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Factors associated with persistent colonisation with methicillin-resistant *Staphylococcus aureus*

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SUMMARY

We conducted a prospective cohort study between 1 January 2010 and 31 December 2012 at five adult and paediatric academic medical centres to identify factors associated with persistent methicillin-resistant *Staphylococcus aureus* (MRSA) colonisation. Adults and children presenting to ambulatory settings with a MRSA skin and soft tissue infection (i.e. index cases), along with household members, performed self-sampling for MRSA colonisation every 2 weeks for 6 months. Clearance of colonisation was defined as two consecutive negative sampling periods. Subjects without clearance by the end of the study were considered persistently colonised and compared with those who cleared colonisation. Of 243 index cases, 48 (19·8%) had persistent colonisation and 110 (45·3%) cleared colonisation without recurrence. Persistent colonisation was associated with white race (odds ratio (OR), 4·90; 95% confidence interval (CI), 1·38–17·40), prior MRSA infection (OR 3·59; 95% CI 1·05–12·35), colonisation of multiple sites (OR 32·7; 95% CI 6·7–159·3). Conversely, subjects with persistent colonisation were less likely to have been treated with clindamycin (OR 0·28; 95% CI 0·08–0·99). Colonisation at multiple sites is a risk factor for persistent colonisation and may require more targeted decolonisation efforts. The specific effect of clindamycin on MRSA colonisation needs to be elucidated.

Key words: Methicillin-resistant Staphylococcus aureus (MRSA), MRSA colonisation, persistent MRSA colonisation, MRSA skin and soft tissue infections.

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INTRODUCTION

Over recent years, there has been an increase in the number of emergency department visits for skin and soft tissue infections (SSTI) [1]. This coincides with the rise of community-onset methicillin-resistant *Staphylococcus aureus* (MRSA) infections [1]. MRSA comprises the majority of SSTI for which there is a microbiological diagnosis [2], and the proportion is particularly high in paediatric populations [3, 4].

It has been shown that colonisation with MRSA is an important risk factor for subsequent infection [5, 6]. However, the temporal pattern of MRSA colonisation varies among individuals. Of those screened for MRSA colonisation, it has been estimated that 20% will have persistent colonisation, while approximately 30% will have intermittent colonisation [7–9]. Subjects with persistent colonisation have been shown to be at higher risk for subsequent infection [10]. Additionally, MRSA colonisation can be transmitted among household members [11]. Failure to identify and interrupt colonisation within the household may serve as a barrier to preventing persistent colonisation or repeated infections [12, 13]. Furthermore, past work has demonstrated that specific antibiotic treatment of MRSA SSTI may be associated with subsequent duration of MRSA colonisation [9].

Previous studies examining the risk factors for persistent colonisation have focused exclusively on hospitalised patients and used varying definitions of 'persistence' [8, 14]. Risk factors for persistent MRSA colonisation in the outpatient setting remain unstudied. Our group has demonstrated the factors associated with longer duration of colonisation with MRSA [9] and recurrent colonisation after clearance [15] using a longitudinal systematic surveillance approach in community-dwelling adults and children. The cohort of subjects in those studies revealed that approximately 20% of subjects never cleared colonisation while approximately 45% cleared colonisation without recurrence. Therefore, we sought to compare these groups to identify the factors associated with persistent colonisation. Identification of these factors will aid in better identifying this higher risk population and in devising interventions to decrease the burden of MRSA colonisation and subsequent infection.

METHODS

Study design and study subjects

We conducted a prospective cohort study between 1 January 2010 and 31 December 2012 at five adult and paediatric academic medical centres in Southeastern Pennsylvania. As described previously in [9], adults and children presenting to emergency departments and primary care settings with an acute MRSA SSTI were eligible for entry. MRSA SSTIs identified within 48 h of hospital admission were also considered eligible. To be enrolled, a study subject (i.e. index case) and all members of his/her household were required to agree to participate. All households agreeing to participate were included. Each index case and household was enrolled only once. Informed consent was obtained from all adult index cases and household members; subjects 7–17 years of age provided assent; parents provided consent for children younger than 7 years. This study was approved by the Institutional Review Boards of all participating institutions.

