Strategies for Clinical Management of MRSA in the Community: Summary of an Experts' Meeting Convened by the Centers for Disease Control and Prevention

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*Note: The findings and conclusions in this report are those of the authors and do not necessarily represent the views of the funding agency.

ABSTRACT

Methicillin-resistant *Staphylococcus aureus* (MRSA) has emerged as a cause of skin infections and, less commonly, invasive infections among otherwise healthy adults and children in the community. More data are needed in order to fully understand the epidemiology, microbiology, and pathophysiology of these infections and to identify optimal prevention and treatment strategies. This report summarizes strategies for the clinical management of MRSA in the community based on discussions held at an MRSA experts' meeting convened by the Centers for Disease Control and Prevention in July 2004, in conjunction with additional data available as of January 2006.

Methicillin-resistant *Staphylococcus aureus* (MRSA) has emerged in the community with clinical, epidemiologic, and bacteriologic characteristics distinct from healthcare-associated MRSA (HA-MRSA)[2-4]. In July 2004, the Centers for Disease Control and Prevention (CDC) convened a meeting of experts to describe reasonable strategies for the clinical and public health management of MRSA in the community. This report summarizes strategies for the clinical management of MRSA in the community that are based on discussions held at that meeting, in conjunction with additional data available as of January 2006.

Background

Community-associated MRSA (CA-MRSA) refers to an MRSA infection with onset in the community in an individual lacking established MRSA risk factors, such as recent hospitalization, surgery, residence in a long-term care facility, receipt of dialysis, or presence of invasive medical devices[5]. This term has also been used to refer to MRSA strains with bacteriologic characteristics (e.g., genotypes, antimicrobial susceptibility profiles) considered typical of isolates obtained from patients with CA-MRSA infections[6], although an association initially observed between microbiologic characteristics and MRSA transmission in the community versus healthcare settings appears to be breaking down. From a clinical management standpoint, awareness of local resistance patterns for pathogens in the differential diagnosis of specific clinical syndromes is more important than formally categorizing possible MRSA infections as CA-MRSA or HA-MRSA; however, some assessment of healthcare exposure may be useful in predicting isolate resistance to particular antimicrobial agents.

The spectrum of disease caused by CA-MRSA appears to be similar to that of methicillin-susceptible *Staphylococcus aureus* (MSSA) in the community. Skin and soft tissue infections (SSTIs), specifically furuncles (abscessed hair follicles or "boils"), carbuncles (coalesced masses of furuncles), and abscesses, are the most frequently reported clinical manifestations[5, 7, 8] *(Figure 1)*. MRSA skin lesions are frequently confused with spider bites by both patients and clinicians, even in areas of the country where spiders capable of causing necrotic skin lesions are not endemic[9]. The spontaneous appearance of a raised red lesion might lead to this supposition among patients, while the tendency for lesions to develop necrotic areas might confuse clinicians. The role of MRSA in cellulitis without abscess or purulent drainage is less clear since cultures are rarely obtained. The severity of MRSA SSTIs varies from mild superficial infections to deeper soft-tissue abscesses requiring hospital admission for surgical incision and drainage and delivery of parenteral antibiotics[10, 11]. Anecdotal reports suggest that recurrent MRSA skin infections and clustering of infections within a household are relatively common occurrences.

Less commonly, MRSA has been associated with severe and invasive staphylococcal infections in the community, including necrotizing pneumonia and empyema[12-14], sepsis syndrome[15, 16], musculoskeletal infections including pyomyositis and osteomyelitis[15, 17], necrotizing fasciitis[18], purpura fulminans[19, 20], and disseminated infections with septic emboli[14, 15]. Invasive manifestations occur as complications of preceding SSTIs or viral respiratory tract infections (particularly influenza), as well as in otherwise healthy persons without recognized preceding infections or risk factors[21].

