Community-associated methicillin-resistant *Staphylococcus aureus* skin infections in a religious community

F. CORONADO[1,3]*, J. A. NICHOLAS[3], B. J. WALLACE[3], D. J.
KOHLERSCHMIDT[4], K. MUSSER[4], D. J. SCHOONMAKER-BOPP[4], S. M.
ZIMMERMAN[5], A. R. BOLLER[5], D. B. JERNIGAN[2] AND M. A. KACICA[3]

[1] *Epidemic Intelligence Service, and* [2] *National Center for Infectious Diseases, Centers for Disease Control
and Prevention, Atlanta, GA, USA*
[3] *Bureau of Communicable Disease Control, New York State Department of Health, NY, USA*
[4] *Wadsworth Center, New York State Department of Health, NY, USA*
[5] *Esopus Medical, NY, USA*

SUMMARY

In September 2004, an outbreak of community-associated methicillin-resistant *Staphylococcus
aureus* (MRSA) skin and soft tissue infections (SSTI) was reported among members of a religious
community. We conducted a retrospective cohort study on all 175 community members;
performed a nasal carriage survey, and environmental swab testing. We identified 24 MRSA cases
(attack rate 14%). In multivariate analysis, sauna use [odds ratio (OR) 19·1, 95% confidence
interval (CI) 2·7–206·1] and antimicrobial use within 12 months before infection (OR 11·7, 95%
CI 2·9–47·6) were risk factors for infection. MRSA nasal carriage rate was 0·6% (1/174). Nine of
10 clinical isolates and an isolate from an administrative office within the community had the
pulsed-field gel electrophoresis type USA300. Targeted hygiene improvement, wound care, and
environmental cleaning were implemented. We describe the first reported outbreak of MRSA
SSTI in a religious community. Adherence to appropriate personal and environmental hygiene
might be critical factors in controlling transmission.

INTRODUCTION

Methicillin-resistant *Staphylococcus aureus* (MRSA)
has been increasingly identified as a cause of disease
outbreaks in community settings. Although initial
reports of community-onset MRSA disease were
predominantly associated with health care-associated
strains (HA-MRSA), distinct community strains are
now commonly causing disease in regions around the
world [1, 2]. Outbreaks of community-associated
MRSA (CA-MRSA) have been reported in non-
hospitalsettings (e.g. correctional facilities [3], long-
term care institutions [4], Alaskan native and Pacific
Islander communities [5, 6], among military recruits
[7], close-contact sports participants [8], and a local
delicatessen [9]). Outbreaks of CA-MRSA have
occurred among otherwise healthy persons living in a
community and were not associated with traditional
HA-MRSA risk factors (i.e. contact with health-care
facilities) [2, 10, 11]. More recently, previous anti-
microbial use, determined to be a risk factor for HA-
MRSA, might possibly be associated with CA-MRSA

* Author for correspondence: F. Coronado, M.D., M.P.H.,
Centers for Disease Control and Prevention, 1600 Clifton Rd,
NE MS E-92, Atlanta, GA 30333, USA.
(Email: fcoronado@cdc.gov)

infections as well [12]. CA-MRSA strains can cause skin and soft tissue infections (SSTI) among susceptible hosts; additional virulence factors associated with more severe disease have also been reported [13, 14]. In addition, certain strains are reaching a high level of endemicity and are even emerging as causes of health care-associated infection [15].

CA-MRSA strains can often be distinguished from HA-MRSA on the basis of preserved susceptibility to non-β-lactam antimicrobials and by distinct genotypic features [2]. These community strains typically are resistant to β-lactam antimicrobials by virtue of the *mec*-type IV resistance staphylococcal-cassette-chromosome (SCC); in contrast, HA-MRSA strains normally carry different types of *SCCmec* elements, which often code for additional antimicrobial resistance [1, 16]. CA-MRSA strains can also code for unique toxins [e.g. Panton–Valentine leukocidin (PVL) not typically present in hospital strains] [1, 17]. Molecular typing methods enable expanded case finding and more explicit characterization of the pathogen.

In September 2004, the New York State Department of Health (NYSDOH) and the Centers for Disease Control and Prevention (CDC) were notified of an increase in the number of SSTIs reported among otherwise healthy members of a religious community located in rural upstate New York. From 1 May to early September 2004, 27 persons had been diagnosed with a SSTI at the community's health centre. Only seven had their wounds cultured; MRSA was isolated from all seven. NYSDOH initiated an investigation to describe the scope of the outbreak, identify the source and the risk factors for infection, and implement community-wide infection-control and prevention measures.

