Clinical Review

Community-associated methicillin-resistant Staphylococcus aureus infection

Literature review and clinical update

Kassandra Loewen  Yoko Schreiber  MD FRCP  MSc(Epi)  CIP  Mike Kirlew  MD CCFP
Natalie Bocking  MD MIPH  CCFP  Len Kelly  MD MClSc  FCFP  FRRM

Abstract

Objective To provide information on the prevalence and treatment of methicillin-resistant Staphylococcus aureus (MRSA) infections and the distinction between community-associated MRSA and health care–associated MRSA.

Quality of evidence The MEDLINE and EMBASE databases were searched from 2005 to 2016. Epidemiologic studies were summarized and the relevant treatment literature was based on level I evidence.

Main message The incidence of community-associated MRSA infection is rising. Certain populations, including indigenous Canadians and homeless populations, are particularly affected. Community-associated MRSA can be distinguished from health care–associated MRSA based on genetic, epidemiologic, or microbiological profiles. It retains susceptibility to some oral agents including trimethoprim-sulfamethoxazole, clindamycin, and tetracyclines. Community-associated MRSA typically presents as purulent skin and soft tissue infection, but invasive infection occurs and can lead to severe, complicated disease. Treatment choices and the need for empiric MRSA coverage are influenced by the type and severity of infection.

Conclusion Community-associated MRSA is a common cause of skin and soft tissue infections and might be common in populations where overcrowding and limited access to clean water exist.

Infection à Staphylococcus aureus résistants à la méticilline d'origine communautaire

Revue de la littérature médicale et mise à jour clinique

Résumé

Objectif Fournir des renseignements sur la prévalence et le traitement des infections aux Staphylococcus aureus résistants à la méticilline (SARM), de même que sur la distinction entre les SARM d’origine communautaire et les SARM associés aux soins de santé.

EDITOR’S KEY POINTS

• Isolates of methicillin-resistant Staphylococcus aureus (MRSA) that were first identified as hospital acquired are called health care–associated MRSA and are highly antibiotic resistant. Isolates of MRSA that appear in young and otherwise healthy patients are identified as community–associated (previously community-acquired) MRSA (CA-MRSA). Neither of these bacteria exist solely in the community or in hospitals.

• Empiric treatment is the norm for these typically purulent skin and soft tissue infections and includes consideration of severity of illness, access to follow-up, and patient adherence. Clinical practice guidelines for CA-MRSA treatment recommend increasingly aggressive treatment with increased severity of infection.

• Predisposing factors for CA-MRSA infection are varied and include living in a group setting, participation in sports teams, and social determinants of health. Crowded living environments and lack of access to clean water are also associated with increased risk of CA-MRSA infection.

This article has been peer reviewed.

Can Fam Physician 2017;63:512-20
Main message

*Staphylococcus aureus* is a common component of skin flora, and 30% to 50% of healthy adults are colonized with it at any given time.1 Preferred colonization sites include the axillae, anterior nares, pharynx, vagina, rectum, and perineum, and damaged skin.1.2 Colonization with *S aureus* is a commensal, asymptomatic relationship.1 Symptomatic *S aureus* infection is less common and might occur following breaks in skin or mucosal barriers. Its severity is influenced by isolate virulence and host factors.1.3 Diseases caused by *S aureus* range from superficial skin and soft tissue infections (SSTIs) to life-threatening invasive disease, including bacteraemia, endocarditis, and toxic shock syndrome.1 Most *S aureus* infections are caused by methicillin-sensitive *S aureus* (MSSA), which responds to penicillin.8 Methicillin-sensitive *S aureus* infections predominate (75%) in tertiary care centre staphylococcal infections, while some rural hospitals report MRSA accounts for slightly more than half (56%) of staphylococcal infections.4.5 This review will concentrate on strains that are resistant to penicillin (MRSA), for which *methicillin* (or *oxacillin*) is the term used by laboratories to identify penicillin resistance.

