

Epidemiological and Biological Determinants of *Staphylococcus aureus* Clinical Infection in New York State Maximum Security Prisons

Benjamin A. Miko,¹ Montina Befus,² Carolyn T. A. Herzig,^{2,3} Dhritiman V. Mukherjee,¹ Zoltan L. Apa,¹ Ruo Yu Bai,¹ Joshua P. Tanner,¹ Dana Gage,⁴ Maryann Genovese,⁴ Carl J. Koenigsmann,⁴ Elaine L. Larson,^{2,3} and Franklin D. Lowy¹

¹Division of Infectious Diseases, Department of Medicine, College of Physicians and Surgeons, ²Department of Epidemiology, Mailman School of Public Health, Columbia University, ³Columbia University School of Nursing, New York, and ⁴New York State Department of Corrections and Community Supervision, Albany, New York

Background. Large outbreaks of *Staphylococcus aureus* (SA) infections have occurred in correctional facilities across the country. We aimed to define the epidemiological and microbiological determinants of SA infection in prison to facilitate development of prevention strategies for this underserved population.

Methods. We conducted a case-control study of SA infection at 2 New York State maximum security prisons. SA-infected inmates were matched with 3 uninfected controls. Subjects had cultures taken from sites of infection and colonization (nose and throat) and were interviewed via structured questionnaire. SA isolates were characterized by *spa* typing. Bivariate and multivariable analyses were conducted using conditional logistic regression.

Results. Between March 2011 and January 2013, 82 cases were enrolled and matched with 246 controls. On bivariate analysis, the use of oral and topical antibiotics over the preceding 6 months was strongly associated with clinical infection (OR, 2.52; $P < .001$ and 4.38, $P < .001$, respectively). Inmates with clinical infection had 3.16 times the odds of being diabetic compared with inmates who did not have clinical infection ($P < .001$). Concurrent nasal and/or oropharyngeal colonization was also associated with an increased odds of infection (OR, 1.46; $P = .002$). Among colonized inmates, cases were significantly more likely to carry the SA clone *spa* t008 (usually representing the epidemic strain USA300) compared to controls (OR, 2.52; $P = .01$).

Conclusions. Several inmate characteristics were strongly associated with SA infection in the prison setting. Although many of these factors were likely present prior to incarceration, they may help medical staff identify prisoners for targeted prevention strategies.

Keywords. molecular epidemiology; bacterial infections; infection control; incarceration.

The United States has the highest incarceration rate in the world [1]. Over 2.3 million Americans are currently imprisoned and an additional 5 million are under community supervision [2]. Inmates are disproportionately drawn from low-income backgrounds and have a high prevalence of homelessness, medical and psychiatric comorbidities, and illicit drug use [3]. Crowding in

American prisons has been associated with transmission of methicillin-resistant *Staphylococcus aureus* (MRSA), hepatitis C, human immunodeficiency virus (HIV), and tuberculosis [3–6]. Several large outbreaks of MRSA have occurred in correctional facilities in California, Texas, Missouri, Georgia, and Mississippi [7–10]. Many inmates enter the prison with a confluence of established community-associated (CA)-MRSA risk factors including illicit drug use, low socioeconomic status, tattoos, immunosuppression from HIV/AIDS, and other chronic health conditions [3–11]. Upon admission, they are frequently subjected to crowding, high-risk social networks, and variable hygiene conditions that further increase their risk. Taken together, these factors place incarcerated individuals at elevated risk

Received 10 October 2014; accepted 12 March 2015; electronically published 25 March 2015.

Correspondence: Benjamin A. Miko, MD, MS, 630 West 168th St, Box 82, New York, NY 10032 (bm2266@columbia.edu).

Clinical Infectious Diseases® 2015;61(2):203–10

© The Author 2015. Published by Oxford University Press on behalf of the Infectious Diseases Society of America. All rights reserved. For Permissions, please e-mail: journals.permissions@oup.com.

DOI: 10.1093/cid/civ242

of MRSA colonization and infection [4]. To date, the epidemiology of staphylococcal infections in prisons has received comparatively less attention than that of jails [12]. It should also be noted that the majority of studies on staphylococcal epidemiology in the community setting have focused on MRSA. Whether principles of MRSA epidemiology can be applied to methicillin-susceptible strains remains unclear at present.