Longitudinal follow-up and data collection

Index cases and household members performed self-sampling for MRSA from three anatomic sites (nares, axillae and groin) every 2 weeks for 6 months to assess for MRSA colonisation, for a total of 14 potential sampling periods. The rationale and protocol were described previously [9]. Briefly, subjects obtained specimens by placing one swab in both nares, then placing a second swab in both axillae followed by the groin. If the initial skin lesion was present, then that site was sampled with a third swab. The ESwab™ System (Copan Diagnostics Inc, Murrieta, CA) was used for sample collections. Subjects then mailed swabs to the study laboratory.

Demographic data, comorbidities, medications, number of people in the household and antibiotic use were collected via self-report at the initial home interview and updates requested at each sampling period. These data, along with data on the diagnosis and treatment of the presenting SSTI were confirmed or expanded with medical record review, including prescription records.

Laboratory testing

Swab samples were plated to BBL™ ChromAgar® MRSA medium (BD, Sparks, MD) and processed according to manufacturer's instructions [16]. Testing for *in vitro* susceptibility of *S. aureus* to oxacillin, penicillin, erythromycin, clindamycin, levofloxacin, chloramphenicol, gentamicin, trimethoprim–sulfamethoxazole, rifampin and vancomycin was performed using the Vitek 2 automated identification and susceptibility

testing system with AES (Advanced Expert System) (bioMerieux, Inc.) and interpreted according to established criteria [17]. Isolates that were erythromycin-resistant but clindamycin-susceptible were routinely tested for inducible macrolide–lincosamide–streptogramin resistance by the disk diffusion method (*D*-test) [17].

Data analysis

Only index cases who returned samples for at least the first two consecutive sampling periods were included in the analysis (to permit determination of clearance of colonisation). Clearance of colonisation was defined as two consecutive sampling periods with no positive MRSA surveillance cultures. Subjects who did not meet the definition of clearance of colonisation by the end of follow-up were categorised as having persistent colonisation. Subjects who initially met the definition of clearance but then had recurrent colonisation were excluded.

Antibiotic exposure in the index case was assessed in three distinct time periods: the year prior to diagnosis of SSTI, excluding the 14 days prior to SSTI diagnosis as this was assumed to be empiric treatment for the SSTI (past use), the 14 days following SSTI diagnosis (treatment), and day 15 after enrolment through end of follow-up (post-treatment period). Presence of colonisation in household members was analysed at baseline (i.e. first sampling period) using three different measures: at least one household member with MRSA colonisation, number of household members colonised with MRSA and proportion of household members with MRSA colonisation. The proportion of household members with MRSA colonisation was *a priori* determined to be the primary measure and forced into the final model, but secondary analyses of the other measures were also conducted.

Bivariable analyses were performed to evaluate factors independently associated with persistent colonisation in the index case using Pearson's χ^2 or Fisher's exact test for categorical variables and student's t test for continuous variables. Multivariable logistic regression was used to determine the association between factors and persistent MRSA colonisation. Variables were included in the model if they were associated with persistent MRSA colonisation on bivariable analysis (P value $\leqslant 0{\cdot}20$) [18] and were maintained in the final model if they remained significantly associated with the outcome using manual backward deletion. An odds ratio (OR) and 95% confidence interval (CI) were calculated to evaluate the strength of associations.

For all calculations, a P value (two-tailed) $<0{\cdot}05$ was considered significant. Statistical calculations were performed using commercially available software (Stata 14·1, StataCorp, College Station, TX).

RESULTS

During the 6-month study period, a total of 349 households provided informed consent. Of these enrolled households, 243 (69·6%) index cases returned samples for the first two sampling periods (permitting a calculation of duration of colonisation) and were included in the initial analysis. The only significant difference between the included and excluded index cases was in the proportion of white subjects (42·8% of included index cases *vs.* 26·4% of excluded index cases, $P = 0{\cdot}004$). However, there were no differences observed in other demographic factors, household size or comorbidities between included and excluded subjects.

Among the 243 index cases, 110 (45·3%) were determined to have clearance of MRSA colonisation without recurrence during the study period, while 48 (19·8%) met the definition of persistent colonisation. The median age of index cases was 19·5 years (interquartile range (IQR) 3·9–47·5) and 99 (62·7%) were female. Thirty-two (20·3%) index cases reported a history of prior MRSA infection; however, 46 (29·1%) index cases did not provide a response to this question. Subjects who reported prior MRSA infection were not more likely to have received a prescription for decolonisation agents (topical mupirocin or bleach baths/chlorhexidine) ($P = 0{\cdot}950$).

