Prevalence of Methicillin-Resistant *Staphylococcus aureus* Within Spring Arbor University Fall Athletes

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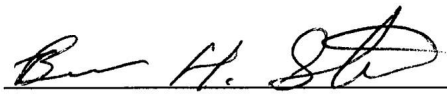

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Acknowledgments

I want to thank Dr. Michael Buratovich for trusting me to continue a research project that has been in progress for several years and for carefully reviewing this manuscript for submission. His guidance as my research and academic advisor has been a valuable resource during my time at Spring Arbor University. I would also like to thank the athletic department for its involvement in this project. Without the coaches, athletes, and athletic trainers being involved, this project would not have been possible. Finally, I would like to thank Mrs. Kathleen Schaeffer for her willingness to communicate with the different athletic teams and ask crucial questions to continue this research.

List of Abbreviations

IRB - Institutional Research Board

MRSA - Methicillin-Resistant *Staphylococcus aureus*

MSSA - Methicillin Susceptible *Staphylococcus aureus*

PBP2a - Penicillin-Binding-Protein 2a

Abstract

Spring Arbor University fall athletes were tested for methicillin-resistant *Staphylococcus aureus* carriage by culturing nasal swabs shortly after their arrival on campus. Should an athlete test positive, they were contacted and recommended to receive treatment provided at the Holton Health Center on the Spring Arbor University campus. The athletes were again tested in the middle and towards the end of their seasons, and the results compared to the first tests. Data comparison of these three testing periods will help us better understand how these bacteria are transmitted between athletes and how preventative measures can be implemented to reduce transmission.

Introduction

History of MRSA

The bacterial genus *Staphylococcus* was first observed in 1880 by Sir Alexander Ogston, a Scottish surgeon, within pus from a knee joint abscess. Upon microscopic analysis of the pus, Ogston described the infecting bacteria as "masses [that] looked like bunches of grapes." Ogston had accurately summarized the distinctive microscopic morphology of this bacterial genus. The name of this genus stems from the Greek *staphyle* (oTcwpvA,f|; a bunch of grapes) and KOKKÓ<; (berry). In 1884, Friedrich Julius Rosenbach, a German physician, differentiated the *Staphylococcus* species by the color of their colonies. *S. aureus* (from the Latin *aurum* for gold) and 5. *albus* (Latin for white). 5. *albus* would eventually be renamed *S. epidermidis* due to its seemingly universal presence on human skin and negative coagulase test.[1,7]

5. *aureus* is a common commensal meaning found asymptomatically on the human body, very commonly on the skin and nose. It is a Gram-positive, coagulase-positive microorganism. This bacterium synthesizes an assortment of virulence factors but can also gain resistance to antibiotics such as aminoglycosides, macrolides, and fluoroquinolones. It is one of the most common causes of infections in humans and can survive in various environmental conditions. 5. *aureus* is prevalent in hospitals and communal-living spaces due to the number of shared surfaces and areas.[8] One virulence factor found in *S. aureus* is protein A. Protein A is a cell wall anchored protein that allows the bacteria to interact with host components and cause higher infection rates.[12] A second virulence factor present in S. *aureus* is Clumping factor A (ClfA).

ClfA is a cell-wall anchored protein that promotes bacterial adhesion to the blood plasma protein fibrinogen via currently unspecified molecular forces.[13]

Methicillin is a semisynthetic 0-lactamase-resistant penicillin and was first introduced in 1959. Before, a drug called benzylpenicillin, a 0-lactam antibiotic, successfully treated *S. aureus* infections. 0-lactam antibiotics target penicillin-binding proteins (PBPs) in the cell membrane. By targeting PBPs, 0-lactam antibiotics inhibit the synthesis of the peptidoglycan that forms the bacterial cell wall. Preventing cell wall synthesis and remodeling causes the bacteria to be vulnerable to molecular pressures and water, causing the cell to die quickly. Peptidoglycan is formed in the cytoplasm, and the steps take place on the cytoplasmic membrane's inner and outer sides. In this biosynthesis pathway, the Park nucleotide, or UDP-N-acetylmuramyl pentapeptide, is flipped to the outside of the membrane by a bactoprenol carrier and flippase enzyme. The Park nucleotide was activated prior to being flipped the outside by the attached UDP. The PBPs will then make the 0-1-4 glycosidic linkage and the transpeptidase linkage that joins peptidoglycan monomers. By the late 1950s, resistant strains began emerging that produced 0-lactamase. This enzyme inactivates 0-lactam antibiotics by hydrolyzing the four-membered beta-lactam ring that binds to the bacterial transpeptidase. Methicillin was produced in response to the concerns associated with these strains and works by having methoxy groups that cause steric hindrance around its amide bond and reduce the affinity for the staphylococcal 0-lactamase (Fig l).[8]