Incidence of CA-MRSA varies geographically in the United States[5]. To date, reported CA-MRSA infections have disproportionately affected children and young adults and individuals from racial minority groups or low socioeconomic status[5, 22, 23]. Transmission of MRSA has occurred among inmates in correctional facilities[24-26], competitive sports participants[27-31], military recruits[32], day care attendees[33, 34], men who have sex with men[21, 35], and Native Americans[7, 36]. Factors common to these settings that facilitate the spread of infection include crowding, frequent skin-to-skin contact between individuals, participation in activities that result in compromised skin surfaces, sharing of personal items that may become contaminated with wound drainage, and challenges in maintaining personal cleanliness and hygiene. Limited access to health care[25] and frequent antibiotic exposure[29, 37] may also facilitate spread of infection in some settings. While outbreaks have frequently been reported among members of defined groups, most patients do not have recognized CA-MRSA or HA-MRSA risk factors and are not linked to an outbreak[38].

Isolates obtained from patients with MRSA infections described as CA-MRSA based on epidemiologic criteria have been noted to possess bacteriologic characteristics distinct from those of isolates from patients meeting epidemiologic criteria for HA-MRSA, although this situation is evolving. CA-MRSA isolates tend to be resistant to fewer antimicrobial classes, possess different toxin genes, and carry a different type of the gene complex known as staphylococcal cassette chromosome *mec* (SCC*mec*), which contains the *mec*A methicillin-resistance gene[23, 39]. Pulsed-field gel electrophoresis (PFGE) and other strain-typing methods have identified a small number of molecular types that have accounted for most CA-MRSA isolates characterized in the United States[40].

Unlike HA-MRSA isolates, which are usually resistant *in vitro* to multiple classes of antimicrobial agents, many CA-MRSA isolates to date have been resistant only to beta-lactams (the antimicrobial class that includes penicillins and cephalosporins) and macrolides / azalides (e.g., erythromycin, clarithromycin, azithromycin)[5]. However, resistance to other classes of antimicrobial agents, such as fluoroquinolones and tetracyclines, occurs and may be increasing in prevalence[5, 10]. Most CA-MRSA isolates to date have been susceptible to trimethoprim-sulfamethoxazole (TMP/SMX), gentamicin, tetracycline, and clindamycin, although some *S. aureus* isolates that appear erythromycin-resistant and clindamycin-susceptible by routine susceptibility testing exhibit *in vitro* resistance to clindamycin during therapy ("inducible resistance")[41].

Inducible clindamycin resistance can be detected through a specialized laboratory test called the D-zone test[42] *(Figure 2)*. *S. aureus* strains with the inducible resistance phenotype, termed "inducible macrolide-lincosamide-streptogramin B resistance" (iMLS), have a high rate of mutation to constitutive clindamycin resistance, a trait which would confer a selective advantage during clindamycin therapy. Although erythromycin is used to induce clindamycin resistance in the D-zone test, pre-treatment or co-treatment with erythromycin is not needed for iMLS strains to express clindamycin resistance *in vivo* during a course of therapy. The clinical implications of inducible clindamycin resistance are unclear[43]. There have been case reports of clindamycin treatment failures[44, 45] in patients with invasive *S. aureus* infections caused by iMLS strains. However, clinical successes have also been reported in patients infected with iMLS strains and

treated with clindamycin[46, 47]. Treatment failure may be more likely to occur in patients with deep-seated infections requiring prolonged therapy; however, data are scant.

CA-MRSA isolates commonly possess genes for the Panton-Valentine leukocidin (PVL) toxin, rarely identified in HA-MRSA isolates[23, 48]. Presence of PVL genes in *S. aureus* isolates has been associated with primary skin infections[49], severe necrotizing pneumonia[49, 50], and increased complications of hematogenous osteomyelitis[17]; however, the role of PVL in the pathogenesis of *S. aureus* infections has not been fully elucidated.

