

BACTERIAL SPORT-RELATED SKIN AND SOFT-TISSUE INFECTIONS (SSTIs): AN ONGOING PROBLEM AMONG A DIVERSE RANGE OF ATHLETES

Justin J. Mitchell, MD

J. Mark Jackson, MD

Azam Anwar, MD

Steven B. Singleton, MD

Investigation performed at The Steadman Philippon Research Institute, Vail, Colorado

Abstract

» Skin and soft-tissue infections have become increasingly common in the sports world. Recent reports have shown that these infections are prevalent throughout all arenas of sport, and efforts to decrease colonization of bacteria and fungi are now essential for preventing the development of SSTIs.

» Education on cleansing and hygiene are a vital part of this process, and, as such, the team physician and the team athletic trainers play an important role in the education of the athlete and all members of the athletic team.

» The impact of SSTIs on individuals and the athletic team may be severe and have the potential for notable consequences, including loss of playing time, hospitalization, and even surgery. Continued efforts to better understand and to prevent the development of SSTIs are paramount.

Skin and soft-tissue infections (SSTIs) often occur in athletes during training for competitive sports or during actual competition, and the majority of these infections are bacterial or fungal in origin¹. SSTIs can have an immediate impact on athletic eligibility and could threaten the ability to compete or to participate in games during the course of a single season or an entire career. The consequences resulting from restricted participation due to an SSTI may impact win-loss records as well as financial incentives such as scholarships or contracts². In addition to athletes who participate in competitive sports, millions of people regularly visit fitness centers or recreational gymnasiums in an attempt to stay fit and healthy. Studies have shown that these gymnasiums are sources of large quantities of bacteria that could cause SSTIs, which may then impact the lives and work of many people in any community^{3,4}.

The present review describes the mechanisms by which bacterial SSTIs occur in healthy athletes, the prevalence of SSTIs among players in various sports (including the effect of player position and the sites of the body commonly infected in each sport), the mechanisms by which SSTIs are spread, and hygiene measures that are recommended to prevent the spread of infection. Extrapolating these lessons into the general population of so-called weekend warriors or fitness enthusiasts may then help us to better understand, prevent, and treat these increasingly complicated infections.

Mechanisms by Which SSTIs Occur in Healthy Athletes

Studies of the human microbiome have shown that billions of bacteria, fungi, and other microbes inhabit the skin and that these microorganisms vary between individuals and between different sites on the

COPYRIGHT © 2017 BY THE JOURNAL OF BONE AND JOINT SURGERY, INCORPORATED

Disclosure: There was no external funding source. The **Disclosure of Potential Conflicts of Interest** forms are provided with the online version of the article.

skin⁵⁻⁷. In fact, each region of the body's skin is home to its own distinct community of microbes. This microbe "biome" may extend several centimeters out and away from the skin of the body and is subject to temperature, moisture, wind, and movement^{5,6,8}. The characteristics of these various colonies depend on the surrounding environment. Factors such as skin characteristics, sebaceous gland concentration, moisture content, temperature, host genetics, and exogenous environmental factors (including hygiene practices) can influence each community⁹.

A recent study of the human skin microbiome showed that skin-to-skin contact during a sporting event can alter the microbial composition of the skin environment¹⁰. The investigators hypothesized that sports in which contestants have substantial skin-to-skin collisions might disrupt these ecosystems on the skin and allow microbes to be shared among players. They selected roller derby as the model high-contact sport to study. Skaters from 3 geographically different areas were competing in a derby tournament. The researchers took swab samples from the exposed upper arms of skaters from each of the teams before and after matches. DNA analysis following these matches demonstrated that similarities in bacterial communities predicted team membership. Furthermore, when competing teams participated in an hour-long match, their microbial communities became more homogeneous, suggesting that simple contact was enough to change the microbial community makeup of a player, at least temporarily.

Contact sports were also the focus of a study by Jiménez-Truque et al.¹¹. Those investigators evaluated 377 male and female varsity athletes with monthly swabs of the nares and throat at regular intervals over 2 academic years. The study group consisted of 224 athletes participating in contact sports (e.g., lacrosse, basketball, wrestling, football, and soccer) and 153 athletes participating in noncontact sports (e.g., golf, cross country, and baseball). Results showed

that collegiate contact athletes were carriers of methicillin-resistant *Staphylococcus aureus* (MRSA) more than twice as frequently as were noncontact athletes or similar individuals from the general population. In that 2-year study, MRSA colonization was found in 8% to 31% of contact athletes, compared with 0% to 23% of noncontact athletes. In comparison, roughly 5% of the community at large was positive for MRSA colonization. Furthermore, MRSA was acquired more quickly and colonization lasted for greater periods in contact athletes than in noncontact athletes¹¹.

Maceration of the skin due to sweating also occurs during athletic competition, and this change in skin integrity yields a hospitable reproductive environment for bacteria and other organisms. Trauma implicit to sports participation can also damage the cutaneous barrier, allowing microorganisms to penetrate into deeper tissue layers¹². In addition, strenuous training or competition may make athletes temporarily more susceptible to infections¹³. Extended periods of intense exercise temporarily depress certain aspects of the immune system, including natural killer cells, neutrophils, lymphocytes, immunoglobulin levels, and interleukin-2 levels¹⁴⁻¹⁸. During this window of altered immunity, the body demonstrates an increased vulnerability to environmental pathogens, and microbes may take advantage of this altered state and cause infection^{19,20}.

Mechanisms by Which SSTIs Are Spread

As SSTIs are widespread throughout various sports, it is important to understand both the mode of transmission and methodologies for the prevention of spread. Transmission of bacterial organisms typically results from skin-to-skin collision with a person who has an SSTI, as previously suggested. Therefore, SSTIs are commonly found in athletes who participate in contact sports, such as those in which skin-to-skin physical contact is implicit. Athletes who participate in these collision or contact sports

often sustain injuries involving cutaneous barriers (e.g., turf burns, lacerations, and abrasions), which could facilitate the entry of pathogens^{11,21}.

For athletes participating in events without contact or with limited skin-to-skin collision (e.g., marathon running and swimming), other factors may contribute to SSTI transmission, including sharing of bars of soap, shower cloths, water bottles, uniforms, shaving products, whirlpools, and so on²². In addition, locker rooms, workout areas, and fitness-center equipment surfaces may be contaminated with bacteria and fungi that can cause an SSTI^{3,4,23,24}.

S. aureus Infections in the Athletic Population

S. aureus is the most frequently isolated bacterial pathogen in all athletic groups and deserves special mention from the outset. *S. aureus* may lead to a wide array of infections, ranging from cutaneous or superficial skin infections to more severe invasive infections that can be fatal. Recently, *S. aureus* infections have demonstrated resistance to methicillin or similar penicillin-type antibiotics, which has made treatment and cure difficult. These difficult strains are now referred to as MRSA.

Despite the recent rise in MRSA isolates, 20% to 30% of the general population is also asymptotically colonized with methicillin-susceptible *S. aureus* (MSSA)^{25,26}. Sites most commonly colonized by either pathogen include the nostrils (anterior nares)²⁷, upper airway, digestive tract, skin, and genital mucosa (Fig. 1)²⁷. This asymptomatic colonization does not necessarily pose an immediate risk to the carrier; however, it may be a risk factor for transmission to other susceptible individuals as well as for the development of later symptomatic infection²⁶⁻²⁸.

When first recognized, MRSA colonies were typically isolated to hospitals (hospital-acquired [HA]-MRSA), health-care facilities, and patients frequently seeking care in these environments. As a result, MRSA infections rarely occurred in healthy individuals.



