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# Role of Nasal *Staphylococcus aureus* Carriage in Transmission Among Contact Athletes

Kotaro Suzuki

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## Abstract

This chapter focuses on *Staphylococcus aureus* (SA) infections in athletes. Previous SA infection studies performed starting in the 1980s examined close physical contact athletes, with a focus primarily on injured skin. However, more recent studies of skin SA transmission in athletes were conducted using molecular epidemiology. When participants in sports having a greater duration of competition were examined, results indicated that there was prolonged contact between athletes on the same team and athletes from other teams. These findings demonstrate that effective measures for preventing SA infections are urgently needed. Factors that can affect skin SA infections include high rates of SA nasal colonization, the type of “position on a team,” repeated skin-to-skin contact, and perspiration that occurs during exercise in SA nasal carriers. Thus, it should be possible to utilize molecular typing methods to assess skin-to-skin contact in athletes. This study summarizes the current understanding of SA infections in athletes. In order to develop preventive strategies, it will be necessary to further elucidate the predisposing factors and mechanisms behind SA infections and the subsequent transmission in athletes.

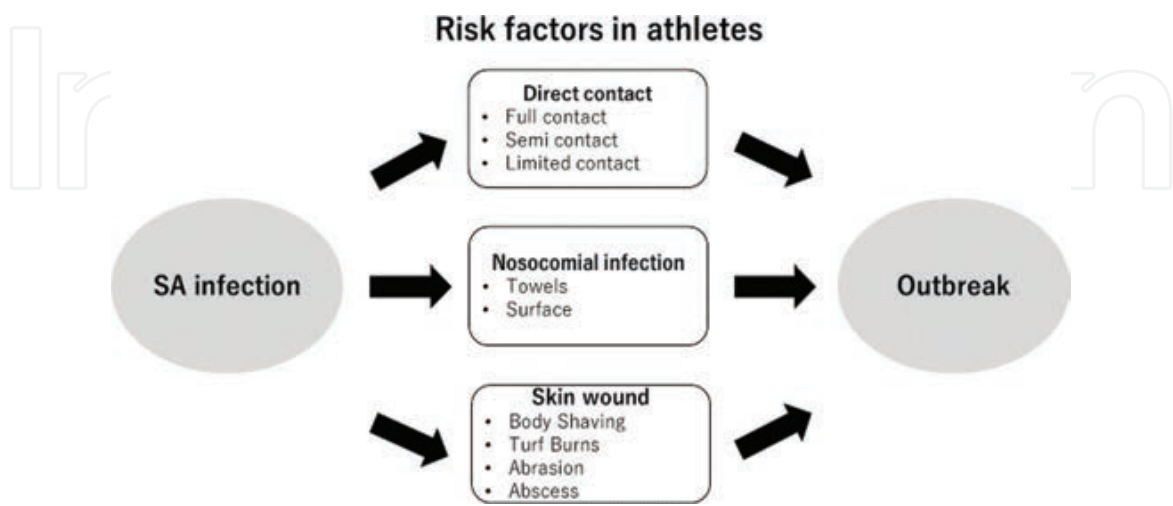
**Keywords:** athletes, transmission, physical contact, genotyping

## 1. Introduction

*Staphylococcus aureus* (SA) infections and its transmission among athletes have long been of interest to sports medicine scientists. SA is very well adapted to colonize the human skin, as the human body provides major ecological niches for the species. Although originally thought to be a nosocomial pathogen, it has become a rapidly emerging, problematic infection in athletes [1]. When outbreaks occur, the infection is spread through repeated skin-to-skin contact, especially due to physical contact between the broken skin of players during games and practices. In addition, sharing contaminated equipment [2], turf burns, and shaving [3]

also contribute to the high incidence of infection among athletes. A review of past studies of SA infections in athletes suggested that the risk factors associated with the outbreaks could be classified into three categories (**Figure 1**). These include direct contact (which refers to “contact sports” where physical contact between players is an acceptable part of the sport), nosocomial infections, and skin wounds. The infections that occur during these outbreaks can also disrupt or potentially eliminate the opportunity for a team to compete at the highest level of their sport. Furthermore, outbreaks of infectious diseases can additionally spread to the player’s social contacts and propagate within their communities [4, 5]. However, the association between direct physical contact and the SA transmission has yet to be fully understood.