Data from controlled clinical trials are needed to establish optimal therapy for MRSA SSTIs. Various antimicrobial agents, including clindamycin, TMP/SMX, tetracyclines, and linezolid have been used for empiric outpatient treatment of SSTIs possibly caused by MRSA[51-56]. Incision and drainage alone may be adequate therapy for some previously healthy patients with cutaneous abscesses and no systemic signs of infection. In recent investigations, receiving an antimicrobial agent to which the infecting isolate was later found to be resistant was not associated with adverse outcomes among immune-competent patients with CA-MRSA SSTIs[5, 57]. Furthermore, in a recent randomized, placebo-controlled trial in adult patients with deep skin abscesses and surrounding cellulitis, the majority of which were caused by MRSA, treatment success rates were over 90% for patients treated with incision and drainage alone and those treated with incision and drainage plus cephalexin (D. Young, University of California, San Francisco, submitted). It is not known if antimicrobial therapy of less serious skin infections prevents invasive complications.

The epidemiology of MRSA colonization in the community and the association between colonization and infection need to be better elucidated, as does the role of attempted decolonization in the clinical management of CA-MRSA infections. In a nationally representative U.S. survey of non-institutionalized individuals ≥1 year old, the prevalence of *S. aureus* and MRSA nasal colonization were 32.4% and 0.8% respectively in 2001 and 2002[58]. Other data indicate the prevalence of MRSA colonization may be increasing in some community settings[59]. However, nasal MRSA colonization is not invariably present in individuals with active MRSA infections[10].

Nasal colonization with *S. aureus* has been identified as a risk factor for infection, and carriage of MRSA as opposed to MSSA has posed an increased risk of infection in various healthcare settings[60-62]. Few data are available on the association between MRSA colonization and infection in the community[63]. MRSA colonization also occurs at sites other than the nose (e.g., pharynx, axilla, rectum, perineum)[34, 64], and may be important in development and transmission of infection as well as in persistence or reappearance of colonization after use of nasal decolonization agents.

Regimens to eliminate *S. aureus* colonization have been used in healthcare settings in an effort to prevent autoinfection among colonized patients and control MRSA outbreaks[65]. More recently, decolonization regimens have been employed in MRSA outbreaks in community settings[66]. These regimens have included various combinations of topical and systemic antimicrobial agents and antiseptic body washes and have typically been used as part of multi-faceted infection control interventions, making it difficult to evaluate the effectiveness of any individual component.

Data from healthcare settings indicate that intranasal mupirocin can be effective at eliminating *S. aureus* colonization in the short term; however, recolonization is common[67]. The effectiveness of decolonization therapy of any kind for preventing

S.aureus infections in individual patients has not been well-established[67, 68]. There are few data on the effectiveness of decolonization regimens to eliminate colonization or prevent infection in community settings or within families[69, 70]. Compliance with decolonization regimens has been poor in some community settings[66]. Additionally, development of resistance to systemic and topical agents during decolonization therapy has been described[68], causing concern about widespread use of these interventions.

The epidemiologic and molecular features of MRSA are evolving such that characteristics that initially distinguished CA-MRSA from HA-MRSA appear to be changing. For example, several reports have described transmission in healthcare settings of MRSA strains indistinguishable from those associated with community transmission[6, 71, 72]. Eventually, these strains could become the predominant strains in both community and healthcare settings. In addition, the prevalence of *in vitro* resistance to non-beta-lactam antimicrobial agents may be increasing among MRSA strains associated with community transmission[73, 74].

Additional data from well-designed studies are needed in order to fully understand the epidemiology, microbiology, and pathophysiology of CA-MRSA infections and identify optimal prevention and treatment strategies. Given the information that is currently available, participants in the CDC-convened experts' meeting identified the following strategies as reasonable.

Reasonable Strategies for Clinical Management of MRSA in the Community, with a Focus on Skin and Soft Tissue Infections

1. MRSA should be considered in the differential diagnosis of SSTIs compatible with *S. aureus* infection, such as skin abscesses. A presenting chief complaint of "spider bite" should raise suspicion of a *S. aureus* infection.

