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SKIN BARRIER DYSFUNCTION IN ENDURANCE ATHLETES: A NARRATIVE REVIEW OF MECHANISMS, ENVIRONMENTAL STRESSORS, AND CLINICAL IMPLICATIONS

Abstract: The skin of the endurance athlete functions as a load-bearing, evaporative, antimicrobial, and photoprotective interface that is repeatedly stressed during prolonged training and competition. This narrative review examines how endurance-training exposures disturb the skin barrier and translate into clinical skin disease in runners, ultramarathoners, triathletes, and cyclists. PubMed, PubMed Central, and publisher sources were searched for athlete-specific and mechanistic skin-barrier literature, with priority given to work published from 2016 onward and evidence organised by mechanism and exposure rather than by diagnosis. Prolonged, repetitive mechanical loading, sweat and hyperhydration, occlusion by clothing and equipment, ultraviolet radiation, and thermal and water exposure, together with transient post-exercise shifts in surface pH, lipids, antimicrobial peptides, and microbial ecology, generate a recurrent pattern of friction blisters, chafing, intertrigo,

folliculitis, saddle sores, tinea, irritant and contact dermatitis, xerosis, sunburn, and cumulative photodamage rather than a single uniform disorder. Effective prevention is mechanism-based, combining sport-specific friction control, rapid removal of wet garments, gentle cleansing, barrier-supportive moisturisation, early recognition of infection, and redundant photoprotection. Prospective studies linking objective barrier biomarkers to clinical outcomes are needed to individualise prevention.

Keywords: endurance athletes; skin barrier; stratum corneum; transepidermal water loss; sweat; chafing; friction blisters; saddle sores; microbiome; photoprotection

1. Methods

This article is a narrative review rather than a PRISMA-based systematic review. It addresses how endurance-training exposures alter the physical, chemical, immunologic, and microbial components of the skin barrier, and how those changes translate into clinically relevant skin problems in runners, ultramarathoners, triathletes, cyclists, and comparable endurance populations. PubMed, PubMed Central, publisher websites, and the reference lists of relevant articles were searched using combinations of terms covering the skin barrier, endurance disciplines, sweat, transepidermal water loss, stratum-corneum hydration, surface pH, ceramides, natural moisturizing factor, antimicrobial peptides, the microbiome, and the principal sports dermatoses, including friction blisters, chafing, intertrigo, saddle sores, tinea, folliculitis, and photodamage. Priority was given to work published from 2016 onward, while older studies were retained when they remained foundational.

Because endurance dermatology spans both direct athlete studies and indirect skin-barrier science, evidence was synthesised on two levels: direct evidence from race cohorts, runner surveys, athlete physiology studies, cyclist saddle-sore literature, and sport-related infection reviews; and indirect evidence from stratum-corneum lipid biology, barrier-measurement methodology, moisturiser mechanisms, microbiome ecology, and photobiology, drawn upon only where the mechanism was biologically relevant to endurance exposures. The synthesis is organised by mechanism and exposure rather than by diagnosis, because blisters, chafing, folliculitis, saddle sores, tinea, dermatitis, and sunburn frequently coexist in the same athlete through shared upstream drivers such

as shear, wetness, heat, occlusion, altered pH, and delayed barrier recovery. As a narrative review it does not pool prevalence estimates or grade certainty with GRADE; it provides a clinically oriented synthesis and a research agenda rather than definitive estimates of incidence, treatment efficacy, or biomarker thresholds for every endurance sport.

2. Introduction

The skin of an endurance athlete is not merely a covering surface. During long training sessions and races it becomes a load-bearing interface, an evaporative cooling organ, an antimicrobial boundary, and a photoprotective tissue that must function under moving clothing, wet shoes, helmets, gloves, wetsuits, saddle pressure, wind, heat, cold, and sunlight. The stratum corneum provides the main permeability barrier through corneocytes embedded in organized extracellular lipids, while surface acidity, natural moisturizing factor, antimicrobial peptides, and the microbiome contribute to chemical and immune defense (1,2,3).

Endurance sport stresses these systems in a distinctive way because the exposure is prolonged and repetitive. A sprinter may experience high force for seconds, but a marathoner, ultrarunner, or cyclist may expose the same anatomical sites to thousands of shear cycles, prolonged sweat film formation, prolonged occlusion, and repeated UV exposure before the athlete can wash, dry, cool, or change clothing. The clinical result is not one disease but a recurrent pattern of barrier insults that can manifest as friction blisters, chafing, jogger's nipple, intertrigo, folliculitis, acne mechanica, tinea pedis, irritant dermatitis, saddle sores, sunburn, and actinic damage (4,5,6).

Available prevalence data support the clinical importance of this topic. In a cross-sectional study of road runners in Southern Brazil, the most prevalent sports-related lesions were blisters, chafing, calluses, nail shedding, tinea pedis, ingrown nails, and cheilitis, and several conditions increased with higher weekly mileage (4). A recent long-distance-running review similarly emphasized traumatic, environmental, infectious, inflammatory, and UV-related skin disorders as recurring problems in distance runners (6).

Endurance events also expose athletes to acute medical encounters in which apparently minor skin problems can influence race completion, gait, equipment tolerance, sleep, and infection risk. In large road-race surveillance, dermatologic complaints are not usually the leading life-threatening

events, but they form part of the broader spectrum of musculoskeletal, heat, fluid, and environmental problems that medical teams encounter during endurance races (7).

The barrier-dysfunction frame is useful because it connects prevention to mechanism. A friction blister is not merely a superficial bubble; it reflects repetitive shear deformation through layers of skin, often intensified by moisture, heat, and footwear interface problems. Chafing is not merely soreness; it is repeated abrasion of an already wet or salt-contaminated surface. Intertrigo and folliculitis are not only infections; they are enabled by maceration, occlusion, altered pH, and microtrauma that change microbial opportunity (8,9,10).

The field has an important evidence gap. Dermatology has mature methods for measuring TEWL, hydration, pH, corneometry, impedance, NMF, lipids, cytokines, antimicrobial peptides, and microbial communities, yet endurance studies often stop at questionnaires, clinical observation, or small short-term exercise experiments. As a result, practical guidance must combine athlete-specific clinical evidence with mechanistic skin science until prospective biomarker studies become available (11,12,13).

3. Normal Skin Barrier Functions Relevant to Endurance Exercise

The physical barrier of the skin is centered in the stratum corneum, where flattened corneocytes provide mechanical resilience and extracellular lipids create lamellar structures that limit uncontrolled water loss and entry of irritants or microbes. Ceramides, cholesterol, and free fatty acids are the major lipid classes of this matrix, and changes in lipid composition or organization can weaken permeability barrier function (1,2).

Endurance athletes repeatedly challenge this physical barrier through shear, bending, pressure, stretch, and impact. In running, the plantar skin and toes experience repetitive loading inside footwear, while the nipples, axillae, groin, waistband, bra line, and inner thighs experience textile-skin or skin-skin friction. In cycling, the perineal and gluteal skin experiences a combined pressure, friction, and humidity environment at the saddle interface (4,14).

