FOCUS: MRSA IN THE COMMUNITY (CMRSA)

APUA One on One
Public Health Perspectives on CMRSA
Interview with Scott Fridkin, M.D., CDC

At the Centers for Disease Control and Prevention, Scott Fridkin tracks an epidemic whose origins are elusive and contours ever-shifting. The threat of community-onset MRSA (methicillin-resistant Staphylococcus aureus) recently entered public consciousness with a February 2003 MMWR report describing three Los Angeles County outbreaks. But health officials have been watching cases ominously rise for nearly a decade.

Q: Between 1997 and 1999, community-onset MRSA killed four children in North Dakota and Minnesota. Are the organisms behind the recent MRSA outbreaks the same?
A: The outbreak strains from Los Angeles County, and from several other recent community outbreaks, are actually very distinct from the 1997 isolates. Most of the community MRSA strains that we’ve been able to evaluate fall into two main clonal groups. The 1997 North Dakota/Minnesota strains are from one, and the L.A. County strains another. It has yet to be determined if non-outbreak, or endemic, MRSA in the U.S. also belongs to these two groups.

Q: How prevalent is community-onset MRSA disease?
A: Somewhere between 5 and 15 percent occurs in persons without established risk factors.

Tracking the Spread of CMRSA

Henry F. Chambers, M.D., Professor of Medicine, University of California, San Francisco

Methicillin-resistant Staphylococcus aureus (MRSA), first encountered in 1961, is primarily a nosocomial pathogen. More recently, however, strains from community-onset infections have appeared in patients apparently lacking any nosocomial risk factors. These community-onset infections have occurred in the context of dramatically rising MRSA in US hospitals with prevalence rates approaching 50%.

A potential explanation for community cases of MRSA, therefore, is that nosocomial risk factors have been overlooked or that colonization has occurred through contact with an MRSA source case who has nosocomial risk factors. S. aureus carriage rates range between 25 and 50% and colonization may be transient or persistent, lasting for years.

Although transmission of MRSA from a hospital source into the community is an attractive and reasonable hypothesis, it fails to account for five unique features that differentiate community MRSA isolates from endemic hospital strains: 1) community isolates tend to be susceptible to antibiotic classes other than beta-lactam antibiotics; 2) genotypes of community isolates are not the same as local hospital isolates; 3) community strains harbor a novel methicillin resistance cassette element, not previously identified among endemic hospital strains; 4) community isolates are more likely to encode a putative virulence factor, Panton-Valentine leukocidin; and 5) community isolates occur in patients lacking typical risk factors for MRSA.

The methicillin resistance gene, mecA, encodes penicillin-binding protein 2a (PBP 2a), which has low affinity for binding beta-lactam antibiotics and confers class resistance by substituting for the normal, high affinity PBPs when these are inactivated by a beta-lactam antibiotic. mecA is located within a gene complex, SCCmec (staphylococcal cassette chromosome mec), a mobile element that precisely integrates into and excises from a specific site within a specific open reading frame, orfX, of the staphylococcal chromosome. While SCCmec encodes several genes, only mecA is required for methicillin resistance.

The four SCCmec types described thus far differ with respect to which of the several elements are present and how they are arranged within SCCmec (Table). Also present are two genes, ccrA and ccrB (cassette chromosome recombinase genes A and B), which have homology to DNA recombinases of the invertase-resolvase family. ccrA and ccrB catalyze the site-specific excision of SCCmec from its site- and orientation-specific integration into the S. aureus chromosome.

Before the debut of community

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Introduction

S. aureus has been, and continues to be, a significant human pathogen. In the influenza pandemic of 1918, S. aureus was the major bacterial cause of death among those who developed secondary bacterial pneumonia. Not long after the introduction of penicillin, S. aureus developed resistance to the antibiotic, becoming an important cause of serious nosocomial infection in the 1950s. The timely discovery of beta lactamase-resistant cephalosporins and later the semisynthetic beta lactam antibiotics (methicillin, oxacillin and naftillin) returned control to the clinician for the next 10-15 years. In the 1970s, sporadic reports of methicillin-resistant S. aureus (MRSA) began to appear, particularly in facilities with extremely ill patients and intense antibiotic usage. Over the subsequent 20-30 years, we have seen the widespread emergence of MRSA infections in hospitals in certain regions of Europe, throughout the United States, as well as in Japan and the Western Pacific, where the prevalence has reached 50%.