Community features

The community is part of a regional and international network of faith-based communities. Approximately 2500 persons reside in 12 communities in the United States, Germany, and Australia, all sharing a philosophy of communal living, close proximity, and sharing. The community experiencing the outbreak described in this report includes 175 full-time residents. Families live in a rural setting in one of six multifamily dwellings; bathrooms and kitchens are shared among several families. Personal hygiene items (e.g. nailbrushes, combs) are frequently shared among family members. Most meals are taken in a communal dining hall. Employment occurs primarily within the community and includes office and manufacturing jobs, child care, and teaching. Primary medical care is provided by two community member clinicians on-site. Children attend grade school within the community; most complete their secondary education at a local public high school. Clothing, towels, and linens from all families are laundered together weekly in a centralized on-site industrial laundry. Recreational activities include use of an outdoor swimming pool, year-round use of a community sauna, and fishing and occasional swimming in a local lake.

METHODS

Case finding

A confirmed CA-MRSA case was defined as a SSTI with culture-confirmed MRSA in a community member during the outbreak period of 1 May–30 November 2004. A probable case was defined as a SSTI during the outbreak period that required incision and drainage (I&D) but for which no culture was collected. Case-finding was accomplished through active surveillance by the community's clinicians, medical record review, and community outreach and education, encouraging residents to report any skin lesions. All medical evaluations, procedures, and treatment occurred at the community's clinic.

Cohort study

To identify risk factors for infection, we conducted a retrospective cohort study on community members. We designed a standard data-collection questionnaire and collected information on demographic characteristics, hygiene practices, contact with persons with skin infections, management of skin lesions, use of recreational facility activities, health-care exposures, and antimicrobial use during the 12 months preceding the outbreak period. Antimicrobial use was defined as the receipt of systemic antimicrobials from the on-site community clinic in the 12 months preceding the diagnosis of SSTI in an individual patient. The questionnaire was self-administered at the end of the outbreak period. Questionnaires for children aged <12 years were completed by their parents. Data were entered into a Microsoft Access database. Univariate relative risks were analysed by using the SAS statistical package (SAS Institute Inc., Cary, NC, USA),

with the primary outcome of interest defined as confirmed or probable MRSA case status. χ^2 ($P < 0.05$) was used to identify risk factors on the univariate analyses. Exposures associated with illness on the univariate analyses were investigated by using logistic regression.

Laboratory study

Patient isolates and nasal carriage study

A total of 17 patients had confirmed MRSA isolates during the outbreak period. Ten primary swabs were obtained from SSTIs of community members and processed at the NYSDOH laboratories. Prior to the NYSDOH investigation, all MRSA-positive specimens were processed at a private laboratory. One additional MRSA isolate was obtained after the outbreak period. Specimen sources included cultures from hip, thigh, knee, foot, hand, elbow, nose, and perirectal areas. In addition, we conducted a nasal carriage survey by obtaining anterior nares swabs from community members. Specimen collection was conducted at the end of November 2004. An MRSA carrier was defined as an asymptomatic community member with positive MRSA nasal culture.

Environmental study

To identify any environmental sources of exposure, we sampled selected surfaces and shared items in recreational and other communal areas. Environmental sites for sampling were selected based on hypothesized mechanisms for transmission within the community (e.g. shared places: sauna). Pre-moistened polyester swabs and wipes were used for surface sample collection [18]. All environmental sampling was conducted after infection-control procedures described in the following were in place.

Laboratory tests

Patient isolates evaluated by NYSDOH laboratories were cultured by using trypticase soy agar plates with 5% sheep's blood. Environmental samples were cultured by using Baird–Parker agar, mannitol salt agar containing 4μg/ml oxacillin, and a selective enrichment broth containing 10% NaCl and 1% sodium pyruvate. Plates were examined for colonies resembling *S. aureus*. Identification of *S. aureus* was confirmed by Gram stain, catalase, coagulase and

AccuProbe® (Gen-Probe Inc., San Diego, CA, USA) testing and *nuc* gene detection using polymerase chain reaction (PCR) [19]. Methicillin resistance was confirmed by oxacillin disk diffusion and E-test® (AB Biodisk, Solna, Sweden) MIC testing, according to interpretive criteria of the Clinical and Laboratory Standards Institute (formerly the National Committee for Clinical Laboratory Standards), and by *mec*A gene detection by using PCR. Thirteen MRSA isolates from 11 patients and two environmental isolates were analysed by pulsed-field gel electrophoresis (PFGE) [20]. Fragments were separated using a linear pulse ramp of 5–50 s over 20 h.

