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Infectious skin diseases in athletes, with particular reference to contact and combat sports: a narrative review of epidemiology, prevention and treatment

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ABSTRACT

Introduction and purpose. The skin is the body's largest organ and its principal barrier against the external environment. In athletes—especially those in contact and combat sports—continuous mechanical, thermal and microbial stress predisposes to cutaneous infection and to team-wide transmission. This review synthesises current evidence on the epidemiology, clinical features, diagnosis, prevention and treatment of infectious skin diseases in athletes, with emphasis on wrestling, judo, boxing and mixed martial arts.

Material and methods. A narrative review of peer-reviewed reviews, epidemiological and microbiological studies, outbreak investigations and case reports addressing bacterial, viral, fungal and parasitic skin infections in athletes.

Results. Bacterial infections—chiefly Staphylococcus aureus, including community-associated methicillin-resistant strains, and Streptococcus pyogenes—together with herpes gladiatorum (herpes simplex virus type 1) and dermatophytoses (tinea gladiatorum, predominantly Trichophyton tonsurans) dominate the clinical picture. Transmission occurs mainly through direct skin-to-skin contact, with fomites playing a secondary role. Diagnosis is largely clinical but is strengthened by potassium hydroxide microscopy, culture and polymerase chain reaction. Management combines targeted antimicrobial therapy, incision and drainage of purulent

lesions, isolation and, for herpes gladiatorum and tinea gladiatorum, chemoprophylaxis; return to competition is governed by defined criteria.

Conclusions. Early recognition, accurate diagnosis, prompt treatment and isolation, rigorous hygiene, environmental disinfection and evidence-based prophylaxis are the cornerstones of minimising morbidity and interrupting transmission of skin infections in athletes.

Keywords: infectious skin diseases; contact sports; combat sports; herpes gladiatorum; tinea gladiatorum; methicillin-resistant Staphylococcus aureus; prevention.

1. Introduction

The skin and its subcutaneous tissue constitute the outermost protective barrier of the human body, shielding internal organs from mechanical trauma, ultraviolet radiation, thermal extremes and, most relevant to the present discussion, invasion by pathogenic micro-organisms [1]. Beyond this barrier role, the skin performs immunological, thermoregulatory, excretory and sensory functions; free fatty acids maintain an acidic surface pH of roughly 4.7–5.3, and the skin-associated lymphoid tissue together with a diverse commensal microbiome forms a "protective coat" that competitively excludes pathogenic flora and instructs cutaneous immunity [1,2]. In athletes this equilibrium is repeatedly challenged. Intense and prolonged exertion, profuse sweating, friction and pressure from clothing and equipment, recurrent micro-trauma and, in many disciplines, sustained skin-to-skin contact with opponents all conspire to compromise barrier integrity and to facilitate both the acquisition and the onward transmission of infectious agents [3,4].

Infectious skin diseases are, accordingly, among the most frequently encountered medical problems in sport. Dermatological conditions account for approximately 8.5% of health problems reported by high-school athletes and 20.9% of those reported by collegiate athletes [5,6]. The burden is heavily concentrated in contact and combat disciplines—wrestling, judo, sumo, rugby and American football—where direct cutaneous contact and frequent abrasions provide an efficient route for pathogen transfer [6,7]. A review of infectious-disease outbreaks in competitive sport between 2005 and 2010 found that skin and soft tissue was the most common site of infection (71% of outbreaks), that MRSA (33%) and tinea, or trichophytosis, (29%) were the leading pathogens, and that most outbreaks occurred in close-contact sports, chiefly combat sports and American football [8]. In wrestling in particular, cutaneous infections have historically been responsible for roughly one-fifth of all practice time lost to medical conditions, and skin infections account for approximately 20% of sports-related time-loss incidents among wrestlers, driven chiefly by herpes simplex virus (about 40.5%), bacterial infections (about 24.9%) and dermatophytosis (about 22.1%) [6,9].

The spectrum differs by competitive level. Among high-school wrestlers the most common infectious dermatosis is impetigo (approximately 30%), followed by herpes simplex virus infection (20%) and dermatophytosis (20%); among collegiate wrestlers, herpes virus infection predominates (47.1%), followed by impetigo (36.8%), tinea corporis (7.4%), cellulitis (5.9%) and methicillin-resistant Staphylococcus aureus infection (2.9%) [6]. These conditions are more than a cosmetic nuisance: in addition to individual morbidity they cause disqualification from

competition, mandate antimicrobial therapy, and carry the risk of team-wide or tournament-wide outbreaks [6,10].

Descriptive epidemiological data reinforce this concentration of risk in wrestling and clarify the relative frequency of the responsible pathogens. Analysis of National Collegiate Athletic Association surveillance from the 2009-2010 through 2013-2014 seasons recorded an overall skin-infection rate of 14.23 per 10,000 athlete-exposures, of which viral infections were the most common (rate 6.35 per 10,000), predominantly herpes simplex virus type 1, which alone accounted for 41.1% of all infections; bacterial infections (rate 3.68) and fungal infections (rate 3.05) followed [10]. Nearly a quarter of infections (22.3%) were recurrent, most occurred during the regular season and were identified at practice rather than competition, and the great majority clustered within a small number of team-seasons—65.2% arose in just five of 35 team-seasons—vividly illustrating the contagious, outbreak-prone nature of these conditions [10]. Notably, in that dataset the most frequent fungal infection was tinea versicolor rather than ringworm, a divergence from earlier reports explained by the inclusion of non-time-loss infections, since all tinea versicolor cases involved no lost time and would have been excluded under stricter case definitions [10].

A national survey of United States high-school athletes similarly documented a preponderance of skin infections in wrestling and estimated bacterial infections at 53.8% and herpes at 6.7% of infections, although providers unfamiliar with the sport tend to overestimate bacterial disease at the expense of herpes; long-term surveillance at national wrestling tournaments has repeatedly shown that inadequate suspicion and culturing lead to over-diagnosis of bacterial infection and under-diagnosis of herpes gladiatorum [11]. Wrestling, football and hockey athletes are at higher risk than participants in most other sports because of shared equipment and direct skin-to-skin contact, and abrasions, cuts and open wounds provide ready portals of entry [11]. These epidemiological realities frame the clinical and preventive priorities that the remainder of this review develops.

Two features peculiar to the athletic setting amplify the clinical challenge. First, presentations are frequently atypical—lesions abraded during competition, examined very early during routine pre-match skin checks, or masked by inappropriate topical corticosteroids—so that diagnostic error is common; in one series more than 90% of herpes gladiatorum cases were misdiagnosed at initial presentation, with bacterial infection consistently over-diagnosed at the expense of viral disease [7,9,11]. Second, asymptomatic carriage and subclinical shedding mean that visibly healthy competitors can seed outbreaks, which limits the effectiveness of visual screening alone [7,12].

The purpose of this narrative review is to synthesise and critically compare the current evidence on infectious skin diseases in athletes, focusing on medical aspects—aetiology, epidemiology, clinical presentation, diagnosis, treatment and prevention—and giving particular attention to contact and combat sports. Where the literature is discordant, competing findings are contrasted and the probable sources of disagreement are examined; where the evidence is strong, its practical implications for the sports physician, dermatologist and athletic trainer are drawn out.

2. Material and methods

This work is a narrative review. The evidence base comprises peer-reviewed narrative and systematic reviews, descriptive and analytical epidemiological studies, outbreak investigations, microbiological and environmental sampling studies, and case reports and case series concerning bacterial, viral, fungal and parasitic skin infections in athletes, retrieved from databases including PubMed, Google Scholar, Scopus, Web of Science and Embase using combinations of terms such as "skin infection", "contact sports", "wrestling", "herpes gladiatorum", "tinea gladiatorum", "methicillin-resistant Staphylococcus aureus", "athletes" and "prevention" [6,12,13]. In keeping with the narrative format, sources were selected to represent the breadth of the field and to permit critical comparison rather than to satisfy the exhaustive, protocol-driven criteria of a systematic review. Findings are organised by pathogen class and, within each class, by aetiology, epidemiology, clinical features, diagnosis, treatment and prevention.

Table 1. *Selected principal studies and reviews informing this narrative review of infectious skin diseases in athletes.*

Study	Type	Population / focus	Key findings relevant to this review
Kucharczyk et al. (2025) [6]	Literature review	Contact-sport athletes	Bacterial, viral and fungal skin infections; MRSA, Trichophyton and HSV predominate; direct contact and shared equipment drive spread; prevention by hygiene, early detection, disinfection.
Peterson et al. (2019) [7]	Clinical review	Contact-sport athletes (wrestling model)	Herpes gladiatorum 20-40% incidence in wrestlers; mats minor vectors; valacyclovir prophylaxis; preferred treatment regimens and NCAA/NFHS return-to-play criteria.
Herzog et al. (2017) [10]	Descriptive epidemiology	NCAA men's wrestlers, 2009-2014	Overall rate 14.23/10,000 exposures; viral (HSV-1 41.1%) most common; 65.2% of infections in 5 of 35 team-seasons; 22.3% recurrent.
Wilson et al. (2013) [9]	Clinical review	Wrestlers	Detailed diagnosis and dosing for herpes gladiatorum, bacterial SSTIs and tinea gladiatorum; fluconazole prophylaxis (SORT A); PCR/KOH confirmation.
Srisaeng et al. (2025) [14]	Outbreak investigation	Thai-boxing gyms, Phuket, Thailand	First Thai HG outbreak; 9 confirmed cases, all non-Thai from high-income countries; attack rates 4.8-

Study	Type	Population / focus	Key findings relevant to this review
			33.3%; clinching distribution; low seroprevalence/immunity hypothesis.
Nguyen et al. (2005) [15]	Outbreak / case-control	College football team	Recurring CA-MRSA (USA300); linemen attack rate 18%; soap sharing and cuts linked to infection; locker proximity and towel sharing to carriage.
Champion et al. (2014) [16]	Prevalence / molecular	University student athletes	34.9% MRSA carriage; 76% in wrestlers; 47% of carriers not colonised in nares; SCCmec IV; dynamic strain variation over 12 weeks.
LaBelle et al. (2020) [17]	Cohort / quality improvement	High-school and collegiate training rooms	Three-phase protocol cut bacterial APC by 94.7%; MRSA/VRE 24% to 0%; influenza 25% to 0; education phases decisive.
Zalewski et al. (2022) [12]	Systematic review	Contact-sport athletes (tinea gladiatorum)	Prevalence 2.4-100%; T. tonsurans predominant; diagnosis, treatment and prophylaxis; mats vector not reservoir.
Bassiri-Jahromi & Khaksar (2008) [18]	Epidemiological study	Wrestlers, Tehran, Iran	T. tonsurans in 68.5% of wrestlers; family involvement 10.8%; skin-to-skin transmission; head/neck/upper-limb lesions.
Akhoundi et al. (2022) [19]	Experimental (laboratory)	Dermatophyte conidia on textiles	Laundering at 60 C eliminates conidia; heat drying, freezing and direct dry heat ineffective; rinsing decisive for fomite decontamination.
Pilz et al. (2025) [20]	Retrospective multicentre	Dermatophytoses, Germany	T. tonsurans emerging; tinea capitis doubled; barbershop transmission; terbinafine recommended empirically; historical wrestling link.
Ptak & Szyc (2024) [21]	Narrative review	Tinea pedis and onychomycosis	Terbinafine effective topically and orally; single-dose film-forming solution; laser adjuncts; 18.6% terbinafine resistance emerging; diabetes a risk factor.

Study	Type	Population / focus	Key findings relevant to this review
Sfyri et al. (2025) [22]	Retrospective survey	1047 Greek competitive swimmers	Impetigo 10.9%, pitted keratolysis 3.2%, folliculitis 2.7%; equipment sharing and bench use significant; many trained during treatment.
Pawluszkiewicz et al. (2024) [13]	Narrative review	MRSA prevention in sport	~6% asymptomatic athlete carriage; decolonisation effective but temporary; multifaceted hygiene, screening and education essential.

