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Infectious Diseases Associated With Organized Sports and Outbreak Control

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Participation in organized sports has a variety of health benefits but also has the potential to expose the athlete to a variety of infectious diseases, some of which may produce outbreaks. Major risk factors for infection include skin-to-skin contact with athletes who have active skin infections, environmental exposures and physical trauma, and sharing of equipment and contact with contaminated fomites. Close contact that is intrinsic to team sports and psychosocial factors associated with adolescence are additional risks. Minimizing risk requires leadership by the organized sports community (including the athlete's primary care provider) and depends on outlining key hygiene behaviors, recognition, diagnosis, and treatment of common sports-related infections, and the implementation of preventive interventions.

abstract

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To cite: Davies HD, Jackson MA, Rice SG, AAP COMMITTEE ON INFECTIOUS DISEASES, AAP COUNCIL ON SPORTS MEDICINE AND FITNESS. Infectious Diseases Associated With Organized Sports and Outbreak Control. *Pediatrics.* 2017;140(4):e20172477

INTRODUCTION

The definition of organized sports includes traditional team sports commonly acknowledged as well as other types of sports (Table 1). Participation in organized sports provides the benefits of (1) physical activity, by engaging in vigorous exercise, achieving fitness, and learning athletic skills; (2) socialization, by experiencing camaraderie and learning teamwork and sportsmanship; and (3) competition, by challenging oneself to perform against others, by striving to continually improve oneself toward achieving one's full athletic potential, and by learning to win and lose with grace and dignity.[1] Organized sports participation, however, can result in the acquisition of a variety of infectious diseases and conditions. Physical contact among athletes, sharing of equipment (such as worn personal protective equipment or braces plus towels, drinking vessels, showers, and locker rooms), and contact with athletic surfaces (mats, artificial turf, dirt, grass, and gym or weight room equipment) can all be responsible for transmission of infection.[2–9] In addition, certain organized sports carry specific additional risks; for example, wrestlers

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practicing in close quarters are especially vulnerable to skin infections.[10,11]

Athletes should be taught proper personal hygiene (eg, hand-washing, showering, and proper laundering of uniforms and practice clothing on a daily or regular basis).[12–15] Avoidance of sharing of drinking vessels (water bottles, ladles, or cups), mouth guards, towels, braces, batting helmets, personal protective equipment, bars of soap, bath sponges, razors or electric hair shavers, and callus trimmers is also important in reducing infectious risk.[2–9] In addition, athletic programs should ensure regular (daily, weekly, and monthly) cleaning of facilities and equipment (eg, weight room, railings, mats, blocking dummies, locker rooms, and showers).[16–19] Those who manage sports programs and facilities should develop a plan for proper cleaning and maintenance of a sanitary sporting environment by using guidelines such as those published by the American College of Sports Medicine.[20]

Special attention should be paid to proper management of blood and other body fluids.[21] Just as hospitals in the United States have concentrated on preventing hospital-associated infections in recent years, the same level of focus on infection prevention and control needs to be present within the organized sports community, including among athletes, parents, coaches, athletic directors, equipment managers, certified athletic trainers, administrators, janitorial staff, team physicians, facility managers, and league officials.

Although the primary care pediatrician may appear to be peripheral in this athletic milieu of organized sports, leadership from physicians has always been welcome and expected regarding issues of public health and safety. Furthermore, because pediatricians need to provide medical clearance to athletes to participate in organized sports, the preparticipation physical examination is an opportunity to verify that the athlete does not have a skin condition or infection that could be transmitted to others. This visit between the physician and the student athlete allows the primary care pediatrician to deliver anticipatory guidance. Ensuring that immunizations are current per recommendations of the Centers for Disease Control and Prevention, the Advisory Committee on Immunization Practices, and the American Academy of Pediatrics is important, and pediatric providers should identify and document cases in which vaccines are refused or incomplete because of medical exemptions (eg, serious allergy to a vaccine component). Coaches and trainers are primarily responsible for reviewing and stressing to the athlete the key hygiene behaviors needed to minimize the risk of obtaining or spreading infection in organized sports. However, primary care pediatricians can help reinforce such educational messages.

ORGANISMS ASSOCIATED WITH INFECTIONS IN ATHLETES

An athlete can acquire many different infections by participating in organized sports. The pathogens include many that are prominent in outbreaks typically seen in crowded communities or closed community settings or that are facilitated by certain exposures specific to the sport.