Outbreak control interventions

Prior to the involvement of NYSDOH health officials in September 2004, the community had instituted infection-control measures focused on enhancing personal hygiene and limiting the sharing of personal items (e.g. sauna, pool towels). Chlorhexidine showers were prescribed for case-patients with active infections and for their direct contacts and family members; case-patients' homes were regularly and meticulously cleaned with disinfectant solutions. Additionally, use of antibacterial soap and disposable hand towels were instituted community-wide, and alcohol-based hand cleaners were made available in public areas and case-patients' homes. The sauna was cleaned 2–3 times a week with a chlorine bleach solution. Because most community members have no health insurance, only families with the highest incidence of MRSA infection were offered intranasal mupirocin by the on-site clinicians following previously reported recommendations [21]. Early in the outbreak, wound care and dressing changes were performed by case-patients or their families at home. Later in the outbreak, dressing changes were performed in the community's health clinic by healthcare staff.

After the involvement of NYSDOH officials, the sauna was closed to all use in October 2004.

RESULTS

Characteristics of case-patients

During the outbreak period, we identified 17 confirmed and seven probable case-patients among 175 residents (attack rate 14%), representing a threefold increase in the baseline monthly frequency of SSTIs

Table 1. *Demographic characteristics of members of a religious community including methicillin-resistant* Staphylococcus aureus *patients, 2004*

	Community overall	MRSA case-patients	Non-cases
No. of members (%)	175 (100)	24 (14)	151 (86)
Male (%)	83 (47)	17 (71)	66 (44)
Median age (range)	21 (1–82 yr)	13 (2–54 yr)	23 (1–82 yr)
Underlying illnesses (%)	Unknown	1* (4)	Unknown
Median number of families per household (range)	5 (2–7)	6 (2–7)	5 (2–7)
Median number of persons per family (range)	5 (1–10)	7 (1–10)	4 (1–10)
Median number of persons per bedroom (range)	2 (1–6)	3 (2–6)	2 (1–6)

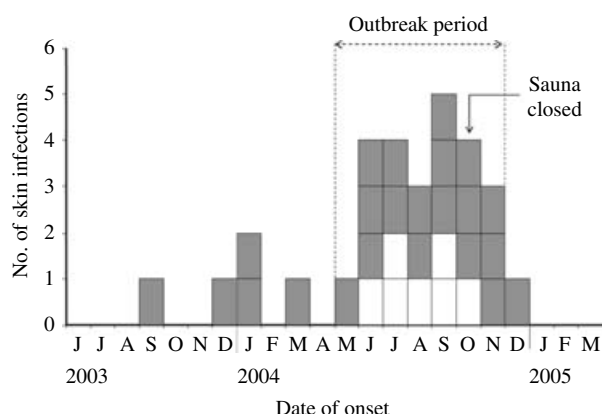* Refers to the patient with chronic folliculitis on the neck and scalp.

Fig. 1. Date of onset of MRSA SSTI among members of a religious community. □, Probable case; ■, confirmed case.

Fig. 2. Illustration of an abscess present on a case-patient's forearm.

reported before May 2004 (Fig. 1). Twenty-two other clinically diagnosed SSTIs not requiring I&D were also identified but are not represented in the figure. The median age of case-patients was 13 years (range 2–54 years); 17 (71%) were male. Crowded living conditions were also observed among case-patients (Table 1). All confirmed and probable case-patients presented clinically with abscesses (Fig. 2), 18 (75%) with lesions located below the waist. Lesions typically appeared initially as a deep, erythematous, tender nodule. Within the first 24 h of appearance, the majority of lesions were painful, typically out of proportion to the soft-tissue changes. By 48 h, a clearly defined deep abscess, ~4 mm below the skin surface formed and was amenable to I&D. A clinician performed I&D on 15 of 17 confirmed case-patients. No hospitalizations were required. Nineteen case-patients (79%) received antimicrobial agents (e.g. penicillin, fluoroquinolone, cephalosporin and macrolides) during the 12 months preceding a MRSA SSTI for

indications including otitis media, tonsillitis, Lyme disease, and upper respiratory infection. One case-patient reported extensive chronic folliculitis on the neck and scalp and had required surgical treatment and multiple courses of antimicrobials during the previous 10 years. As for community SSTIs that occurred before the defined outbreak period, all five patients who required I&D also had cultures that yielded MRSA. Antimicrobial agents used for treatment of MRSA infection included: amoxicillin and clavulanate; ciprofloxacin; dicloxacillin; trimethoprim–sulfamethoxazole; vancomycin and clindamycin; combination of tetracycline with fluoroquinolones such as levofloxacin, gatifloxacin, and ciprofloxacine; combination of trimethoprim–sulfamethoxazole and rifampin; and combination of levofloxacin and rifampin. The last MRSA-positive

Table 2. *Univariate and multivariate analyses of risk factors for methicillin-resistant* Staphylococcus aureus *infection among members of a religious community, 2004*