In the 158 households, there were a total of 491 household members. The median age of household members was 23 (IQR 9–38), 198 (40·3%) were 18 years or younger and 279 (56·8%) were females. Median duration of follow up was 106·5 days (IQR 56–186) for index cases. Sampling was completed for a median of 7·5 episodes (IQR 4–13) out of a possible 14. At least two swabs were returned 98% of the time. There was a difference in median follow-up time between index cases who met the definition of persistent MRSA colonisation and those who cleared (87·5 days (IQR 36·5–183) *vs.* 125·5 days (IQR 63–187), respectively). Similarly, those with persistent colonisation returned samples for a median 5·5 sampling episodes (IQR 3–12·5) while those who cleared colonisation returned swab samples for a median of 8 sampling periods (IQR 4–13).

Table 1. *Baseline characteristics of study population*

Variable *N* (%)	Cleared (110)	Persistent (48)	*P* value
Mean age (s.d.)	23·1 (23·6)	34·9 (24·3)	0·005
Over 18	47 (42·7)	33 (68·8)	0·003
Female	72 (65·5)	27 (56·3)	0·271
Race			
White	38 (34·6)	26 (54·2)	0·021
Black/African-American	56 (50·9)	22 (45·8)	
Hispanic/Latino	2 (1·8)	0 (0)	
Asian	2 (1·8)	0 (0)	
American Indian/Alaska Native	2 (1·8)	0 (0)	
Mixed/other	7 (6·4)	0 (0)	
No answer	3 (2·7)	0 (0)	
Hospital			0·081
HUP	27 (24·6)	18 (37·5)	
CHOP	57 (51·8)	14 (29·2)	
PPMC	12 (10·9)	6 (12·5)	
PAH	1 (0·9)	2 (4·2)	
HMC	13 (11·8)	8 (16·7)	
Medical setting			0·752
Emergency Department	74 (67·3)	35 (72·9)	
Outpatient	29 (26·4)	10 (20·8)	
Inpatient	7 (6·4)	3 (6·3)	
Comorbidities*			
Hepatic disease	5 (4·6)	3 (6·3)	0·701
Diabetes	10 (9·2)	9 (18·8)	0·090
Renal disease	1 (0·9)	4 (8·3)	0·031
Malignancy	6 (5·5)	7 (14·6)	0·057
Organ transplant	0 (0)	2 (4·2)	0·092
Prior MRSA infection†	19 (22·9)	13 (44·8)	0·024
Mean household size (s.d.)	4·3 (2·6)	3·8 (2·2)	0·243
Single-person	11 (10·0)	5 (10·4)	
Two-person	14 (12·7)	12 (25·0)	
Three-person	17 (15·5)	9 (18·8)	
Four-person	31 (28·2)	5 (10·4)	
Five-person	15 (13·6)	9 (18·8)	
>Five-person	22 (20·0)	8 (16·7)	

HUP, Hospital of the University of Pennsylvania; CHOP, Children's Hospital of Philadelphia; PPMC, Penn Presbyterian Medical Center; PAH, Pennsylvania Hospital; HMC, Penn State Milton S. Hershey Medical Center; MRSA, methicillin-resistant *Staphylococcus aureus*.

*Based on available data: 157 index cases.

† Based on available data: 112 index cases.

On bivariable analysis, index cases who met the definition of persistent colonisation were older, were more likely to be of white race, to have had a MRSA infection and to have renal disease (Table 1). Those with persistent colonisation had a higher proportion of household members colonised with MRSA at baseline with borderline statistical significance (23·5% *vs.* 11·1%; $P = 0·064$) (Table 2). Subjects with persistent colonisation were significantly more likely to be colonised at one or more sites and at all three sites than those who cleared colonisation (Table 3). There were no significant differences in the types of sites that were colonised, but these were small numbers (Table 3). Finally, those with persistent colonisation were less likely to be prescribed clindamycin as treatment for the presenting SSTI (22·9% *vs.* 60·9%, $P < 0·001$). Although subjects with persistent colonisation appeared to receive prescriptions for decolonisation agents more frequently, there was no statistical difference in prescription of these agents between the two groups. There were also no differences noted between the groups in patterns of antibiotic exposure during pre-treatment and post-treatment time periods (Table 4).

