacillus rhamnosus* Skinbac™ SB06, and to assess the antiperspirant and deodorant efficacy of a deodorant spray formulation containing 1% SB06 in a controlled clinical study. **Methods:** In vitro studies assessed cytotoxicity (MTT/LDH assays), Aquaporin-3 (AQP3) expression, reactive oxygen species (ROS) production, antipathogen activity against *Staphylococcus aureus* (AlamarBlue assay), cytokine modulation (TNF- α , IL-6, IL-8, IL-23) in Normal Human Epidermal Keratinocytes (NHEK) and Peripheral Blood Mononuclear Cells (PBMCs), and axillary microbiome compatibility against *Corynebacterium striatum*, *Staphylococcus epidermidis*, and *Staphylococcus hominis* by viable plate count (CFU/mL). Clinically, a randomized split-body study ($n = 20$) evaluated antiperspirant effectiveness by gravimetric sweat collection and deodorant efficacy by expert olfactory panel (Likert 1–5) at 24 and 48 h. **Results:** In vitro testing confirmed the safety of SB06 (MTT and LDH, both non-significant vs. control). SB06 significantly increased AQP3 expression (+20%, $p < 0.001$) and significantly reduced ROS production (−48%, $p < 0.05$). Antipathogen testing showed significant reduction in *S. aureus* planktonic viability (−7%, $p < 0.05$). Microbiome compatibility testing on selected axillary-associated strains showed a differential compatibility profile, with the strongest inhibitory effect observed for *C. striatum* (13% residual viability at T24h, corresponding to 87% inhibition), near-complete preservation of *S. epidermidis* (92% residual viability at T48h), and a mild reduction in *S. hominis* (−15% at T48h). Cytokine modulation showed significant IL-8 and IL-23 reduction in NHEK (both $p \leq 0.05$) and immunostimulatory activity in PBMCs. Clinically, SB06 reduced sweat production vs. placebo by −21.8% at T24 ($p = 0.0009$) and −10.0% at T48 ($p = 0.0495$), with significantly lower odor intensity at both timepoints (median score 3 vs. 4, $p < 0.0001$). **Conclusions:** Heat-treated *L. rhamnosus* SB06 showed a multimodal in vitro profile including antipathogen, anti-inflammatory, antioxidant, and AQP3-upregulating activities, and was associated with statistically significant antiperspirant and deodorant effects in a randomized controlled split-body study. These findings are consistent with SB06 being a functional postbiotic ingredient with potential for deodorant and antiperspirant applications, pending confirmation in larger controlled studies.



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Keywords: Aquaporin-3; antiperspirant; axillary microbiome; body odor; cytokine modulation; deodorant spray; heat-treated probiotics; *Lactocaseibacillus rhamnosus*; postbiotics; skin microbiome

1. Introduction

The axillary region constitutes a unique anatomical microenvironment characterized by warmth, moisture, occlusion, and high densities of both eccrine and apocrine sweat glands, creating optimal conditions for a specialized resident microbial community [1,2]. Unlike other skin sites, axillary skin is continuously exposed to apocrine secretions containing odorless precursor molecules—including steroids, fatty acid conjugates, and sulfanyl alkanols—that are transformed by resident bacteria into the volatile compounds responsible for characteristic axillary malodor, including 3-methyl-2-hexenoic acid, 3-hydroxy-3-methylhexanoic acid, and 3-methyl-3-sulfanylhexasan-1-ol [3–5].

The axillary microbiome is dominated by *Corynebacterium*, *Staphylococcus*, and *Cutibacterium*, whose relative abundances determine odor intensity and quality [2,6]. *Corynebacterium* species are considered the primary producers of thioalcohol-derived malodor through C-S lyase activity, while coagulase-negative staphylococci are associated with milder odor profiles [5,7]. *Staphylococcus aureus*, although typically a minor component, exerts a disproportionate odorigenic and inflammatory influence when its relative abundance increases: it converts amino acid conjugates and fatty acid precursors into potent volatile malodorous compounds, forms resilient biofilms, and promotes local barrier disruption and inflammatory responses [7–9]. In addition, epidermal tight junctions contribute to cutaneous barrier homeostasis and are relevant to the maintenance of skin integrity in sites exposed to repeated chemical and mechanical stress, such as the axilla [10]. Current antiperspirant and deodorant strategies rely predominantly on aluminum-based salts for sweat reduction and antimicrobial agents for odor control [11]. These approaches, while effective, can induce axillary microbiome dysbiosis by reducing protective species and enabling recolonization by more odorigenic populations [12,13]. Growing consumer demand for microbiome-compatible, naturally derived ingredients has directed cosmetic research toward biological alternatives targeting malodor at its microbial source without collateral disruption of the resident microbiota [14].

Postbiotics—defined as preparations of non-viable microorganisms and/or their components that confer a health benefit on the host [15]—represent an emerging class of cosmetic active ingredients with significant potential in this context. Heat inactivation of probiotic strains generates structurally intact bacterial cell corpses retaining bioactive cell wall components, metabolites, lipoteichoic acids, and peptidoglycans that mediate immunomodulatory, antimicrobial, antioxidant, and barrier-enhancing effects [16,17]. Thermal inactivation further confers practical formulation advantages including ambient stability, extended shelf life, and regulatory simplicity compared to live microorganism delivery [17,18].

Lactocaseibacillus rhamnosus (formerly *Lactobacillus rhamnosus*) is among the most extensively studied *Lactobacillus* species for both gastrointestinal and cutaneous applications [19,20]. Its production of organic acids contributes to local acidification inhibitory to pathogens, while its surface components facilitate competitive exclusion at epithelial adhesion sites [21]. In cutaneous applications, *L. rhamnosus* strains have demonstrated anti-inflammatory properties through pro-inflammatory cytokine downregulation, barrier-supporting tight junction upregulation, and oxidative stress reduction in keratinocytes [20,22], making heat-treated *L. rhamnosus* a candidate of mechanistic in-

terest for deodorant formulations requiring simultaneous antipathogen efficacy, local anti-inflammation, and skin conditioning.

Aquaporin-3 (AQP3), the predominant water channel in the epidermis, mediates water and glycerol transport across keratinocyte membranes, supporting stratum corneum hydration, barrier repair, and cellular proliferation [23,24]. Its expression is suppressed by inflammatory cytokines and oxidative stress [25]—conditions relevant to axillary skin subjected to microbiome dysbiosis or repeated conventional deodorant use. AQP3 upregulation by postbiotic ingredients may therefore contribute to improved skin conditioning in the underarm region, particularly in users experiencing dryness or sensitivity.

This study aimed to provide a comprehensive in vitro and in vivo characterization of heat-treated *L. rhamnosus* Skinbac™ SB06 for deodorant formulation development. In vitro objectives were to: (I) establish safety by cytotoxicity assays; (II) quantify AQP3 modulation; (III) assess antioxidant capacity by ROS quantification; (IV) evaluate antipathogen activity against *S. aureus* planktonic cells and biofilm; and (V) characterize cytokine modulation in NHEK and PBMCs. The in vivo objective was to evaluate the antiperspirant effectiveness and deodorant efficacy of a 1% SB06 deodorant spray formulation compared to a matched placebo vehicle in a randomized split-body controlled clinical study.

2. Materials and Methods

2.1. Cell Culture

Normal Human Epidermal Keratinocytes (NHEK-Ad, Cat. No. 00192627; Lonza, Basel, Switzerland) were cultured in Keratinocyte Growth Medium (Cat. No. 00192060; Lonza) at 37 °C/5% CO₂. Cells were seeded at 5×10^4 cells/well in 24-well plates and allowed to adhere overnight before treatment. Cryopreserved peripheral blood mononuclear cells were obtained from Lonza (Basel, Switzerland). Cells were thawed according to the manufacturer's protocol, washed twice with pre-warmed complete RPMI-1640 medium, and resuspended in complete medium supplemented with 10% heat-inactivated fetal bovine serum (FBS; Thermo Fisher Scientific, Waltham, MA, USA) and 2 mM L-glutamine. Cells were seeded at 1×10^6 cells/mL and used as a model of systemic immune response.