*Methicillin-resistant S aureus: 2 distinct origins.*

Methicillin-resistant *S aureus* was first identified at a hospital in the United Kingdom in 1961, shortly after the introduction of methicillin.6.9 In Canada, MRSA was first documented in 1964 and the first outbreak occurred in 1978 at the Royal Victoria Hospital in Montreal, Que.9 From the time of its emergence until the 1980s, MRSA was essentially a hospital-acquired pathogen.8 Today, these isolates of MRSA are called *health care–associated MRSA* (HA-MRSA) and are highly resistant to most oral antibiotics.

In the late 1980s and early 1990s cases of MRSA in young and otherwise healthy patients without any health care–related risk factors were reported.2,7,8,10 Some of the earliest reports of such infections in Canada and Australia came from isolated indigenous communities.11-14 Today, these isolates of MRSA have been identified as *community-associated* (previously *community-acquired*) MRSA (CA-MRSA).

Community-associated MRSA and HA-MRSA can be differentiated in several ways. These include presumed location of acquisition (ie, community or hospital),15 antibiotic susceptibility pattern,16 and genotyping,17.19 the latter being the most definitive. Our review included many articles with genotyped definitions, but some smaller studies use antibiotic susceptibility patterns.

Some newer, highly resistant strains have arisen, but they are rare in Canada and are currently limited to tertiary care centres. They include vancomycin-intermediate *S aureus* (VISA), heterogeneous VISA, and vancomycin-resistant *S aureus**20,21
Comparing CA-MRSA and HA-MRSA. Community-associated MRSA and HA-MRSA are genetically, epidemiologically, and phenotypically distinct (Table 1).²,⁴,⁶-⁸,¹⁰-¹⁵,¹⁹,²²-³⁴

Contemporary advances in laboratory technology have demonstrated that methicillin resistance was acquired through different genes in CA-MRSA and HA-MRSA isolates. Specifically, staphylococcal chromosomal cassette mec (SCCmec) types I, II, and III confer methicillin resistance in HA-MRSA whereas SCCmec types IV and V confer methicillin resistance in CA-MRSA.²,²³-²⁷

The SCCmec types carried by CA-MRSA are larger than those carried by CA-MRSA and confer resistance to additional non-β-lactam antibiotics. Community-associated MRSA is therefore susceptible to a broader range of antibiotics than HA-MRSA is.¹,²⁷,³⁰,³³ A study of pathogens isolated at Canadian hospitals between 2007 and 2009 found the susceptibility of CA-MRSA to trimethoprim-sulfamethoxazole (100.0%), gentamicin (98.7%), and clindamycin (86.1%) to be greater than that of HA-MRSA (86.5%, 85.5%, and 27.8%, respectively).⁴ Antibiotic sensitivity profiles can consequently be used as an inexpensive means of classifying MRSA as health care associated or community associated.¹⁶,³⁵ For example, clindamycin susceptibility is predictive of CA-MRSA with 95% sensitivity, 80% specificity, and a likelihood ratio of 4.86.³⁶ Methicillin-resistant Staphylococcus aureus isolates that are resistant to 3 or more non-β-lactam antibiotics can safely be categorized as HA-MRSA.¹⁶

Before advances were made in laboratory genetic technologies, epidemiologic risk factors were used to differentiate cases of HA-MRSA and CA-MRSA infection: the location of acquisition (ie, community or hospital) provided its designation.²⁶,²⁷ In the contemporary context, this method of differentiating HA-MRSA and CA-MRSA no longer aligns with clinical reality, as CA-MRSA has found its way into hospitals and is becoming an increasingly prevalent hospital pathogen.²,³² An American study found that community-associated strains of MRSA are increasing both in communities and in hospitals.¹⁵ In Canada, more than 20% of nosocomial MRSA infections are caused by CA-MRSA.¹⁷,³⁶ A recent study from Alberta found 27.6% of such hospital-onset MRSA infections were caused by CA-MRSA and 27.5% of community-associated infections were caused by HA-MRSA.³⁶ Both communities and hospitals have become antibiotic-rich environments and are apparently exchanging bacterial isolates.