Infectious pathogens include those spread by skin contact (eg, *Staphylococcus aureus*, group A streptococcal skin infections, *Bacillus cereus*, herpes simplex virus [HSV], *Tinea capitis*, *Tinea corporis*, *Tinea pedis*, *Tinea cruris*, *Pediculosis capitis*, *Pediculosis corporis*, and *Pediculosis pubis*), by contaminated food or water (eg, Shiga-toxin producing *Escherichia coli*, *Shigella* species, *Giardia* species, *Cryptosporidium* species, and norovirus, which is further propagated by the person-to-person route), by respiratory droplet (eg, influenza, pertussis, *Neisseria meningitidis*, group A streptococcal pharyngitis, mumps), by airborne particles (eg, varicella, measles), or by certain vectors (eg, ticks) (Table 2). In the case of Epstein-Barr virus infection, close contact is required for transmission, and endemic disease within adolescent group settings has been reported (Table 2). Although biologically plausible, there have been no validated reports of infections from transmission of bloodborne pathogens, including hepatitis B, hepatitis C, or HIV during athletic competitions. Nonetheless, the American Academy of Pediatrics has previously issued specific detailed guidelines for management of infections spread by blood and body fluids, including guidance for athletes who are infected with HIV, hepatitis B virus, or hepatitis C virus, and these will not be reiterated in this statement.[21]

Transmission of a specific infectious agent may be affected by a variety of psychosocial (sexually transmitted infection), physical (trauma, closed community contact), and environmental (soil, food, water, vector) factors, especially in an immunologically naïve population. This policy will be focused on diagnosis, treatment, and prevention of the most common infections that may be encountered in the athlete participating in organized sports, with an additional focus on factors that are potentially modifiable. It should be noted that some of the organisms discussed can be transmitted in multiple fashions. Transmission of pathogens spread by contaminated food or water, by

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respiratory droplets, and by vectors are similar to what can be expected under nonsporting conditions[51] and are beyond the scope of this report. Although several of these pathogens are summarized in Table 2, detailed descriptions in this clinical report are provided only for the organisms transmitted primarily by contact, manifesting primarily on the skin, and those that are airborne.

INFECTIONS PRIMARILY SPREAD BY CONTACT TRANSMISSION

Most sports-related skin infections are spread by contact and have been associated with 10% to 15% of time-loss injuries among athletes at the collegiate level.[52] For this reason, routine screening of athletes participating in contact sports during practices and before competitions is important. The Sports Medicine Advisory Committee of the National Federation of State High School Associations (NFHS),[53] the National Collegiate Athletic Association (NCAA),[54] and the National Athletic Trainers Association (NATA)[55] have published guidelines for screening and when to return to athletic participation for several conditions (summarized in Table 3). It is noteworthy that many of the recommendations by these organizations are more stringent than ordinary infection control practices for similar conditions in which the likelihood of the type of close bodily contact is not as significant.

S Aureus

Community-acquired methicillin-resistant *S aureus* (MRSA) is a cause of outbreaks of skin infections among high school and collegiate athletes participating in contact sports, particularly among football players and wrestlers, and is associated with significant morbidity. It manifests primarily as cellulitis or skin abscesses but may lead to invasive disease such as bacteremia, septic arthritis, osteomyelitis, myositis, fasciitis, and pneumonia in up to 10% of cases.[56,57] Other noninvasive forms of disease including impetigo, staphylococcal ecthyma, pustulosis, and folliculitis may also pose a risk for transmission of the organism from those so affected. Players who have preexisting skin diseases such as atopic dermatitis may have chronic colonization with *S aureus* and may be predisposed to recurrent secondary infection. Between 10% and 23% of football players or wrestlers have developed signs and symptoms during outbreaks.[5,58–60] Risk factors for infection include skin breaks associated with turf burns or trauma,[58] skin-to-skin contact, sharing of equipment or clothing (towels), and higher BMI.[5,19,59] Although 4% to 23% of athletes have been found to have colonization with MRSA, high colonization alone does not appear sufficient to trigger an outbreak.[61] Wrestling mats, artificial turfs, and football training equipment have been documented with MRSA colonization.[19,62,63]

Incidence estimates of MRSA-related skin and soft tissue infections in Nebraska student athletes have ranged from 11.3 to 20.9 per 10 000 football players and 28.1 to 60.8 per 10 000 wrestlers from 2008 to 2012.[64] Among 190 high school football players in northeast Ohio who were managed prospectively with nasal swab cultures, 23% displayed methicillin-susceptible *S aureus* colonization (none carried MRSA). Of the participating athletes, 10 (5.3%) developed skin infections, including 7 with impetigo and 1 with folliculitis barbae during the course of the 2008 season. None of the cultured specimens tested positive for MRSA.[65]

Group A *Streptococcus*

Group A β-hemolytic *Streptococcus* (GABHS) has been associated with outbreaks of skin infections after indoor association football tournaments,[66] rugby,[67,68] and other organized sports. The organism can cause localized skin infections such as pyoderma, cellulitis, or impetigo or invasive infections such as thrombophlebitis, myositis, and sepsis. GABHS has also been associated with outbreaks of pharyngitis among university students (median age 19.5 years) participating in judo.[69] Within a 15-day period, 12 of 23 club members in Tokyo, Japan, presented with sore throat and high fever. Diagnosis was made by use of either a rapid streptococcal antigen test or positive throat culture for GABHS when the clinical presentation was suggestive of pharyngitis. Occasionally, outbreaks of invasive GABHS disease have been reported in members of high school football teams,[70] likely attributable to sharing of equipment and water bottles.