The chemical barrier includes the acidic surface pH, sweat constituents, sebum lipids, enzymes involved in desquamation, and NMF components that help maintain corneocyte flexibility. A relatively acidic surface supports barrier homeostasis and influences microbial ecology, whereas

elevation of skin-surface pH can impair enzymes and favor microbial shifts in susceptible environments (3,11).

The immunologic barrier includes resident immune cells, antimicrobial peptides, secretory molecules, and rapid epithelial responses to injury. Endurance exercise may transiently alter local defense markers: in a cycling exercise model, high-intensity endurance exercise reduced skin secretory IgA and changed staphylococcal counts and human beta-defensin 2, suggesting a short-term disturbance of epidermal barriers against microbial invasion (15).

The microbial barrier is increasingly understood as a functional component rather than a passive contaminant. The skin microbiome differs across dry, moist, and sebaceous regions, and commensal organisms interact with host immunity, antimicrobial peptides, pH, lipids, and moisture. Athletic activity can alter these microenvironments through sweating, contact surfaces, shared facilities, clothing, and repeated washing (16,13).

These barrier dimensions overlap during endurance activity. For example, a wet toe box changes mechanical friction, hydration, pH, thermal conditions, and microbial growth opportunity at the same time. A salt-contaminated sports bra line produces abrasive crystals, local inflammation, occlusion, and delayed drying. A hot cycling bib creates shear and pressure while also altering humidity and microbial density at follicular units (4,14,5).

The concept of barrier reserve is clinically helpful. A healthy athlete may tolerate a long run without symptoms when mileage, equipment, heat, humidity, UV exposure, and recovery are moderate. The same athlete may develop blisters, chafing, folliculitis, or dermatitis when multiple subclinical insults stack: new shoes, wet socks, higher training volume, hot weather, salt accumulation, delayed showering, or a prior irritant reaction (9,10).

4. Exercise-Induced Changes in Skin Hydration, pH, Sebum, and Immune Markers

Direct studies in athletes and exercise models suggest that a single session can measurably alter surface physiology. In a quantitative evaluation of 60 athletes, sportive activity was associated with increased stratum-corneum hydration, higher skin-surface pH, and changes in sebum content, which the authors linked to increased sweat production during activity (17).

Exercise-induced sweating can also change facial biophysical parameters in non-athlete populations. Wang and colleagues reported that sweating after exercise affected facial sebum,

stratum-corneum hydration, and skin-surface pH, supporting the idea that sweat is both a cooling fluid and a transient modifier of the cutaneous surface environment (18).

The direction of hydration change is not always simple. Immediately after exercise, surface and stratum-corneum water content may rise because sweat hydrates the outer layers, but later evaporation, washing, wind, or low ambient humidity can leave the skin feeling tight or dry. Eda and colleagues observed changing moisture values after high-intensity endurance exercise, with site-specific post-exercise patterns and concurrent changes in immune markers (15).

TEWL is attractive for sports research because it estimates water flux through the skin and is commonly used as an indicator of barrier integrity. However, TEWL is sensitive to room temperature, humidity, recent washing, product use, sweating, acclimatization, body site, and measurement technique; these issues are especially important in athletes who arrive after training or competition (12).

Electrical impedance spectroscopy, corneometry, pH measurement, and tape-stripping assays may complement TEWL, but they do not remove the need for strict protocols. A useful endurance study should standardize time since exercise, time since showering, product use, acclimatization period, body site, ambient temperature, humidity, and measurement sequence; otherwise, the recorded value may reflect the measurement context more than the athlete baseline (3,12).

Changes in pH have special clinical relevance because microbial ecology and barrier enzymes are pH-sensitive. Sweating, occlusion, detergents, alkaline cleansers, and prolonged wet clothing can all shift the local surface environment; in flexures, shoes, and cycling shorts, these changes may help explain why maceration, intertrigo, folliculitis, and tinea appear during heavy training blocks (18,10).

Sebum and surface lipids add another layer. Reduced lipid film may increase dryness, while occlusion and heat may promote follicular plugging in sebaceous regions. This is one reason athletes can show apparently opposite complaints: dry, stinging skin on exposed limbs after wind and washing, but acne mechanica or folliculitis under helmets, straps, bib shorts, or tight technical clothing (5,11).

Taken together, these studies do not prove that endurance athletes chronically have impaired skin barriers. They show that intense activity can transiently alter hydration, pH, sebum, antimicrobial

molecules, and microbial counts, and that repeated exposure may be clinically important when combined with friction, UV, clothing, heat, and delayed recovery (15,17,18).

5. Mechanical Stress: Friction, Shear, Chafing, and Blister Formation

Mechanical stress is the most visible barrier insult in endurance sport. The classic explanation of friction blisters emphasizes repeated shear forces that separate layers of epidermis, producing a fluid-filled cavity after sufficient cycles of deformation. Modern blister models have shifted attention from surface rubbing alone to repetitive shear deformation generated by bone motion, high friction force, and repeated loading events (8,9).

Moisture modifies this mechanism. Hydrated stratum corneum can become more deformable, and wet socks or shoes can increase movement between the foot and footwear. At the same time, salt, sand, dust, seams, and wrinkled fabric can create focal areas of higher shear. This explains why blisters often cluster at toes, heels, metatarsal heads, and other sites where motion and pressure concentrate (8,9).

Chafing is closely related but clinically distinct. It usually reflects abrasion at skin-skin or skin-textile interfaces such as the inner thighs, axillae, inframammary folds, waistbands, sports-bra lines, hydration-vest edges, and nipples. Road-runner data show that chafing and blisters are among the most frequent running dermatoses, and weekly distance can influence several trauma-related skin outcomes (4).

Jogger nipple illustrates how a small surface area can become a performance-limiting lesion. Repeated textile movement across the nipple-areola complex can produce erythema, fissuring, bleeding, pain, and secondary irritation. The same mechanical logic applies to hydration-pack straps, heart-rate-monitor bands, wetsuit seams, and compressive garments when prolonged movement occurs in wet conditions (5,6).

Preventive evidence is strongest for some taping approaches but remains incomplete for many common strategies. In the Pre-TAPED II randomized prevention trial, paper tape applied to blister-prone areas reduced both incidence and frequency of foot blisters in ultramarathon runners (19). By contrast, a systematic review of friction-blister prevention in outdoor pursuits found that evidence across socks, antiperspirants, and barrier strategies was heterogeneous and often limited (20).

Recent critical appraisal stresses that many widely used blister-prevention practices are plausible but insufficiently tested. Footwear fit, lubricants, powders, toe socks, padding, textile changes, orthoses, and gait interventions may help some athletes, but robust comparative evidence is sparse and individual blister anatomy varies substantially (21).

Clinical management should therefore begin with mapping the actual shear source. The clinician or coach should ask what changed: shoe model, sock fiber, insole, lacing, foot swelling, rain, heat, trail camber, new orthotic, race pace, pack weight, or total hours on feet. Blister prevention is more successful when it identifies a reproducible friction point instead of applying a generic product to every possible site (9,21).

