Community-Associated Methicillin Resistant *Staphylococcus areus* Infections

in Two Collegiate Crew Teams

by

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Dedication

This thesis is dedicated to my late mother, Mrs. Molly Ann Wood (Duffy) who died from metastatic breast cancer during this project. Without her unconditional love and support I would not have been able to be where I am today.

The thesis of William Garin Wood is approved:

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History of *Staphylococcus aureus* and antibiotic resistance.

Staphylococcus aureus is a Gram positive coccus, which appears as grape-like clusters when viewed through a microscope and has large, round, golden-yellow colonies, usually with β-hemolysis, when grown on blood agar plates. *S. aureus* is catalase positive and thus able to convert hydrogen peroxide (H_2O_2) to water and oxygen, which makes the catalase test useful to distinguish staphylococci from enterococci and streptococci. It was discovered in Aberdeen, Scotland in 1880 by the surgeon Sir Alexander Ogston in pus from surgical abscesses.

Penicillin binding proteins (PBPs) are necessary for synthesis of the bacterial cell wall. These PBPs are capable of being blocked by the antibiotic penicillin, which effectively prevents the formation of the cell wall. Since the cell wall is not synthesized, the cell will lyse. The molecular structure of penicillin contains a beta-lactam ring. The principle difference in the drugs within this family is distinguished by the R-groups that are covalently bonded to the beta-lactam ring.

About sixteen years after the discovery of penicillin by Sir Alexander Fleming in 1928, a strain of *S. aureus* was discovered to be resistant to penicillin [1]. These *S. aureus* produced penicillinase, a specific type of beta-lactamase that hydrolyzes penicillin. Methicillin was the first developed penicillinase-resistant antibiotic in the beta-lactam family of antibiotic drugs, and was introduced in 1959 by a British pharmaceutical company, Beecham, which is now known as GlaxoSmithKline. The United Kingdom was able to isolate the first strain of methicillin-resistant *Staphylococcus aureus*(MRSA) in 1961 and the United States saw its first methicillin-resistant *S. aureus* case in 1968 [2].

The mechanism for resistance to methicillin is attributed to an alteration of the PBP2 to that of PBP2a. PBP2a is derived from the expression of the *mecA* gene, which is a 2,130-bp segment of DNA that is usually carried on a larger piece of DNA called a staphylococcal cassette chromosome *SCCmec* [3]. PBP2a has a reduced affinity for all antibiotics containing the beta-lactam ring, thus allowing the bacterium to synthesize its cell wall normally. MRSA is resistant to all beta-lactam antibiotics, including all penicillins and cephalosporins. There are five distinct clones of MRSA that have been identified, which are determined from the sequencing of the SCCmec gene. By convention, *S. aureus* strains that are susceptible to methicillin and other beta-lactam antibiotics are termed methicillin susceptible *S. aureus* (MSSA).

Staphylococcal infections, including MRSA, occur most frequently among persons in hospitals and other healthcare facilities (such as nursing homes and dialysis centers) to which the name, hospital-associated MRSA (HA-MRSA) infections is used (www.cdc.gov). Today, staphylococcal infections remain one of the most important nosocomial and community associated pathogens. In a large retrospective study by the National Nosocomial Infections Surveillance System, MRSA rates were measured in US Hospitals from 1975-1991 (**Figure 1**). This study found that the percentage of MRSA in the hospital beds among all sizes of hospitals rose from 2.4% in 1975 to 29% in 1991 [4]. The National Nosocomial Infections Surveillance System followed up this study with another study in ICU patients from 1995-2004, also demonstrating an alarming trend of increased rates of nosocomial MRSA infections. This report revealed that MRSA accounted for a mean of 53 percent of *S. aureus* isolates recovered from intensive care

unit (ICU) patients, 46 percent from non-ICU patients, and 31 percent of *S. aureus* recovered from outpatients (**Figure 2**)[5].

Figure 1: National Nosocomial Infections Surveillance System measurements of nosocomial MRSA by hospital bed size, 1975-1991 [4].

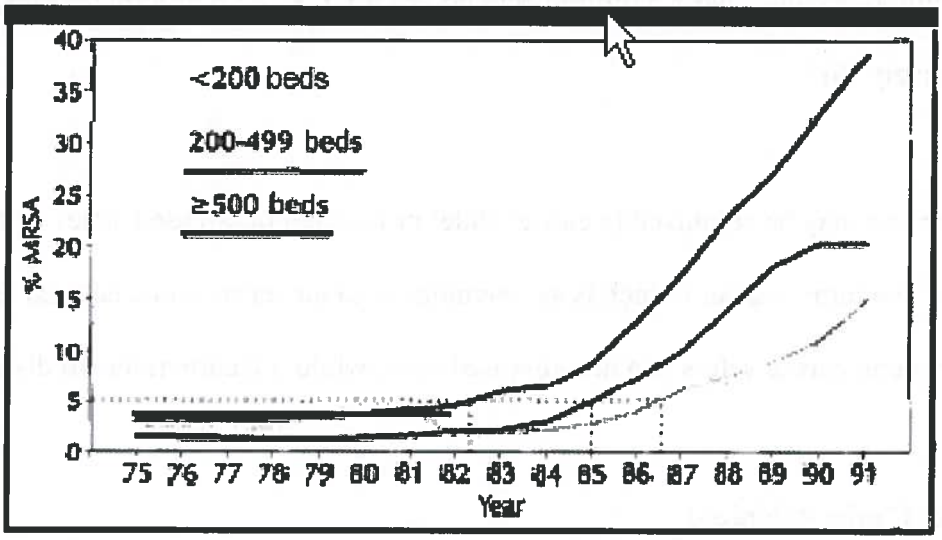
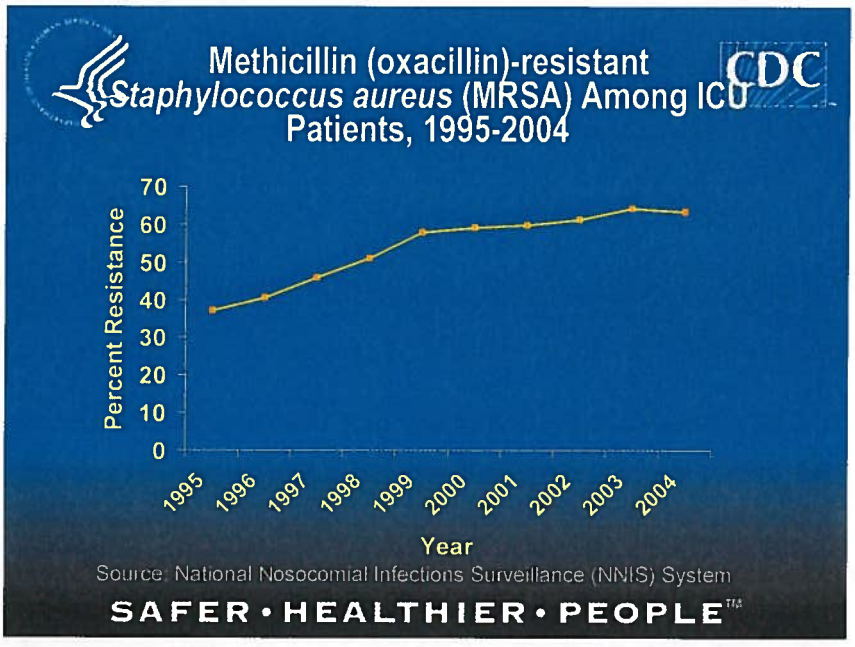