Management of *S Aureus* and GABHS Outbreaks

Management of MRSA and GABHS outbreaks has been accomplished through meticulous focus on hygiene education, good hygiene practices, prompt identification of infected people, limiting exposure to infected people and contaminated surfaces and objects, decontamination of the environment, and proper treatment and close follow-up of infected people.[4,5,7,59,60,69,71] Of particular importance for management of MRSA outbreaks is screening of players for carriage along with use of topical mupirocin for those found to have colonization, use of chlorhexidine washes, and enhancement of personal hygiene practices.[7,19,72] Bleach baths (Clorox: regular 6.0% hypochlorite, 5 mL, added to 1 gallon of water) used twice weekly reduced the recurrence rates among children with community-associated *S aureus* infections by 20% compared with control children managed with routine hygienic measures, but this

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was not a statistically significant reduction.[73] Screening for *S aureus* and GABHS requires knowledge of what body sites would have colonization. Nasal, skin, vaginal, and rectal carriage are the primary reservoirs for *S aureus*. In the context of sports-related infections, the primary culture sites should be the nares and any open skin lesions.

GABHS screening should be performed through vigorous swabbing of a pair of swabs on both tonsils and the posterior pharynx for rapid antigen detection and culture as well as culture of any skin lesions, especially those that are oozing or macerated.[23] Measurement of sequential streptococcal antibody titers may also be used to diagnose a recent infection, but this is not recommended for routine use.[74]

Skin abscesses are best managed by incision and drainage, with culture of the wound for identification of causative agent and antimicrobial susceptibility pattern along with empirical antibiotic coverage pending culture results. Antibiotic choices should be guided by knowledge of the local patterns of susceptibility of *S aureus*, especially local rates of MRSA. Methicillin-susceptible *S aureus* typically is treated with oral penicillinase-resistant β-lactam drugs, such as a first- or second-generation cephalosporin. For patients who are allergic to penicillin, or if MRSA is a significant consideration, the alternatives are trimethoprim-sulfamethoxazole, doxycycline, or clindamycin for susceptible isolates.[22] Doxycycline can be used safely in children ages 2 years and older when given for durations less than 2 weeks. Trimethoprim-sulfamethoxazole should not be used as a single agent in the initial treatment of cellulitis because of the possibility it is caused by group A *Streptococcus* and the possibility of intrinsic resistance of this organism.[75–77] Topical mupirocin may be used for localized and nonbullous impetigo. During outbreaks, attempts at eradication of *S aureus* infections to limit spread may be accomplished by use of topical nasal mupirocin therapy (twice daily for 5–7 days) among people with colonization. However, low-level (minimum inhibitory concentration, 8–256 μg/mL) and high-level (minimum inhibitory concentration, >512 μg/mL) resistance to mupirocin have been identified in *S aureus*. High-level resistance has been associated with subsequent failure of decolonization.

GABHS typically is susceptible to penicillin, and this is the usual first-line therapy.[74] An oral macrolide or azalide (eg, erythromycin, clarithromycin, or azithromycin) is acceptable for patients who are allergic to penicillin. Duration of treatment is 10 days, with the exception of azithromycin, which is indicated for 5 days. Local mupirocin[78–80] or retapamulin[81–83] ointment may be useful for limiting person-to-person spread of nonbullous impetigo and for eradicating localized GABHS disease. Guidelines from the NCAA, NFHS, and NATA for when the infected athlete can return to competition for both GABHS and *S aureus* are summarized in Table 3.

Prevention of *S Aureus* and GABHS Outbreaks

Prevention of infections caused by MRSA and GABHS is achieved primarily through good hygiene practices, not sharing equipment and water bottles, limiting exposure to infected people and contaminated surfaces and objects, decontamination of the environment, and prompt identification, proper treatment, and close follow-up of infected people.[4,5,7,59,60,69,71] Athletes with GABHS pharyngitis or skin infections should not return to competitive sports for at least 24 hours after beginning appropriate oral antimicrobial therapy.[23] Table 3 summarizes recommendations of the NCAA, NFHS, and NATA for return to competition for patients with *S aureus* infections, including MRSA.

Herpes Gladiatorum and Herpes Rugbiorum

HSV (primarily type 1) has been identified as a cause of outbreak of skin infections among wrestlers (herpes gladiatorum [HG]) and rugby players (herpes rugbiorum [HR]) on numerous occasions, affecting up to 2.6% of high school and 7.6% of college wrestlers in the United States.[84–86] During outbreaks, up to 34% of all high school wrestlers have been documented to be infected.[87] Risk factors for development of HG-related cutaneous HSV lesions include direct skin-to-skin exposure to opponents with cutaneous lesions.[84] There is a range of 4 to 11 days, with an average of 6.80 ± 1.70 days from onset of exposure to development of skin lesions. Most outbreaks (96%) occur on the ventral surface of the body, with up to three-quarters of the cases occurring on areas in direct contact when wrestlers are engaged in the lock-up position (head, face, and neck). Other body areas frequently involved are the extremities (42%) and trunk (28%).[16] HSV conjunctivitis (5%) and blepharitis have also been reported.[87] Between 25% and 40% of patients with HG and HR will develop constitutional symptoms including fever, chills, sore throat, and headaches.[85,87] There are data associating acquired antibody to HSV type 1 infection with protection from acquiring HG, but the association is very weak.[84]