Once skin has failed, the prevention goal changes. The athlete needs protection of the blister roof if intact, clean drainage only when clinically appropriate, dressing that reduces further shear, and monitoring for cellulitis or purulence. Race-medical settings must balance continued participation with infection risk, pain, gait alteration, and the possibility that a small skin lesion will become a larger soft-tissue problem during prolonged events (8,10).

6. Sweat, Hyperhydration, Salt, and Maceration

Sweat is essential for thermoregulation, but it is also a chemical and mechanical modifier of the skin surface. During endurance exercise, sweat wets the stratum corneum, changes friction behavior, transports electrolytes and organic solutes to the surface, and can alter pH and microbial conditions at sites where evaporation is blocked by clothing or equipment (17,18).

Hyperhydration of the stratum corneum has two faces. Short-term hydration can improve flexibility, but prolonged wetness under socks, gloves, tight shorts, wetsuits, or sports bras can produce maceration, reduce mechanical resistance, and make the tissue more vulnerable to fissuring and abrasion. This is especially relevant during ultramarathons, wet trail races, long indoor trainer sessions, and triathlon transitions where the skin remains damp for hours (4,6).

Salt accumulation is clinically important even though it is rarely quantified in endurance-dermatology studies. Evaporated sweat can leave abrasive crystals on clothing and skin, and those crystals may increase focal irritation at moving textile interfaces. Athletes often recognize this phenomenon after hot races when white salt marks appear on clothing and the skin burns under straps or seams (18,5).

Sweat may also affect photobiology. In the classic experiment by Moehrle and colleagues, sweating reduced the minimal erythema dose, implying that athletes may become more UV-sensitive during exactly the conditions in which they are outdoors, uncovered, and difficult to reapply sunscreen (22).

Post-exercise cleansing is therefore a barrier intervention, not only a hygiene habit. Prompt removal of sweat, salt, soil, sunscreen residue, and occluded clothing can reduce irritant load and microbial opportunity, provided that cleansing is not overly harsh. Repeated use of aggressive detergents, hot water, and abrasive scrubbing may worsen dryness and barrier disruption, particularly in athletes who train twice daily (11,10).

Practical prevention should separate three tasks: managing sweat during exercise, removing sweat after exercise, and restoring the barrier after washing. During exercise, fabric choice, fit, seam placement, anti-chafe products, and sock changes may reduce wet friction. After exercise, rapid showering and drying reduce irritant and microbial exposure. After washing, a bland moisturizer can support barrier recovery when the athlete is prone to xerosis, irritant dermatitis, or fissuring (11,23).

The evidence does not support a universal anti-sweat strategy for endurance athletes. Sweating is physiologically necessary, and antiperspirants may be useful only for selected focal problems such as excessive foot sweating or recurrent maceration. Any intervention that blocks sweating over large areas during heat exposure could create thermoregulatory concerns and should not be treated as routine endurance advice (20,24).

7. Occlusion, Clothing, Equipment, and Microclimate

Occlusion converts the endurance athlete environment from open-air evaporation to a local microclimate. Shoes, socks, gloves, helmets, wetsuits, cycling bibs, sports bras, compression garments, hydration packs, and adhesive dressings can trap heat and moisture, increase contact time with sweat, alter pH, and concentrate mechanical force at edges or seams (5,6).

Equipment-related dermatoses frequently arise where mechanical and occlusive factors overlap. Acne mechanica can occur under helmets, straps, pads, and tight synthetic clothing; folliculitis may appear where sweat, occlusion, friction, and follicular trauma coincide; contact dermatitis can arise from adhesives, rubber, neoprene, dyes, detergents, or topical products used before racing (25,26).

Footwear creates one of the most complex microclimates. During long runs, foot volume may change, socks become wet, heat accumulates, and shoe-upper pressure may shift with terrain. A shoe that is comfortable for one hour may become a blister generator at four hours, especially when downhill running, rain, stream crossings, or edema increase movement and shear (8,9).

Cycling adds a unique saddle microenvironment. Saddle sores are commonly described as lesions in the saddle area, and the literature links them to pressure, friction, moisture, follicular inflammation, and sometimes secondary infection. A scoping review found inconsistent definitions and limited high-quality prevalence evidence, but it confirmed that saddle sores are an important and under-researched problem for cyclists (14).

Female cyclists have additional exposure patterns related to saddle shape, pelvic anatomy, vulvar pressure, clothing, menstruation, and genital soft-tissue vulnerability. A scoping review on female competitive cyclists found limited empirical evidence and highlighted the need for better prevalence, prevention, and treatment research in this population (27).

Wetsuits and triathlon clothing create another form of occlusive stress. Neoprene can rub the neck, axillae, and shoulders, while a wet tri-suit may remain on the skin for hours through cycling and running. This combination can produce neck abrasions, salt irritation, groin chafing, and delayed drying at flexural sites (26,24).

Indoor endurance training deserves attention. Long stationary cycling sessions may reduce natural movement off the saddle, increase continuous pressure, increase local sweat accumulation, and delay garment changes because the athlete is training at home. These conditions are plausible contributors to saddle-area irritation even though direct comparative studies between indoor and outdoor cycling skin outcomes are scarce (14,13).

Clinical advice should therefore be equipment-specific. It is insufficient to tell a cyclist with recurrent saddle sores to improve hygiene if saddle height, saddle tilt, shorts fit, chamois friction, training load, and continuous sitting time are driving the lesion. Similarly, a runner with recurrent toe blisters needs a footwear and terrain analysis, not only a recommendation to apply more lubricant (14,21).

8. Environmental Stressors: Heat, Cold, Wind, Water, and Ultraviolet Radiation

Heat amplifies sweating, wet friction, vasodilation, and fatigue-related changes in movement. In hot endurance events, athletes often experience simultaneous thermal strain, salt accumulation, clothing wetness, sunscreen degradation, and higher UV exposure. These factors interact, so the skin problem after a hot race may reflect more than one insult (28,24).

Cold and wind create a different barrier challenge. Wind exposure can increase evaporative cooling and leave exposed skin dry, tight, and fissured, while cold weather clothing can trap sweat close to the body during high-intensity efforts and then chill the athlete during lower-intensity periods. The same session can therefore produce both occlusion-related maceration and exposed-site dryness (11,6).

Water exposure is especially relevant in triathlon and open-water training. Swimming before cycling and running prolongs skin wetness and may increase friction at wetsuit edges, sock interfaces, and groin seams. In a study comparing swimmers and non-water-sport athletes, a swimming session increased TEWL immediately after exercise, supporting the idea that water environments can measurably alter barrier function (29).

Ultraviolet radiation is the best documented environmental hazard in outdoor endurance sport. A review of photoprotection in outdoor sports concluded that outdoor athletes receive substantial UV exposure and recommended combined protection through behavior, clothing, sunscreen, scheduling, shade, and education (24).

Triathlon provides a striking dose example. In the Ironman World Championship study, three athletes received a mean personal UV exposure of 8.3 minimal erythema doses during 8 hours 43 minutes to 9 hours 44 minutes of racing, and sunburn occurred despite water-resistant sunscreen use (28).