APC, aerobic plate count; CA-MRSA, community-associated methicillin-resistant Staphylococcus aureus; HG, herpes gladiatorum; HSV, herpes simplex virus; NCAA, National Collegiate Athletic Association; NFHS, National Federation of State High School Associations; SORT, Strength of Recommendation Taxonomy; SSTI, skin and soft-tissue infection; VRE, vancomycin-resistant enterococcus.

3. The cutaneous barrier, the skin microbiome and mechanisms of infection in athletes

3.1. Structure and protective functions of the skin

The skin is composed of three layers—epidermis, dermis and hypodermis—of distinct embryological origin and function [1]. The keratinised, multilayered epidermis provides the physical barrier; the dermis houses vasculature, nerve endings, mechano- and thermoreceptors and the skin appendages; and the subcutaneous tissue offers thermal insulation, energy storage and cushioning against injury [1]. Physiologically, the skin maintains water and electrolyte balance and body temperature, participates in vitamin D synthesis and in excretion of metabolites, and functions as an immunocompetent organ through the skin-associated lymphoid tissue, which integrates innate and adaptive responses to cutaneous antigens [1]. Critically for athletes, the acid mantle generated by free fatty acids and the resident microbiome together resist colonisation by pathogenic organisms; disruption of this microenvironment—an event that is especially common during contact sports—predisposes to the development or worsening of skin disease [1].

3.2. The dual role of sweat

Sweating illustrates the double-edged nature of the athletic cutaneous environment. Eccrine secretions deliver water, electrolytes, lactate and urea that hydrate the stratum corneum and contribute to the natural moisturising factor, and they transport antimicrobial peptides such as dermcidin, cathelicidin and lactoferrin, together with immunoglobulins and cytokines, to the skin surface, thereby reinforcing innate defence [4]. In physically active individuals, exercise-induced sweating can therefore transiently strengthen barrier function and microbial homeostasis [4]. However, under conditions of prolonged moisture, friction and occlusion—precisely those generated by training gear and sustained contact—sweat promotes maceration of the stratum corneum, disrupts the barrier, raises local humidity and shifts surface pH, creating a microenvironment conducive to microbial overgrowth, irritation and infection [4]. Excessive or prolonged sweat exposure is associated with increased susceptibility to bacterial and fungal

infection, including dermatophytosis and trichomycosis axillaris, and hyperhidrosis itself has been linked to a higher risk of treated skin infection [4]. Maceration weakens the epidermal barrier and facilitates dermatophyte penetration, which explains why moist, occluded and repeatedly abraded sites are characteristic locations of sports-related fungal disease [4].

A more detailed understanding of sweat physiology clarifies why the athletic environment is simultaneously protective and hazardous. Three gland types contribute to cutaneous secretion: eccrine glands, the most numerous (an estimated 2-4 million, densest on the palms and soles), which produce hypotonic sweat under sympathetic cholinergic control and drive thermoregulation; apocrine glands, which open into hair follicles in the axillary, mammary, perineal and genital regions, become active at puberty, and secrete a viscous, lipid-rich fluid that is odourless until bacterial decomposition; and apoeccrine glands of the axilla, which combine features of both and contribute substantially to axillary sweating [4]. Eccrine secretion carries not only water, sodium chloride and metabolites but also immunoglobulins (IgG, IgA, IgE), antimicrobial peptides (dermcidin, cathelicidin, lactoferrin), cytokines and proteolytic enzymes that constitute an important arm of innate cutaneous defence [4]. Dermcidin in particular is constitutively expressed in eccrine glands and processed into peptides active against Staphylococcus aureus, Escherichia coli, Enterococcus faecalis and Candida albicans, while also modulating innate immune responses [4]. Increased sweating during exercise enhances delivery of these protective molecules to the surface, temporarily improving stratum corneum hydration and barrier integrity [4].

The same physiology, under adverse mechanical and environmental conditions, predisposes to a family of sweat-related and infection-relevant dermatoses that are common in athletes. Dermatophytosis is favoured because increased moisture and temperature macerate the stratum corneum, weaken the epidermal barrier and facilitate fungal penetration, a process amplified by occlusive clothing, frequent skin-to-skin contact and shared facilities [4]. Intertrigo arises within opposing skin folds where friction, occlusion and persistent moisture compromise the barrier and permit overgrowth of opportunistic organisms such as Candida, Corynebacterium minutissimum and group A beta-haemolytic streptococcus; prolonged physical activity, repetitive motion, tight clothing and profuse sweating intensify these processes and predispose to more severe or recurrent disease [4]. Trichomycosis axillaris, a superficial infection of the hair shaft by aerobic Corynebacterium species, is driven by excessive sweating that promotes bacterial proliferation, adhesion and biofilm-like sheath formation around axillary hairs, so that intense physical activity, occlusive clothing and repeated friction render athletes particularly susceptible [4]. Dyshidrotic eczema, a vesicular eruption of the palms and soles associated with immune dysregulation, may be worsened by prolonged excessive sweating and carries an increased risk of secondary infection through barrier disruption [4].

Sweat may also aggravate pre-existing inflammatory dermatoses in ways that heighten infection risk. In atopic dermatitis, sweat components penetrating a defective barrier deliver pro-inflammatory cytokines (IL-1 alpha, IL-1 beta, IL-31, IL-8) that activate keratinocytes; altered surface pH and increased colonisation by Staphylococcus aureus further amplify inflammation, and scratching perpetuates the cycle [4]. Because both barrier failure and staphylococcal overgrowth predispose to secondary bacterial infection, these conditions are directly relevant

to the infectious burden of sport. Accordingly, attentive skin care is a shared preventive measure: prompt cleansing with gentle products after exercise removes sweat and micro-organisms; dry, breathable, moisture-wicking clothing and frequent changing of damp garments limit the proliferation of fungi and bacteria in folds and interdigital spaces and reduce maceration; and regular emollient use supports barrier restoration in athletes with atopic dermatitis [4]. These simple measures lower the risk of the very infections—trichomycosis axillaris, dermatophytosis and intertrigo—that a compromised, sweat-laden barrier invites [4].

The structural and immunological properties of the skin itself complete this picture. The epidermis, composed of keratinocytes arranged in the stratum basale, spinosum, granulosum and corneum, is the physical barrier; melanocytes within it produce melanin, whose quantity determines phototype and whose UV-induced products, if unchecked, are potentially carcinogenic, so that photoprotection (protective clothing and sunscreen of at least SPF 30) is relevant to outdoor athletes [1]. The dermis provides collagen (chiefly type I, about 80%, and type III, about 20%) that confers elasticity and resistance to injury, together with vasculature and the sensory apparatus [1]. Immunologically, the skin-associated lymphoid tissue integrates a rapid, non-antigen-specific innate response (chemotaxis, phagocytosis, complement) with a slower, memory-bearing adaptive response, the two acting in concert to defend the integument; disturbance of the commensal microbiome—most often during contact sports—removes an important competitive barrier and permits the development or worsening of skin disease [1]. It is against this backdrop of barrier physiology and cutaneous immunity that the specific infectious agents discussed below exert their effects.

3.3. The skin microbiome of contact-sport athletes

Molecular characterisation of the skin microbiome supports the view that contact sport itself reshapes cutaneous ecology. In a 16S ribosomal RNA study of young wrestlers, kickboxers and non-athlete peers, the athletes' microbiome differed from that of controls, and bacteria linked to skin infection—including Aliterella, Arthrobacter and Empedobacter species—were present in roughly 30% of wrestlers and kickboxers yet were absent from controls [2]. Wrestlers, who experience the most frequent skin-to-skin contact and wear the least covering sportswear, showed a distinct profile from both kickboxers and non-athletes, whereas kickboxers—whose sportswear covers more of the body and whose striking-based movements involve less grappling—more closely resembled controls [2]. The authors also documented sexual dimorphism in microbiome composition [2]. Although such associations are cross-sectional and cannot establish causation, the consistent finding of infection-associated genera in a substantial minority of contact-sport athletes, but not in controls, reinforces the epidemiological picture of heightened infection risk and argues for regular skin monitoring in these disciplines [2].

At the level of individual genera, the same study found that Enhydrobacter and Staphylococcus were the most abundant members of the youth skin microbiome, followed by important commensals including Corynebacterium, Cutibacterium, Acinetobacter, Micrococcus, Paracoccus, Psychrobacter and Streptococcus—organisms that ordinarily contribute to defence against pathogens, control of inflammation and microenvironment regulation [2]. Significant sexual dimorphism was evident: males showed higher relative abundance of genera associated with respiratory and nosocomial infection (Corynebacterium, Luteimonas, Paracoccus,

Psychrobacter, Brevundimonas), whereas females showed higher abundance of genera associated with skin inflammation and infection (Cutibacterium, Kocuria, Micrococcus, Pseudomonas, Streptococcus, Escherichia-Shigella) [2]. Comparing athletes with controls, the relative abundance of Psychrobacter was far higher in athletes, while Staphylococcus—paradoxically—was higher in non-athletes; wrestlers carried more Acinetobacter and Brevundimonas than either kickboxers or controls, and both athlete groups exceeded controls in Micrococcus and Enhydrobacter [2]. Several of these organisms are opportunistic pathogens implicated in skin and soft-tissue infection in immunocompromised hosts, and their enrichment in contact-sport athletes, together with the presence of overt infection-associated genera absent from controls, led the authors to recommend appropriate antiseptic skin-care product selection and routine microbiological monitoring to reduce infection and contact-related contamination [2].

The mechanisms underlying these sex differences are not fully understood, but experimental work suggests that androgens can suppress type 2 innate lymphoid cell activity through an androgen–innate-lymphoid-cell–dendritic-cell axis, modulating cutaneous immunity and disease susceptibility, while differences in hygiene practices, clothing choices and hormonal milieu may further shape the male and female skin microbiome [2]. The wrestlers' high-friction, minimally clothed and grappling-intensive environment appears to foster microbial exchange and dysbiosis to a greater degree than the striking-based, more fully clothed environment of kickboxing, which may explain why kickboxers' microbiome more closely resembled that of non-athletes [2]. These findings are associative and derived from a cross-sectional design, so they cannot establish causation; longitudinal study is required to determine whether the microbiome shifts and pathogen enrichment observed in contact-sport athletes prospectively predict clinical infection [2].

3.4. Routes of transmission and risk factors

Transmission of any infectious agent requires three elements: a source, a susceptible host and a mode of transmission [23]. In the athletic setting the dominant mode is direct skin-to-skin contact; indirect (fomite) transmission via mats, shared equipment, towels, razors and clothing is documented but generally secondary, and droplet or airborne routes are comparatively minor for cutaneous disease [7,23]. Athletes are unusually susceptible hosts because their skin sustains constant assault to its integrity and because they engage in high-risk, high-contact activities; the source may exhibit obvious active lesions or may be an asymptomatic carrier in the incubation period, so that individuals should always be assumed to be potential carriers [23]. Recognised risk factors include abrasions, cuts and turf burns that breach the barrier, sweaty and macerated skin, sharing of equipment and lockers, close proximity of an athlete's locker to that of an infected teammate, and crowded living arrangements during camps and travel [6,7,15].

The relative contribution of fomites versus direct contact and of grooming practices remains debated, and recent work has begun to quantify individual behaviours. In a survey of 109 collegiate and club wrestlers, frequent scratching carried the highest odds ratio for reported skin infection (odds ratio 2.70; 95% confidence interval 0.87–8.37), exceeding any measured mat-cleaning variable, which led the authors to propose that scratch frequency—and by extension

nail hygiene and cutaneous micro-trauma—may be a stronger predictor of infection than mat-cleaning practices [24]. Although this trend did not reach statistical significance in a small self-reported sample, it aligns with the broader consensus that direct contact and skin breaches, rather than environmental surfaces, drive most transmission [7,24].