Although prisons and communities operate as distinct environments, transmission of MRSA between the 2 settings occurs on a regular basis [8, 9, 11, 13]. Several large epidemiological investigations have identified recent incarceration or contact with incarcerated individuals as important risk factors for the development of MRSA infection in the community setting [14, 15]. These findings have led some investigators to consider correctional facilities as “amplification zones” that are capable of accelerating the MRSA epidemic in the community at large [16]. MRSA infection control interventions in correctional settings have been almost exclusively in response to outbreaks [4, 17–20]. Despite some success in the implementation of multifactorial response measures, there remain great opportunities for prevention on the individual, institutional, and system-wide levels. The objective of this study was to characterize the epidemiological and microbiological determinants of *S. aureus* (SA) clinical infection in maximum security prisons to facilitate the development of effective prevention strategies for this underserved population.

METHODS

Study Sites

We conducted a case-control study of SA infection at 2 New York State (NYS) maximum security prisons: Sing Sing Correctional Facility (housing approximately 1800 men) and Bedford Hills Correctional Facility (housing approximately 900 women). Average length of incarceration is greater at Bedford Hills than Sing Sing (38 months v. 21 months, respectively) [21]. The majority of inmates at both prisons are serving sentences for violent or drug-related felonies committed in NYC.

Study Design, Subject Enrollment, and Data Collection

Participation in this study was voluntary; compensation is not permitted for prison inmates in NYS. Eligibility requirements included the ability to provide informed consent and age ≥ 16 years (emancipated adults in NYS prisons). Case subjects were ascertained by prison-based medical staff who were trained on the signs and symptoms of purulent skin infections. Providers were instructed to refer all confirmed or suspected SA skin infections to our study team for further evaluation. Case subjects with positive SA cultures were specified as “confirmed”; those without culture-proven SA were considered “probable.” Three control subjects were matched by gender and time of

infection with each case in a contemporaneous fashion. Controls were randomly selected through our ongoing investigation of SA colonization in NYS prisons [21, 22]. Male controls were recruited directly from public locations in the prison (training and counseling buildings, dining halls); female controls were called to the prison medical facility prior to being invited to speak with a researcher.

Cases and controls had cross-sectional data collected on a number of factors relating to demographics, behavior (including illicit drug use, hygiene, recreational activities), and health status (including medical comorbidities, past infections, and past antibiotic use) [22]. In addition to information collected by research assistants using a standardized questionnaire, our study team had access to prison medical records and the centralized prison database that included information on duration of incarceration and prison transfer history. At the time of the study interview, research assistants collected cultures from the anterior nares and oropharynx of both cases and controls. When obtained, clinical cultures were collected by prison-based medical providers prior to referral for study enrollment. The study was approved by the institutional review boards of Columbia University Medical Center and the NYS Department of Corrections and Community Supervision.

Processing and Analysis of Cultures

Samples for culture were collected on rayon tipped swabs and processed in our laboratory as previously described [21, 22]. To determine the clonal type of each strain, isolates were *spa* typed by polymerase chain reaction sequencing. *Spa* typing allows for the categorization of strains based on heterogeneity of *spa* tandem repeats [23, 24]. Sequence analysis was performed using Ridom bioinformatics software (Ridom GmbH, Germany) by comparing different strain profiles through an international database. All clinical isolates underwent antimicrobial susceptibility testing by the Kirby-Bauer disc diffusion method. The antimicrobial agents tested included clindamycin, erythromycin, levofloxacin, linezolid, penicillin, oxacillin, and vancomycin [25].

Data Analysis and Statistical Power

Data from both facilities were pooled together for a single risk factor analysis. The outcome of clinical infection was tested using bivariate conditional logistic regression for each potential risk variable. Predictors determined to be significantly related to the development of infection (either statistically or clinically) were fitted into a multivariable conditional logistic regression model. The dependent variable was the dichotomous indicator of infection. Independent variables included demographic, behavioral, and biomedical characteristics of the individual and (when applicable) their colonizing isolates. The maximum likelihood estimates were obtained with the use of an expectation-maximization

algorithm. Heterogeneity by gender was assessed by stratified analysis. Using a 3-to-1 match, our study had 99% power to detect larger effect determinants (odds ratio [OR] = 2.5), 95% power for medium effect determinants (OR = 2), and 60% power for smaller effect determinants (OR = 1.5). Power analysis was not conducted for analysis of clonal heterogeneity. All statistical analysis was conducted using SAS software version 9.4 (SAS Institute, Cary, North Carolina).