2. MRSA should be considered in the differential diagnosis of other syndromes compatible with *S. aureus* infection, including sepsis syndrome, osteomyelitis, septic arthritis, and pneumonia that is severe or follows an influenza-like illness, as well as in the differential diagnosis of some severe syndromes not typically associated with *S. aureus*, such as necrotizing fasciitis and purpura fulminans.

3. Clinicians are encouraged to collect specimens for culture and antimicrobial susceptibility testing from all patients with abscesses or purulent skin lesions, particularly those with severe local infections, systemic signs of infection, or history suggesting connection to a cluster or outbreak of infections among epidemiologically linked individuals. Culture and susceptibility results are useful both for management of individual patients and to help determine local prevalence of *S. aureus* susceptibility to beta-lactam and non-beta-lactam agents. In an outbreak within a defined cohort, cultures should be obtained from all new onset cases at least until the susceptibility pattern of the outbreak strain has been determined. In a community where empiric therapy for SSTIs has been modified to provide coverage for MRSA, obtaining cultures of purulent SSTIs is still important to monitor trends in susceptibility of *S. aureus* to non-beta-lactam agents.

   a. Appropriate clinical specimens include: (1) fluid from a purulent lesion or abscess cavity, (2) respiratory secretions (e.g., sputum, tracheal aspirations, bronchoscopic aspirations) or pleural fluid from a patient with pneumonia, (3) blood from a moderately or severely ill patient with signs and symptoms of systemic infection, and (4) other specimen from a normally sterile site suspected to be a focus of infection (e.g., joint or bone).

   b. It is not necessary to routinely collect nasal cultures in all patients presenting with possible MRSA infection.

4. At the present time, there is no information to suggest that molecular typing or identification of toxin genes should impact clinical management decisions.

5. Incision and drainage constitutes a primary therapy for furuncles, other abscesses, and septic joints, and should be performed routinely. If a clinician is unsure whether pus is present in a lesion, an attempt can be made to aspirate fluid from the lesion using an adequate size needle and syringe (e.g., a 16- to 19-gauge needle on a 10cc syringe). For small furuncles not amenable to incision and drainage or collection of material for culture, moist heat may be satisfactory to promote drainage[56].

6. For some patients with purulent skin lesions, empiric antimicrobial therapy may be administered in addition to incision and drainage. Factors that may influence the clinical decision to supplement incision and drainage with antimicrobial therapy include: (1) severity and rapidity of progression of the SSTI or the presence of associated cellulitis (in one study[57], an infected site of >5 cm in diameter was associated with failure of incision and drainage without effective antimicrobial therapy; however, in a recent placebo-controlled trial, a greater than 90% success rate was achieved for deep skin abscesses with associated cellulitis treated with incision and drainage alone (D. Young, University of California, San Francisco, submitted)), (2) signs and symptoms of systemic illness, (3) associated patient co-morbidities or immune suppression (e.g., diabetes mellitus, neoplastic disease, HIV infection), (4) extremes of patient age, (5) location of the abscess in an area that may be difficult to drain completely or that can be associated with septic phlebitis of major vessels (e.g., central face), and (6) lack of response to initial treatment with incision and drainage alone.

7. When empiric antimicrobial therapy is provided for treatment of an SSTI compatible with *S. aureus* infection, local susceptibility data should be used to guide treatment. A beta-lactam agent (anti-staphylococcal penicillin or cephalosporin) is still a reasonable option for first-line therapy in a patient with mild to moderate illness and no significant co-morbidities if the local prevalence of methicillin-resistance among community *S. aureus* isolates is low. There are no data to determine a specific MRSA prevalence rate that warrants a change in

empiric therapy for SSTIs; a prevalence of >10-15% of community *S. aureus* isolates has been suggested by some experts[53]. No reliable criteria (other than a history of recurrent MRSA infection) have been identified for predicting MRSA in an individual patient presenting with an SSTI. In some settings, local health departments may be able to provide information on groups that are at increased risk for CA-MRSA infection in their area.