Two additional transmission considerations broaden the clinical picture. First, some pathogens are zoonotic or occupationally acquired: animals harbour both ringworm and community-associated MRSA, so athletes who work as butchers, veterinarians, hunters or farm workers may acquire these organisms through direct animal contact, and wrestlers with recalcitrant or recurrent tinea should have family members and pets examined as potential reservoirs for reinfection [11,23]. Second, although this review concerns cutaneous disease, the athletic environment also transmits infections by common-source and airborne routes—shared water bottles have been implicated in enteroviral outbreaks, and mass gatherings in confined training facilities in influenza and measles—so a comprehensive infection-control programme must address more than the skin, even though direct and indirect cutaneous contact remains the dominant route for the dermatoses that are the focus here [25,26].

4. Bacterial infections

Skin and soft-tissue infections caused by Staphylococcus and Streptococcus species are among the most common conditions affecting humans and are especially frequent in contact-sport athletes, where close physical interaction, shared equipment and skin injury combine to facilitate spread [6,11]. Clinical syndromes range from superficial impetigo to erysipelas, cellulitis, folliculitis, furuncles, carbuncles and abscesses [7,9].

4.1. Staphylococcus aureus and methicillin-resistant strains

Staphylococcus aureus is a Gram-positive, coagulase-positive commensal that colonises the skin, glands and mucous membranes, most notably the anterior nares; approximately 20% of people are persistent or intermittent nasal carriers, and this colonisation constitutes a reservoir that greatly increases the likelihood of subsequent infection [6]. Methicillin-resistant S. aureus (MRSA) was first described in the 1960s as a nosocomial pathogen; community-associated MRSA (CA-MRSA) emerged in the 1980s and has since become a major public-health concern [6,13]. CA-MRSA strains typically carry the smaller staphylococcal cassette chromosome mec types IV and V, are resistant to fewer non–β-lactam classes than hospital strains, and frequently express Panton–Valentine leukocidin, a cytotoxin associated with necrotising soft-tissue infection and pneumonia [6,13]. Athletes are recognised as a high-risk group alongside neonates, military personnel, detainees and other groups characterised by crowding and close contact [13].

Colonisation studies frame the epidemiology. A meta-analysis found that approximately 6% of asymptomatic athletes are colonised with MRSA, rising to about 13% among collegiate athletes, with rates varying by sport and country [13]. Colonisation is not confined to the nares: in a 12-week multi-site study of 223 university athletes and staff, 34.9% carried MRSA, wrestlers had the highest carriage (76%), and 47% of MRSA-positive individuals were not colonised in the anterior nares—so that nasal culture alone identified only about half of carriers [16]. That study also demonstrated that carriage is a dynamic process, with strains and toxin-

gene profiles varying and alternating within individuals over time, and confirmed transmission between teammates in wrestling and baseball [16]. Because colonised athletes are approximately seven times more likely than non-colonised athletes to develop a bacterial skin infection, this asymptomatic reservoir is central to outbreak dynamics [7].

Outbreak investigations illustrate the practical consequences. During the 2003 National Football League season, five of 58 St. Louis Rams players (about 9%) developed MRSA infection, all at sites of turf abrasion, while environmental sampling recovered methicillin-sensitive S. aureus from taping gel and whirlpools [13,25]. In a college football team, Nguyen and colleagues traced a recurring CA-MRSA (USA300) outbreak across consecutive seasons in which linemen had the highest attack rate (18%); case–control analysis implicated sharing bars of soap and pre-existing cuts or abrasions in infection, and locker proximity to an infected teammate and towel sharing in nasal carriage [15]. Such reports establish direct contact and shared fomites—rather than any single environmental source—as the dominant drivers, and they underline the difficulty of eradicating the organism once it is established in a team [15,25].

Molecular characterisation adds depth to this epidemiology. In the multi-site university study, all typable isolates carried the community-associated staphylococcal cassette chromosome mec type IV, with USA300 and USA400 clones together comprising roughly 69% of characterised isolates, although USA700 and USA800 strains were also recovered; the Panton-Valentine leukocidin genes were present in 81% of isolates, exfoliative toxin genes in about 34%, and toxic shock syndrome toxin in 28% [16]. The most common spa type was t008, corresponding to the epidemic USA300 clone, and toxin-gene profiles and even clone identity varied within individuals over the 12-week study, indicating that carriage is a dynamic rather than a static state [16]. Crucially, despite the frequency of virulence genes, only one athlete developed clinical infection during the study, so that colonisation and toxin carriage—while necessary conditions—were not sufficient to trigger disease, and additional predisposing factors (skin breach, moisture, contact) are required [16]. This observation is echoed by longitudinal surveillance showing that nasal MRSA carriage increases during the competitive season without a proportionate rise in infection, cautioning against equating carriage with imminent disease [13].

Transmission dynamics have been dissected in complementary studies. In the football-team outbreak, direct contact and shared personal items dominated, and turf abrasions provided the portal of entry, with methicillin-sensitive S. aureus recovered from taping gel and whirlpools rather than MRSA from the environment [13,15]. Environmental sampling in fitness facilities has found S. aureus on 38.2% of surfaces, with particularly high contamination of weightlifting equipment and treadmill handles, and experimental work has shown that viable S. aureus can survive on volleyballs and basketballs for at least 72 hours, underscoring the potential vector role of shared objects [13]. Even so, contact with an infected or colonised individual remains the dominant route; studies of healthcare providers demonstrate that MRSA is transferred to gloves in about 17% of contacts, and that gloving reduces transferred colony counts roughly 30-fold, supporting the use of gloves and hand hygiene without displacing their fundamental importance [13]. Cosmetic body shaving, common among athletes, has been shown to increase CA-MRSA risk more than sixfold and is therefore specifically discouraged [13,23].

Distinguishing community-associated from healthcare-associated MRSA is clinically useful. Healthcare-associated strains typically carry the larger staphylococcal cassette chromosome mec types I, II or III, rarely express Panton-Valentine leukocidin, affect older patients with comorbidities and cause pneumonia, bacteraemia and invasive disease; community-associated strains carry types IV or V, frequently express the leukocidin, affect younger healthy individuals and cause predominantly skin and soft-tissue infection, although they may produce necrotising pneumonia and severe sepsis [13]. Athletes overwhelmingly encounter the community-associated form, which explains both the predominance of soft-tissue disease and the occasional occurrence of severe, rapidly progressive infection in otherwise healthy competitors [6,13]. Against this background, decolonisation regimens—daily chlorhexidine washes combined with intranasal mupirocin, sometimes as part of universal decolonisation protocols—can reduce the risk of infection by approximately one-third, but the effect is temporary: initial eradication may approach 100%, yet re-colonisation is common in the persistently exposed athletic setting and non-compliance with hygiene protocols contributes to recurrence [13]. Decolonisation must therefore be embedded within continuous preventive practice rather than regarded as curative [9,13].

Diagnosis of a skin and soft-tissue infection is usually clinical, but because no specific sign reliably distinguishes CA-MRSA from methicillin-sensitive disease—CA-MRSA lesions are often mistaken for a "spider bite"—culture and antimicrobial susceptibility testing of purulent lesions is recommended when antibiotics are used, when systemic toxicity or multiple or large lesions are present, when there is water exposure, when initial treatment fails, or during a suspected outbreak [9,11,23]. Culture-guided management is particularly valuable in the team context: in a case series of four wrestlers from one school, culture and sensitivity testing established that all shared an identical, easily treatable MRSA strain, allowing consistent oral therapy and prompting notification of the state athletic and dermatological associations [27].

Treatment follows standard principles. Purulent lesions—furuncles, carbuncles and abscesses—require incision and drainage, which for localised, uncomplicated collections may suffice without antibiotics; non-purulent infection (erysipelas, cellulitis, folliculitis) is treated with an oral β-lactam such as cephalexin or dicloxacillin when MRSA is not suspected [9,11]. When CA-MRSA is likely, empirical oral options include trimethoprim–sulfamethoxazole, clindamycin or doxycycline, usually combined with topical mupirocin, with linezolid reserved for organisms resistant to first-line agents; severe or systemically toxic infection warrants hospitalisation and intravenous therapy [7,9,11]. Decolonisation—daily chlorhexidine washing plus intranasal mupirocin for five to ten days—has a limited and controversial role; the evidence in athletes is weak, re-colonisation is common, and the intervention is best reserved for recurrent infection, culture-confirmed close contacts and outbreak settings [9,11,13].

Specific outpatient regimens have been standardised for these infections. For impetigo, topical mupirocin 2% three times daily is used for limited disease, with oral options including cephalexin or dicloxacillin 250 mg four times daily, clindamycin 300-400 mg three times daily, or erythromycin where susceptibility allows [9]. Non-purulent methicillin-sensitive infections such as erysipelas, cellulitis and folliculitis are treated with cephalexin or dicloxacillin 500 mg four times daily or doxycycline 100 mg twice daily, whereas suspected community-associated

MRSA is covered with doxycycline 100 mg twice daily, trimethoprim-sulfamethoxazole one to two double-strength tablets twice daily, clindamycin 300-450 mg three times daily, or linezolid 600 mg twice daily, generally combined with topical mupirocin [7,9]. For isolated cellulitis without abscess when MRSA is not suspected, 5 to 10 days of an oral beta-lactam covering beta-haemolytic streptococci is appropriate; poor response or systemic toxicity should prompt reconsideration of MRSA, and cases with systemic toxicity, rapid progression or failure of oral therapy after 48 hours warrant hospitalisation and intravenous antibiotics, with infections overlying joints assessed for septic bursitis or septic arthritis [9]. Randomised evidence indicates that antibiotics after incision and drainage of uncomplicated purulent MRSA lesions do not significantly reduce short-term treatment failure, although trimethoprim-sulfamethoxazole may reduce subsequent lesions within 30 days, so the decision to add antibiotics after drainage should follow disease severity and consensus guideline criteria rather than reflex prescribing [9,11].

4.2. Streptococcus pyogenes, impetigo and related pyodermas

Streptococcus pyogenes (group A streptococcus) causes minor infections such as impetigo and pharyngitis as well as invasive disease including necrotising fasciitis and toxic shock–like syndrome [6]. Impetigo, caused by S. aureus, S. pyogenes or a mixed flora, is the archetypal superficial pyoderma of athletes: it begins as vesiculopustular eruptions that dry into honey-coloured crusts, most often on the face and extremities, and spreads readily among wrestlers, swimmers and football players [5,6,9]. First-line treatment is topical mupirocin for limited disease and oral antibiotics (for example clindamycin, cephalexin or dicloxacillin) for extensive or resistant lesions [5,9]. Ecthyma represents a deeper, ulcerative form of impetigo caused by group A β-haemolytic streptococci and is treated similarly but with oral antibiotics [5]. Folliculitis, furuncles and carbuncles arise when hair follicles become infected, usually by S. aureus; a distinct entity, "hot-tub" folliculitis, is caused by Pseudomonas aeruginosa after exposure to inadequately disinfected whirlpools and is generally self-limiting [5,6,9]. Erysipelas and cellulitis, non-purulent infections of the superficial and deeper dermis respectively, present with erythema, warmth and often fever, and are treated with systemic antibiotics [5,9].

Folliculitis merits particular attention because its three common subtypes have different implications for the athlete. Staphylococcal folliculitis is usually pruritic and localised and may become painful; Pseudomonas or hot-tub folliculitis presents as pruritic urticarial papules or pustules after exposure to inadequately disinfected whirlpools and typically resolves within five to seven days without systemic spread; and pseudofolliculitis barbae, the razor-bump reaction to shaving, is not a true infection but an inflammatory response around shaved follicles, more common in individuals with curly hair, that requires no participation restriction unless secondarily infected [5,9]. For staphylococcal and Pseudomonas folliculitis a 48-hour/72-hour rule applies—no new lesions for 48 hours and 72 hours of antibiotic therapy—with dry, non-oozing lesions permitted to be covered for medium- and low-risk contact sports [5]. Abscesses, furuncles and carbuncles, being purulent, require incision and drainage as the primary intervention, with antibiotics added when there is surrounding cellulitis, systemic toxicity, immunocompromise or failure of drainage alone; small furuncles may respond to moist heat,

and athletes may generally return once lesions are dry [5,9]. This graded approach—matching the intervention to the depth and purulence of the lesion—minimises both under-treatment of deep infection and unnecessary antibiotic exposure for lesions that drainage alone will resolve [5,11].