The primary goal of the review presented in this chapter is to provide a better understanding of SA infections and the potential relationship with the associated sports activity. This chapter is divided into four parts, with the first section summarizing the latest insights into the sports activity related to SA infections and risk factors. In the second part, we focus on the latest insights into the determinants of SA nasal carriage and skin infections. As nasal carriers may be the reservoir responsible for the transmission in athletes and teams, this section describes the first high-throughput SA nasal carriage effort for large numbers of SA from athletes. As nasal carriers may be the reservoir responsible for the transmission in athletes and teams, this section describes the first high-throughput SA nasal carriage genotyping effort that has been used to examine the SA transmission in athletes. Previous reports have shown that nasal carriage may play a key role in the epidemiology and pathogenesis of SA infections [6, 7]. The third part presents information on our current understanding on how SA can thrive on the skin and be easily transmitted from person to person via sweat. Thus, when a sport involves physical contact, this route is likely to be the major mode of transmission between the athletes. In the final section, we discuss the high-throughput genotyping effort that has been undertaken in order to investigate SA transmission in athletes.



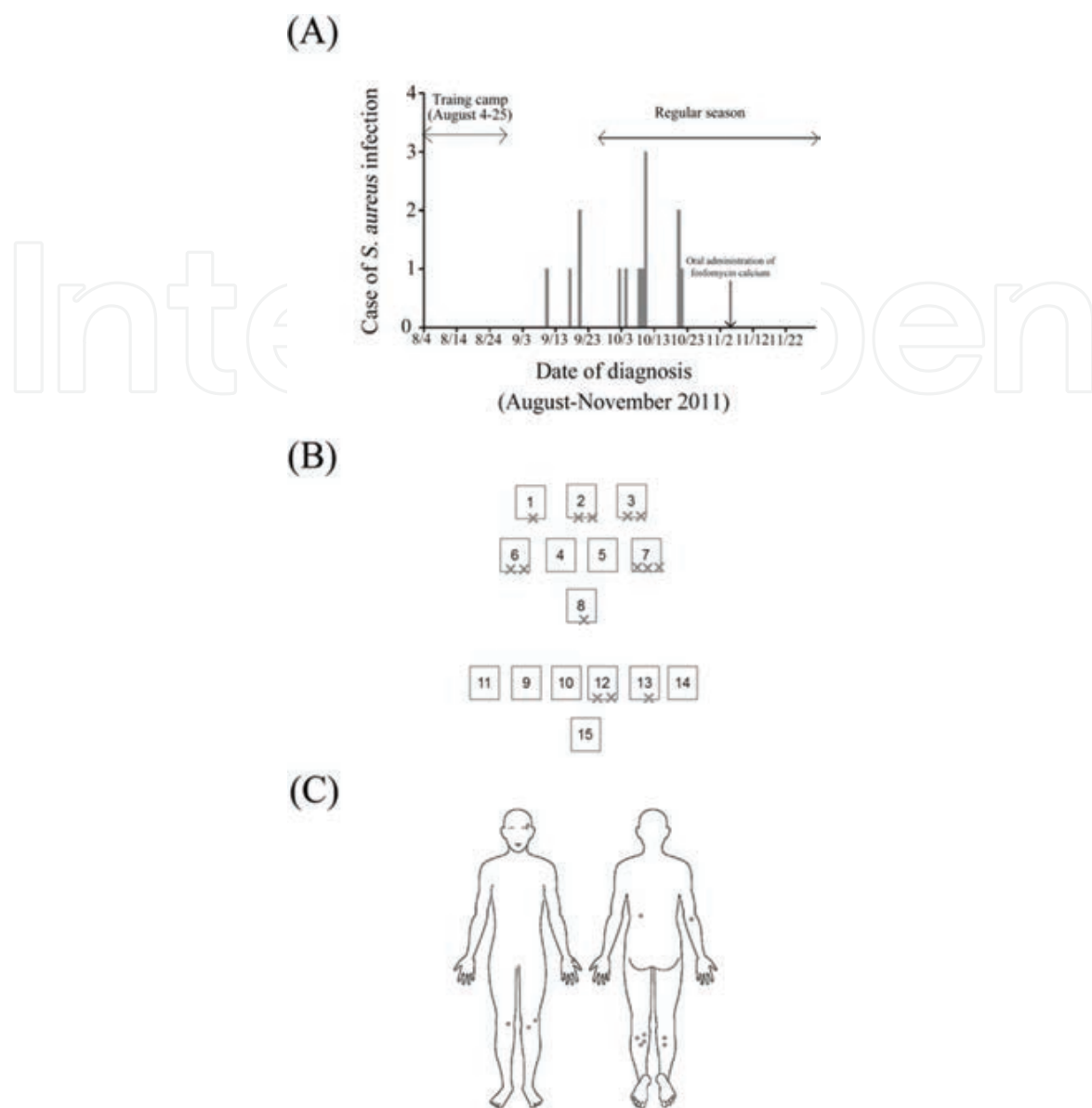
**Figure 1.** A hypothetical example of a *Staphylococcus aureus* infection resulting in an outbreak within an athletic setting. *Staphylococcus aureus* outbreaks are classified into three categories, which include “direct contact,” “nosocomial infection,” and “skin wound.” These hypothetical schemes will need to be further examined in future experiments.