Figure 2: National Nosocomial Infections Surveillance System measurements of nosocomial MRSA in ICU's 1995-2004 [5].



For 2001–2002, national *S. aureus* and MRSA colonization prevalence estimates were 32.4% (95% confidence interval [CI], 30.7%–34.1%) and 0.8% (95% CI, 0.4% 1.4%), respectively, and population estimates were 89.4 million persons (95% CI, 84.8 94.1 million persons) and 2.3 million persons (95% CI, 1.2–3.8 million persons), respectively [6].

Humans may be colonized (a carrier state) or infected (a diseased state) with S. aureus. The terms carrier (which is synonymous with the term colonized) and infected are independent: carrier refers to a non-diseased state, while infection refers to disease.

(1a) Carrier/colonized

The definition of carrier (colonized) is that a host has a particular organism that is viably living upon it. In the example of *S. aureus*, this organism can viably live on human skin and other parts of the human body. The most common locations of colonization are: anterior nares, axillae, vagina, anus and damaged skin. A newly discovered site of colonization of *S. aureus*, the oral cavity and throat has been demonstrated.

Being a *S. aureus* carrier significantly elevates a person's risk of infection when compared to noncarriers [7]. This phenomenon of higher risk of infection when colonized with MRSA has also been demonstrated within the hospital setting [8]. Being a carrier does not always mean that infection will occur. Wertheim et al. [9] studied the incidence of bacteremia in carriers as well as non-carriers in a nonsurgical patient population (n =

14,008). This study found a significantly increased risk for *S. aureus* nasal carriers for acquiring a nosocomial *S. aureus* bacteremia, compared to noncarriers, with a relative risk of 3.0. (95% CI: 2.0–4.7) [14]. The bacteremic strain of the carriers had the same genotype as the nasal strain in approximately 80% of the cases.

Colonization may be intermittent or persistent and can last for years [10]. Between 10 and 35% of healthy individuals almost always carry one strain of *S. aureus* and are called persistent carriers. A larger proportion (20 to 75%) of individuals harbor *S. aureus* intermittently, and are called intermittent carriers. Finally, between 5 and 50% almost never carry *S. aureus* and are called noncarriers. Cross-sectional studies yield a prevalence of approximately 35% in the general population, which is actually a mix of persistent and intermittent carriers at that time point [7]. Because no systematic, population-based surveillance of community isolates of *S. aureus* exists, the true prevalence of MRSA cannot be determined [11].

(1b) Treatment of Colonization

The term "decolonization" rather than "eradication" may be more appropriate because eradication of MRSA carriage is not often successful and, if successful, often not permanent. The effect of any eradication or decolonization lasts 90 days at most [12]. A recently demonstrated randomized, double-blind placebo-control trial with soldiers (n=3447) included 134 participants initially colonized with CA-MRSA. Despite eradication attempts using mupirocin, a topical antibiotic active against MRSA, this study

showed no decrease in infections in the either mupirocin treated individuals or within their control group [13].

(2) Infected/diseased

The definition of being infected/diseased with a *S. aureus* infection is a result of the bacteria causing pathology in the human host. A person has *S. aureus* disease, whether the infection is located cutaneously or is systemic. These people will also be subsequently colonized. MRSA is one of the few pathogens routinely implicated in nearly every type of nosocomial infection, ranging from pneumonia to bacteremia.

A multinational survey, which included data from the U.S. from 1997-1999 that found 15,439 *S. aureus*–infected patients indicated that MRSA was the most commonly implicated source of nosocomial infections in the lung (46% of lung infections), the urinary tract (38% of urinary tract infections), the bloodstream (30% of bloodstream infections), and skin/soft tissues (30% of skin/soft tissue infections) [14] (**Table 1**).

Table 1: Percentage of bloodstream, lower respiratory tract, and skin/soft-tissue infections due to *Staphylococcus aureus* at SENTRY centers in the United States, Canada, Latin America, Europe, and the Western Pacific region, 1997–1999 [14].

No. (%) of infections due to S. aureus						
Site of infection	United States	Canada	Latin America	Europe	Western Pacific	All regions
Bloodstream	4405/17,399 (25.3)[a]	739/3840 (19.2)[a]	1092/5295 (20.6)[a]	2014/10,815 (18.6)[b]	679/3148 (21.6)[a]	8929/40,497 (22.0)[a]
Lower respiratory tract	1709/6711 (25.5)[a]	379/1659 (22.8)[a]	413/1914 (21.6)[b]	526/2572 (20.4)[a]	344/1696 (20.3)[b]	3371/14,552 (23.2)[a]
Skin/soft tissue	969/2328 (41.6)[a]	278/633 (43.9)[a]	432/1353 (31.9)[a]	880/2371 (37.1)[a]	369/789 (46.8)[a]	2928/7474 (39.2)[a]

[a] Rank of S. aureus among all other pathogens, 1.
[b] Rank of S. aureus among all other pathogens, 2.

There have been many reports of more serious diseases as result from MRSA infections in the community [15-18]. Some of the more serious, organ affected diseases include pneumonia, meningitis, cerebritis, brain abscess, osteomyelitis, endocarditis, septic arthritis. There are also diseases caused by the release of exotoxin from *S. aureus* including gastroenteritis and toxic shock syndrome.

Skin and soft-tissue infections represent the majority of the community-associated MRSA disease burden. Necrotic skin lesions are a common presentation and are often incorrectly attributed to bites by brown recluse spiders (even in areas where these spiders do not live) or insect bites. In addition, other pathological processes have been described in association with community-associated MRSA including, necrotizing pneumonia, pleural empyema, necrotizing fasciitis, septic thrombophlebitis with pulmonary

embolization, myositis, and severe sepsis with purpura fulminans and the Waterhouse–Friderichsen syndrome [19].