Community-acquired MRSA: genetic differences

More recently, reports of true community-acquired MRSA infections have appeared.\(^1\)\(^2\) Empiric treatment of some of these with conventional agents resulted in disastrous outcomes before it was determined that the etiologic agent was MRSA. Two lessons come from these dramatic treatment failures. First, these infections did not respond to the types of agents that most would prescribe for community-acquired S. aureus infections. Second, these MRSA were severe, highly virulent infections, resulting in death. It was assumed that patients in the community with MRSA infections must have had some type of contact with health care facilities, occult antibiotic exposure, or with someone with hospital-acquired MRSA. There is now clear genetic-based evidence that community-acquired MRSA strains are distinct from hospital-acquired MRSA\(^2\)\(^3\) (see Chambers in this issue).

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Are community-acquired MRSA strains more virulent?

Some investigations have demonstrated a two-fold increase in the prevalence of TSST-1 and staphylococcal enterotoxin in MRSA strains compared to methicillin-sensitive S. aureus strains.\(^5\) In fact, recent reports from Japan suggest an increase in the frequency of staphylococcal toxic shock syndrome caused by MRSA.\(^6\)\(^7\) In addition, the propensity of MRSA to cause more severe soft tissue infections is...
suggested by the fatal cases recently reported from the American midwest. Similarly, severe staphylococcal infections in France have been reported to be associated with the Panton-Valentine leukocidin. Toxin was found in 96% of community-acquired MRSA strains compared to 0% of methicillin-sensitive strains.

Recognition and management of community-acquired MRSA infections

How can we translate these basic science findings from the laboratory into sound clinical practice? Community-acquired staphylococcal infections fall along a broad spectrum (see Table). We should be prepared to see toxic shock syndrome associated with any type of staphylococcal infection. In the future, staphylococcal toxic shock syndrome may not be limited to menstrual cases or post-surgical cases involving packing material. Pneumonia may also be more severe, according to recent studies in France. Finally, necrotizing soft-tissue infections may become more common due to the presence of the Panton-Valentine leukocidin.

The empiric choice of antimicrobial treatment

When the prevalence of MRSA in hospitals increased to 40%, empiric treatment of staphylococcal infection demanded administration of agents such as vancomycin, to which the organism remained susceptible. Selection of an effective antibiotic regimen also required that clinicians know the prevalence of MRSA in their specific hospitals. The same need holds true in community-acquired MRSA infections.

In evaluating seriously ill patients with community-acquired staphylococcal infection, we cannot assume that nafcillin or a cephalosporin will be an effective treatment. It will be prudent to assume that the infection is caused by MRSA and begin empiric treatment with agents effective against this organism. In less seriously ill patients, conventional treatment may be sufficient, but such therapy may fail and careful clinical follow-up will be necessary. More than ever, making a correct diagnosis and obtaining good culture material for susceptibility testing will be crucial for rational antibiotic selection and switching. Should community-acquired MRSA strains be isolated, careful follow-up, monitoring of the patient's clinical course, and a prompt change of therapy will be necessary to ensure good outcomes.

Considerations in the selection of specific anti-MRSA antimicrobial agents

- Skin and soft tissue infections: Clinical trials have documented the efficacy of linezolid, vancomycin, and quinupristin-dalfopristin in skin and soft tissue infections.
- Pneumonia: There may be some advantage for linezolid due to its excellent penetration into lung alveolar fluid.
- Bacteremia: Linezolid and vancomycin have been equivalent in treatment of MRSA-related bacteremia. Other studies are underway with quinupristin-dalfopristin.
- Endocarditis: Sadly, new information is lacking. Most clinicians favor using bactericidal agents such as vancomycin or quinupristin-dalfopristin. There have been numerous papers describing failures with vancomycin, but little comparative data with the other agents.
- Toxic Shock Syndrome: No clinical trial has specifically addressed antibiotic treatment of staphylococcal toxic shock syndrome. Suppression of staphylococcal toxin production has been demonstrated with linezolid in vitro, and this may be an advantage in

**Table: Treatment of Suspected Community-Acquired MRSA Infection**

<table>
<thead>
<tr>
<th>Non Toxic</th>
<th>Toxic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Localized Soft-Tissue Infection</strong></td>
<td><strong>Pneumonia</strong></td>
</tr>
<tr>
<td>Linezolid</td>
<td>Linezolid</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>Quinupristin-dalfopristin</td>
</tr>
<tr>
<td>Trimethoprim-sulfamethoxazole(a)</td>
<td>Tetacycline(a)</td>
</tr>
</tbody>
</table>

(a) Many strains of MRSA are susceptible, but individual results should be verified
(b) Linezolid reaches very high alveolar space concentrations
(c) Efficacy of linezolid and vancomycin were equivalent for MRSA bacteremia
(d) Need more data; most would favor a bactericidal agent such as vancomycin, quinupristin-dalfopristin, or daptomycin
(e) No clinical data