8. Several antimicrobial agents have been proposed as alternatives to beta-lactams for outpatient treatment of SSTIs when an oral regimen with activity against MRSA is desired. These include clindamycin, tetracyclines (including doxycycline and minocycline), trimethoprim-sulfamethoxazole (TMP-SMX), rifampin (used only in combination with other agents), and linezolid. There are advantages and disadvantages to each of these agents. More data are needed from controlled clinical trials to establish optimal regimens for the treatment of MRSA SSTI. *Clostridium difficile*-associated disease (CDAD) can occur in association with numerous antimicrobial agents[75]. While uncommon, there have been recent reports of severe CDAD in otherwise healthy adults and children in the community[76], highlighting the need to use antimicrobial agents only when necessary. Clinicians should consult product labeling for a complete list of potential adverse effects associated with a particular agent.

   a. <u>Clindamycin:</u> Clindamycin is FDA-approved for the treatment of serious infections due to *S. aureus*. Although not specifically approved for the treatment of infections due to MRSA, clindamycin has been used widely in the treatment of SSTIs[77, 78] and there are reports of clindamycin being used successfully to treat CA-MRSA infections[54, 79-81].

      i. A D-zone test should be performed to identify inducible clindamycin resistance in erythromycin-resistant, clindamycin-susceptible *S. aureus* isolates[42]. If empiric clindamycin therapy has been initiated and inducible clindamycin resistance is detected, response to therapy should be assessed. Clinicians should consider changing to another agent if response to therapy has been unsatisfactory and should monitor the patient closely to assure resolution of the infection if clindamycin therapy is continued.

      ii. Although there are no direct comparative data from prospective trials, observational data suggest CDAD may occur relatively more frequently in association with clindamycin as compared to other antimicrobial agents [76]. However, CDAD is a rarely reported complication of antimicrobial therapy, particularly among children under the age of eight years, for whom options for oral MRSA therapy are most limited.

   b. <u>Tetracyclines (e.g., tetracycline, doxycycline, minocycline)</u>: Doxycycline is FDA-approved for the treatment of *S. aureus* skin infections, but not

specifically for those caused by MRSA. There is little information in the medical literature on the use of tetracyclines for the treatment of MRSA infections. In a small case series, the long-acting tetracyclines, doxycycline and minocycline, appeared to be adequate for the treatment of MRSA SSTIs caused by tetracycline-susceptible isolates, but data were not sufficient to support their use in treatment of invasive infections[82].

   i. Tetracyclines are generally not recommended during pregnancy or for children under the age of eight years, due to potential for tooth enamel hypoplasia and discoloration and decreased bone growth.

   ii. Most U.S. laboratories test *S. aureus* isolates for susceptibility to this class of agents using tetracycline; however, this may overestimate the prevalence of resistance to doxycycline and minocycline. The two major tetracycline resistance genes in *S. aureus* are *tetM*, which confers resistance to all agents in the class, and *tetK,* which confers resistance to tetracycline specifically[83]. While the prevalence of tetracycline resistance remains low among MRSA isolates in the community, resistance described thus far in predominant community strains has been associated with *tetK*[84], indicating that doxycycline and minocycline may remain viable treatment options. However, there is little information available on clinical outcomes associated with the use of minocycline or doxycycline to treat infections caused by *S. aureus* strains with *in vitro* resistance to tetracycline[85]. Replacement of tetracycline with doxycycline or minocycline on commercial susceptibility testing panels for *S. aureus* may be desirable in the future, particularly if the prevalence of tetracycline resistance increases.