RESULTS

Epidemiological Data

Between March 2011 and January 2013, 82 case subjects and 246 control subjects were enrolled. Participant ages ranged between 18 and 71 years; 51% were women. The median time between diagnosis of infection and study enrollment/screening for colonization was 1 day with a range of 1 to 20 days (interquartile range: 1, 5 days). Half of the case subjects (n = 41) had SA confirmed by culture; the remainder had either negative cultures or no cultures taken. All but 3 of the case patients were diagnosed with abscesses, although 5 did have concurrently documented cellulitis. The 3 nonabscess cases were (1) a human bite wound, (2) a pacemaker wound infection, and (3) staphylococcal impetigo. All of these were culture positive.

Cases and controls differed over several hypothesized risk factors (Table 1). It should be noted that analysis of these determinants stratified by study site (Sing Sing or Bedford Hills) showed similarity in the direction of risk relationships (Supplementary Data) suggesting comparability of the 2 prison populations. Variables included in the final multivariable model were age, body mass index (BMI), history of piercings, shaving of the legs, participation in group activities, diagnosis of diabetes, history of skin infections, previous antibiotic use, and SA colonization (Table 2). There was no evidence of multicollinearity among independent variables. The only variable with heterogeneity with regard to gender was BMI, although stratified analysis was not significant ($P = .062$).

Microbiological Data

Among the 41 definite cases with clinical cultures positive for SA, 19.5% (n = 8) were not colonized, 29.3% (n = 12) were colonized only in the anterior nares, 19.5% (n = 8) were colonized only in the oropharynx, and 31.7% (n = 13) were colonized at both sites ("dual colonization"). Those with dual colonization were further classified as concordant (eg, identical strain types at each site, n = 8/13, 61.5%) or discordant (eg, different strain types at each site, n = 5/13, 38.5%). As 2 of the 41 SA-positive clinical infection cultures were non-typable, they could not be compared with colonization strains for genetic similarity. For the 39 definite case subjects for whom the clinical infection isolate was typed, 31 were positive for colonization at one or more

sites. Among these, 71% (n = 22) had identical staphylococcal strain types between the clinical site and at least one of the colonization sites. Twelve (54.5%) of these 22 subjects were identical with the nasal colonization isolate, 5 (22.7%) were identical with the oropharyngeal isolate, and 5 (22.7%) were identical with concordant sites of dual colonization. Among inmates who had discordant dual colonization, none had an isolate associated with a clinical infection that was the same as the throat carriage isolate. As such, if one of the colonization isolates had the same clonal type as the clinical infection isolate, it was always that of the anterior nares. Among those with dual colonization, staphylococcal carriage isolates from the oropharynx only matched those from the clinical site if there was concordant dual colonization with the anterior nares.

In sum, 103 control subjects were colonized with SA at 1 or more sites (32 nasally alone, 50 oropharyngeally alone, and 21 dually colonized). Both sites of colonization had significant clonal heterogeneity. Of the 37 different *spa* types found in the anterior nares of control subjects, the most common were t008 (n = 6), t334 (n = 6), t922 (n = 4), and t002 (n = 3). Of the 45 unique *spa* types found in the oropharynx of controls, the most prevalent were t002 (n = 11), t008, t0334, t922, and t359 (n = 3 each).

Antibiotic Treatment and Susceptibility

All but 15 of the case patients were treated with systemic antibiotics at the time of study enrollment. Patients not prescribed systemic antimicrobials were managed with a combination of local therapy (ie, drainage, wound care) and topical antibiotics (most commonly mupirocin). Among those who received oral antibiotics, the most common agents used were trimethoprim/sulfamethoxazole (n = 31), cefazolin (n = 18), doxycycline (n = 7), clindamycin (n = 5), amoxicillin/clavulanate (n = 5), and ciprofloxacin (n = 5). Fifteen patients were treated with a combination of 2 or more antibiotics.

The majority of clinical infection isolates (73.2%, n = 30) were nonsusceptible (resistant or intermediate) to oxacillin; 92% of isolates (n = 38) were nonsusceptible to penicillin. The majority of isolates (70.7%, n = 29) were also nonsusceptible to erythromycin. Although nonsusceptibility was much less common for clindamycin (7.3%, n = 3), these isolates were not tested for inducible resistance by D-testing. Nonsusceptibility was also common for levofloxacin (36.6% of isolates, n = 15). No isolates were found to be intermediate or resistant to gentamicin, vancomycin, or linezolid.