The discovery of SA outbreaks in athletes taking part in physical contact sports is not new. In 1982, Bartlett et al. published the first scientific paper on SA infections in athletes [8]. This study examined 26 players of a high school football team and reported finding a total of 55 lesions, with two players found to have Methicillin-Susceptible *Staphylococcus Aureus* (MSSA), while 24 had methicillin-resistant SA (MRSA). There was no pathogen growth observed for any of the players. There were three essential findings observed and confirmed by the authors in subsequent studies. First, the majority of the lesions observed were located on the extremities and in areas not usually covered by a football uniform or other apparels. Second, 61% of the affected players reported the development of a furuncle at the site of a previously open wound, while 27% reported the development of a furuncle at the site of a previous bruise. Third, cultures obtained from the lesions of two players grew SA that was sensitive to nafcillin, clindamycin, erythromycin, cephalosporin, tetracycline, and sulfa and resistant to penicillin and ampicillin. This is of importance, as infections with drug-resistant bacteria may lead to longer and more costly hospital care, in addition to an increase in the risk of dying from the infection.

Since this initial report, various infectious disease outbreaks have been reported [9]. Sosin et al. [10] additionally reported an outbreak of furuncles in athletes in the state of Kentucky in the USA. The outbreak involved members of the high school football and basketball teams, with a total of 62 lesions reported in these affected athletes. In this school, the basketball season overlapped with the end of the football season, with the two teams sharing a locker room. In addition, six of the players participated on both of the teams. Based on these findings, the authors hypothesized that close physical contact was a risk factor for SA transmission in athletes. The majority of affected players were treated with oral antibiotics, with three players developing infections that did not respond to oral therapy, thereby requiring hospitalization for intravenous antibiotic therapy. One of these hospitalized players subsequently developed a disseminated SA infection and a lung abscess. A total of 81% of the observed lesions were found on the extremities. Moreover, players who sustained a skin injury were three times more likely to develop an infection compared to those who did not report any skin injury. The use of the school showers and locker room and the sharing of clothing and towels were not found to be risk factors for SA infections. Although SA was isolated from 14 of 52 (27%) nasal cultures collected from the athletes, this was not higher than the proportion of SA-positive nasal cultures found in a group of student controls.

These studies provided valuable information and have helped encourage the development of subsequent investigations into additional components affecting SA skin infections. Furthermore, the majority of all of the studies performed because the initial publication has focused on investigations of the effects of physical contact on SA skin infection.