Presently, there are reports of MRSA infections occurring in all realms of the health care system, from health care workers to surgery patients and those in outpatient programs. Costs are higher for patients with MRSA infections with the main factor driving excess hospital costs attributable to the duration of hospitalization [20]. In a study with 479 patients with surgical site infections, the control subjects with no infections the cost was $29,455, $52,791 for patients with MSSA infections, and $92,363 for patients with MRSA infections [p <.001 for all group comparisons [21].

MRSA is now community acquired/associated (CA-MRSA) and not just hospital acquired (HA-MRSA).

The known distinguishing characteristics between HA-MRSA and CA-MRSA are listed in **Table 2**. CA-MRSA strains can also be distinguished, to a certain extent, by molecular typing methods such as pulse-field gel electrophoresis (PFGE) and multilocus sequence typing [22].PFGE is used to differentiate particular strain profiles such as the USA 300 strain, commonly seen in CA-MRSA. Sequencing SCC*mec* from many MRSA strains has revealed that there are five SCCmec types (I-V) that vary in genetic makeup and size. A majority of healthcare-associated MRSA clones have type I, II or III SCC*mec* and are multidrug resistant. In contrast, most community-associated MRSA (CA-MRSA) strains have type IV SCC*mec* and are not multidrug resistant, although SCC*mec* type IV

has been described in some HA-MRSA strains as well [23]. Some CA-MRSA strains have SCC*mec* type V; these isolates have no antibiotic resistant genes other than *mecA*.

During the middle of the 1990's, MRSA infections were beginning to be detected in the community, outside of the health care setting. These infections are distinguished as community-associated MRSA (CA-MRSA) and have not been found to have any relationship with the health care setting [24]. There is some debate in the literature today about the blurring of the descriptions of CA-MRSA and HA-MRSA. Some have argued that CA-MRSA must have originated from HA-MRSA, as others think the CA-MRSA strains have evolved independently. Today, we are seeing CA-MRSA causing outbreaks in health-care settings, usurping HA-MRSA. Although many have disagreed on how to properly categorize the strains of MRSA, all have agreed that it is a serious pathogen and one that is affecting patients in health-care settings as well as in the community.

Table 2: Comparing the Characterstics of CA-MRSA and HA-MRSA. Derived from Daum, RS 2007 & Beam, JT 2005 [19, 25].

Characteristic	CA-MRSA	HA-MRSA
Usually susceptible to clindamycin	+	-
Less often multiple-resistant to other non-β-lactam antibiotics	+	-
High prevalence of genes encoding the two-component Panton-Valentine leukocidin [26]	+	-
Small DNA cassettes mediating methicillin resistance	+	-
Toxin-producing	+	-
Health Care Exposure	-	+
SCC mec type	IV	II
Lineage	USA 300, USA 400	USA 100, USA 200

Described populations and risk factors for CA-MRSA colonization and infection

CA-MRSA has been described in several well-defined populations, such as children, incarcerated persons, Alaskan Natives, Native Americans, Pacific Islanders, sports participants, intravenous drug users, and military personnel [22]. Until more research is performed, it remains unclear why the above mentioned ethnic groups have higher rates of MRSA infections. Some of the known factors that facilitate the spread of infection have been shown and include crowding, frequent skin-to-skin contact between individuals, participation in activities that result in compromised skin surfaces, sharing of personal items that may become contaminated with wound drainage, and challenges in maintaining personal cleanliness and hygiene [16].

A number of studies have assessed certain risk factors for CA-MRSA acquisition; these risk factors included recent hospitalization, outpatient visit, nursing home admission, antibiotic exposure, chronic illness, injection drug use, and close contact with a person with risk factor(s). The most common risk factors assessed were recent hospitalization and chronic illness requiring health care visits[25]. In Bartlett's 2007 review of CA-MRSA [27], he has labeled the 5 C's as the risk factors associated with infection of CA-MRSA as:

1. Contact;
2. Crowding;
3. Contaminated items;
4. Compromised skin integrity; and
5. Cleanliness.

Background Literature about Sports Teams

Sports in General

In sports outbreaks, environmental sources, such as sharing of clothing, equipment, towels, balms, lubricants, razors, and soaps; improper care of skin trauma; direct skin-to-skin contact with MRSA lesions; and crowded living conditions were identified as possible risk factors for MRSA acquisition[22, 25].

It has been reported and originally accepted that MRSA outbreaks/clusters have occurred on sports teams in which physical contact between players is an accepted part of the game. The hypothesis is that there must be physical contact during competition and/or training for transmission of pathogens to occur. The most commonly reported risk factor for developing a CA-MRSA infection is injury to the skin The majority of infections occur in areas of the body most susceptible to skin trauma [28]. The bulk of these reports have been from outbreaks of American football teams, (high school, college and professional), wrestling, basketball and rugby [29-41] (**Table 3 in appendix**).

In the largest outbreak of CA-MRSA (USC football) infection reported among competitive athletes, the sharing of soap bars with teammates was identified as the strongest risk factor for disease acquisition during a retrospective analysis (OR = 15.0, 95% CI = 1.69-180)[33]. In this study, other determined risk factors for infection were preexisting cuts or abrasions of players. The investigators of this study also measured carrier-control rates and determined the highest risk factors for being a carrier (carrier being a positive MRSA culture obtained from anterior nasal swabs) was associated with

having a locker near a teammate with a skin and soft tissue infection (SSTI), sharing towels, and living on campus.

We are now seeing outbreaks of skin and soft tissue infections (SSTIs) as a result from *Staphylococcus aureus* in traditionally accepted non-contact sports, including river rafting guides, volleyball, weight lifters, surfers and fencers [17, 18, 39, 42, 43].

Some have argued that the source of exposure of MRSA on a sports team must be from an exposure to HA-MRSA. The argument is that most MRSA colonization and infection was the result of having health care-associated risk factors or contact with others with risk factors [25, 44]. This means that an athlete would have to be in direct contact with either someone who works in the health field and/or a patient from a hospital setting. Although most athletic injuries and/or illnesses do not require hospitalization, athletes are often referred to outpatient clinical services, including surgery, rehabilitation, radiology, and laboratory offices. There is also evidence of transmission of MRSA from one team to another team. This team-to-team spread has been documented in contact sports (football and rugby) [31, 37].

