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MICROBIOME ALTERATIONS OF ATHLETES' SKIN: IMPACT OF OCCLUSIVE SPORTSWEAR AND SWEATING ON CUTANEOUS IMMUNITY – A NARRATIVE REVIEW

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ABSTRACT

Background: The skin microbiome is a site-specific, dynamic ecosystem tightly linked to barrier integrity and cutaneous immune regulation. Athletes are repeatedly exposed to high sweat loads, friction, and prolonged occlusion from sportswear and protective gear, alongside shared environments that facilitate microbial transfer.
Aim: To synthesize and critically evaluate evidence on how sweating and occlusive sportswear alter the athletes' skin microbiome and to discuss implications for cutaneous immunity and sport-related dermatoses.
Material and methods: This narrative review synthesizes current evidence from foundational and contemporary literature addressing skin microbiome ecology and its modulation under athletic conditions. Evidence was organized thematically across: baseline microbiome biogeography and stability, immune–microbiome interactions (including antimicrobial peptides), sweat and pH physiology, textile–microbiome interactions, and athlete-relevant clinical outcomes (MRSA colonization, dermatophytosis, acne mechanica).
Results: Sweating and heat adaptation shape skin microclimates and secretions that act as ecological filters on microbial communities. Occlusion increases hydration and temperature, perturbs pH, and can compromise barrier function—conditions that favor dysbiosis and facilitate survival and transfer of microbes via textiles. Deodorant/antiperspirant use measurably shifts axillary microbiota. Athlete-relevant risks include increased Staphylococcus aureus carriage and outbreaks in contact settings, dermatophyte transmission (tinea gladiatorum), and friction-occlusion inflammatory dermatoses (acne mechanica).
Conclusions: Sweat and occlusion synergistically create a "high-risk microenvironment" for microbiome imbalance and cutaneous immune stress. Prevention should prioritize microclimate management (rapid de-occlusion, laundering, moisture control), barrier-sparing cleansing, and sport-specific infection control while avoiding unnecessary antimicrobial pressure.

KEYWORDS

Skin Microbiome, Athletes, Sweating, Occlusive Sportswear, Cutaneous Immunity, Dermcidin, pH, Textile Microbiology, MRSA, Tinea Gladiatorum

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1. Introduction

The last decade has transformed the understanding of human skin from a passive surface to a complex ecological and immunological interface. Culture-independent mapping studies established that microbial communities differ markedly by body site and are shaped by local microenvironments, with measurable temporal patterns and individuality [3,6–9]. Foundational syntheses clarified that these microorganisms participate in immune calibration and barrier homeostasis, and that dysbiosis is associated with inflammatory and infectious dermatoses [1,2,12]. In parallel, sports medicine has long recognized that athletes experience characteristic cutaneous problems—ranging from friction injuries and follicular disorders to transmissible infections in contact sports—yet mechanistic explanations have often been fragmented across dermatology, microbiology, and sports hygiene guidance [28–33].

Athletes are a particularly informative population to study host–microbe interactions because they experience repeated, predictable perturbations of skin microclimate: intense sweating, elevated temperature, mechanical shear, and prolonged occlusion from tight sportswear and protective equipment. These factors occur in high frequency cycles (training sessions and competitions) and coincide with shared environments (locker rooms, mats, benches, protective gear), creating conditions conducive to microbial transfer and persistence on fabrics. The plausibility of a causal chain—**sweat/occlusion → microclimate shift → microbiome alteration → immune/barrier consequences → clinical dermatoses**—is strong, but the

evidence base requires careful synthesis and critical appraisal, especially given methodological variability in microbiome studies.

This narrative review integrates verified evidence on skin microbiome ecology, cutaneous immunity, sweat and pH physiology, and textile microbiology to clarify how occlusive sportswear and sweating may alter skin microbial communities and influence disease risk in athletes. Particular attention is given to practical implications: hygiene policies, sportswear choices, and microclimate management that can reduce morbidity while minimizing unnecessary antimicrobial selection pressure.

Review question: How do sweating and occlusive sportswear modify the athletes' skin microbiome and cutaneous immunity, and what evidence-based prevention strategies can be derived?

2. Material and methods

2.1. Design and scope

This manuscript is a narrative review, chosen because the available literature spans heterogeneous domains (skin microbiome biogeography, immune mechanisms, sweat physiology, textile microbiology, and athlete infection epidemiology) that are not readily comparable for meta-analysis.

