REFERENCES

1. Griffith CJ, Cooper RA, Gilmore J, Davies C, Lewis M. An evaluation of hospital cleaning regimes and standards. *J Hosp Infect* 2000;45:19–28.
2. Wilson F, Wells A. Evaluating environmental cleanliness in hospital and other healthcare settings. *Healthc Infect* 2012;17:70.
3. Boyce JM, Havill NL, Dumigan DG, Golebiewski M, Balogun O, Rizvani R. Monitoring the effectiveness of hospital cleaning practices by use of an adenosine triphosphate bioluminescence assay. *Infect Control Hosp Epidemiol* 2009;30:678–684.
4. Carling PC, Parry MM, Rupp ME, Po JL, Dick B, Von Beheren S; Healthcare Environmental Hygiene Study Group. Improving the environment surrounding patients in 36 acute care hospitals. *Infect Control Hosp Epidemiol* 2008;29:1035–1041.
5. Boyce JM, Havill NL, Havill HL, Mangione E, Dumigan DG, Moore BA. Comparison of fluorescent marker systems with 2 quantitative methods of assessing terminal cleaning practices. *Infect Control Hosp Epidemiol* 2011;32:1187–1193.
6. Lewis T, Griffith CJ, Gallo M, Weinbren M. A modified ATP benchmark for evaluating the cleaning of some hospital environmental surfaces. *J Hosp Infect* 2008;69:156–163.
7. Mulvey D, Redding P, Robertson C, et al. Finding a benchmark for monitoring hospital cleanliness. *J Hosp Infect* 2011;77:25–30.
8. Whiteley GS, Derry C, Glasbey T. The comparative performance of three brands of portable ATP-bioluminometer intended for use in hospital infection control. *Healthc Infect* 2012;17:91–97.
9. Aiken ZA, Wilson M, Pratten J. Evaluation of ATP bioluminescence assays for potential use in a hospital setting. *Infect Control Hosp Epidemiol* 2011:32;507–509.
10. Shama G, Malik DJ. The uses and abuses of rapid bioluminescence-based ATP assays. *Int J Hyg Environ Health* 2013;216:115–125.
11. Carrick K, Barney M, Navarro A, Ryder D. The comparison of four bioluminometers and their swab kits for instant hygiene monitoring and detection of microorganisms in the brewery. *J Inst Brew* 2001;107:31–37.

Is Environmental Contamination Associated with *Staphylococcus aureus* Clinical Infection in Maximum Security Prisons?

Over the past decade, large outbreaks of community-associated methicillin-resistant *Staphylococcus aureus* (CA-MRSA) infections have occurred in correctional facilities across the country.[1,2] Although many have been managed with aggressive interventions, response to standard infection control procedures has been variable, highlighting our incomplete understanding of staphylococcal transmission in this setting.[2] Environmental contamination has recently emerged as a possible target for novel prevention and control strategies.[3,4] This study sought to characterize the relationship between environmental contamination and clinical infection in this vulnerable population.

We conducted a case-control study of *S. aureus* environmental contamination at 2 New York State (NYS) maximum security prisons: Sing Sing (men) and Bedford Hills (women). Prisoners with documented *S. aureus* skin infections were identified by medical personnel at each prison. For every case, 2 uninfected controls—1 nasally and/or oropharyngeally colonized with *S. aureus* and 1 noncolonized—were selected from the same prison in a contemporaneous fashion. These were identified through our research group's ongoing study of *S. aureus* colonization in NYS prisons.[5]

Consenting study participants had a standardized set of environmental surfaces cultured within 1 week of infection diagnosis (cases) or selection (controls). These included bed sheets, sink handles, toilet flushes, toilet seats, cell bars, light switches, soap dishes, window handles, locker handles, and radios but varied on the basis of the prisoner's cell contents. Cultures were also obtained from shared gymnasiums in each prison at study initiation.

All samples were collected using premoistened rayon-tipped swabs and qualitatively cultured as described elsewhere.[5] *S. aureus* isolates were typed by polymerase chain reaction sequencing of the *spa* (staphylococcal protein A) gene.[6] SAS (ver. 9.2; SAS Institute) was utilized for data analysis. The study was approved by the Columbia University and NYS Department of Corrections Institutional Review Boards.