DISCUSSION

Although little is known about the epidemiology of SA infections in prisons, several important investigations on staphylococcal disease have been conducted within county jails [8, 11–13, 26–28].

Table 1. Bivariate Analysis of Hypothesized Determinants of *Staphylococcus aureus* Clinical Infection Among Prisoners

Variable	All N = 328* (%)	Cases N = 82* (%)	Controls N = 246* (%)	OR	P Value
Female	168 (51.2)	42 (51.2)	126 (51.2)		1.00
Age, years					
18–38	175 (53.4)	35 (42.7)	140 (56.9)		
39–48	98 (29.9)	36 (43.9)	62 (25.2)		
49–58	43 (13.1)	10 (12.2)	33 (13.4)		
59–71	12 (3.7)	1 (1.2)	11 (4.5)		.009
Race/ethnicity*					
Non-Hispanic white	76/327 (23.2)	19/82 (23.1)	57/245 (23.3)		
Non-Hispanic black	161/327 (49.2)	47/82 (57.3)	114/245 (46.5)		
Hispanic	68/327 (20.8)	12/82 (14.6)	56/245 (22.9)		
Other	22/327 (6.7)	4/82 (4.9)	18/245 (7.3)		.26
Education					
Some high school	102 (31.1)	28 (34.1)	74 (30.1)		
High school grad	121 (36.9)	32 (39)	89 (36.2)		
Any college	105 (32)	22 (26.8)	83 (33.7)		.52
BMI					
≤24.9	63 (19.2)	12 (14.6)	51 (20.7)		
25.0–29.9	148 (45.1)	31 (37.8)	117 (47.6)		
30.0–34.9	74 (22.6)	23 (28.0)	51 (20.7)		
≥35	43 (13.1)	16 (19.5)	27 (11)		.09
Current cigarette use	202 (61.6)	56 (68.3)	146 (59.3)	1.15	.14
Past marijuana use*	244/327 (74.6)	61/82 (74.4)	183/245 (74.7)	1	.94
Past crack cocaine use	104 (31.7)	27 (32.9)	77 (31.3)	1.05	.78
Heroin use ever	50 (15.2)	14 (17.1)	36 (14.6)	1.17	.49
Injection drug use ever	25 (7.6)	8 (9.8)	17 (6.9)	1.41	.41
Tattoos ever	194 (59.1)	50 (61)	144 (58.5)	1.04	.70
Piercings ever	166 (50.6)	33 (40.2)	133 (54.1)	0.74	.02
Sexual activity past 6 mo ^a	49 (14.9)	14 (17.1)	35 (14.2)	1.2	.53
Physical fight in past 6 mo	11 (3.3)	3 (3.7)	8 (3.2)	1.13	.85
Showers per week*					
0–7	228/326 (69.9)	60/81 (74.1)	166/245 (67.8)		
8–14	77/326 (23.6)	19/81 (23.5)	58/245 (23.7)		
15+	21/326 (6.4)	2/81 (2.5)	21/245 (8.6)		.17
Shave (past 2 wks)					
Any body part	257 (78.4)	66 (80.5)	191 (77.6)		.61
Arms	28 (8.5)	6 (7.3)	22 (8.9)		.62
Legs	82 (25)	15 (18.3)	67 (27.2)		.059
Share any personal items*	36/327 (11)	9/82 (11)	27/245 (11)	1	.83
Gym use	100 (30.5)	22 (26.8)	78 (31.7)	0.85	.36
Yard use	199 (60.7)	46 (56.1)	153 (62.2)	0.9	.30
Participation in group activities ^b	229 (69.8)	38 (46.3)	191 (77.6)	0.6	<.001
Contact with anyone with <i>S. aureus</i> infection*	8/319 (2.5)	4/82 (4.9)	4/237 (1.7)	2.9	.12
Fair/poor general health*	70/327 (21.4)	24/82 (29.3)	46/245 (18.8)	1.56	.058
Diabetes*	33/322 (10.2)	17/81 (21)	16/241 (6.6)	3.16	<.001
HIV infection*	14/321 (4.4)	5/80 (6.2)	9/241 (3.7)	1.67	.36
Duration of incarceration (years)					
<1	159 (48.5)	40 (48.8)	119 (48.4)		
1	44 (13.4)	9 (11)	35 (14.2)		
2–3	47 (14.3)	16 (19.5)	31 (12.6)		
4–9	57 (17.4)	10 (12.2)	47 (19.1)		
≥10	21 (6.4)	7 (8.5)	14 (5.7)		.26

Table 1 continued.