Table 2. *Measures of household member colonisation status at baseline*

Measure	Cleared (110)	Persistent (48)	*P* value
Mean (S.D.) proportion of household members colonised	11·1 (20·8)	23·5 (35·0)	0·064
Mean (S.D.) number of household members colonised	0·43 (0·84)	0·77 (1·13)	0·071
At least one household member colonised; *N* (%)	33 (30·0)	20 (41·7)	0·153

Table 3. *Number and types of sites colonised at baseline (index case)**

Sites	Cleared (110)	Persistent (48)	*P* value
Number of sites			
No sites	98 (89·9)	14 (30·4)	<0·001
One site	7 (6·4)	15 (32·6)	<0·001
Multiple sites	4 (3·7)	17 (37·0)	<0·001
All sites	0 (0)	5 (10·9)	<0·001
Type of sites†			
Nares	7 (63·6)	16 (50)	0·501
Axilla/groin	2 (18·2)	11 (34·4)	0·456
Lesion	6 (54·5)	12 (37·5)	0·323
Nares only	4 (36·4)	7 (21·8)	0·430
Axilla/groin only	0 (0)	4 (12·5)	0·558
Lesion only	3 (27·3)	4 (12·5)	0·347
Nares + axilla/groin	1 (9·1)	4 (12·5)	1·00
Nares + lesion	2 (18·2)	5 (15·6)	1·00
Axilla/groin + lesion	1 (9·1)	3 (9·4)	1·00

* Based on available data: 155 index cases.
† Proportions calculated from total positive sites (i.e. 11 in cleared group, 32 in persistent group).

In multivariable analyses (Table 5), persistent MRSA colonisation was associated with white race (adjusted OR (aOR) 4·90; 95% CI 1·38–17·40; $P = 0·014$), prior MRSA infection (aOR 3·59; 95% CI 1·05–12·35; $P = 0·042$), having multiple sites colonised (aOR 32·7; 95% CI 6·7–159·3; $P < 0·001$). Conversely, index cases who received treatment of the presenting SSTI with clindamycin were less likely to have persistent MRSA colonisation (aOR 0·28; 95% CI 0·08–0·99; $P = 0·049$). Having a higher proportion of household members colonised at baseline was not significantly associated with persistent colonisation in the multivariable model (aOR 1·01; 95% CI 0·98–1·03; $P = 0·471$). Finally, when substituting the other measures of household colonisation in the multivariable model, adjusting for white race, prior MRSA infection, multiple sites colonised and clindamycin prescription, the other measures of householdmember colonisation were not associated with persistent colonisation (total number of household members positive: aOR 1·37; 95% CI 0·65– 2·86; P = 0·407; at least one household member positive: aOR 1·49; 95% CI 0·41–5·37; P = 0·541).

Owing to the decreased number of subjects reporting on prior MRSA infection and the importance of this variable, we performed a secondary analysis using only those subjects who provided an answer to that question. This cohort comprised 112 (70·9%) subjects. Of these, 29 (25·9%) of subjects met the definition of persistent colonisation and 83 (74·1%) of subjects had clearance of colonisation. The results of these analyses were identical to those of the primary analysis (data not shown).

DISCUSSION

In this longitudinal analysis of subjects presenting with MRSA SSTI and their household members, we found that 20% of index cases remained persistently colonised at the end of the study period. We identified several risk factors for persistent colonisation, including white race, prior MRSA infection and colonisation of multiple sites. We also found that treatment with clindamycin was associated with a decreased risk of persistent colonisation.

Similar to past studies of duration of MRSA colonisation, we found that 20% of subjects screened will have persistent colonisation. However, previous studies used varying definitions of persistence and studied different populations. For example, Robicsek *et al.* evaluated hospitalised patients with previous clinical culture or surveillance culture with MRSA on readmission and determined that 48% of subjects had persistently positive surveillance cultures at 1 year and 21% at 4 years [8]. Similarly, Scanvic *et al.* also examined patients with previous positive MRSA surveillance cultures and found that 40% of those readmitted within 10 months had a subsequent positive MRSA surveillance culture [14]. Another study of hospitalised patients followed their MRSA colonisation status monthly after discharge to home health care and reported that 19% of those who had MRSA carriage in the hospital continued to be colonised at 1 year [19]. Finally, in the community setting, it has been noted that 25% of healthy high school students with MRSA colonisation will have persistent colonisation defined, in that study as $\geqslant 7$ swabs (of possible 8) over an 11-month study period