Fig. 1

Left: Clinical photograph of the left side of the face and neck of a patient presenting with impetigo caused by an *S. aureus* infection. *Right:* Clinical photograph of the left axilla of a patient presenting with the same infection.

However, beginning in the late 20th century, epidemiologists and physicians noted a substantial increase in MRSA infections among healthy people in the general population, and especially in athletes (Fig. 2)²⁹⁻³³. A recent survey of 364 members the National Athletic Trainers Association revealed that more than half (53%) of all athletic trainers had treated MRSA infections in their athletes, with the majority (92%) being SSTIs³⁰. The dramatic increase in community-acquired MRSA (CA-MRSA) has necessitated a need for orthopaedic surgeons to become more familiar with the identification of the organism and to develop an awareness of treatment algorithms²⁹.

CA-MRSA and HA-MRSA are characterized by their genetic mechanisms of resistance and virulence. Two separate components define the CA-MRSA bacterial strains. The first component, which is similar to that

seen in HA-MRSA, allows for resistance to antibiotics such as methicillin and other beta-lactam agents. The second element, atypical of HA-MRSA, is a cytotoxin that enhances tissue necrosis. The latter is of most concern and is thought to come from a virulent protein factor known as Pantone-Valentine leukocidin (PVL). Several studies have shown this protein to be present in nearly all known CA-MRSA strains but in <5% of HA-MRSA isolates³¹⁻³³. The PVL cytotoxin has the ability to lyse white blood cells and to cause necrosis of mucosa and skin. This property likely allows many CA-MRSA infections to progress to abscess formation and to transmit infection through exposed soft tissue³¹⁻³³.

Despite the preponderance of *S. aureus* and CA-MRSA in athletes, other infections are also common and can present similar challenges. In the following sections, we will review common types of bacterial infections among a

myriad of professional and recreational athletes.

Prevalence of SSTIs Among Players in Various Sports

American Football

Professional

Bacterial infections have been widely reported among professional football players. The media have noted a large number of MRSA infections in National Football League (NFL) players, including members of the Cleveland Browns³⁴⁻³⁶, New York Giants³⁷, Miami Dolphins³⁷⁻³⁹, Tampa Bay Buccaneers³⁷⁻³⁹, Washington Redskins⁴⁰, Indianapolis Colts⁴¹, and New England Patriots⁴². Despite this wide reporting, we found only 2 reports on MRSA SSTIs outside of the lay press^{43,44}.

Throughout the course of 1 season, 8 MRSA infections were found in 5 (9%) of 58 St. Louis Rams players⁴³.

Fig. 2

Clinical photograph demonstrating the typical presentation of MRSA abscesses on the ventral surface of the abdomen of a member of a recreational gymnasium.



Infections were noted at the sites of skin lesions (turf burns) over the elbows, knees, or arms (Fig. 3) of offensive linemen, defensive linemen, and 1 linebacker. All of these infections eventually required operative treatment with incision and drainage as they failed to respond to antibiotic treatment and evolved into abscesses measuring 5 to 7 cm in diameter. While none of the players required hospital admission, 3 players missed between 1 and 12 days (total, 17 working days) of competition secondary to treatment of the infection. These findings are not necessarily unexpected as a recent cohort study revealed that players participating as linemen or linebackers demonstrated a significantly increased relative risk of infection with MRSA (10.6 [95% confidence interval (CI), 1.3 to ∞], $p = 0.02$) when compared with players in skill positions such as quarterbacks or running backs⁴³. Players with cutaneous MRSA infections demonstrated significantly higher body-mass indices (BMIs), and had more frequently used antibiotics during the prior 12 months (relative risk, 7.8; 95% CI, 0.5 to ∞) than those who completed the season without evidence of infection, although the difference was not significant⁴³.

In an attempt to avoid, treat, and prevent MRSA outbreaks during NFL

competition, 108 players and associated personnel from the San Francisco 49ers underwent index nasal swabs at the start of the season and had follow-up cultures of specimens from wounds and nares in cases of suspected MRSA infections throughout the season⁴⁴. Despite this careful surveillance program and aggressive preventative measures to limit the spread of bacteria, 5 (4.6%) of the 108 individuals were found to have new-onset culture-confirmed MRSA skin infections throughout the season. None of these 5 team members had MRSA-positive nasal cultures during index testing, suggesting the difficulty of controlling spread in such high-contact environments.

College

MRSA outbreaks among college football teams also have been well documented in the literature^{11,45-53}. In 2000, a MRSA SSTI outbreak occurred in 10 members of a collegiate football program in Pennsylvania⁴⁵. Seven (70%) of the affected patients required hospitalization and intravenous antibiotics to treat the infection. These MRSA infections were thought to be associated with minor skin trauma resulting from turf abrasions or shaving and the sharing of unlaundered shower towels. Similarly, 2 players from a collegiate team in Los

Angeles were hospitalized for the treatment of MRSA skin infections within the same week during the 2002 season⁴⁵. One of these players did not respond to antibiotic therapy and required extensive surgical debridement, leading to a subsequent skin-grafting procedure for cutaneous coverage. Players from that team reported infrequent treatment or coverage of cutaneous wounds (occurring only 50% of the time), despite frequent and recurrent trauma to the skin. Staff from the team further suggested that the sharing of skin balms and lubricants contributed to the spread of infection.

Between 2000 and 2010, multiple cases of MRSA infections were reported throughout an array of collegiate teams. While many were simple skin infections that were treated with oral antibiotics, there were several reports of small abscesses requiring surgical decompression as well as 1 case of necrotizing fasciitis leading to multiple surgical interventions and prolonged hospitalization^{11,46-53}. The majority of infections occurred in areas of exposed skin, and common sites of infection included the elbow, knee, leg, and forearm. In situations in which infections occurred in areas typically covered by clothing (e.g., thigh or torso), transmission was attributed to at least biweekly

Fig. 3
Clinical photograph demonstrating the typical appearance of a MRSA infection on the left side of the abdomen/flank region.



sharing of a cold tub or whirlpool⁴⁶. The prevalence of infection was higher in linemen and linebackers, which was postulated to be consistent with substantial skin-to-skin contact relating to play at these positions^{11,46-53}. Other risk factors found to be associated with MRSA transmission included abrasions from turf or artificial playing surfaces (turf burns) and shaving of body hair. An overview of these groups is shown in Table I.

Despite the known risk factors, there have been reports of the ability to reduce and control infections in these collegiate settings. Studies have demonstrated that, following the institution of a campaign to promote education, the use of hexachlorophene-containing soap, increased presence of hand-sanitizing agents, disposable towels, cleansing and decontamination of training and weight-room equipment, and bathing or showering prior to the use of athletic training facilities, a substantial reduction in the rate of infection transmission is possible⁴⁷⁻⁴⁹.

High School

There are fewer reports of SSTI outbreaks in high school football players. Over a 5-year span, 3 reports demonstrated that between 10% and 14% of players on evaluated teams were affected by skin infections (Table II)⁵⁴⁻⁵⁶. The majority of the cases were either culture-proven MRSA or suspected MRSA, and linemen carried a 4 times greater likelihood of infection than those playing other positions⁵⁵. Furthermore, in 1 report, the risk of MRSA infection was 8 times higher among athletes who shared towels or washcloths with others than among those who used only their own items. The subset of athletes who were infected with MRSA were found to have an increased BMI when compared with those without infection^{55,56}, and athletes who were found to have proven cases of *S. aureus* infection also admitted to dressing in the same game uniform or practice apparel as many as 11 times without washing between uses.