Risk factor or characteristic	Cases (n=24)	Non-cases (n=151)	AR %	RR (univariate analyses)	Multivariate analyses		
					OR	95% CI	P value
Age, ≤15 years	14	51	22	2·4	0·4	0·07–2·5	0·30
Male sex	17	66	20	2·7	2·4	0·6–9·4	0·20
≥5 persons in family	15	54	22	2·8	2·8	0·6–13·2	0·21
Used antimicrobials within 12 months before infection	**19**	**50**	**28**	**5·2**	**11·7**	**2·9–47·6**	**0·0006**
Shared clothes with family members	8	26	24	2·2	3·1	0·6–16·5	0·18
Shared hand creams	17	70	20	2·4	2·7	0·6–11·8	0·16
Shared towels with other community members	14	49	22	2·7	2·9	0·8–10·8	0·10
Shared razor/clippers	15	60	20	2·2	0·6	0·1–2·6	0·53
Used pool	24	126	16	Undef.	Undef.	Undef.	1·0
Contact with water in lake	10	24	29	3·0	2·7	0·5–15·8	0·26
Used sauna	**23**	**86**	**21**	**14·0**	**19·1**	**2·7–206·1**	**0·001**

AR, Attack rate; RR, risk ratio; OR, odds ratio; CI, confidence interval; Undef., undefined or incalculable value.
* Values in **bold** are significant ($P<0.05$).

skin infection was reported in December 2004 in a girl aged 2 years.

Cohort study

All 175 (100%) community members were enrolled in the study. On univariate analysis, the following characteristics were identified as potential risk factors for confirmed or probable CA-MRSA ($P<0.05$): male sex; age <15 years (cut-off defined because in this community, members of high-school age entered local public high schools in their community, and have a different risk factor profile than community dependants under the age of 15 years); antimicrobial use within 12 months of infection; >5 family members (cut-off defined as median number of family members in each household); sharing towels, clothes, hand creams, and razors or clippers; sauna or pool use; and contact with lake water. After the multivariate analysis, only sauna use [odds ratio (OR) 19·1, 95% confidence interval (CI) 2·7–206·1) and antimicrobial use within 12 months before infection (OR 11·7, 95% CI 2·9–47·6) remained significant risk factors for CA-MRSA infection (Table 2). Among sauna users, frequent sauna use (more than once a week) was an additional risk factor for CA-MRSA infection (OR 2·3, 95% CI 1·1–4·5). In addition, case-patients were three times as likely to recall having used the sauna with a pre-existing cut or abrasion than those who did not become ill (OR 3·2, 95% CI 1·5–6·9).

Restricting the analysis to confirmed cases yielded similar statistically significant findings.

Laboratory study results

A total of 17 case-patients were confirmed MRSA during the outbreak period. Wound isolates were available to NYSDOH laboratory for 11 confirmed case-patients; one of those outside the described outbreak period. All were *mec*A-positive by PCR. Susceptibility testing of the isolates indicated that they were resistant to oxacillin and erythromycin. Nine of these isolates were indistinguishable by PFGE and belonged to the PFGE type USA300, a strain that falls within the ST8 grouping by multilocus sequence typing [22]. One patient's isolate differed by less than three bands when compared to the outbreak strain, which indicate they are closely related.

Nasal carriage study results

Of 175 residents, 174 (99%) had a nasal swab taken for culture. Of those, 36 (21%) yielded *S. aureus*. Thirty-five of these nasal isolates were susceptible to oxacillin by disk diffusion. One was resistant to oxacillin and was positive for *mec*A (overall community MRSA carriage rate of 0·6%). The PFGE pattern of the MRSA isolate was indistinguishable from the pattern of the outbreak strain (USA300).
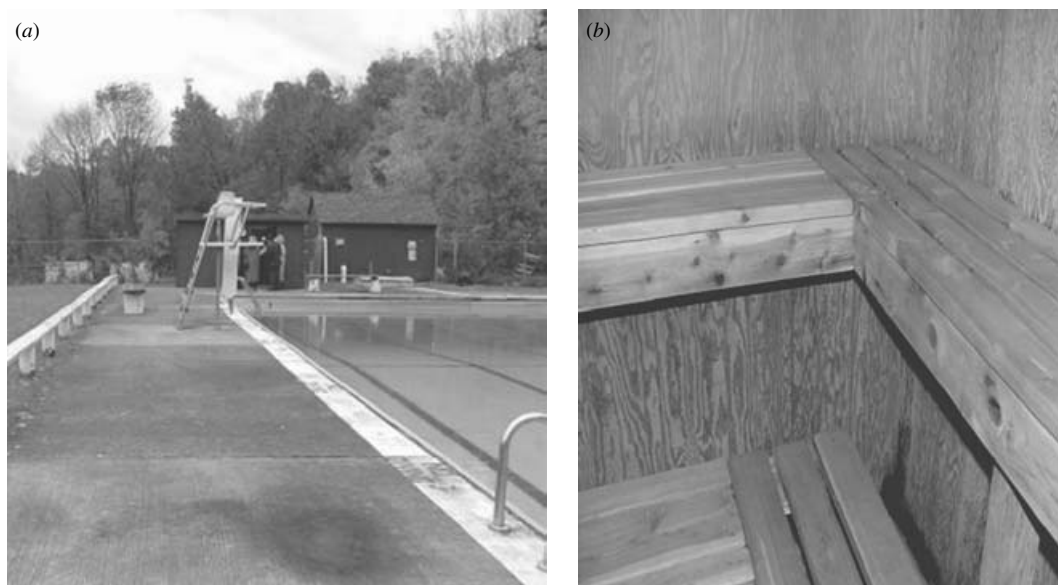
Fig. 3. Photograph of the community's swimming pool and sauna area (*a*) and benches at the sauna (*b*).