Herpes Outbreak and High-Altitude Skiing

High-altitude skiing also has been associated with relapses of orofacial herpes, presumably because of solar UV radiation exposure, with a median onset of 3.5 days after

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exposure (strong evidence).[88] Although sunscreen with a sun protection factor of 15 was shown to prevent experimental UV light-induced reactivation of herpes labialis compared with placebo,[89] it has not been shown to influence the reactivation rate among high-altitude skiers.[88]

Management of HG and HR Outbreaks

There is strong evidence that prompt identification and 3 to 8 days of isolation of infected wrestlers during primary outbreaks of HG and HR with suspension of competition can help contain outbreaks in more than 90% of cases.[87,90,91] Diagnosis involves a combination of clinical recognition and may be coupled with cell culture, histologic examination, or rapid diagnostic tests such as direct fluorescent antibody staining, enzyme immunoassay, or polymerase chain reaction (PCR) of vesicular lesion scrapings in complex, lingering, or unclear cases.[26] Valacyclovir, 500 mg, every day or twice a day for 7 days, when given within 24 hours of symptoms onset, has been shown to shorten the duration of time until HSV PCR clearance from lesions of adolescent and adult wrestlers with recurrent HG by 21% (from ~8.1 days with placebo to 6.4 days with valacyclovir).[92] Wrestlers receiving valacyclovir should be advised about the importance of good hydration to minimize the risk of nephrotoxicity. Competitors often do not recognize or may deny possible infection. As a result, efforts to reduce transmission should include (1) examination of wrestlers and rugby players for vesicular or ulcerative lesions on exposed areas of their bodies and around their mouths or eyes before practice or competition by a person familiar with the appearance of mucocutaneous infections (including HSV, herpes zoster, and impetigo), (2) excluding athletes with these lesions from competition until all lesions are fully crusted or production of a physician's written statement indicating that their condition is noninfectious, and (3) cleaning of wrestling mats with a freshly prepared solution of household bleach (1 quarter cup of bleach in 1 gallon of water) applied for a minimum contact time of 15 seconds at least daily and preferably between matches.[26] NCAA, NFHS, and NATA guidelines for when the infected athlete can return to competition are summarized in Table 3.

Prevention of HG and HR Outbreaks

Athletes with a history of recurrent HG, HR, or herpes labialis should be considered for suppressive antiviral therapy. There is strong evidence that nucleoside analogues (valacyclovir) can suppress recurrent outbreaks of herpes. In a study involving 42 male wrestlers aged 13 to 31 years in Minnesota combining double-blind randomization followed by an open enrollment onto treatment, participants with recurrent HG were treated during the first half of the season with either 500 mg of valacyclovir or placebo and in the second half with 1000 mg of valacyclovir. The 500 and 1000 mg doses of valacyclovir suppressed recurrence of outbreaks among 100% (7 out of 7 and 12 out of 12) of participants whose last recurrence was more than 2 years before. However, the doses were slightly less successful among those with recurrences within 2 years (11 out of 14 and 23 out of 25, respectively), with better results with the 1000 mg dosing.[93]

Similarly, in a Minnesota study of 332 male wrestlers 13 to 20 years of age who participated at a 28-day wrestling camp, once-a-day prophylactic valacyclovir (1000 mg) starting 1 week before camp and continuing throughout camp reduced the incidence of clinical HG outbreaks by 87%. Among 55 of these wrestlers who were HSV type 1 immunoglobulin G seronegative at the beginning of the camp and had postcamp serologic testing performed, none developed detectable immunoglobulin M against HSV type 1 or HSV type 2.[94] However, there is need for a detailed risk–benefit analysis of using valacyclovir for prophylaxis in wrestlers who are seronegative for herpes.

Molluscum Contagiosum

Molluscum contagiosum is a common, benign viral skin infection presenting as skin-colored papules that develop a central umbilication as they age. Molluscum contagiosum affects 5% to 11% of children 0 to 16 years of age[95] and most commonly affects the trunk, face, and extremities. Molluscum contagiosum is mostly asymptomatic but may present with pain, itching, redness, or occasionally bacterial superinfection. Outbreaks of molluscum contagiosum have most often been described in association with exposure to swimming in public pools and underlying eczema.[3,95–97] Other factors associated with infection include young age (highest incidence in children younger than 14 years), living in close proximity, skin-to-skin contact, sharing of fomites, and residence in tropical climates.[3,98]

Management of Molluscum Outbreaks

Resolution of uncomplicated molluscum contagiosum typically occurs spontaneously in 6 to 12 months, although complete resolution of lesions can take up to 4 years. Although no regimen has proven highly successful, 10% potassium hydroxide and cryotherapy with liquid nitrogen have been used to treat lesions that occur in locations that are cosmetically bothersome to patients or for patients with underlying skin conditions such as eczema. Both forms of treatment appear to have similar efficacy in

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children, but cryotherapy may be associated with postinflammatory hyperpigmentation or, uncommonly, scarring.[99,100] Imiquimod was not shown to be of benefit compared with placebo in randomized controlled trials.[101,102] Open-label and observational studies[103,104] indicate that cantharidin can be an effective treatment of molluscum contagiosum; however, in 1 small randomized controlled trial of 29 patients, the improvement seen with cantharidin, although greater than with placebo, was not found to be statistically significant.[100] Guidelines from the NCAA, NFHS, and NATA for when the infected athlete can return to competition for molluscum are summarized in Table 3.