ASK the EXPERT continued on page 6
M. RSA and the subsequent discovery of type IV SCCmec, horizontal transfer of methicillin resistance in nature was thought to be a relatively rare event. All MRSA strains worldwide were considered descendants from a single ancestral clone. However, using pulsed field gel electrophoresis (PFGE) plus ribotyping, Hiramatsu identified at least three distinct chromosomal backgrounds or lineages, A, B, and C that had acquired SCCmec. Of the three known SCCmec types, I, II and III, lineage A was found to harbor any of the three; B and C each harbored type II. Thus, MRSA worldwide could be assigned to one of five "clonotypes."

SCCmec types I, II and III, the first three to be identified, were almost exclusively from nosocomial MRSA isolates collected prior to 1990. These SCCmec types are relatively large, 34,000 bp or greater. Too large for bacteriophage transduction, they are thought to be poorly transferable, and without a sequence that would allow co-transfer by a conjugative plasmid. On the other hand, type IV SCCmec is potentially transferable. The lack of transposons, integrated plasmids, and other antibiotic resistance genes (Table) explains in part the greater susceptibility of community MRSA compared to nosocomial strains, and makes the element small enough for horizontal transfer by phage transduction.

As more strains were examined and higher resolution methodologies employed, the mobility of SCCmec became more apparent.

As more strains were examined and higher resolution methodologies employed, the mobility of SCCmec became more apparent. Fitzgerald, et al analyzed 369 MRSA (once again, mostly nosocomial) strains collected from 20 countries between 1961 and 1999. They found 11 major MRSA clones (i.e., a specific sequence type, or genetic background, plus the specific SCCmec type integrated into this background). The sequence types could be ordered into one of five groups of closely related genetic backgrounds constituting five clonal complexes: CC5, CC8, CC22, CC30, and CC45. Types I and III SCCmec were restricted to two clonal clusters, CC5 and CC8; type II was found in all but CC22; type IV was associated with all five types and one additional sequence type, ST59, that appeared unrelated to the other five clonal complexes. MLST analysis of community MRSA isolates from the United States and Australia has shown that, although many are related to known methicillin-resistant clonal complexes (e.g., CC8 and CC30), many are members of yet another one, CC1, and that type IV SCCmec is present in almost all. In fact, no type I, II or III was identified among community strains.

### Table: Elements Found Within the Four Types of SCCmec.

<table>
<thead>
<tr>
<th>GenBank Number</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. aureus</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Size</td>
<td>34,364 bp</td>
<td>53,017 bp</td>
<td>66,896 bp</td>
<td>20,920 bp</td>
</tr>
<tr>
<td>mecA</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>mecR1-mecI</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>ccrAB</td>
<td>+*</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>pUB110</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>pT181</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>IS431</td>
<td>1</td>
<td>2</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Tn554</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>ermA</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>tetK1, Hg resistance</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>aadD</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

plus sign (+) = present, minus sign (-) = absent; mecA = gene encoding PBP 2a, the low binding affinity penicillin-binding protein that mediates methicillin resistance; mecR1-mecI = sensor-transducer and repressor genes that regulate production of inducible PBP 2a; ccrAB = cassette chromosome recombinases A and B that mobilize the mec element; pUB110 = integrated plasmid that encodes aadD, the gene for tobramycin and kanamycin resistance; pT181 = integrated plasmid that encodes tetracycline resistance; IS431 = insertion sequence; Tn554 = erythromycin resistance-encoding transposon; tetK1 = tetracycline resistance gene; Hg = mercury resistance determinant.

* Present but incapable of excising and mobilizing SCCmec.

In addition to these differences, community MRSA contain the gene for Panton-Valentine leukocidin, a toxin associated with severe, necrotizing pneumonia in children and a propensity to cause skin and soft tissue infections. This toxin, and perhaps others, are speculated to account for the deaths reported in children with community-onset MSRA infection, as well as for...
reported outbreaks of abscesses, often persistent and unresponsive to therapy.6

Although urgently needed prevalence rates are hard to come by, MRSA in the community appears to be increasing, particularly in some geographical regions in the United States. Alongside the heavy burden of hospital MRSA, which are major contributors of MRSA to the community, is a parallel epidemic of true community strains. The mobility of SCCmec type IV, in addition to strain mobility, is likely fueling the emergence of MRSA in the community.