Environmental study results

A total of 47 environmental samples were obtained and analysed. Samples were collected from the sauna (Fig. 3); swimming pool area; communal kitchens; bathrooms; certain household items (e.g. hand creams and nailbrushes); office computers; keyboards and telephones; and environmental samples of lake, pool, and sauna water. Wood samples from the sauna area, changing area, and a perimeter boardwalk were also collected. Two environmental samples exhibited growth of MRSA. One *mec*A-positive, oxacillin-resistant isolate was cultured from an administrative office's computer keyboard used by a co-worker of the case-patient with chronic folliculitis. The MRSA strain of this environmental sample was identical to the MRSA outbreak strain (USA300). The second MRSA isolate was obtained from a sauna bench. This isolate was *mec*A-negative but was resistant to oxacillin by disk diffusion and the E-test MIC method. The strain differed by more than three PFGE bands from the outbreak strain and was classified as USA400 lineage. Eleven other environmental samples yielded *S. aureus* that were *mec*A-negative and oxacillin-susceptible. These methicillin-sensitive *S. aureus* (MSSA) isolates were obtained from the sauna area, including benches, floor, changing room, door handle, and walls. Thirty-four of the environmental samples exhibited no growth of *S. aureus*. These included samples from bathroom facilities, the swimming pool, and lake water.

DISCUSSION

We investigated an outbreak of skin abscesses, caused by an emerging community clone of MRSA, among members of a communal religious group. This is the first reported outbreak of MRSA SSTIs in such a setting. Personal use of antimicrobials agents in the preceding 12 months and use of the community's sauna were associated with disease. Transmission of infection ended after closure of the sauna, environmental cleaning and targeted treatment and decolonization of case-patients and their contacts. Our investigation indicates that previous personal antimicrobial use was strongly associated with subsequent MRSA infection among the members of this community. Although this finding has been well documented in cases of HA-MRSA [23], and more recently as a risk factor for MRSA colonization [24, 25], personal antimicrobial use might also be a risk factor for acquisition of a circulating CA-MRSA strain as well [5, 17]. Although community-wide antimicrobial use can be expected to promote the emergence of resistant community strains, personal antimicrobial use may confer a specific risk to a person, through disruption of normal competing bacterial flora, altered host defences, or promotion of colonization by resistant staphylococcal strains [26]. *In vitro* evidence demonstrates that under specific circumstances, antimicrobials can increase the virulence of selected microbial pathogens by enhancing microbial virulence adhesion factors and toxin

production [27, 28]. At a minimum, antimicrobial prescribing patterns have been linked to the carriage of resistant staphylococci by patients and their contacts [29]. Although the relationship between antimicrobial use and MRSA is intricate [30], our study demonstrates that both community and personal level health reasons require using antimicrobials judiciously in community settings.

Community and environmental factors probably played a substantial role in the extent and persistence of this outbreak. Previous community outbreaks have been described in other communal settings, including prisons [3], military barracks [7, 25], and sports facilities [8], and often ascribed to skin-to-skin contact and breakdown in personal hygiene. Community life among this cohort provided multiple opportunities for close contact. Common areas are shared, and shared resources and personal items (e.g. nailbrushes, hand creams, and laundry facilities) place community members at increased risk for infection. As in other reports [12, 31, 32], compromised skin was a risk factor for experiencing MRSA infection, underscoring the importance of promoting personal hygiene and appropriate wound care. Community educational programmes regarding the importance of personal hygiene with a focus on correct hand-washing techniques, and environmental cleaning were strongly emphasized in this community by the community clinicians upon outbreak recognition. Standardized skin infection treatment measures were also instituted at that time, including a mechanism to prevent transmission of MRSA during dressing changes. Interestingly, MRSA transmission continued after the institution of these interventions with which the community appeared remarkably compliant. In other studies, lack of hygiene has been a consideration for MRSA infection [12, 32]; however, in this community, ongoing MRSA transmission could not be attributed to substantial lack of personal hygiene. Close proximity among its members made possible frequent contact with items and surfaces that had been recently touched by case-patients. This characteristic feature of community life for these persons necessitated vigilant adherence to recommended interventions to prevent the spread of infection.

