

Dear Editor:

We read with interest the article by Oller et al<sup>1</sup> on *Staphylococcus aureus* recovery from the environment and human locations in 2 collegiate athletic teams, and we welcome the publication of articles emphasizing the importance of proper hygiene in sport. Although we agree with the key points of the article, we suggest that the data warrant further interpretation.

The authors found that the rate of colonization of *Staphylococcus* was highest among wrestlers (n = 16/25, 64%). Table 1 ("Sample Human Locations That Tested Positive for *Staphylococcus aureus*") states that 15 of these 16 staphylococci had a negative coagulase test result, suggesting that the isolates were not *S aureus* but coagulase-negative staphylococci, which were unlikely to be significant isolates in this group. If this is the case, the true rate of *S aureus* colonization in the wrestlers was 4%, a surprisingly low rate but similar to that of the control group. It should be noted that the prevalence of *S aureus* in the control group was well below carriage rates reported previously.<sup>2,3</sup> Only one coagulase result was presented for the non-methicillin-resistant staphylococci isolated from the football players, and this, too, was coagulase negative (sample number 801105). If this was the case for other isolates in this group, the rate of *S aureus* colonization among football players was also much lower.

Seven football players (of 70 tested) and 1 wrestler (of 25 tested) were reported to harbor methicillin-resistant *S aureus* (MRSA). Two strains, differentiated on the basis of antibiotic susceptibilities, were identified among the footballers by the authors. However, the susceptibility profiles of the MRSA isolates presented in Table 2 suggest that 6 MRSA strains were isolated among the footballers, with an additional strain isolated in the wrestler. The only similar MRSA strains among the football players were the isolates from sample numbers 801137 and 801167, suggesting a possible multiclonal MRSA problem, with very little evidence of MRSA cross-transmission between players. Without adequate typing data, such as pulsed field gel electrophoresis, for methicillin-susceptible and methicillin-resistant isolates from both the environment and from humans, no further inferences can be made about transmission in this study.

In the absence of evidence of transmission, the relevance of the survey addressing sharing of personal items is questionable. Furthermore, the choice of controls for the survey is also debatable. The authors found that footballers were more likely to share water bottles and that wrestlers were more likely to share towels, soap, and deodorants than controls. However, the control group consisted of on-campus nonathletes. A significant difference was likely in the sharing of these items, regardless of whether the athletes were colonized or infected with *S aureus* or not. An association between sharing of items and *S aureus*

transmission cannot be proven or implied. It might have been more appropriate to study footballers and wrestlers who were not colonized or infected as controls for those that were. Without adequate controls and confirmed transmission, the survey findings are of limited use.

Finally, there are concerns over the methods used to determine vancomycin resistance because one MRSA isolate (sample number 801163) and one coagulase-negative staphylococcus (sample number 801183) were documented as vancomycin resistant. Vancomycin-resistant *S aureus* (VRSA) and, to a lesser extent, vancomycin-resistant coagulase-negative staphylococci are extremely rare.<sup>4,5</sup> The isolation of a true VRSA, in particular, poses significant infection control and public health problems; guidance on investigation and control is offered by the Centers for Disease Control and Prevention (CDC).<sup>6</sup> It appears that disk diffusion was used to determine vancomycin resistance in staphylococci in this study, which alone is not sufficient to confirm vancomycin resistance. We suggest that the CDC's algorithm for testing *S aureus* for vancomycin resistance should be followed to confirm that sample number 801163 is a true VRSA.<sup>7</sup>

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## REFERENCES

1. Oller AR, Province L, Curless B. *Staphylococcus aureus* recovery from environmental and human locations in 2 collegiate athletic teams. *J Athl Train*. 2010;45(3):222–229.
2. Kluytmans J, van Belkum A, Verbrugh H. Nasal carriage of *Staphylococcus aureus*: epidemiology, underlying mechanisms, and associated risks. *Clin Microbiol Rev*. 1997;10(3):505–520.
3. Kuehnert MJ, Kruszon-Moran D, Hill HA, et al. Prevalence of *Staphylococcus aureus* nasal colonization in the United States, 2001–2002. *J Infect Dis*. 2006;193(2):172–179.
4. Sievert DM, Rudrik JT, Patel JB, McDonald LC, Wilkins MJ, Hageman JC. Vancomycin-resistant *Staphylococcus aureus* in the United States, 2002–2006. *Clin Infect Dis*. 2008;46(5):668–674.
5. Srinivasan A, Dick JD, Perl TM. Vancomycin resistance in staphylococci. *Clin Microbiol Rev*. 2002;15(3):430–438.
6. Hageman JC, Patel JB, Carey RC, Tenover FC, McDonald LC. Investigation and control of vancomycin-intermediate and -resistant *Staphylococcus aureus*: a guide for health departments and infection control personnel. [http://www.cdc.gov/ncidod/dhqp/pdf/ar/visa\\_vrsa\\_guide.pdf](http://www.cdc.gov/ncidod/dhqp/pdf/ar/visa_vrsa_guide.pdf). Accessed December 13, 2010.
7. Centers for Disease Control and Prevention. Vancomycin-intermediate/resistant *Staphylococcus aureus* laboratory testing algorithm. [http://www.cdc.gov/HAI/settings/lab/visa\\_vrsa\\_algorithm.html](http://www.cdc.gov/HAI/settings/lab/visa_vrsa_algorithm.html). Accessed December 13, 2010.