Outdoor runners may know about skin cancer but still underuse protection. In a study of outdoor runners, sun protection behavior and skin cancer literacy were assessed, and the authors concluded that outdoor runners had room for improvement in protective behavior despite the known risk profile of outdoor sport (30).

A systematic review of sun-exposure knowledge, behavior, and attitudes in sportspeople found that knowledge and protective behavior are often insufficient, and that athletes may prioritize

performance, comfort, appearance, or routine over photoprotection during training and competition (31).

Older melanoma data remain relevant as a warning signal. A study of marathon runners reported more atypical melanocytic nevi and solar lentigines than controls, and the authors discussed UV exposure and exercise-related immune effects as possible contributors to skin-cancer risk in this population (32).

Photoprotection must be adapted to endurance realities. Sunscreen should be broad-spectrum, water-resistant, applied before exposure, and reapplied when feasible, but athletes also need UPF clothing, hats or visors, UV-protective eyewear, shade planning, and race scheduling that avoids unnecessary peak UV exposure. Sunscreen alone may fail when sweating, wiping, water immersion, and long event duration are present (28,24).

9. Microbiome, Infection Susceptibility, and the Barrier-Infection Interface

The skin microbiome occupies a living ecological surface. Dry sites, moist sites, and sebaceous sites host different microbial communities, and these communities interact with host immunity, antimicrobial peptides, sweat, sebum, pH, and environmental exposure. Exercise changes the conditions of that surface through sweating, skin temperature, hygiene patterns, clothing, shared facilities, and repeated contact with equipment (16,13).

Endurance athletes are not contact-sport athletes in the classic outbreak sense, but they still experience infection-relevant exposures. Wet socks and shoes favor tinea pedis; occlusive shorts and saddle friction favor folliculitis and furunculosis; skin breaks after blisters or chafing create portals of entry; delayed showering after travel or racing prolongs contact with sweat, soil, sunscreen, and microorganisms (4,10).

The Eda exercise study is important because it connects endurance exercise to local immune markers. After high-intensity endurance exercise, skin secretory IgA decreased, staphylococcal colony counts changed, and human beta-defensin 2 increased, suggesting that intense exercise can disturb and compensate within the antimicrobial barrier during the recovery period (15).

Sport-related infection reviews emphasize bacterial, fungal, and viral infections in athletes, with special attention to Staphylococcus aureus, MRSA, impetigo, folliculitis, cellulitis, dermatophyte infection, herpesvirus infections, and return-to-play concerns. Although much of the outbreak

literature comes from contact sports, the diagnostic and prevention principles are relevant when endurance athletes develop infected abrasions, folliculitis, or tinea (33,10).

Barrier disruption changes infection probability. A blister roof that has torn, a chafed groin fold, a fissured heel, or a saddle sore can allow bacterial entry, and occlusion may then support growth. This is why endurance dermatology should not separate mechanical injury from infection risk; the mechanical lesion often precedes the infectious complication (5,10).

Tinea pedis is particularly relevant to runners because shoes create a warm, moist, occluded environment and because road-runner data found tinea pedis among prevalent sports-related dermatoses. The clinical implication is simple but often neglected: dry feet, rotate shoes, change wet socks, use breathable footwear when possible, and treat fungal disease early to reduce fissuring and secondary bacterial entry (4,10).

Folliculitis and acne mechanica require attention to pressure and occlusion, not only antimicrobial treatment. Recurrent lesions under cycling shorts, helmets, straps, or tight clothing should prompt review of garment fit, laundering, lubricant or chamois cream composition, training volume, and time spent in wet clothing after exercise (14,5).

The microbiome literature also cautions against over-sterilizing the athlete. Excessive antiseptic use, harsh detergents, and repeated stripping cleansers may irritate the barrier and disrupt commensal ecology. Hygiene should remove sweat, soil, and pathogens without creating a cycle of dryness, irritant dermatitis, and further barrier vulnerability (11,13).

10. Clinical Phenotypes of Barrier Dysfunction in Endurance Athletes

Friction blisters are the archetypal endurance skin injury. They are common on the feet of runners and hikers, can alter gait, and can become infected if the roof is disrupted. Their mechanism is best understood as repetitive shear deformation rather than simple rubbing, and prevention should focus on reducing the force, repetition, or tissue movement that creates shear at the vulnerable site (8,9).

Chafing is another high-frequency phenotype. It is often dismissed as minor, but severe chafing can cause bleeding, burning pain, altered movement, sleep disturbance after ultramarathons, and secondary infection. Sites vary by sport, sex, clothing, and body habitus, but common areas include nipples, axillae, thighs, groin, waistband, bra line, hydration-pack edges, and wetsuit contact zones (4,6).

Intertrigo reflects inflammation in opposed skin surfaces where heat, sweat, friction, and poor ventilation coexist. In endurance athletes, the groin, inframammary folds, axillae, and gluteal cleft are common risk zones, especially during hot weather, long events, and prolonged use of tight synthetic clothing. Secondary Candida, dermatophyte, or bacterial involvement should be considered when erythema is persistent, malodorous, exudative, or satellite lesions are present (5,10).

Folliculitis and furunculosis often appear where follicles are stressed by occlusion and friction. Cycling shorts, compression garments, backpacks, helmets, and straps can create follicular trauma and local humidity. Lesions that become painful, fluctuant, spreading, or associated with fever require medical evaluation because Staphylococcus aureus and MRSA are important sport-related pathogens (5,10).

Saddle sores represent a cycling-specific cluster rather than a single diagnosis. The term may include folliculitis, furuncles, nodules, abrasions, cysts, ulceration, and deeper soft-tissue inflammation in the saddle contact region. Reviews emphasize inconsistent definitions, limited prevalence data, and the need to address pressure, friction, moisture, fit, hygiene, and recovery time (27,14).

Acne mechanica is induced or worsened by friction, pressure, heat, and occlusion. Endurance examples include helmet straps, heart-rate monitor bands, hydration packs, compression clothing, wetsuits, and tight cycling garments. The barrier relevance is that follicular occlusion and inflammation often coexist with sweat and textile friction, so treatment should include mechanical modification rather than topical therapy alone (26,5).

Contact dermatitis may be irritant or allergic. Irritant dermatitis can result from sweat, salt, repeated washing, friction, antiseptics, adhesives, or topical products. Allergic dermatitis can arise from rubber accelerators, dyes, fragrances, preservatives, topical antibiotics, adhesives, sunscreen ingredients, or neoprene-related exposures. Patch testing may be needed when dermatitis is recurrent, sharply patterned, or resistant to standard barrier care (25,11).

Xerosis and fissuring occur when water loss, lipid disruption, cold weather, wind, repeated washing, or harsh cleansers outpace repair. Heel fissures, hand fissures in cyclists, and exposed-leg dryness after winter running can become painful and create portals for infection. Moisturizers

containing humectants, emollients, and occlusives can support barrier recovery when matched to the athlete and site (34,11).

Photodamage includes sunburn, tanning, lentigines, actinic keratoses, and increased long-term skin-cancer risk. Endurance athletes may accumulate UV exposure over many years, and the immediate race environment can undermine protection through sweat, water, wiping, and long intervals without reapplication. This makes skin-cancer prevention a core endurance-health issue, not a cosmetic add-on (32,24).