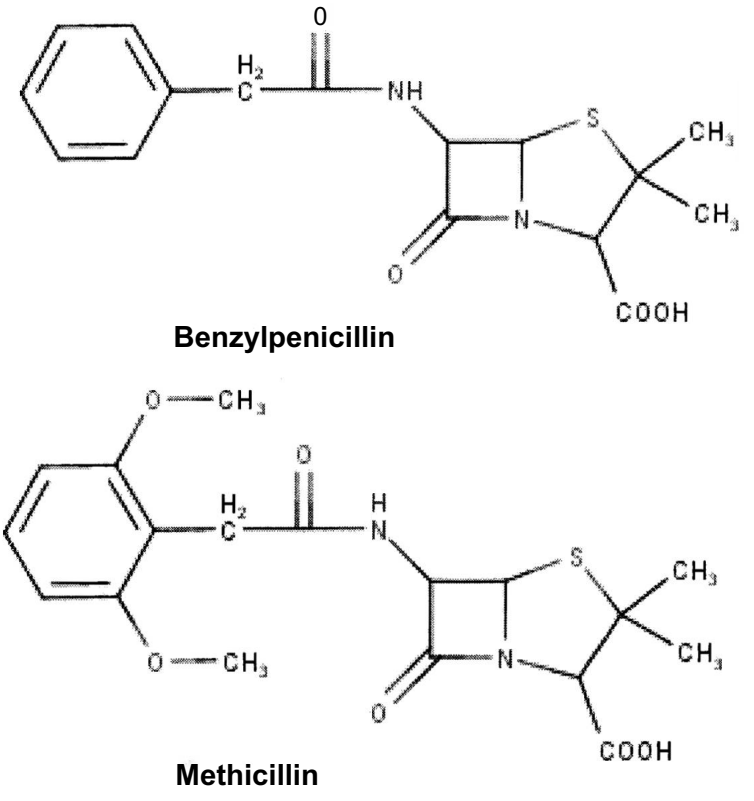

Figure 1. Structures of benzylpenicillin and methicillin.[8]

Methicillin-resistant *S. aureus* (MRSA) was first recognized in 1960. This discovery occurred only one year after methicillin was first utilized to treat S. *aureus* infections. The first outbreak of MRSA in the United States occurred in 1968 at a Boston hospital and has occurred frequently in individuals who have been in health care settings or have invasive medical devices, such as insulin pumps. Infections are also commonly found in athletes involved in sports where they frequently encounter another individual's skin or surfaces that have been contaminated.

While the name MRSA is still used to describe the *S. aureus* strains resistant to all penicillin antibiotics, methicillin has been largely replaced in clinical settings. The drug was found to cause interstitial nephritis and has been replaced by cloxacillin, dicloxacillin, and flucloxacillin.

Resistance

MecA Gene

The presence of the *mecA* gene is required for S. *aureus* to develop and display methicillin resistance. The structural component, *mecA,* encodes the penicillin-binding protein 2a (PBP2a) and creates resistance to methicillin and other semisynthetic penicillinase-resistant beta-lactams. The gene also consists of two regulatory components in some cases, the *mecRl-mecI* and the beta-lactamase genes *(blal, blaRI, and blaZ). mecRl-mecI* is a negative regulator of *mecA* transcription, and mutations can result in more resistant strains. The beta-lactamase genes can also downregulate *mecA* transcription due to sequence similarity (Fig 2). These genes produce resistance by hydrolyzing the beta-lactam ring.[9]

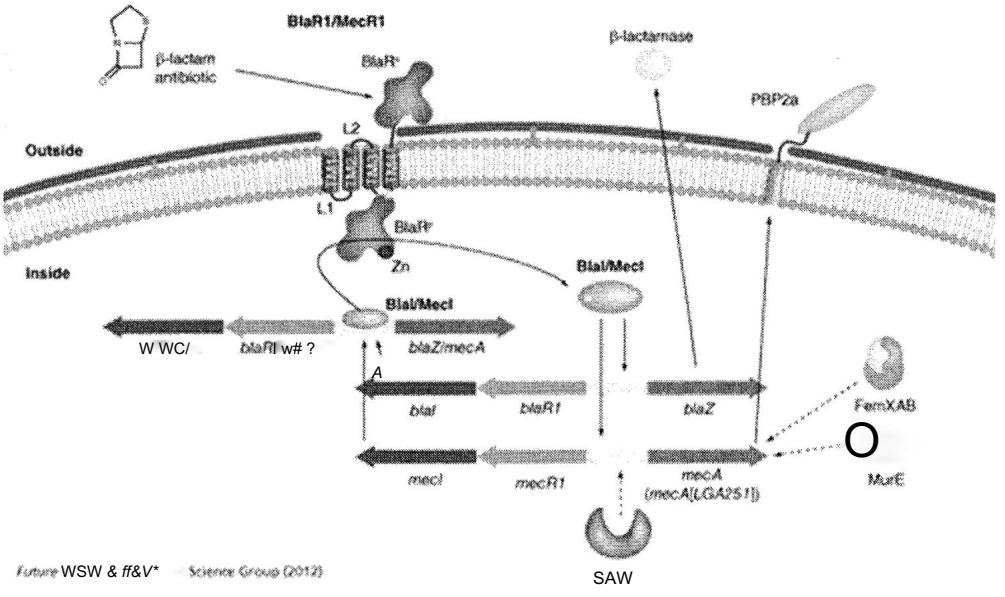

Figure 2. Operon containing mecA.[14]