### Table 1. Comparison of CA-MRSA and HA-MRSA

<table>
<thead>
<tr>
<th>CHARACTERISTIC</th>
<th>CA-MRSA</th>
<th>HA-MRSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time and location of emergence</td>
<td>1980s-1990s, in the community</td>
<td>1960s, in hospitals</td>
</tr>
<tr>
<td>Genotype</td>
<td>SCCmec types IV and V</td>
<td>SCCmec types I, II, and III</td>
</tr>
<tr>
<td>Virulence factors</td>
<td>Panton-Valentine leukocidin often present; other virulence factors believed to exist</td>
<td>Uncommon</td>
</tr>
<tr>
<td>Common subtypes</td>
<td>CMRSA-10 (USA300), CMRSA-7 (USA400)</td>
<td>CMRSA-2 (USA100)</td>
</tr>
<tr>
<td>Predominant type of infection</td>
<td>Skin and soft tissue infections</td>
<td>Respiratory tract, urinary tract, bloodstream, and postsurgical infections</td>
</tr>
<tr>
<td>Infection onset</td>
<td>Typically in the community in young, healthy individuals</td>
<td>Typically in hospital, often associated with older age, intensive care unit stay, and central lines</td>
</tr>
<tr>
<td>Antibiotic susceptibility</td>
<td>Susceptible to a range of antibiotics</td>
<td>Limited range of antibiotic susceptibility</td>
</tr>
<tr>
<td>Risk factors</td>
<td>• Living or working in a group setting (such as military barracks, subsidized housing, or a shelter)</td>
<td>• Surgery, hospitalization, residence in a long-term care facility, or dialysis within the past 12 months</td>
</tr>
<tr>
<td></td>
<td>• Use of illegal drugs within the past year</td>
<td>• The presence of an indwelling percutaneous catheter</td>
</tr>
<tr>
<td></td>
<td>• History of CA-MRSA infection or colonization</td>
<td>• Being hospitalized for more than 48 hours at time of first positive culture</td>
</tr>
<tr>
<td></td>
<td>• Regular contact with somebody who lives or works in a group setting, has used drugs in the past year, or has a history of CA-MRSA</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Absence of in-home water service</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Recent antibiotic use</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Being HIV positive</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Playing contact sports</td>
<td></td>
</tr>
</tbody>
</table>

There is consistent evidence that CA-MRSA is more likely than HA-MRSA to be associated with SSTIs. Community-associated MRSA is more likely than HA-MRSA to carry Panton-Valentine leukocidin, a known virulence factor often associated with tissue necrosis SSTIs.

Methicillin-resistant *S. aureus* SSTIs are associated with higher mortality rates, longer hospital admissions, and greater hospital costs than SSTIs caused by MSSA strains are. The reason for this is unclear, but might involve greater virulence of MRSA relative to MSSA or increased effectiveness of β-lactam antibiotics against MSSA.

In 2012, Golding reported a high rate of CA-MRSA infection in northern Saskatchewan (168.1 cases per 10,000 population in 2006). A compilation of 8 years of data from this region, including 2731 cases, shows that most cases (78.2%) are SSTIs, followed distantly by ear infections (6.7%), urogenital infections (2.4%), respiratory infections (1.1%), and joint or blood infections (0.4%) (Figure 1).

A community and hospital study done in northern Ontario documented that 56% of the burden of staphylococcal illness was caused by CA-MRSA.

The predominant strains of CA-MRSA identified are Canadian epidemic strain (CMRSA) 10 (also known as USA300) and CMRSA-7 (also known as USA400). The predominant strain of HA-MRSA is CMRSA-2 (also known as USA100). Health care–associated MRSA is more likely to be associated with respiratory tract, urinary tract, bloodstream, and postsurgical infections.

