The Threat of MRSA

A report from the American Society for Microbiology

American Academy of Microbiology
Recognizing Scientific Excellence
The Threat of MRSA

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Edited by Jeffrey Fox

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Cover: Staphylococcus aureus bacteria, MRSA. Image retrieved from Microbe World.
FAQ: The Threat of MRSA

What is *Staphylococcus aureus* and what types of infections does it cause?

Bacteria, also known as microorganisms or as microbes, play a substantial role in our lives, acting beneficially but also causing harm. Some bacterial species help in making vitamins in our gastrointestinal tract, assist in breaking down foodstuffs, and stimulate our immune systems in a healthy manner. However, disease-causing bacteria called pathogens, can make us ill and, if not treated and controlled, can kill us.

One such bacterial pathogen is *Staphylococcus aureus* (SA). Although these bacteria sometimes live harmlessly on the human body, enjoying a status known as commensal, they can gain access into the body through a cut, abrasion, or wound, resulting typically in minor skin and tissue infections in healthy individuals. If the infection goes unchecked, however, SA can invade more deeply, leading to more serious disease, including complicated soft tissue infections as well as infections of the bone, blood, and heart-valves. These infections can progress, leading to organ failure and death. The extent of these complications is dependent upon both the properties of the infecting SA bacteria and the immune responses of the infected individual. In that sense, SA can be considered an opportunistic pathogen, one that awaits the right opportunities to shed its commensal status, invading our bodies and causing frank disease.

*Bolded words are defined on p 5*

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**National Estimated Incidence Rates of Invasive MRSA Infections, Stratified by Epidemiologic Category**

<table>
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<th>Year</th>
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<th>Health care-associated community onset</th>
<th>Hospital onset</th>
<th>Community associated</th>
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Data are given for methicillin-resistant *Staphylococcus aureus* (MSRA) infections reported to the Emerging Infections Program-Active Bacterial Core surveillance (United States, 2005-2011).

*Defined as MRSA isolated from a normally sterile source.*

Image retrieved from Dantes et al 2013 (15).
The principal habitat of SA as a commensal of humans is the nose, but this organism is found at other anatomical sites, including the skin and throat of healthy people. According to the Centers for Disease Control and Prevention (CDC), one in three people is colonized with SA in the nose (1). Although individuals who are carriers of SA have these bacteria on and in the body, they do not show signs of infection. Individuals who are SA carriers can serve as reservoirs from which this microorganism is disseminated to other people. Moreover, some people may become colonized with SA for a few hours or days, while others are colonized for months to years.

Being colonized with SA generally precedes becoming infected with this microorganism. However, all humans, irrespective of their carrier status, are capable of being infected with SA. SA infections in healthy persons typically are superficial skin and soft tissue infections such as skin sores, pimples, boils, and mild ulcers, although severe types of infections can arise from those originating in the skin, including complicated deep soft tissue infections and also bone, blood, and heart-valve infections.

Figure 1

Crude schematic of a Staphylococcus aureus bacterial cell illustrating its mechanisms for attachment to human host cells and virulence.

Image adapted from Lowy 1998 (2).
SA is capable of causing such infections because it encodes virulence factors in its DNA, including toxins such as α-hemolysin, α-type phenol soluble modulins, and Panton Valentine Leukocidin (PVL). SA also produces adhesion molecules that enable these bacteria to attach to human cells (See Figure 1 and Text box 1). These virulence factors act in several different ways to damage our cells including the induction of damaging pro-inflammatory responses by the human immune system, and the prevention of our immune systems from recognizing these bacteria and mounting an attack against them (2).

The scientific name “staphylococcus” derives from the Greek, “stafle,” meaning wine grape because, under a light microscope, these bacteria look like grape clusters after they are subjected to a procedure known as the Gram stain (Figure 3A). The “aureus” component of its name means golden, reflecting its appearance when it is cultured in the laboratory. Clinical laboratories use a gel-like growth medium for culturing organisms, on which SA appears as round golden spots, called colonies (Figure 3B).