Collection of environmental samples was conducted after extensive community-wide cleaning (particularly in the sauna), which may have limited our capacity to identify the outbreak strain. However, the presence of the outbreak strain on an office computer keyboard and of another MRSA strain in the sauna area indicates that the environment may have played a key role in the ongoing transmission of MRSA among community members. In addition, the presence of MSSA in some of the areas after environmental cleaning suggests a limited ability to completely eradicate S. aureus strains from environmental sites. Although substantial environmental reservoirs of MRSA are not thought to be common, computer terminals have been reported to harbour MRSA [33, 34], as have blood pressure cuffs, showers [35], and furniture [36], In addition, sauna use has been associated previously with MRSA infection in rural Alaska [5, 17]. Certain communal living factors such as sauna use, crowding (e.g. shared bedrooms and bathrooms), and sharing of potentially contaminated surfaces and items (e.g. sauna benches or towels, particularly by children) probably contributed to the transmission of MRSA and high morbidity in the community. This particular sauna was constructed from a combination of materials, with plywood walls, cedar wood benches, and an unfinished concrete floor. The porous nature of these materials and the tendency to retain moisture may have made them particularly vulnerable to persistent bacterial colonization. Other identified potential contributing factors to sauna infection included failure to shower either before or after sauna use, group use by school-aged children, and failure to place seating barriers on the benches (e.g. clean towels). The lower extremity distribution of SSTI on case-patients is consistent with the conclusion that the sauna bench was a major source and mode of MRSA transmission.

Colonization swab surveys have been used in other MRSA investigations; however, data are insufficient to recommend their routine use in controlling outbreaks. In addition, data from randomized controlled trials are too limited to conclude that decolonization regimens are effective in limiting an outbreak of MRSA. The yield of colonization studies varies widely in different published reports. In investigations among football players, findings have ranged from no detection of MRSA nasal carriage [12, 31] to MRSA carriage rates as high as 8% [32]. In our investigation, we determined that 20% of community members carried MSSA, but only one carried MRSA. The colonization swab survey was conducted at the end of the outbreak period after the implementation of infection-control measures, which may have limited MRSA transmission and colonization in the community. Only anterior nares swabs were performed;

MRSA carriage at other sites is also possible. A community-wide decolonization regimen was not pursued; other combined standard infection-control interventions (e.g. chlorhexidine showers, disposable towels, and frequent hand washing) were chosen and appear to have been successful at stopping transmission.

The PFGE pattern USA300 is an emerging lineage associated with CA-MRSA outbreaks and sporadic cases throughout the United States, indicating that it may be widely distributed [12]. This clone may be more likely to lead to clinical infection, particularly to SSTIs, that can be explained by the production of the PVL toxin [24], a cytotoxin also identified in certain MSSA strains. *S. aureus* strains that produce PVL have been associated with necrotic skin lesions and severe necrotizing pneumonia, although the relationship between this toxin and risk for severity of clinical disease has not been fully characterized [14, 37]. The USA300 and USA400 lineages, which are unrelated by PFGE, appear to be responsible for the majority of CA-MRSA SSTIs in the United States [22]. From our investigation, it appears possible that the patient with chronic folliculitis served as the community reservoir for this USA300 strain because of frequent antimicrobial use and a chronically infected skin lesion. Although the antimicrobial susceptibility profile obtained from this case-patient before the outbreak investigation matched to other case-patients, we were unable to obtain a specimen from him during the outbreak and therefore unable to determine if the PFGE pattern of his potential isolate was consistent with the outbreak strain. However, cultures from multiple members of this case-patient's family did indeed have the outbreak strain.

Because certain outbreak control measures were enacted concurrently, some before our investigation, the termination of this outbreak cannot be attributed to any one specific intervention. Multiple strategies were employed, including closing the sauna, treating affected community members with systemic antimicrobials, decolonization with nasal mupirocin in clinically affected members, applying skin antiseptics to case-patients, enhancing community hygiene and awareness, and increasing surveillance for SSTIs among community members. Past studies have not been designed to quantify the independent benefits of individual interventions (e.g. decolonizing agents or infection control measures); further studies are needed for development of evidence-based strategies for intervention. Community commitment to adhering to recommended measures was impressive, and was an important factor to outbreak resolution. Our findings indicate that the outbreak was terminated after interventions that did not require community-wide decolonization strategies (e.g. chlorohexidine body washes or intranasal mupirocin for all members of the cohort). Further surveillance will be pursued to detect any re-emergence of MRSA in this community.