As Suzuki and Tagami [11] discussed in detail, physical contact contributes to SA transmission. Their study identified several factors including an outbreak of SA skin infection in a collegiate men's rugby team. The athletes examined had all started the rugby season with a training camp that was in close proximity to where they lived and which was conducted between August 4 and 25 in 2011 (**Figure 2A**). SA infections were found in 14 (20%) of the 69 healthy rugby players between September 10 and October 21 of 2011 (**Table 1**). One team member required hospitalization during October 2011 in order to treat an abscess that was secondary to the SA infection. As other members of the team also developed skin infections,



**Figure 2.** Epidemic curve of the initial skin infections due to *Staphylococcus aureus* among collegiate rugby players (A). Field position diagram of players who developed *Staphylococcus aureus* infection (B). See **Table 1** for position-specific attack rates. 1 and 3, prop; 2, hooker; 4 and 5, lock; 6 and 7, flanker; 8, number 8; 9, scrum half; 10, fly half; 11 and 14, wings; 12 and 13, center back; and 15, fullback. Players infected with *Staphylococcus aureus* (x). Anatomical locations of infection sites (C). Front and back side of body. Filled circles, skin lesions (unpublished data).

screening was begun on October 6. The dispersion of the outbreak was trimodal, with 28, 50, and 21% occurring between September 10 and 20, between October 2 and 10, and between October 20 and 21 (**Figure 2A**). The infections developed in 11 forwards and three back-positioned players (**Figure 2B**). The infection rate was higher among the forwards versus the back positions (28 vs. 7%) (**Table 1**), with the flanker position exhibiting a greater likelihood of becoming infected compared to the other players. The forehead, back, elbow, and thumb comprised the primary sites of SA infection (**Figure 2B**). Infections tended to occur most frequently on the areas that were not covered by athletic apparel, such as the elbows, forearms, knees, and lower legs [1, 8]. These trends suggest that competitive practices lead to repeated direct contact. Nine crural abscesses were located on the front and back of the legs near or on

Position		No. of infected players (%) <i>n</i> = 14	Total no. on team (%) <i>n</i> = 69	Position-specific attack rate (%)*
Forwards	Props	3 (21)	7 (10)	28
	Hooker	2 (14)	5 (7)	
	Locks	0	10 (14)	
	Flankers	5 (35)	14 (20)	
	Number 8	1 (7)	2 (2.8)	
Backs	Scrum half	0	6 (8.6)	7
	Fly half	0	4 (5.7)	
	Wings	0	5 (7.2)	
	Fullback	0	7 (9.8)	
	Center back	3 (21)	9 (13)	

\*Attack rate = no. of infected players/total no. on team, per position.  
The overall attack rate = 10%.

**Table 1.** Position-specific attack rates of clinical and *Staphylococcus aureus* and soft tissue infections among members of a rugby team (unpublished data).

the knee. In addition, most of the lesions were located on the extremities in areas that are not usually covered by the rugby uniform. However, SA isolates that resulted from the sharing of items, such as the contact bag, tackle bag, and bibs, were limited. Starting on November 3, oral fosfomycin calcium (1 g, two tablets) was administered three times daily for 5 days. After 22 days, there was a decrease in the number of SA nasal carriers. This intervention prevented further dissemination of the SA infection among the team members.

This report shows that the epidemic curves can provide considerable information about the outbreaks such as the pattern of the spread, magnitude, time trend, and exposure and disease incubation periods. The epidemic curve in the present study was trimodal, with continuous, intermittent exposure and gradual increases in the numbers of infections. This type of epidemic curve is typical of person-to-person spread [12]. Classic epidemic curves from propagating outbreaks comprise successively taller peaks, distanced one incubation period apart. The two most common sources of SA spread were contaminated hands and physical contact with athletes [13, 14]. Multimodal peaks are representative of SA outbreaks and comprise a risk factor for such outbreaks during physical contact sports [8]. These previous studies support a person-to-person contact method for the transmittal of the disease, with skin injuries serving as an entrance point for the infectious organisms.

## 2. When do *S. aureus* outbreaks occur in athletes?

Outbreaks of SA infections in an athletic setting have also occurred during the regular season (with the term “regular season” referring to the sport’s league competitive period) [15]. During the regular season, there are likely to be more opportunities for contact with the others that



subsequently result in wounds. Creech et al. examined an American men's football team and found a high rate of SA infection during the regular season [15]. Kazakova et al. also reported an outbreak of MRSA in a men's football team during the regular season [1]. Thus, evaluations of players taking part in physical contact sports during the regular season are critical for prevention and control of SA infections. In addition, individuals with SA-infected abscesses need to be carefully evaluated, as they may also serve as an SA "reservoir" that facilitates transmission to uninfected players. Our current analyses indicated that the regular season is intrinsically associated with risk factors for SA outbreaks (**Figure 2A**). Individuals with SA-infected abscesses might also be SA reservoirs that facilitate transmission to uninfected players.