For an athlete (or anyone) to become infected with MRSA there must be two occurrences, (1) the athlete must have exposure to the bacteria, either directly touching another person who is infected and/or a carrier or indirect contact via a fomite that has the bacteria upon it AND (2) there must be some level of trauma to the skin, either micro or macro (bruise, laceration, chafing, razor use, burn, etc.). Close contact between athletes during high intensity training periods, which usually is the weeks preceding the start of the season, has also been reported as a possible risk factor in many studies [29, 33, 35].

These times of training (for upcoming seasons) are similar to the times when new military recruits that live in close quarters during intensive training and MRSA outbreaks have been described in the military [45, 46]. Military recruits often have minor cuts or abrasions that increase the risk of developing skin infections. Such injuries would be expected during physically demanding activities such as running, hiking, and negotiating obstacle courses. Most MRSA infections in this population occur on exposed surfaces such as arms, legs, and knees [46].

Sports associated *S. aureus* infections

In most reported cases of MRSA and MSSA outbreaks amongst sports teams, the site of infection has been cutaneous. Most of these SSTIs are of the mild form, consisting of impetigo, folliculitis, furuncles and carbuncles [15]. Large, fluctuant furuncles and carbuncles require incision and drainage combined sometimes with systemic antimicrobial therapy and close follow-up [47]. Some infections persist and require hospitalization. To date, there is little evidence of *S. aureus* causing outbreaks of bacteremia, sepsis or other invasive diseases on sports teams as a result of athletic competition there is some, including deaths. Amongst sports participants SSTIs are the most prevalent types of disease caused by *S. aureus*, but in another closely related gram positive coccus, *Streptococcus pyogenes,* there has been a report of nephritogenic pyoderma on a rugby team [48]. A fencer in 2003 suffered from paraspinal myositis as a complication of bacteremia of *S. aureus* and had to be hospitalized for eleven days. In a 2007 review of MRSA infections on sports teams by Patel et. al., the authors reported a Division III football receiver who contracted a CA-MRSA infection that progressed to

bacteremia, sepsis and death [49]. These infections are far more serious than Freeman & Bergfeld reported in their 1977 review in CUTIS of skin diseases of football and wrestling participants [50]. Freeman & Bergfeld report Staphylococcal infections as, "…may be a disabling disorder, but it does not spread in epidemic proportions."

Water Sports

Sports in which participants use water, (either salt or fresh) in their training and in competition (i.e. water polo, rowing, swimming, surfing, etc.) have different risks than those of non-water sports. Moisture will contribute towards the growth of microorganisms on the skin. After an athlete removes themselves from the water environment, their skin is susceptible to microinjuries such as the skin becoming too dry from the loss of its natural oils resulting in an irritating, drying effect of the skin and hair [51]. If the skin is chronically dried and re-wetted, it can become cracked allowing for microorganisms to invade. In 2003, Sam Sunshine argued in his review of surfing injuries, that repeated water exposure impairs the skin's healing properties and that a healing wound should at minimum be kept out of water for 2 days [43]. Decker et al. [18] have argued that frequent and prolonged immersion in water delays healing and enhances the ability of *S. aureus* to cause infection. Wounds of the skin in a marine environment are often contaminated with organisms such as *Vibrios, Alternomonas, Mycobacterium*, and *Pseudomonas* species [52, 53]. *S. aureus* is not found naturally occurring in water, it is only found living on its host, in this case, human skin or upon fomites in which human contact has occurred.

Interventions used for sports teams with MRSA and MSSA outbreaks

Of the seventeen described outbreaks on sports teams in the literature, eleven describe various interventions that were administered and/or recommended for sports teams with players infected with *S. aureus* (**Table 4**). These interventions fall into two categories; those designed to control an emerging outbreak on a team and those that are designed for the prevention of future outbreaks. The techniques described and/or implemented may or may not be mutually exclusive of either prevention or immediate control of outbreaks. For example, the technique of educating athletes about the importance of hand washing after practice is both a measure used for preventing outbreaks as well as for the control of an already established outbreak.

The most prevalent methods of intervention used were determining colonization status of teams and/or staff and increasing the frequency of cleaning of training facilities. Only interventions that were administered, i.e. were conducted on the teams, were counted, thus excluding recommended interventions. Seven of the seventeen studies gave recommendations they felt would be the most useful interventions for their studied teams.

Table 4: Frequency of administered interventions of sports teams with *S. aureus* outbreaks.

Intervention	Number of Studies
Colonization/Decolonization Techniques	
Nasal cultures of all players and/or staff	6
Intranasal mupirocin administration a. carriers[1] b. entire team	 a. 1 b. 1
All players were questioned for mupirocin compliance	1
Sanitation Techniques	
Increased frequency of cleaning training facilities	6
Disinfection of whirlpools	2
Cessation of whirlpool use	1
Team Education	
Hygiene behavior education	3
Disseminated CA-MRSA educational materials to staff and team	1
Posted hand hygiene signs in locker room	1
Player Hygiene Techniques	
Installation and use of antibiotic soap dispensers[2]	5
Towel use a. increased towel availability b. cessation of towel usage c. washing towels with water consistently >70 degrees Celsius	 a. 1 b. 1 c. 1
Active/weekly inspection of infectious lesions	3
Skin lesions fully covered during contact (football)	1
Careful bandaging of the skin before adhesive tape application	1
Banned the use of communal containers of emollients (e.g. petroleum jelly)	1
Frequent changes of socks and T-shirts	1
Properly fitted shoes to avoid blistering	1
Infected Players Role on Team	
Players banned/restricted from practice	3
Skin lesions fully covered during contact	1
Patients treated with cotrimoxazol until infections were healed	1

[1] One study had patients use chlorohexidine, gluconate scrub, providone iodine and mupirocin ointment for 5 days, Huijsdens et. al. 2006.
[2] Chlorohexidine; hexachlorophene/phisohex.

There does not seem to be an agreement in the literature as to whether sports participants with MRSA infections should be allowed to practice/train and/or compete. In fact, three of the seventeen studies (football, wrestling and rugby) either banned or restricted players infected with *S. aureus* from practicing until their infections were medically treated [32, 34, 37]. It is now generally accepted that the decision whether to allow infected players on the field or in training should be made on a case by case basis [15], although some would argue that a more cautious approach be implemented. In the 2007 review by Sedgwick et. al. of bacterial dermatoses in sports, the authors argue that since there are no concrete recommendations of return to play, that the rules for wrestling be followed, even in sports that are non-contact or collision, which share equipment such as gymnastics or aquatic sports [54]. These wrestling rules are instated from two governing bodies, The National Collegiate Athletic Association and The National Federation of High Schools. They state that if any athlete has dermatosis symptoms, which include impetigo, folliculitis, furuncles, abscesses, erysipelas, and/or cellulites that return to play shall be granted only after no new lesions for 48 hours, completion of 48-72 hours of oral antibiotic treatment and no moist, oxidative/oozing/draining lesions. These guidelines are summarized in **Table 5**.