**Risk factors.** The original epidemiologic definition of HA-MRSA infection captures its principal risk factors: hospitalization, other prolonged exposure to a health care environment, or the presence of a percutaneous device such as a central line.

Predisposing factors for CA-MRSA infection are more varied and are intimately associated with social determinants of health. Frequent skin-to-skin contact, wound contact, and poor sanitation facilitate the transmission of CA-MRSA. Crowded living environments, including military barracks, homeless shelters, subsidized housing, and prisons, are associated with increased risk of CA-MRSA infection.

A study of the relationship between in-home pressurized water service and infectious diseases among Alaska Natives found that regions with limited access to clean water had significantly higher rates of MRSA infections (rate ratio = 7.1; 95% CI 3.6 to 14.0) and hospitalization for skin infections (rate ratio = 2.7; 95% CI 1.8 to 4.1). Socially disadvantaged minority populations are consistently associated with higher rates of CA-MRSA infection, including African Americans, Canadian First Nations communities, and the indigenous populations of Australia and New Zealand. Homelessness is another recognized risk factor for CA-MRSA infection, as is intravenous drug use.

**Epidemiology.** During the 2000s, increasing incidence rates of CA-MRSA infections were widely reported by researchers in the United States and Canada, along with a corresponding increase in SSTIs caused by *S. aureus*. Rates of

---

**Figure 1.** Rates of community-associated methicillin-resistant *Staphylococcus aureus* infections in northern Saskatchewan: *N = 2731.*

- Skin and soft tissue infections
- Ear infections
- Urogenital infections
- Respiratory infections
- Joint or blood infections
- Not specified
CA-MRSA infection are increasing, while HA-MRSA infection rates are generally reported to be in decline.\textsuperscript{19,53,57}

Several studies documenting the epidemiology of MRSA in indigenous populations have been published. Studies from communities in the United States,\textsuperscript{5,34} Canada,\textsuperscript{5,19,39,41,50,51} Australia,\textsuperscript{11,16} and New Zealand\textsuperscript{52} demonstrate high and increasing rates of CA-MRSA infection in the indigenous populations, where HA-MRSA is rare.

In Canada, Muileboom et al found the proportion of \textit{S aureus} isolates demonstrating methicillin resistance isolated from cultures obtained in one northern Ontario laboratory increased from 31\% in 2008 to 56\% in 2012.\textsuperscript{5}

Kirlew et al reported an incidence rate of MRSA bacteremia of 41.1 cases per 100000 person-years in northwestern Ontario.\textsuperscript{51} In northern Saskatchewan, Golden et al found that the rate of CA-MRSA infection increased from 8.2 cases per 100000 person-years in 2001 to 168.1 cases per 100000 person-years in 2006.\textsuperscript{61} A previous study found that 99.5\% of MRSA isolates from these remote communities were CA-MRSA.\textsuperscript{60} A 1-year study at the Children's Hospital of Winnipeg in Manitoba found that 79\% of patients from outside of Winnipeg who presented with community-onset \textit{S aureus} infection lived in rural communities in northern Manitoba, southern Nunavut, or northwestern Ontario.\textsuperscript{39} Among these patients, the rate of MRSA infection was relatively high (61\%).\textsuperscript{39} A large study assessing MRSA infection rates among children across Canada between 1995 and 2007 found that 25\% of all cases occurred in First Nations children.\textsuperscript{19}

Like their counterparts in Canada, indigenous populations in the United States, Australia, and New Zealand face disproportionately high rates of MRSA-associated infection and hospitalization.\textsuperscript{6,11,16,52}

The confluence of environmental and host factors might explain the disproportionate MRSA burden in indigenous communities. Environmental conditions associated with social and material deprivation, such as overcrowding and inadequate access to in-home pressurized water service, are associated with the transmission of MRSA and the development of MRSA-associated SSTIs.\textsuperscript{34} These same environmental conditions are pressing concerns in indigenous communities around the world.\textsuperscript{11,16,34,51} Additionally, the prevalence of host factors increasing vulnerability to infection by modulating the immune response (such as diabetes mellitus) or providing a portal of entry (skin disease, injection drug use) might be elevated in some indigenous communities.\textsuperscript{59-63}