References

Q: Have you seen hot spots in the U.S.?
A: Outbreaks have occurred across the U.S. in different populations. It is not likely that community-onset MRSA occurred in one single place at one time and spread. The fact that there are at least two different clonal groups suggests that the emergence of this smaller resistance element in the community occurred probably around the same time in distinct places. Without ongoing active surveillance, it's impossible to say where it occurred first or where the hotspot is.

Settings that appear to amplify transmission include correctional facilities and competitive sports teams. We've had outbreaks on native American reservations, both in Alaska and in the lower 48 states. Most recently, the L.A. County health department has been evaluating men who have sex with men.

Q: Are any individuals or population groups especially at risk?
A: It is unclear if any particular individuals may be at higher risk for carrying this community MRSA. What is clear is that not only do you need to be exposed to MRSA to develop disease, but you need to have some sort of inoculation injury: breakdown in skin. With competitive sports, there are lots of opportunities for skin breakdown. The same may be true for inmates as well as men who have sex with men. We need to identify what behaviors may be associated with disease — tattooing is something that we're looking at. At the same time, we also see MRSA disease in persons without established risk factors who are not part of an outbreak.

Q: How are the new community MRSA strains transmitted?
A: Staph aureus is transmitted through close contact — we've known that for decades. In the community, substantial close contact with infected material, such as pus, is probably the greatest risk. Investigations have found that close contact, touching infected wounds or boils, changing someone else's dressings, and sharing personal items were all risk factors for subsequent development of MRSA skin disease. We need more awareness about avoiding contact with potentially infectious materials such as pus or infected wounds, proper handwashing, and removal of barriers to appropriate hygiene.

Q: How can our public health system better track community MRSA?
A: MRSA is not a reportable disease. And there's no requirement for strains to be submitted and analyzed systematically. But there's been a major shift in interest among public health authorities. The Council of State and Territorial Epidemiologists will be discussing a resolution this summer regarding making MRSA reportable in some fashion. That would greatly aid our ability to track MRSA and systematically study MRSA isolates to determine if there are biologic differences that explain changing epidemiology. We're currently collaborating with health departments in over 20 states to systematically look at MRSA by pulsed field gel electrophoresis.

Q: How will community-onset MRSA evolve?
A: It will likely increase unless we aggressively identify effective prevention measures. Clinicians need to be aware that MRSA is in the community, and they must use appropriate diagnostics and therapeutic measures. The worst case scenario is a dramatic change in the way we approach patients in the community who have skin and soft tissue disease. If we assume they have MRSA, it’s going to make treatment more difficult and more expensive.

Dr. Scott Fridkin, M.D., Medical Epidemiologist, Division of Healthcare Quality Promotion, National Center for Infectious Diseases, U.S. Centers for Disease Control and Prevention was recently interviewed by APUA Newsletter associate editor Madeline Drexler.
APUA National Roundtable on UTIs

On June 10, 2003, APUA convened a group of leading experts in Boston at a meeting entitled “UTI Treatment at a Turning Point: Improving Antibiotic Prescribing for Uncomplicated UTIs in an Era of Increasing Antibiotic Resistance”. Participants will collaborate on several forthcoming scientific articles. Look for meeting summary on the APUA website: www.apua.org.

New APUA Advisory Board Member

APUA welcomes a distinguished new member to its Scientific Advisory Board. Dr. Thomas E. Wellem is Acting Chief, Laboratory of Malaria and Vector Research, NIAID, NIH.

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APUA Honors Leaders at ICAAC

APUA will hold a member reception and present the 2003 APUA Leadership Awards at the 43rd Annual Interscience Conference on Antimicrobial Agents and Chemotherapy (ICAAC), September 14-17, 2003, in Chicago, IL. The reception will be held at the Hyatt Regency McCormack Place in the Atrium from 6:00 to 7:30 pm on September 15. Award recipients are selected based on leadership and collaboration to preserve the power of antibiotics. On September 16, the Steering Committee for the Global Advisory on Antibiotic Resistance Data (GAARD) will meet to discuss current surveillance projects. Additionally, APUA will exhibit jointly with CDC at ICAAC.

Funds Available to Investigate ABR in Commensal Organisms

Reservoirs of Antibiotic Resistance (ROAR II), funded by the NIAID and coordinated by APUA, is calling for research proposals that will address the hypotheses that commensal organisms serve as reservoirs for the emergence and proliferation of antibiotic resistance genes in disease-associated bacteria, and that commensal bacteria can donate antibiotic resistance elements to disease-associated bacteria. Awards up to $60,000 for 1 year, with possible renewal for the 2nd year are considered pilot grants to obtain data useful in addressing these hypotheses and in developing a more comprehensive study. For more information on the ROAR project, the request for proposals (RFP) and proposal guidelines, visit our web site at www.apua.org or contact at Allison.Hodges@tufts.edu 617-636-4021

Deadline for submission: October 15, 2003

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References
News from the APUA International Chapter Program

Recently, Iruka Okeke, Ph.D., APUA specialist on AMR for Africa, spoke on Voice of America Television (VOA) for Africa about antimicrobial resistance.