2.2. Evidence base

The review is based exclusively on the 35 peer-reviewed references provided by the author (all with verified DOIs). These sources include: foundational skin microbiome mapping studies [3,6–9], fungal/bacterial topography [4], immune–microbiome mechanistic work [11–13], antimicrobial peptides in sweat (dermcidin) [14,15], commensal-derived antimicrobials relevant to pathogen control [16], pH and barrier physiology [17–19], heat adaptation and sweating physiology [20], effects of deodorants/antiperspirants on axillary microbiota [21–23], textile/sportswear microbiology and transfer [24–27], and athlete-relevant clinical outcomes (skin disease guidance, MRSA colonization, *S. aureus* carriage mechanisms, tinea gladiatorum, acne mechanica) [28–34]. A strain-level study in psoriasis is included to support the importance of within-species diversity and functional shifts [35]. One integrative microbiome paper is included as conceptual background for microbiome-health relationships across body sites [10].

2.3. Synthesis approach

Findings were organized into a thematic framework: baseline ecology and stability; immune interface and antimicrobial effectors; sweat, pH, and barrier changes under athletic conditions; occlusion and textile reservoirs; and athlete-relevant disease translation. Throughout, emphasis is placed on critical evaluation—where evidence is direct, where it is inferential, and where research gaps remain.

3. Results: thematic synthesis of the literature

3.1. Baseline skin microbiome ecology and stability: why "microclimate" matters

Skin microbiome studies consistently demonstrate that microbial communities are structured by local habitat features: moisture, sebum, pH, and exposure [1,3,6–9]. The classic topographical study by Grice and colleagues showed that bacterial diversity varies strongly by body site and that the same individual carries distinct site-specific communities [3]. Subsequent work emphasized that individuality and geography shape not only taxonomic composition but also functional potential of the skin metagenome [7]. The axilla, groin, and other moist regions—precisely where athletes often experience occlusion and sweating—tend to support taxa adapted to higher humidity, including *Corynebacterium* and *Staphylococcus* species [1,2,7].

Temporal behavior is central to understanding athletes because training imposes recurring perturbations. Longitudinal analyses in healthy individuals show that the skin microbiome can be relatively stable over time at the community level, yet includes meaningful fluctuations and site-dependent variability [6]. Stability should not be interpreted as immutability: the microbiome exhibits resilience (recovery after perturbation) and plasticity (shift under sustained environmental change). Importantly, the skin is also a mixed bacterial–fungal ecosystem. Findley and colleagues demonstrated that fungal communities have distinct topographical patterns and interact with bacterial ecology, implying that athlete-related moisture and occlusion could affect both domains [4].

Age and developmental stage also shape microbiome patterns. A study comparing healthy children and adults documented shifts in skin and nares microbiota across age groups, reinforcing that host physiology and behavior influence microbial communities [5]. While athletes are often young adults, sports cohorts include adolescents and older athletes; thus, age-related baseline variation may modify the impact of sports exposures.

Critical evaluation: The strongest evidence in this section is descriptive and ecological: it explains what "normal" looks like and why microclimate shifts can plausibly alter microbial communities. However, it does not, by itself, prove that athletic exposures cause clinically important dysbiosis. The translation step requires integrating immune mechanisms, barrier physiology, and known athlete dermatoses.

3.2. Immune–microbiome interactions: commensals as regulators of cutaneous immunity

Mechanistic studies demonstrate that skin commensals shape immune function in compartmentalized and context-dependent ways. Naik and colleagues showed that resident commensals can control skin immunity in localized compartments, underscoring that immune modulation is not uniform across the body surface [11]. Linehan and colleagues further demonstrated that non-classical immunity controls microbiota impact on skin immunity and tissue repair, linking microbial presence to wound repair pathways—highly relevant in athletes who experience microtrauma and barrier disruption [13]. These studies establish a causal direction: microbes influence immune responses and tissue outcomes.

Sanford and Gallo summarized key functional roles of the skin microbiota in health and disease, including colonization resistance, immune education, and modulation of inflammatory thresholds [12]. This framework is crucial when considering athletes: repeated physical stressors may not only change which microbes are present but also alter the "tone" of immune responsiveness, potentially shifting the balance between tolerance (to commensals) and inflammation (to tissue stress and opportunists).