Prevention of Molluscum Contagiosum Outbreaks

Given the known associations of molluscum contagiosum, the best method of prevention would involve avoiding of skin-to-skin contact with people known to have lesions (covering lesions), not sharing towels and other fomites, and limiting exposure to swimming pools that have recently been associated with known outbreaks.

Tinea Infections

T corporis and *T capitis* infections have been reported more frequently among high school wrestlers and judo practitioners (*T corporis* gladiatorum and *T capitis* gladiatorum) than among other athletes.[8,105–112] Studies of the prevalence of *T corporis* gladiatorum have involved use of potassium hydroxide examination to aid diagnosis. In 1 such study, 24% of 29 wrestlers had lesions of *T corporis*, versus 0 in a control group of track team members ($P = .005$).[106] In another study, *T corporis* was detected in 10 of 19 boys (53%) 15 to 17 years of age belonging to a judo club in Kyoto, Japan.[112] The most common cause of *T capitis* gladiatorum is *Trichophyton tonsurans*, accounting for more than 80% of cases, but it may also be caused by *Trichophyton rubrum* and *Trichophyton mentagrophytes*.[113,114] *T tonsurans* (primarily), *T mentagrophytes*, and *Microsporum canis* have also been isolated in high rates during outbreaks of *T capitis* (ringworm) among high school wrestlers in a wrestling boarding school in Turkey.[109,115,116] These organisms have been isolated frequently from individual skin lesions and from wrestling mats.[108,109]

Management of *T Corporis* and *T Capitis* Outbreaks

Both skin-based and oral medications are available for the treatment of *T corporis*, but *T capitis* is managed exclusively with oral medications, often with simultaneous application of topical treatment to the scalp (because of the need for hair follicle penetration). Skin-based preparations for *T corporis* include but are not limited to azole creams (clotrimazole, econazole, ketoconazole, oxiconazole), allylamine creams and gels (1% gel of terbinafine and butenafine), and hydroxypyridone (ciclopirox) preparations. Although these all are reasonably effective, some preparations in some studies demonstrate superior cure rates. For example, terbinafine emulsion gel has a mycological cure rate superior to that of ketoconazole cream (94% versus 69%, respectively) with similar adverse events rates.[117]

Oral agents also have proven efficacious in the treatment of most cases of *T corporis*. Different doses and durations of itraconazole have been used in studies. Itraconazole, 100 mg, given orally once a day, was superior to griseofulvin, 500 mg, orally, once a day, when given for 15 days (87% mycological cure rate versus 57%, respectively, at the end of 2 weeks after completion of therapy) to adolescents and adults.[118] In contrast, 200 mg of itraconazole was found to be superior to 250 mg of terbinafine when given for 7 days respectively in terms of clinical response rates when administered to adolescents and adults.[119]

For *T capitis*, the traditional treatment in children had been oral griseofulvin, 10 mg/kg per day, microsize formulation, given once daily for 6 to 8 weeks, although doses of griseofulvin as high as 20 to 25 mg/kg per day have been used.[120] Terbinafine, 125 or 250 mg (adjusted for age), given for 2 to 8 weeks, has recently been shown to have efficacy that is at least as good, with fewer adverse effects and fewer recurrences.[116,121–123] Cochrane reviews have concluded that 4 weeks of terbinafine was equivalent to 8 weeks of griseofulvin.[124,125] Similarly, oral fluconazole given for 4 weeks had similar efficacy to oral griseofulvin given for 6 weeks,[126,127] although at least 1 study revealed the efficacy to be low for both regimens.[128] Itraconazole given for 2 weeks is also similar in efficacy to 6 weeks of griseofulvin, whereas terbinafine and itraconazole appear to have similar efficacy for treatment periods of 2 to 3 weeks.[124] However, terbinafine, itraconazole, and fluconazole are significantly more expensive than griseofulvin. Furthermore, griseofulvin appears to be superior for infections attributable to certain species of *Microsporum*, which may require 4 weeks' duration or more of therapy with terbinafine and itraconazole.[122,129,130] Terbinafine appears superior for *T tonsurans*.[125]

Prevention of *T Corporis* and *T Capitis* Outbreaks

Fluconazole, 100 mg per day for 3 days, given prophylactically before initiation of competitive interscholastic high school wrestling and given again 6 weeks into the season, has been reported to significantly reduce the incidence

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of *T corporis* from 67.4% to 3.5%.[30] However, the risk-benefit analysis of giving fluconazole prophylactically in this manner has not been determined, and its use should be in consultation with an infectious diseases expert.