2.2. Preparation of Heat-Treated Probiotic Strain

Lactocaseibacillus rhamnosus Skinbac™ SB06 was thermally inactivated at temperatures exceeding 75 °C for 30–90 min and spray-dried. Enumeration was performed by flow cytometry using Thiazole Orange/Propidium Iodide staining (TO/PI, Cat. No. 349483; BD Biosciences, Franklin Lakes, NJ, USA) to determine total fluorescent units (TFU), standardized to 10^9 TFU/g. The resulting powder was stored at 4 °C until use. All in vitro experiments were conducted at 10^7 TFU/mL.

2.3. In Vitro Testing

2.3.1. Safety Assessment

Cytotoxicity was evaluated using MTT tetrazolium reduction and Lactate Dehydrogenase (LDH) release assays. NHEK cells were treated with 10^7 TFU/mL SB06 for 24 h at 37 °C. LDH release was quantified from supernatants per manufacturer instructions (CytoTox 96®, Promega, Madison, WI, USA, Cat. No. G7891). For MTT, cells were incubated with 0.5 mg/mL MTT (Sigma-Aldrich, St. Louis, MO, USA, Cat. No. M-5655) for 4 h; formazan was dissolved in DMSO (Sigma-Aldrich, Cat. No. D5879) and absorbance read at 570 nm on a VarioskanLUX microplate reader (Thermo Fisher Scientific, Waltham, MA, USA). All experiments were performed in triplicate.

2.3.2. AQP3 Expression

NHEK cells were treated with 10^7 TFU/mL SB06 for 24 h at 37 °C. Following incubation, protein extraction was performed and AQP3 protein levels were quantified by ELISA (Human AQP3 ELISA Kit, Cat. No. HUF100733; Assay Genie, Dublin, Ireland). Results are expressed as percentage relative to untreated control cells. All experiments were performed in triplicate.

2.3.3. Antioxidant Activity—ROS Quantification

NHEK cells were stimulated with 10^7 TFU/mL SB06 and incubated for 24 h at 37 °C. Supernatants were collected and ROS levels quantified by cytochrome C reduction colorimetric assay (Sigma-Aldrich, Cat. No. C3131; absorbance 550 nm). Results are expressed as percentage reduction relative to untreated controls. All experiments were performed in triplicate.

2.3.4. Antipathogen Activity Against *Staphylococcus aureus*

Staphylococcus aureus (SA) was plated at $OD_{600} = 0.05$ in 48-well plates and immediately co-incubated with SB06 at 10^7 TFU/mL. After 72 h, planktonic cells were separated from biofilm by medium aspiration and metabolic activity of both fractions assessed by Alamar-Blue assay (resazurin sodium salt, 0.015%; Sigma-Aldrich). Fluorescence was recorded (excitation 560 nm/emission 590 nm) and expressed as Relative Fluorescence Units (RFU) relative to *S. aureus* monoculture controls. All experiments were performed in triplicate.

2.3.5. Axillary Microbiome Compatibility

The microbiome compatibility profile of SB06 was assessed against selected axillary-resident microbial strains of interest: *Staphylococcus epidermidis* ATCC 14990, *Staphylococcus hominis* ATCC 27844, and *Corynebacterium striatum* ATCC 6940 (ABICH S.r.l., Verbania, Italy; Report REL/1082/2025). Each strain was prepared from frozen stock and inoculated at $OD_{600} \approx 0.2$ in Tryptic Soy Broth (TSB) medium, then immediately co-incubated with SB06 at 10^7 TFU/mL at 37 °C. Phosphate-buffered saline (PBS) served as the vehicle control. Microbial growth was monitored by absorbance at 600 nm and confirmed by viable plate counts (CFU/mL) at T0, T2h, T4h, T6h, T24h, and T48h. All conditions were tested in triplicate. Results are reported at the timepoints of primary interest: T4h and T48h for *S. epidermidis*, T24h for *C. striatum*, and T48h for *S. hominis*.

2.3.6. Cytokine Modulation in NHEK and PBMCs

Immunomodulatory properties were assessed in NHEK (skin model) and PBMCs (systemic immune model). For the NHEK model, UVC irradiation (30 J/cm^2) was applied concurrently with SB06 stimulation to mimic an inflammatory skin stress condition, consistent with the methodology previously employed for this strain series [26]; cells were co-stimulated with 10^7 TFU/mL SB06 and UVC for 24 h. PBMCs were stimulated with 10^7 TFU/mL SB06 alone for 24 h. At the end of incubation, supernatants were collected and concentrations of TNF- α , IL-6, IL-8, and IL-23 were quantified by ELISA (R&D Systems, Minneapolis, MN, USA) according to the manufacturer's instructions. Results are expressed as fold increase relative to untreated control cells (basal level = 1). Statistical comparisons were performed using Student's *t*-test. All experiments were performed in triplicate.

2.4. Clinical Study

2.4.1. Study Design and Population

A randomized split-body open-label controlled study was conducted by ABICH S.r.l. (Clinical and Cosmetological Trials Center, Vimodrone, Italy; Report REL/3452/2025) on 20 healthy volunteers of both sexes, aged 18–65 years, presenting intense axillary sweating (minimum 100 mg of sweat collected from each axilla in 20 min under controlled

conditions at screening, prior to randomization) (Table 1). Eligible participants were healthy adults considered suitable for participation by the investigators. Exclusion criteria included pregnancy or breastfeeding, local or systemic medication potentially affecting skin response, signs of irritation at the test site, active skin disease that could interfere with study outcomes, participation in other concurrent clinical studies, and any condition judged by the investigators to make the subject unsuitable for participation.

Table 1. Demographic and study design characteristics of the clinical study population. Mean age $\pm$ SD and sex distribution were not reported in the final clinical report provided by ABICH S.r.l. (Report REL/3452/2025).

Characteristic	Value
Study design	Randomized within-subject (split-body), controlled, open-label
Sample size (n)	20
Sex	Male and female
Age range (years)	18–65
Inclusion criterion—sweating	≥ 100 mg axillary sweat/20 min under controlled conditions
Washout period	≥ 17 days (no antiperspirant use)
Study duration	~33 days per volunteer
Adverse events/dropouts	None/0

All participants underwent a pre-treatment washout period of at least 17 days during which the use of any antiperspirant product was prohibited. The study was conducted in accordance with the principles of Good Clinical Practice (GCP) and the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment. Data were processed in compliance with EU Regulation 679/2016 (GDPR).

Volunteers were randomized so that half applied the test product (Deo Vapo Base + SB06, COSM25_001SA) to the left axilla and the placebo (Deo Vapo Base, COSM25_001S) to the right axilla; the remaining half were assigned in the opposite manner. The INCI composition of the active formulation was: Aqua, Maltodextrin, PEG-40 Hydrogenated Castor Oil, PPG-26-Buteth-26, Lactobacillus, Glycerin, Sodium Benzoate, Potassium Sorbate, Citric Acid (SB06 included at 1% concentration). The placebo vehicle contained: Aqua, PEG-40 Hydrogenated Castor Oil, PPG-26-Buteth-26, Glycerin, Sodium Benzoate, Potassium Sorbate, Citric Acid. Both formulations were aqueous spray solutions; the active formulation appeared as a turbid aqueous solution owing to the postbiotic active ingredient.

2.4.2. Product Application

Both products were applied once daily for 4 consecutive days at a dose of 2 mg/cm². For each sniff test session, volunteers washed both axillae in the laboratory using a fragrance-free, non-antibacterial neutral soap provided by ABICH prior to product application. Products were applied by trained ABICH technicians under standardized conditions.

2.4.3. Antiperspirant Effectiveness Assessment

Antiperspirant effectiveness was assessed gravimetrically at 24 h (T24) and 48 h (T48) after the last daily treatment. Subjects were placed in a controlled hot room (temperature 40 °C, relative humidity 30–40%) for a 40-min warm-up period to thermally induce perspiration, followed by a 20-min collection period during which pre-weighed absorbent underarm pads (One-Time Underarm Sweat Pad, 113 mm $\times$ 123 mm) were placed under both axillae. Pad weights were measured before and after the collection period using an analytical balance (LA214i) to determine sweat mass in milligrams. The percentage reduction in sweat production on the treated side versus the placebo side was calculated as the primary endpoint.