In addition to the limitations noted previously, other limitations might have affected our findings. Cultures were not performed by clinicians on all skin infections because of the relative cost to this uninsured community. Not all MRSA isolates were available for PFGE, because certain early isolates were processed at a private laboratory and subsequently discarded. Recall bias, particularly as it relates to self-reported risk factors (e.g. recall of compromised skin integrity prior to infection), probably interferes with the accuracy of some of our findings. Finally, limited or quickly resolving skin lesions were less likely to be reported. These factors suggested that the number of true case-patients is likely to be larger than the numbers we report here.

Our investigation documented the termination of transmission of CA-MRSA within a semi-closed religious community. Health-care providers should consider the emergence of CA-MRSA when evaluating SSTIs in community settings. Additional targeted studies are needed to identify the best approaches for control of CA-MRSA. To better characterize the epidemiology and prevention of CA-MRSA, New York State, in collaboration with CDC and other public health partners, is participating in active population-based surveillance for invasive MRSA in eight geographic locations in the United States [38]. In addition, guidelines and educational materials for prevention of MRSA infections have been developed for specific populations (e.g. athletic trainers and those providing care in correctional facilities) [3, 12].

DECLARATION OF INTEREST

None.

REFERENCES

1. **Vandenesch F, et al.** Community-acquired methicillin-resistant *Staphylococcus aureus* carrying Panton-Valentine leukocidin genes: worldwide emergence. *Emerging Infectious Diseases* 2003; **9**: 978–984.

2. **Chambers HF.** The changing epidemiology of *Staphylococcus aureus*? *Emerging Infectious Diseases* 2001; **7**: 178–181.

3. **Centers for Disease Control and Prevention.** Methicillin-resistant *Staphylococcus aureus* infections in correctional facilities – Georgia, California and Texas, 2001–2003. *Morbidity and Mortality Weekly Report* 2003; **52**: 992–996.

4. **Borer A, et al.** Community-acquired methicillin-resistant *Staphylococcus aureus* in institutionalized adults with developmental disabilities. *Emerging Infectious Diseases* 2002; **8**: 966–970.

5. **Baggett HC, et al.** An outbreak of community-onset methicillin-resistant *Staphylococcus aureus* skin infections in southwestern Alaska. *Infection Control & Hospital Epidemiology* 2003; **24**: 397–402.

6. **Centers for Disease Control and Prevention.** Community-associated methicillin-resistant *Staphylococcus aureus* infections in Pacific Islanders – Hawaii, 2001–2003. *Morbidity and Mortality Weekly Report* 2004; **53**: 767–770.

7. **Zinderman CE, et al.** Community-acquired methicillin-resistant *Staphylococcus aureus* among military recruits. *Emerging Infectious Diseases* 2004; **10**: 941–944.

8. **Centers for Disease Control and Prevention.** Methicillin-resistant *Staphylococcus aureus* infections among competitive sports participant – Colorado, Indiana, Pennsylvania, and Los Angeles County, 2000–2003. *Morbidity and Mortality Weekly Report* 2003; **52**: 793–795.

9. **Jones TF, et al.** An outbreak of community-acquired illness caused by methicillin-resistant *Staphylococcus aureus*. *Emerging Infectious Diseases* 2002; **8**: 82–84.

10. **Moreno F, et al.** Methicillin-resistant *Staphylococcus aureus* as a community organism. *Clinical Infectious Diseases* 1995; **21**: 1308–1312.

11. **Herold BC, et al.** Community-acquired methicillin-resistant *Staphylococcus aureus* in children with no identified predisposing risk. *Journal of the American Medical Association* 1998; **279**: 593–598.

12. **Kazakova SA, et al.** A clone of methicillin-resistant *Staphylococcus aureus* among professional football players. *New England Journal of Medicine* 2005; **352**: 468–475.

13. **Baba T, et al.** Genome and virulence determinants of high virulence community-acquired MRSA. *Lancet* 2002; **359**: 1819–1827.

14. **Gillet Y, et al.** Association between *Staphylococcus aureus* strains carrying gene for Panton-Valentine leukocidin and highly lethal necrotising pneumonia in young immunocompetent patients. *Lancet* 2002; **359**: 753–759.

15. **Saiman L, et al.** Hospital transmission of community-acquired methicillin-resistant *Staphylococcus aureus* among postpartum women. *Clinical Infectious Diseases* 2003; **37**: 1313–1319.

16. **Carleton HA, et al.** Community-adapted methicillin-resistant *Staphylococcus aureus* (MRSA): population dynamics of an expanding community reservoir of MRSA. *Journal of Infectious Diseases* 2004; **190**: 1730–1738.

17. **Baggett HC, et al.** Community-onset methicillin-resistant *Staphylococcus aureus* associated with antibiotic use and the cytotoxin Panton-Valentine leukocidin during a furunculosis outbreak in rural Alaska. *Journal of Infectious Diseases* 2004; **189**: 1565–1573.