Nail and toe disorders overlap with barrier dysfunction because footwear pressure, wetness, and repetitive impact can injure the nail unit and surrounding skin. Road-runner data identified nail shedding and ingrown nails among prevalent running dermatoses, and these lesions can increase pain and infection risk when combined with tinea pedis or periungual trauma (4,6).

11. Translating Mechanisms Into Sport-Specific Risk Profiles

Distance running concentrates risk in the feet, nails, nipples, thighs, groin, waist, and exposed skin because these sites combine repetitive impact, textile motion, sweat, salt, and sunlight. The same training week may therefore produce plantar blisters, interdigital maceration, jogger's nipple, thigh chafing, tinea pedis, and sunburn without a single unifying diagnosis other than overloaded barrier function (4,6).

The running foot deserves special attention because it is a closed biomechanical system during most sessions. With every stride, the skeleton moves inside a sock and shoe envelope, and the soft tissues must tolerate pressure, shear, swelling, heat, and moisture. When the athlete adds rain, trail debris, downhill running, new socks, or shoe changes, the local shear threshold may be crossed even if the shoe was previously comfortable (8,9,21).

Ultramarathon and stage-race athletes extend the exposure from hours to days. Repeated wet-dry cycles, limited hygiene, sand or dust, sleep deprivation, and reduced attention to early hot spots can turn minor irritation into open erosions. This is one reason blister prevention in ultradistance events has been studied with paper tape and why race medical teams treat foot care as performance-relevant rather than cosmetic (8,19).

Road cycling concentrates risk at the saddle interface, hands, feet, face, and UV-exposed limbs. The saddle area is distinctive because pressure and shear are applied while the skin is occluded by

bib shorts and often coated with chamois products. A cyclist with recurrent saddle lesions therefore needs assessment of saddle height, saddle tilt, reach, shorts construction, chamois hygiene, hair removal practices, indoor trainer time, and return-to-ride decisions (27,14).

Triathlon combines three exposure modes in sequence. Swimming hydrates the stratum corneum and may increase TEWL after water exposure; cycling then adds saddle pressure and prolonged UV exposure; running adds impact, foot shear, and chafing while the athlete may still be wet or salt-covered. The triathlete therefore illustrates how skin-barrier stressors are cumulative across disciplines, not isolated within one sport segment (29,24,14).

Open-water athletes and swim-run competitors add friction at the neck, axillae, shoulders, wrists, ankles, and zipper lines. Neoprene-related rubbing can produce sharp linear erosions that are then exposed to salt water, lake water, sweat, sunscreen, and repeated movement. Although most direct TEWL research in athletes has focused on controlled training sessions, the mechanistic concern is consistent with evidence that water environments can alter barrier readings (29,5).

Indoor endurance training changes the risk profile rather than removing it. Indoor cycling and treadmill sessions may eliminate UV exposure, but they can increase sweat accumulation because airflow is reduced and athletes may delay changing clothing. Long trainer rides also create prolonged saddle pressure without the natural posture variation of outdoor terrain, which may contribute to saddle-area irritation in susceptible cyclists (14,13).

Cold-weather endurance sport should not be interpreted as low-sweat sport. Athletes often overdress early, sweat into base layers, and then experience wind or cold exposure when intensity drops. The result can be maceration under clothing and xerosis or fissuring at exposed sites in the same athlete, which supports a dual prevention strategy of moisture management during exercise and barrier repair after cleansing (17,11).

Hot-weather endurance sport has the opposite temperature profile but a similar cumulative logic. Heat increases sweat, wet friction, salt deposition, sunscreen loss, and fatigue-related equipment shifts. It also increases the practical difficulty of reapplication and clothing coverage, so the skin is simultaneously exposed to friction, chemical irritation, and UV radiation for many hours (28,24).

Trail running, gravel cycling, and adventure racing add soil, plant material, insects, water crossings, and uneven surfaces. These exposures can increase abrasion, contaminate small wounds, and make footwear or clothing fit less predictable. The clinical history should therefore ask not only about

distance and pace but also about terrain, weather, pack load, water immersion, and opportunities for mid-event sock or clothing changes (6,10).

Commuter athletes and recreational participants may have different risks from elite athletes. They may train before or after work, remain in wet clothing during travel, reuse shoes before they dry, or lack access to showers and skin-care supplies immediately after exercise. These constraints matter because delayed removal of sweat, salt, and occlusive garments can prolong the period of barrier stress (4,5).

The sport-specific profile is also useful for prevention counseling. A runner with recurrent heel blisters needs a different plan from a cyclist with recurrent perineal nodules, a triathlete with neck wetsuit abrasions, or an outdoor runner with repeated sunburn. Barrier-centered care begins with the anatomy and exposure map, then selects the intervention that matches the dominant mechanism (8,28,14,11).

12. Clinical Assessment and Differential Diagnosis

A barrier-centered consultation should start with location, timing, repetition, and threshold. The clinician should ask where the lesion appears, how long into exercise it begins, whether it occurs only in specific weather, whether the athlete changed equipment, and whether symptoms improve with rest, drying, or different clothing. Reproducible timing and location often reveal the mechanical or environmental driver (3,5).

Inspection should distinguish intact erythema, erosion, vesicles, bullae, pustules, scale, fissures, nodules, crust, ulceration, and pigment change. These morphologic details matter because a friction blister, allergic contact dermatitis, tinea, folliculitis, herpesvirus infection, cellulitis, and early pressure injury may all be described by athletes as a skin sore or rash (5,10).

Pain quality is diagnostically useful. Burning under a moving seam suggests irritant chafing, focal pressure pain under the saddle suggests mechanical compression or follicular inflammation, itching and scale suggest eczema or fungal disease, and rapidly increasing tenderness with warmth or swelling raises concern for bacterial infection. The history should also include fever, malaise, drainage, and recurrent boils (14,10).

The foot examination should include shoe wear pattern, sock marks, callus distribution, nail length, toe deformity, interdigital scale, heel fissures, and blister roof status. Because blisters are caused

by repetitive shear deformation, the visible lesion should be interpreted together with the underlying movement pattern rather than treated as an isolated surface defect (35,8,9).

The saddle-area examination requires sensitivity and specificity. Athletes may underreport perineal, vulvar, or gluteal lesions because of embarrassment, normalization within cycling culture, or fear of being told to stop riding. The clinician should use neutral language, ask permission, and evaluate pressure points, follicular lesions, abrasions, nodules, asymmetry, and signs of infection (27,14).

A dermatitis pattern may reveal the source. Linear dermatitis under tape suggests adhesive exposure; symmetric rash under a sports bra may suggest textile, detergent, sweat, or friction; sharply demarcated neck dermatitis after wetsuit use may suggest neoprene or friction; and facial or arm dermatitis after sunscreen can reflect irritant or allergic reaction to a product used for photoprotection (25,26).