4.3. Corynebacterial infections and bacterial dermatoses of water sports

Several bacterial dermatoses reflect the moist, occluded conditions of athletic footwear and aquatic environments. Pitted keratolysis, caused by Corynebacterium species and Kytococcus sedentarius, produces malodorous crateriform pits on weight-bearing plantar surfaces and is promoted by hyperhidrosis and excess humidity; erythrasma, caused by Corynebacterium minutissimum, produces well-demarcated reddish-brown patches in intertriginous areas and shows coral-red fluorescence under Wood's light [1,6]. Water-sport athletes are additionally exposed to Pseudomonas-associated otitis externa ("swimmer's ear"), leptospirosis from contaminated fresh water, and swimming-pool granuloma caused by Mycobacterium marinum entering through damaged skin [1,6]. The largest relevant epidemiological study, a retrospective survey of 1047 Greek competitive swimmers, self-reported folliculitis in 2.7%, impetigo in 10.9% and pitted keratolysis in 3.2%; infections correlated with age category, and behaviours such as placing towels and clothing on locker-room benches and sharing equipment were significantly associated with impetigo and pitted keratolysis [22]. Notably, many affected swimmers continued to train during treatment, a practice that may perpetuate transmission [22].

4.4. Environmental burden and the evidence for surface interventions

The athletic training facility is itself a reservoir. Quality-improvement studies show that structured programmes can substantially reduce environmental bio-burden: a three-phase protocol combining hand and surface disinfectants with athlete and trainer education across two high schools and two colleges reduced overall bacterial aerobic plate counts by 94.7%, eliminated detectable MRSA and vancomycin-resistant enterococcus (present on 24% of surfaces before intervention), and abolished detectable influenza (initially on 25% of surfaces) over one academic year [17]. Complementary work on wrestling mats found that residual disinfectants reduced bacterial load by roughly 76% compared with non-residual cleaners, and that alcohol-based hand gel used before practice reduced bacterial load on sporting surfaces by about 78% [25,27]. These data indicate that environmental interventions are effective at lowering surface contamination; whether that reduction translates into fewer clinical infections—given the primacy of direct contact—remains incompletely resolved and is discussed further below [17,25].

Aquatic sports merit separate consideration because water introduces its own pathogens. Leptospirosis is acquired through contact with water contaminated by infected animal urine or tissue; it usually begins as a mild febrile illness with chills, headache and myalgia but progresses in about 10% of cases to severe disease with hepatic and renal failure and bleeding, and outbreaks have occurred among rafters, swimmers, kayakers and triathletes, with treatment by doxycycline, amoxicillin or ceftriaxone and prevention by protective clothing [6]. Otitis externa (swimmer's ear), caused mainly by Pseudomonas aeruginosa but also by Staphylococcus aureus or Aspergillus, occurs several times more frequently in swimmers than non-swimmers because

prolonged water exposure softens the canal and removes protective cerumen; treatment combines cleaning with topical antimicrobials and, in severe cases, systemic therapy [1,6]. Swimming-pool granuloma, caused by Mycobacterium marinum entering through damaged skin, produces indolent nodules and ulcers on the hands, fingers and legs and is treated with oral clarithromycin or minocycline [1,6].

The largest epidemiological study of superficial bacterial infection in an aquatic sport quantified these risks and their behavioural determinants. Among 1047 Greek competitive swimmers, impetigo was the most frequent condition (10.9%), peaking in summer and predominating in the youngest age group, followed by pitted keratolysis (3.2%) and folliculitis (2.7%) [22]. Multivariate analysis linked specific behaviours to specific infections: sharing fins increased the odds of folliculitis (odds ratio 2.611), while placing clothing on locker-room benches (odds ratio 2.097), sharing kickboards (odds ratio 1.894) and sharing flip-flops (odds ratio 2.109) roughly doubled the odds of impetigo, and barefoot walking and bench use were associated with pitted keratolysis [22]. Pseudomonas-associated hot-tub folliculitis accounts for about 80% of water-related folliculitis, thriving in warm, humid conditions at temperatures up to 41 degrees Celsius [22]. A recurrent and concerning finding was that many infected swimmers continued to train during treatment, potentially facilitating transmission, which reinforces the need for education on hygiene, transmission pathways and return-to-play [22].

The evidence that structured environmental programmes reduce microbial burden is now reasonably strong, even if the downstream effect on clinical infection is less firmly established. The three-phase athletic-training-room protocol proceeded from installation of hand and surface disinfectants, through the addition of posters and daily checklists, to targeted education of trainers, coaches and athletes; overall bacterial aerobic plate counts fell by 94.7% (95% confidence interval 72.6-99.0), with reductions of 96.3% in high schools and 92.2% in colleges, MRSA and vancomycin-resistant enterococcus fell from 24% of surfaces to zero, and influenza, initially detected on 25% of surfaces, became undetectable after the educational phase [17]. Importantly, the introduction of products alone (phase one) produced only a modest, non-significant reduction and was accompanied by a slight rise in MRSA and VRE detection, because athletes did not consistently use the newly available resources; the significant improvement followed the educational phases, echoing survey findings that about 35% of athletic trainers performed inadequate hand hygiene and many were unaware of appropriate disinfectants [17]. On wrestling mats specifically, residual disinfectants reduced bacterial load by 76% relative to non-residual cleaners, and pre-practice alcohol-based hand gel reduced surface bacterial load by roughly 78%, providing practical, evidence-based targets for facility protocols [25,27].

4.5. Environmentally acquired infections in outdoor athletes

Athletes who train and compete outdoors—cross-country runners, triathletes, adventure racers and others in rural settings—may acquire infections with prominent cutaneous manifestations from arthropod vectors and contaminated water. Tick-borne diseases include Lyme borreliosis, whose early sign is the expanding erythema migrans rash, and the rickettsial illnesses, of which Rocky Mountain spotted fever characteristically produces a maculopapular rash spreading in a centripetal pattern accompanied by fever, headache and myalgia; as many as 60-75% of

rickettsial cases are initially misdiagnosed because they mimic viral syndromes, and Lyme disease generally requires at least 24 hours of tick attachment for transmission [26]. Prevention relies on understanding regional epidemiology and seasonality, wearing protective light-coloured clothing with trousers tucked into socks, applying appropriate repellents to skin and permethrin to clothing, and performing careful tick checks with correct tweezer removal after exposure [26]. Mosquito-borne flaviviruses such as West Nile virus and water-associated bacteria such as Leptospira further extend the environmental exposures of outdoor and aquatic athletes, reinforcing that the differential diagnosis of an athletic rash must include the training environment as well as the pathogens transmitted by contact [6,26].

5. Viral infections

5.1. Herpes gladiatorum

Herpes gladiatorum, cutaneous infection with herpes simplex virus (HSV), is the signature viral infection of wrestling; between 94% and 97% of cases are due to HSV type 1 [7,9]. The estimated annual incidence in collegiate wrestlers is 20–40%, and from 1993 to 2004 herpes gladiatorum was the single most common cutaneous infection causing lost practice time in National Collegiate Athletic Association wrestlers, accounting for 40.5% of such infections [7,9]. Transmission is almost exclusively by direct skin-to-skin contact; multiple studies indicate that mats and other fomites do not significantly contribute to spread [7,9]. A crucial epidemiological feature is the discrepancy between infection and awareness: 29–30% of high-school wrestlers are infected with or colonised by HSV but only about 3% are aware of it, and up to 87% of outbreaks are subclinical, so that asymptomatic shedding seeds large outbreaks at inopportune times such as training camps and post-season tournaments [7,11].

Clinically, primary herpes gladiatorum often begins with systemic prodromal features—malaise, low-grade fever, sore throat and tender cervical lymphadenopathy—followed within one to two days by clusters of vesicles on an erythematous base [7,9]. Lesions predominate on body surfaces that contact the opponent (face, head, neck, ears and upper extremities) and frequently reflect the wrestler's handedness and preferred tie-up position; most infections manifest within eight days of exposure [7,9]. Ocular involvement is a serious concern, as herpetic keratitis can cause corneal scarring and, rarely, blindness [7,9]. Because lesions are often abraded during competition, presentations are atypical and misdiagnosis is common—up to 90% at first presentation—so microbiological confirmation is strongly recommended; polymerase chain reaction offers near-perfect specificity and high sensitivity, while viral culture is cheaper but less sensitive, and both are preferred over serology [7,9]. Treatment is with oral antivirals (aciclovir, valaciclovir or famciclovir) begun within 24 hours of symptom onset, which hastens resolution and reduces transmission; recurrent outbreaks are milder, shorter and require lower doses [7,9,11]. Season-long chemoprophylaxis is a distinctive and evidence-based feature of wrestling medicine: a 10-year study at a 28-day camp demonstrated that daily oral valaciclovir reduced recurrent herpes gladiatorum by about 89.5% and prevented acquisition in HSV-naïve wrestlers, with dosing of 500 mg daily for wrestlers whose most recent infection was more than two years earlier and 1 g daily for more recent infection [7,9,11].

Several practical points refine the diagnosis and testing of herpes gladiatorum. The incubation period from exposure to lesion appearance is short—typically four to seven days in outbreak settings, with most infections manifesting within eight days—so post-exposure vigilance is time-limited but critical [7,9]. When testing, an intact vesicle should be unroofed and its base scraped within the first 24 to 48 hours, using a swab without calcium alginate (which inhibits HSV) and with a plastic or metal shaft (wood may be toxic to the virus in culture) [9]. Viral-culture sensitivity varies with lesion stage—about 52-93% for vesicles, 41-72% for ulcers and only 19-27% for crusted lesions—whereas real-time polymerase chain reaction offers 98-100% sensitivity and specificity at higher cost, so the choice should be guided by lesion stage, availability and expertise, with sampling performed as early as possible [9]. Ocular vigilance is essential: in a Minnesota high-school outbreak approximately 8% of infected wrestlers developed ocular involvement, and herpetic keratitis is a recognised infectious cause of blindness, so the eyes and the oral and nasal cavities should always be examined when herpes gladiatorum is suspected [7,9]. Because up to 87% of outbreaks are subclinical and roughly 1-5% of seropositive individuals shed virus asymptomatically, direct testing of active lesions is preferred over serology, which cannot reliably distinguish active disease [7,9].

The history of herpes gladiatorum is essentially a history of dramatic camp and team outbreaks that shaped current practice. The term, coined in 1964, gained prominence after a 1989 report in which 60 of 175 wrestlers at a 28-day high-school wrestling camp contracted the virus, forcing the public-health department to close the camp; similar outbreaks in 1999, 2001 and 2007 repeatedly closed the same camp [6,7]. In 2007 the Minnesota State High School League mandated an eight-day suspension of wrestling when 24 wrestlers across multiple teams developed the disease, and by the end of the quarantine 56 had been diagnosed [7]. Reviews of outbreaks across competitive sport confirm that herpes simplex, together with Staphylococcus aureus, has historically been among the most common outbreak pathogens, while the emergence of community-associated MRSA has more recently shifted the balance toward bacterial and dermatophyte outbreaks [7,8]. These recurrent episodes exemplify the twin drivers of transmission—high subclinical prevalence and the intense, sustained contact of camps and tournaments—and they justify both the aggressive isolation policies and the season-long antiviral prophylaxis now recommended for at-risk wrestlers, ideally begun at least five days before a season, camp or tournament to ensure adequate ganglionic drug levels [7,9].