\textbf{Treatment.} Empiric treatment is the norm for infections and must take into consideration information about likely infecting agents, severity of illness, access to follow-up, patient adherence, and other factors. Published guidelines, original research, and knowledge of local epidemiology might assist clinicians in making clinical judgments that adhere to principles of antimicrobial stewardship.\textsuperscript{52,62-65}

The current clinical practice guidelines for CA-MRSA and HA-MRSA treatment from the Infectious Diseases Society of America recommend increasingly aggressive treatment with increased severity of infection.\textsuperscript{66}

A distinction is made between purulent and non-purulent SSTIs. Uncomplicated abscesses without evidence of systemic toxicity might be treated by incision and drainage without antibiotics (level I evidence).\textsuperscript{2,22,28,65} Evidence from 3 randomized controlled trials and a systematic review indicates not providing antibiotics to patients who undergo incision and drainage for uncomplicated abscesses is associated with lower re-infection rates and comparable wound healing (level I evidence).\textsuperscript{22,66-69} Empiric treatment of purulent cellulitis, when needed, might include oral clindamycin, trimethoprim-sulfamethoxazole, tetracyclines, or linezolid (level II evidence).\textsuperscript{65} Nonpurulent cellulitis is generally caused by \textit{Streptococcus} (group A, C, or G), while purulent cellulitis is substantially more likely to be caused by \textit{S aureus}, most commonly CA-MRSA.\textsuperscript{70-73} Treatment of nonpurulent cellulitis should therefore target streptococcal species with a \textit{\beta}-lactam antibiotic, without routine addition of an agent active against MSSA or MRSA. Most, if not all, MRSA encountered by family physicians will be CA-MRSA, as it occurs primarily in the community context and is distinct from its highly drug-resistant relative, HA-MRSA (Table 2).\textsuperscript{66,74}

Complicated SSTIs and invasive MRSA infections, including bacteremia, septic arthritis, endocarditis, meningitis, and pneumonia, are typically treated with parenteral vancomycin (level I and III evidence).\textsuperscript{28,65} Susceptibility to clindamycin, trimethoprim-sulfamethoxazole, and tetracyclines is often retained in CA-MRSA isolates\textsuperscript{75} and these agents can be considered in nonsevere infection or as step-down therapy. These agents have good oral bioavailability.

\begin{table}[h]
\centering
\begin{tabular}{|l|l|}
\hline
\textbf{SSTI} & \textbf{TREATMENT*} \\
\hline
Simple cutaneous abscess (in a low-risk patient not involving face, hands, or genitalia) & Incision and drainage alone; obtain culture \\
\hline
Purulent cellulitis (without abscess): treat for CA-MRSA if risk factors present & Tetracycline, trimethoprim-sulfamethoxazole, or clindamycin \\
\hline
Nonpurulent cellulitis (no exudate): treat for \textit{\beta}-hemolytic streptococcus & \textit{\beta}-Lactam antibiotic (cloxacillin or first-generation cephalosporin) \\
\hline
\end{tabular}
\caption{Table 2. Treatment of outpatient SSTI in the era of CA-MRSA}
\end{table}

\*A detailed management algorithm is available within the Infectious Diseases Society of America guidelines 2014 update on SSTIs.\textsuperscript{59} All recommendations are level II evidence, adapted from the Infectious Diseases Society of America 2011 guidelines.\textsuperscript{65}
Alternatives to vancomycin for the treatment of severe or invasive MRSA infection include linezolid, daptomycin, and tigecycline. Newer agents recently approved or developed that have shown promise are the cephalosporins ceftaroline and ceftobiprole; the lipoglycopeptides telavancin, dalbavancin, and oritavancin; and the oxazolidinone tedizolid. Pharmacologic and clinical considerations for each antimicrobial agent are listed in Table 3. Telavancin, oritavancin, and dalbavancin might be of particular interest to community-based health care services because of their once-daily, one-time, and weekly dosing, respectively (only dalbavancin is currently available in Canada).