2.4.4. Deodorant Efficacy Assessment—Olfactory Sensory Analysis (Sniff Test)

Deodorant efficacy was assessed by olfactory sensory analysis (sniff test) performed by a panel of three trained expert evaluators from ABICH S.r.l. working in a well-ventilated, odor-free environment. Evaluators independently scored the intensity of axillary sweat odor immediately after product application (T0), and at 24 (T24) and 48 (T48) hours after application, using a Likert scale from 1 to 5 (1 = no sweat odor; 2 = light; 3 = moderate; 4 = intense; 5 = very intense). Median scores across the three evaluators were used as the summary statistic per volunteer and timepoint. Olfactory assessments at T0 served as the post-wash baseline, with both axillae starting at a score of 1. Because the study was conducted under an open-label design, evaluators were not blinded to treatment allocation.

2.5. Statistical Analysis

In vitro data are presented as mean $\pm$ standard deviation (SD). Normality was assessed using the Kolmogorov–Smirnov test. Comparisons between treatment groups and controls were performed using paired or independent *t*-tests as appropriate. Statistical significance was set at $p < 0.05$. No correction for multiple comparisons was applied in the exploratory in vitro analyses; results should be interpreted as hypothesis-generating. All in vitro experiments were performed in biological triplicate, consistent with standard practice for exploratory cell-based assays; findings should be interpreted as preliminary and require independent replication. In vitro analyses were performed using GraphPad Prism 9.0 (GraphPad Software, San Diego, CA, USA).

For the clinical study, antiperspirant effectiveness was analyzed by paired *t*-test comparing mean sweat mass (in mg) between product and placebo axillae at T24 and T48. Deodorant efficacy (olfactory scores) within each group over time was analyzed by the Friedman test (non-parametric repeated measures), with between-side comparisons at T24 and T48 performed using a paired statistical test. Statistical analyses for the clinical study were performed by ABICH S.r.l. using certified laboratory software according to FDA guidelines for antiperspirant effectiveness testing. *p*-values and significance levels are reported as follows: ns ($p > 0.05$), * ($p \leq 0.05$), ** ($p \leq 0.01$), *** ($p \leq 0.001$), **** ($p \leq 0.0001$).

3. Results

3.1. In Vitro Safety Assessment

The safety profile of SB06 was evaluated in NHEK cells (Figure 1). The MTT assay demonstrated maintained metabolic activity (100% relative to untreated control), and the LDH assay showed no significant increase in membrane permeability following 24-h treatment with 10^7 TFU/mL SB06. Both assays confirm the absence of cytotoxic effects at the tested concentration, establishing the safety foundation required for topical formulation development.

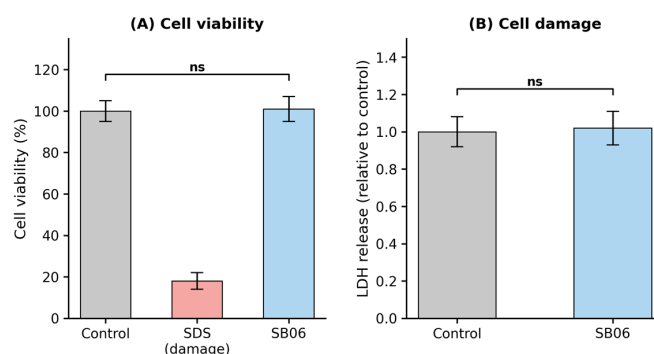


Figure 1. Safety assessment of SB06 in NHEK cells. (A) Cell viability by MTT assay; (B) Cytotoxicity by LDH release assay after 24-h treatment (10^7 TFU/mL). Data represent mean $\pm$ SD of three independent experiments. ns = not significant vs. untreated control.

3.2. AQP3 Expression

SB06 treatment induced a statistically significant increase in AQP3 protein expression in NHEK cells compared to untreated controls (+20% relative to control, $p < 0.001$; Figure 2). This upregulation was achieved without associated cytotoxic effects, indicating a specific biological response.

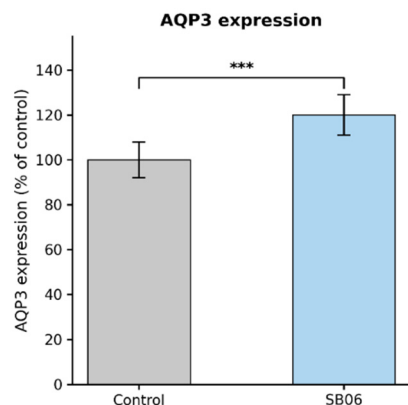


Figure 2. AQP3 protein expression in NHEK cells after 24-h treatment with SB06 (10^7 TFU/mL), measured by ELISA. Data represent mean $\pm$ SD of three independent experiments. *** $p < 0.001$ vs. untreated control.

3.3. Antioxidant Activity—ROS Quantification

SB06 induced a statistically significant reduction in ROS production in NHEK cells (−48%, $p < 0.05$; Figure 3). This magnitude of antioxidant activity is relevant for attenuating oxidative-stress-driven skin damage associated with axillary dysbiosis and inflammatory episodes.

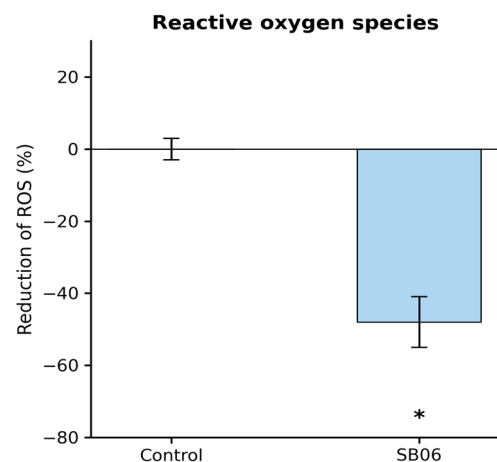


Figure 3. ROS production in NHEK cells after 24-h treatment with SB06 (10^7 TFU/mL), quantified by cytochrome C reduction assay. Results expressed as percentage reduction vs. untreated control. Data represent mean $\pm$ SD of three independent experiments. * $p < 0.05$ vs. untreated control.

3.4. Antipathogen Activity Against *Staphylococcus aureus*

Co-incubation with SB06 for 72 h produced a statistically significant reduction in *S. aureus* planktonic cell metabolic activity compared to the monoculture control (−7%, $p < 0.05$; Figure 4). A directional reduction in biofilm metabolic activity was also observed (−8%), which did not reach statistical significance ($p \approx 0.1$) and should not be interpreted as evidence of biological activity in the absence of formal significance. These results, while modest in magnitude, suggest a directional inhibitory effect of SB06 against the tested *S. aureus* model and warrant confirmation in adequately powered, strain-specific studies.

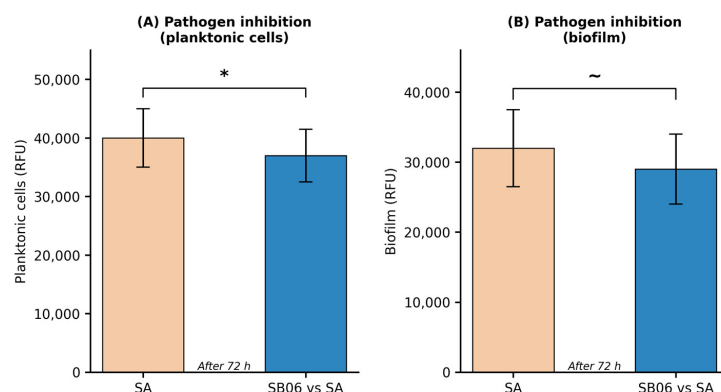


Figure 4. Activity of SB06 against the tested *S. aureus* model after 72-h co-incubation. **(A)** Planktonic cell metabolic activity (RFU). **(B)** Biofilm metabolic activity (RFU). Data represent mean $\pm$ SD of three independent experiments. * $p < 0.05$ vs. *S. aureus* monoculture control (statistically significant); $\sim p = 0.1$ vs. *S. aureus* monoculture control (not significant, directional trend only).

3.5. Axillary Microbiome Compatibility

The microbiome compatibility profile of SB06 against selected axillary-resident species is shown in Figure 5. Within this selected panel, SB06 showed a differential effect across species, with the strongest inhibitory activity observed for *C. striatum*.