Herpes simplex virus type 1 is highly prevalent in the general population—an estimated 40-63% of United States adults carry it—so the wrestler's disease is a contact-specific manifestation of a ubiquitous virus rather than a sport-specific pathogen [5]. After primary infection the virus travels to the sensory dorsal root ganglion and establishes lifelong latency, and reactivation can be triggered by stress, cold, fever, ultraviolet exposure and corticosteroids; viral shedding may persist for weeks after visible lesions resolve, and recurrence occurs at sites of prior involvement [5]. Clinically, HSV-1 produces several patterns relevant to athletes: herpes labialis around the lips, herpetic keratitis of the eye, herpetic whitlow on the fingers, herpetic sycosis of the bearded area, and herpes gladiatorum of the torso, head and neck [5]. The incubation period for herpes gladiatorum is 2 to 20 days, with vesicles appearing three to five days after contact; recurrent disease has been identified in about 29% of high-school and

20-40% of collegiate wrestlers, and prophylactic valaciclovir in wrestling camps has reduced recurrent outbreaks by approximately 87% [5]. Suppressive therapy is therefore recommended for seropositive individuals for the duration of the season, and, as with all HSV infections, merely covering an active lesion is insufficient to permit competition [5,9].

The first documented outbreak of herpes gladiatorum in Thailand, investigated across three Phuket Thai-boxing gyms in 2022, illustrates several of these principles in a combat-sport context distinct from wrestling [14]. Of 114 individuals examined, ten had rashes consistent with herpes gladiatorum and nine were confirmed by reverse-transcription polymerase chain reaction; all cases were non-Thai nationals from high-income countries, with attack rates among non-Thai trainees ranging from 4.8% to 33.3% while no Thai trainer or trainee was affected [14]. Lesions predominated on the right forearm (78%) and right side of the face (56%), a distribution matching the clinching techniques in which trainees grasp the opponent's neck, echoing the handedness-related pattern seen in wrestling [14]. Environmental sampling detected HSV-1 on target pads, a boxing-ring rope and an uncleaned sandbag, but only at high cycle-threshold values and without culturable virus, indicating inactive remnants and a low surface-transmission risk consistent with the wrestling literature [14]. Genomic sequencing placed all isolates in the East Asian Clade II, suggesting local acquisition, and two epidemiologically linked Gym II cases shared a phylogenetic node [14]. The investigators proposed that lower hygiene compliance and lower HSV-1 seroprevalence—hence lower immunity—among affluent non-Thai trainees, rather than any environmental factor, explained their higher attack rates, and they highlighted that economic pressure led two infected fighters to compete with active lesions despite pre-fight examination rules [14]. This last observation is a pointed reminder that biological control measures can be undermined by socioeconomic and regulatory failures [14].

The Phuket investigation warrants closer examination because it is the most detailed recent outbreak study and illuminates host factors rarely captured elsewhere. The joint investigation team applied graded case definitions—suspected cases with rash, confirmed cases with positive reverse-transcription polymerase chain reaction for HSV-1, and risk contacts with direct skin contact or shared equipment—and tested specimens for HSV-1, HSV-2, mpox virus, varicella-zoster virus and Treponema pallidum, all of which except HSV-1 were negative [14]. Attack rates differed markedly between gyms (21.4% at Gym I, 11.5% at Gym II and 2.6% at Gym III) and were consistently higher among non-Thai trainees [14]. All three non-Thai trainees whose blood was tested had undetectable HSV-1 antibodies, consistent with declining HSV-1 seroprevalence in high-income countries, whereas the unaffected Thai trainers, of lower socioeconomic status, would be expected to have higher antibody levels and greater immunity; differences in hygiene behaviour (four of nine non-Thai trainees rarely showered before training, versus nine of ten Thai trainers who regularly did) may have compounded this immunological gap [14]. Several cases were initially misdiagnosed as bacterial skin infection by clinics or pharmacies, delaying diagnosis and permitting continued transmission, and the cluster was first notified as suspected mpox—an illustration of how heightened surveillance for one rash-causing disease can surface another [14]. The public-health response included isolation and health education, distribution of educational materials to all Phuket gyms and

stadiums, establishment of rash-disease surveillance, and recommendations for stricter pre-fight examinations and rules barring trainees with active infection from training and competition [14].

5.2. Human papillomavirus infections (verrucae)

Common, plantar and flat warts, caused by human papillomavirus and transmitted by direct contact or via contaminated surfaces, are frequent in athletes; moist environments and epidermal micro-trauma favour their development, and plantar warts are especially associated with swimming-pool and shower-room floors [1,5,7]. In the survey of Greek competitive swimmers, plantar warts were reported by 36.5%—far above general-population estimates of roughly 7–12%—and were significantly associated with walking barefoot on the pool deck, placing towels on benches and sharing fins [28]. Treatments include curettage, cryotherapy, salicylic and other topical acid preparations, cantharidin and, for resistant lesions, intralesional agents; for return to competition, lesions are curetted or covered, with facial lesions covered by a mask [1,5,7]. Because ablative techniques may delay return to sport, topical therapy is often preferred for athletes who cannot miss training [1].

5.3. Molluscum contagiosum

Molluscum contagiosum, caused by a poxvirus, presents as small, dome-shaped, umbilicated flesh-coloured papules and is transmitted by skin-to-skin contact among wrestlers and by pool exposure among swimmers [1,5,7]. Because no single medical therapy is convincingly superior for immunocompetent individuals, physical destruction by curettage is the preferred treatment in the training-room setting, after which the site is covered to allow return to play; cryotherapy and topical agents such as imiquimod are alternatives [1,7,9].

The wider range of destructive and topical options reflects how frequently these viral lesions occur in athletes and the competing priorities of eradication and prompt return to play. For warts, curettage, electrocoagulation, carbon-dioxide laser, cryotherapy and photodynamic therapy are effective but may delay return to sport, so topical salicylic or lactic acid, imiquimod or cantharidin—carrying a lower risk of side effects—are often preferred for athletes who cannot miss training, with flat warts treated by topical retinoids and resistant warts by intralesional Candida antigen, bleomycin, 5-fluorouracil or cidofovir [1,9]. For molluscum contagiosum, curettage, electrocoagulation, cryosurgery or laser therapy achieve rapid physical removal, while topical imiquimod, 5% potassium hydroxide, tretinoin, cantharidin, trichloroacetic acid or 5-fluorouracil are non-destructive alternatives; untreated lesions may persist for six to nine months, so athletes should be instructed in self-examination and prompt reporting to minimise both lost training time and onward spread [1,7]. Across all these viral dermatoses, the recurring management principle is to treat early, cover or remove the lesion to permit safe participation, and isolate until the athlete is no longer contagious, balancing individual welfare against the risk to opponents [7,9].

5.4. Blood-borne viruses and other viral considerations

Although the risk of transmitting human immunodeficiency virus during sport is extremely low—estimated at less than one in 85 million game contacts in American football, with only isolated documented cases—universal blood-borne pathogen precautions remain essential:

bleeding wounds should be covered, contaminated surfaces cleaned with appropriate disinfectants, and gloves worn when handling bodily fluids [7,26]. Hepatitis B virus is considerably more transmissible than either human immunodeficiency virus or hepatitis C virus, with documented sports transmission including an outbreak among sumo wrestlers, and is preventable by vaccination [7,26]. Reviews of infectious disease in athletic facilities additionally note that emerging viral pathogens transmitted by close contact, such as mpox, may present in contact-sport athletes and should be considered in the differential diagnosis of atypical rashes [9,25].

Varicella-zoster virus, though less common than herpes simplex, causes competition-disqualifying disease in athletes. Primary infection (chickenpox) produces a diffuse pruritic eruption of erythematous papules and surface vesicles creating a dew-drop-on-petal appearance, arising in successive crops; reactivation produces dermatomal herpes zoster [9]. Infected individuals remain contagious until all lesions have crusted, usually four to five days after rash onset, and both forms may carry systemic manifestations; return-to-competition criteria require lesions to be covered by a firm adherent crust with no secondary bacterial infection [9]. Widespread childhood vaccination has reduced varicella incidence substantially, affording many contemporary athletes primary protection, and early oral aciclovir can shorten the course and hasten return [9].

Human papillomavirus produces three principal patterns of cutaneous wart relevant to athletes: common warts (frequently HPV-2) as hyperkeratotic papules on the hands; plantar warts (HPV-1, the myrmecia type) as painful endophytic lesions especially in swimmers whose macerated soles favour spread; and flat warts (HPV-3 and HPV-10) arranged linearly through the Köbner phenomenon on the hands and face [1,9]. The high plantar-wart prevalence in swimmers (36.5% in the Greek cohort, against roughly 7-12% in the general population) reflects communal showers, barefoot walking on abrasive pool surfaces and equipment sharing, with a winter and spring peak coinciding with intensive training [28]. The same swimmer cohort illustrates that ultraviolet exposure, rather than the pool itself, drives herpetic disease: herpes labialis affected 8.2%, and sunburn was strongly associated with herpes labialis (odds ratio 3.156), genital herpes (odds ratio 14.103) and herpes zoster (odds ratio 12.513), while non-use of sunscreen independently raised the risk of herpes labialis (odds ratio 1.493) [28]. Herpes zoster (1.9%) and the sexually transmitted herpes simplex and papillomavirus infections occurred chiefly in adult swimmers and were linked to sun exposure, physical fatigue and competitive stress, all of which may impair immune control of latent virus—observations that extend the relevance of ultraviolet protection and stress management to the prevention of viral dermatoses in outdoor and aquatic athletes [28].

Molluscum contagiosum, a poxvirus infection, is more prevalent in swimmers (about 7.5%) than non-swimmers (about 3.6%) and spreads by skin-to-skin contact among wrestlers; because no medical therapy is convincingly superior, curettage of the umbilicated papules with subsequent covering is preferred for prompt return to play [1,5,7]. Blood-borne viral infections, though feared, are rare in sport. The risk of transmitting human immunodeficiency virus in American football has been estimated at less than one in 85 million game contacts, with only isolated documented cases, whereas hepatitis B virus—present at far higher blood

concentrations and more stable on surfaces—is 50 to 100 times more transmissible, with documented outbreaks among sumo wrestlers and American football players; percutaneous transmission risk in healthcare is 0.2-0.5% for human immunodeficiency virus but 2-40% for hepatitis B virus [7,26]. Hepatitis B is vaccine-preventable, and for all blood-borne pathogens universal precautions—covering bleeding wounds, cleaning contaminated surfaces with soap and water or a 1:10 bleach solution, and gloving—remain the mainstay, with no major governing body restricting participation for athletes infected with human immunodeficiency virus or hepatitis C [7,26].

6. Fungal infections

6.1. Tinea gladiatorum

Tinea gladiatorum—dermatophytosis of contact-sport athletes—is the most widespread fungal skin infection in this population and, in wrestlers, the second most common cutaneous infection overall after herpes gladiatorum [12]. The predominant causative organism is the anthropophilic dermatophyte Trichophyton tonsurans, which invades keratinised tissue and spreads chiefly by skin-to-skin contact, favoured by abrasions, occlusion and the often asymptomatic course of the disease [9,12]. A systematic review of 22 studies found that the prevalence of positive mycological results among wrestlers ranged extraordinarily widely—from 2.4% in Iran to 100% in the United States—with comparable heterogeneity within single countries, a variability that reflects differences in screening intensity, diagnostic method and the degree of team penetration required to identify a team for study [12]. Reported outbreaks have similarly documented penetration of one-half to three-quarters of a team's members once infection is present [18,23]. Trichophyton tonsurans accounted for more than 90% of isolates in the largest single series, from Tehran, where infection was also documented in family members of infected wrestlers, underscoring a domestic dimension to contagiousness [12,18].

Lesions are typically well-demarcated, erythematous, scaly annular plaques with raised borders and central clearing, located on the trunk, head, neck and upper extremities—the points of contact between competitors—rather than the lower limbs [9,12]. In athletes, however, the classic ring is frequently absent because lesions are examined early or abraded during competition, and the infection may mimic impetigo, atopic dermatitis, psoriasis or early herpes gladiatorum [9,12]. Two case reports illustrate diagnostic pitfalls: tinea corporis gladiatorum may present as a Majocchi granuloma when inappropriate topical corticosteroids drive dermatophytes deeper into the follicle, delaying diagnosis for years, and inappropriate corticosteroid use may also produce an atypical, extensive "tinea incognito" in which mycological confirmation and prompt antifungal therapy are decisive [29,30]. Asymptomatic carriers, with the scalp serving as a reservoir, sustain circulation of the pathogen within teams and complicate control [9,12]. A single case in a mixed-martial-arts fighter whose training partners had recently been diagnosed underscores that the disease is not confined to wrestling but extends across grappling and striking disciplines [31].