T Pedis

T pedis (athlete's foot) presents as a fine scaly or vesiculopustular eruption that is often itchy. The lesions may involve all areas of the foot but commonly include the fissures and scaling between toes. Increased rates of *T pedis* have been well documented and common among swimmers and runners (especially marathon runners), with documented infections in up to 22%. The predominant causes are *T rubrum* and *T mentagrophytes*.[131] Spread via direct contact with the organism, *T pedis* is prevalent in warm, humid environments and affects men more than women.[132] Obesity and diabetes are additional risk factors for *T pedis*.[133]

Management of *T Pedis* Outbreaks

Numerous treatments (creams and oral medications) have been evaluated for treating *T pedis*. In randomized controlled trials in adults, ciclopirox olamine (a broad-spectrum hydroxypyridone antifungal with proven efficacy against *T rubrum*, *T mentagrophytes*, and *Epidermophyton floccosum*) cream or gel (0.77%) applied twice daily to the affected areas for 4 weeks has been shown to be effective in eradicating *T pedis* and superior to 1% clotrimazole cream or ciclopirox vehicle in achieving both clinical and mycological cure (~60% for cyclopirox olamine cream versus 6% for its vehicle only at end of treatment, and 85% versus 16% two weeks after treatment).[134,135] There are no published studies of use of ciclopirox to treat *T pedis* in children, but a dosage of topical application twice a day is recommended for use in children older than 10 years.[28] Similarly, naftifine ointment applied twice daily for 4 weeks and sulconazole nitrate 1% cream applied daily for 4 to 6 weeks have been associated with significantly higher mycological clearance rates (57%–66%) compared with their vehicles alone (13%–34% cleared) and were associated with fewer relapses in treatment of *T rubrum*–related *T pedis*.[136,137] However, sulconazole is not approved for use in children in the United States. More recently, topical azoles such as fenticonazole powder have been used in adults with up to 100% cure rates.[138] Terbinafine 1% cream applied daily for 1 week also has been used effectively to treat *T pedis*, with >93% mycological cure rate at 4 weeks, and is approved for children 12 years and older.[139] Luliconazole was recently reported as a 1% cream trial for *T pedis* in adults, but less than 50% of the participants were cured on this regimen.[140] It is only approved by the US Food and Drug Administration for use in adults.

Terbinafine offers the advantage of once-daily dosing and can be given for briefer periods than skin-based treatments. Oral terbinafine, 250 mg, given once daily for 1 week, has similar efficacy (based on mycological cure rates at week 4) to 4 weeks of clotrimazole 1% cream applied twice a day but with faster clinical resolution.[141] Oral terbinafine, 250 mg, also is similar in mycological efficacy to itraconazole, 100 mg, when given over a 2-week duration but may have a slightly lower rate of relapse. Terbinafine is well tolerated in children, with the most concerning potential adverse events being occasional isolated neutropenia and rare liver failure, typically in people with preexisting liver disease.

Prevention of *T Pedis* Outbreaks

The use of foot powder after bathing has been associated with a decline in the rates of *T pedis* in a random sampling of users of a swimming bath in Scotland from 8.5% to 2.1% over a 3.5-year period,[142] mostly attributable to the decline in rates of *T mentagrophytes* from 5.3% to 0.5%. Experts believe that careful and thorough drying between the toes after showers, daily changes of socks, and periodic cleaning of athletic footwear can be helpful.

T Cruris

T cruris (jock itch or crotch rot) is a common pruritic fungal infection of the groin and adjacent skin.[133] Heat, humidity, and hyperhidrosis are predisposing factors, as is wearing of tight-fitting or wet clothing. Similar to *T pedis*, obesity and diabetes are additional risk factors for *T cruris*.[133]

The predominant cause is *T rubrum* followed by *E floccosum* and *T mentagrophytes*.[143,144] It may be spread by contaminated fomites (contaminated towels, hotel bed sheets) or autoinoculation from hands or feet infected with *Tinea* from another body site (*Tinea unguium*, *T pedis*, or *Tinea manuum*).[133]

Management of *T Cruris* Infection

Similar to *T pedis*, terbinafine 1% cream applied daily for 1 week has been used effectively to treat *T cruris* with a mycological cure rate of approximately 94% and is approved for children 12 years and older.[117] Butenafine (a benzylamine derivative of clotrimazole) applied twice daily for 2 weeks and clotrimazole applied twice weekly for 4 weeks are also over-the-counter alternatives, but butenafine is only approved in adults.[145–148] Oral itraconazole (100 mg daily for 2 weeks or 200 mg daily for 1 week) has been shown to be effective in adults for treating *T cruris* and superior to oral griseofulvin (500 mg daily for 2 weeks).[118,149,150] Several other azole topical formulations (oxiconazole,[151] luliconazole,[151] sertaconazole,[144] eberconazole[152]) have been shown to

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be effective in adults, but none have been studied in any detail in children.