Rugby

We are aware of only 2 reports on bacterial SSTIs affecting rugby players in the medical literature^{57,58}. In 1 report, 5 (33%) of 15 members of a rugby team in the United Kingdom (all of whom played the forward position) presented to the team physician because of a cutaneous MRSA infection⁵⁷, and all of them had recently played in a match against a visiting team from the South Pacific. In the subsequent 10 days, all of the infections progressed to large abscesses measuring several centimeters in width at various body locations, including the shoulder region, head, neck, and back. In the second report, 37 suspected cases of skin infection were observed in participants from 4 different Belgian rugby clubs, and cultures of specimens from 5 of these wounds were positive for *Streptococcus pyogenes emm* type 81, suggesting that many had been exposed to similar modes of infection⁵⁸.

Soccer

There have been several reports of MRSA outbreaks in soccer teams^{59,60}. A 26-member Slovenian soccer team was affected by an outbreak of a highly virulent and contagious MRSA SSTI⁵⁹. Eleven team members and the team masseuse (46%) were diagnosed with cutaneous MRSA infections. Bilateral staphylococcal furunculosis of the lower extremity was seen in 10 players, nasal colonization was found in 1 player who subsequently developed a MRSA-positive perianal abscess, and eczematous dermatitis of the hands developed in the masseuse. During the 2005-2006 season, members of a Dutch soccer club along with close team associates experienced an outbreak of MRSA infections⁶⁰. Several players had skin infections, and 1 member of the team was hospitalized because of an abscess that was attributed to MRSA. Forty-two members of the soccer club (all of whom utilized the same shower facilities, locker rooms, and training equipment) along with 14 of their roommates were screened for the organism. Of the 56 individuals who were screened, 11 were

found to have a MRSA infection, including 9 (21%) of 42 soccer players and 2 (14%) of 14 roommates. The genesis of this outbreak was later attributed to contact with a member from a visiting team with an exposed boil that tested positive for MRSA following completion of the match.

Wrestling

With extensive and continuous skin-to-skin collision during grappling and maneuvering, a large number of bacterial SSTIs are plausible in wrestlers. However, a majority of reported infections are fungal in nature⁶¹⁻⁷³, and we are aware of only 2 reports of MRSA SSTIs among wrestlers^{45,74}. In 1 report, 7 (22%) of 32 members of a high school wrestling team in Vermont, as well as 6 other individuals who were closely related to the athletes, were found to be infected with MRSA during an outbreak between January 1993 and February 1994⁷⁴. Of those infected, 6 wrestlers sought medical attention and were found to have boils on the arms or legs that were positive for MRSA on culture. Another patient was found to have positive results on culture of specimens from the anterior nares. One infected team member had progressive cellulitis that required admission to the local hospital and was found to have lymphangitis and septicemia associated with the cutaneous infection. Although the cause of this outbreak was indeterminate, members of the team typically practiced as a group on the same mats and wrestling floors without clothing covering the legs or arms for 6 days each week, and contact in these exposed areas was thought to play a contributing role. A second report revealed that 2 wrestlers from the same high school team in Indiana had cutaneous MRSA infections⁴⁵. Those teammates competed in different weight classes, and as such had not wrestled against each other, even in a practice setting. This finding suggested that in sports such as wrestling, in which skin frequently comes in contact with a common playing surface, cleansing of shared spaces and limiting

TABLE 1 Reports of MRSA SSTIs Among College Football Players*

Population	Year(s)	No. of Athletes Infected	Infection Site(s)	Comments
College football players ⁴⁵	2000	10	NR	<ul style="list-style-type: none"> Shaving and turf burns resulting in trauma to the skin and sharing of unwashed bath towels were suspected as potential risk factors for transmission
College football players ⁴⁵	2002 to 2003	2	NR	<ul style="list-style-type: none"> Both players were hospitalized for MRSA skin infections within the same week; 1 received surgical debridement and skin grafts Shared items such as balms and lubricants were identified as potential risk factors for transmission
College football players ⁴⁶	2003	10 (10%) of 100	Elbow, thigh, hip, chin, forearm, wrist, knee, tibial plateau	<ul style="list-style-type: none"> 10 players developed 13 MRSA skin infections (9 abscesses and 4 cellulitis) Hospitalization was required for 2 players who had recurrent MRSA infections. The other 8 players were managed as outpatients; all but 1 required at least 7 days of frequent visits for wound care 8 of the 10 infected players were cornerback defensive backs or wide receivers. The 2 remaining infected players were a linebacker and a tight end Other risk factors deemed to be associated with transmission of MRSA infection included abrasions from artificial grass (turf burns), cosmetic body shaving In 3 of the 4 players whose infection was located at a covered site (e.g., hip or thigh), transmission was attributed to sharing the cold whirlpool at least twice each week
College football players ⁴⁷⁻⁴⁹	2002 to 2004	2 (1.9%) of 107 in 2002; 17 (15.9%) of 107 in 2003; 1 (0.96%) of 104 in 2004	Elbow, shin, ankle, forearm, knee, buttock, and chin	<ul style="list-style-type: none"> The spectrum of disease ranged from a small abscess requiring outpatient surgical drainage to necrotizing fasciitis requiring hospitalization and multiple procedures 8 players required hospitalization
College football players ⁵⁰	2005	13		
College football players ⁵¹	2006	33 (6.7%) of 491	Elbow, knee, leg, and forearm	<ul style="list-style-type: none"> Cutaneous manifestations included abscess (70%), cellulitis (16%), folliculitis, impetigo, and necrotizing fasciitis 90% of the infections were treated with surgical drainage, whereas 27% were treated with intravenous antibiotics No differences were seen in occurrence by player position
College football players ⁵²	2006	25 (22.9%) of 109	Distal arm, distal leg, neck, chest, shoulder, axilla, foot, and groin	<ul style="list-style-type: none"> 13 (52%) of the 25 cases occurred in offensive linemen. Other positions included defensive lineman (4), tight end (2), linebacker (2), defensive back (2), quarterback (1), and wide receiver (1)
College football players ⁵³	2007	8 (7.3%) of 110		<ul style="list-style-type: none"> A particularly virulent strain of MSSA with an unusual resistance profile (resistant to erythromycin and ciprofloxacin) was responsible for this outbreak
College football players ¹¹	2008 to 2010	9 (7.2%) of 125		<ul style="list-style-type: none"> 5 of the 9 infections were identified as MRSA, 1 was identified as MSSA, and 3 were not <i>S. aureus</i> abscesses

*NR = not reported.

TABLE II Reports of MRSA SSTIs Among High School Football Players*

Population	Year(s)	No. of Athletes Infected	Infection Site(s)	Comments
High school football players (Pittsburgh, Pennsylvania) ⁵⁴	2003	13 (14.4%) of 90	Arm, elbow, forearm, knee, leg, face, neck, and abdomen	<ul style="list-style-type: none"> Playing a lineman position carried a fourfold greater likelihood of infection than playing other positions
High school football players (Illinois) ⁵⁵	2004	4 players (team size unknown)	NR	<ul style="list-style-type: none"> 4 cases of MSSA infection were reported in 4 football players, 1 of whom was hospitalized All 4 players reported wearing a game or practice uniform as many as 11 times without laundering the uniform between uses during the season
High school football players (Brooklyn, New York) ⁵⁶	2007	6 (10.2%) of 59	NR	<ul style="list-style-type: none"> 3 cases involved abscesses that required incision and drainage The team had attended a preseason football training camp, where all 59 players on the team lived together in the school gymnasium The risk for MRSA infection was 8 times higher among those who reported sharing towels during the training camp than among those who did not The 6 players with MRSA infections had a mean BMI that was significantly higher than that for those who were not infected

*NR = not reported.

the use of communal hygiene products are of utmost importance.