Tinea should remain in the differential for recurrent foot, groin, and fold complaints. Occlusion and moisture can favor dermatophyte growth, and road-runner data show tinea pedis among common sports-related findings. KOH microscopy or fungal culture can prevent prolonged misclassification as eczema or simple chafing (4,10).

Bacterial infection should be suspected when lesions are pustular, purulent, expanding, warm, increasingly painful, or accompanied by systemic symptoms. Sport-related infection reviews emphasize Staphylococcus aureus and MRSA among important athletic pathogens, and a torn blister or saddle abrasion can become a portal of entry even in a non-contact endurance sport (33,10).

Not every recurrent lesion is caused by poor hygiene. Persistent saddle sores may reflect bike fit and pressure; recurrent blisters may reflect shear and foot anatomy; and recurrent dermatitis may reflect allergy or harsh cleansing. Over-attributing skin disease to cleanliness can delay the mechanical or allergologic diagnosis and encourage excessive washing that worsens the barrier (17,14).

Conversely, hygiene should not be dismissed. Athletes who remain in wet gear, share towels, delay showering after group travel, or train with open draining lesions can increase microbial risk. A balanced assessment distinguishes hygiene as one modifiable exposure among many rather than as the sole explanation (33,10).

Medication and medical history may change the threshold for barrier problems. Atopy, prior eczema, acne treatment, isotretinoin, topical retinoids, systemic immunosuppression, diabetes, prior MRSA, vascular disease, and frequent antibiotic use can alter dryness, infection risk, wound healing, or microbiome composition. These factors should be included when endurance athletes present with recurrent or severe lesions (17,10).

Skin cancer assessment belongs in endurance dermatology. Outdoor athletes with high cumulative UV exposure, repeated sunburn, numerous nevi, actinic damage, fair phototype, family history, or poor photoprotection should receive counseling and appropriate examination. Marathon-runner melanoma data are older, but they remain relevant because endurance exposure accumulates over years (32,24).

A practical clinical note should document the lesion and the exposure that probably produced it. For example: medial heel blister after new trail shoe during wet 30 km run; saddle-area follicular nodules after increased indoor cycling and unchanged bib shorts; inframammary chafing after hot marathon with sports-bra seam; recurrent interdigital scale in high-mileage runner. This format turns diagnosis into prevention planning (5,10).

13. Special Populations, Sex-Specific Issues, and Accessibility

Women endurance athletes may have specific barrier concerns related to sports bras, breast and inframammary chafing, vulvar and perineal saddle symptoms, menstruation, hair removal, clothing fit, and underreporting of genital or saddle-area pain. Reviews of female cyclist saddle sores show that evidence is limited despite the practical importance of the problem (27).

Sports-bra related chafing is a typical example of a sex-specific mechanical issue. Seam position, band tension, moisture retention, breast movement, and salt accumulation may combine to produce linear erosions or painful abrasions. Prevention may require garment redesign, size reassessment, seam avoidance, lubricant placement, and rapid post-exercise cleansing rather than a single topical remedy (5,6).

Male athletes are not protected from intimate-site barrier problems. Groin chafing, scrotal irritation, cycling saddle sores, folliculitis, and jogger's nipple can be severe, and cultural norms may also lead men to delay care. Clinicians should ask neutrally about these areas in all endurance athletes when the exposure pattern makes them plausible (14,5).

Athletes with darker skin phototypes may have lower sunburn susceptibility but still accumulate UV damage and can develop skin cancers that are diagnosed later. Photoprotection recommendations should therefore not be limited to athletes who burn easily. Outdoor sport reviews recommend photoprotection across skin phototypes, with attention to both skin and eye exposure (24).

Athletes with atopic dermatitis or a history of sensitive skin may have lower tolerance for sweat, detergents, friction, wool, adhesives, and repeated washing. Barrier-supportive care is especially important in this group because lipid organization, NMF, pH, immune signaling, and irritant susceptibility can differ from those of athletes without barrier disease (2,11).

Masters athletes may accumulate decades of UV exposure and may also have drier skin, slower recovery from abrasions, and more comorbidities or medications that influence healing. Their dermatologic prevention plan should include skin-cancer screening awareness, moisturization after cleansing, careful footwear fit, and early evaluation of infected or nonhealing lesions (32,24).

Youth endurance athletes require guidance that is practical for families and teams. They may have acne, eczema, developing hygiene routines, rapidly changing shoe sizes, and dependence on adults for sunscreen and clothing choices. Pediatric sports dermatology emphasizes that clinicians should recognize sport-related skin disorders and return-to-play implications in young athletes (33).

Para-endurance athletes and athletes using adaptive equipment may have additional pressure, strap, prosthetic, wheelchair, or brace interfaces. These interfaces can create sustained occlusion and shear at sites that are not typical for non-disabled athletes. Although direct endurance-barrier research is sparse, the same principles of pressure relief, moisture management, inspection, and early wound care apply (8,11).

Economic accessibility matters. Some prevention advice assumes that athletes can buy multiple shoes, high-cost garments, specialty sunscreens, or premium bike fits. A useful clinical plan should prioritize high-value actions such as drying, laundering, avoiding untested race-day changes, using affordable paper tape at proven hot spots, and choosing UPF coverage when feasible (19,24).

Cultural and occupational constraints also shape adherence. Athletes who commute, work long shifts, travel to races, lack shower access, or train in clubs may not be able to follow ideal post-exercise routines. Prevention plans should therefore be realistic: dry clothing in a bag, gentle wipes

when a shower is delayed, spare socks, and post-session moisturizer may be more achievable than a complex protocol (4,5).

14. Prevention and Management: A Barrier-Centered Clinical Framework

Prevention should begin before the athlete is injured. The main objective is to reduce cumulative load on the skin barrier by identifying personal high-risk sites, testing equipment in training, avoiding new shoes or garments on race day, adjusting clothing and footwear to weather conditions, and planning drying, cleansing, and photoprotection around the expected duration of exposure (6,21).

The first step is anatomical mapping. The clinician, coach, or athlete should identify whether the dominant problem occurs at the foot, nail unit, nipple-areola complex, axilla, groin, inframammary fold, waistband, saddle area, exposed limbs, face, or wetsuit contact zone. Each location suggests a different combination of friction, pressure, sweat, salt, occlusion, ultraviolet exposure, water exposure, and delayed recovery. A recurrent medial-heel blister, a saddle-area nodule, a neck wetsuit abrasion, and repeated sunburn are not variants of the same problem; they require different prevention plans (8,28,14).

Friction control should be anatomical and situational. Paper tape has randomized evidence for blister-prone foot sites in ultramarathon runners, but taping is not a universal solution and should be tested before competition. Lubricants, powders, toe socks, padding, footwear changes, lacing strategies, orthoses, and textile changes may help selected athletes, but the choice should follow the specific shear mechanism rather than routine product use (19,20,21).

For chafing, the preventive hierarchy is fit, fabric, friction reduction, and early response. Garments should avoid seams at high-motion sites, remain stable when wet, and not compress folds in a way that traps sweat. Anti-chafe products can reduce friction but may fail when applied too thinly, washed off, contaminated with grit, or used under clothing that continues to move excessively. Recurrent chafing should therefore prompt garment reassessment rather than repeated topical treatment alone (4,5).