Prevention of *T Cruris* Infection

Because of risk of spread from *T pedis*, covering active foot lesions with socks before wearing undershorts may reduce the likelihood of direct contamination. Furthermore, complete drying of the crural folds after bathing, and use of separate (clean) towels for drying the groin and other parts of the body may help reduce contamination.

Verruca Vulgaris

Verruca vulgaris (common skin warts) are benign epithelial proliferations of the skin and are caused by human papillomaviruses. They are typically painless, multiple in number, and occur on any epithelial surface, although most commonly on the hands, feet, and around and under the nails.[33] They may be distinguished from calluses and corns by the presence of black dots (clotted blood vessels that have grown into the wart) when they are pared down as well as the associated loss of overlying dermatoglyphs. Although reports indicate that warts may occur in outbreaks among athletes, there are no published data on the prevalence. Reported risk factors appear to include sharing of equipment and exposure of unshod feet in common shower areas.[34] In a study of 146 adolescents who used locker rooms, 27% of those who used communal showers on a regular basis were found to have plantar warts versus only 1.25% of those who only used the locker rooms.[34] However, because the children using communal showers were also members of a swim club, it is unclear whether the communal shower or the swimming was the major contributing factor.

Management of Verruca Vulgaris Infections

Most cases of common warts will eventually spontaneously regress, with 30% regressing within 6 months and approximately 60% within 2 years. Treatment modalities are usually geared toward chemical or physical destruction of the infected epithelium and include techniques such as freezing with liquid nitrogen, application of salicylic acid-based products or tretinoin (retinoic acid) cream, surgical (paring) or laser removal, and use of topical immunomodulating agents.[34] The more destructive methods may lead to pain, which may inhibit athletic activity. More recently, cantharidin combined with podophyllotoxin-salicylic acid has been used in adults and reported to be effective but associated with pain and blistering.[153,154]

Prevention of Verruca Vulgaris Infections

Precise mechanisms of preventing common warts are unknown. However, like most infections that are transmitted by contact, avoidance of contact with people known to have common warts, not sharing equipment and towels, and wearing rubber soled flip-flops or sandals in communal showers may minimize risk.

Scabies and Lice

Scabies (caused by *Sarcoptes scabiei*) and lice (*P capitis*), although not commonly reported in sports, can be disqualifying if identified in children participating in organized, especially contact, sports. They are transmitted primarily through person-to-person contact. The scabies parasite can survive on clothing for up to 4 days without skin contact.[35] Lice do not survive away from the scalp more than 1 to 2 days without a blood meal, and although uncommon, can be transmitted by hair brushes, combs, hats, and hair ornaments.[37] The transmission, risk factors, recognition, diagnosis, treatment, and prevention are summarized in Table 2. Successful treatment of scabies can be achieved topically with permethrin 5% cream or oral ivermectin (not for pregnant women), but resistance has been reported to both.[155,156] Pregnant women and infants may be treated with topical crotamiton or precipitated sulfur ointment. Head lice treatment requires attention not only to the live lice but also to eggs that may subsequently hatch. Treatment of head lice can be started with over-the-counter 1% permethrin lotion or with pyrethrin combined with piperonyl butoxide, both of which have good safety profiles.[36] Resistance to these over-the-counter agents is commonplace in many parts of the United States, and as such, alternative Food and Drug Administration–approved treatments may be necessary. For lice resistant to over-the-counter medications, treatment options include spinosad suspension, benzyl alcohol lotion, malathion, or ivermectin lotion. Spinosad and ivermectin lotion are ovicidal, and a single treatment may be adequate, but no treatment is 100% ovicidal.[155,156] Suffocant treatments, such as benzyl alcohol lotion or malathion lotion, require retreatment approximately 1 week later to kill any new lice that hatched from nits. For lice resistant to all topical agents, oral ivermectin in a single dose of 200 or 400 μg/kg may be used in infants weighing over 15 kg, with a second dose given after 9 to 10 days.[36]

INFECTIONS PRIMARILY SPREAD BY AIRBORNE OR DROPLET ROUTE

Varicella-Zoster Virus

Although varicella has been reported as a cause of airborne sports-related infections,[157] such reports are rare in the era of immunization with the live attenuated vaccine against the virus. The varicella-zoster virus (VZV) manifests primarily as a generalized, pruritic, vesicular rash consisting of 250 to 500

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lesions in different stages (crops) of development and crusting.[39] There is usually an associated low-grade fever, and there may be other systemic symptoms. Disease in vaccinated children is often milder and atypical in nature compared with the wild virus infection and requires a high index of suspicion. Diagnosis is usually made on the basis of a typical clinical picture coupled with history of exposure, but vesicular fluid or scab scraping can be used for confirmation by using PCR, direct fluorescent antibody assay, or VZV-specific culture. A significant increase in serum varicella immunoglobulin G antibody between acute and convalescent serum samples can also assist in diagnosis.