Diagnosis rests on clinical examination supported by potassium hydroxide microscopy of skin scrapings (sensitivity approximately 88%, specificity approximately 95%) and, where necessary, fungal culture, which is more specific but requires up to three weeks; molecular methods and, for scalp disease, dermoscopy provide additional accuracy [9,12]. Treatment of localised tinea corporis is with topical antifungals for at least two weeks—fungicidal allylamines such as terbinafine being more efficacious than fungistatic imidazoles—while diffuse, inflammatory or scalp disease requires oral therapy with terbinafine, itraconazole, fluconazole or griseofulvin, with monitoring for hepatotoxicity [9,12,23]. Because compliance with topical regimens is poor and disease is often widespread, systemic therapy and, importantly, chemoprophylaxis have strong support: a 10-year prospective study of high-school wrestlers found that oral fluconazole prophylaxis reduced the incidence of tinea gladiatorum from 67.4% to 3.5% without significant adverse events, and itraconazole pulse therapy has likewise prevented active disease over a season [7,9,12]. Prophylactic fluconazole for in-season wrestlers carries a high level-of-evidence rating in dermatology guidance for sport [9].

The role of the wrestling mat as a source of infection remains genuinely contested and exemplifies the value of a critical, comparative reading of the literature. Some investigators repeatedly isolate T. tonsurans from mats and infer a meaningful role in transmission, whereas others recover no dermatophytes at all; a study of Isfahan sport centres recovered Trichophyton rubrum and Trichophyton mentagrophytes—not T. tonsurans—from 57% of sampled mats, complicating any simple mat-to-athlete narrative [18,32]. The prevailing synthesis is that mats may act as a vector but not as a reservoir if cleaning is adequate, and that the predominance of trunk, scalp and facial lesions—rather than the plantar distribution that constant foot–mat contact would predict—argues against surfaces as the principal route and in favour of direct contact [12,32]. This inference is reinforced by laundering data: controlled experiments show that machine laundering at 60 °C reliably eliminates T. tonsurans, T. rubrum and T. interdigitale conidia from contaminated textiles, whereas heat drying, freezing to −20 °C and direct dry heat at 60 °C do not, indicating that hot-water laundering—rather than drying or freezing—is the decisive hygienic step for fomites [19]. Finally, the epidemiology of T. tonsurans is not static: a multicentre German study documented a marked rise in T. tonsurans infections between 2018 and 2023, linked in part to barbershop transmission and affecting predominantly young males, a shift that has prompted calls to adapt empirical treatment of tinea capitis toward terbinafine and that signals the organism's continuing spread beyond the traditional wrestling niche [20].

6.2. Tinea pedis, tinea cruris and tinea corporis of non-contact origin

Beyond gladiatorum-pattern disease, athletes are prone to "athlete's foot" (tinea pedis), tinea cruris and tinea corporis through warm, moist, occluded environments and communal facilities [1]. Tinea pedis, most often caused by Trichophyton rubrum and Trichophyton interdigitale, presents in interdigital, hyperkeratotic (moccasin) and vesiculobullous forms, may spread to the nails as onychomycosis, and predisposes to secondary bacterial infection through epidermal fissuring [1,21]. Management combines topical antifungals—imidazoles, allylamines or ciclopirox—with systemic terbinafine, itraconazole or fluconazole for extensive, chronic or recalcitrant disease, alongside footwear disinfection and rigorous drying [1,21]. A focused review of terbinafine concluded that both topical and oral formulations are effective and

generally safe for tinea pedis and onychomycosis, that a novel film-forming solution can achieve cure after a single application, and that fractional carbon-dioxide and neodymium-doped yttrium-aluminium-garnet lasers may usefully augment terbinafine for onychomycosis in patients unsuited to systemic therapy [21]. Crucially, that review also flagged emerging resistance—18.6% of dermatophyte isolates in a United States laboratory showed terbinafine resistance, chiefly Trichophyton rubrum and Trichophyton indotineae—and identified diabetic athletes as a group requiring particular vigilance, themes that resonate with the resistance concerns raised for tinea gladiatorum [12,21].

6.3. Pityriasis versicolor

Pityriasis versicolor, a superficial mycosis caused by overgrowth of commensal Malassezia yeasts, is relevant to athletes because the predisposing factors—heat, humidity, hyperhidrosis and increased physical activity—are intrinsic to sport [33]. It presents as hypo- or hyperpigmented macules on seborrhoeic areas of the trunk, arms and neck, mainly in adolescents and young adults, with prevalence ranging from about 1% in temperate regions to as high as 50% in the tropics [33]. Diagnosis is usually clinical, confirmed where necessary by potassium hydroxide microscopy showing the characteristic "spaghetti-and-meatballs" appearance, by yellow-green fluorescence under Wood's light, or by dermoscopy [33]. Topical azoles, selenium sulphide, zinc pyrithione and terbinafine are first-line for localised disease, while oral itraconazole or fluconazole is reserved for extensive or recurrent cases; oral terbinafine is ineffective because it does not reach adequate concentrations in the stratum corneum through sweat [33]. High recurrence rates—frequently exceeding 40% within a year—and the absence of a universally accepted gold-standard regimen make pityriasis versicolor a persistent management challenge, and prophylactic strategies remain an area of active investigation [33].

6.4. Pathophysiology, risk factors and diagnosis of tinea gladiatorum

Trichophyton tonsurans is a keratinophilic, keratinolytic anthropophilic dermatophyte that adheres to keratinised tissue and adapts to the local environment through fungal enzymes—collagenolytic, elastolytic and mucolytic proteases, lipases and nucleotidases—whose activity is regulated in response to host factors such as skin pH [12]. Heat-shock proteins and transcription factors enable the organism to tolerate the initially acidic surface and to gradually alkalinise it, whereupon keratinolytic proteases operate at maximum efficiency and sustain the infection [12]. In the scalp the fungus invades the hair shaft as endothrix hyphae that convert to arthroconidia, producing the black-dot appearance, and melanin synthesis by the fungus may act as an antioxidant that protects it from ultraviolet damage in sun-exposed lesions [12]. Recognised risk factors include age, sex, hyperhidrosis, failure to wear headgear, infrequent laundering of clothing and bedding, and training in tight, enclosed, humid spaces; the disease has a domestic dimension, since it may spread to family members, an important consideration in crowded households [12,18]. Like other visible dermatoses, tinea gladiatorum carries a quality-of-life burden—embarrassment, low self-esteem, anxiety and depression—with a mean Dermatology Life Quality Index of about 13 in one study of dermatophytosis, influenced by disease severity and body-surface involvement [12].

Diagnosis proceeds from history and examination to laboratory confirmation. Direct microscopy of skin scrapings, hair or nail in 10-20% potassium hydroxide reveals septate hyphae; culture on Sabouraud dextrose agar with cycloheximide identifies the species but takes up to three weeks; Wood's light screens for Microsporum, and dermoscopy distinguishes tinea capitis by features such as comma-shaped hairs [9,12]. Molecular methods—polymerase chain reaction fingerprinting, restriction-fragment-length polymorphism and ribosomal DNA sequencing—and matrix-assisted laser desorption/ionisation time-of-flight mass spectrometry offer rapid, accurate identification, the latter comparable to sequencing though dependent on reference-spectra libraries [12]. Because early or corticosteroid-modified lesions are frequently atypical, mycological confirmation is emphasised; the Majocchi granuloma and tinea incognito described above are the direct clinical consequence of omitting it and treating dermatophytosis with topical steroids [12,29,30]. The two-case series of a basketball player with extensive, corticosteroid-modified tinea corporis and a Kung-Fu athlete with three small annular lesions on the right side of the body illustrates how sport-specific exposure and individual factors shape the same infection into different clinical pictures, both caused by T. tonsurans [30].

Treatment stratifies by extent. Localised tinea corporis responds to topical antifungals applied for at least two weeks; a meta-analysis of 65 randomised trials found no statistically significant difference between agents in end-of-treatment mycological cure, but for durable recovery the allylamines butenafine and terbinafine were superior to clotrimazole, oxiconazole and sertaconazole, and terbinafine was superior to ciclopirox [12]. Diffuse, inflammatory, scalp or self-inaccessible disease requires oral therapy—itraconazole 100-200 mg daily or terbinafine 250 mg daily for one to two weeks, with fluconazole 150-200 mg weekly or griseofulvin as alternatives requiring longer courses—under monitoring for hepatotoxicity [9,12]. In-vitro testing of T. tonsurans from wrestlers found tolnaftate and itraconazole most potent and fluconazole weakest, a discordance with clinical practice that reflects the additional pharmacokinetic and adherence considerations of real-world use [12]. Of growing concern is antifungal resistance and tolerance, mediated by drug efflux, target mutation, chaperone overexpression and biofilm formation and promoted by long or interrupted therapy; cross-resistance between allylamines and azoles has been reported, and a terbinafine-resistance rate of 32% was found among Trichophyton interdigitale isolates, foreshadowing therapeutic challenges also seen in tinea pedis [12].

6.5. The mat-contamination debate and the decontamination of fomites

The role of surfaces in transmitting dermatophytes has been examined repeatedly with conflicting results, and a careful reading exposes the reasons. The large Tehran series confirmed T. tonsurans in 612 of 893 wrestlers (68.5%), most between 10 and 20 years of age, with tinea corporis in 10.8% of family members and clustering in clubs of southern and south-eastern Tehran, and attributed transmission primarily to skin-to-skin contact [18]. Environmental sampling has yielded discordant findings: some investigators recover T. tonsurans from mats and infer a transmission role, whereas a study of Isfahan sport centres recovered Trichophyton rubrum and Trichophyton mentagrophytes—not T. tonsurans—from 57% of sampled mats, and other authors recover no dermatophytes at all [18,32]. The predominance of trunk, scalp and facial lesions, rather than the plantar distribution that constant foot-mat contact would predict,

argues against surfaces as the principal route, and the consensus is that mats may serve as a vector but not as a reservoir when cleaning is adequate [12,32].

Controlled decontamination experiments give this debate a practical resolution. Testing the survival of T. tonsurans, T. rubrum and T. interdigitale conidia on contaminated textiles, machine laundering at 60 degrees Celsius reliably eliminated all three species, whereas laundering at 40 degrees, heat drying in domestic or laundromat machines, freezing at minus 20 degrees for 24 hours to one week, and direct dry heat at 60 degrees for 10 to 90 minutes all failed to kill the conidia [19]. Temperature monitoring revealed that the drying and heating processes rarely reached or sustained 60 degrees, and the authors concluded that repeated rinsing and water discharge during laundering—rather than heat, drying or freezing alone—is the decisive factor in removing dermatophytes from fomites [19]. The practical implication is unambiguous: hot-water laundering, not tumble-drying or freezing, is the reliable hygienic step for contaminated clothing, towels and bedding, complementing the direct-contact precautions that address the primary route [12,19].

Finally, the epidemiology of the causative organism is shifting in ways that extend its relevance beyond the wrestling room. A multicentre German study comparing 2018 with 2023 found that, while Trichophyton rubrum remained the overall dominant dermatophyte (falling from 78.7% to 66.3% of isolates), tinea capitis doubled from 4.3% to 9.3% of dermatophytoses, and T. tonsurans emerged as the leading agent of tinea capitis (67.6% of cases in 2023) and the second most common in tinea corporis (26.3%), affecting predominantly young males [20]. The rise was linked in part to transmission through shared shavers in barbershops, prompting recommendations to adopt terbinafine for empirical treatment of tinea capitis and to intensify screening and hygiene [20]. Historically, local German outbreaks of T. tonsurans were associated with wrestling, so its spread into the general population through grooming practices represents a continuation of the same transmissible organism into new niches and reinforces the wider public-health importance of the sport-derived literature [12,20].