### 3. The role of fomites in SA infection outbreaks in athletes

Data suggest that athletes with SA skin infection are more likely to experience a recurrence if the fomites were contaminated with SA. For example, an investigation of an outbreak of a MRSA infection in two different football teams revealed that the responsible MRSA clone was not found in the nares of any of the infected players, uninfected teammates/staff, or the environment [1, 3]. In a retrospective study that examined community-onset MRSA skin infections among professional football players, Kazakova et al. [1] did not find any MRSA in the nasal swabs or environmental cultures, even though 42% of the players were nasal carriers of the MSSA strains. Apart from these highly selected populations, it remains questionable whether the results from these studies can be extrapolated to the general population [16]. These findings suggest that the strain responsible for the infection was acquired from a non-nasal endogenous source or environmental sources. Moreover, the MRSA infection observed in these outbreaks was associated with exposures to various contaminated fomites, including whirlpools, shared razors, and shared towels. Other fomites implicated in the outbreaks of sports team-associated MRSA infections include benches, body sites worn by fencers, and even a bar of soap [1, 12, 17]. In non-outbreak settings, it has been reported that close contact with a person who has a skin infection was also associated with the SA infection [11].

Aggressive control of SA strains in the environment has contributed to effective strategies that can be used to prevent SA infection. For example, the National Collegiate Athletic Association (NCAA) has implemented prevention programs [18] that encourage hand hygiene [9], surveys of environmental contamination [12], showering the entire body with an antimicrobial soap and water immediately after each practice and game [19], discouraging cosmetic body shaving [3], and cleaning and disinfecting shared items [18]. As a result of active surveillance (a way of carefully monitoring of SA nasal carriage), consensus has been reached concerning the optimal ways for controlling infection among athletes [15], with nares screening for SA critical for preventing skin and soft tissue infection.

### 4. Nasal carriage and SA infection in athletes

After the reports of the initial studies on SA infection, sports medicine scientists have focused on the SA nasal carriage and skin infection. In human beings, the nose is the main ecological

niche where SA resides [20]. The primary reservoir of SA is thought to be the anterior nares, and 30% of individuals carry nasal SA at any given time [6]. The association between SA nasal carriage and staphylococcal disease was first reported by Danbolt in 1931, who studied furunculosis [21].

Epidemiologic studies found higher SA nasal carriage rates and skin lesions in players of various sports. Sports activities that can cause skin lesions are also correlated with higher SA nasal carriage rates. These include river rafting [22] and football [1]. Decker et al. reported that higher nasal carriage rates were correlated with SA skin infections [22]. They postulated that maceration of the skin caused by prolonged contact with water in conjunction with repeated small cuts or skin injuries might have been the cause of the infections. Begier et al. [3] examined the sports activity of the players for an American football team and found that 97 of 100 players were positive for SA nasal carriage. Supporting these data is a further study that found repeated skin punctures in drug users and diabetics appeared to be the source of higher SA nasal carriage rates [6]. In addition, a retrospective analysis demonstrated that infection rates tended to peak among rugby forwards [23], American football linemen [1, 12, 14], cornerbacks, and wide receivers [3], all of whom have frequent contact with other players. All of these athletes play in the front lines, engage in frequent and aggressive skin-to-skin contact during matches, and are expected to engage the opposing team in the blind side of the scrum and tackle other players. This high-frequency, rough physical contact causes the skin abrasions that are associated with SA soft tissue infections [8].

Persistent SA nasal carriage is an established risk factor for cutaneous infection in physical contact sports [15]. Despite various proposals, there has yet to be a standard definition regarding the number of cultures that need to be taken or what fraction should be positive when determining the carrier status [24]. However, attempts to define persistent carriers have been problematic, as most SA infections originate from lines that are specific to carriers and hands, which are often the primary vectors for transmitting nasal bacteria in athletes [13]. Moreover, there are a number of infectious diseases that can be spread from one person to another by contaminated hands and body sites in athletes [13]. Therefore, the current consensus is that SA resides in the anterior nares of individuals and which serve as reservoirs that predispose players to subsequent infections.