Management of VZV Infections

Isolation should be instituted for those suspected to have chickenpox until the diagnosis is either ruled out or all the lesions are crusted over or in vaccinated children when there are no new lesions within a 24-hour period.[39] Oral acyclovir or valacyclovir given within 24 hours of rash onset results in only a modest decrease in symptoms and is not routinely recommended for most healthy children. It should be considered in otherwise healthy children who are at increased risk of moderate to severe varicella, including people older than 12 years, people with chronic cutaneous or pulmonary disorders, those receiving long-term salicylate therapy, and those on short, intermittent, or aerosolized courses of corticosteroids.[39]

Prevention of VZV Infections

The most effective proven means of preventing VZV is via primary immunization with the live attenuated vaccine.[39] Children exposed to VZV should have their immunity evaluated, either through vaccination records or through serologic testing.

Measles

Measles is characterized by a prodrome of cough, coryza, and conjunctivitis with fever followed by maculopapular or morbilliform rash that begins on the face and spreads downward to the trunk and out to the extremities. Koplik spots, which are considered pathognomonic, also appear during the prodrome.[40] Patients are contagious 4 days before the rash to 4 days after the rash appears. Measles outbreaks have been reported during many different types of sporting events ranging from gymnastics to skiing and fencing,[158–163] highlighting the importance of adequate vaccination of all athletes. Up to 5% of people who have received a single dose of vaccine at 12 months or older have vaccine failure. Among previously immunized people, primary vaccine failure (inadequate response to vaccine) is a more common cause of failure than waning immunity. As a result, the current recommendations call for a 2-dose vaccine schedule for children and high-risk adults.[40]

Mumps

Mumps is a systemic illness that presents with swelling of 1 or more of the salivary glands, typically the parotid glands. Up to one-third of mumps cases do not cause salivary gland swelling, presenting instead as a respiratory tract infection. Orchitis is a common complication after puberty, but it rarely leads to sterility. Approximately 10% of patients have an associated viral meningitis, and numerous other complications occur rarely, including permanent hearing loss, myocarditis, endocardial fibroelastosis, arthritis, thrombocytopenia, thyroiditis, mastitis, glomerulonephritis, pancreatitis, and oophoritis. The virus has been isolated from saliva from 7 days before through 8 days after onset of salivary gland swelling. Before introduction of mumps vaccine, outbreaks of mumps were common in the United States, primarily in crowded settings including schools, prisons, orphanages, and military facilities. Although the incidence of the disease has declined significantly with the introduction of the 2-dose schedule of the measles-mumps-rubella (MMR) vaccine, outbreaks still periodically occur, primarily because of incomplete vaccination.

Management of Measles and Mumps Outbreaks

Immunization is the cornerstone of managing measles and mumps outbreaks. All suspected cases of measles or mumps should be reported immediately, and every effort should be made for laboratory confirmation of the infection either through serologic testing or detection of virus from clinical specimens (throat washings, nasopharyngeal secretions, urine, and blood for measles and buccal swabs, throat washings, saliva, or cerebrospinal fluid, if relevant, for mumps). In an outbreak setting, the MMR vaccine should be given to all people (>12 months) who lack evidence of immunity. For mumps, a second MMR dose should be offered to all students (including those in postsecondary school) who have received only 1 dose of MMR vaccine. A second MMR dose should also be considered for both conditions in children 1 to 4 years of age if they have only received 1 dose and there is an ongoing outbreak affecting preschool-aged children with community-wide transmission.[40]

Prevention of Measles and Mumps Outbreaks

The most important method to prevent measles and mumps outbreaks is routine immunization of all children with a live attenuated vaccine, such as MMR or MMR-varicella vaccine, at age 12 through 15 months, with a second dose at 4 to 6 years of age, and people who are not documented to have been

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vaccinated during these periods.[40] People who have altered immunity (except HIV infection, unless they have severe immunosuppression) should not receive MMR vaccine. Furthermore, MMR-varicella vaccine should not be given to patients with HIV (even if these people have little to no immune compromise) because of a lack of safety data at the present time.

The epidemiology and outbreak control considerations for other conditions primarily transmitted via droplet and contaminated food and water are summarized in Table 2.

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ABBREVIATIONS

GABHS: group A β-hemolytic streptococcus
HG: herpes gladiatorum
HR: herpes rugbiorum
HSV: herpes simplex virus
MMR: measles-mumps-rubella
MRSA: methicillin-resistant *Staphylococcus aureus*
NATA: National Athletic Trainers Association
NCAA: National Collegiate Athletic Association
NFHS: National Federation of State High School Associations
PCR: polymerase chain reaction
VZV: varicella-zoster virus

DOI: https://doi.org/10.1542/peds.2017-2477

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PEDIATRICS (ISSN Numbers: Print, 0031-4005; Online, 1098-4275).

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FINANCIAL DISCLOSURE: The authors have indicated they have no financial relationships relevant to this article to disclose.

FUNDING: No external funding.

POTENTIAL CONFLICT OF INTEREST: The authors have indicated they have no potential conflicts of interest to disclose.

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Pediatrics originally published online September 25, 2017;

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Pediatrics originally published online September 25, 2017;

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