Post-exercise care should be prompt but gentle. Athletes should remove wet clothing quickly, wash with a mild cleanser, dry folds, feet, and saddle areas thoroughly, and apply a bland moisturizer to sites prone to xerosis, fissuring, or irritant dermatitis. This sequence removes sweat, salt, soil,

sunscreen residue, and microorganisms while avoiding excessive stripping of lipids and natural moisturizing factor-dependent hydration (11,23).

Infection prevention requires early recognition rather than indiscriminate antiseptic use. Athletes should seek medical evaluation for spreading erythema, warmth, swelling, increasing pain, pus, red streaking, fever, recurrent boils, genital ulceration, lesions near the eye, or wounds that fail to improve. Continuing to train through infected or draining lesions can worsen the individual outcome and increase transmission risk in shared facilities. Conversely, excessive antiseptics, harsh exfoliation, aggressive callus removal, chronic topical antibiotics, and unmonitored occlusive dressings may worsen irritation or disrupt commensal ecology (33,13,10).

Photoprotection should be redundant. Sunscreen is important, but outdoor endurance athletes also need UPF clothing, hats or visors, sunglasses, shade planning, timing strategies, and reapplication protocols when feasible. Sweat, wiping, water immersion, body marking, long race duration, and limited access to supplies can all reduce the practical effectiveness of sunscreen alone. Photoprotection should therefore be treated as part of race preparation, not as an optional cosmetic behavior (31,24).

Barrier prevention can be organized across the training and race timeline. During training, athletes should test shoes, socks, shorts, wetsuits, packs, bras, sunscreens, lubricants, and tapes under realistic distance and weather conditions. During build phases, prevention should escalate before symptoms become severe: rotate shoes, replace damaged socks, address callus edges gradually, test tape at known hot spots, adjust saddle setup, and create a post-session cleaning and moisturization routine. The week before an event should avoid aggressive callus shaving, chemical exfoliation, waxing, untested adhesives, new sunscreen, and new chamois products. Race-morning preparation should be simple and rehearsed: protect known hot spots, lubricate chafe-prone sites, apply sunscreen before clothing and body marking interfere, and start with dry feet and event-appropriate socks (19,34,24).

During the event, athletes should respond early to warning symptoms. A hot spot represents an early pre-blister signal rather than a trivial symptom. Briefly stopping to smooth a sock, add tape, change socks, rinse grit, adjust lacing, or correct a moving garment may prevent progression to a more extensive lesion. In hot events, the plan should include salt and sweat management, saturated garment changes when possible, rinsing of salt-contaminated sites, and sunscreen reapplication. In

wet events, athletes should assume that shoes, socks, adhesives, and skin friction will behave differently. In cold events, layering should balance insulation with moisture escape, because overdressing can cause sweat accumulation under clothing while exposed sites become dry and fissured (22,20,11).

Post-race care should be staged. First, remove wet and contaminated garments. Second, wash gently and dry folds, feet, and saddle areas. Third, inspect lesions before applying occlusive products. Fourth, protect open areas with appropriate dressings. Fifth, monitor for infection over the next 24 to 72 hours, when pain, warmth, swelling, and redness should not be escalating. Recovery days should also be treated as barrier-repair days; athletes may need reduced friction, moisturization, and temporary modification of training mode until erosions, fissures, or chafed areas have re-epithelialized (34,10).

Integrated clinical scenarios illustrate this approach. A marathon runner with recurrent medial-heel blisters after 25 to 30 km requires assessment of shoe fit, sock moisture, heel movement, downhill running, lacing, swelling, and tested hot-spot taping rather than a generic lubricant recommendation. A cyclist with recurrent saddle-area nodules requires review of saddle height, saddle tilt, reach, shorts construction, chamois hygiene, hair removal practices, indoor trainer time, and return-to-ride decisions rather than hygiene advice alone. A triathlete with neck abrasions after open-water swimming requires wetsuit fit assessment, seam and zipper inspection, lubricant testing, and post-swim cleansing. An outdoor runner with repeated sunburn requires combined photoprotection through sunscreen, UPF clothing, timing, shade, and reapplication planning rather than education alone (28,24,14,21).

The practical summary is simple: identify the anatomical site, identify the dominant exposure, reduce friction and occlusion, remove sweat and wet clothing promptly, restore the barrier after cleansing, recognize infection early, and use redundant photoprotection. Skin-barrier protection should be considered part of responsible endurance training because barrier failure can compromise comfort, performance, participation, and long-term health even when it is not life-threatening (7,20,12).

15. Research Gaps, Biomarker-Oriented Interpretation, and Future Directions

The main limitation of current endurance-sport dermatology is the lack of prospective, endurance-specific studies that combine objective barrier measurements with clinical outcomes. Most direct athlete studies are small, cross-sectional, focused on selected sports, or limited to short post-exercise observation. Conversely, many advanced barrier measures, including transepidermal water loss, electrical impedance spectroscopy, corneometry, skin pH, natural moisturizing factor, ceramide profiling, antimicrobial peptides, cytokines, and microbiome sequencing, have been developed mainly in dermatology populations rather than in endurance athletes (12,13).

Future work should therefore distinguish direct endurance-athlete evidence from indirect mechanistic evidence. Direct evidence is strongest for common clinical phenotypes such as friction blisters, chafing, tinea pedis, saddle-area complaints, and ultraviolet exposure in outdoor sport. Evidence is weaker for sport-specific changes in ceramides, natural moisturizing factor, antimicrobial peptides, and microbiome composition. These mechanisms are biologically plausible and clinically relevant, but they should be interpreted as research targets rather than validated diagnostic markers for individual athletes (15,2,16).

A useful next generation of studies should follow runners, ultrarunners, triathletes, cyclists, and comparable endurance athletes across training blocks and target events. Baseline measurements should be collected after standardized rest and under controlled environmental conditions, followed by repeated measurements after long sessions, heat exposure, rain exposure, water immersion, and competition. Outcomes should include incident blisters, chafing, intertrigo, folliculitis, tinea, saddle sores, irritant or allergic dermatitis, xerosis, sunburn, treatment-seeking behavior, missed training, and performance-limiting symptoms (4,7,6).

Biomarker interpretation should be anatomical and exposure-specific. A high transepidermal water loss value at the forearm after sun and wind exposure does not carry the same meaning as interdigital maceration after wet running shoes or follicular inflammation under cycling shorts. Similarly, microbiome shifts after training may be adaptive, neutral, or harmful depending on the body site, symptoms, pH, moisture, antibiotic exposure, and lesion status. For this reason, future studies should combine instrument values with symptom maps, equipment data, environmental exposure, lesion morphology, and clinically relevant outcomes rather than interpreting biomarkers in isolation (16,12,13).

Table 1 summarises an operational barrier-assessment matrix, including preferred measurement fields, the pre-analytical controls each field requires, and research-use interpretation points relevant to endurance athletes.

Table 1. Operational barrier-assessment matrix for endurance-athlete research and clinical translation.