Penicillin-Binding Protein 2a

Encoded by *mecA,* PBP2a is an inducible protein that establishes resistance to the semisynthetic penicillinase-resistant beta-lactams: methicillin, nafcillin, oxacillin, and all

cephalosporins. PBP2a has a lower affinity for beta-lactam antibiotics than other PBPs. In strains sensitive to methicillin, the antibiotic covalently binds to PBPs 1-3 and inactivates enzyme activity, preventing transpeptidation, and ultimately causing cell death. However, PBP2a can compensate for the other proteins' inactivity and allow for the completed synthesis of peptidoglycan (Fig 3).[9]

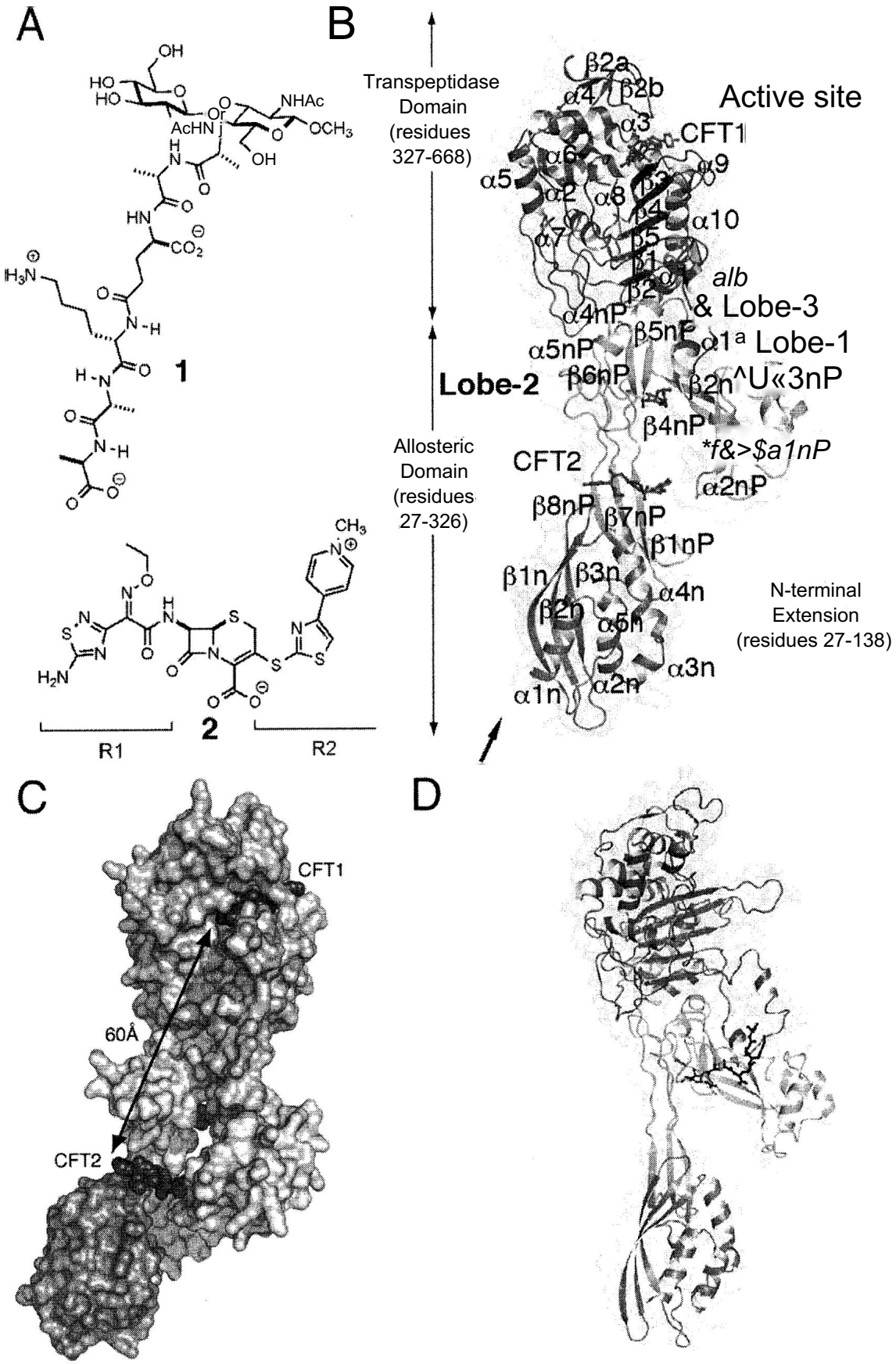
A
B
C
D
1
2
R1
R2
Transpeptidase Domain (residues 327-668)
Allosteric Domain (residues 27-326)
Active site
CFT1
CFT2
Lobe-2
alb & Lobe-3
Lobe-1
N-terminal Extension (residues 27-138)
60Å