6.6. Tinea pedis, onychomycosis and the terbinafine evidence base

Tinea pedis and its frequent companion onychomycosis illustrate both the effectiveness and the emerging limitations of antifungal therapy in athletes. A focused review of terbinafine, an allylamine that inhibits squalene epoxidase and accumulates in the stratum corneum and nail plate, summarised extensive trial evidence: oral terbinafine 250 mg daily for two weeks achieved negative mycological findings in about 86% of patients with moccasin-type tinea pedis, and 125 mg daily for four weeks yielded negative results in up to 95% of hyperkeratotic cases; in the Spanish SETTA study, 94% of tinea pedis patients reported significant improvement and 52.7% achieved complete cure [21]. Topical terbinafine outperformed clotrimazole with a shorter one-week regimen, and a novel film-forming solution achieved negative mycological findings in 72-86% of patients after a single application, improving adherence [21]. For onychomycosis, oral terbinafine outperformed topical lacquers, and combining terbinafine with fractional carbon-dioxide or long-pulsed neodymium-doped yttrium-aluminium-garnet laser improved outcomes, offering an option for patients unsuited to systemic therapy [21]. However, the review flagged rising resistance—18.6% of dermatophyte isolates in a United States laboratory over 2021-2022 were terbinafine-resistant, chiefly

Trichophyton rubrum and the emerging Trichophyton indotineae, associated with squalene-epoxidase mutations such as F397L—and a disappointing 30% cure rate in an Indian cohort, signalling that terbinafine may need to yield to alternative therapies in coming years [21]. Diabetes was identified as an independent risk factor for tinea pedis and, through neuropathy, for onychomycosis, so diabetic athletes warrant particular vigilance and structured foot care [21].

Pityriasis versicolor, discussed above as a sweat- and heat-favoured mycosis, has its own well-characterised therapeutic literature that is directly applicable to athletes. Non-specific topical agents (selenium sulphide, zinc pyrithione) act by removing infected stratum corneum or altering cell turnover, while specific agents provide direct antifungal activity: ketoconazole 2% cream achieved mycological cure in about 84% of patients in a placebo-controlled trial, ketoconazole 2% shampoo and topical terbinafine 1% solution (about 81% cure) are effective, and among systemic agents itraconazole 200 mg daily for five to seven days relieves symptoms in roughly 80% of patients while fluconazole 300 mg weekly achieved 93% mycological cure [33]. Two caveats are important for the sports physician: oral terbinafine is ineffective against Malassezia because it is not excreted in sweat and does not reach adequate stratum-corneum concentrations, and oral ketoconazole is no longer recommended for cutaneous mycoses because of hepatotoxicity (approximately one in 500 patients) [33]. Given recurrence rates frequently exceeding 40% within a year, maintenance regimens such as weekly ketoconazole shampoo or monthly fluconazole are used in predisposed individuals, and phototherapy is reserved for patients with contraindications to antifungals [33]. Because heat, humidity and hyperhidrosis—intrinsic to sport—drive recurrence, sustained preventive skin care is as important as the antifungal course itself [4,33].

7. Ectoparasitic infestations

Although less common than bacterial, viral and fungal disease, scabies (infestation with Sarcoptes scabiei var. hominis) and pediculosis (head and body lice) are transmissible by direct contact in contact sports and are competition-disqualifying [7,9]. Scabies presents with intensely pruritic, nonspecific papules and, characteristically, linear burrows in skin folds and interdigital spaces; diagnosis is confirmed by microscopic identification of mites, eggs or faeces in skin scrapings, and treatment is with topical permethrin (with treatment of close contacts and laundering of linens) or oral ivermectin [7,9]. Pediculosis is diagnosed by identifying lice or viable nits and treated with permethrin or pyrethrin preparations, repeated after seven days, together with mechanical removal and laundering [7,9]. Governing-body rules require a negative scabies preparation or confirmed treatment response before return to wrestling [9].

Two features make ectoparasitic infestations easy to miss in the athletic context. First, symptoms are delayed: pruritus and rash in scabies may not appear for three to four weeks after acquisition, so an athlete can transmit the mite before recognising infestation, and the reliable dermatological sign of burrowing tracks is frequently absent [7]. Second, nits alone do not confirm active pediculosis, which requires visualisation of living lice or viable eggs, so treatment decisions should rest on direct identification rather than the presence of empty nit shells [7]. Eradication requires treating not only the athlete but also close contacts and infested

fomites: bedding, towels and clothing should be washed in hot water—the Centers for Disease Control and Prevention recommend temperatures above roughly 50 degrees Celsius—to prevent reinfestation, an environmental principle that parallels the laundering evidence for dermatophytes [7,9]. Although these infestations are uncommon in wrestling relative to bacterial, viral and fungal disease, they are competition-disqualifying and are readily controlled once recognised, so awareness during routine skin checks is the key to limiting spread [7,9].

8. Return-to-competition regulations and their limitations

Because skin infections are readily transmissible during contact, governing bodies—chiefly the National Collegiate Athletic Association (NCAA) and the National Federation of State High School Associations (NFHS)—have codified return-to-play criteria, which the sports physician must apply alongside clinical judgment [5,9,23]. For bacterial infections, both organisations generally require no new lesions for 48 hours, at least 72 hours of antibiotic therapy, and no moist, exudative or draining lesions; active infections may not simply be covered to permit participation [5,7,9]. For herpes gladiatorum, criteria include resolution of systemic symptoms, no new vesicles for 72 hours, all lesions surmounted by a firm adherent crust, and a defined course of systemic antiviral therapy—at least 120 hours for the NCAA—again with the stipulation that active lesions cannot be covered for competition [5,9,23]. For tinea, at least 72 hours of topical antifungal therapy is required for tinea corporis and at least two weeks of systemic therapy for tinea capitis, with lesions then covered by a gas-permeable dressing; molluscum contagiosum and verrucae require curettage or secure covering [5,9,23].

The purpose of these criteria is to balance the athlete's health and prompt return against the risk of transmission, and the disease burden they seek to contain is substantial. Skin infections account for approximately 20% of the time-loss conditions in wrestling, and although around 70% of affected athletes lose less than one week of practice, the cumulative effect of disqualification, antimicrobial therapy and inconveniently timed outbreaks is considerable [6,10]. Severe infections are relatively uncommon—only four of 112 skin infections in one collegiate surveillance cohort resulted in more than three weeks of lost time—but their consequences can be serious, and the timing of transmission is often unfavourable: infections contracted at regional or sectional competition typically become symptomatic 8 to 10 days later, just before championship events, so that athletes may be withheld from the most important competitions of their season [7,10]. This intersection of biological incubation and competitive calendar is a recurring source of post-season confusion and controversy, and it is precisely why early recognition, culturing and, where indicated, prophylaxis carry such practical weight [7,10].

A recurring and clinically important caveat is that covering an active lesion does not render an athlete eligible—an explicit provision aimed at the temptation to conceal contagious disease to permit competition [5,9]. The limits of regulation are exposed when enforcement fails: in the Phuket outbreak, two infected fighters competed with active herpes lesions—one with an open forehead wound—because of financial pressure and inadequate enforcement of pre-fight examination rules, demonstrating that even well-designed return-to-play criteria depend on surveillance, education and socioeconomic conditions that permit athletes to rest when unwell

[14]. Weekly skin checks that exceed minimum competition requirements, prompt referral of suspicious lesions, and a culture in which athletes report rather than hide lesions are therefore integral to the effectiveness of any return-to-play framework [9,10].

The two national frameworks differ in emphasis in ways the clinician should know. For primary herpes, the National Collegiate Athletic Association requires firm adherent crusts, no new blisters for 72 hours, resolution of systemic symptoms and at least 120 hours of antiviral therapy, whereas the National Federation of State High School Associations requires all lesions to be scabbed, no new lesions for 48 hours, and 10 to 14 days of antiviral therapy for cutaneous lesions (or longer with systemic symptoms); for recurrent herpes both require crusting and about 120 hours of therapy [7,9]. For tinea, the collegiate guideline specifies at least 72 hours of topical therapy for tinea corporis and two weeks of systemic therapy for tinea capitis, permitting covered lesions to compete, while the high-school guideline is broadly similar [5,9]. Molluscum contagiosum requires curettage and covering, and verrucae require covering or curettage, with multiple uncoverable facial lesions grounds for disqualification under some rules [5,7]. USA Wrestling additionally requires final clearance by the team physician and reiterates that merely covering active lesions is insufficient [5,9].

Two principles cut across these detailed criteria. First, the decision to withhold an athlete should be based on the individual's health and only then on transmission risk, and it must be individualised by sport: a tinea infection in a tennis player carries far less transmission risk than the same infection in a wrestler in constant contact with an opponent, so identical lesions may justify different decisions [7,11]. Second, criteria depend on being applied honestly; the temptation to conceal or cover contagious lesions is real, and the Phuket experience—in which fighters competed with active herpes lesions under financial pressure and lax enforcement—demonstrates that even sound regulations fail without surveillance, education and the socioeconomic conditions that allow athletes to rest [11,14]. For this reason, weekly skin checks that exceed minimum competition requirements, promotion of self-examination and prompt referral are recommended as adjuncts to formal criteria [10,11].

9. Prevention

Prevention of infectious skin disease in athletes rests on several complementary pillars: personal hygiene, environmental disinfection, screening and early isolation, education, immunisation and, for selected pathogens, chemoprophylaxis [13,23,26]. Personal hygiene measures include showering with antimicrobial soap immediately after every practice and game, frequent hand hygiene, daily laundering of clothing and equipment, avoidance of shared towels, razors, water bottles and protective gear, and prompt covering of wounds; athletes should be discouraged from cosmetic body shaving, which has been shown to increase the risk of CA-MRSA more than sixfold [6,13,23]. Environmental measures include documented cleaning schedules for mats, benches, treatment tables and floors using appropriately registered disinfectants applied for the correct contact time; a diluted bleach solution is a widely used option, and residual disinfectants outperform non-residual cleaners on wrestling mats [23,25]. Hot-water laundering at or above 60 °C is specifically effective against dermatophyte conidia, whereas drying and freezing are not [19].

Beyond individual behaviour, organisational commitment is a prerequisite for effective infection control. The athletic-training position statement on skin diseases calls for adequate fiscal and human resources, enhanced custodial staffing, provision of antimicrobial liquid (not bar) soap at showers and sinks, written infection-control policies within institutional manuals, and, where feasible, a contracted team dermatologist to assist with diagnosis and management [23]. A clean environment must be maintained through documented cleaning schedules for high-contact surfaces—wrestling mats, treatment tables, locker-room benches and floors—using registered disinfectants applied at the correct dilution and contact time, with a 1:10 household-bleach solution a commonly cited option [23]. Hand hygiene is identified as the single most important practice, and the correct technique is specified: wetting the hands, applying an appropriate amount of antimicrobial product, rubbing vigorously for at least 15 seconds, rinsing and drying with a disposable towel, with alcohol-based rubs acceptable when the hands are not visibly soiled [23]. Athletes should report all abrasions, cuts and lesions for prompt cleansing and dressing, and open, uninfected wounds should be covered with a semiocclusive or occlusive dressing until healed to prevent contamination from infected lesions or surfaces [23]. Because adherence to precautions is demonstrably higher in groups that understand their rationale, education of administrators, coaches, athletes and custodial staff is woven through every recommendation, and a shared culture of institutional safety is regarded as essential to controlling infectious disease [23].

Screening and isolation are central to contact sports: systematic head-to-toe skin examinations before practice and competition allow early identification and removal of infected athletes, and any suspicious lesion should be triaged the same day to a physician or dermatologist [9,23]. Education of administrators, coaches, athletes and custodial staff underpins adherence, since knowledge of transmission routes and correct hygiene demonstrably improves compliance, and structured educational protocols have measurably reduced bio-burden in training rooms [17,23]. Immunisation contributes to prevention of several relevant infections—hepatitis B, varicella, influenza, measles–mumps–rubella and meningococcal disease—particularly given the crowding and travel that characterise elite sport [25,26]. Chemoprophylaxis is uniquely well supported for two pathogens of contact sport: season-long valaciclovir for wrestlers with a history of herpes gladiatorum and oral fluconazole (or itraconazole pulse therapy) for wrestlers prone to tinea gladiatorum, both of which markedly reduce recurrence [7,9,12]. Decolonisation of MRSA carriers, by contrast, is of limited durability: although initial eradication may approach 100%, re-colonisation is common in the persistently exposed athletic environment, so decolonisation must be paired with continuous hygiene rather than viewed as a definitive solution [9,13].