Variable	All N = 328* (%)	Cases N = 82* (%)	Controls N = 246* (%)	OR	P Value
Probable history of skin infections	101 (30.8)	66 (80.5)	35 (14.2)	5.66	<.001
Definite history of skin infections	64 (19.5)	29 (35.4)	35 (14.2)	2.49	<.001
Indicator of history of infections ^c	174 (53)	72 (87.8)	102 (41.5)	2.12	<.001
Previous antibiotic use ^d	151 (46)	66 (80.5)	85 (34.6)	2.33	<.001
Systemic*	133/326 (40.8)	61/82 (74.3)	72/244 (29.5)	2.52	<.001
Topical	64 (19.5)	38 (46.3)	26 (10.6)	4.38	<.001
Colonized with any <i>S. aureus</i> clonal type	153 (46.6)	50 (61)	103 (41.9)	1.46	.002
Colonized with <i>spa</i> t008 (USA300) ^e					
Yes	49 (14.9)	23 (28)	26 (10.6)		
No	104 (31.7)	27 (32.9)	77 (31.3)		
Not colonized	175 (53.3)	32 (39)	143 (58.1)		<.001

All data are number (percent); "high school grad," graduation from high school or certification of high school equivalency.

Abbreviations: BMI, body mass index; HIV, human immunodeficiency virus; OR, odds ratio.

* Total numbers of subjects as per column heading unless noted by star; if subject data is missing on particular variables, the correct denominator appears within the chart.

^a Piercings reflects 1 or more of the following sites: ear, nose, other facial location, abdomen.

^b Group participation included one or more of the following activities: counseling services, vocational activities, educational activities, athletic activities, social activities, ethnic groups, hobby groups, gang membership.

^c Indicator of history of infection is composite variable measuring whether an individual had one or more of the following characteristics: use of systemic antibiotics in the previous 6 months, use of topical antibiotics in the previous 6 months, documented history of skin infections.

^d Previous antibiotic use measures either topical or systemic antibiotic used in the previous 6 months.

^e *S. aureus* clonal type *spa* t008 often corresponds to the epidemic strain USA300 (a pulsed-field gel electrophoresis designation).

Jails and prisons, which are often referred to interchangeably, typically represent distinct environments with unique populations. American jails are locally operated and hold individuals awaiting

Table 2. Final Multivariable Model of Hypothesized Determinants of *Staphylococcus aureus* Clinical Infection Among Prisoners

Variable	OR (95% CI)
Age, years	
18–38	Ref
39–48	3.4 (1.28–8.90)
49–58	0.6 (.16–2.37)
59–71	0.2 (.02–1.72)
BMI	
≤24.9	Ref
25.0–29.9	0.8 (.25–2.82)
30.0–34.9	0.9 (.23–3.28)
≥35	1.4 (.34–5.86)
Piercings ever	0.4 (.15–.90)
Shave legs	0.32 (.09–1.16)
Participation in group activities	0.1 (.02–.24)
Diabetes	3.6 (.88–14.8)
Definite history of skin infections	2.7 (1.02–7.34)
Previous antibiotic use	23.33 (7.77–70.84)
Colonization with any <i>S. aureus</i> clonal type	3.26 (1.26–8.45)

Abbreviations: BMI, body mass index; CI, confidence interval; OR, odds ratio; Ref, reference.

trial and those sentenced to short terms of incarceration (usually less than 1 year). In contrast, prisons incarcerate convicted individuals, often for several years. Administered by states or the federal government, prisons hold inmates that are less transient and possibly more amenable to longitudinal public health interventions. To date, the largest characterization of MRSA clinical infection in jails was reported by Maree et al in 2010 [11]. This case-control study, which was performed in the Los Angeles County Jail, enrolled 60 inmates with MRSA infection and 102 uninfected controls. MRSA nasal colonization was found to be significantly more prevalent among case subjects compared to control subjects (35% vs 11%, $P < .001$). Apart from asymptomatic colonization, MRSA infection was associated with lower educational level, lack of knowledge about "Staph" infections, lower rates of showering in jail, recent skin infection, sharing soap with other inmates, and less pre-incarceration contact with the healthcare system. As several of these factors related to education and hygiene were potentially modifiable, the authors proposed jail-based interventions including increased access to daily showers, availability of liquid soap, and education about SA infections. Smaller reports have shown such measures to be effective in decreasing staphylococcal infection in correctional and athletic settings [9].