Ten cases were enrolled in this study. Twenty controls were selected, but 2 did not meet inclusion criteria. There were no significant associations between case status and the demographic and exposure variables assessed (sex, age, race/ethnicity, self-perceived health, shower frequency, and gym use). The proportion of subjects with *S. aureus* contamination on 1 or more surfaces did not vary appreciably on the basis of infection status (3/10 cases [30%] vs 6/18 controls overall [33.3%]; Table 1). Despite this, environmental contamination of controls varied depending on their colonization status. Surface contamination, when present, was more frequent among cases than among controls (13/18 surfaces from 3 cases [72.2%] vs 20/43 surfaces from 6 controls [46.5%]; $P = .07$). Six clonal types were identified on surfaces of the 9 contaminated cells; only 1 cell had more than 1 clone present. None of the infectious, colonization, or personal environmental isolates were methicillin resistant.

Of the 20 items sampled in the Sing Sing gymnasium, 8 (40%) were positive for *S. aureus*. These included the gym door handle, boxing gloves, basketballs, abdominal crunch machine, seated and upright leg presses, and hand sanitizer dispenser. Among these surfaces, 6 clonal types were found (*spa* t002, t008, t334, t701, t1510, and t2334), and all were methicillin susceptible. The Bedford Hills gymnasium was not heavily contaminated; 2 (7.7%) of 26 surfaces were positive, 1 with methicillin-resistant *spa* t008.

Few studies have assessed the prevalence and significance of bacterial surface contamination in jails or prisons. In 2009, Felkner et al[7] cultured 132 surfaces from a Texas jail in a nonoutbreak setting. *S. aureus* was recovered from 10 surfaces (7.6%), with the majority of isolates (8/10) resistant to meth-

TABLE 1. Environmental Contamination of Cases and Controls

	Cases (n = 10)	Colonized controls[a] (n = 10)	P[b]	Noncolonized controls (n = 8)	P[c]	P[d]
Environmental contamination						
By subject	3/10 (30)	6/10 (60)	.37[e]	0/8 (0)	.22[e]	1[e]
By surface	13/71 (18.3)	20/69 (29)	.14[f]	0/57 (0)	<.001[f]	.66[f]
By surface of contaminated subject's cells	13/18 (72.2)	20/43 (46.5)	.07[f]	NA	NA	NA
Environmental *spa* types represented	*spa* t148, t334, t571	*spa* t002, t148, t164, t334, t2094	NA	NA	NA	NA
Surfaces contaminated	Sheets, light switch, door handle, toilet seat, cell bars	Sheets, door handle, toilet seat, sink	NA	NA	NA	NA

NOTE. Data are proportion (%), unless otherwise indicated. Clones represented in infectious isolates were *spa* t064, t126, t174, t216, t571, and t3169; clones represented in asymptomatic colonization were *spa* t002, t008, t015, t017, t073, t084, t148, t164, t334, t359, t571, t3169, and t10062. NA, not applicable.

[a] Of the 10 colonized subjects, 3 were nasally colonized, 4 were oropharyngeally colonized, and 3 were colonized at both sites.

[b] For the comparison of cases with colonized controls.

[c] For the comparison of cases with noncolonized controls.

[d] For the comparison of cases with the 2 control groups combined.

[e] Calculated by the 2-sided Fisher exact test.

[f] Calculated by the Pearson χ^2 test.

icillin. A subset of isolates (6/10) underwent pulsed-field gel electrophoresis, and two-thirds were found to be identical to the USA300 epidemic strain (*spa* t008). Inmates at Sing Sing and Bedford Hills are known to have high rates of asymptomatic colonization with MRSA (11.2% and 11.1%, respectively) and USA300 (10% and 12.4%, respectively).[5] Although this study documented a high prevalence of staphylococcal contamination, only 1 (0.4%) of the 283 environmental cultures was positive for MRSA. The etiology of this discrepancy is unclear. As previous studies have shown effective survival of USA300 and MRSA in the environment,[6,8] the comparatively low prevalence of surface contamination with these clones may be related to infection control strategies within the prison. Since USA300 is a common cause of skin abscesses, systemic antibiotics administered to those with active infection may reduce asymptomatic carriage and subsequent environmental contamination with this clone. Similarly, aggressive environmental hygiene may be differentially applied to locations highly contaminated with this strain if they are associated with purulent skin infections. Prisoners are responsible for disinfecting their own environments using quaternary ammonium products on a weekly basis. Despite this, the true frequency and intensity of cleaning may vary on the basis of prisoner preferences. Our finding of increased environmental contamination among colonized controls compared with that among noncolonized controls suggests that asymptomatic nasal and/or oropharyngeal carriage correlates with environmental reservoirs.