An important immune effector is the production of antimicrobials—both host-derived AMPs and microbe-derived antimicrobials. Nakatsuji and colleagues showed that human skin commensals produce antimicrobials that protect against *Staphylococcus aureus*, and that these protective functions are deficient in atopic dermatitis [16]. Although athletes are not necessarily AD patients, the study provides a mechanistic template: perturbations that reduce commensal antimicrobial capacity can open ecological space for pathogens. In sports settings where *S. aureus* exposure is high, maintaining commensal-mediated defense may be especially important.

Critical evaluation: These mechanistic papers provide strong causal evidence that commensals shape immunity and tissue repair, and that commensal antimicrobial capacity can constrain *S. aureus* [11–13,16]. The limitation is external validity: controlled experimental settings differ from athlete conditions. Nevertheless, the mechanisms plausibly generalize to any context where barrier disruption and pathogen exposure occur, including sport.

3.3. Sweat and heat adaptation: an ecological and immunological filter

Sweating is among the defining physiological features of sport exposure. Heat adaptation influences sweating capacity and thermoregulation; Taylor's review on human heat adaptation provides a physiological backdrop for understanding variability in sweat rate and composition across athletes and environments [20]. From a microbiome standpoint, sweat changes hydration, solute concentration, and nutrient availability on the skin surface; from an immune standpoint, sweat delivers antimicrobial peptides and can influence pH.

A landmark discovery relevant to sweating is dermcidin, an antibiotic peptide secreted by sweat glands [14]. Dermcidin-derived peptides exhibit antimicrobial activity and represent a direct link between sweat physiology and innate defense. Rieg and colleagues' work (as cited in the reference set) further supports the relevance of dermcidin-derived peptides to sweat antimicrobial function [15]. In athletes, increased sweating could increase dermcidin delivery, suggesting a potential protective effect; however, athletes simultaneously experience prolonged moisture, occlusion, and friction, which may offset or override antimicrobial benefits by promoting maceration, follicular occlusion, and microinjury.

Sweat also interacts with skin pH and barrier. While sweat is often slightly acidic to neutral depending on conditions, prolonged moisture and occlusion can shift surface pH, and cleansing practices post-exercise can further alter pH and lipid organization. Because pH is a major determinant of enzyme activity involved in barrier maintenance, persistent deviation from physiologic acidity may weaken barrier function and alter microbial selection [17–19].

Critical evaluation: The evidence linking sweat to antimicrobial defense via dermcidin is robust [14,15]. However, direct athlete studies that measure sweat composition, AMP activity, pH, and microbiome changes together are scarce within this reference set. Thus, the review necessarily synthesizes mechanistic plausibility and indirect evidence from related contexts.

3.4. Skin pH and barrier integrity under occlusion and repeated cleansing

The acid mantle is a core feature of skin barrier physiology and microbial ecology. Schmid-Wendtner and Korting reviewed how skin surface pH impacts barrier function, emphasizing that pH modulates lipid processing enzymes and antimicrobial activity [17]. Lambers and colleagues provided empirical evidence that natural skin surface pH is on average below 5, reinforcing that acidity is a physiological baseline rather than a niche phenomenon [18]. Proksch and colleagues highlighted the skin as an indispensable barrier, with structural and biochemical features that prevent transepidermal water loss and limit microbial invasion [19].

Athletes face repetitive cycles of sweating followed by cleansing. Both the sweat episode and the cleansing episode can influence pH and lipids. Post-exercise hygiene practices vary widely: some athletes use strong soaps or frequent antiseptics, while others delay washing due to training schedules. The athlete-specific consequence is that pH fluctuations and lipid disruption may become chronic rather than transient, potentially predisposing to irritation, inflammatory dermatoses, and altered microbial communities.

In the axilla and groin, occlusion from tight garments and protective equipment can increase hydration and temperature, which can weaken barrier integrity mechanically and biochemically. Elevated hydration can soften the stratum corneum, increasing susceptibility to friction damage and microfissures. These barrier disruptions can intensify immune activation and influence microbial competitiveness, potentially increasing the probability of colonization by opportunists when exposure is high.

Critical evaluation: The pH and barrier evidence is strong and generalizable [17–19]. The key limitation is that direct athlete intervention data linking pH manipulation to disease reduction are limited in this set. Still, the mechanistic direction supports barrier-sparing hygiene and microclimate management as rational preventive strategies.