To the best of our knowledge, this investigation is the first case-control study of SA clinical infection in the prison setting. Our study identified several statistically and clinically significant

inmate-level factors associated with staphylococcal infection. While our investigation evaluated similar behavioral factors to the Maree study [11], we did not find level of education, frequency of showering, or sharing of hygiene products to be associated with clinical infection. This likely relates to differences in our study populations. As jails hold large and transient populations, crowding may contribute to poor hygiene habits that increase the risk of SA infection. Interestingly, 2 behavioral variables (piercings and participation in group activities) were found to be protective against staphylococcal infection. Although the mechanisms underlying these associations remain unclear, it is possible that the protection of group activities is explained by reverse causation as infected inmates are often in isolation and/or not allowed to mix with the general prison population. Given the lack of hygiene or educational determinants in our study, possible interventions proposed for the jail setting may be ineffective when applied to prisons.

Although no demographic factors evaluated reached statistical significance, we noted a consistent increase in odds of infection with rising BMI. Previous reports have noted similar associations between obesity and both community-associated and hospital-associated SA infections [29, 30]. Identification of obese individuals as those at elevated risk of infection may facilitate the development of targeted prophylactic and preemptive strategies. Similarly, the biomedical variables found to be associated with infection (diabetes, fair/poor health status, history of skin infection, previous systemic and topical antibiotic use, and staphylococcal colonization status) may also be useful in identifying those inmates at high risk of clinical disease. Taken together, such information may be helpful in the design and implementation of infection control initiatives (eg, targeted nasal decolonization) aimed at those individuals with the greatest potential for benefit.

Several microbiological findings of this study are of particular importance. First, our study demonstrated that community-onset MRSA driven by clonal type *spa* t008 (often corresponding to the epidemic strain USA300) causes the bulk of clinical infections in the prison setting. Second, the pattern of asymptomatic SA colonization may have important implications for clinical infection. The oropharynx has only recently been recognized as a common site of staphylococcal carriage in community populations, with some studies showing a greater burden of colonization at this site compared to the anterior nares [21]. Despite this, the significance of oropharyngeal colonization remains poorly understood, and its association with clinical disease has yet to be determined. Although our study showed a concordance between isolates colonizing the nose and those causing clinical infection, it was not adequately powered to determine the significance of this association. Larger studies may be better able to define the clinical significance of oropharyngeal

carriage in the pathogenesis of staphylococcal disease in the community setting. Such information may have significant implications on infection prevention strategies such as screening and decolonization protocols.

Some important limitations of this study must be acknowledged. Our case ascertainment, although standardized between prison facilities, relied upon provider recognition of probable infections. Our study team addressed this limitation through quarterly meetings with prison-based health providers focusing on signs, symptoms, and treatment of staphylococcal infections. Although all medical providers received the same training, it is likely that there was variability in the referral pattern between practitioners, or perhaps by the same practitioner depending on different circumstances. As surveillance for purulent skin infections is not performed at either prison, there is no documentation of the total number of incident staphylococcal infections at our sites during the study period. Because of this, we are unable to formally define the differences between the total population of inmates with skin and soft tissue infections and those included in our specific sample. Another threat to representative sampling was our exclusion of inmates placed in solitary confinement. While this was necessary for protection of study subjects and research personnel, it may have excluded both case and control participants. Inclusion of both probable (those without positive clinical cultures) and confirmed (those with positive clinical cultures) cases is a potential source of misclassification. Previous studies in the community setting have shown SA to be the cause of a large majority of purulent skin and soft tissue infections [14]. A sensitivity analysis repeating the same multivariable analyses with probable and confirmed cases separately showed identical associations, supporting our method of case ascertainment.

Additional considerations for interpretation of study findings relate to our use of case-control methodology and its employment of retrospective data. Several variables analyzed for their association with clinical infection relied upon self-reporting of past or current events. As such, reporting or recall bias may have impaired our ability to accurately classify the presence or absence of hypothesized risk factors. With regard to microbiological data, the retrospective nature of case-control studies impairs our ability to define the direction of causation. For example, our study demonstrated a significant association between staphylococcal colonization and clinical infection. While this suggests that nasal and/or oropharyngeal colonization may be a risk factor for incident infection, one could also argue that staphylococcal clinical disease is a potential cause of asymptomatic colonization. Further complicating the association between infection and colonization, individuals receiving systemic antibiotics at the time of nasal and oropharyngeal sampling may have had transient suppression of

SA colonization. This, however, would bias data toward the null. As MRSA nasal colonization is believed to be a risk factor for subsequent MRSA infection (especially in hospitalized individuals), concerns regarding causal inference are partially mitigated through comparison with the existing staphylococcal literature [4, 31, 32].

Despite these limitations, this study provides tools to address an urgent public health problem among a large and underserved population. As has been demonstrated in several studies, prisoners carry a high burden of SA colonization and infection. Upon their release, they likely contribute to a community-based reservoir of SA among lower income populations. The prison environment provides a unique opportunity to identify, screen, and treat high-risk individuals that could otherwise be missed in the community setting. Our study results have direct applicability to the design of such measures and may potentially contribute to the health promotion of both prisoners and the communities in which they reside.

Supplementary Data

Supplementary materials are available at *Clinical Infectious Diseases* online (<http://cid.oxfordjournals.org>). Supplementary materials consist of data provided by the author that are published to benefit the reader. The posted materials are not copyedited. The contents of all supplementary data are the sole responsibility of the authors. Questions or messages regarding errors should be addressed to the author.

Notes

Financial support. This research was supported by a grant from the U.S. National Institutes of Health to F. D. L. and E. L. L. [R01 AI082536].

Potential conflicts of interest. All authors: No potential conflicts of interest.

All authors have submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Conflicts that the editors consider relevant to the content of the manuscript have been disclosed.

References

1. International Centre for Prison Studies. Entire world - prison population rates per 100 000 of the national population. World Prison Brief. Available at: http://www.prisonstudies.org/info/worldbrief/wpb_stats.php?area=all&category=wb_poprate. Accessed 30 September 2013.
2. Glaze LE, Herberman EJ. Correctional Population in the United States, 2012. Bureau of Justice Statistics Bulletin (Publication No. NCJ 243936). Washington, DC: US Department of Justice, 2013.
3. Aiello AE, Lowy FD, Wright LN, Larson EL. Methicillin-resistant *Staphylococcus aureus* among US prisoners and military personnel: review and recommendations for future studies. *Lancet Infect Dis* 2006; 6:335–41.
4. David M, Daum R. Community-associated methicillin-resistant *Staphylococcus aureus*: epidemiology and clinical consequences of an emerging epidemic. *Clin Microbiol Rev* 2010; 23:616–87.
5. Leh S. HIV infection in U.S. correctional systems: its effect on the community. *J Community Health Nurs* 1999; 16:53–63.
6. Turabelidze G, Lin M, Wolkoff B, Dodson D, Gladbach S, Zhu BP. Personal hygiene and methicillin resistant *Staphylococcus aureus* infection. *Emerg Infect Dis* 2006; 12:422–7.
7. Methicillin-resistant *Staphylococcus aureus* skin or soft tissue infections in a state prison—Mississippi, 2000. *Morb Mortal Wkly Rep* 2001; 50:919–22.
8. Pan ES, Diep BA, Carleton HA, et al. Increasing prevalence of methicillin-resistant *Staphylococcus aureus* infection in California jails. *Clin Infect Dis* 2003; 37:1384–8.
9. Methicillin-resistant *Staphylococcus aureus* infections in correctional facilities—Georgia, California, and Texas, 2001–2003. *Morb Mortal Wkly Rep* 2003; 52:992–6.
10. Outbreaks of community-associated methicillin-resistant *Staphylococcus aureus* skin infections—Los Angeles County, California, 2002–2003. *Morb Mortal Wkly Rep* 2003; 52:88.
11. Maree CL, Eells SJ, Tan J, et al. Risk factors for infection and colonization with community-associated methicillin-resistant *Staphylococcus aureus* in the Los Angeles County jail: a case-control study. *Clin Infect Dis* 2010; 51:1248–57.
12. David MZ, Mennella C, Mansour M, Boyle-Vavra S, Daum RS. Prevalence of methicillin-resistant *Staphylococcus aureus* among pathogens causing skin and soft tissue infections in a large urban jail: risk factors and recurrence rates. *J Clin Microbiol* 2008; 46:3222–7.
13. Okano JT, Blower S. Are correctional facilities amplifying the epidemic of community-acquired methicillin-resistant *Staphylococcus aureus*? *Nat Rev Microbiol* 2010; 8:83.
14. Moran GJ, Krishnadasan A, Gorwitz RJ, et al. Methicillin-resistant *S. aureus* infections among patients in the emergency department. *N Engl J Med* 2006; 355:666–74.
15. Jacobus C, Lindsell C, Leach S, et al. Prevalence and demographics of methicillin resistant *Staphylococcus aureus* in culturable skin and soft tissue infections in an urban emergency department. *BMC Emerg Med* 2007; 7:19.
16. Wootton SH, Arnold K, Hill HA, et al. Intervention to reduce the incidence of methicillin-resistant *Staphylococcus aureus* skin infections in a correctional facility in Georgia. *Infect Control Hosp Epidemiol* 2004; 25:402–7.
17. Goldstein E, Hradecky G, Vilke G, Chan TC. Impact of a standardized protocol to address outbreak of methicillin-resistant *Staphylococcus aureus* skin infections at a large, urban county jail system. Available at: [http://lapublichealth.org/acd/docs/Impact_of_Standardized_Protocol_for_MRSA\[1\].pdf](http://lapublichealth.org/acd/docs/Impact_of_Standardized_Protocol_for_MRSA[1].pdf). Accessed 30 September 2013.
18. Mandeville M. Making Strides with MRSA. The Corrections Connection. Available at: <http://www.corrections.com/articles/579-making-strides-with-mrsa>. Accessed 30 September 2013.
19. Federal Bureau of Prisons. Management of methicillin resistant *Staphylococcus aureus* (MRSA) infection: Clinical Practice Guidelines. Available at: <http://www.bop.gov/resources/pdfs/mrsa.pdf>. Accessed 30 September 2013.
20. Bick JA. Infection control in jails and prisons. *Clin Infect Dis* 2007; 45:1047–55.
21. Lee CJ, Sankaran S, Mukherjee DV, et al. *Staphylococcus aureus* oropharyngeal carriage in a prison population. *Clin Infect Dis* 2011; 52:775–8.
22. Mukherjee DV, Herzig CT, Jeon CY, et al. Prevalence and risk factors for *Staphylococcus aureus* colonization in individuals entering maximum-security prisons. *Epidemiol Infect* 2014; 142:484–93.
23. Shopsin B, Gomez M, Montgomery SO, et al. Evaluation of protein A gene polymorphic region DNA sequencing for typing of *Staphylococcus aureus* strains. *J Clin Microbiol* 1999; 37:3556–63.
24. Mellmann A, Weniger T, Berssenbrugge C, et al. Based Upon Repeat Pattern (BURP): an algorithm to characterize the long-term evolution of *Staphylococcus aureus* populations based on *spa* polymorphisms. *BMC Microbiol* 2007; 7:98.
25. Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing; twenty-second informational supplemental, M100-S22. Wayne, PA: CLSI, 2012.
26. David MZ, Siegel J, Lowy FD, et al. Asymptomatic carriage of sequence type 398, *spa* type t571 methicillin-susceptible *Staphylococcus aureus* in an urban jail: a newly emerging, transmissible pathogenic strain. *J Clin Microbiol* 2013; 51:2443–7.

27. Elias AF, Chaussee MS, McDowell EJ, Huntington MK. Community-based intervention to manage an outbreak of MRSA skin infections in a county jail. *J Correct Health Care* **2010**; 16:205–15.
28. Deger GE, Quick DW. The enduring menace of MRSA: incidence, treatment, and prevention in a county jail. *J Correct Health Care* **2009**; 15:174–8.
29. Olsen K, Danielsen K, Wilsgaard T, et al. Obesity and *Staphylococcus aureus* nasal colonization among women and men in a general population. *PLoS One* **2013**; 8:e63716.
30. Kaye KS, Marchaim D, Chen TY, et al. Predictors of nosocomial bloodstream infections in older adults. *J Am Geriatr Soc* **2011**; 59:622–7.
31. Wertheim HF, Melles DC, Vos MC, et al. The role of nasal carriage in *Staphylococcus aureus* infections. *Lancet Infect Dis* **2005**; 5:751–62.
32. Fritz SA, Epplin EK, Garbutt J, Storch GA. Skin infection in children colonized with community-associated methicillin-resistant *Staphylococcus aureus*. *J Infect* **2009**; 59:394–401.