Table 4 provides a list of additional agents active against MRSA that are not available in Canada.

Failure of vancomycin therapy has been documented in the context of resistant strains (heterogeneous VISA, vancomycin-resistant *S aureus*), but these are unlikely

### Table 3. Antibiotics relevant in the treatment of MRSA

<table>
<thead>
<tr>
<th>AGENT</th>
<th>ROUTE</th>
<th>ACTIVITY</th>
<th>DOSAGE FOR MRSA INFECTIONS</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lincosamides</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clindamycin</td>
<td>Oral or IV</td>
<td>Bacteriostatic</td>
<td>300-450 mg orally 4 times daily or 600-900 mg IV every 8 h</td>
<td>Increasing resistance among community-associated MRSA and methicillin-sensitive <em>Staphylococcus aureus</em>; inducible resistance in MRSA</td>
</tr>
<tr>
<td><strong>Sulfonamides</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trimethoprim-sulfamethoxazole</td>
<td>Oral</td>
<td>Bactericidal</td>
<td>1-2 double-strength tablets (160 mg and 800 mg) orally twice daily</td>
<td>Contraindicated in severe renal or hepatic dysfunction; multiple drug interactions (including ACEIs and ARBs)</td>
</tr>
<tr>
<td><strong>Tetracyclines</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tetracycline</td>
<td>Oral</td>
<td>Bacteriostatic</td>
<td>250-500 mg orally 4 times daily</td>
<td>Caution about teratogenicity</td>
</tr>
<tr>
<td>Doxycycline</td>
<td>Oral</td>
<td>Bacteriostatic</td>
<td>100 mg orally twice daily</td>
<td>Caution about teratogenicity</td>
</tr>
<tr>
<td>Minocycline</td>
<td>Oral</td>
<td>Bacteriostatic</td>
<td>100 mg orally twice daily</td>
<td>Caution about teratogenicity</td>
</tr>
<tr>
<td>Tigecycline</td>
<td>IV</td>
<td>Bacteriostatic</td>
<td>100-mg IV loading dose, then 50 mg IV every 12 h</td>
<td>Caution about teratogenicity; indicated for SSTI and intra-abdominal infections (unfavourable outcomes in community-associated pneumonia)</td>
</tr>
<tr>
<td><strong>Oxazolidinones</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Linezolid</td>
<td>Oral or IV</td>
<td>Bacteriostatic</td>
<td>600 mg orally twice daily or 600 mg IV every 12 h</td>
<td>Indicated for SSTI; multiple drug interactions, risk of myelosuppression if used 2 wk or longer; high cost</td>
</tr>
<tr>
<td><strong>Lipopeptides</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Daptomycin</td>
<td>IV</td>
<td>Bactericidal</td>
<td>4 mg/kg IV every 24 h for SSTI; 6 mg/kg IV every 24 h for bacteremia or right-sided endocarditis, up to 12 mg/kg IV every 24 h</td>
<td>Indicated for SSTI, endocarditis, and bloodstream infection; not indicated for pneumonia unless from haematogenous origin; might cause eosinophilic pneumonia, abnormal coagulation, myopathy, and rhabdomyolysis</td>
</tr>
<tr>
<td><strong>Lipoglycopeptides</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vancomycin</td>
<td>IV</td>
<td>Bactericidal</td>
<td>15-20 mg/kg per dose every 8-12 h; consider loading dose of 25-30 mg/kg in seriously ill patients</td>
<td>Dose monitoring; target levels vary with site and severity of infection</td>
</tr>
<tr>
<td>Telavancin</td>
<td>IV</td>
<td>Bactericidal</td>
<td>10 mg/kg IV every 24 h (if creatinine clearance &gt; 50 mL/min)</td>
<td>Indicated for SSTI; increased mortality observed in chronic kidney disease</td>
</tr>
</tbody>
</table>