3.5. Deodorants, antiperspirants, and the axillary microbiome: a modifiable factor in athletes

Athletes frequently use deodorants and antiperspirants to manage odor and sweating, particularly in team settings. These products can alter axillary microclimate and microbial communities. Urban and colleagues showed that habitual and experimental antiperspirant/deodorant use affects the armpit microbiome, providing evidence that topical product use can reshape microbial communities in a real-world manner [21]. Callewaert and colleagues similarly demonstrated that deodorants and antiperspirants affect the axillary bacterial community [22]. A more recent review of trends in active agents in deodorants and antiperspirants outlines evolving formulations and antimicrobial strategies, relevant to modern athlete product use [23].

From an ecological perspective, antiperspirants reduce sweat availability and change humidity and salt composition, potentially shifting selection pressures. Deodorants often include antimicrobial components that may suppress odor-associated taxa but may also reduce commensal diversity. Whether these changes are beneficial or harmful depends on context. In athletes, reduced sweat may decrease moisture-driven dysbiosis in the axilla, but antimicrobial pressure may also select for resistant organisms or alter commensal protection against pathogens.

Critical evaluation: These studies provide direct evidence that axillary microbiota are modifiable and that products can meaningfully shift community composition [21–23]. However, clinical endpoints (infection rates, dermatitis incidence) are not consistently measured. For athletes, product choice should be individualized and balanced against risks of irritation and microbial disruption, especially in those prone to folliculitis or contact dermatitis.

3.6. Sportswear, occlusion, and textiles as microbial reservoirs and transfer vectors

A central athlete-specific factor is the role of textiles. The iScience review on sweat and odor in sportswear emphasizes that textiles interact with sweat chemistry and microbes, shaping malodor formation and microbial persistence [24]. Importantly, malodor is not merely cosmetic; it is a functional signature of microbial metabolism on fabric. Persistence of odor after laundering suggests persistence of microbial communities or metabolites, raising concern that inadequately cleaned sportswear can serve as a recurring source of exposure and reinoculation.

Sanders and colleagues synthesized the microbiology of clothing, describing how garments accumulate microbes from skin and environment, and how fabric type, humidity, and laundering influence microbial load and community composition [25]. Procopio and colleagues examined transferability of the human and environmental microbiome on clothes, indicating that clothing can carry signatures and facilitate microbial transfer across contexts [26]. Callewaert and colleagues' work on skin microbiome–textile interactions further

supports the concept that textiles and skin form an integrated system where microbial exchange and selection pressures occur bidirectionally [27].

For athletes, these findings imply that sportswear is not neutral. Tight synthetic garments often increase occlusion and moisture retention, while repeated use (especially with incomplete drying between sessions) can promote microbial persistence. Additionally, shared equipment (pads, helmets, mats) may act similarly to textiles in storing and transferring microbes. The implication is that laundering practices, drying time, and fabric choice can be legitimate targets for disease prevention.

Critical evaluation: The textile evidence supports reservoir and transfer mechanisms [24–27], but translating this to quantified infection risk in athletes remains challenging. Many textile studies rely on DNA-based measures that do not directly assess viability. Moreover, laundering conditions vary widely. Nonetheless, the mechanistic coherence and repeated observations justify practical recommendations for laundering and microclimate control.

3.7. Athlete-relevant infectious outcomes: MRSA, *Staphylococcus aureus* carriage, and tinea gladiatorum

Athletes, particularly in contact sports, experience higher risk of transmissible skin infections. Zinder and colleagues' position statement remains a key reference for identification, management, and prevention of skin diseases in athletic settings [28]. These guidelines emphasize early recognition, covering lesions, hygiene protocols, and exclusion policies to prevent outbreaks.

MRSA colonization and infection in athletes has been studied systematically. Karanika and colleagues conducted a meta-analysis on MRSA colonization among athletes, supporting that athletes can be asymptomatic carriers and that carriage relates to infection risk and outbreak potential [29]. Understanding carriage requires a mechanistic framework: Kluytmans and colleagues reviewed nasal carriage of *S. aureus*, outlining epidemiology, mechanisms, and risks, which can be extrapolated to athletic settings where close contact and microtrauma facilitate transmission and invasion [30].

Fungal infections are also prominent in contact sports. Zalewski and colleagues reviewed tinea gladiatorum, a dermatophytosis prevalent in wrestlers and other grappling athletes, emphasizing epidemiology, clinical presentation, and management strategies [31]. Moisture, occlusion, and skin-to-skin contact create ideal conditions for dermatophyte transmission. The microbiome lens adds nuance: barrier integrity and commensal competition may influence fungal colonization, while textiles and shared mats can serve as reservoirs.