Immunisation is an under-appreciated component of prevention in sport, where crowding, travel and international competition raise exposure. Hepatitis B vaccination, given as a three-dose schedule with 90-95% efficacy and durable immunity, is particularly relevant given the virus's high transmissibility and documented sports outbreaks; annual influenza vaccination (70-90% efficacy in those under 65) reduces cases within teams; meningococcal conjugate vaccine is recommended for dormitory-resident students at elevated risk; and measles-mumps-rubella, varicella and, for eligible individuals, human papillomavirus vaccines contribute to herd

immunity that protects the athletic community [25,26]. Athletes traveling to endemic regions require destination-specific immunisation planned months in advance, and blood-borne and sexually transmitted infections are further mitigated by universal precautions and barrier protection [26]. Concerns that vaccination might impair performance are not supported by evidence: studies in elite athletes show no performance decrement and only transient minor side effects from influenza and severe acute respiratory syndrome coronavirus 2 vaccines, and exercise exerts a net positive immunomodulatory effect on the vaccine response, so that vaccination reduces training days lost to infection more than it costs [25].

Education underpins every other measure. Cross-sectional analysis found that about 35% of athletic trainers did not practise appropriate hand hygiene before assessing each athlete and that 10% were unaware of the infection risk associated with whirlpool use, gaps that structured programmes can close [25]. The consolidated guidance from public-health authorities, synthesised across the athletic literature, is practical and consistent: athletes and staff should wash hands frequently for at least 20 seconds or use alcohol-based gel; shower with soap and water immediately after every training session or game; never share towels, bar soap, razors, clothing or topical ointments from open containers; launder uniforms, towels and clothing after each use following manufacturer instructions (with dermatophyte-contaminated textiles requiring hot-water laundering at or above 60 degrees Celsius); cover cuts and wounds with clean dry dressings until healed; use a barrier such as a towel or clothing on shared surfaces; avoid public water facilities when carrying an open wound or active infection; and ensure regular disinfection of high-contact surfaces with residual disinfectants [13,19,23,25]. Because these measures address the athlete, the team and the environment simultaneously, their combined and consistently enforced application—rather than any single intervention—offers the best prospect of reducing the burden of infectious skin disease in sport [13,17].

10. Critical synthesis and directions for future research

Several themes emerge from a comparative reading of this literature. The first is the striking heterogeneity of reported prevalences—from 2.4% to 100% for tinea gladiatorum and across a similarly broad range for MRSA carriage—which is largely an artefact of differing screening intensity, diagnostic methods (self-report versus clinical examination versus culture or molecular testing) and study populations rather than a true measure of biological variability [12,13,16]. Self-reported studies, such as those in Greek swimmers, are efficient for large cohorts but are subject to recall bias and diagnostic misclassification, whereas culture-based surveillance is more accurate but resource-intensive and prone to selection toward teams already known to be affected [12,22]. Readers should therefore interpret single prevalence figures cautiously and weigh the methodology behind them.

A second theme is the consistent primacy of direct skin-to-skin contact over fomite transmission across pathogen classes. For herpes gladiatorum, mats and equipment contribute little; for tinea gladiatorum, the anatomical distribution of lesions and the discordant mat-sampling results argue against surfaces as the main route; and for MRSA, outbreak investigations repeatedly implicate contact and skin breaches over environmental sources [7,12,15]. This does not render environmental measures useless—training-room disinfection demonstrably lowers bio-

burden—but it does explain why hygiene programmes that neglect direct-contact factors (skin checks, isolation, wound care, avoidance of shared items) achieve limited control, and why interventions targeting the athlete's own skin, such as reducing scratching and micro-trauma, deserve further study [17,24].

A third theme concerns the strength and gaps of the prophylaxis evidence. Chemoprophylaxis for herpes gladiatorum and tinea gladiatorum is among the best-supported interventions in the field, with large longitudinal and controlled studies demonstrating substantial reductions in recurrence [7,9,12]. In contrast, the evidence for MRSA decolonisation in athletes is weak and its benefit transient, and the translation of surface-disinfection gains into reductions in clinical infection has not been definitively established [13,17]. Emerging antimicrobial resistance—both terbinafine resistance in dermatophytes and the continued evolution of CA-MRSA—threatens current regimens and underscores the need for stewardship and for culture- and susceptibility-guided therapy [12,21,27].

It is useful to grade the strength of the principal recommendations, as the sports-dermatology literature does through the Strength of Recommendation Taxonomy. The use of oral fluconazole prophylaxis to reduce the incidence of tinea gladiatorum is supported by consistent, high-quality evidence and carries the strongest rating; microbiological testing of skin lesions to determine the responsible pathogen and guide treatment is likewise strongly supported [7,9]. Oral valaciclovir, both to hasten resolution of herpes gladiatorum begun within 24 hours of symptoms and to prevent recurrence in previously infected wrestlers, and incision and drainage as the treatment of choice for purulent skin and soft-tissue infection, rest on good but somewhat less consistent evidence [7,9]. By contrast, MRSA decolonisation in athletes and several environmental interventions are supported chiefly by expert opinion or limited data and warrant a more cautious rating [9,13]. This gradation should guide clinical emphasis: interventions with the strongest evidence—confirmatory testing, targeted antimicrobial therapy and, for the two classic wrestling pathogens, chemoprophylaxis—deserve priority, while measures of lesser or uncertain benefit should be applied thoughtfully and studied further [7,9,13].

Finally, the literature reveals several under-examined dimensions. Host factors beyond behaviour—including HSV-1 seroprevalence, socioeconomic status and baseline immunity—may materially influence attack rates, as the Phuket outbreak suggested, yet are rarely measured [14]. The skin microbiome of athletes is only beginning to be characterised, and its prospective relationship to clinical infection, together with sex differences in susceptibility, warrants longitudinal study [2]. Female athletes, non-wrestling combat disciplines and aquatic sports remain comparatively understudied relative to male collegiate wrestling, from which much of the evidence base is derived [10,22]. As a narrative review, the present synthesis is itself limited by the non-systematic selection of sources and by the predominance of observational and North American data; these constraints should temper the generalisation of specific figures even as the broad clinical principles remain robust.

A further practical theme is the divergence between the infection spectra of contact and aquatic sports, which should shape screening and prevention. In wrestling and other combat disciplines the dominant threats are herpes gladiatorum, staphylococcal and streptococcal infections and tinea gladiatorum, all favoured by direct skin-to-skin contact and abrasion; in swimming and

other aquatic sports the spectrum shifts toward plantar warts, impetigo, pitted keratolysis, folliculitis and, distinctively, ultraviolet-triggered herpetic reactivation, favoured by moisture, communal wet surfaces, shared equipment and sun exposure [6,22,28]. A prevention programme optimised for one setting is therefore not directly transferable to the other: combat sports demand rigorous skin checks, isolation and chemoprophylaxis, whereas aquatic sports demand attention to footwear, pool-deck hygiene, equipment sharing and photoprotection [22,28].

Finally, the clinician must situate infectious dermatoses within the broader differential diagnosis of athletic skin disease, since misclassification in either direction is harmful. Non-infectious conditions common in athletes—irritant and allergic contact dermatitis from equipment and disinfectants, mechanical lesions such as friction blisters, acne mechanica and talon noir, and inducible urticarias including cholinergic and cold urticaria—may mimic or coexist with infection, and inflammatory dermatoses such as eczema and psoriasis both resemble infection and predispose to it [3,5]. Conversely, the over-diagnosis of bacterial infection at the expense of herpes gladiatorum, repeatedly documented, delays appropriate antiviral therapy and isolation and permits onward transmission [11,14]. Accurate, and where necessary laboratory-confirmed, diagnosis is thus not merely an academic nicety but the pivot on which correct treatment, timely isolation and effective outbreak control all turn [7,9].

11. Key clinical recommendations

The following evidence-informed recommendations distil the practical implications of this review for the physician, dermatologist and athletic trainer caring for athletes in contact and combat sports.

- Maintain a high index of suspicion and confirm atypical or abraded lesions with potassium hydroxide microscopy, culture or polymerase chain reaction before committing to treatment, because over-diagnosis of bacterial infection at the expense of herpes gladiatorum delays antiviral therapy and isolation [9,11].
- Perform systematic head-to-toe skin checks before every practice and competition, exceeding minimum competition requirements where feasible, and isolate athletes with suspicious lesions until they are evaluated and cleared [10,23].
- Treat purulent bacterial lesions primarily with incision and drainage, and guide antibiotic choice for suspected community-associated MRSA by culture, susceptibility testing and local resistance patterns rather than reflex prescribing [9,11].
- Begin oral antiviral therapy within 24 hours for suspected herpes gladiatorum, and offer season-long valaciclovir prophylaxis to previously infected wrestlers, starting several days before camps and tournaments [7,9].
- Use topical allylamines for localised tinea and oral antifungals for diffuse, inflammatory or scalp disease, and consider fluconazole or itraconazole prophylaxis in wrestlers prone to tinea gladiatorum [9,12].
- Launder contaminated clothing, towels and bedding in hot water at or above 60 degrees Celsius, recognising that tumble-drying and freezing are unreliable for eliminating dermatophyte conidia [19].

- Enforce personal and environmental hygiene: shower after every session, avoid sharing towels, razors and equipment, discourage cosmetic body shaving, and disinfect high-contact surfaces with residual disinfectants [13,23].
- Apply return-to-play criteria strictly and consistently; merely covering an active, contagious lesion does not permit participation [5,9].
- Ensure appropriate immunisation—against hepatitis B, influenza, varicella and other vaccine-preventable diseases—and observe universal blood-borne pathogen precautions during bleeding injuries [7,26].
- Educate athletes, coaches, trainers and custodial staff, since adherence to preventive practices and the resulting reduction in microbial burden improve when the rationale is understood [17,23].

12. Conclusions

Infectious skin diseases are a common, consequential and largely preventable problem in athletes, and they fall disproportionately on contact and combat sports, where direct skin-to-skin contact, abrasions, sweat, occlusion and shared environments create ideal conditions for transmission [4,6,7]. The dominant entities are bacterial infections due to Staphylococcus aureus—including community-associated MRSA—and Streptococcus pyogenes, herpes gladiatorum caused by herpes simplex virus type 1, and dermatophytosis due chiefly to Trichophyton tonsurans, complemented by warts, molluscum contagiosum, pityriasis versicolor and, in aquatic sports, a characteristic set of bacterial and viral dermatoses [9,12,22]. Diagnosis is primarily clinical but is frequently confounded by atypical presentations, so that microbiological confirmation—potassium hydroxide microscopy, culture and polymerase chain reaction—should be used liberally when the picture is unclear [7,9].

Effective management combines targeted, increasingly susceptibility-guided antimicrobial therapy, incision and drainage of purulent lesions, and prompt isolation of infected athletes, applied within clearly defined return-to-play criteria that cannot be circumvented by merely covering active lesions [5,9,11]. Prevention is multifaceted and must integrate rigorous personal hygiene, environmental disinfection, systematic skin screening, education, immunisation and, for herpes gladiatorum and tinea gladiatorum, well-evidenced chemoprophylaxis [12,23,26]. The greatest gains are likely to come not from any single measure but from coherent, enforced programmes that address the athlete, the environment and the team simultaneously—recognising that direct contact drives most transmission, that asymptomatic carriage sustains outbreaks, and that socioeconomic and regulatory realities determine whether infected athletes actually rest [10,13,14]. Future research should prioritise prospective and interventional studies—particularly in female athletes, non-wrestling combat disciplines and aquatic sports—the durability of decolonisation, the emergence of antimicrobial resistance, and the clinical significance of the athletic skin microbiome, so that prevention can be placed on an ever firmer evidential footing [2,10,21].

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