Other Sports

Skin infections also have been reported in athletes participating in college basketball^{21,75}, volleyball²¹, tennis, and weightlifting^{21,76}. While SSTIs also have been reported in athletes participating in marathon running, swimming, and judo, we are not aware of any cases of bacterial infection in such athletes; instead, all of the reported cases involved only fungal infections⁷⁷⁻⁸⁷. Although the majority of bacterial infections are due to cutaneous beta-hemolytic streptococci or MRSA, pitted keratolysis (a condition characterized by foul-smelling pits on the plantar or dorsal aspect of the foot) also has been seen¹². The latter is typically provoked by gram-positive bacterial species, such as *Corynebacterium* or *Micrococcus*, and is treated with topical antibiotics such as erythromycin or clindamycin. Another effective antimicrobial agent is topical benzoyl

peroxide, which not only kills the infecting organisms but also creates a difficult environment for bacterial growth by making the skin dry. Athletes with such infections should avoid wearing cotton socks as such socks keep the foot warm and moist¹².

Measures to Prevent the Spread of Infection

Given that infections occur in association with a variety of sporting activities, a thorough knowledge of mechanisms to prevent spread is of utmost importance. The Centers for Disease Control and Prevention (CDC)⁴⁵, the Infectious Diseases Society of America (IDSA)⁸⁸, the National Athletic Trainers' Association (NATA)⁸⁹, and the National Collegiate Athletic Association (NCAA)⁹⁰ have all provided guidelines for steps that should be taken to avoid bacterial and fungal infections and outbreaks in locker rooms, private and school gymnasiums, and fitness centers.

Because even limited contact with infected areas can lead to spread, athletes should be excluded from participation if wounds cannot be properly covered by a securely attached bandage or dressing that contains all drainage and remains intact during activity⁹¹. Even in the setting of properly covered wounds, appropriate hygiene measures must be stressed to prevent spread. An athlete also may be excluded at the discretion of the physician if participation poses a health risk to the infected athlete (such as injury to the infected area), even if the infection can be properly covered⁹¹.

Personal Hygiene

Treating abrasions or cuts involving the skin is an important preventative mechanism. All cutaneous lesions should be cleaned with soap and water and covered with a clean, dry bandage before the return to sports participation is considered²⁹. These hygiene practices, along with avoidance of contact with any draining or weeping skin lesions of other

athletes, are the most effective means for preventing the spread of SSTIs⁴⁵. All wounds, including all cuts and scrapes, should be covered adequately before participation; if the wound cannot be completely and securely protected, the athlete should be excluded from participation in practice or games until the lesions are fully healed or are small enough to be appropriately dressed⁴⁵. All athletes with open wounds, abrasions, or lacerations should be excluded from whirlpools or common tubs, and spaces that are jointly used should be sanitized between uses⁴⁵. Some studies have also revealed that, in individuals with *S. aureus* SSTIs, the addition of chlorhexidine body washes^{92,93} or “bleach baths” (i.e., soaking in a bathtub containing a dilute solution of household bleach) to routine hygiene measures may help to decrease the colonization of organisms and prevent recurrence^{92,94,95}.

The most effective behavioral practice in preventing of spread of SSTIs such as MRSA is hand washing²⁹. All athletes, including individuals who exercise at fitness centers, should frequently and thoroughly wash the hands with soap or an alcohol-based hand sanitizer to limit the transmission of bacterial pathogens⁸⁹. The use of plain or antimicrobial soap products is equally effective, but hand washing with liquid soap is preferred over bar soap in order to limit multi-person use⁹⁶. Both hands should be cleansed before and after sporting activities, and the practice should especially be followed when using shared weight-training equipment, when using communal showers or locker rooms, after providing wound care, and when changing bandages⁹⁶. Hand washing is also particularly important after palpation of infected skin or after using an object that has come into direct contact with a draining wound⁸⁸.

Athletes should shower in soapy warm or hot water as soon as possible after the completion of a workout, practice, or game competition^{29,45,89}. Individuals who exercise at health clubs

should also shower immediately after working out on shared fitness equipment⁹⁴. Personal items such as towels, clothing, razors, and bar soap should not be shared^{45,89}.

Environmental Hygiene

All athletes, including individuals who work out at fitness centers, should place a barrier (e.g., an item of clothing or a cloth towel) between bare skin and commonly shared surfaces, including exercise machines, massage tables, weight-training devices, and sauna and steam-room benches⁹⁶. All workout clothing, including team-supplied uniforms and towels used for sports and exercise, should be washed after each use in hot water with bleach or detergent^{29,97}. To avoid moist environments that promote bacterial growth, complete drying of clothes in a dryer is recommended⁹⁶. Locker rooms should not provide communal jars of ointments that athletes apply by placing the hands into an open container⁹⁶. In gymnasiums, health clubs, and fitness centers, bar soap should be replaced with liquid soap to limit sharing⁹⁰. Individual or disposable, single-use products should replace commonly used communal towels during practice or competition^{2,47-49}.

Environmental interventions aimed at controlling the spread of bacterial and fungal infections in athletes focus primarily on thorough cleansing and sterilization of shared or frequently used equipment. Cleansing is the key to decreasing colonization²⁹, and a focus on surfaces that have frequent contact with skin (e.g., benches, door handles, water coolers, showers, tubs, and toilet seats) is of utmost importance⁸⁸. All shared equipment and athletic gear (e.g., helmets, protective gear, and wrestling mats) should be cleaned prior to, and following, each individual use, practice, or competition²⁹.

Disinfectants and detergents designed specifically for athletic equipment are commercially available. All of these agents are simple and effective solutions for routine cleansing of high-

contact surfaces⁸⁸. Care should be exercised, however, as the overuse of prepackaged antibiotic wipes or towel-ettes may actually lead to an increased potential for the development of resistant organisms, so appropriate use as recommended by the manufacturers of these items and label guidelines should be followed⁸⁸. If prepackaged products are not available for disinfection of equipment, a dilute bleach solution made by combining 1 gallon (3.8 L) of water with 1/4 cup (60 mL) of regular household bleach (i.e., a 1:100 dilution equivalent to 500 to 615 ppm of available chlorine) can be used to produce similar results^{29,90,92}. If athletic equipment is damaged to an extent that impervious surfaces are breached and appropriate cleansing is prevented, replacement, repair, or disposal of these items is recommended⁹⁰.

Treatment of SSTIs in Athletes

While the most important method for the avoidance of SSTIs is appropriate prevention, athletes who have contracted an infection should be managed according to the most recent guidelines put forth by the IDSA⁸⁸. For athletes with nonpurulent cellulitis, oral beta-lactam antibiotics providing coverage for beta-hemolytic streptococci, including penicillin and cephalosporins, are recommended as a first-line treatment. Patients who do not respond to beta-lactam agents should be managed empirically for CA-MRSA with oral clindamycin, trimethoprim-sulfamethoxazole (TMP-SMX), a tetracycline such as doxycycline or minocycline, or linezolid^{88,98}. The antibiotic regimen is typically continued for 5 to 10 days but should be individualized on the basis of patient symptoms and response to treatment. If concurrent treatment for both beta-hemolytic streptococci and CA-MRSA is desired, the use of isolated oral clindamycin, TMP-SMX, or linezolid is typically effective. However, a combination of a tetracycline agent and a beta-lactam also can be utilized^{88,98}.