Domain	Preferred field or clinic assessment	Pre-analytical control needed	Interpretive use for endurance athletes
Permeability barrier	TEWL plus symptom map at foot, thigh, groin, saddle region, and exposed forearm	No exercise, shower, moisturizer, or sunscreen immediately before measurement; room acclimatization recorded	Identifies sites where training or equipment may increase water flux; compare within athlete across seasons rather than relying only on population norms (12)
Hydration and maceration	Corneometry or capacitance hydration with standardized visual scoring of maceration	Record sock or garment wet time, rain, water immersion, shoe changes, and drying interval	Distinguishes dry-barrier complaints from overhydrated vulnerable sites after long wet exposure (17,29)
Surface chemistry	Skin pH at flexures, toe webs, saddle region, and exposed sites	Avoid cleanser, antiseptic, deodorant, and topical product confounding before sampling	Flags environments where alkaline shift may interact with irritation, microbial overgrowth, or delayed repair (18,3)
Lipid and NMF reserve	Tape stripping for ceramide profile and	Standardize tape pressure, anatomical site, season, product	Tests whether recurrent endurance dermatitis reflects

	NMF components in research settings	washout, and recent UV exposure	lipid or NMF depletion rather than friction alone (2,11)
Immune defense	Secretory IgA, beta-defensin 2, cytokines, and clinical infection surveillance	Sample before exercise, immediately after, and during recovery using the same site and time-of-day	Links transient immune-barrier changes to folliculitis, impetigo, cellulitis, or recurrent infected abrasions (15,10)
Microbiome ecology	16S/ITS sequencing or targeted culture when clinically indicated	Separate dry, moist, sebaceous, and lesion sites; document antibiotics and antiseptics	Determines whether training load, occlusion, and hygiene shift microbial communities at problem sites (16,13)
UV burden	Personal UV dosimetry, exposure diary, sunscreen reapplication log, and skin examination	Record cloud cover, altitude, water, sweat, clothing coverage, and race start time	Connects cumulative exposure behavior to sunburn, lentigines, actinic damage, and screening need (28,24)

The matrix is intended for protocol design and clinical translation and should not be interpreted as a set of validated diagnostic cut-offs for individual athletes.

Environmental measurement should become routine. Temperature, humidity, wind, ultraviolet index or personal ultraviolet dose, altitude, precipitation, water immersion, and garment wet time may explain why the same athlete tolerates one session but develops severe chafing, blisters, folliculitis, or sunburn during another. Without these data, the relationship between training load and skin-barrier failure remains difficult to interpret (28,24).

Intervention studies should be pragmatic and sport-specific. In runners and ultrarunners, trials should compare paper tape with other commonly used blister-prevention strategies, sock systems,

footwear modifications, lubricants, and combined approaches at anatomically defined hot spots. In cyclists, trials should evaluate saddle fit, shorts construction, chamois products, standing intervals, hygiene timing, and graded return-to-ride protocols after saddle-area lesions. Photoprotection studies should test systems rather than knowledge alone, including sunscreen stations, UPF clothing, coach-led reminders, wearable ultraviolet prompts, race-start timing, and transition-area reapplication protocols (19,24,14,21).

Implementation research is also needed. Many athletes already know that they should shower, dry the skin, use sunscreen, avoid untested race-day equipment, and respond early to hot spots, yet adherence is limited by cost, discomfort, aesthetics, perceived performance effects, time pressure, sponsor clothing, travel, race logistics, and fear that preventive products may irritate the skin. Future prevention protocols should therefore be simple, low-cost, sport-specific, and compatible with real training and competition routines (30,31,5).

The field also needs consensus outcome measures. A minimum dataset should include athlete profile, training load, equipment exposure, environmental exposure, barrier measurements, microbial or infection data when relevant, and clinical outcomes that affect participation. Such a dataset would allow prospective cohorts and nested intervention trials to move the field from plausible advice toward individualized, evidence-based barrier protection.

Table 2. Proposed minimum dataset for future endurance-skin-barrier studies.

Dataset block	**Variables to capture**	**Reason for inclusion**
Athlete profile	Age, sex, sport, training age, atopy history, medication, prior infections, Fitzpatrick phototype	Allows stratification of baseline barrier risk and UV susceptibility.
Training exposure	Weekly volume, longest session, intensity distribution, terrain, indoor/outdoor ratio, race duration	Separates cumulative load from single-event exposure.
Equipment exposure	Shoes, socks, saddle, shorts, wetsuit, pack, bra, helmet, adhesives, topical products	Links lesions to modifiable contact systems.

Environment	Temperature, humidity, wind, UV index, altitude, precipitation, water immersion, garment wet time	Explains why the same athlete may develop lesions only in specific conditions.
Barrier measures	TEWL, hydration, pH, EIS, NMF, ceramide subclasses, antimicrobial peptides, cytokines	Connects symptoms to physical, chemical, lipid, and immune barrier mechanisms.
Microbial data	Targeted cultures when infected, 16S/ITS sequencing in research, antibiotic and antiseptic exposure	Distinguishes colonization, dysbiosis, and clinically significant infection.
Clinical outcomes	Blisters, chafing, intertrigo, folliculitis, tinea, saddle sores, dermatitis, sunburn, treatment, missed training	Anchors biomarkers to outcomes that matter to athletes and clinicians.

The immediate aim should not be a single diagnostic biomarker, but an integrated model that links sport, anatomy, environment, equipment, barrier physiology, microbial ecology, and clinical outcome. Until such data exist, biomarker language should remain cautious: transepidermal water loss, pH, hydration, ceramides, natural moisturizing factor, antimicrobial peptides, and microbiome data can guide hypotheses, but clinical decisions still require careful history, lesion morphology, equipment review, environmental context, and follow-up (17,3,10).

16. Conclusion

Skin barrier dysfunction in endurance athletes is best understood as a convergence of physical, chemical, immunologic, microbial, and environmental stressors rather than as a single disease. The same athlete may experience friction blisters, chafing, folliculitis, saddle sores, irritant dermatitis, tinea, and sunburn because prolonged exercise combines shear, sweat, occlusion, pH change, microbial opportunity, UV exposure, and delayed recovery (19,24,14,3).

The most defensible clinical approach is mechanism-based prevention. Reduce shear at known hot spots, keep wet and occluded skin from remaining wet longer than necessary, cleanse gently after training, restore dry or fissured skin with barrier-supportive moisturizers, identify infection warning signs early, and use redundant photoprotection for outdoor training and racing (17,24,10).

The evidence base is clinically useful but incomplete. Recent studies and reviews clarify photoprotection, saddle sores, sports infections, microbiome concepts, and blister-prevention uncertainty, yet endurance-specific data on TEWL, NMF, ceramides, pH trajectories, antimicrobial peptides, and microbiome shifts remain limited. The next step is integrated prospective research that connects objective barrier measurements to real clinical outcomes and practical prevention strategies (18,6,13).

For now, clinicians, coaches, and athletes should treat the skin as an endurance organ. Although barrier failure is rarely life-threatening in most events, it can compromise performance, comfort, participation, and long-term health. Protecting the barrier is therefore part of responsible endurance training, not an optional cosmetic routine (7,20,12).

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