ACEI—angiotensin-converting enzyme inhibitor, ARB—angiotensin receptor blocker, IV—intravenous, MRSA—methicillin-resistant *Staphylococcus aureus*, SSTI—skin and soft tissue infection.
to be commonly encountered.\textsuperscript{20,22} Treatment of these infections is beyond the scope of this article.\textsuperscript{52,82}

For patients colonized with MRSA, decolonization treatment can be considered under special circumstances, such as recurrent infections in an individual or household (level III evidence).\textsuperscript{22,28,65} Decolonization regimens might involve nasal administration of mupirocin, daily 4% chlorhexidine soap baths, and a course of doxycycline and rifampin (level I).\textsuperscript{72,83} Success rates are modest (<50%) at best and largely influenced by comorbidities, and thus decolonization is not routinely recommended.\textsuperscript{3,47,84,85} It is recommended that household contacts and patients exercise good hand-washing practices. Household members should avoid sharing razors and other personal hygiene equipment; however, family bedding, clothing, and dishes can be washed together as usual. Aside from covering open wounds, there is no need to isolate persons colonized with MRSA within a household or to wear personal protective equipment when engaging with the colonized individual. However, gloves should be used when handling wounds.\textsuperscript{47}

**Future research directions.** This is an evolving science, and there is much to learn about community spread of CA-MRSA. As HA-MRSA primarily involves inpatients, it lends itself more easily to study. As CA-MRSA began entering the hospital setting it now lends itself to hospital-based research. While specific clinical questions around initial drug choice and duration remain, regional population studies are needed to inform empirical treatment for the community-based clinician.

**Conclusion**

The prevalence of CA-MRSA appears to be on the rise globally, and disadvantaged communities with overcrowded housing and homeless populations are disproportionally affected. Community-associated MRSA can be found in both hospitals and the community and is predominantly associated with purulent SSTIs.

Treatment of endemic CA-MRSA infections needs to be balanced with the principles of antibiotic stewardship.\textsuperscript{54}

**Ms Loeven** is a research intern in the Anishinaabe Bimaadiziwin Research Program in Sioux Lookout, Ont. Dr Schreiber is Assistant Professor at the University of Ottawa in the Ottawa Hospital in Ontario, Clinical Investigator in the Ottawa Hospital Research Institute, and a visiting faculty member at the Northern Ontario School of Medicine in Sioux Lookout. Dr Kiri\textsuperscript{ev} is Assistant Professor at the Northern Ontario School of Medicine and a community physician in Sioux Lookout. Dr Bocking is a public health physician in the Sioux Lookout First Nations Health Authority. Dr Kelly is a research consultant for the Anishinaabe Bimaadiziwin Research Program.

**Contributors**

All authors contributed to the literature review and interpretation, and to preparing the manuscript for submission.

**Competing interests**

None declared

**Correspondence**

Dr Len Kelly, e-mail kelly@mcmaster.ca

**References**


---

**Table 4. Additional agents active against MRSA not available in Canada**

<table>
<thead>
<tr>
<th>AGENT</th>
<th>ROUTE</th>
<th>STATUS (AT TIME OF WRITING)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tedizolid</td>
<td>Oral or IV</td>
<td>Received NOC; not yet marketed</td>
</tr>
<tr>
<td>Cefetiboprol medocaril</td>
<td>IV</td>
<td>Received NOC; never marketed</td>
</tr>
<tr>
<td>Cefdartaroline</td>
<td>IV</td>
<td>Not available</td>
</tr>
<tr>
<td>Dalbavancin</td>
<td>IV (weekly)</td>
<td>Not available</td>
</tr>
<tr>
<td>Oritavancin</td>
<td>IV (1-time dose)</td>
<td>Not available</td>
</tr>
</tbody>
</table>

*IV—intravenous, MRSA—methicillin-resistant *Staphylococcus aureus*, NOC—Health Canada Notice of Compliance.*