Critical evaluation: The athlete infection literature is clinically grounded and directly relevant [28–31]. However, linking these infections to specific microbiome states remains an emerging field. Most infection studies focus on pathogen carriage rather than full community ecology. Future work should integrate pathogen surveillance with microbiome profiling and barrier measures to identify predictive signatures of outbreak risk.

3.8. Friction-occlusion inflammatory dermatoses: acne mechanica and environmental acne drivers

Beyond infections, athletes commonly experience friction-occlusion dermatoses. Acne mechanica is a classic entity associated with repetitive mechanical stress, heat, and occlusion. The original description by Kligman and Mills established the concept and remains relevant for modern sports where helmets, straps, pads, and tight garments create persistent pressure and friction [32]. More recent work in pediatric dermatology highlights that friction-related lesions and acne mechanica can occur in young athletes, emphasizing the relevance to adolescent sports populations [33]. Environmental factors in acne—including occlusion, humidity, and mechanical stress—have been reviewed, providing a broader framework for acne exacerbation outside classical hormonal drivers [34].

Mechanistically, occlusion and friction can promote microcomedone formation, disrupt follicular keratinization, and increase local inflammation. Sweat and trapped heat can intensify these effects by increasing hydration and swelling of the stratum corneum. Microbiome changes may contribute via shifts in follicular microenvironment and microbial metabolic activity, though direct athlete-specific follicular microbiome studies are limited within this reference set.

Critical evaluation: The acne mechanica evidence is strong clinically and mechanistically [32–34]. The main gap is direct integration with contemporary microbiome sequencing in athletes. Nonetheless, the available data support preventive strategies focused on reducing friction/occlusion and optimizing microclimate.

3.9. Why strain-level and functional diversity matters: lessons from inflammatory skin disease

Microbiome research increasingly shows that disease associations may be driven by strain-level differences rather than genus-level shifts. Tett and colleagues demonstrated unexplored diversity and strain-level structure in the skin microbiome associated with psoriasis, highlighting that functionally relevant shifts can occur beneath the level of broad taxonomy [35]. For athletes, this matters because outbreak risk and transmissibility may depend on specific *S. aureus* strains or toxin profiles rather than mere presence of *S. aureus*.

Similarly, atopic dermatitis studies show that microbial communities shift during flares and treatment [8]. While athletes may not have AD, these studies illustrate that inflammatory states and barrier disruption can remodel microbiome structure dynamically, and that treatment can drive rapid shifts. This supports a general model: **barrier stress (from friction/occlusion) + microclimate change (sweat/heat) → community and functional shifts → altered immune responses and disease susceptibility**.

4. Discussion

4.1. An integrated model for athletes: the sweat–occlusion–friction triad

Synthesizing the evidence, athletes repeatedly experience a triad that plausibly drives microbiome and immune shifts:

1. **Sweat and heat exposure** (influenced by heat adaptation) alters hydration, salt concentration, nutrient availability, and delivers AMPs such as dermcidin [14,15,20].
2. **Occlusion from sportswear/protective gear** traps sweat and heat, prolongs high humidity, and modifies pH dynamics—conditions known to influence barrier enzymes and microbial selection [17–19,24].
3. **Friction and microtrauma** compromise barrier integrity, increase inflammation, and create microfissures that facilitate pathogen invasion and dysbiosis [19,32–34].

These factors are synergistic rather than additive. Occlusion amplifies the impact of sweat by prolonging moisture and heat, while friction amplifies the impact of both by damaging the barrier in a hydrated, softened state. The microbiome response is predicted to be site-specific: axilla/groin/trunk under tight clothing may shift toward moist-site communities and higher microbial activity, while exposed dry sites may remain more stable [1,3,6,7].

4.2. Protective vs harmful effects of sweat: a nuanced view

Sweat is often viewed as a risk factor because it increases moisture and maceration. Yet sweat also contains dermcidin, a host defense peptide with antimicrobial activity [14]. The net effect depends on context. In a well-ventilated environment with rapid drying and intact barrier, sweat may support defense and transiently suppress microbial overgrowth. Under occlusion, sweat's protective effects can be undermined because high humidity and heat promote microbial growth and biofilm formation on skin and textiles, and pH changes may reduce barrier resilience [17–19,24–27]. Thus, sweat is best conceptualized as a **dual-role ecological factor**: protective in physiology, but potentially pathogenic in microclimate-trapped environments.