**Coagulase** – an enzyme made by SA that assists in clotting blood; this activity of SA is so distinctive that it is considered a traditional marker in the clinical laboratory for identifying SA and differentiating this microorganism from other staphylococcal species (Figure 2)

**Clumping factor** – an enzyme from SA that, in combination with coagulase and in the presence of blood, creates fibrin, a meshwork that prevents the binding of immune cells at infection sites

**Collagen binding protein** – this protein promotes adherence of SA to collagen in the human host; important in the pathogenesis of septic arthritis

**Fibronectin binding protein** – attaches to human host cells and more specifically the human host cell protein, fibronectin

α-hemolysin – creates a pore in human blood cells that causes a water imbalance resulting in death of the cell; induces pro-inflammatory effects

α-type phenol soluble modulins – recruits immune cells to the site of infection and then disrupts them

Panton Valentine Leukocidin (PVL) – causes immune cell destruction and tissue necrosis

**Enterotoxin** – released by SA and causes food poisoning symptoms

**Toxic Shock Syndrome Toxin-1** (TSST-1) – released by SA and causes toxic shock syndrome

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**Text box 1.**

**Characteristics of SA that contribute to its success as a pathogen**

**Protein A** – extends from the surface of the bacterium and prevents the opsonization function of the immune system. Opsonization is an immune system response during which foreign molecules are coated before being digested and eliminated by immune cells. Protein A camouflages SA, enabling it to avoid opsonization.

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**Figure 2**

Coagulase test commonly used in the clinical laboratory to differentiate *S. aureus* from other less pathogenic species of *Staphylococcus*. *S. aureus* is a coagulase-positive organism (indicated by the top image, cloudy appearance; A) whereas all the other staphylococcal species that colonize humans are coagulase negative (bottom image, clear appearance; B).

MRSA, an acronym for methicillin-resistant *Staphylococcus aureus*, is shorthand for a type of SA that is resistant to the beta (\(\beta\))-lactam class of antibiotics. According to the Centers for Disease Control and Prevention (CDC), MRSA is among the highest of all threats to humans of antibiotic resistant pathogens (3). Although MRSA causes serious infectious disease, many antibiotics remain available for treating it effectively. As the numbers of severe invasive MRSA infections acquired in healthcare settings continue to decline, the numbers of such infections among the general public continue to rise (3) (See chart p 1).

Historically, SA rapidly developed resistance to antibiotics as they were brought successively into clinical use, and doctors came to recognize resistance to each such new drug within months to years of its introduction. Thus, for example, penicillin was first used for treating SA infections in 1940. The first penicillin resistant SA pathogens emerged by 1942, and SA remains resistant to that original type of penicillin and to other members of this class of antibiotics (4).

**Figure 3**

Microscopic and phenotypic representations of *Staphylococcus aureus*. A) Grape-like (circular) clusters B) Gold colonies displayed on enriched culture media.

Less than one year after methicillin was developed and put into clinical use, resistance among SA bacteria was being widely detected. SA becomes MRSA after acquiring genes that encode resistance to the broader class of β-lactam antibiotics. The specific gene taken up by SA to make it resistant to methicillin is called meca. This gene is carried together with a larger set of genes, known as SCCmec (staphylococcal cassette chromosome), that is mobile and capable of inserting into the DNA of other SA bacteria. MRSA strains can also carry resistance to antibiotics other than members of the β-lactam class. This multidrug resistance makes the infections caused by these SA strains very difficult to treat (See Figure 5, showing the evolution of resistance within SA and MRSA strains).

In addition to producing toxins and virulence factors, MRSA bacteria can generate biofilms, sticky layers and clumps of cells that enable bacteria within them to adhere to medical devices such as catheters and implants. A biofilm protects the bacteria within it from the actions of antibiotics and of the human immune system (4). The protective strategies used by MRSA strains to evade the host immune system in

Figure 4

SA and MRSA colonization hotspots. These are areas that are typically warm and moist.

Key terms used throughout text.

**Anthroponosis** – a disease that can be transmitted from a human to an animal

**Beta (β)-lactam antibiotics** – antibiotics with a characteristic 4-membered, ring structure that block synthesis of bacterial cell walls

**Clone** – genetically identical bacteria that arose from a single common bacterial ancestor

**Colonization** – the presence of bacteria on or within the body but without signs of illness or disease (asymptomatic). Common SA colonization sites include anywhere on the surface of the skin but with preference for warm, moist regions such as the nose, armpits, and groin. No treatment is necessary for colonization (Figure 4).