Figure 3. Domains of PBP2a and key ligands. (X) The chemical structures of a synthetic NAG-NAM(pentapeptide) (1) and ceftaroline (2). The R1 and R2 groups of 2 are labeled. (B) Ribbon representation of PBP2a acylated by ceftaroline. The N-terminal extension is colored in green, the remaining allosteric domain is colored in gold, and the transpeptidase (TP) domain is colored in blue. These domain colors are retained in all other figures. Two molecules of ceftaroline (capped sticks in red) are found in complex with protein: one covalently bound as an acyl-enzyme in the TP domain (CFT1) and one intact at the allosteric domain (CFT2). A muramic acid saccharide (capped sticks in magenta) is found at the center of the allosteric domain. The arrow indicates the point of attachment of the membrane anchor. (C) The solvent-accessible surface representation for PBP2a is shown. The distance between the two ceftaroline molecules is 60 Å. (D) Ribbon representation of PBP2a in complex with 1 (black sticks). This view is rotated ~45° on the y axis compared with the view of *C.* Taken from https://www.pnas.org/doi/10.1073/pnas.1300118110.[31]

MRSA in Athletic Populations

MRSA is the leading cause of infectious diseases that are spread amongst athletes. Outbreaks of MRSA are more likely to occur during the competitive portion of the season due to the increased opportunity of contact between players and the consequential spread of infection. A 2003 study of the St. Louis Rams football team showed that eight MRSA infections occurred among 5 of the 58 players (9%) during their season. All infections developed at turf abrasion sites. During nasal and environmental swabbing, no cases of MRSA were observed; however, 35 of the 84 (42%) nasal swabs of staff and players showed MSSA colonization. MSSA was also recovered from hot tubs and taping gels.[15]

Another study that sheds light on the issue of MRSA within athletic populations was conducted in Taiwan, the first of its kind in the country in 2017. Of 259 students, 120 non-athletes, and 139 athletes, 54 cases of MSSA (21%) and 4 cases of MRSA were reported. Surprisingly, this study showed a higher carriage rate in non-contact sports than in contact sports. MRSA colonization rates were not high enough to determine risk factors within this population.[17]

One study examined American collegiate athletes at Vanderbilt University in Tennessee. The study swabbed 100 football players, 98% of the team, over a year during eight sampling periods. Nasal colonization rates ranged from as low as 4% during summer off-season to as high as 19% at the end of their regular season. It was found that MRSA colonization rates were higher during the regular season than in spring training (16.5% vs. 8.4%), off-season (16.5% vs. 4.4%), and post-season (16.5% vs. 7.7%). During the study, 37% of athletes had at least one positive nasal culture. This study also analyzed the women's lacrosse team and sampled all 26 players. This team was not on campus during the summer, so only 6 sample periods were used for these athletes. MRSA nasal colonization rates ranged from 11% to 23%, with relative peaks during the spring season (15.4%) and fall season (23.1%).[18]

It has been recognized that skin infections are more likely to reoccur if the fomites, objects, or materials likely to carry infection are contaminated with 5. *aureus.* Athletes can exhibit repeated skin-to-skin contact, sharing spaces, and a lack of hygiene, such as a lack of washing hands or taking a shower after training or post-race and sharing tools and toilets. While it is common that the microorganism is commonly spread through droplets transported from the area, direct contact with the nasal secretions or fomites plays a key role in the spread of infection.[15]

Athletic training rooms are commonly scrutinized once an outbreak occurs. In a study examining NCAA Division I university therapeutic whirlpool facilities, S. *aureus* was identified in 22% (52/240) of the samples and MRSA in 0.8% (2/240). *S. aureus* appeared in and around more whirlpools when multiple athletes used the whirlpool. However, bacteria was present regardless of whether multiple athletes used a whirlpool or no athletes used it. Therapeutic equipment such as this need to have strict sanitation procedures that are strictly followed in order to reduce the risk of infections and transmission among athletic populations within universities and other athletic teams.[16]

Identification

Mannitol Salt Agar

Using mannitol salt agar to detect *staphylococci* has been standard practice since 1945. The agar contains peptones and beef extract to supply nutrients essential for bacterial growth. The agar has a sodium chloride concentration of 7.5%, resulting in partial or complete growth inhibition of bacteria other than *staphylococci.* Mannitol is added to the agar as a fermentable carbohydrate and will produce acid upon its fermentation by bacteria, such as MRSA. The phenol red indicator in the agar will turn yellow in the presence of coagulase-positive *Staphylococci* and remain red in the presence of coagulase-negative *Staphylococci.* Inducers can also be added to the agar to induce growth-promoting properties of the bacteria, such as cefoxitin to induce the *mecA* gene in MRSA.[10]

Coagulase Test

The coagulase test is used to differentiate between different *Staphylococci spp.* This test identifies the presence of bound coagulase or clumping factor on the cell wall of bacteria such as MRSA. The coagulase on a positive bacteria will react with the fibrinogen in a plasma serum

mixture and cause the fibrinogen to aggregate and form clumps. Coagulase-negative bacteria will not cause fibrinogen aggregation.