C. striatum showed a marked reduction in viable counts at T24h: SB06-treated cultures retained only 13% residual viability relative to the PBS vehicle control at the same timepoint, corresponding to an 87% inhibition of viable counts. This represents the strongest inhibitory effect observed across the tested species and is consistent with mechanistic relevance to the deodorant application context, given the established role of *C. striatum* as the primary thioalcohol-producing taxon in the axillary microbiome [5,7].

S. epidermidis exhibited a transient reduction at T4h (62% residual viability relative to control; -38%) with near-complete recovery at T48h (92% residual viability relative to control), demonstrating that the effect on this beneficial commensal is transient and does not persist over the full 48-h observation period.

S. hominis showed a consistent reduction in viable counts at T48h (85% residual viability relative to control; -15%), indicating a sustained, albeit moderate, inhibitory effect on this thioalcohol-contributing species over the 48-h observation period. A single type strain per species was tested; broader isolate panels are required before a general axillary-microbiome-compatibility claim can be supported.

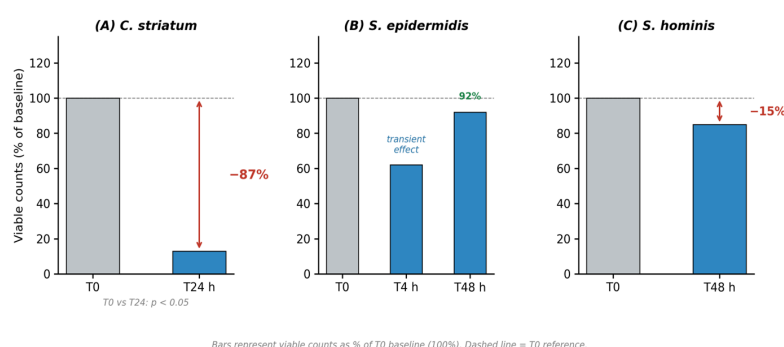


Figure 5. Viable counts expressed as percentage of T0 baseline (T0 = 100%) are shown for SB06-treated cultures relative to the corresponding PBS control at the same timepoint for **(A)** *C. striatum* at T0 and T24h, **(B)** *S. epidermidis* at T0, T4h, and T48h, and **(C)** *S. hominis* at T0 and T48h. Values represent means from triplicate experiments conducted under standardized conditions. Replicate-level variability data were not reported in the primary laboratory report (ABICH S.r.l., Report REL/1082/2025); error bars are therefore omitted. Annotated percentages indicate residual viability or inhibition relative to the PBS control at the same timepoint.

3.6. Cytokine Modulation in NHEK and PBMCs

The cytokine modulation profile of SB06 was characterized across two cellular models (Table 2). In NHEK cells, SB06 induced a significant reduction in IL-8 ($0.2\times$, $p < 0.01$) and IL-23 ($0.2\times$, $p < 0.05$), while TNF- α was unaffected and IL-6 showed a non-significant increase ($1.9\times$, $p \approx 0.1$). In PBMCs, SB06 induced a marked upregulation of TNF- α ($13.3\times$, $p < 0.001$) and IL-6 ($7\times$, $p < 0.001$) alongside a significant reduction in IL-8 ($0.94\times$, $p < 0.01$). IL-23 in PBMCs was not significantly altered.

Table 2. Cytokine modulation profile of SB06 in NHEK and PBMC models. Results expressed as fold increase $\pm$ SD relative to untreated basal level (=1).

Cytokine	Cell Model	Basal Level	SB06	p-Value
TNF- α	PBMCs	1 $\pm$ 0.05	13.3 $\pm$ 0.02	$p < 0.001$
	NHEK	1 $\pm$ 0.01	1.1 $\pm$ 0.1	ns
IL-6	PBMCs	1 $\pm$ 0.18	7 $\pm$ 0.2	$p < 0.001$
	NHEK	1 $\pm$ 0.04	1.9 $\pm$ 0.01	$p \approx 0.1$
IL-8	PBMCs	1 $\pm$ 0.06	0.94 $\pm$ 0.01	$p < 0.01$
	NHEK	1 $\pm$ 0.01	0.2 $\pm$ 0.1	$p < 0.01$
IL-23	PBMCs	1 $\pm$ 0.02	1 $\pm$ 0.04	ns
	NHEK	1 $\pm$ 0.00	0.2 $\pm$ 0.03	$p < 0.05$

ns = not significant.

3.7. Clinical Efficacy

3.7.1. Antiperspirant Effectiveness

Gravimetric sweat collection data from 20 volunteers are summarized in Table 3 and Figure 6. At T24, the SB06-treated axilla produced a mean of 477 mg of sweat compared to 609 mg in the placebo-treated contralateral axilla, corresponding to a statistically significant reduction of -21.8% ($p = 0.0009$, ***). At T48, mean sweat production was 639 mg (SB06) versus 710 mg (placebo), representing a significant reduction of -10.0% ($p = 0.0495$, *). Individual-level data confirmed that the majority of volunteers responded to treatment in the direction of reduced sweat production at both timepoints. Effect size analysis indicated a large effect at T24 (Cohen's $d = 0.85$; 95% CI for mean difference: -206 to -60 mg) and a medium effect at T48 (Cohen's $d = 0.56$; 95% CI: -130 to -12 mg), supporting the practical magnitude of the observed antiperspirant effect at both timepoints, within the limitations of this exploratory study. Individual-level data are presented in Figure 10. At T24, 17 of 20 volunteers (85%) showed lower sweat production on the SB06-treated side compared to the placebo-treated contralateral axilla; at T48, this was observed in 16 of 20 volunteers (80%), confirming a majority-responder pattern at both timepoints.

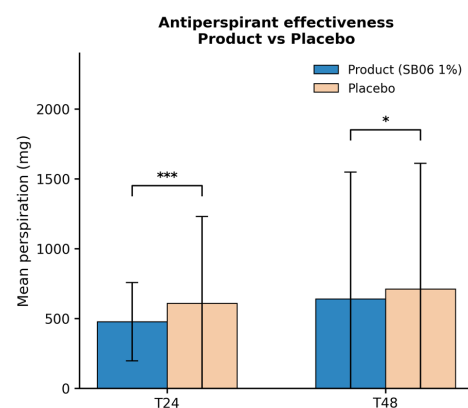


Figure 6. Mean axillary perspiration (mg) in the SB06-treated and placebo-treated axillae at T24 and T48 after the last daily treatment. Statistical significance: *** $p \leq 0.001$; * $p \leq 0.05$ (paired t -test, SB06 vs. placebo).

Table 3. Mean axillary sweat collected (mg) and percentage reduction for SB06 versus placebo at T24 and T48.

Timepoint	SB06 (mg)	Placebo (mg)	% Reduction vs. Placebo	95% CI (mg)	Cohen's d	p-Value
T24	477	609	−21.8%	[−206, −60]	0.85	0.0009 (***)
T48	639	710	−10.0%	[−130, −12]	0.56	0.0495 (*)

3.7.2. Deodorant Efficacy—Olfactory Sensory Analysis

Olfactory sensory evaluation results are summarized in Table 4 and Figures 7–9. At T0 (immediately after washing and product application), both axillae were rated at a median score of 1 (no sweat odor), confirming an equivalent and odor-free baseline for both conditions. At T24, the median odor intensity score in the SB06-treated axilla was 3 (moderate odor) compared to 4 (intense odor) on the placebo side. At T48, the same pattern persisted: SB06 3 versus placebo 4. Both T24 and T48 comparisons between product and placebo reached high statistical significance ($p < 0.0001$, **** for both timepoints).

Within-group analysis (Friedman test) confirmed that odor intensity increased significantly over time for both conditions: for the SB06 product, T0 vs. T24 ($p = 0.0024$, **) and T0 vs. T48 ($p < 0.0001$, ****); for the placebo, T0 vs. T24 ($p = 0.0002$, ***) and T0 vs. T48 ($p < 0.0001$, ****). Importantly, the rate and magnitude of odor increase were attenuated in the SB06-treated axilla versus the placebo at both measurement points, confirming a genuine deodorant effect of the postbiotic formulation.