Table 5: Wrestling return to play guidelines for bacterial dermatoses. Adapted from Sedgwick et. al. 2007.

Dermatosis	National Collegiate Athletic Association Guidelines	National Federation of High School Guidelines
Impetigo, folliculitis, furuncles, abscesses, erysipelas, cellulites	No new lesions for 48 h	Oral antibiotics for 48 h
	Completed 72 h of oral antibiotics No moist, exudative, or draining lesions.	No drainage, oozing, or moist lesions

Patel et. al. [49] have recommended a decontamination protocol for the entire team, staff and athletic facility personnel which includes daily chlorohexidine washes and intranasal mupirocin ointment application twice daily for 5 days. The CDC recommends these guidelines for any person with a SSTI:

1. Keep wounds that are draining covered with clean, dry, bandages.
2. Clean hands regularly with soap and water or alcohol-based hand gel (if hands are not visibly soiled). Always clean hands immediately after touching infected skin or any item that has come in direct contact with a draining wound.
3. Maintain good general hygiene with regular bathing.
4. Do not share items that may become contaminated with wound drainage, such as towels, clothing, bedding, bar soap, razors, and athletic equipment that touches the skin.
5. Launder clothing that has come in contact with wound drainage after each use and dry thoroughly.

6. If you are not able to keep your wound covered with a clean, dry bandage at all times, do not participate in activities where you have skin to skin contact with other persons (such as athletic activities) until your wound is healed.

7. Clean equipment and other environmental surfaces with which multiple individuals have bare skin contact with an over the counter detergent/disinfectant that specifies *Staphylococcus aureus* on the product label and is suitable for the type of surface being cleaned.

The California Department of Public Health through the California Department of Health Services has published a document intended for the use of athletic departments about "Participation in Sports" when dealing with CA-MRSA [55] Listed below are their recommendations:

- An athlete who has a draining wound and is in a sport where there is regular physical contact with others should be evaluated by a physician or qualified health provider for participation in that sport. Considerations for continuing participation in the sport while the wound is still leaking fluid would include:
  - Ability to completely contain the drainage with a clean, dry bandage;
  - Stability of equipment/padding that covers the wound
  - Amount of drainage
  - Location of the draining wound

– The nature of the contact. Frequent pressure on a bandaged wound (for example, against a piece of athletic equipment) may both delay healing and contaminate the point of contact.

Awareness, Attitudes & Beliefs about MRSA infections

To-date there have been no studies describing the opinion of athletes regarding allowing infected players to return to the playing field. The allowance of athletes participating in practice and in competition is multifactorial. First and foremost is the requirement of the safety of all those participants involved. Health status of athletes falls under the safety motif. This safety concern is directly related to what is known of the morbidity and mortality rates of MRSA infections. As health care providers, we must think of obtaining the optimal health possible for our patients. Sports teams, especially at the professional levels, and to some extent the collegiate level have higher expectations of producing winning records and teams. These expectations may have an impact into treatment of players with certain diseases. Awareness of MRSA infections is extremely important for reducing risk factors for acquiring infections.

In a 2005 study by Gill et. al. [56] amongst 50 patients and 100 National Health Service (NHS) employees (25 doctors, 25 nurses, 25 domestics and 25 porters) in the UK, a questionnaire was given to determine the level of awareness of MRSA infections. There was a high level of awareness of MRSA among both patients/visitors (94%) and NHS employees (100%). The media was the most common source of information for patients/visitors (68%) compared with 24% of NHS employees ($P<0.01$). Perceived risk

of contracting MRSA was very similar among patients/visitors and NHS employees (34% vs. 35%, P<0.10). Fifty-two percent of doctors felt that they were at risk compared with 13% of domestic employees. This study showed a high level of awareness among the general public and healthcare workers alike. In a similar study by Hamour et. al. in 2003 [57], 113 surgical out-patients completed a questionnaire stating whether they had heard of either superbugs or MRSA. Fifty patients (44%) had heard of superbugs or MRSA mainly via the media (58%) or from hospital staff (44%). The majority would feel either angry or afraid if they acquired MRSA in hospital, but there was good awareness of both methods of infection control and the consequences of infection. The authors of this study concluded that the media is at least as important as health professionals in providing information. The question still remains on how those infected with MRSA perceive their infections and isolation.

In a 2001 hospital setting study conducted in the UK by Newton et. al. [58], participants were asked about their perceptions of being infected with MRSA together with their experience of source isolation. Participants were unclear about the nature of MRSA, and generally did not perceive the infection to have a significant impact upon their life (either in terms of the presence of symptoms or in restriction of activities). Despite this, roughly half the sample thought that an MRSA infection was 'serious'. Only one participant clearly viewed their MRSA as hospital-acquired, most being uncertain about the mode of transmission or viewing it as unrelated to the behavior of care staff. Few respondents displayed an accurate knowledge of the reasons for source isolation and barrier nursing. Isolation was viewed as having advantages and disadvantages. There was

little evidence of a detrimental psychological effect of isolation. Patients infected with MRSA appear to understand little about their condition or the necessity for barrier nursing and source isolation.

These studies demonstrated the levels of perceptions that both patients and health care providers have about MRSA and might be extrapolated for the sports community context. In sports, a player may be ostracized and/or isolated from their team during a period of having a MRSA infection. It is important to ascertain the levels of perceptions from those who are on sports teams about MRSA infections. The understanding of these perceptions, will yield another tool used for the prevention and treatment of MRSA outbreaks on sports teams.

In summary, the history of *S. aureus* and its antibiotic resistance in this country has been well documented. Now, we are seeing MRSA infections outside of hospital settings. There are known high risk populations in the community. One of the known high risk populations is those of contact sports, particularly football, wrestling and rugby. It is known that sports teams have particular risk factors for MRSA outbreaks. One of these risk factors is the colonization status of the athletes and coaches on the teams, with those being colonized having a higher risk of infections. Many different interventions have been used in preventing outbreaks from occurring on sports teams. One of the particular interventions is not allowing infected athletes from practicing and/or competing with the team. This method is not proven effective and may possibly have detrimental impacts on the infected athletes being removed from their teams.

It is important to understand the relationship between outbreaks and colonization, and the relationships between knowledge, practices and beliefs and outbreaks on sports teams. These relationships have potential efficacy in the designing of specific interventions for preventing MRSA outbreaks from occurring in the future as well as stopping extant outbreaks.