Table 4. *Antibiotic and steroid use in study population*

Drug	Cleared (110)	Persistent (48)	*P* value
Pre-treatment period			
Any antibiotic	10 (10·9)	10 (20·8)	0·097
Amoxicillin	5 (4·6)	2 (4·2)	1·00
Amoxicillin–clavulanate	1 (0·9)	2 (4·2)	0·219
Azithromycin	1 (0·9)	3 (6·3)	0·054
First generation cephalosporin	1 (0·9)	1 (2·1)	0·517
Clindamycin	7 (6·4)	0 (0)	0·102
Doxycycline	1 (0·9)	0 (0)	1·00
Fluoroquinolone	0 (0)	0 (0)	NA
Trimethoprim–sulfamethoxazole	2 (1·8)	2 (4·2)	0·585
Mupirocin	0 (0)	2 (4·2)	0·091
Bleach bath/chlorhexidine	0 (0)	1 (2·1)	0·304
Oral steroid	7 (6·4)	4 (8·3)	1·00
Nasal steroid	8 (7·3)	5 (10·4)	0·508
Treatment period			
Any antibiotic	103 (93·6)	40 (83·3)	0·042
Amoxicillin	2 (1·8)	0 (0)	1·00
Amoxicillin–clavulanate	2 (1·8)	2 (4·2)	0·585
First generation cephalosporin	4 (3·6)	4 (8·3)	0·247
Clarithromycin	2 (1·8)	1 (2·1)	1·00
Clindamycin	67 (60·9)	11 (22·9)	<0·001
Doxycycline	7 (6·4)	3 (6·3)	1·00
Fluoroquinolone	2 (1·8)	1 (2·1)	1·00
Trimethoprim–sulfamethoxazole	38 (34·6)	22 (45·8)	0·179
Mupirocin	19 (17·3)	14 (29·2)	0·091
Bleach bath/chlorhexidine	19 (17·3)	13 (27·1)	0·158
Oral steroid	2 (1·8)	2 (4·2)	0·585
Nasal steroid	6 (5·5)	4 (8·3)	0·493
Post-treatment period			
Any antibiotic	16 (14·6)	14 (29·2)	0·031
Amoxicillin	1 (0·9)	1 (2·1)	0·517
Amoxicillin–clavulanate	0 (0)	0 (0)	NA
Azithromycin	0 (0)	1 (2·1)	0·304
First generation cephalosporin	0 (0)	0 (0)	NA
Clindamycin	6 (5·5)	1 (2·1)	0·676
Doxycycline	3 (2·7)	1 (2·1)	1·00
Fluoroquinolone	1 (0·9)	3 (6·3)	0·084
Trimethoprim–sulfamethoxazole	10 (5·1)	6 (12·5)	0·065
Mupirocin	2 (1·8)	4 (8·3)	0·070
Bleach bath/chlorhexidine	2 (1·8)	3 (6·3)	0·165
Oral steroid	3 (2·7)	1 (2·1)	1·00
Nasal steroid	6 (5·5)	4 (8·3)	0·493

NA, not applicable.

[7]. Our study contributes to the current knowledge by examining the rate of persistent colonisation among community-dwelling adults and children. In addition, we focused on a clinically relevant population of patients, those with confirmed MRSA SSTI, as these are patients at higher risk of subsequent infection. Lastly, the longitudinal analysis of MRSA colonisation status allowed for a more specific determination of persistent colonisation.

Subjects with persistent MRSA colonisation have higher bacterial loads, likely resulting in higher risk of subsequent infection and transmission [10, 20]. The finding in this study that subjects with persistent colonisation were significantly more likely to report prior MRSA infection confirms the higher risk of recurrent infection in this population. The identification of factors associated with persistent colonisation is critical to identify this higher risk group of patients

Table 5. *Multivariable model of risk factors for persistent MRSA colonisation*

Variable	Odds ratio (95% CI)	*P* value
White race	4·90 (1·38–17·40)	0·014
Prior MRSA infection	3·59 (1·05–12·35)	0·042
Multiple sites colonised	32·7 (6·7–159·3)	<0·001
Treatment of SSTI with clindamycin	0·28 (0·08–0·99)	0·049
Proportion of household members colonised	1·01 (0·98–1·03)	0·471

CI, confidence interval; MRSA, methicillin-resistant *Staphylococcus aureus*; SSTI, skin and soft tissue infections.

and to determine modifiable factors to decrease the risk of persistent colonisation and, ultimately, recurrent infection and/or transmission to others.