The quantity of the SA colony-forming units (CFU) that can be recovered from swabs used to examine the noses of carriers varies widely, with numbers reported to range from the single digits to millions [25, 26]. In addition, other studies have reported that there is a strong association between high cell counts and persistent carriage [24, 27, 28]. Furthermore, evidence from various studies has led to the postulate that persistent carriers represent a separate group that is distinct from the intermittent and noncarriers [27, 29]. (Most studies that have examined SA nasal carriage have used a cross-sectional design with a single-nasal culture in order to determine whether an individual is a carrier. However, longitudinal studies have distinguished at least three SA nasal carriage patterns in healthy individuals: persistent carriage, intermittent carriage, and non-carriage [6, 30–32]. In addition, some studies make a further distinction between occasional and intermittent carriers [33, 34].) Even though the reasons remain unknown, the basic determinants of persistent and intermittent carriage are thought to be different. Persistent carriers are often colonized by a single strain of SA over



long time periods, whereas intermittent carriers may carry different strains over time [31, 33, 35]. Furthermore, the load of SA is higher in persistent carriers, which results in increased dispersal and a higher risk of infection [24, 29]. Nasal carriers who are also persistent carriers are reported to have higher SA loads and disperse more SA [21, 36, 37].

Approaches that use high-throughput nasal swab data can also be applied to help in our understanding of the bacterial spread. While many studies have focused on nasal swab data, it is still unknown whether nasal SA colonization alone can trigger an SA outbreak. By achieving a deeper understanding of the repercussions of carrying nasal SA, this should help to refine and optimize strategies for risk control among athletes, thereby reducing SA infections.

## 5. Skin surface of *S. aureus* in athletes

The skin is the largest organ of the human body, representing more than 10% of the body mass [38]. In athletes who participate in contact and collision sports, the risk of transmission of SA has been shown to be particularly high [39–41]. It has been hypothesized that “skin-to-skin contact” might be the main cause of SA transmission in athletes, with the physical contact inducing SA dissemination in these athletes [1]. The average area of the skin surface of a human adult is 2 m<sup>2</sup> [42]. Although a dry, salty, low-pH skin surface discourages SA growth [43], the skin of an athlete is usually soaked in sweat, which provides a moist and nourishing environment that is suitable for SA growth. Therefore, skin sweat has been considered to be a key point of transmission during physical contact [44].

Recent evidence suggests that nasal SA has a high propensity to colonize the skin surface [45]. This idea is supported by the finding that colonization often simultaneously disappears from other body sites if an intranasal topical antibiotic is used to temporarily eliminate the SA nasal carriage [46]. Furthermore, cutaneous investigations that examined sweat glands, sebaceous glands, and hair follicles have reported that these areas are likely to be associated with their own unique microbiota [47]. Sebaceous glands secrete lipid-rich sebum, with this hydrophobic coating able to protect and lubricate the hair and skin. In general, sebum serves as an antibacterial coating and acts as a molecular defense mechanism [48]. However, the relationship between exercise-induced sweating and SA transmission in physical contact sports among athletes remains unclear.

It has been reported that the nasal cavity is the primary reservoir for SA and that these carriers are an established risk factor for transmission. Two factors may be involved in the SA transmission in an athletic setting. First, nasal carriers also carry the organism on their hands. Thus, not only are contaminated hands considered to be a likely source for causing the transmission, the hands actually serve in many cases as the primary vectors for transmitting the nasal SA. Second, SA can also live on the skin, which makes it easy to transmit from one person to another via sweat. This route is considered to be the major mode of transmission. The reason for the presence of a higher density of SA on the skin surface is due to the sweat that occurs during exercise in nasal carriers [11].