c. <u>TMP-SMX</u>: TMP-SMX is not FDA-approved for the treatment of any form of staphylococcal infection. However, the medical literature contains several case reports of the successful use of TMP-SMX in the treatment of *S. aureus* infections, including MRSA[86-92]. In a case-series of CA-MRSA skin infections in Los Angeles, California[93], prompt resolution of symptoms was achieved in six (50%) of twelve patients initially treated with double strength TMP/SMX alone (in addition to incision and drainage of abscesses) and in all of six patients treated initially with a combination of TMP/SMX and rifampin. A single randomized-controlled trial compared TMP-SMX to vancomycin for the treatment of a variety of serious (predominantly invasive) *S. aureus* infections in intravenous drug users, and found TMP-SMX to be inferior to vancomycin in that setting[94].

   i. There are no data indicating that TMP-SMX is clinically effective for the treatment of SSTIs due to group A streptococcus (GAS), another common cause of SSTIs, and GAS isolates are commonly resistant to TMP-SMX, as evidenced by the use of this agent in

culture media selective for GAS[95]. Clinicians should consider addition of an agent for GAS coverage, such as a beta-lactam agent or clindamycin, if this is an etiologic consideration (e.g., in patients with cellulitis).

ii. TMP-SMX is not recommended in women in the third trimester of pregnancy or in infants less than two months of age.

d. <u>Rifampin (Should not be used as a single agent)</u>: Resistant strains of *S. aureus* are observed rapidly when rifampin is used as a single agent[96]. Rifampin has been used in combination with other antimicrobial agents that are active against *S. aureus* to treat staphylococcal infections[93]. Because rifampin achieves high concentrations in mucosal surfaces[97], a theoretical benefit of including it in a treatment regimen for active MRSA infections is that it may promote eradication of MRSA carriage[62]. However, there is little information available on the incremental benefit of adding rifampin for the treatment of staphylococcal infections. Drug-drug interactions are common with rifampin.

e. <u>Linezolid (Consultation with an infectious disease specialist suggested)</u>: Linezolid is FDA-approved for the treatment of complicated skin infections and hospital-acquired pneumonia due to MRSA in adults. Clinical experience with linezolid in children is limited. Apparent failures of linezolid to treat or prevent endocarditis in patients with intravascular MRSA infection have been reported[98-100]. Linezolid use has been associated with a risk of dose- and duration-dependent reversible myelosuppression, principally thrombocytopenia, prompting recommendations for monitoring of complete blood counts in patients receiving linezolid for >2 weeks[101, 102]. There have also been case reports of peripheral and optic neuropathy and lactic acidosis in patients receiving prolonged therapy with linezolid[103-105]. Linezolid is costly compared to other alternative agents. Although rare, resistance to linezolid has been described in *S. aureus*[106]. To limit potential for widespread resistance, clinicians should consider reserving linezolid for use in more severe infections in consultation with an infectious disease specialist.

9. Because of a relatively high prevalence of resistance among *S. aureus* isolates in the community or the potential for rapid development of resistance, some antimicrobial agents are not optimal choices for the empiric treatment of community-associated SSTIs possibly caused by *S. aureus*. These include fluoroquinolones and macrolides.

a. <u>Fluoroquinolones</u>: Ciprofloxacin and levofloxacin are FDA-approved for the treatment of complicated skin infections in adults. These agents, plus moxifloxacin and gatifloxacin, are FDA-approved for the treatment of uncomplicated skin infections caused by *S. aureus*. However, none of the

fluoroquinolones are FDA-approved for treatment of MRSA infections. A major limitation of fluoroquinolones is that resistant mutants can be selected with relative ease, leading to relapse and treatment failure[107, 108]. MRSA strains are especially adept at developing fluoroquinolone resistance, and such resistance is already found among MRSA isolated from patients with CA-MRSA infections in some areas of the United States[5]. While minimal inhibitory concentrations (MICs) tend to be lower for newer (e.g., moxifloxacin, gatifloxacin) as compared to older (e.g., ciprofloxacin, levofloxacin) fluoroquinolone agents, genes conferring fluoroquinolone resistance in *S.aureus* confer resistance to the entire class of agents[109].