White race has been found to be associated with carriage of *S. aureus* [21] and specifically MRSA [22] in other studies as well. The reasons for this finding are unclear. However, given that white race has been found to be associated with *S. aureus* colonisation in multiple studies suggests that there may be genetic differences in hosts that determine risk of colonisation and persistent colonisation. These host differences should be further studied.

The site colonised plays a role in persistence. An increased number of colonised sites was associated with persistent colonisation in the present study as well as in a prior study evaluating the factors associated with persistent MRSA carriage in subjects who participated in a clinical trial of mupirocin for eradication of nasal carriage of MRSA [23]. The number of subjects with colonisation at each type of site was too small to be able to distinguish differences between site types in this study. However, it has previously been shown that colonisation of the rectum in addition to the nares is likely associated with persistence of MRSA colonisation [24]. Rectal swabs were deemed infeasible for self-collection in our study, but this association should be further explored. Identification of patients with multiple sites colonised will help target decolonisation efforts toward those at highest risk. Furthermore, more directed decolonisation (i.e. mupirocin or retapamulin) beyond anti-septic washes and dilute bleach baths at specific sites other than the nares may be useful and should be evaluated.

We found that presence of an increased proportion of household members colonised with MRSA was not associated with persistent colonisation in index cases. The role of colonised household members in transmission [25–27], duration [9, 28] and recurrence [15] of MRSA colonisation has become increasingly clear. However, it does not seem to play an important role in persistent colonisation. This highlights that patients with persistent colonisation are distinct from those with intermittent colonisation and the factors associated with persistence may be more related to host differences rather than strain/environmental factors.

Treatment of the initial MRSA SSTI with clindamycin was significantly associated with decreased risk of persistent MRSA colonisation. We found the same association between clindamycin and earlier clearance of MRSA colonisation [9] as well as decreased risk of recurrent MRSA colonisation [15]. One small study noted that patients with staphylococcal skin infections treated with clindamycin for 3 months resulted in recurrent abscesses in 2 of 11 patients as compared with 7 of 11 patients who received placebo [29]. Clindamycin has been included as part of decolonisation protocols, with high success rates for eradication [30, 31]. Other agents with activity against MRSA, such as trimethoprim–sulfamethoxazole and doxycycline also have resulted in similar eradication rates [32]. However, these bundles included multiple components, including topical decolonisation agents, so the roles of specific components are not clear. Our study did not find an association between other MRSA-active antibiotics and persistent colonisation, but there may not have been sufficient power to study specific agents (e.g. doxycycline). Future studies are needed to confirm this effect of clindamycin and its potential use for decreasing the burden of MRSA colonisation and recurrent infections.

This study has several potential limitations. Recall bias is a concern because some data were obtained by self-report from the subjects. This most likely affected the ascertainment of prior antibiotic use and use of decolonisation methods, such as mupirocin or chlorhexidine. However, these data were confirmed using medical record review. Furthermore, potential interviewer bias was minimised by using a structured data abstraction form completed by study team members who were blinded to the subject's colonisation status. Index cases may have been misclassified in terms of clearance or persistence of colonisation. However, defining clearance of colonisation as all samples negative for two consecutive sampling periods decreased the possibility that we were missing true clearance of colonisation. As this is an observational

study, there may be unmeasured confounders that could account for the findings of this study. Also, other household and community factors not assessed in this study, such as home surface contamination and pet carriage with MRSA, which have been shown to be associated with MRSA transmission within households [33]. In addition, rates and patterns of antibiotic resistance may vary across regions and this variation may result in differences in the distribution of risk factors. Nevertheless, this study was conducted at multiple sites comprising a geographically, racially and ethnically diverse population of both adults and children, which should improve the generalisability of these findings.

In summary, we found that 20% of subjects who initially presented with MRSA SSTI had persistent MRSA colonisation at the end of the study period. White race, prior MRSA infection and colonisation at multiple sites were associated with increased risk of persistent MRSA colonisation, while treatment of the MRSA SSTI with clindamycin was associated with a decreased risk of persistent colonisation. Future studies should evaluate the molecular epidemiology of MRSA to identify strain-specific factors that lead to persistence and could inform future interventions. Additionally, evaluation of host-specific genetic or immune factors that are associated with persistent colonisation is needed. Finally, the association between clindamycin and decreased risk of persistent colonisation needs to be clarified and its potential role in decolonisation efforts should be examined.

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DECLARATION OF INTEREST

None.

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