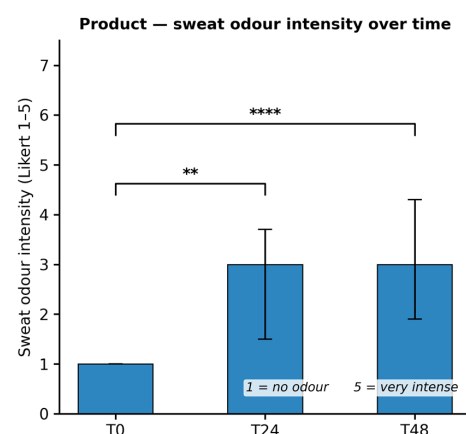
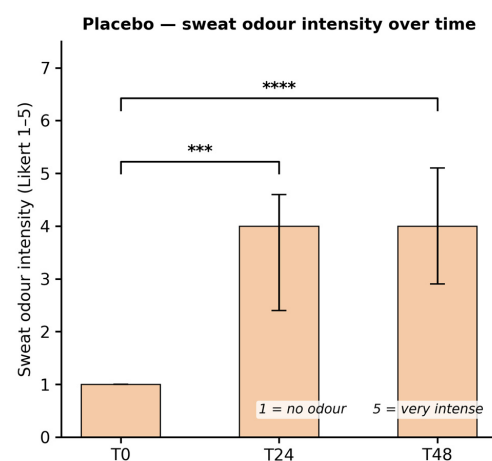
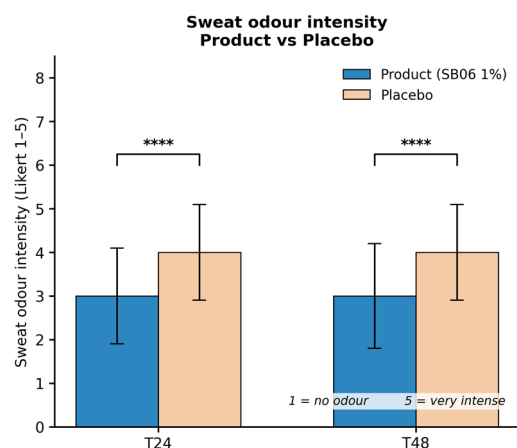
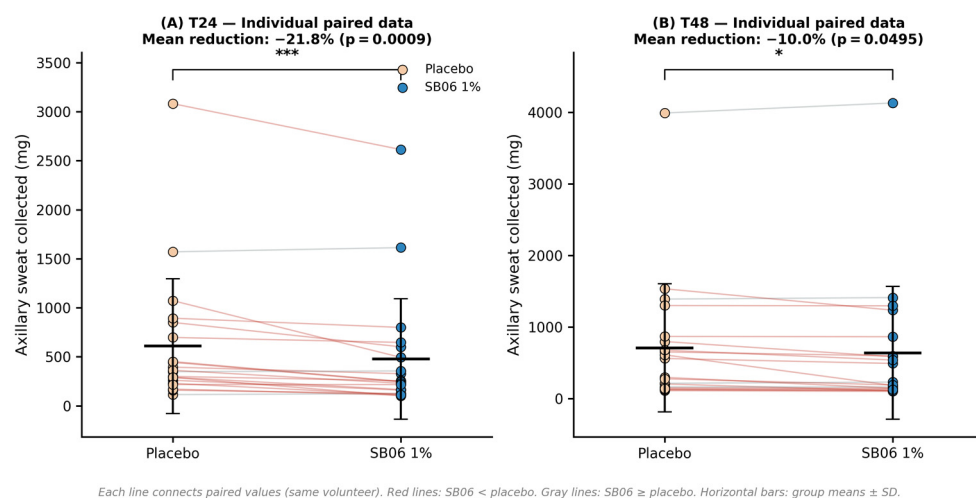
**Figure 7.** Variation in sweat odor intensity (Likert 1–5) in the SB06-treated axilla over time (T0, T24, T48). Within-group Friedman test: T0 vs. T24 $p = 0.0024$ (**); T0 vs. T48 $p < 0.0001$ (****).**Figure 8.** Variation in sweat odor intensity in the placebo-treated axilla over time (T0, T24, T48). Within-group Friedman test: T0 vs. T24 $p = 0.0002$ (***); T0 vs. T48 $p < 0.0001$ (****).

Table 4. Median sweat odor intensity scores (Likert 1–5) for SB06 and placebo at T0, T24, and T48.

Timepoint	SB06 (Median)	Placebo (Median)	SB06 vs. Placebo <i>p</i> -Value
T0	1 (no odor)	1 (no odor)	—(baseline)
T24	3 (moderate odor)	4 (intense odor)	<0.0001 (***)
T48	3 (moderate odor)	4 (intense odor)	<0.0001 (***)

*** $p < 0.0001$.**Figure 9.** Direct comparison of sweat odor intensity between SB06 and placebo at T24 and T48. Paired comparison: both $p < 0.0001$ (****).**Figure 10.** Individual paired sweat production data (mg) at T24 (A) and T48 (B). Each line connects placebo and SB06 values from the same volunteer; red lines indicate volunteers in whom sweat production was lower on the SB06-treated side (T24: 17/20; T48: 16/20), gray lines indicate volunteers in whom sweat production on the SB06-treated side was equal to or higher than on the placebo-treated side (T24: 3/20; T48: 4/20). Horizontal bars represent group means $\pm$ SD. Statistical significance by paired *t*-test: *** $p \leq 0.001$ (T24); * $p \leq 0.05$ (T48).

4. Discussion

This study provides a comprehensive characterization of heat-treated *L. rhamnosus* SB06 as a postbiotic ingredient for deodorant applications, integrating mechanistic in vitro evidence with controlled clinical data that collectively support its potential value as a functional active ingredient with in vitro microbiome-compatible properties. The combination of a split-body randomized design with matched placebo control and two orthogonal clinical endpoints—gravimetric antiperspirant assessment and expert olfactory

evaluation—substantially strengthens the clinical interpretation relative to uncontrolled single-arm studies.

The confirmed safety of SB06 in NHEK cells established through complementary MTT and LDH assays represents an essential prerequisite for topical deodorant formulation. MTT reflects mitochondrial metabolic integrity while LDH quantifies membrane permeability, together excluding primary and secondary cytotoxicity at the tested concentration [27,28].

The statistically significant AQP3 upregulation (+20%, $p < 0.001$) contributes a skin conditioning dimension to the SB06 profile that is of relevance for axillary applications. AQP3 mediates water and glycerol transport across keratinocyte membranes, contributing to stratum corneum hydration, barrier repair, and cellular proliferation [23,24,29]. Its suppression by inflammatory cytokines and oxidative stress [25] is directly relevant to axillary skin subjected to repeated conventional deodorant application or microbiome dysbiosis.

AQP3 upregulation by SB06 suggests a potential contribution to skin hydration and barrier-supporting processes in the axillary region, independently of any mechanistic link to the observed antiperspirant effect. However, the present study was not designed to directly evaluate skin barrier function, and no direct relationship can be inferred between AQP3 modulation and the antiperspirant effect observed in the clinical study.

The statistically significant ROS reduction achieved by SB06 (−48%, $p < 0.05$) is notable compared to data on related heat-treated strains: whereas *L. plantarum* SB01 and *B. animalis* spp. lactis SB05 showed a non-significant directional ROS reduction (−33%, $p = 0.06$) at comparable concentrations [30], SB06 achieves formal statistical significance, suggesting a more potent or mechanistically distinct antioxidant activity for this specific strain. Postbiotic-derived antioxidant activity may be mediated by radical scavenging from cell wall components including exopolysaccharides and peptidoglycan fragments, and by indirect activation of endogenous antioxidant enzymes [31,32]. In the axillary context, ROS reduction may attenuate inflammation-driven malodor amplification and limit oxidative degradation of skin lipids serving as volatile compound precursors [33]. The observed activity of SB06 against the tested *S. aureus* model is mechanistically relevant to the deodorant application context, although confirmation across additional strains would strengthen generalizability. The statistically significant reduction in planktonic cell viability ($p \leq 0.05$) and consistent directional biofilm trend ($p \approx 0.1$) indicate competitive or antagonistic pressure on this odorigenic species, possibly mediated by organic acid production, competitive adhesion site exclusion, or bacteriocin-like substances released during heat inactivation [16,34]. While a 7% reduction in planktonic viability may appear modest in absolute terms, its biological relevance should be interpreted in the context of the axillary microenvironment rather than against conventional antimicrobial benchmarks. In the axillary ecosystem, *S. aureus* typically represents a minority taxon whose odorigenic and inflammatory influence is disproportionate to its relative abundance [7,8]. Partial competitive suppression of *S. aureus*—combined with the concurrent anti-inflammatory activity observed in NHEK cells—may be sufficient to attenuate its contribution to malodor and local irritation without requiring bactericidal eradication. Furthermore, the postbiotic mechanism of action is inherently distinct from that of chemical antimicrobials: rather than direct killing, it likely involves competitive exclusion and microenvironmental modulation, for which even modest reductions in viable counts may translate to meaningful ecological effects [16,34].