Even though SA is found on the skin, the nose appears to be the primary reservoir for its replication and transmission to other body sites. This hypothesis is supported by studies that have demonstrated that the use of an intranasal topical antibiotic will temporarily eliminate the transmission of the SA from the nasal carriage to the colonized body site [46]. Pulsed-field gel electrophoresis (PFGE) has also shown that nasal SA isolates are often identical to the strains that later cause clinical infections [49, 50]. Since 10% of the nasal SA carriers exhibited more than one genotype or phage type in their nose, this suggests that many of the infections might be of endogenous origin [32, 51].

Direct physical contact with bodily fluids is believed to be one source of SA dissemination [52]. Examples of direct contact in rugby occur in the scrum, when making tackles, shaking hands, or coming in contact with perspiration and skin lesions. To determine the factors behind SA transmission in physical contact sports, Suzuki and Tagami [11] examined the skin surface SA before and after exercise. The findings of this study showed that the density of the nasal SA was correlated with that of the skin surface SA in nasal carriers with perspiration on the skin surface after exercise, which indicates that perspiring during exercise promotes the appearance of SA in nasal carriers. Eda et al. also provided direct evidence of skin surface SA in healthy adult males after participating in high-intensity endurance exercises [53]. Perspiring during exercise appears to be a key part in the self-infection and transmission of SA in nasal carriers. Thus, the chances of team players transmitting SA to other team members would be increased during practices and while taking part in other exercises.

## 6. Genotyping

Although there is a low risk of the SA infection in team sports, early detection and an awareness of possible pathways of SA transmission could play a huge part in reducing social and economic impacts if an outbreak was to occur in a particular type of sports. Studies that have examined team sports have reported on the importance of early detection in the prevention of the spread of SA [1]. When there is an SA outbreak among a sports team, the first goal should be to identify all of the carriers, which includes both players and the coaching staff. However, it can be difficult to directly obtain such information at the present. Since the SA isolation test is the most reliable and sensitive method that can be used in these identifications, the use of these tests is essential for accurate surveillance of SA outbreaks. However, it should be noted that these tests also isolate many nonspecific SA from the anterior nares of the nose or the wound. Thus, the lack of SA specificity could hinder the surveillance. At present, both mannitol salt agar with egg yolk and Baird-Parker agar media are specifically used for SA isolation. Since these media require a large amount of time for the preparation, this raises the labor costs. In addition, the mannitol salt agar with egg yolk and Baird-Parker media exhibit weak reactivity against other different bacteria, and thus, this test requires appropriate proficiency in the discerning of the colony. Therefore, a reliable method that can be readily adopted by general diagnostic laboratories will need to be developed in order to improve the diagnostic ability of these tests. The examination methodology used is central to the SA surveillance.

Recently, molecular typing methods have greatly improved our understanding of SA transmission, provided powerful tools for tracing the transmission of individual strains and revealed methicillin-resistant SA (MRSA) strains [17]. Since there is a lack of data on the prevalence of SA transmission among athletes, this has prevented effective surveillance, thereby leading to the failure of preventing infections. In the infection control field, our understanding of SA transmission is limited by the methods used to determine the relatedness of microorganisms in the context of time and space. Conventional typing methods, such as phage typing, multi-locus sequence typing (MLST) and pulsed-field gel electrophoresis (PFGE) [54], spa typing [55, 56], and multi-locus enzyme electrophoresis (MLEE) [57], have all been successfully used to describe the global population structure of SA. In addition, this methodology has been used to provide a framework for the description of the major lineages associated with healthcare-associated infections in different countries and to monitor their emergence, dispersal, and decline in different settings [58]. However, when attempting to investigate the finer details of infection outbreaks, these conventional typing methods have serious limitations [59]. Phage-open reading frame typing (POT) has been developed as a genotyping tool based on multiplex polymerase chain reaction (PCR) [60]. These POT methods have been applied to investigate nosocomial MRSA outbreaks, with the discriminatory power of the method shown to be excellent [61, 62]. Although strategies that use molecular genotyping have been able to successfully detect the presence of SA colonization within a few hours, at the present time, they cannot help in predicting the carrier state. In addition, these methods are expensive as compared to that for standard cultures.

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## Author details

Kotaro Suzuki

Address all correspondence to: koutarousuzuki55@gmail.com

Faculty of Health and Sports Sciences, University of Tsukuba, Tsukuba, Ibaraki, Japan

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