Gram-staining

Gram staining allows for the differentiation of two varieties of bacteria based on the composition of their cell walls. This test involves three stages: staining with a water-soluble dye called crystal violet, decolorization with acid-alcohol, and counterstaining with safranin. Gram-positive bacteria, such as MRSA, will stain a violet color due to a thick layer of peptidoglycan in their cell wall. This component will retain the crystal violet dye during the decolorization process. However, Gram negative-bacteria will not retain the crystal violet due to their thinner peptidoglycan wall and stain a pink/red color.[11]

Materials and Methods

Ethical Approval

Approval was given by the SAU Institutional Research Board (IRB) before sampling began.

Participant Consent

Each participant had to read through and sign an informed consent form before any samples were taken. A copy of this waiver is included on the last page of this document.

Nasal Swabbing and Sample Collection

Samples were taken from each athlete using a different sterile cotton nasal swab in each nostril. The inside of each nostril was swabbed in a circular motion for 10 seconds. The swab for each nostril was then streaked in a zig-zag pattern onto a BBL CHROMagarTM MRSA II plate.

These plates contain a rich trypticase soy-base with two percent agar, cefoxitin (5.2 mg/L), salt, and a chromogen mixture. Each sample was incubated at 37°C for a minimum of 48 hours before visual analysis was conducted while looking for light pink/red colonies. If no colonies of this color were present, the plate was returned to the incubator to allow another 24 hours for possible growth. Further testing was conducted on plates possessing light pink/red colonies.

Mannitol/Oxacillin Plates

The original colonies, which had a light pink/red color, were then transferred to Thermo Scientific™ Remel™ Mannitol Salt Agar w/Oxacillin (4pg/mL) plates with a sterile inoculating loop to confirm the bacteria's identity further (Fig 4). The streaked mannitol plates were placed into the 37°C incubator for 48 hours and then visually analyzed. The plates were set aside for further analysis if the bacteria had fermented the mannitol. If the bacteria had not fermented mannitol at this point, the plates were placed back into the incubator for an additional 24 hours and then reexamined. The plates were then discarded if no fermentation had occurred.

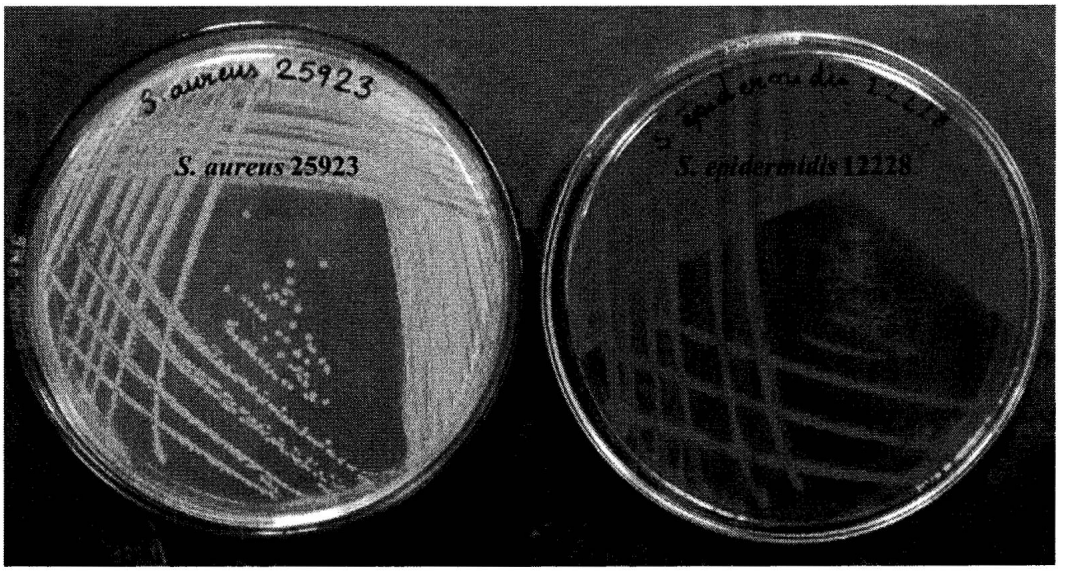

Figure 4. Image showing the positive result (change to yellow) of *S. aureus* on mannitol plate (left) and negative result of S. *epidermidis* with no color change (right).[4]

Gram-staining

Gram stains of each bacterial isolate used a sterile inoculating loop containing the bacteria. The bacterial cells were smeared into a drop of water on top of a glass slide. The slide was allowed to air dry, and the sample was heat-fixed to the slide by passing it over a flame. Crystal violet dye was then flooded onto the slide and allowed to sit for 60 seconds. After the allotted time, the slide was gently rinsed with DI water until all excess crystal violet was removed. Grams iodine was flooded onto the slide and allowed to sit for 60 seconds. The slide was then rinsed to remove excess stain. Acid alcohol, a decolorizing agent, was dripped across the slide for roughly 15 seconds until it stopped leaching the stain. The slide was then flooded with safranin, a counterstain, for 60 seconds and then rinsed off. Lens paper was then used to gently blot off any excess liquid, and the slide was viewed under a microscope using oil immersion. If the bacteria were Gram-positive (purple) and round (Fig 5), they matched the characteristics of 5. *aureus* and were kept for further analysis.