Jawahar S. Bapna, M.D., Ph.D., APUA-India chapter coordinator, discussed antimicrobial resistance issues in India on VOA.

APUA and the European Society of Clinical Microbiology and Infectious Diseases (ESCMID)

Anibal Sosa, APUA International Program Director, participated as faculty in the 22nd ESCMID Post-Graduate Education Course “Measuring, Auditing and Improving Antimicrobial Prescribing,” held in Troon, Scotland, May 9-10, 2003. The course was coordinated by Dominique L. Monnet, Pharm.D., Ph.D. from Statens Serum Institut in Denmark and Prof. Inge C. Gyssens, from the Erasmus CC Rotterdam, The Netherlands.

APUA Regional Chapter Meetings

An APUA chapter network meeting was held in Glasgow, Scotland on May 11, 2003. Dr. Gunnar Simonsen, a member of the WHO AMR team, spoke.

APUA chapters in attendance were: Croatia, United Kingdom, Poland, Greece, Bulgaria, Italy, Germany, Rumania and Saudi Arabia.

APUA at the Pan American Congress of Infectious Diseases

On May 14, 2003, APUA coordinated a symposium in collaboration with the Pan American Health Organization (PAHO) and the Pan American Society of Infectious Diseases. Symposium chairs were Anibal Sosa, M.D., APUA International Program Director and Liliana Clara, M.D. See www.apua.org for proceedings and presenters.

APUA Welcomes Four New Chapters

APUA United Kingdom:

In collaboration with the British Society for Antimicrobial Chemotherapy (BSAC) and led by Prof. Peter Davey, Professor in Pharmacoeconomics MEMO, Department of Clinical Pharmacology, Ninewells Hospital, Dundee, Scotland, BSAC has developed a web page for the APUA UK chapter. Visit www.bsac.org.uk.

APUA Philippines

Led by Célia C. Carlos, M.D., Chair, Committee on Antimicrobial Resistance Surveillance, Research Institute for Tropical Medicine, Department of Health, Filinvest Corporate City, Alabang, Muntinlupa, Metro Manila, Philippines.

APUA Fiji Islands

Led by Dr. Kamal Kishore, M.D., Senior Lecturer, Medical Microbiology, Fiji School of Medicine, Fiji Islands.

APUA Lebanon

Led by Dr. Jacques Mokhbat, M.D., Chairman of the Department of Medicine, Lebanese University Faculty of Medical Sciences, Dekwaneh, Lebanon.

APUA Grassroots Research in Developing World

APUA-Nepal

Judd Walson, M.D., APUA specialist on AMR for the Asian region, has traveled to Kathmandu, Nepal to work with Shyam P. Lohani, MPharm on an analysis of antibiotic use indicators in national care health facilities in Kathmandu.

APUA-Lebanon

APUA provided support for APUA-Lebanon’s study entitled “Carriage of MRSA in healthy medical students.”

APUA-Guatemala

In collaboration with AB Biodisk ARTIST project, APUA has provided support for a study “Risk Factors for Antibiotic Resistance of Strptococcus pneumoniae among Guatemalan Children from Different Socio-Economic Strata and Health Care Delivery Systems: Preliminary Surveillance for a Program for the Judicious Use of Antibiotics in the Outpatient Setting.”

For more information on APUA international grassroots research, visit our website at www.APUA.org/chapters.
Alliance for the Prudent Use of Antibiotics  
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If you are concerned about the public health threat of antibiotic resistance, become part of the solution. Make a tax-deductible contribution and join our global network of citizens, clinicians, researchers and policy makers.

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Founded in 1981 as a nonprofit organization, APUA is the only organization in the world solely dedicated to strengthening society's defenses against infectious diseases through research and education on antibiotic use and antibiotic resistance. APUA's mission is to improve infectious disease treatment and control worldwide through promoting appropriate antibiotic access and use and reducing antibiotic resistance. With members in over 100 countries and numerous foreign affiliated chapters, APUA provides a unique network to support country-based activities and facilitate international planning and communication. APUA's resources include an international scientific advisory board with members of national academies of medicine and science and a professional staff with specialized expertise. APUA's global network of affiliated chapters serves to tailor interventions to local customs and practices.

... preserving the power of antibiotics™ throughout the world.

★ Headquarters
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