4.3. Textiles as intervention targets: beyond "clean clothes"

Textile microbiology suggests that sportswear can be both reservoir and selective surface [25–27]. The iScience review highlights that fabric properties and laundering influence microbial persistence and malodor production [24]. For athletes, this supports concrete intervention levers:

- **De-occlusion and drying:** immediate changing after training reduces microclimate duration, limiting selection for moisture-adapted taxa.
- **Laundering efficacy:** sufficient temperature, detergent selection, and full drying may reduce viable microbial load and residual metabolites that sustain odor and microbial survival.
- **Fabric selection:** moisture-wicking, breathable fabrics may reduce humidity duration; however, "antimicrobial fabrics" should be approached cautiously due to potential selection pressures and skin irritation.

Current evidence supports textiles as a meaningful component of the exposure pathway but does not yet quantify which textile interventions yield the greatest clinical benefit. The lack of athlete-specific randomized trials is a major gap.

4.4. Hygiene and topical products: microbiome-sparing strategies

Athletes often increase washing frequency. While hygiene is essential for preventing transmission (especially in contact sports), repeated harsh cleansing can disrupt the acid mantle and barrier lipids [17–19]. Axillary product studies show that deodorants/antiperspirants can shift the microbiome [21–23]. This suggests that "more antimicrobial" is not always better. In teams, evidence-based infection control should be paired with barrier-sparing practices: gentle cleansers, avoidance of unnecessary antiseptics, and targeted measures for outbreaks rather than indiscriminate antimicrobial exposure.

A practical framework is **risk stratification**:

- High-risk settings (wrestling, judo, rugby, shared mats): prioritize lesion screening, infection-specific protocols, and strict equipment disinfection [28,31].
- Lower-risk endurance sports: prioritize microclimate management (moisture/friction reduction), textile hygiene, and prevention of acne mechanica [24,32–34].

4.5. Clinical translation: how microbiome science informs return-to-play

Sports medicine needs actionable criteria. The NATA position statement provides return-to-play considerations for common skin diseases [28]. Microbiome science refines prevention: rather than focusing only on treating disease once it occurs, it supports upstream strategies that reduce dysbiosis triggers—prolonged occlusion, persistent humidity, and barrier damage.

For MRSA, meta-analytic evidence supports the relevance of colonization among athletes [29]. Integrating carriage concepts [30] with textile reservoir mechanisms [25–27] suggests that outbreak control must address both person-to-person transmission and environmental reservoirs (clothing, towels, shared equipment). For tinea gladiatorum, the dermatology literature emphasizes rapid treatment and environmental control [31]; microbiome-aware strategies (drying, barrier protection, laundering) are complementary.

4.6. Research gaps and future directions

Key gaps emerge from this synthesis:

1. **Integrated measurements:** Athlete studies should combine microbiome sequencing with sweat metrics (rate, composition), dermcidin/AMP profiling, skin pH, and barrier measures (e.g., transepidermal water loss). Current evidence often isolates one domain [14,17,24].
2. **Strain-level resolution:** Strain-level analysis may better predict pathogen transmission and inflammation risk, as highlighted in inflammatory skin disease research [35].
3. **Textile intervention trials:** Randomized or pragmatic trials comparing laundering protocols, fabric types, and changing behaviors with clinical endpoints (folliculitis, tinea, *S. aureus* carriage) are needed [24–27].
4. **Sport-specific risk models:** Different sports have distinct exposure profiles. Contact sports require transmission-focused research; endurance sports require friction-occlusion and microclimate studies [28,31–34].
5. **Balance between antimicrobial control and microbiome preservation:** Future guidelines should weigh infection prevention against potential harms of excessive antimicrobial topical exposure, using axillary microbiome data as precedent [21–23].

5. Conclusions

Athletic training exposes the skin to repeated cycles of sweating, heat, occlusion, and friction that can reshape local microclimates and plausibly shift the skin microbiome. Mechanistic evidence shows that commensals regulate cutaneous immunity and tissue repair [11–13], that sweat provides antimicrobial defense through dermcidin [14,15], and that barrier integrity and acidic pH are central to microbial selection and host protection [17–19]. Sportswear and textiles represent critical, modifiable reservoirs and vectors for microbial persistence and transfer [24–27]. Clinically, these mechanisms align with athlete-relevant conditions: increased risk of *S. aureus*/MRSA carriage and outbreaks in contact settings [28–30], dermatophyte transmission such as tinea gladiatorum [31], and friction-occlusion dermatoses including acne mechanica [32–34].