b. <u>Macrolides / Azalides</u>: Erythromycin, clarithromycin, and azithromycin are all FDA-approved for the treatment of uncomplicated skin infections caused by *S. aureus*. However, there are no specific data concerning the treatment of MRSA infections with these drugs. Furthermore, resistance to macrolides is common among MRSA isolates, including community strains[5], limiting their usefulness as alternative agents for empiric treatment of SSTIs in areas where the prevalence of MRSA is high.

10. As with methicillin-resistance, prevalence of resistance to non-beta-lactam agents varies geographically and is likely to change over time. Local susceptibility patterns of community *S. aureus* isolates should be monitored and the information used to guide empiric management decisions.

11. Intravenous antimicrobial agents are appropriate for patients with severe staphylococcal infections, particularly patients requiring hospitalization. Vancomycin remains a first-line therapy for severe infections possibly caused by MRSA. Other intravenous agents such as clindamycin[54], daptomycin[110], linezolid[111], quinopristin-dalfopristin[112], tigecycline[113], and TMP/SMX[94] may be appropriate to consider in some circumstances. Some experts advocate the addition of nafcillin or oxacillin for optimal MSSA coverage in patients who are severely ill[114], although toxicity may be increased with combination therapy; clinicians should weigh the risks and benefits in individual patients. Consultation with an infectious disease specialist should be sought. Final therapy decisions should be based on results of cultures and antimicrobial susceptibility testing.

12. Patient education is a critical component of SSTI case management. Clinicians should educate patients or their care-takers, and when possible, household members, on methods to limit further spread of infection in their household and among other close contacts *(Figure 3)*. Patients that can not maintain adequate hygiene and keep wounds covered with clean, dry bandages should be excluded from activities where close contact with other individuals occurs, such as daycare or athletic practice, until their wounds are healed.

13. Clinicians should routinely ask about similar cases of SSTI in household members and other close contacts. If a potential outbreak of cases in a defined cohort outside of a single household (e.g., school, athletic team) is identified, the local public health department should be notified.

14. To date, there are no data to support the use of agents to eliminate *S. aureus* colonization, such as nasal mupirocin and antiseptic body washes (e.g., chlorhexidine), for patients with MRSA infection or their close contacts. Recognizing that efficacy data are lacking, it may be reasonable to administer decolonization regimens when (1) an individual patient has multiple documented recurrences of MRSA infection or (2) ongoing MRSA transmission is occurring in a well-defined, closely-associated cohort (such as a household). However, this should be considered only after reinforcing the standard prevention measures *(Figure 3)* and documenting that this has been unsuccessful at interrupting transmission. Consultation with an infectious disease specialist may be helpful. The local health department should be consulted if administration of decolonization regimens to a larger cohort (e.g., classroom, athletic team, group home, correctional facility) is being considered. Appropriate decolonization regimens (agents and administration schedules) have not been established for community settings. When attempting to eliminate MRSA colonization in members of a defined cohort, it has not been established whether it is preferable to obtain colonization cultures from all members of the cohort and target decolonization regimens to members with confirmed colonization or to provide regimens universally to all members of the cohort. However, all members of a cohort that receive decolonization regimens should do so simultaneously. To decrease the potential for emergence of resistance, decolonization agents should be administered only in short courses and targeted to individuals or members of well-defined cohorts with documented recurrent infections. The use of systemic antimicrobial agents for decolonization should be limited to patients with concurrent active infections.

