**Methicillin-Resistant Staphylococcus aureus**

By Karla Givens

**Means of Transmission and Usual Reservoirs**

Staphylococcus aureus is part of normal flora and can be found on the skin and in the noses of one in every three people. In most instances, it is harmless, but does have the potential to cause serious or even fatal infections. Unfortunately, the overuse of antibiotics has given rise to methicillin-resistant *Staphylococcus aureus*, or MRSA, which is a subspecies of staphylococcus aureus that is resistant to beta-lactam antibiotics. The CDC estimates that two in every 100 people carry the bacterium.

MRSA can be spread by skin-to-skin contact between individuals or contact with contaminated objects. While it is mostly hospital acquired (HA-MRSA), it can also be community acquired by healthy individuals, such as sports team members who frequently have close contact. (CA-MRSA)

**Etiologic Agent, Its General Characteristics and Key Tests for Identification**

As with Staphylococcus aureus, MRSA is a gram-positive, catalase positive, coccal bacterium, which causes infection when it enters a skin lesion or wound. The cocci are approximately 0.5-1.5 μm in diameter and form clusters. The bacteria is non-spore forming, facultative anaerobic, and non-motile.

MRSA is detected using broth microdultion. The Clinical and Laboratory Standards Institute also recommends the cefoxitin disk screen test, the latex agglutination test for PBP2a, or Mueller-Hinton agar with 4% NaCl and 6 μg/ml of oxacillin. Selective chromogenic agars are also available. Molecular tests, such as the PCR, are also available and can detect the presence of MSRA within hours by locating genetic markers for S. aureus and the mecA gene, which confers resistance to most popular antibiotics.

**Historical Information**

Staphylococcus aureus was discovered in 1880 by Scottish surgeon Sir Alexander Ogston. He used the greek words Staphyl (bunch of grapes) and kokkos (berries) to describe the bacteria. For years later, German physician Friedrich Julius Rosenbach added aureus (gold) to the strain, which appears in gold colonies when cultured on media.

Infections frequently resulted in death until 1941 when penicillin was discovered and used for treatment. The bacterium developed a resistance to penicillin throughout the 1950’s, and
methicillin was introduced in 1961 as a better alternative. The first MRSA strains were isolated in Britain late that year and first reported in the United States in 1968.5

**Signs and Symptoms of MRSA**

Infection can be described as small red bumps, boils, or sores on the skin that is red, swollen, painful, warm, or accompanied by pus, often resembling a spider bite.1 The bumps can become deep abscesses or make their way into the body and cause greater infection.2 Soft tissue, respiratory, bone, joint, and endovascular infections may occur, as well as sepsis.8

**Microbial Virulence Mechanisms Contributing To The Disease Process**

MRSA strains are always accruing further antibiotic resistance, leading to highly virulent bacterial strains. Mobile genetic elements called staphylococcal cassette chromosome mec (SCCmec) are DNA fragments that include the mecA gene. The mecA gene codes for low affinity binding protein PBP2a, which is a protein that cannot be inhibited by beta-lactam antibiotics, and thus making the bacteria resistant to methicillin. There are currently 11 types of SCCmec elements.8

There are 5 colonial complexes of MRSA clones: 5, 8, 22, 30, and 45. Many of these clones are also resistant to other antibiotics such as erythromycin, clindamycin, ciprofloxacin, and tetracycline.8

The virulence of MRSA can be attributed to toxins, adhesion of surface proteins, regulators, and immune evasion. MGE-encoded toxins vary among strains and include toxic shock syndrome toxin, Panton-Valentine leukocidin, and exfoliative toxins. Global regulatory system, Agr, controls the expression of numerous toxin genes as well as surface proteins, which can be mutated and cause significant differences in pathogenic potential. Staphylococcus aureus surface protein X (SasX) binds to nasal epithelial cells. The horizontal gene transfer is shown to contribute to nasal colonization, biofilm formation, and immune evasion and virulence.8 In the last decade, CA-MRSA has gained greater virulence than HA-MRSA. The reasons as to why this occurs are still being debated.8

**Control, Treatment, and Prevention for the Disease**

MRSA can be controlled by taking certain precautionary steps to avoid infection. The CDC recommends covering all wounds, washing hands often, not sharing personal items such as razors and clothing, and washing sheets and clothes often and efficiently.1 The Mayo Clinic also recommends showing after activities with physical contact, such as sporting events.7

Healthcare providers can drain an abscess or boil as a first line of defense to treat skin infections. Often, antibiotics may not be needed. For severe infections, vancomycin may be administered intravenously.2 Few cases of vancomycin resistance have been documented.8

Last month, a new antibiotic called teixobactin was shown to successfully treat mice affected with MRSA. Similar to vancomycin, the new drug works by breaking down the cell wall of bacteria. Researchers say it also attacks growth processes, which may prolong resistance.9
The CDC provides contact precaution guidelines for healthcare providers to prevent or reduce the incidence of MRSA. These include patient placement, wearing disposable gloves and gowns, limiting patient transfers, using disposable patient equipment, and limiting group activities for those possibly infected.\(^1\)

### Current Cases or Outbreaks

The CDC’s Emerging Infectious Program and National Healthcare Safety Network provide data to estimate the prevalence of MRSA in our nation in the table below.

<table>
<thead>
<tr>
<th>Epidemiologic Category</th>
<th>Estimated Cases of Infection</th>
<th>Incidence Rate (Confidence Interval)(^2)</th>
<th>Estimated No.</th>
<th>Incidence Rate (Confidence Interval)(^3)</th>
<th>Estimated No.</th>
<th>Incidence Rate (Confidence Interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CA</td>
<td>15,138</td>
<td>4.82 (3.69-6.42)</td>
<td>NA</td>
<td>NA</td>
<td>15,138</td>
<td>4.82 (3.69-6.42)</td>
</tr>
<tr>
<td>HCA</td>
<td>44,771</td>
<td>14.29 (12.40-16.62)</td>
<td>14,041</td>
<td>3262.39 (2495.82-4247.12)</td>
<td>58,812</td>
<td>18.74 (15.81-22.42)</td>
</tr>
<tr>
<td>HCA-HO</td>
<td>11,493</td>
<td>3.67 (2.73-5.02)</td>
<td>1,408</td>
<td>327.24 (131.31-739.07)</td>
<td>12,901</td>
<td>4.11 (2.90-6.02)</td>
</tr>
<tr>
<td>HCA-HACO</td>
<td>61,268</td>
<td>10.62 (9.06-12.55)</td>
<td>12,633</td>
<td>2936.08 (2215.08-3976.65)</td>
<td>45,911</td>
<td>14.63 (12.09-17.85)</td>
</tr>
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<td>Overall(^c)</td>
<td>65,296</td>
<td>19.5(^4) (17.24-22.31)</td>
<td>14,041</td>
<td>3263.31 (2495.12-4248.98)</td>
<td>75,309</td>
<td>23.99 (20.64-28.10)</td>
</tr>
</tbody>
</table>

Since 2005, HA-MRSA cases have decreased by 54.2% while CA-MRSA only decreased by 5.0%. A total of 30,800 fewer cases occurred in 2011 compared to 2005. Despite the reduction in infection numbers, MRSA continues to be one of the most antimicrobial-resistant pathogens. While improvements in hospitals are being made to decrease incidence, concern is heightening for community acquired MRSA throughout the world.\(^10\)

Around the globe, different strains can be seen affecting different areas. This can be attributed to different health practices.\(^8\)
Below, The Center for Disease Dynamics, Economics and Policy shows most recent data for MRSA prevalence in 2004.\textsuperscript{11}

References