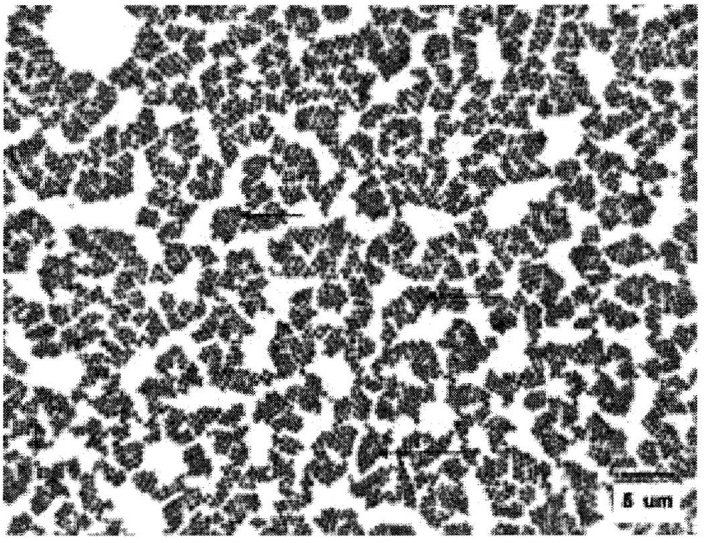

Figure 5. Image showing standard Gram stain result of S. aureus.[5]

Coagulase Test

The bacterial sample was removed from a growth plate using a sterile wooden stick and mixed into a drop of physiological saline on a testing card. A drop of plasma was added to one of the bacterial mixtures, and the other was left as a negative control to differentiate any granular appearance of the organism from coagulase clumping (Fig 6). A positive result, clumping of organisms, would be visible after 10-30 seconds, and the sample was kept for further analysis. If no clumping was observed, the sample was discarded.

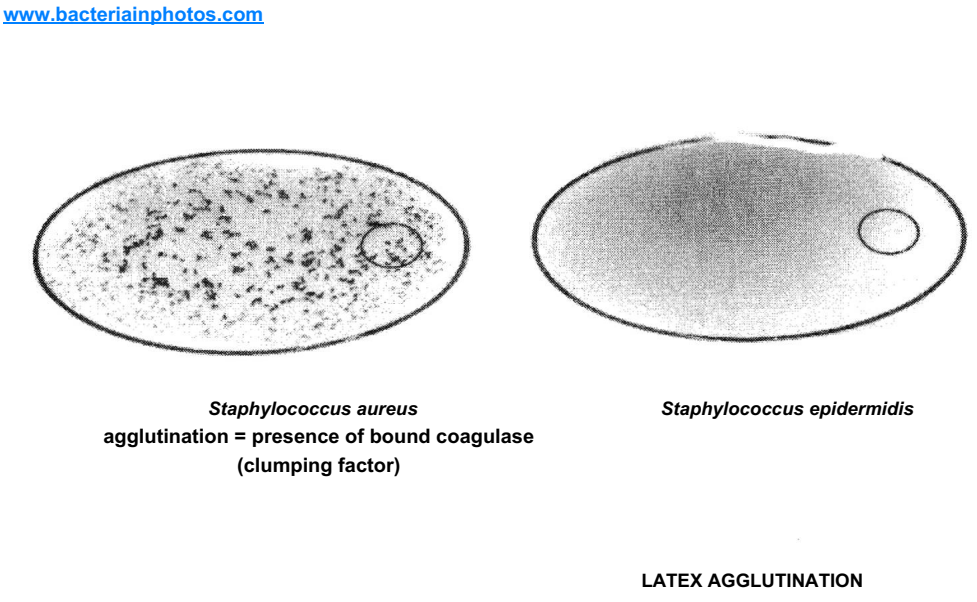

Figure 6. Image comparing positive coagulase test of *S. aureus* to negative result from *S. epidermidis.* [6]

Results

The study consisted of 120 athletes, and of those tested, there were seven positive results by the end of the study. The men's and women's golf teams were only involved in the first two testing rounds because their season was shorter than the other sports but had one athlete test positive. Both tennis teams were only involved in the first two rounds due to a shorter season than the remaining sports, but they yielded no positive results during this time. The men's soccer team participated in all three rounds of swabbing and had 0 positive results during the preseason, two after the midseason round, and four after the end of the season round. The volleyball team also participated in all three rounds and had 0 positive results during the preseason round, one after the midseason round, and one after the end-of-season round; however, the volleyball athlete who tested positive during the end-of-season round was not the same athlete who tested positive during the midseason round. Men's and women's cross-country teams participated in all three rounds but yielded 0 positive results during the study (Table 1).