Introduction

During the 2005-2006 academic year, I was informed of an outbreak of MRSA infections amongst the University of California, Berkeley (UCB) intercollegiate men's crew team. Seven members of the men's crew team were treated for SSTIs that were confirmed to be MRSA (personal communication). Most of these infections occurred on the skin of the hands and legs. This knowledge prompted questions pertaining to why a possible MRSA outbreak occurred on this team and what immediate interventions could be used to prevent future outbreaks. To-date, there have been no previous reports of MRSA outbreaks on crew teams. This is a descriptive study designed to explain the prevalence of *S. aureus* and MRSA carriers on the UCB men's and women's crew teams, as well as to show what's known about the two teams' knowledge, practices and beliefs of communicable disease outbreaks and MRSA infections. A few weeks after confirmation of this outbreak, the men's team was visited in their off-campus training facility by Dr. Cindy Chang, Director of Intercollegiate Athletics of the University Health Services, Tang Center of UCB and Dr. John Swarzberg, Director of the UC Berkeley - UC San Francisco Joint Medical Program. Dr. Swarzberg is also an Infectious Disease

consultant to the Tang Center. The purpose of this visit was to give immediate education to the entire team addressing their current MRSA outbreak. This included a brief discussion about MRSA and infectious disease outbreaks and a question and answer session. A tour of the facility was conducted to find any possible sources of risks for initiating MRSA infections.

Because of the known prior outbreak on the men's crew team, I hypothesized that this team would have a higher prevalence of MRSA carriers when compared to the women's team. I also hypothesized that the men's team would have more risks for MRSA infections based on their practices, lower knowledge about MRSA and outbreak control as well as different beliefs about MRSA infections as it pertains to training and practice when compared to the knowledge, practices and beliefs of the women's team.

Knowledge? is defined as knowing what MRSA is and how it is spread, as well as what should be done to prevent possible infections and/or outbreaks from occurring. Practice is defined as what risk factors someone has that are known to be risk factors for MRSA infection (e.g. antibiotic usage, prior hospitalization, injectable drug use, etc.) Beliefs are defined as what a person feels about an athlete with known MRSA infection allowing participation in practice/training and competing.

Materials & Methods

Sample Population

The populations chosen for this study were the UCB men's and women's crew teams during the 2005-2006 academic year. Permission to include these teams in this study was initially granted by the head coaches of each team, medical staff and trainers. This study was reviewed and approved by a full board committee review in the University of California, Berkeley's Office for the Protection of Human Subjects (IRB CPHS Protocol #2005-9-24). Both the men's and women's teams have a varsity team and a junior varsity team and in any given year there are approximately 60 players and 6 coaches on each team. These teams, like most intercollegiate teams, train year round, but have more rigorous training when the academic year begins (August) throughout the season, ending around the later part of May. The intercollegiate competitions (meets) occur during the spring semesters (January to June). The men's and women's teams train in the boats at separate facilities that are both located off campus but when training on campus, both teams use the same facility. Four initial meetings were arranged with each team (2 junior varsity and 2 varsity) to explain the study, answer all questions, and deliver consent forms. The idea of coercion of volunteering for this study was explained by detailing that participation in this study is strictly on a volunteer basis and that participation in it or lack there of will yield no effect on team status. A second meeting was arranged with all four teams to collect consent forms, take nasal swabs and administer surveys.

Survey

A written survey was derived and administered to all participants in this study (see appendix for copy). The questions in this survey were derived from new, unpublished CA-MRSA infection guidelines intended for California sports teams. These guidelines were developed by Dr. Jon Rosenberg of the California Department of Health Services (now the California Department of Public Health, http://www.dhs.ca.gov/) using published guidelines for controlling staphylococcal infections in athletic departments and in non-hospital settings from the Texas Department of State Health Services, Infectious Disease Control Unit, and from the Los Angeles County Department of Health Services.

The survey has questions that ask about demographics (e.g. age, self-identified ethnicity, etc.); knowledge about MRSA and recognizing as well as preventing staphylococcal infections (e.g. hygiene knowledge, what is MRSA, etc.); practices that will put an athlete at risk for the development of staphylococcal infections (e.g. antibiotic usage history, intravenous drug use, etc.); and beliefs as to whether athletes should be allowed to train and/or compete with MRSA or MSSA infections. Administration of the survey as well as swabs from the anterior nose was done with each participant while in the teams' training facility (shared by both the men's and women's teams). Each team was instructed to take the survey individually (i.e. to not share answers or hold discussions) and progress through each question as if someone were asking them these questions. This announcement was made in attempt to maintain autonomy of answers as

well as to prevent participants from changing their answers to questions by using other questions as possible sources of knowledge.

These data from the surveys were inputted into a Microsoft Excel spreadsheet and calculations for frequencies of answers were completed for each team. These data included demographic information for each participant.

Laboratory Methods

Collecting Nasal Swabs

An initial informational meeting was set-up with the men's and women's teams, in which the study was proposed and consent forms were distributed for collection during the second meeting. This second meeting was used for two purposes: the first was the administration and collection of a survey and secondly, to obtain a nasal swab. These meetings were at the men's and women's crew training facility, held on the campus of UC Berkeley. Both teams use the same facility during their training on campus.

All of the following collections and lab work were performed by the principal investigator. To obtain a nasal swab, each participant was invited into a separate room from the rest of the team. While in privacy, a sterile swab was held with a gloved hand between the thumb and index finger, which was rolled while in the participant's left and right external nares. These nasal swabs were intended to be collected as uniform as

possible. The swab was then immediately rolled onto a blood agar plate that was labeled with the matching number of the participant's survey.

The newly collected samples were immediately taken into the laboratory for work-up. The first step was to streak-out the area that was rolled with the nasal swab and incubate these plates overnight at 35°C, which is the optimum temperature for *S. aureus* growth. *S. aureus* is a β-hemolytic organism, therefore each colony that had complete clearing on the blood agar (i.e. turned the blood agar color from red to yellow/clear) were then selected and transferred to mannitol salt agar plates (MSA) to be incubated overnight at 35°C. If a blood agar plate was found to contain no colonies that cleared the color, then two colonies were picked and also transferred to MSA plates for overnight incubation. MSA selects for organisms that can tolerate high salt and differentiates based on mannitol fermentation. *S. aureus* is a mannitol fermentor, turning the MSA plates yellow. Positive and negative controls were also plated. The positive controls were obtained from the laboratory of George Sensabaugh in the School of Public Health at UC Berkeley. Negative controls were from sterile saline.

Individual colonies from the yellow MSA plates were carefully picked without touching the surrounding agar and streaked on blood agar plates for overnight incubation. Purity of these plates was essential as there may possibly be cells of other organisms that grow slow on MSA, yet can not be seen.