The broader microbiome selectivity profile of SB06, including selective activity against *Corynebacterium striatum*, is discussed in the following paragraph.

The microbiome compatibility data obtained on the selected axillary-associated strains are consistent with a differential activity profile that may support microbiome-compatible

deodorant development. The marked reduction in *C. striatum* viable counts at T24h (13% residual viability relative to the PBS control at the same timepoint, corresponding to an 87% inhibition) is relevant given that *C. striatum* is recognized as the primary thioalcohol-producing taxon in the axillary microbiome, responsible for the conversion of odorless cysteine conjugates into the volatile sulfanylalkanols characteristic of intense underarm malodor [5,7,33]. *S. hominis*, which contributes to thioalcohol release through distinct enzymatic pathways [7], also showed a consistent inhibitory trend at T48h (−15% residual viability relative to control). By contrast, *S. epidermidis*—a beneficial commensal involved in skin barrier homeostasis, antimicrobial peptide production, and competitive exclusion of opportunistic pathogens [9,10,35]—showed only a transient reduction at T4h (62% residual viability) with recovery to 92% of control viability at T48h, confirming that SB06 does not persistently suppress this key protective species. This differential profile—characterized by stronger inhibition of *C. striatum* together with limited and partly transient effects on selected commensal species—is consistent with a microbiome-compatible mode of action in vitro and may differentiate postbiotic-based formulations from conventional broad-spectrum antimicrobials. It should be noted, however, that no direct microbiological assessment of the axillary microbiome was performed during the clinical study, and whether similar microbial modulation occurs under in vivo conditions remains to be established [12,13].

The cytokine modulation profile of SB06 reveals a context-dependent immunomodulatory pattern coherent for a deodorant ingredient. In NHEK cells, the significant reductions in IL-8 and IL-23 support a local anti-inflammatory effect: IL-8 is a potent neutrophil-attracting chemokine overproduced during axillary inflammatory episodes contributing to erythema and irritation [36], and its suppression suggests improved axillary skin comfort during product use. The concurrent IL-23 downregulation supports attenuation of the Th17-axis, a pathway implicated in chronic skin inflammatory conditions [37]. In PBMCs, upregulation of TNF- α and IL-6 reflects a pattern of innate immune activation, functionally distinct from tissue-pathological inflammation and consistent with the capacity for enhanced antimicrobial surveillance demonstrated by the antipathogen data [38,39]. The simultaneous IL-8 reduction in PBMCs supports a targeted rather than dysregulated immune response [36]. It should be explicitly noted that the pronounced TNF- α and IL-6 induction observed in PBMCs (13.3 $\times$ and 7 $\times$, respectively) may represent a cell-type-specific inflammatory activation pattern in this in vitro model, distinct from the anti-inflammatory profile observed in keratinocytes. However, two considerations limit the clinical relevance of this interpretation for a leave-on topical product. First, systemic exposure of circulating mononuclear cells to a topically applied deodorant spray is negligible under normal use conditions, making the PBMC model a worst-case surrogate rather than a physiologically representative one. Second, the local keratinocyte compartment—the tissue directly exposed during axillary application—showed a clearly anti-inflammatory profile (IL-8 $\downarrow$, IL-23 $\downarrow$), which is the endpoint most directly relevant to the safety and tolerability of the formulation. No adverse events or signs of local inflammation were observed in any of the 20 clinical volunteers, further supporting the absence of pro-inflammatory effects under actual use conditions. Given the exploratory nature of the in vitro analyses and the absence of multiple testing correction, these findings should be interpreted as mechanistic hypothesis-generating observations rather than definitive conclusions.

The clinical results are consistent with the in vitro mechanistic profile and provide controlled pilot support for its translational relevance. The −21.8% reduction in sweat production at T24 ($p = 0.0009$) and −10.0% at T48 ($p = 0.0495$) represent statistically significant and methodologically rigorous antiperspirant effects, assessed according to FDA guidelines using pre-weighed absorbent pads under standardized heat stress conditions in a split-body randomized design. The attenuation of the antiperspirant effect from T24

(−21.8%) to T48 (−10.0%) is consistent with the expected duration of action of a postbiotic formulation applied once daily. Unlike aluminum-based antiperspirants, which form physical plugs within eccrine ducts that persist for 24–72 h, postbiotic-based formulations likely act through biological mechanisms—including competitive modulation of axillary bacteria and local immunomodulation—whose effects are inherently transient and dependent on sustained application. The observed waning at T48 therefore reflects the natural dissipation of a single-dose biological effect rather than loss of intrinsic activity and would be expected to stabilize with continued daily use over longer application periods. This interpretation is consistent with the study design, which assessed efficacy after only 4 days of application—a period likely insufficient to establish the microbiome-modulating effects that may underlie longer-term deodorant efficacy. Future studies employing longer application periods and microbiome profiling will be required to characterize the full duration-of-action profile of SB06-based formulations.

The deodorant efficacy results were consistent across both assessment timepoints: the SB06-treated axilla maintained a significantly lower odor intensity score (median 3, “moderate”) compared to the placebo-treated contralateral axilla (median 4, “intense”) at both T24 and T48, with a between-group difference reaching $p < 0.0001$ at both timepoints. These findings support a deodorant effect of the postbiotic formulation relative to the base vehicle over 48 h following the final application. It is noteworthy that neither the product nor the placebo fully prevented odor development over 48 h—both arms showed significant within-group increases from T0 (Friedman test)—which reflects the physiological inevitability of progressive odor development in high-sweating individuals over this period. However, the SB06 formulation consistently attenuated this progression relative to the vehicle-only control, suggesting a cosmetically relevant effect under the present test conditions.

Taken together, the in vitro and in vivo data suggest that heat-treated *L. rhamnosus* SB06 possesses biological activities that may be relevant for deodorant applications, including antipathogen activity against the tested *S. aureus* model, modulation of inflammatory markers in keratinocytes, antioxidant activity, AQP3 upregulation, and a reduction in sweat production and odor intensity under placebo-controlled conditions. While these findings are consistent with a multifactorial mechanism of action, the relative contribution of each pathway to the observed clinical effects remains to be established.

This multimodal profile distinguishes SB06 from conventional deodorant actives and aligns with the growing demand for non-aluminum alternatives with potential microbiome-compatible properties [14].

Study Limitations

Several limitations should be acknowledged. The in vitro models, while well-validated for skin research, do not fully recapitulate the complexity of the axillary microenvironment including multispecies microbial interactions, eccrine and apocrine secretion chemistry, and the physical–chemical milieu of the underarm. Antipathogen activity against *S. aureus* was assessed using the AlamarBlue metabolic assay, which is methodologically distinct from the OD600/CFU viability approach used in Section 3.5; direct cross-assay comparison is therefore not appropriate. The modest magnitude of *S. aureus* planktonic inhibition and the non-significant biofilm trend require confirmation in mixed-species biofilm systems representative of the axillary environment. The immunostimulatory PBMC signals (TNF- α , IL-6) were not accompanied by functional antimicrobial killing readouts, and their translational relevance to topical application—where systemic PBMC exposure is negligible—should be interpreted in context.