Practical prevention should target microclimate duration and barrier preservation: rapid de-occlusion, effective laundering and drying, thoughtful fabric choices, gentle cleansing, and sport-specific infection control protocols. Future research must integrate microbiome profiling with functional immune and barrier measures and apply strain-level methods to predict infection and inflammation risk more precisely.

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REFERENCES

1. Grice, E. A., & Segre, J. A. (2011). The skin microbiome. *Nature Reviews Microbiology, 9*(4), 244–253. https://doi.org/10.1038/nrmicro2537
2. Byrd, A. L., Belkaid, Y., & Segre, J. A. (2018). The human skin microbiome. *Nature Reviews Microbiology, 16*, 143–155. https://doi.org/10.1038/nrmicro.2017.157
3. Grice, E. A., Kong, H. H., Conlan, S., et al. (2009). Topographical and temporal diversity of the human skin microbiome. *Science, 324*(5931), 1190–1192. https://doi.org/10.1126/science.1171700
4. Findley, K., Oh, J., Yang, J., et al. (2013). Topographic diversity of fungal and bacterial communities in human skin. *Nature, 498*(7454), 367–370. https://doi.org/10.1038/nature12171
5. Oh, J., Conlan, S., Polley, E. C., Segre, J. A., & Kong, H. H. (2012). Shifts in human skin and nares microbiota of healthy children and adults. *Genome Medicine, 4*(10), 77. https://doi.org/10.1186/gm378
6. Oh, J., Byrd, A. L., Park, M., Kong, H. H., & Segre, J. A. (2016). Temporal stability of the human skin microbiome. *Cell, 165*(4), 854–866. https://doi.org/10.1016/j.cell.2016.04.008
7. Oh, J., Byrd, A. L., Deming, C., et al. (2014). Biogeography and individuality shape function in the human skin metagenome. *Nature, 514*, 59–64. https://doi.org/10.1038/nature13786
8. Kong, H. H., Oh, J., Deming, C., et al. (2012). Temporal shifts in the skin microbiome associated with disease flares and treatment in children with atopic dermatitis. *Genome Research, 22*, 850–859. https://doi.org/10.1101/gr.131029.111
9. Costello, E. K., Lauber, C. L., Hamady, M., et al. (2009). Bacterial community variation in human body habitats across space and time. *Science, 326*(5960), 1694–1697. https://doi.org/10.1126/science.1177486
10. Clemente, J. C., Ursell, L. K., Parfrey, L. W., & Knight, R. (2012). The impact of the gut microbiota on human health: An integrative view. *Science, 336*(6086), 1262–1267. https://doi.org/10.1016/j.cell.2012.01.035
11. Naik, S., Bouladoux, N., Wilhelm, C., et al. (2012). Compartmentalized control of skin immunity by resident commensals. *Science, 337*(6098), 1115–1119. https://doi.org/10.1126/science.1225152
12. Sanford, J. A., & Gallo, R. L. (2013). Functions of the skin microbiota in health and disease. *Seminars in Immunology, 25*(5), 370–377. https://doi.org/10.1016/j.smim.2013.09.005
13. Linehan, J. L., Harrison, O. J., Han, S. J., et al. (2018). Non-classical immunity controls microbiota impact on skin immunity and tissue repair. *Cell, 172*(4), 784–796.e18. https://doi.org/10.1016/j.cell.2017.12.033
14. Schittek, B., Hipfel, R., Sauer, B., et al. (2001). Dermcidin: A novel human antibiotic peptide secreted by sweat glands. *Nature Immunology, 2*(12), 1133–1137. https://doi.org/10.1038/ni732
15. Rieg, S., Steffen, H., Seeber, S., et al. (2005). Deficiency of dermcidin-derived antimicrobial peptides in sweat. *The Journal of Immunology, 174*(12), 8003–8010. https://doi.org/10.4049/jimmunol.174.12.8003
16. Nakatsuji, T., Chen, T. H., Narala, S., et al. (2017). Antimicrobials from human skin commensal bacteria protect against *Staphylococcus aureus* and are deficient in atopic dermatitis. *Science Translational Medicine, 9*. https://doi.org/10.1126/scitranslmed.aah4680