Perform latex agglutination test

To determine if the colonies that grew successfully on the MSA plates were actually *S. aureus,* a plasma coagulase test ("Staphaurex" by Remel [59]) was used. These colonies, which were grown on blood agar after successful growth on MSA plates were mixed with the polystyrene latex particles that are coated with fibrinogen and IgG. When mixed on a slide, reaction of clumping factor with the fibrinogen, and/or protein A with the IgG causes rapid, strong agglutination of the latex particles. A negative result is no agglutination after 5 minutes time.

Those that are positive, grown on antibiotic plates

All of the plates that yielded positive latex agglutination tests were saved and colonies were selected to be re-grown on blood agar plates overnight. Three colonies were then re-suspended in 2 mL of sterile 0.85% NaCl solution, of which 10 μL drops were placed onto blood agar plates containing oxacillin that are MRSA selective. Positive and negative controls were also placed on each oxacillin plate. These plates were checked daily while being allowed to grow for 5 days at 35°C.

Results

Study Population

There were a total of 63 participants in this study (59 athletes, 3 coaches and one trainer). Amongst the women's crew team, there were 42 athletes (median age of 19, 18-22) and 3 coaches recruited to participate in this study. The men's team had 17 athletes participating (median age of 20, range 20-26), one of whom was a woman. One athletic

trainer that interacts and treats both teams participated. The frequencies and percentages of the self-identified race/ethnicity are listed in **Table 6** below.

Table 6: Frequencies and percentages of self-reported race/ethnicity amongst the participants of the men's and women's crew teams.

	Frequency, %					
Team (N)	White/Caucasian	Asian	Mixed-Irish & Chinese	Mixed-Caucasian/Mexican-American	Mixed-non-specific	Other
Women (45*)	39, 87%	4, 9%	1, 2%	-		1, 2%
Men (17)	15, 88%	-	-	1, 6%	1, 6%	-

*42 athletes and 3 coaches.

Colonization Frequencies

Colonization of *S. aureus* in the anterior nares was assessed and is as follows: 11 of the 42 (26.2%) women's crew team participants were MSSA positive with one person being MRSA colonized; the men's team had 5 of 17 (29.4%) colonized with MSSA and no MRSA carriers; the coaches/trainer group had 1 of 4 (25%) colonized with MSSA and no MRSA carriers (**Table 7**). When counting the one person with MRSA colonization and the three women's team coaches and trainer, then there would be a total of 13 colonized of 46 (28.3%). These calculated carrier status means were not significantly different when comparing the entire women's team (including the one MRSA carrier, coaches/trainer, and athletes) and the men's team ($p = 0.4696$, 1-tailed T-test) or when comparing the women's team athletes with the men's team athletes ($p = 0.4549$, 1-tailed T-test).

Survey Results

The survey results were counted for frequencies with respect to questions pertaining to knowledge, practices and beliefs.

For the knowledge-based questions, the averages of the total correct answers were very similar between the participants of the men's and women's crew teams. The women's team answered 74% of the questions correctly, and the men's team had 76% of the answers correct (**Table 7**). These averages between the teams were not significantly different (P = 0.4473, 1-tailed T-test).

The overall prevalence of risk factors (practices) for obtaining a MRSA infection amongst the participants was low, around 10% (**Table 7**). This number was calculated with the frequency of answers to all risk factor questions on the survey per team. The most prevalent risk factor reported on the surveys was that of having previous antibiotic usage in the past twelve months (i.e. the number of players on each team that used antibiotics in this period) 40% for women's crew and 29% for the men's crew respectively.

When asked to report previous "Staph" infections, 7% of the women's crew team responded yes and another 5% were unsure, while one participant had a single previous skin infection but chose to respond no when asked of having any previous "Staph" infections. Amongst the men's crew team, 18% admitted having a previous "Staph" infection and another 24% were unsure.

Table 7: *S. aureus* carriers and correct answers of knowledge based questions and practices/risk factors for obtaining MRSA infections.

Group (n)	Prevalence of *S. aureus* carriers (%)	Correct Knowledge Answers, %	Risk Factors, %
Women's Crew (42)	12 (28.6)	74	10
Men's Crew (17)	5 (29.4)	76	8
Coaches/Trainer (4)	1 (25.0)	74	11

$p = 0.4473$ (1-tailed T-test) When comparing the knowledge answers of the women's and men's crew teams.
$p = 0.3682$ (1-tailed T-test) When comparing the reported risk factors of the women's and men's crew teams.

Beliefs about whether or not athletes should be cleared to train/practice and compete are listed in **Table 8**.

Table 8: Survey answers about the beliefs concerning return to train/practice and competing in the crew teams and the coaches/trainer groups.

Answers to Questions About Known MRSA Infection Status (%)	Yes	No	Unsure
Someone else is MRSA+; should they be cleared to train/practice?			
Women's crew	19	40	40
Men's Crew	47	24	29
Coaches/Trainer	75	0	25
Someone else is MRSA+; should they be cleared to compete?			
Women's crew	14	40	45
Men's Crew	47	24	29
Coaches/Trainer	75	0	25
You are MRSA+; should you be cleared to train/practice?			
Women's crew	31	31	38
Men's Crew	47	18	35
Coaches/Trainer	75	0	25
You are MRSA+; should you be cleared to compete?			
Women's crew	26	38	36
Men's Crew	47	18	35
Coaches/Trainer	75	0	25

Survey Results of *S. aureus* Carriers vs. Non-carriers

S. aureus carriage status **(n)**	Correct Answers, %	Risk Factors, %
Carriers (18)	78	12
Non-carriers (45)	73	9

$p = 0.3106$ (1-tailed T-test) When comparing the knowledge answers of the carriers and the non-carriers.
$p = 0.3555$ (1-tailed T-test) When comparing the reported risk factors of the carriers and the non-carriers.

Discussion

Colonization rates

Both teams had roughly the same percentage colonized with *S. aureus* that is in the community (~30%). This is strong evidence against the hypothesis that the men's crew team would have a higher carrier rate than the women's crew team. A caveat is that the sample size of 17 participants from a team of 60, it is still possible that there could be a higher percentage of carriers on the men's team. There was only one person found to be colonized with MRSA, which is not different from what you will also find in the community.

Knowledge, Practices and Beliefs

The survey answers demonstrating knowledge and practices were not significantly different between the two teams. This finding was inconsistent with my hypothesis because of the knowledge of a previous MRSA outbreak on the men's team. A confounding feature in this study is that there was some education made to the teams in the form of interventions (e.g. education, soap dispensers, more surveillance of wound

care, etc.) before the survey questions were administered to these teams. The risk factors (practices questions) between the teams were also found to be not significantly different, a finding that was not consistent with my hypothesis.