Regarding the clinical study, a specific limitation of the deodorant efficacy assessment is the absence of evaluator blinding: olfactory assessors were aware of which axilla received

the active product, introducing potential assessment bias that cannot be excluded. This represents a meaningful limitation of the olfactory endpoint and should be considered when interpreting the odor intensity scores. Future studies should implement blinded olfactory assessment protocols, for example through coded sample presentation or independent panel evaluation, to minimize this source of bias. The sample size of 20 volunteers, while adequate for exploratory evaluation according to FDA antiperspirant testing guidelines, limits statistical power for detecting small between-subgroup differences and precludes definitive conclusions regarding responder characteristics. The biological basis of the observed antiperspirant effect remains unclear. Although SB06 significantly increased AQP3 expression in keratinocytes, AQP3 is a water channel expressed in epidermal keratinocytes and is not known to directly regulate eccrine sweat gland secretion. The study was not designed to investigate the mechanistic basis of the antiperspirant effect, and no causal link can be established between AQP3 modulation and reduced perspiration. The antiperspirant effect observed clinically may reflect distinct biological mechanisms that remain to be elucidated. Dedicated mechanistic studies will be required to clarify this observation. The clinical study population was not fully characterized in terms of sex distribution and mean age, as these variables were not systematically recorded in the study protocol; future studies should prospectively collect complete demographic data to allow subgroup analyses. Additional baseline variables including BMI, smoking status, physical activity level, and dermatological history were not systematically collected in the study protocol and therefore cannot be assessed as potential confounding factors for the sweating and body odor endpoints. Future studies should prospectively record these variables to enable covariate-adjusted analyses. The inclusion of volunteers requiring a minimum baseline sweat threshold (100 mg/20 min collected from each axilla under controlled conditions at screening) ensures methodological rigor for the antiperspirant endpoint but may reduce generalizability to the broader population of light sweaters. A major limitation of the present work is the absence of direct microbiological assessment of the axillary microbiome during the clinical study. Although the *in vitro* experiments demonstrated selective activity against representative axillary-associated species, it cannot be assumed that similar microbial shifts occurred *in vivo* under real-use conditions. Consequently, the proposed microbiome-mediated mechanism underlying the observed deodorant efficacy should be considered biologically plausible but not directly demonstrated. Future studies incorporating longitudinal microbiome profiling (e.g., 16S rRNA sequencing or metagenomic analysis) before and after treatment will be required to establish whether the *in vitro* selectivity profile translates to measurable microbiome modulation *in vivo* and whether clinical effects are associated with changes in microbial composition and function. Finally, the study duration of 4 application days, while consistent with standard antiperspirant efficacy testing protocols, does not permit assessment of effects associated with longer-term daily use.

5. Conclusions

This study suggests that heat-treated *L. rhamnosus* Skinbac™ SB06 possesses a multimodal activity profile that is mechanistically coherent and clinically supported for deodorant applications. *In vitro*, SB06 showed: (I) confirmed safety in NHEK; (II) significant AQP3 upregulation (+20%, $p < 0.001$); (III) statistically significant antioxidant activity (−48% ROS, $p < 0.05$), with a stronger signal than that previously observed for related postbiotic strains under comparable experimental conditions; (IV) antipathogen activity against *S. aureus* planktonic cells ($p < 0.05$) with a consistent directional biofilm effect; (V) a dual immunomodulatory profile combining local keratinocyte anti-inflammatory activity (IL-8↓, IL-23↓ in NHEK) with immune activation capacity in PBMCs; and (VI) a preferen-

tial inhibitory effect against *C. striatum* (13% residual viability at T24h, corresponding to 87% inhibition), the principal axillary malodor taxon, together with near-complete preservation of the beneficial commensal *S. epidermidis* (92% residual viability at T48h). Clinically, a 1% SB06 deodorant spray formulation was associated with statistically significant antiperspirant effectiveness versus a matched placebo in a split-body randomized study: −21.8% sweat reduction at T24 ($p = 0.0009$) and −10.0% at T48 ($p = 0.0495$). Deodorant efficacy was confirmed by olfactory sensory analysis, with the SB06-treated axilla showing significantly lower sweat odor intensity than placebo at both T24 and T48 (median score 3 vs. 4, both $p < 0.0001$).

These findings, obtained across in vitro assays and selected axillary-associated strains together with a controlled clinical study, are consistent with heat-treated *L. rhamnosus* SB06 being a functional postbiotic ingredient with potential for deodorant and antiperspirant formulations with in vitro microbiome-compatible properties that target malodor-associated microbial activity while also supporting skin conditioning. Larger, double-blind controlled studies incorporating axillary microbiome profiling and extended application periods are warranted to fully characterize the mechanistic and clinical potential of SB06-based deodorant formulations.

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References

1. Haykal, D.; Cartier, H.; Dréno, B. Dermatological Health in the Light of Skin Microbiome Evolution. *J. Cosmet. Dermatol.* **2024**, *23*, 3836–3846. [[CrossRef](#)] [[PubMed](#)]
2. Callewaert, C.; Ravard Helffer, K.; Lebaron, P. Skin Microbiome and its Interplay with the Environment. *Am. J. Clin. Dermatol.* **2020**, *21*, 4–11. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]