Athletes presenting with purulent cellulitis are considered to be positive for CA-MRSA; in such cases, culture specimens should be obtained and empiric antibiotic treatment should be initiated. In such settings, treatment for beta-hemolytic streptococci typically is not required as such infections are unlikely to cause purulent discharge⁸⁸. For purulent lesions originating from a cutaneous abscess, the recommended initial treatment is incision and drainage. Isolated incision and drainage typically is adequate for the treatment of a simple abscess; however, the currently available literature does not clearly define the role of, or need for, antibiotic therapy in these settings⁸⁸. Nevertheless, there are clearly defined circumstances in which adjunctive treatment of an abscess with antibiotics is appropriate, including cases involving severely purulent or large abscesses; extensive involvement of multiple body locations; accelerated advancement of disease associated with cellulitis, systemic illness, an immunosuppressed state, or associated comorbid conditions; abscesses in very young or very old patients; abscesses located in areas that are difficult to treat with incision and drainage (such as the face, web space of the hand, and genitalia); concomitant septic phlebitis; or failure to completely respond to isolated incision and drainage^{88,98}. To ensure appropriate treatment, culture specimens should be obtained when the patient has an abscess meeting the aforementioned criteria, when the patient has an inadequate response to initial treatment, or when there is concern for an outbreak or cluster infection.

Special considerations should be observed when managing children who have SSTIs. In children and adolescents presenting with superficial cutaneous infections such as impetigo, or in the setting of secondarily infected skin lesions related to eczema, ulcers, or lacerations, topical mupirocin 2% ointment can be used effectively⁸⁸. Tetracycline agents are not recommended, and should be avoided, for

children under 8 years of age as they may cause permanent tooth discoloration or affect growth⁸⁸.

If an athlete develops recurrent SSTIs despite appropriate treatment and preventative measures, referral to an infectious-disease specialist and concomitant skin and nasal decolonization may be considered^{88,98-100}. These strategies can be offered in conjunction with the ongoing recommended treatment. The best initial option for decolonization of the skin and nares is treatment with bleach baths and intranasal mupirocin, respectively; chlorhexidine and intranasal mupirocin also can be utilized as the first option can be cumbersome and has demonstrated poor compliance^{99,100}. However, recent evidence also has suggested that the use of sodium hypochlorite body wash is associated with improved compliance and decolonization success¹⁰¹.

Overview

SSTIs have become increasingly common in the sports world. Efforts to decrease colonization of bacteria and fungi are now essential for preventing the development of SSTIs. Education on cleansing and hygiene are a vital part of this process. The team physician and the team athletic trainers play the most important role in the education of the athlete and all members of the athletic team. The concept of the athletic team also includes anyone in contact with the athlete or athletic training facility, such as all members of the coaching and training staff, as well as the families in contact with the participants. The impact of SSTIs on individuals and the athletic team may be severe and has the potential for notable consequences, including loss of playing time, hospitalization, and even surgery. Providers should stay up to date on the information regarding the recognition, prevention, and treatment of SSTIs, and online guidelines published by the CDC can be very useful for providers in their continued efforts to better understand and to prevent the development of SSTIs^{91,96,102}.

Justin J. Mitchell, MD¹,
J. Mark Jackson, MD²,
Azam Anwar, MD³,
Steven B. Singleton, MD¹

¹The Steadman Clinic and Steadman Philippon Research Institute, Vail, Colorado

²Division of Dermatology, University of Louisville, Louisville, Kentucky

³CLn Skin Care, Top MD Skin Care, Dallas, Texas

E-mail address for S.B. Singleton: stevensingleton@hotmail.com

References

- Collins CJ, O'Connell B. Infectious disease outbreaks in competitive sports, 2005-2010. *J Athl Train*. 2012 Sep-Oct;47(5):516-8.
- Grindstaff TL, Saliba SA, Mistry DJ, Macknight JM. Community-associated methicillin-resistant *Staphylococcus aureus*. *N Am J Sports Phys Ther*. 2007 Aug;2(3):138-46.
- Mukherjee N, Dowd SE, Wise A, Kedia S, Vohra V, Banerjee P. Diversity of bacterial communities of fitness center surfaces in a U.S. metropolitan area. *Int J Environ Res Public Health*. 2014 Dec; 11(12):12544-61. Epub 2014 Dec 3.
- Markley JD, Edmond MB, Major Y, Bearman G, Stevens MP. Are gym surfaces reservoirs for *Staphylococcus aureus*? A point prevalence survey. *Am J Infect Control*. 2012 Dec;40(10): 1008-9. Epub 2012 May 22.
- Grice EA, Segre JA. The skin microbiome. *Nat Rev Microbiol*. 2011 Apr;9(4):244-53.
- Kong HH, Segre JA. Skin microbiome: looking back to move forward. *J Invest Dermatol*. 2012 Mar;132(3 Pt 2):933-9. Epub 2011 Dec 22.
- Schommer NN, Gallo RL. Structure and function of the human skin microbiome. *Trends Microbiol*. 2013 Dec;21(12):660-8. Epub 2013 Nov 12.
- Findley K, Oh J, Yang J, Conlan S, Deming C, Meyer JA, Schoenfeld D, Nomicos E, Park M, Kong HH, Segre JA; NIH Intramural Sequencing Center Comparative Sequencing Program. Topographic diversity of fungal and bacterial communities in human skin. *Nature*. 2013 Jun 20;498(7454):367-70. Epub 2013 May 22.
- Chen YE, Tsao H. The skin microbiome: current perspectives and future challenges. *J Am Acad Dermatol*. 2013 Jul;69(1):143-55. Epub 2013 Mar 13.
- Meadow JF, Bateman AC, Herkert KM, O'Connor TK, Green JL. Significant changes in the skin microbiome mediated by the sport of roller derby. *PeerJ*. 2013;1:e53. Epub 2013 Mar 12.
- Jiménez-Truque N, Saye EJ, Soper N, Saville BR, Thomsen I, Edwards KM, Creech CB. Longitudinal assessment of colonization with *Staphylococcus aureus* in healthy collegiate athletes. *J Pediatric Infect Dis Soc*. 2016 Jun;5(2): 105-13. Epub 2014 Nov 5.
- Adams BB. Skin infections in athletes. *Dermatol Nurs*. 2008 Feb;20(1):39-44.
- Brenner IK, Shek PN, Shephard RJ. Infection in athletes. *Sports Med*. 1994 Feb;17(2):86-107.