There were a higher percentage of participants on the men's crew team that believed that either they or someone else should be allowed to train and/or compete knowing they have a MRSA infection. Interestingly, ~30% of the women's team participants felt that if they had a MRSA infection, that they should be allowed to compete and practice, whereas only 14% thought that someone else with MRSA should be allowed to compete and 19% should be allowed to practice. These differences between the two teams may be attributed to the men's team having a familiarity with MRSA due to their prior outbreak of MRSA. These feelings may have arisen with some interventions that were already administered prior to this study. Also, reassurances from their coaches and medical staff have played a role in their understanding of the problem.

This study had a much lower participant rate then was originally anticipated. This can be partly explained because of the very low participant turnout from the men's crew team. The team originally consented to participate, and then decided against being part of this study, and finally deciding for participation, although only 17 athletes and no coaches consented. The men's team decisions were entirely out of the study's control. This study was originally intended for two surveys, an initial, baseline survey, and then a follow-up survey after more interventions were performed.

This study found that there was no difference in carriage amongst the UCB men's and women's crew teams, and no difference in knowledge or practices. The beliefs were different between the teams, with the men's team having a higher percentage believing that either they or someone else with a MRSA infection should be allowed to train/practice and compete. Future studies into why MRSA outbreaks occur on crew teams should try and answer questions around behavior. This was the first study of this kind demonstrating MRSA colonization rates on a crew team, as well as surveying the team to find their knowledge, practices and beliefs about MRSA infections. This study serves as a pilot study for those interested in preventing MRSA outbreaks on sports teams, with emphasis on non-contact sports and water sports.

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Appendix

Table 3 (on following pages): Reported CA-MRSA Outbreaks within Sports Adapted from Turberville et al (2006)

Author and Date	Sport	No. of Athletes (Attack Rate, %)	Age (Range)	Organism	Site of Infection/Disease	Source/Mode of Infection	Comments
Huijsdens et al (2006)	Dutch Soccer team (including the roommates of players), The Netherlands	9 players + 2 roomates = 11/56 (20)	31 (18-43)	CA-MRSA ST80-IV	Skin	Undetermined but hypothesized as by skin-to-skin contact, sharing equipment or personal items.	The same strain of MRSA was found to cause infection in a player from a neighboring team after a game with the studied team.
Kazakova et al (2005)	Professional football team, St. Louis Rams	5/58 (9)	27 (23-33)	CA-MRSA USA 300-0114	Skin	Skin breaks and turf burns; suboptimal hand washing and personal hygiene may also have contributed	Large abscesses that required surgical intervention and IV antibiotics; no players were hospitalized
Nguyen et al (2005)	Collegiate football team, Los Angeles	11/107 (10)	20	CA-MRSA USA 300	Skin	Sharing of personal items such as soap and towels with other teammates	4 athletes were hospitalized; 9 players required surgical incision and drainage; linemen had the highest attack rate (18%)
Rihn et al (2005)	High School football team, Pennsylvania	13/90 players (14); 0/12 staff members	NR (9^{th} – 12^{th} grade)	CA-MRSA	Skin	Person to person including playing a lineman position; holding junior class status	Attempts at controlling the MRSA outbreak with mupirocin failed; recurrent infections occurred in 6 players.

Centers for Disease Control and Prevention (2003)	Fencing club, Colorado	5/62 (8)	31 (11-51)	MRSA	Skin; 1 with paraspinal myositis	Common source: contaminated sensor wire shared by athletes	3 were hospitalized; 1 patient for 11 days
Centers for Disease Control and Prevention (2003)	High School Wrestling, Indiana	2	NR	MRSA	Skin	Most likely indirect transmission because wrestlers were on different teams	Case report; isolates were not available for PFGE
Lindenmayer et al (1998)	High School Wrestling, Vermont	7/32 (22)	14-18	MRSA	Skin	Person to person; source case was likely a wrestler colonized with MRSA (positive anterior nares culture)	
Stacey et al (1998)	Rugby team, England	5/15 (33)	NR	MRSA phage group I + III	Skin; large cutaneous infections on arms, back, neck, and face	Likely transmitted from a player with positive nasal culture	Identical strain; first community outbreak of MRSA in England
Sosin et al (1989)	High School football/basketball, Kentucky	31/124 (25)	NR	*S. aureus*	Skin, scalp, arms, and legs	Person to person	3 athletes required hospitalization; no outbreak strain was identified
Decker et al, (1986)	River rafting guides, Tennessee, North Carolina, and South Carolina	19/38 (50)	18-35	*S. aureus*	Skin	Frequent minor skin wounds acquired while rafting, and prolonged close contact among the persons with wounds.	Repeated immersion in water likely enhanced the development of infections.

Begier et al (2004)	Collegiate Football team, Connecticut	10/100 (10)	17-22	CA-MRSA USA 300	Skin	Person to person; skin breaks and turf burns facilitated infection; contaminated whirlpool may also have contributed	2 players required hospitalization
Saben (2004)	Collegiate Football team, Davis, California	6	NR	MRSA	Skin	Unknown	Case Report; Immediate response to this outbreak included prevention of towel sharing, additional waterless antibacterial hand was dispensers in the training room, initial suspension of whirlpool use for 3 months followed with daily chlorine monitoring and draining with disinfecting the whirlpools
Centers for Disease Control and Prvention (2003)	Collegiate Football, Pennsylvania	10	NR	MRSA	Skin	Skin trauma from turf burns, shaving, and sharing unwashed towels	7 of 10 athletes were hospitalized

Bartlett et al (1982)	High School football, Illinois	26/142 (18)	NR	*S. aureus*	Skin; furunculosis; upper extremities	Person to person; no common source identified	
Baron (1977) see Bartlett et al	High school football, North Carolina	18/76 (24)	NR	*S. aureus*	Skin; furunculosis; forearms, elbows, and knees	Case source likely player with positive nasal swab	Unpublished data; three players, two of whom had boils, had nasal swabs positive for the same phage type of *S. aureus* as was responsible for the boils
Pollard (1966)	Collegiate football, New Hampshire	34	NR	*S. aureus*	Skin; upper and lower extremities	Inadequate sterilization of locker room facility	Cases reported over 3 seasons; 11 players hospitalized
Centers for Disease Control and Prevention (1962)	High School Wrestling, Iowa	8	NR	*S. aureus*	Skin	Likely source was contaminated wrestling mats	