Sport	1st Swab (# of positives)	2nd Swab (# of positives)	3rd Swab (# of positives)
Men's Soccer (n=31)	0	2	4
Women's Tennis (n=14)	0	0	N/A
Men's Tennis (n=13)	0	0	N/A
Volleyball (n=14)	0	1	1
Men's Cross-Country (n=ll)	0	0	0
Women's Cross Country (n=13)	0	0	0
Golf (Men and Women; n = 24)	1	N/A	N/A

Table 1. Positive results after each swabbing period

Discussion

The Problem of MRSA

Since its discovery, MRSA has become a scourge in health care and community settings. The prevalence of methicillin resistance among *S. aureus* isolates in intensive care units in the United States is 60 percent,[19] and more than 90,000 invasive infections due to MRSA occurred in the United States in 2005.[20]

MRSA outbreaks also occur in collegiate athletes. One study of 277 college student-athletes in East Tennessee older than 18 years showed a prevalence of CA-MRSA nasal carriage of 1.8%, similar to what has been reported in the general population (1.5%).[21] A 2017 study of college athletes in Taiwan also showed similar carriage frequencies and failed to demonstrate differences between athletes and non-athletes.[22] However, a 2014 study by Anna Champion and her colleagues showed a higher rate of MRSA nasal carriage in college athletes. It demonstrated transient colonization of various skin sites throughout the season. Such transient colonization of athletes can potentially lead to a long-term MRSA carriage.[23] Also, MRSA colonization of athletes can lead to MRSA contamination of residence halls.[24] MRSA colonization of college athletes can also cause an outbreak of staphylococcal skin infections.[25,26] Some MRSA outbreaks can even cause high morbidity infections,[27-28] and there is also one case of an athlete dying from an MRSA infection.[29] Therefore, preventing MRSA spread in SAU athletes is integral to the university's commitment to our student-athletes.

Determining Possible Transmission Sites

The end-of-season nasal swabs showed that the number of athletes who carried MRSA in their nasal cavity had increased since the preseason round of swabbing. Therefore, the athletes were contracting MRSA during their athletic seasons. To reduce the danger this poses to all student-athletes, we first had to consider the objects and environments where the transmission was most likely occurring. These places and objects included the athletic training room, the two fitness centers, locker rooms, and various athletic equipment. Contact was made with the coaches and other athletic staff to determine the current sanitation procedures, if any, that are followed for each area and set of equipment. It was clear that adjustments could be made to create a safer and sanitary environment for the athletes to train.

The athletic training room has a strict sanitation protocol that is followed and became stricter during the COVID-19 pandemic. However, during intervals when the staff experience high volumes of athletes visiting them for before and after practice treatment, the sanitization standards become less of a priority. Examples include benches/beds not being sanitized after an athlete has occupied it for a significant period before another athlete is placed at that station. There is direct skin-to-surface contact depending on the treatment being performed. These treatment protocols create a significant risk of transmission if the surface is not sanitized, especially if the athlete has a break in their skin or an open wound.

The two fitness centers include one that is open to the public and another for SAU athletes only. The one open to the public is watched by a student employee in charge of filling the sanitation wipe stations and other cleaning and management tasks, depending on the time of day they are working. There are three stations for wipes spread across the room, but it is common to find only one of them containing wipes. Wiping down exercise equipment is the only

method to sanitize exercise equipment in the fitness center. If no wipes are available, the surface will not be sanitized until the janitorial staff comes in for their daily cleaning of the area. The second fitness center was transitioned to an athlete-only facility after completing the new center in 2019. Once this transition occurred, there were no student workers in charge of maintaining this facility, and the sanitizing was left to the discretion of the athletes who used the equipment.

We also inquired about the sanitation of equipment used by various athletic teams. This equipment included the items used by the track and field teams during their practices and events, balls utilized by the volleyball and tennis teams, the hardwood court where the volleyball team plays, and the buses that bring athletes to their events. It was found that the track and field equipment had no current sanitation protocol that was being used. The balls and courts used by the volleyball team are cleaned frequently and have a low risk for transmission. The buses are sanitized after each team is finished with them and are cleaned before the next team uses them.

Plan to Reduce Transmission

The fitness centers seem to be utilized by all the athletic teams with the most inconsistent sanitation practices. This disparity can be improved by having the student workers be required to do hourly cleaning rounds of the equipment that is not currently being utilized and simply wiping the surfaces down with the currently implemented alcohol wipes. Also, it would be beneficial to produce an informational document posted throughout the room with standard fitness center cleaning procedures that all should follow.

Another area that needs to be always sanitary that is utilized by all athletic teams is the athletic training room. While the trainers have strict protocols that they follow, they sometimes cannot keep up with sanitation procedures during high traffic hours of the day. Athletes should be provided with sanitation items, such as alcohol wipes, at the door to improve the sanitation of

this room. Athletes should sanitize the station they will be utilizing before contacting these surfaces.