14. Grosset-Janin A, Nicolas X, Saroux A. Sport and infectious risk: a systematic review of the literature over 20 years. *Med Mal Infect.* 2012 Nov;42(11):533-44. Epub 2012 Oct 29.
15. Eda N, Shimizu K, Suzuki S, Lee E, Akama T. Effects of high-intensity endurance exercise on epidermal barriers against microbial invasion. *J Sports Sci Med.* 2013;12(1):44-51. Epub 2013 Mar 1.
16. Nieman DC. Exercise immunology: practical applications. *Int J Sports Med.* 1997 Mar; 18(Suppl 1):S91-100.
17. Shephard RJ, Shek PN. Potential impact of physical activity and sport on the immune system—a brief review. *Br J Sports Med.* 1994 Dec;28(4):247-55.
18. Shephard RJ, Rhind S, Shek PN. Exercise and the immune system. Natural killer cells, interleukins and related responses. *Sports Med.* 1994 Nov;18(5):340-69.
19. Kakani MW, Peake J, Brenu EW, Simmonds M, Gray B, Hooper SL, Marshall-Gradsnik SM. The open window of susceptibility to infection after acute exercise in healthy young male elite athletes. *Exerc Immunol Rev.* 2010;16:119-37.
20. Pedersen BK, Bruunsgaard H. How physical exercise influences the establishment of infections. *Sports Med.* 1995 Jun;19(6):393-400.
21. Cohen PR. Cutaneous community-acquired methicillin-resistant *Staphylococcus aureus* infection in participants of athletic activities. *South Med J.* 2005 Jun;98(6):596-602.
22. Oller AR, Province L, Curless B. *Staphylococcus aureus* recovery from environmental and human locations in 2 collegiate athletic teams. *J Athl Train.* 2010 May-Jun;45(3):222-9.
23. Ryan KA, Ifantides C, Bucciarelli C, Saliba H, Tuli S, Black E, Thompson LA. Are gymnasium equipment surfaces a source of staphylococcal infections in the community? *Am J Infect Control.* 2011 Mar;39(2):148-50.
24. Wood M, Gibbons SM, Lax S, Eshoo-Anton TW, Owens SM, Kennedy S, Gilbert JA, Hampton-Marcell JT. Athletic equipment microbiota are shaped by interactions with human skin. *Microbiome.* 2015;3:25. Epub 2015 Jun 19.
25. Gorwitz RJ, Kruszon-Moran D, McAllister SK, McQuillan G, McDougal LK, Fosheim GE, Jensen BJ, Killgore G, Tenover FC, Kuehnert MJ. Changes in the prevalence of nasal colonization with *Staphylococcus aureus* in the United States, 2001-2004. *J Infect Dis.* 2008 May 1;197(9):1226-34.
26. Graham PL 3rd, Lin SX, Larson EL. A U.S. population-based survey of *Staphylococcus aureus* colonization. *Ann Intern Med.* 2006 Mar 7;144(5):318-25.
27. Wertheim HF, Melles DC, Vos MC, van Leeuwen W, van Belkum A, Verbrugh HA, Nouwen JL. The role of nasal carriage in *Staphylococcus aureus* infections. *Lancet Infect Dis.* 2005 Dec;5(12):751-62.
28. Fritz SA, Epplin EK, Garbutt J, Storch GA. Skin infection in children colonized with community-associated methicillin-resistant *Staphylococcus aureus*. *J Infect.* 2009 Dec;59(6):394-401. Epub 2009 Sep 9.
29. Cohen PR. The skin in the gym: a comprehensive review of the cutaneous manifestations of community-acquired methicillin-resistant *Staphylococcus aureus* infection in athletes. *Clin Dermatol.* 2008 Jan-Feb;26(1):16-26.
30. Brinsley-Rainisch KJ, Goding AM, Sinkowitz-Cochran RL, Pearson M, Hageman J. MRSA infections in athletics: perceptions and practices of certified athletic trainers [abstract]. In: The 17th Annual Scientific Sessions of the Society for Healthcare Epidemiology of America; 2007 Apr 14-17. Baltimore, MD: Society for Healthcare Epidemiology of America. Abstract no. 70.
31. Jevons MP. Celbenin-resistant *Staphylococci*. *Br Bed J.* 1961 Jan 14;1(5219):124-5.
32. Moran GJ, Amii RN, Abrahamian FM, Talan DA. Methicillin-resistant *Staphylococcus aureus* in community-acquired skin infections. *Emerg Infect Dis.* 2005 Jun;11(6):928-30.
33. Marcotte AL, Trzeciak MA. Community-acquired methicillin-resistant *Staphylococcus aureus*: an emerging pathogen in orthopaedics. *J Am Acad Orthop Surg.* 2008 Feb;16(2):98-106.
34. Stroud R, Auman G. Ex-Browns tell of staph infection anguish. 2012 Aug 23. <http://www.tampabay.com/sports/football/bucs/ex-browns-tell-of-staph-infection-anguish/2138098>. Accessed 2016 Feb 23.
35. Cabot MK. Former Cleveland Browns receiver Joe Jurevicius sues team, doctors, Cleveland Clinic over staph infection. 2009 Jun 26. http://www.cleveland.com/browns/index.ssf/2009/06/joshua_gunter_the_plain_dealer.html. Accessed 2016 Feb 23.
36. Barr M. LeCharles Bentley lawsuit: I almost died because of Browns negligence. 2011 May 25. http://www.huffingtonpost.com/2010/07/22/lecharles-bentley-lawsuit_n_656099.html. Accessed 2016 Feb 23.
37. Lannetti R. 'Superbug' MRSA worries doctors, athletes. 2007 Mar 6. <http://abcnews.go.com/Health/Primetime/story?id=410908>. Accessed 2016 Feb 23.
38. Patra K. Johnthan Banks third Bucs player with MRSA infection. 2013 Oct 12. <http://www.nfl.com/news/story/0ap2000000260608/article/johnthan-banks-third-bucs-player-with-mrsa-infection>. Accessed 2016 Feb 23.
39. Volin B. MRSA infections growing concern in NFL locker rooms. 2013 Oct 27. <https://www.bostonglobe.com/sports/2013/10/26/buccaneers-dealing-with-mrsa-outbreak/2JxLajA7nNdZXec8kKq4kl/story.html>. Accessed 2016 Feb 23.
40. Noble B. Brandon Noble. 2006 Mar. http://www.idsociety.org/Brandon_Noble/. Accessed 2016 Feb 23.
41. Associated Press. Colts say Manning's initial surgery prompted by staph infection. 2012 Jul 26. <http://www.nfl.com/news/story/09000d5d80be7619/article/colts-say-mannings-initial-surgery-prompted-by-staph-infection>. Accessed 2016 Feb 23.
42. Gever J. Infection complicates knee surgery recovery for Patriots' QB Tom Brady. 2008 Oct 24. <http://www.medpagetoday.com/PublicHealthPolicy/PublicHealth/11458>. Accessed 2016 Feb 23.
43. Kazakova SV, Hageman JC, Matava M, Srinivasan A, Phelan L, Garfinkel B, Boo T, McAllister S, Anderson J, Jensen B, Dodson D, Lonsway D, McDougal LK, Arduino M, Fraser VJ, Killgore G, Tenover FC, Cody S, Jernigan DB. A clone of methicillin-resistant *Staphylococcus aureus* among professional football players. *N Engl J Med.* 2005 Feb 3;352(5):468-75.
44. Garza D, Sungar G, Johnston T, Rolston B, Ferguson JD, Matheson GO. Ineffectiveness of surveillance to control community-acquired methicillin-resistant *Staphylococcus aureus* in a professional football team. *Clin J Sport Med.* 2009 Nov;19(6):498-501.
45. Centers for Disease Control and Prevention (CDC). Methicillin-resistant *Staphylococcus aureus* infections among competitive sports participants—Colorado, Indiana, Pennsylvania, and Los Angeles County, 2000-2003. *MMWR Morb Mortal Wkly Rep.* 2003 Aug 22;52(33):793-5.
46. Begier EM, Frenette K, Barrett NL, Mshar P, Petit S, Boxrud DJ, Watkins-Colwell K, Wheeler S, Cebelinski EA, Glennen A, Nguyen D, Hadler JL; Connecticut Bioterrorism Field Epidemiology Response Team. A high-morbidity outbreak of methicillin-resistant *Staphylococcus aureus* among players on a college football team, facilitated by cosmetic body shaving and turf burns. *Clin Infect Dis.* 2004 Nov 15;39(10):1446-53. Epub 2004 Oct 26.
47. Romano R, Lu D, Holtom P. Outbreak of community-acquired methicillin-resistant *Staphylococcus aureus* skin infections among a collegiate football team. *J Athl Train.* 2006 Apr-Jun;41(2):141-5.
48. Nguyen DM, Mascola L, Brancoft E. Recurring methicillin-resistant *Staphylococcus aureus* infections in a football team. *Emerg Infect Dis.* 2005 Apr;11(4):526-32.
49. Centers for Disease Control and Prevention (CDC). Public health dispatch: outbreaks of community-associated methicillin-resistant *Staphylococcus aureus* skin infections—Los Angeles County, California, 2002–2003. 2003 Feb 7. <http://www.cdc.gov/mmwr/preview/mmwrhtml/mm5205a4.htm>. Accessed 2016 May 25.
50. Archibald LK, Shapiro J, Pass A, Rand K, Southwick F. Methicillin-resistant *Staphylococcus aureus* infection in a college football team: risk factors outside the locker room and playing field. *Infect Control Hosp Epidemiol.* 2008 May;29(5):450-3.
51. Bowers AL, Huffman GR, Sennett BJ. Methicillin-resistant *Staphylococcus aureus* infections in collegiate football players. *Med Sci Sports Exerc.* 2008 Aug;40(8):1362-7.
52. Hall AJ, Bixler D, Haddy LE. Multiclonal outbreak of methicillin-resistant *Staphylococcus aureus* infections on a collegiate football team. *Epidemiol Infect.* 2009 Jan;137(1):85-93. Epub 2008 Apr 18.
53. Fontanilla JM, Kirkland KB, Talbot EA, Powell KE, Schwartzman JD, Goering RV, Parsonnet J. Outbreak of skin infections in college football team members due to an unusual strain of community-acquired methicillin-susceptible *Staphylococcus aureus*. *J Clin Microbiol.* 2010 Feb;48(2):609-11. Epub 2009 Dec 9.
54. Rihn JA, Posfay-Barbe K, Harner CD, Macurak A, Farley A, Greenawalt K, Michaels MG. Community-acquired methicillin-resistant *Staphylococcus aureus* outbreak in a local high school football team unsuccessful interventions. *Pediatr Infect Dis J.* 2005 Sep;24(9):841-3.
55. Borchardt SM, Yoder JS, Dworkin MS. Is the recent emergence of community-associated methicillin-resistant *Staphylococcus aureus* among participants in competitive sports limited to participants? *Clin Infect Dis.* 2005 Mar 15;40(6):906-7.
56. Centers for Disease Control and Prevention (CDC). Methicillin-resistant *Staphylococcus aureus* among players on a high school football team—New York City, 2007. *MMWR Morb Mortal Wkly Rep.* 2009 Jan 30;58(3):52-5.
57. Stacey AR, Endersby KE, Chan PC, Marples RR. An outbreak of methicillin resistant *Staphylococcus aureus* infection in a rugby football team. *Br J Sports Med.* 1998 Jun;32(2):153-4.

- 58.** Quoilin S, Lambion N, Mak R, Denis O, Lammens C, Struelens M, Maes S, Goossens H. Soft tissue infections in Belgian rugby players due to *Streptococcus pyogenes* emm type 81. *Euro Surveill.* 2006;11(12):E061221.2. Epub 2006 Dec 21.
- 59.** Müller-Premru M, Strommenger B, Alikadic N, Witte W, Friedrich AW, Seme K, Kucina NS, Smrke D, Spik V, Gubina M. New strains of community-acquired methicillin-resistant *Staphylococcus aureus* with Panton-Valentine leukocidin causing an outbreak of severe soft tissue infection in a football team. *Eur J Clin Microbiol Infect Dis.* 2005 Dec;24(12):848-50.
- 60.** Huijsdens XW, van Lier AM, van Kregten E, Verhoef L, van Santen-Verheulveel MG, Spalburg E, Wannet WJ. Methicillin-resistant *Staphylococcus aureus* in Dutch soccer team. *Emerg Infect Dis.* 2006 Oct;12(10):1584-6.
- 61.** Ahmadijad Z, Alijani N, Mansori S, Ziaee V. Common sports-related infections: a review on clinical pictures, management and time to return to sports. *Asian J Sports Med.* 2014 Mar; 5(1):1-9. Epub 2014 Jan 26.
- 62.** Kohl TD, Lisney M. Tinea gladiatorum: wrestling's emerging foe. *Sports Med.* 2000 Jun;29(6):439-47.
- 63.** Kohl TD, Giesen DP, Moyer J Jr, Lisney M. Tinea gladiatorum: Pennsylvania's experience. *Clin J Sport Med.* 2002 May;12(3):165-71.
- 64.** Bassiri-Jahromi S, Khaksar AA. Outbreak of tinea gladiatorum in wrestlers in Tehran (Iran). *Indian J Dermatol.* 2008;53(3):132-6.
- 65.** Beller M, Gessner BD. An outbreak of tinea corporis gladiatorum on a high school wrestling team. *J Am Acad Dermatol.* 1994 Aug;31(2 Pt 1): 197-201.
- 66.** Hradil E, Hersle K, Nordin P, Faergemann J. An epidemic of tinea corporis caused by *Trichophyton tonsurans* among wrestlers in Sweden. *Acta Derm Venereol.* 1995 Jul;75(4):305-6.
- 67.** el Fari M, Gräser Y, Presber W, Tietz HJ. An epidemic of tinea corporis caused by *Trichophyton tonsurans* among children (wrestlers) in Germany. *Mycoses.* 2000;43(5):191-6.
- 68.** Adams BB. Tinea corporis gladiatorum: a cross-sectional study. *J Am Acad Dermatol.* 2000 Dec;43(6):1039-41.
- 69.** Hedayati MT, Afshar P, Shokohi T, Aghili R. A study on tinea gladiatorum in young wrestlers and dermatophyte contamination of wrestling mats from Sari, Iran. *Br J Sports Med.* 2007 May; 41(5):332-4. Epub 2006 Nov 30.
- 70.** Ergin S, Ergin C, Erdoğan BS, Kaleli I, Evliyaoglu D. An experience from an outbreak of tinea capitis gladiatorum due to *Trichophyton tonsurans*. *Clin Exp Dermatol.* 2006 Mar;31(2):212-4.
- 71.** Aghamirian MR, Ghiasian SA. A clinico-epidemiological study on tinea gladiatorum in Iranian wrestlers and mat contamination by dermatophytes. *Mycoses.* 2011 May;54(3): 248-53.
- 72.** Ilkit M, Ali Saracli M, Kurdak H, Turac-Bicer A, Yuksel T, Karakas M, Schuenemann E, Abdel-Rahman SM. Clonal outbreak of *Trichophyton tonsurans* tinea capitis gladiatorum among wrestlers in Adana, Turkey. *Med Mycol.* 2010 May;48(3):480-5.
- 73.** Ahmadijad Z, Razaghi A, Noori A, Hashemi SJ, Asghari R, Ziaee V. Prevalence of fungal skin infections in Iranian wrestlers. *Asian J Sports Med.* 2013 Mar;4(1):29-33. Epub 2012 Sep 15.
- 74.** Lindenmayer JM, Schoenfeld S, O'Grady R, Carney JK. Methicillin-resistant *Staphylococcus aureus* in a high school wrestling team and the surrounding community. *Arch Intern Med.* 1998 Apr 27;158(8):895-9.
- 75.** Stevens MP, Bearman G, Rosato A, Edmond M. Community-acquired methicillin resistant *Staphylococcus aureus* in a women's collegiate basketball team. *South Med J.* 2008 Oct;101(10): 1067-8.
- 76.** Cohen PR. Community-acquired methicillin-resistant *Staphylococcus aureus*: skin infection presenting as an axillary abscess with cellulitis in a college athlete. *Skinmed.* 2005 Mar-Apr;4(2):115-8.
- 77.** Purim KS, Bordignon GP, Queiroz-Telles Fd. Fungal infection of the feet in soccer players and non-athlete individuals. *Rev Iberoam Micol.* 2005 Mar;22(1):34-8.
- 78.** Pickup TL, Adams BB. Prevalence of tinea pedis in professional and college soccer players versus non-athletes. *Clin J Sport Med.* 2007 Jan; 17(1):52-4.
- 79.** Auger P, Marquis G, Joly J, Attye A. Epidemiology of tinea pedis in marathon runners: prevalence of occult athlete's foot. *Mycoses.* 1993 Jan-Feb;36(1-2):35-41.
- 80.** Lacroix C, Baspeyras M, de La Salmonière P, Benderdouche M, Couprie B, Accoceberry I, Weill FX, Derouin F, Feuillade de Chauvin M. Tinea pedis in European marathon runners. *J Eur Acad Dermatol Venereol.* 2002 Mar;16(2):139-42.
- 81.** Attye A, Auger P, Joly J. Incidence of occult athlete's foot in swimmers. *Eur J Epidemiol.* 1990 Sep;6(3):244-7.
- 82.** Shiraki Y, Soda N, Hirose N, Hiruma M. Screening examination and management of dermatophytosis by *Trichophyton tonsurans* in the judo club of a university. *Nippon Ishinkin Gakkai Zasshi.* 2004;45(1):7-12.
- 83.** Hirose N, Shiraki Y, Hiruma M, Ogawa H. [An investigation of *Trichophyton tonsurans* infection in university students participating in sports clubs]. *Nippon Ishinkin Gakkai Zasshi.* 2005;46(2):119-23. Japanese.
- 84.** Estève E, Rousseau D, Defo D, Poisson DM. [Outbreak of cutaneous dermatophytosis in the Judo French Programme in Orleans: September 2004-June 2005]. *Ann Dermatol Venereol.* 2006 Jun-Jul;133(6-7):525-9. French.
- 85.** Suganami M, Hirose N, Shiraki Y, Hiruma M, Ikeda S. [Trichophyton tonsurans infection among judo practitioners who attended the National Junior High School Judo Tournament in Japan (2005): incidence and therapeutic response]. *Nippon Ishinkin Gakkai Zasshi.* 2006; 47(4):319-24. Japanese.
- 86.** Shiraki Y, Hiruma M, Hirose N, Sugita T, Ikeda S. A nationwide survey of *Trichophyton tonsurans* infection among combat sport club members in Japan using a questionnaire form and the hairbrush method. *J Am Acad Dermatol.* 2006 Apr;54(4):622-6. Epub 2006 Feb 7.
- 87.** Hirose N, Tamura M, Suganami M, Ogawa YS, Hiruma M. The results of *Trichophyton tonsurans* screening examinations and infection management in University Judo Federation of Tokyo athletes over a 4-year period. *Med Mycol J.* 2012;53(4):267-71.
- 88.** Liu C, Bayer A, Cosgrove SE, Daum RS, Fridkin SK, Gorwitz RJ, Kaplan SL, Karchmer AW, Levine DP, Murray BE, J Rybak M, Talan DA, Chambers HF; Infectious Diseases Society of America. Clinical practice guidelines by the Infectious Diseases Society of America for the treatment of methicillin-resistant *Staphylococcus aureus* infections in adults and children. *Clin Infect Dis.* 2011 Feb 1;52(3): e18-55. Epub 2011 Jan 4.
- 89.** Zinder SM, Basler RS, Foley J, Scarlata C, Vasily DB. National Athletic Trainers' Association position statement: skin diseases. *J Athl Train.* 2010 Jul-Aug;45(4):411-28.
- 90.** National Collegiate Athletic Association. 2014-15 NCAA sports medicine handbook. 2014 Aug. <http://www.ncaapublications.com/productdownloads/MD15.pdf>. Accessed 2016 May 25.
- 91.** Centers for Disease Control and Prevention. Methicillin-resistant *Staphylococcus aureus* (MRSA): coaches, athletic directors, and team healthcare providers. <http://www.cdc.gov/mrsa/community/team-hc-providers/index.html>. March 1, 2016. Accessed 2016 Mar 30.
- 92.** Fritz SA, Camins BC, Eisenstein KA, Fritz JM, Epplin EK, Burnham CA, Dukes J, Storch GA. Effectiveness of measures to eradicate *Staphylococcus aureus* carriage in patients with community-associated skin and soft-tissue infections: a randomized trial. *Infect Control Hosp Epidemiol.* 2011 Sep;32(9):872-80.
- 93.** Viray MA, Morley JC, Coopersmith CM, Kollef MH, Fraser VJ, Warren DK. Daily bathing with chlorhexidine-based soap and the prevention of *Staphylococcus aureus* transmission and infection. *Infect Control Hosp Epidemiol.* 2014 Mar;35(3):243-50. Epub 2014 Jan 24.
- 94.** Kaplan SL, Forbes A, Hammerman WA, Lamberth L, Hulten KG, Minard CG, Mason EO. Randomized trial of "bleach baths" plus routine hygienic measures vs. routine hygienic measures alone for prevention of recurrent infections. *Clin Infect Dis.* 2014 Mar;58(5): 679-82. Epub 2013 Nov 21.
- 95.** Hegggers JP, Sazy JA, Stenberg BD, Strock LL, McCauley RL, Herndon DN, Robson MC. Bactericidal and wound-healing properties of sodium hypochlorite solutions: the 1991 Lindberg Award. *J Burn Care Rehabil.* 1991 Sep-Oct;12(5):420-4.
- 96.** Centers for Disease Control and Prevention. Methicillin-resistant *Staphylococcus aureus* (MRSA): prevention information and advice. <http://www.cdc.gov/mrsa/community/team-hc-providers/advice-for-athletes.html>. Accessed 2015 Nov 18.
- 97.** Kowalski TJ, Barbari EF, Osmon DR. Epidemiology, treatment, and prevention of community-acquired methicillin-resistant *Staphylococcus aureus* infections. *Mayo Clin Proc.* 2005 Sep;80(9):1201-7; quiz 1208.
- 98.** Howe WB. Preventing infectious disease in sports. *Phys Sportsmed.* 2003 Feb;31(2):23-9.
- 99.** Huang JT, Abrams M, Tlougan B, Rademaker A, Paller AS. Treatment of *Staphylococcus aureus* colonization in atopic dermatitis decreases disease severity. *Pediatrics.* 2009 May;123(5):e808-14.
- 100.** Leung TH, Zhang LF, Wang J, Ning S, Knox SJ, Kim SK. Topical hypochlorite ameliorates NF- κ B-mediated skin diseases in mice. *J Clin Invest.* 2013 Dec;123(12):5361-70. Epub 2013 Nov 15.
- 101.** Ryan C, Shaw RE, Cockerell CJ, Hand S, Ghali FE. Novel sodium hypochlorite cleanser shows clinical response and excellent acceptability in the treatment of atopic dermatitis. *Pediatr Dermatol.* 2013 May-Jun;30(3):308-15.
- 102.** Centers for Disease Control and Prevention. Methicillin-resistant *Staphylococcus aureus* (MRSA). 2016 Mar 1. <http://www.cdc.gov/mrsa/index.html>. Accessed 2016 Mar 30.