15. Standard infection control precautions should be used for all patients in outpatient and inpatient healthcare settings. This includes performing hand hygiene (handwashing or using alcohol hand gel) after touching body fluids or contaminated items (whether or not gloves are worn), between patients, and when moving from a contaminated body site to a clean site on the same patient; wearing gloves when managing wounds; and wearing gowns and eye protection as appropriate for procedures that are likely to generate splashes or sprays of body fluids. In addition, contact precautions, which involve greater spatial separation of patients (through placing infected patients in private rooms or cohorting patients with similar infection status), use of gown and gloves for all contact with the patient or their environment, and use of dedicated noncritical patient-care equipment, have been recommended for empiric use in patients with abscesses or draining wounds in which wound drainage can not be contained[115]. Contact precautions have also been recommended for patients in acute care inpatient settings known or suspected to be infected or colonized with MRSA; these

precautions may be modified as appropriate for ambulatory care and other non-acute care inpatient settings based on risk factors for transmission. Exam room surfaces should be cleaned with an EPA-registered hospital detergent/disinfectant, in accordance with label instructions, or a 1:100 solution of diluted bleach (1 tablespoon bleach in 1 quart water)[116].

16. Patients with SSTIs treated on an outpatient basis should be clearly instructed to return promptly if they develop systemic symptoms or worsening local symptoms or if their symptoms do not improve within 48 hours. Ideally, a follow-up visit should be scheduled within 48 hours of the initial visit to confirm adequate response to therapy.

For additional assistance in clinical management of MRSA infections, consult an infectious disease specialist. For questions regarding the epidemiology of MRSA in your community, or to report a possible outbreak, contact your local or state health department.

Appendix A: Participants in the CDC-Convened Expert's Meeting on Management of MRSA in the Community

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Figure 1: MRSA skin infections

MRSA skin lesions may begin as small papules and then develop into larger pustules or abscesses with areas of necrosis and surrounding erythema. Lesions are often confused with spider bites.

Figure 2: D-zone test for inducible clindamycin resistance

Inducible clindamycin resistance can be detected through a D-zone test, which involves placement of erythromycin and clindamycin disks in close proximity on an agar plate inoculated with a standardized suspension of the isolate of interest. Flattening of the clindamycin zone of inhibition in the area between the two disks (resulting in a D-shaped zone of inhibition) indicates the presence of inducible clindamycin resistance (positive D-zone test).
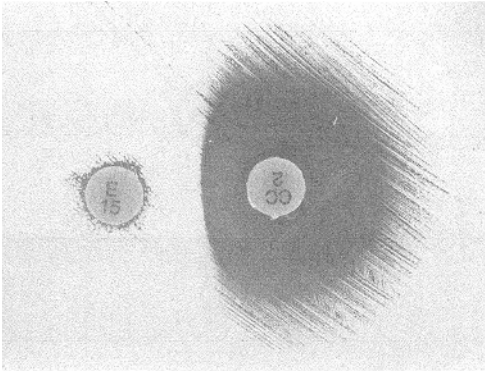
Figure 3: Key prevention messages for patients with Skin and Soft Tissue Infections

Key Prevention Messages for Patients with Skin and Soft Tissue Infections and their Close Contacts

1. **Keep wounds that are draining covered with clean, dry, bandages.**
2. **Clean hands regularly with soap and water or alcohol-based hand gel (if hands are not visibly soiled). Always clean hands immediately after touching infected skin or any item that has come in direct contact with a draining wound.**
3. **Maintain good general hygiene with regular bathing.**
4. **Do not share items that may become contaminated with wound drainage, such as towels, clothing, bedding, bar soap, razors, and athletic equipment that touches the skin.**
5. **Launder clothing that has come in contact with wound drainage after each use and dry thoroughly.**
6. **If you are not able to keep your wound covered with a clean, dry bandage at all times, do not participate in activities where you have skin to skin contact with other persons (such as athletic activities) until your wound is healed.**
7. **Clean equipment and other environmental surfaces with which multiple individuals have bare skin contact with an over the counter detergent/disinfectant that specifies *Staphylococcus aureus* on the product label and is suitable for the type of surface being cleaned[1].**