LaBelle and others reported that distributing MRSA educational materials and making PURELL hand sanitizers readily available decreased MRSA incidence by almost 95 percent.[30] This inexpensive and simple mitigation technique should become a routine part of athletic life at SAU. In this report, researchers handed out educational brochures, hung posters, and posted hand sanitizers throughout the athletic facility. Because student-athletes were constantly reminded of the perils of MRSA infection and the efficacy of hand hygiene, they constantly used hand sanitizers. These strategies effectively dropped MRSA carriage and infection rates.

Future Directions

This research could be continued by testing the athletes' equipment during their practices and events. The surfaces within the fitness centers and the training room could also be swabbed periodically throughout the day to assess how well the surfaces are being maintained by the staff and those utilizing the area.

The study could also be improved by testing both fall and spring athletes upon their arrival on campus in the fall and throughout the year. The fall athletes would be tested upon arrival for their preseason training throughout August and then for the fall and spring semesters. The spring athletes would first be tested upon their arrival for the beginning of courses in the fall, then tested throughout the fall, and then throughout their season in the spring. This testing regimen would give a baseline for all athletes on campus and allow for a better understanding of

how the virus is being spread due to observing athletes who are not currently in season and mostly using shared facilities.

Finally, SAU should employ some of the same techniques successfully used by LaBelle and others to reduce MRSA rates in high and collegiate athletes.

Conclusion

This experiment aimed to determine if athletes were coming to the university with MRSA or if they were being infected with it during their activities on campus. After the initial round of swabbing during the athlete's arrival on campus, only one athlete tested positive. However, after the midseason and post-season rounds of swabbing, there were 3 and 6 positive cases, respectfully. Therefore, most fall athletes are not reporting to campus with MRSA but are being infected with it throughout the season and during their activities on campus.

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Appendix I: Informed Consent Forms

Informed Consent

Below are the guidelines for writing an informed consent, as taken from the Code of Federal Regulations. The second page of this document is intended as a template for writing an informed consent specific to any study involving human subjects and being presented to the SAU IRB for review.

Basic Elements of Informed Consent (from 45 CFR 46.116)

In seeking informed consent, the following information shall be provided to each subject:

1. A statement that the study involves research, an explanation of the purposes of the research and the expected duration of the subject's participation, a description of the procedures to be followed, and identification of any procedures which are experimental.
2. A description of any reasonably foreseeable risks or discomforts to the subject. (NOTE: This includes any information about procedures that might make a subject hesitant to participate.)
3. A description of any benefits to the subject or to others which may reasonably be expected from the research.
4. A disclosure of appropriate alternative procedures or courses of treatment, **if any (if not, eliminate this section on the form),** that might be advantageous to the subject.
5. A statement describing the extent, if any, to which confidentiality of records identifying the subject will be maintained.
6. For research involving more than minimal risk, **if any (if not, eliminate this section on the form),** an explanation as to whether any compensation and an explanation as to whether any medical treatments are available if injury occurs and, if so, what they consist of, or where further information may be obtained.
7. An explanation of whom to contact for answers to pertinent questions about the research and research subjects' rights, and whom to contact in the event of a research-related injury to the subject.
8. A statement that participation is voluntary, that refusal to participate will involve no penalty or loss of benefits to which the subject is otherwise entitled, and that the subject may discontinue participation at any time without penalty or loss of benefits to which the subject is entitled.

Informed consent document should be submitted as an attachment with the application by the principle investigator.

If infants or very young children are involved in a study an informed consent with the child's name must be secured from at least one parent or legal guardian. If the child is of cognitive age an "informed assent" must be secured from the child, along with both the child's and parent's permission signature. Assent is basically the same as consent yet involves minor children who

are not authorized to give legally valid informed consent because of their age. Assent is written in child-friendly language and describes the research participation, risks, benefits, and other elements of consent.

Spring Arbor University
Informed Consent

Title of Study: MSRA Carriage in SAU Athletes

Principle Investigator: Michael Buratovich PhD

Co-Investigator(s):

1. Purpose of the Research:
   To determine if colonization of SAU athletes occurs during the season and if this colonization is long-term.

2. Risks or Discomforts:
   The nasal swab may cause some short-term discomfort, but there are no long-term consequences of such a swab.

3. Benefits to the Research Participants or Others:
   We will determine if SAU athletes have a tendency to become colonized by MRSA during their season.

4. Possible Alternative and Advantageous Procedures or Courses of Treatment: (only relevant for Full Review application)

5. Confidentiality Maintained:
   All samples will be labeled with a code. No names will be used.

6. Greater than Minimal Risk: (only relevant for Full Review application)

7. Contact Person:
   Michael Buratovich - michaelb@arbor.edu; 517-750-6383

8. Voluntary Participation: No student-athlete who does no wish to participate in this study will be compelled to do so. Also athletes may withdraw from the study at any time.

Printed Name of Participant:

Signature of Participant:

Date: