Epidemiol. Infect. (2017), **145**, 2161–2165. © Cambridge University Press 2017
doi:10.1017/S0950268817000942

SHORT REPORT
Correspondence analysis to evaluate the transmission of *Staphylococcus aureus* strains in two New York State maximum-security prisons

M. BEFUS[1]*, D. V. MUKHERJEE[2], C. T. A. HERZIG[1,3], F. D. LOWY[2] AND E. LARSON[1,3]

[1] *Department of Epidemiology, Mailman School of Public Health, Columbia University, New York 10032, USA*
[2] *Department of Medicine, Division of Infectious Diseases, Columbia University, New York 10032, USA*
[3] *School of Nursing, Columbia University, New York 10032, USA*

Received 11 November 2016; Final revision 5 April 2017; Accepted 7 April 2017; first published online 16 May 2017

SUMMARY

Prisons/jails are thought to amplify the transmission of *Staphylococcus aureus* (SA) particularly methicillin-resistant SA infection and colonisation. Two independently pooled cross-sectional samples of detainees being admitted or discharged from two New York State maximum-security prisons were used to explore this concept. Private interviews of participants were conducted, during which the anterior nares and oropharynx were sampled and assessed for SA colonisation. Log-binomial regression and correspondence analysis (CA) were used to evaluate the prevalence of colonisation at entry as compared with discharge. Approximately 51% of admitted ($N = 404$) and 41% of discharged ($N = 439$) female detainees were colonised with SA. Among males, 59% of those admitted ($N = 427$) and 49% of those discharged ($N = 393$) were colonised. Females had a statistically significant higher prevalence (1·26: $P = 0{\cdot}003$) whereas males showed no significant difference (1·06; $P = 0{\cdot}003$) in SA prevalence between entry and discharge. CA demonstrated that some strains, such as *spa* types t571 and t002, might have an affinity for certain mucosal sites. Contrary to our hypothesis, the prison setting did not amplify SA transmission, and CA proved to be a useful tool in describing the population structure of strains according to time and/or mucosal site.

Key words: Carrier state, colonisation, incarceration, *Staphylococcus aureus*, MRSA.

The United States of America (US) accounts for 5% of the world's population but holds 25% of the world's incarcerated population [1]. In 2015, approximately 11 million individuals were admitted to jails, which are correctional facilities operated by local governments that house detainees awaiting trial, sentencing or transfer to state prisons [2]. In addition, approximately 700 000 were admitted to prisons, which house detainees sentenced to a year or more [2]. As of 31 December 2015, state and federal correctional facilities had jurisdiction over 1.5 million prisoners, 600 000 of whom are released each year, and of those released approximately 40% will return to jail and/or prison within 12 months of their release [2]. Vulnerable underserved populations from impoverished communities are at elevated risk of incarceration as well as *Staphylococcus aureus* (SA) colonisation and infection [3]. Given the fluidity that exists between incarcerated settings and vulnerable communities, it is important that we determine whether jails and prisons increase the burden of SA

* Author for correspondence: M. Befus, PhD MPH, Post-doctoral Fellow, Training in Interdisciplinary Research to Prevent Infections, 617W, 168th Street, Room 331, New York, NY 10032, USA. (Email: mbb2143@cumc.columbia.edu)

among detainees and through them the communities to which they are released.

SA remains an important human pathogen [3]. It is also a transient and sometimes persistent component of the resident flora of the anterior nares in a large proportion of the US population [3]. Nasal colonisation with SA is a significant determinant of subsequent infection [4] and a mode of transmission [3, 5]. The importance of extra-nasal colonisation, particularly in the oropharynx, has also been described [5, 6]. Mathematical models of SA, particularly methicillin-resistant SA (MRSA), in both jails and prisons suggest that these settings serve as amplification zones, defined as settings where transmission is increased due to a confluence of risk factors [7]. Unfortunately, these models do little to elucidate transmission at the molecular level and ignore the role of colonisation, particularly extra-nasal. To bridge this gap, we applied stacked correspondence analysis (CA), a multivariate ordination technique, to explore the association between SA and its environment. We hypothesised that traditional regression methods would demonstrate that the prison setting amplified transmission resulting in higher colonisation rates among discharged individuals, and that CA would demonstrate that certain strains interact with colonisation site.

We conducted two independently pooled cross-sectional studies of detainees admitted to, and discharged from, prison in one female and one male maximum-security prison in New York State. The study methodology has been previously described [6]. Briefly, detainees $\geqslant$16 years of age newly admitted to or being discharged from the prisons were eligible for inclusion. It is important to note that in independently pooled studies, observations in one cross-sectional sample are independent of the observations assessed in subsequent cross-sectional samples. More specifically, the men and women assessed at prison admission were different from those assessed at prison discharge. For both cross-sectional samples, trained interviewers used a structured questionnaire to collect demographic, behavioural and medical information from detainees and abstracted information from their medical records after obtaining written informed consent. During the private interview, the anterior nares and oropharynx of participants were sampled and later processed to assess SA colonisation. The institutional review boards of Columbia University Medical Center and the New York State Department of Corrections and Community Supervision approved the study.

Swabs samples were incubated overnight at 35 °C in enrichment broth (6% sodium chloride-supplemented tryptic soy broth) [6] and subsequently plated onto mannitol salt agar before incubation at 37 °C for 48 h. Suspected SA colonies were streaked onto sheep blood agar plates and confirmed by the StaphAurex test (Remel Europe Ltd., Dartford, UK) [8]. Confirmed isolates were characterised by *spa* typing and compared using Ridom StaphType software version 2.2.1 (Ridom GmbH, Würzburg, Germany) [9]. MRSA was determined by the presence and type of staphylococcal chromosomal cassette *mec* using multiplex polymerase chain reaction [10].

The collected data were analysed separately for males and females. Bivariate analyses were conducted using χ^2 test or *t*-test as appropriate. Factors associated with both the exposure (time-point) and outcome (colonisation of the nares and/or oropharynx) with a 10% level of significance were included in the multivariable model. Log-binomial regression was used to assess the association between time-point (admission *vs.* discharge) and SA colonisation. Due to sample size constraints, we were unable to assess the independent effect of time-point on MRSA colonisation.

Stacked CA was used, in addition to the traditional regression methods described above, to describe the influence of time-point as well as colonisation site (anterior nares *vs.* oropharynx) on the distribution of SA strains. CA depicts multivariate data on a two-dimensional plot by implementing a distance matrix to extract underlying factors that drive the data distribution, and graphs the data based on their similarities and dissimilarities [11]. CA results in a geometric map/biplot illustrating how the relative frequencies of SA strain types changed with colonisation site and time point. To interpret the results, the spread of the data points from top to bottom and left to right provide an indication of how similar or dissimilar the data were with the centre of the axis representative of the most common characteristics found in the data. All analyses were conducted using SAS software version 9.3 (SAS institute Inc. Cary, North Carolina, USA).

Over the 5-year study period (2009–2013), 404 female and 427 male participants were recruited at prison admission and 439 female and 393 males were recruited at discharge (Supplementary Table 1). Fifty-one per cent of newly admitted female participants were colonised with SA compared with 41% of those being discharged. With respect to MRSA, approximately 11% of female detainees newly

https://doi.org/10.1017/S0950268817000942 Published online by Cambridge University Press

admitted to the prison and 9% of those being discharged were colonised with MRSA. Of those colonised at admission, 27% were colonised exclusively in the nose, 36% exclusively in the throat and 37% at both sites. Among female detainees colonised at discharge, 35% were exclusively in the nose, 35% exclusively in the throat and 30% at both sites. Female participants entering prison were statistically significantly more likely to be colonised than those being discharged after controlling for age, race, smoking, drug use, topical antibiotic use, obesity and asthma (prevalence ratio = 1·26; 95% confidence interval (CI) 1·08–1·49).

The results differed for male detainees; the proportion colonised at prison entry and discharge were 59% and 49%, respectively, when all SA isolates were considered and 6% and 3% when restricted to MRSA (Supplementary Table 1). Of those colonised, exclusive throat colonisation was higher among males being discharged compared with those being admitted (55% *vs*. 43%). Exclusive nasal colonisation was 26% and 18% at admission and discharge, respectively. There was no statistically significant difference in the prevalence of SA colonisation for male detainees at admission compared with discharge controlling for age, race, education smoking, antibiotic use, obesity, and respiratory disease (prevalence ratio = 1·06; 95% CI 0·92–1·23).

A total of 1068 SA isolates were available for characterisation and included in the stacked CA (Fig. 1); the prevalence of each *spa* type is also shown in the figure. The first dimension (plotted on the vertical axis) extracted for males was the colonisation site and for females, the time-point, indicating the factors that contributed most to variation in the data differed for men and women. Among males (Fig. 1*b*) the vertical axis represents site of colonisation. Moving from the top of the plot to the bottom, affinity for nasal colonisation transitions to affinity for throat colonisation. The horizontal axis represents time-point with affinity for discharge indicated on the right and affinity for admission on the left. In the CA for female detainees (Fig. 1*a*), the vertical axis represents time-point and the horizontal axis represents colonisation site.

The most prevalent *spa* type at both admission and discharge was t008 representing 14% and 11% of all strains in females and males, respectively. *Spa* type t008 is in the proximity of the axis centre demonstrating it has no affinity to a specific colonisation site or time-point. In contrast, *spa* type t002, the third most prevalent type, demonstrated an affinity for throat colonisation among male detainees and was more prevalent at entry as compared with admission for both females and males (Fig. 1). Another observation made is the affinity for some *spa* types such as t571 and t316 for nasal colonisation, suggesting that SA strains may interact differently to different mucosal sites.

Based on the conceptual model of prisons and jails as SA amplification zones, we hypothesised that the prevalence of colonisation would significantly increase at prison discharge compared with at admission. However, we observed a significant decrease in the prevalence of colonisation among female detainees newly admitted to prison compared with those being discharged. Additionally, no statistically significant difference in prevalence was observed between admitted and discharged male detainees.

These findings might be explained by the fact that the majority of female detainees newly admitted to the prison under study were transferred from jails, and could therefore serve as a representative sample of SA colonisation among detainees discharged from jails to prisons. Male detainees were largely transferred from other prisons. This suggests that detainees discharged from jails to prisons and prison detainees have a significantly different prevalence of SA colonisation. Whether transmission is amplified in jails, or detainees entering jails merely present with higher rates of colonisation compared with the general population as reported by Farley *et al*. [12] has yet to be determined. It is important to point out, however, that though mathematical models by Okano *et al*. [7] support the amplification framework, other models proposed by the same group [13] suggest that MRSA outbreaks in jails were sustained primarily due to new admittance of colonised and/or infected detainees rather than within facility transmission. A recent study of SA colonisation in jails conducted by David *et al*. [14] corroborated the latter findings, but could also not directly address within facility transmission. The overall prevalence of SA colonisation amongst the detainees assessed in the latter study was slightly lower than that observed amongst newly admitted female detainees in the current study. To the best of our knowledge, only one other study [15], which had comparable prevalence estimates to both our male and female discharge sample, has assessed SA colonisation in a prison sample distinct from ours. Transmission dynamics were, however, not assessed. More studies are therefore

https://doi.org/10.1017/S0950268817000942 Published online by Cambridge University Press

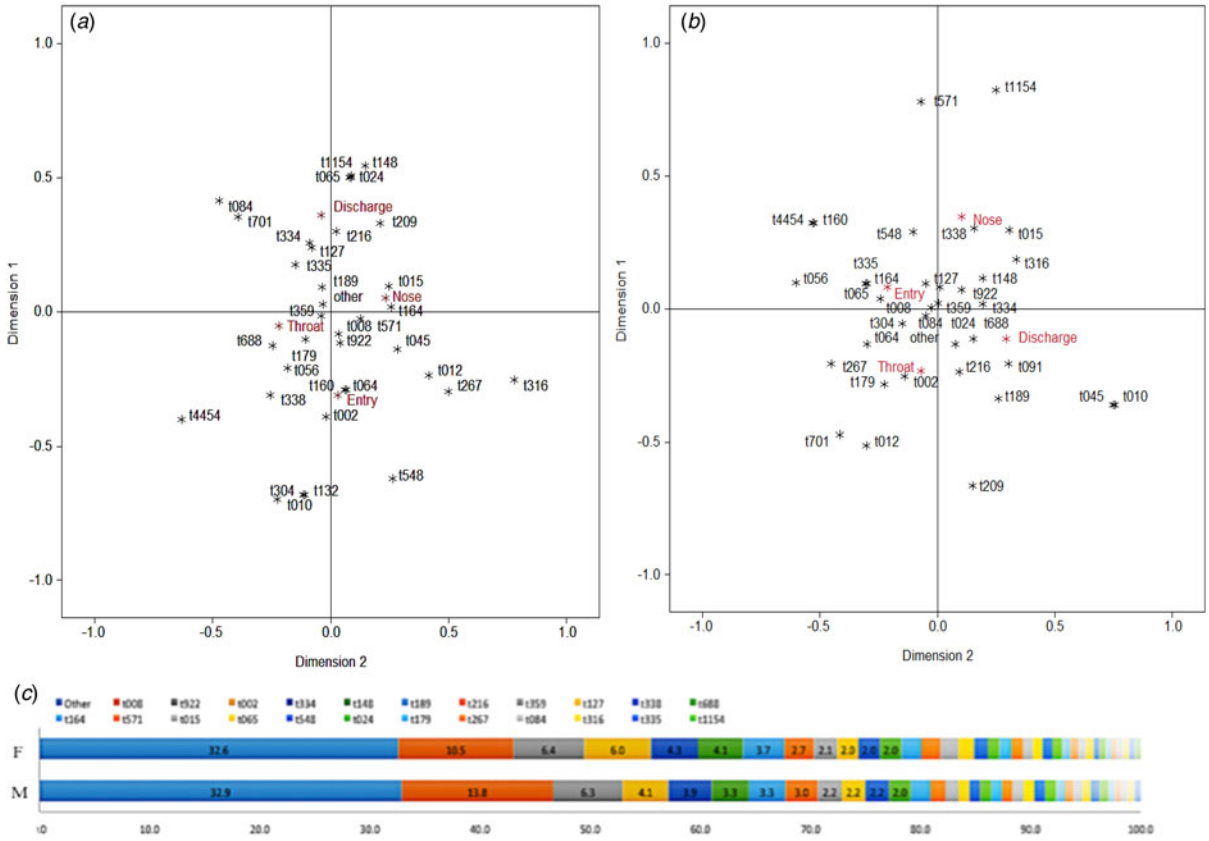

Fig. 1. Geometric biplot of the correspondence analysis of 506 isolates from female (panel (*a*)) and 562 isolates from male (panel (*b*)) New York State detainees. Dimension 1 corresponds to the most important underlying factor driving the distribution of the data points and dimension 2 corresponds to the second most important factor. The spread of the points from top to bottom and left to right provide an indication of how similar or dissimilar the data were. The centre of the axis represents the most common characteristics in the data. Other refers to spa types isolated lest than three times during the study period. The prevalence of each spa type included is given in panel (*c*) for both females (F) and males (M). The prevalence of *spa* types that accounted for <2% of isolates are not numerically displayed.

needed to elucidate the role of incarcerated settings on disease transmission.

It also warrants mention that differences in characteristics such as drug use or hygiene at prison entry compared with discharge could also explain our results. The inclusion of these factors as covariates in the multivariable model, however, ensured that only the independent effect of time point was expressed in the resulting coefficient. Residual and uncontrolled confounding, however, cannot be completely ruled out.

Our results additionally demonstrated that male and female detainees had higher prevalence of exclusive oropharyngeal colonisation than in the nares. Studies have shown higher rates of oropharyngeal SA carriage than in the nares [6], but none have provided an explanation for this observation. The stacked CA approach presented here begins to explain our observations by demonstrating that some *spa* types; e.g. t002, the third most prevalent in the study, demonstrated a greater affinity for the oropharynx. CA also indicated that *spa* type t571 (ST398), a strain frequently associated with livestock, displayed an affinity for nasal colonisation.

Although the CA provided some interesting findings, it is important to note that it is an exploratory technique. Confirmation of these observations, which can be conducted using canonical CA, the hypothesis-driven alternative to CA, should be considered in future studies. Though CA is rarely used in molecular epidemiologic research, it has the potential to bolster molecular analyses for both exploratory and hypothesis testing endeavours. We recommend the use of this analytical tool particularly with the growing interests in microbial composition at different body sites and their interaction with specific environments.

Our study is limited by the use of two independently pooled cross-sectional samples to determine the

https://doi.org/10.1017/S0950268817000942 Published online by Cambridge University Press

epidemiology of SA colonisation at prison admission and discharge, rather than prospective assessment of a single cohort. This limitation is mitigated by the use of multivariable regression analysis that rendered both cohorts similar with respect to measured variables. Additionally, use of CA not only confirmed that time-point was a significant factor among colonised women by its extraction as the first dimension, but it also provided a potential explanation for the high prevalence of oropharyngeal colonisation within the study population.

In conclusion, we found no evidence that the prison setting amplifies SA colonisation. We additionally show that detainees being discharged from prison have a lower prevalence of SA colonisation than do detainees discharged from jails to prisons. Furthermore, the results support the utility of CA in its ability to generate testable hypotheses in microbial epidemiological research that incorporates molecular data.

SUPPLEMENTARY MATERIAL

The supplementary material for this article can be found at https://doi.org/10.1017/S0950268817000942

ACKNOWLEDGEMENTS

The authors would like to thank Zoltan Apa and Jennifer Bai for their diligent work in collecting the data used in this study. They would also like to thank Joshua Tanner and Caroline Lee for processing all of the biological data. This work was supported by the National Institute of Allergy and Infectious Disease at the National Institutes of Health (grant 5R01AI082536) and the National Institute of Nursing Research-funded Training in Interdisciplinary Research to Prevent Infections (TIRI), (grant 5T32NR013454). The contents of the manuscript are solely the responsibility of the authors and do not necessarily reflect the official views of the funding agency.

REFERENCES

1. **Pew Charitable Trusts.** *One in 100: Behind Bars in America 2008*. Washington, DC: The Pew Charitable Trusts, 2008.
2. **Kaeble D, Glaze L.** *Correctional Populations in the United States, 2015*. Washington, DC: US Dept of Justice, Office of Justice Programs, 2016 (https://www.bjs.gov/content/pub/pdf/cpus15.pdf) Accessed 24 January 2017.
3. **David MZ, Daum RS.** Community-associated methicillin-resistant *Staphylococcus aureus*: epidemiology and clinical consequences of an emerging epidemic. *Clinical Microbiology Reviews* 2010; **23**: 616–687.
4. **von Eiff C, *et al.*** Nasal carriage as a source of *Staphylococcus aureus* bacteremia. Study group. *New England Journal of Medicine* 2001; **344**: 11–16.
5. **Knox J, Uhlemann AC, Lowy FD.** *Staphylococcus aureus* infections: transmission within households and the community. *Trends in Microbiology* 2015; **23**: 437–444.
6. **Lee CJ, *et al.*** *Staphylococcus aureus* oropharyngeal carriage in a prison population. *Clinical Infectious Diseases* 2011; **52**: 775–778.
7. **Okano JT, Blower S.** Are correctional facilities amplifying the epidemic of community-acquired methicillin-resistant *Staphylococcus aureus*? *Nature Reviews Microbiology* 2010; **8**: 83.
8. **Fournier JM, *et al.*** New latex reagent using monoclonal antibodies to capsular polysaccharide for reliable identification of both oxacillin-susceptible and oxacillin-resistant *Staphylococcus aureus*. *Journal of Clinical Microbiology* 1993; **31**: 1342–1344.
9. **Mellmann A, *et al.*** Characterization of clonal relatedness among the natural population of *Staphylococcus aureus* strains by using *spa* sequence typing and the BURP (based upon repeat patterns) algorithm. *Journal of Clinical Microbiology* 2008; **46**: 2805–2808.
10. **Milheirico C, *et al.*** Update to the multiplex PCR strategy for assignment of mec element types in *Staphylococcus aureus*. *Antimicrobial Agents and Chemotherapy* 2007; **51**(9): 3374–3377.
11. **Greenacre M.** Correspondence analysis in medical research. *Statistical Methods in Medical Research* 1992; **1**: 97–117.
12. **Farley JE, *et al.*** Prevalence, risk factors, and molecular epidemiology of methicillin-resistant *Staphylococcus aureus* among newly arrested men in Baltimore, Maryland. *American Journal of Infection Control* 2008; **36**: 644–650.
13. **Kajita E, *et al.*** Modelling an outbreak of an emerging pathogen. *Nature Reviews Microbiology* 2007; **5**: 700–709.
14. **David MZ, *et al.*** Hand and nasal carriage of discordant *Staphylococcus aureus* isolates among urban jail detainees. *Journal of Clinical Microbiology* 2014; **52**: 3422–3425.
15. **Lowy FD, *et al.*** *Staphylococcus aureus* colonization and infection in New York State prisons. *Journal of Infectious Diseases* 2007; **196**: 911–918.