Although every effort was made to culture cases' cells immediately after an infection was identified, antimicrobials and disinfection administered immediately after ascertainment may have limited our ability to capture environmental contamination. Our study is further impaired by its small sample size, limited largely by a low incidence of infections over our study period. It is possible that investigations of environmental contamination during prison-based outbreaks could yield different results. Prospective studies with larger enrollments may be more effective in demonstrating small but significant trends in environmental colonization. While mounting evidence suggests a linkage between *S. aureus* surface contamination and clinical infection, data remain conflicting.[3,4,6] Further research into prison-based infectious reservoirs will be essential to effectively protect this important population and the communities in which they reside.

ACKNOWLEDGMENTS

Financial support. This research was supported by grants from the US National Institutes of Health to F.D.L. and E.L.L. (R01 AI082536), A.-C.U. (K08 AI090013), and B.A.M. (T90 NR010824).

Potential conflicts of interest. All authors report no conflicts of interest relevant to this article. All authors submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest, and the conflicts that the editors consider relevant to this article are disclosed here.

Benjamin A. Miko, MD, MS;[1]
Carolyn T. A. Herzig, MS;[2,3]
Dhritiman V. Mukherjee, MS, PhD;[1,2]
Montina Befus, MPH;[1,2] Zoltan L. Apa, BA;[1]
Ruo Yu Bai, BS, BA;[1] Caroline J. Lee, BA;[1]
Anne-Catrin Uhlemann, MD, PhD;[1]
Elaine L. Larson, RN, PhD, FAAN, CIC;[2,3]
Franklin D. Lowy, MD[1]

Affiliations: 1. Division of Infectious Diseases, Department of Medicine, College of Physicians and Surgeons, Columbia University, New York, New York; 2. Department of Epidemiology, Mailman School of Public Health,

Columbia University, New York, New York; 3. Columbia University School of Nursing, New York, New York.

Address correspondence to Benjamin Miko, MD, MS, 630 West 168th Street, Box 82, New York, NY 10032 (bm2266@columbia.edu).

Received October 1, 2012; accepted January 4, 2013; electronically published April 9, 2013.

Infect Control Hosp Epidemiol 2013;34(5):540-542

 0899-823X/2013/3405-0020$15.00. DOI: 10.1086/670218

REFERENCES

1. Aiello AE, Lowy FD, Wright LN, Larson EL. Meticillin-resistant *Staphylococcus aureus* among US prisoners and military personnel: review and recommendations for future studies. *Lancet Infect Dis* 2006;6:335–341.
2. Centers for Disease Control and Prevention. Methicillin-resistant *Staphylococcus aureus* infections in correctional facilities—Georgia, California, and Texas, 2001–2003. *MMWR Morb Mortal Wkly Rep* 2003;52:992–996.
3. Miller LG, Diep BA. Clinical practice: colonization, fomites, and virulence: rethinking the pathogenesis of community-associated methicillin-resistant *Staphylococcus aureus* infection. *Clin Infect Dis* 2008;46:752–760.
4. Davis MF, Iverson SA, Baron P, et al. Household transmission of meticillin-resistant *Staphylococcus aureus* and other staphylococci. *Lancet Infect Dis* 2012;12:703–716.
5. Lee CJ, Sankaran S, Mukherjee DV, et al. *Staphylococcus aureus* oropharyngeal carriage in a prison population. *Clin Infect Dis* 2011;52:775–778.
6. Uhlemann A, Knox J, Miller M, et al. The environment as an unrecognized reservoir for community-associated methicillin-resisant *Staphylococcus aureus* USA300: a case-control study. *PLoS ONE* 2011;6:e22407.
7. Felkner M, Andrews K, Field LH, et al. Detection of *Staphylococcus aureus* including MRSA on environmental surfaces in a jail setting. *J Correct Health Care* 2009;15:310–317.
8. Desai R, Pannaraj PS, Agopian J, Sugar CA, Liu GY, Miller LG. Survival and transmission of community-associated methicillin-resistant *Staphylococcus aureus* from fomites. *Am J Infect Control* 2011;39:219–225.