3. Medrano, A.C.; Cantu, A.; Aviles-Rosa, E.O.; Hall, N.J.; Maughan, M.N.; Gadberry, J.D.; Prada-Tiedemann, P.A. Chemical Characterization of Human Body Odor Headspace Components. *Separations* **2024**, *11*, 85. [\[CrossRef\]](#)
4. James, A.G.; Austin, C.J.; Cox, D.S.; Taylor, D.; Calvert, R. Microbiological and biochemical origins of human axillary odour. *FEMS Microbiol. Ecol.* **2013**, *83*, 527–540. [\[CrossRef\]](#) [\[PubMed\]](#)
5. Troccaz, M.; Gaia, N.; Beccucci, S.; Schrenzel, J.; Cayeux, I.; Starkenmann, C.; Lazarevic, V. Mapping axillary microbiota responsible for body odours using a culture-independent approach. *Microbiome* **2015**, *3*, 3. [\[CrossRef\]](#) [\[PubMed\]](#) [\[PubMed Central\]](#)
6. Urban, J.; Fergus, D.J.; Savage, A.M.; Ehlers, M.; Menninger, H.L.; Dunn, R.R.; Horvath, J.E. The effect of habitual and experimental antiperspirant and deodorant product use on the armpit microbiome. *PeerJ* **2016**, *4*, e1605. [\[CrossRef\]](#) [\[PubMed\]](#) [\[PubMed Central\]](#)
7. Bawdon, D.; Cox, D.S.; Ashford, D.; James, A.G.; Thomas, G.H. Identification of axillary *Staphylococcus* sp. involved in the production of the malodorous thioalcohol 3-methyl-3-sufanylhexan-1-ol. *FEMS Microbiol. Lett.* **2015**, *362*, fnv111. [\[CrossRef\]](#) [\[PubMed\]](#)
8. Herman, R.; Kinniment-Williams, B.; Rudden, M.; James, A.G.; Wilkinson, A.J.; Murphy, B.; Thomas, G.H. Identification of a staphylococcal dipeptidase involved in the production of human body odor. *J. Biol. Chem.* **2024**, *300*, 107928. [\[CrossRef\]](#) [\[PubMed\]](#)
9. Gallo, R.L.; Hooper, L.V. Epithelial antimicrobial defence of the skin and intestine. *Nat. Rev. Immunol.* **2012**, *12*, 503–516. [\[CrossRef\]](#) [\[PubMed\]](#) [\[PubMed Central\]](#)
10. Brandner, J.M. Tight junctions and tight junction proteins in mammalian epidermis. *Eur. J. Pharm. Biopharm.* **2009**, *72*, 289–294. [\[CrossRef\]](#) [\[PubMed\]](#)
11. Callewaert, C.; Hutapea, P.; Van de Wiele, T.; Boon, N. Deodorants and antiperspirants affect the axillary bacterial community. *Arch. Dermatol. Res.* **2014**, *306*, 701–710. [\[CrossRef\]](#) [\[PubMed\]](#)
12. Fredrich, E.; Barzantny, H.; Brune, I.; Tauch, A. Daily battle against body odor: Towards the activity of the axillary microbiota. *Trends Microbiol.* **2013**, *21*, 305–312. [\[CrossRef\]](#) [\[PubMed\]](#)
13. Nakatsuji, T.; Gallo, R.L. The role of the skin microbiome in atopic dermatitis. *Ann. Allergy Asthma Immunol.* **2019**, *122*, 263–269. Erratum in *Ann. Allergy Asthma Immunol.* **2019**, *123*, 529. [\[CrossRef\]](#) [\[PubMed\]](#) [\[PubMed Central\]](#)
14. Han, J.H.; Kim, H.S. Skin Deep: The Potential of Microbiome Cosmetics. *J. Microbiol.* **2024**, *62*, 181–199. [\[CrossRef\]](#) [\[PubMed\]](#)
15. Salminen, S.; Collado, M.C.; Endo, A.; Hill, C.; Lebeer, S.; Quigley, E.M.M.; Sanders, M.E.; Shamir, R.; Swann, J.R.; Szajewska, H.; et al. The International Scientific Association of Probiotics and Prebiotics (ISAPP) consensus statement on the definition and scope of postbiotics. *Nat. Rev. Gastroenterol. Hepatol.* **2021**, *18*, 649–667. Erratum in *Nat. Rev. Gastroenterol. Hepatol.* **2021**, *18*, 671. Erratum in *Nat. Rev. Gastroenterol. Hepatol.* **2022**, *19*, 551. [\[CrossRef\]](#) [\[PubMed\]](#) [\[PubMed Central\]](#)
16. Piqué, N.; Berlanga, M.; Miñana-Galbis, D. Health Benefits of Heat-Killed (Tyndallized) Probiotics: An Overview. *Int. J. Mol. Sci.* **2019**, *20*, 2534. [\[CrossRef\]](#) [\[PubMed\]](#) [\[PubMed Central\]](#)
17. Mehta, J.P.; Ayakar, S.; Singhal, R.S. The potential of paraprobiotics and postbiotics to modulate the immune system: A Review. *Microbiol. Res.* **2023**, *275*, 127449. [\[CrossRef\]](#) [\[PubMed\]](#)
18. Aguilar-Toalá, J.E.; Garcia-Varela, R.; Garcia, H.S.; Mata-Haro, V.; González-Córdova, A.F.; Vallejo-Cordoba, B.; Hernández-Mendoza, A. Postbiotics: An evolving term within the functional foods field. *Trends Food Sci. Technol.* **2018**, *75*, 105–114. [\[CrossRef\]](#)
19. Capurso, L. Thirty Years of *Lactobacillus rhamnosus* GG: A Review. *J. Clin. Gastroenterol.* **2019**, *53*, S1–S41. [\[CrossRef\]](#) [\[PubMed\]](#)
20. Ratanapokasatit, Y.; Laisuan, W.; Rattananukrom, T.; Petchlorlian, A.; Thaipisuttikul, I.; Sompornrattanaphan, M. How Microbiomes Affect Skin Aging: The Updated Evidence and Current Perspectives. *Life* **2022**, *12*, 936. [\[CrossRef\]](#) [\[PubMed\]](#) [\[PubMed Central\]](#)
21. Lopes, E.G.; Moreira, D.A.; Gullón, P.; Gullón, B.; Cardelle-Cobas, A.; Tavaría, F.K. Topical application of probiotics in skin: Adhesion, antimicrobial and antibiofilm *in vitro* assays. *J. Appl. Microbiol.* **2017**, *122*, 450–461. [\[CrossRef\]](#) [\[PubMed\]](#)
22. Habeebuddin, M.; Karnati, R.K.; Shiroorkar, P.N.; Nagaraja, S.; Asdaq, S.M.B.; Khalid Anwer, M.; Fattepur, S. Topical Probiotics: More Than a Skin Deep. *Pharmaceutics* **2022**, *14*, 557. [\[CrossRef\]](#) [\[PubMed\]](#) [\[PubMed Central\]](#)
23. Bollag, W.B.; Aitkens, L.; White, J.; Hyndman, K.A. Aquaporin-3 in the epidermis: More than skin deep. *Am. J. Physiol. Cell Physiol.* **2020**, *318*, C1144–C1153. [\[CrossRef\]](#) [\[PubMed\]](#) [\[PubMed Central\]](#)
24. Qin, H.; Zheng, X.; Zhong, X.; Shetty, A.K.; Elias, P.M.; Bollag, W.B. Aquaporin-3 in keratinocytes and skin: Its role and interaction with phospholipase D2. *Arch. Biochem. Biophys.* **2011**, *508*, 138–143. [\[CrossRef\]](#) [\[PubMed\]](#) [\[PubMed Central\]](#)
25. Karimi, N.; Ahmadi, V. Aquaporin Channels in Skin Physiology and Aging Pathophysiology: Investigating Their Role in Skin Function and the Hallmarks of Aging. *Biology* **2024**, *13*, 862. [\[CrossRef\]](#) [\[PubMed\]](#)
26. Mondadori, G.; Amoruso, A.; Visciglia, A.; Deusebio, G.; Pinto, D.; Pane, M.; Rinaldi, F. Heat-Treated Probiotics' Role in Counteraction of Skin UVs-Induced Damage In Vitro. *Cosmetics* **2025**, *12*, 121. [\[CrossRef\]](#)
27. Mosmann, T. Rapid colorimetric assay for cellular growth and survival: Application to proliferation and cytotoxicity assays. *J. Immunol. Methods* **1983**, *65*, 55–63. [\[CrossRef\]](#) [\[PubMed\]](#)

28. Decker, T.; Lohmann-Matthes, M.L. A quick and simple method for the quantitation of lactate dehydrogenase release in measurements of cellular cytotoxicity and tumor necrosis factor (TNF) activity. *J. Immunol. Methods* **1988**, *115*, 61–69. [[CrossRef](#)] [[PubMed](#)]
29. Schey, K.L.; Wang, Z.; Wenke, J.L.; Qi, Y. Aquaporins in the eye: Expression, function, and roles in ocular disease. *Biochim. Biophys. Acta* **2014**, *1840*, 1513–1523. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
30. Deusebio, G.; Visciglia, A.; Amoroso, A.; Pane, M. Heat-Treated Strains of *Lactiplantibacillus Plantarum* Skinbac™ SB01 and *Bifidobacterium animalis* spp. Lactis Skinbac™ SB05 Visibly Fight Aging Signs Both In Vitro and In Vivo. *Cosmetics* **2026**, *13*, 76. [[CrossRef](#)]
31. Papaccio, F.; D’Arino, A.; Caputo, S.; Bellei, B. Focus on the Contribution of Oxidative Stress in Skin Aging. *Antioxidants* **2022**, *11*, 1121. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
32. Pourzand, C.; Albieri-Borges, A.; Raczek, N.N. Shedding a New Light on Skin Aging, Iron- and Redox-Homeostasis and Emerging Natural Antioxidants. *Antioxidants* **2022**, *11*, 471. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
33. Natsch, A.; Emter, R. The specific biochemistry of human axilla odour formation viewed in an evolutionary context. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* **2020**, *375*, 20190269. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
34. Żółkiewicz, J.; Marzec, A.; Ruszczyński, M.; Feleszko, W. Postbiotics-A Step Beyond Pre- and Probiotics. *Nutrients* **2020**, *12*, 2189. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
35. Nakatsuji, T.; Chen, T.H.; Narala, S.; Chun, K.A.; Two, A.M.; Yun, T.; Shafiq, F.; Kotol, P.F.; Bouslimani, A.; Melnik, A.V.; et al. Antimicrobials from human skin commensal bacteria protect against *Staphylococcus aureus* and are deficient in atopic dermatitis. *Sci. Transl. Med.* **2017**, *9*, eaah4680. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
36. Xu, J.; Lupu, F.; Esmon, C.T. Inflammation, innate immunity and blood coagulation. *Hamostaseologie* **2010**, *30*, 5–6, 8–9. [[CrossRef](#)] [[PubMed](#)]
37. Lunjani, N.; Hlela, C.; O’Mahony, L. Microbiome and skin biology. *Curr. Opin. Allergy Clin. Immunol.* **2019**, *19*, 328–333. [[CrossRef](#)] [[PubMed](#)]
38. Woo, Y.R.; Kim, H.S. Interaction between the microbiota and the skin barrier in aging skin: A comprehensive review. *Front. Physiol.* **2024**, *15*, 1322205. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]
39. Chilicka, K.; Dzieńdziora-Urbińska, I.; Szyguła, R.; Asanova, B.; Nowicka, D. Microbiome and Probiotics in Acne Vulgaris—A Narrative Review. *Life* **2022**, *12*, 422. [[CrossRef](#)] [[PubMed](#)] [[PubMed Central](#)]

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