17. Schmid-Wendtner, M. H., & Korting, H. C. (2006). The pH of the skin surface and its impact on the barrier function. *Skin Pharmacology and Physiology*. https://doi.org/10.1159/000094670
18. Lambers, H., Piessens, S., Bloem, A., Pronk, H., & Finkel, P. (2006). Natural skin surface pH is on average below 5. *International Journal of Cosmetic Science*. https://doi.org/10.1111/j.1467-2494.2006.00344.x
19. Proksch, E., Brandner, J. M., & Jensen, J. M. (2008). The skin: An indispensable barrier. *Experimental Dermatology*. https://doi.org/10.1111/j.1600-0625.2008.00786.x
20. Taylor, N. A. S. (2014). Human heat adaptation. *Comprehensive Physiology*. https://doi.org/10.1002/cphy.c130022
21. Urban, J., Fergus, D. J., Savage, A. M., et al. (2016). The effect of habitual and experimental antiperspirant and deodorant product use on the armpit microbiome. *PeerJ, 4*, e1605. https://doi.org/10.7717/peerj.1605
22. Callewaert, C., Hutapea, P., & Van de Wiele, T. (2014). Deodorants and antiperspirants affect the axillary bacterial community. *Archives of Dermatological Research, 306*(8), 701–710. https://doi.org/10.1007/s00403-014-1487-1
23. Teerasumran, P., et al. (2023). Deodorants and antiperspirants: New trends in active agents. *International Journal of Cosmetic Science*. https://doi.org/10.1111/ics.12852
24. Chang, Y., et al. (2023). Sweat and odor in sportswear (review). *iScience, 26*, 107067. https://doi.org/10.1016/j.isci.2023.107067
25. Sanders, D., Grunden, A., & Dunn, R. R. (2021). A review of clothing microbiology: The history of clothing and the role of microbes in textiles. *Biology Letters, 17*(1), 20200700. https://doi.org/10.1098/rsbl.2020.0700
26. Procopio, R. E., et al. (2024). Transferability of the human and environmental microbiome on clothes. *Genes, 15*(3), 375. https://doi.org/10.3390/genes15030375
27. Broadhead, R., Craeye, L., & Callewaert, C. (2021). The future of functional clothing for an improved skin and textile microbiome relationship. *Microorganisms, 9*(6), 1192. https://doi.org/10.3390/microorganisms9061192
28. Zinder, S. M., Basler, R. S. W., Foley, J., Scarlata, C., & Vasily, D. B. (2010). National Athletic Trainers' Association position statement: Skin diseases. *Journal of Athletic Training, 45*(4), 411–428. https://doi.org/10.4085/1062-6050-45.4.411
29. Karanika, S., Kinamon, T., Grigoras, C., & Mylonakis, E. (2016). Colonization with MRSA among athletes. *Clinical Infectious Diseases*. https://doi.org/10.1093/cid/ciw240
30. Kluytmans, J., van Belkum, A., & Verbrugh, H. (1997). Nasal carriage of *Staphylococcus aureus*: Epidemiology, mechanisms, risks. *Clinical Microbiology Reviews*. https://doi.org/10.1128/CMR.10.3.505
31. Zalewski, P., et al. (2022). Tinea gladiatorum: Epidemiology and clinical aspects. *Journal of Clinical Medicine, 11*(14), 4066. https://doi.org/10.3390/jcm11144066
32. Kligman, A. M., & Mills, O. H. (1975). Acne mechanica. *Archives of Dermatology*. https://doi.org/10.1001/archderm.1975.01630160071007
33. Mazhar, M., Simpson, M., & Marathe, K. (2019). Inner thigh friction as a cause of acne mechanica. *Pediatric Dermatology, 36*(4), 546–547. https://doi.org/10.1111/pde.13817
34. Yang, J., Yang, H., Xu, A., & He, L. (2020). A review of advancement on influencing factors of acne: An emphasis on environment characteristics. *Frontiers in Public Health, 8*, 450. https://doi.org/10.3389/fpubh.2020.00450
35. Tett, A., et al. (2017). Unexplored diversity and strain-level structure of the skin microbiome associated with psoriasis. *npj Biofilms and Microbiomes*. https://doi.org/10.1038/s41522-017-0022-5