18. **Sanderson WT, et al.** Surface sampling methods for *Bacillus anthracis* spore contamination. *Emerging Infectious Diseases* 2002; **8**: 1145–1151.

19. **Tenover F, et al.** Interpreting chromosomal DNA restriction patterns produced by pulsed-field gel electrophoresis: criteria for bacterial strain typing. *Journal of Clinical Microbiology* 1995; **33**: 2233–2239.

20. **Centers for Disease Control and Prevention.** PFGE plug procedure: short form, *Staphylococcus* species. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention, 2000.

21. **Laupland KB, Conly JM.** Treatment of *Staphylococcus aureus* colonization and prophylaxis for infection with topical intranasal mupirocin: an evidence-based review. *Clinical Infectious Diseases* 2003; **37**: 933–938.

22. **McDougal LK, et al.** Pulse-field gel electrophoresis typing of oxacillin-resistant *Staphylococcus aureus* isolates from the United States: establishing a national database. *Journal of Clinical Microbiology* 2003; **41**: 5113–5120.

23. **Graffunder EM, Venezia RA.** Risk factors associated with nosocomial methicillin-resistant *Staphylococcus aureus* (MRSA) infection including previous use of antimicrobials. *Journal of Antimicrobial Chemotherapy* 2002; **49**: 999–1005.

24. **Hidron AI, et al.** Risk factors for colonization with methicillin-resistant *Staphylococcus aureus* (MRSA) in patients admitted to an urban hospital: emergence of community-associated MRSA nasal carriage. *Clinical Infectious Diseases* 2005; **41**: 159–166.

25. **Ellis ME, et al.** Natural history of community-acquired methicillin-resistant *Staphylococcus aureus* colonization and infection in soldiers. *Clinical Infectious Diseases* 2004; **39**: 971–979.

26. **Dancer SJ.** How antibiotics can make us sick: the less obvious adverse effects of antimicrobial chemotherapy. *Lancet Infectious Diseases* 2004; **4**: 611–619.

27. **Bisognano C, et al.** Increased expression of fibronectin-binding proteins by fluoroquinolone-resistant *Staphylococcus aureus* exposed to subinhibitory levels of ciprofloxin. *Antimicrobial Agents Chemotherapy* 1997; **41**: 906–913.

28. **Ohlsen K, et al.** Effects of subinhibitory concentrations of antibiotics on alpha-toxin (*hla*) gene expression of methicillin-sensitive and methicillin-resistant *Staphylococcus aureus* isolates. *Antimicrobial Agents Chemotherapy* 1998; **42**: 2817–2823.

29. **Miller YW, et al.** Sequential antibiotic therapy for acne promotes the carriage of resistant staphylococci on the skin of contacts. *Journal Antimicrobial Chemotherapy* 1996; **38**: 829–837.

30. **Naimi TA, et al.** Comparison of community- and health care-associated methicillin-resistant *Staphylococcus aureus* infection. *Journal American Medical Association* 2003; **290**: 2976–2984.

31. **Begier EM, et al.** A high-morbidity outbreak of methicillin-resistant *Staphylococcus aureus* among players on a college football team, facilitated by cosmetic body shaving and turf burns. *Clinical Infectious Diseases* 2004; **39**: 1446–1453.

32. **Nguyen DM, Mascola L, Bancroft E.** Recurring methicillin-resistant *Staphylococcus aureus* infections in a football team. *Emerging Infectious Diseases* 2005; **11**: 526–532.

33. **Devine J, Cooke RPD, Wright EP.** Is methicillin-resistant *Staphylococcus aureus* (MRSA) contamination of ward-based computer terminals a surrogate marker for nosocomial MRSA transmission and handwashing compliance? *Journal of Hospital Infection* 2001; **48**: 72–75.

34. **Bures S, et al.** Computer keyboards and faucet handles as reservoirs of nosocomial pathogens in the intensive care unit. *American Journal of Infection Control* 2000; **28**: 465–471.

35. **Layton MC, et al.** An outbreak of mupirocin-resistant *Staphylococcus aureus* on a dermatology ward associated with an environmental reservoir. *Infection Control and Hospital Epidemiology* 1993; **14**: 369–375.

36. **Blythe D, et al.** Environmental contamination due to methicillin-resistant *Staphylococcus aureus* (MRSA). *Journal of Hospital Infection* 1998; **38**: 67–69.

37. **Lina G, et al.** Involvement of Panton-Valentine leukocidin-producing *Staphylococcus aureus* in primary skin infections and pneumonia. *Clinical Infectious Diseases* 1999; **29**: 1128–1132.

38. **Fridkin SK, et al.** Methicillin-resistant *Staphylococcus aureus* disease in three communities. *New England Journal of Medicine* 2005; **352**: 1436–1444.