**Commensal** – a microorganism that lives on another species such as an animal or a person without causing it harm

**Culture specimen** – a sample of a bodily fluid such as blood, urine, or sputum, or a swab of an anatomic site or wound that is sent to the laboratory for testing to determine what microorganisms may be present

**Gene** – a unit of heredity that, when passed from a bacterial species to its clones (offspring), imparts a specific characteristic or function

**Genome** – the total genetic material of an organism

**Infection** – the classical signs of clinical illness such as redness, inflammation, localized pain, and fever following invasion of bacteria into the body

**Opportunistic pathogen** – an organism that exists harmlessly on or in its host but may cause disease when it moves to other anatomic sites or when the host immune system becomes compromised

**Pathogen** – a specific causative agent of infectious disease such as a bacterium, virus, fungus, or parasite

**Strain** – a genetic variant or subtype of a microorganism; there are many strains of SA bacteria that vary in terms of virulence, pathogenicity, transmissibility, and resistance to antibiotics. Bacterial “typing” includes methods that distinguish among and thus identify different strains from one another (See Text box 2).

**Zoonosis** – an infectious disease that can be transmitted from an animal to a human (e.g., SA, rabies, anthrax, ringworm)
combination with multidrug resistance all contribute to the virulence of MRSA. Patients with these infections can have high morbidity and mortality rates, and caring for them contributes to high healthcare costs for these patients (due to treatment difficulties and longer hospital stays) (4).

An additional challenging feature of MRSA bacteria is their hardiness. They can survive for prolonged periods on surfaces and objects such as sinks, toilets, door handles, floors, medical devices, bed linens, cleaning equipment, and clothing. Therefore, extensive contact control and disinfection procedures are needed to limit the spread of MRSA in healthcare and other settings.
MRSA strains vary in terms of their virulence, colonization capabilities, transmissibility, and degree of resistance to antibiotics beyond those belonging to the β-lactam class of antimicrobial drugs. The composition of SCCmec, the set of genes containing the methicillin resistance gene, also differs among the many kinds of MRSA strains that circulate in different environments.

MRSA strains can be categorized by using one or more of several widely available analytical methods (See Text box 2). One of these methods is called pulsed-field gel electrophoresis (PFGE). It separates bacterial DNA fragments into specific patterns that are distinctive and thus useful for identifying the bacteria from which they came, much like fingerprints are used to identify specific individuals. MRSA banding patterns typically fall into one of eight categories, designated USA100 – USA800, according to the national PFGE database for SA, established by the CDC (5). Most infections caused by healthcare-associated MRSA (See below) fall under the USA100 category, while those caused by community-associated MRSA (See p 8) fall within the USA300 banding pattern.

The spread of MRSA requires skin-to-skin contact with an individual who either had close contact with MRSA (by touching wounds, dressings, or other objects that came into contact with MRSA-infected patients) or is colonized with this organism. MRSA can be divided into groups, called “epi-vars,” based on the population that the organism tends to infect, its origin, and route of transmission. Here, we describe three major “epi-vars” (See also Table 1):

1. **Healthcare-associated (HA)-MRSA** infections are transmitted commonly in healthcare institutions dealing with acutely ill patients (2). In many countries, MRSA infections are acquired primarily in healthcare settings, including hospitals, long-term care facilities, dialysis centers, and other health-related centers. The proportion of MRSA infections occurring outside healthcare

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**Text box 2. Methods for classifying MRSA strains**

- Pulsed-field gel electrophoresis (PFGE)
- Multi-locus sequence typing (MLST)
- spa typing
- SCCmec typing

* A full description of these terms is beyond the scope of this FAQ. For more information, please read DeLeo and Chambers (2009) (5).
settings has changed since the late 1990s in many countries, especially in North America, where MRSA infections in the community have become increasingly common (6, 7, 8). The primary mechanism for transferring MRSA among patients, caretakers, and others in these settings is through poor hand hygiene and lack of consistent disinfection procedures. In healthcare facilities, items such as catheters, intubation tubes, and other implanted devices provide MRSA a means to invade patients. Surgical and other procedures that disrupt the skin and other body surfaces are additional important avenues for MRSA to access otherwise protected body sites.

2. Community-associated (CA)-MRSA infections are transmitted from sources within a community that are not healthcare-related. The dominant CA-MRSA bacterial strain that continues to circulate in the US is referred to as USA300. As in healthcare settings, MRSA is transmitted within the community through skin contact or the sharing of items with extensive skin contact such as lotions, soaps, cosmetics, towels, razors, hairbrushes, and nail files. Some MRSA strains are shared among household members or among members of athlet-

<table>
<thead>
<tr>
<th>Setting</th>
<th>Risk Factor*</th>
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| Healthcare-associated (HA)-MRSA  | • Prolonged hospital stay  
|                                  | • Previous antibiotic use                        |
|                                  | • Surgical procedure                             |
|                                  | • Burn wounds                                    |
|                                  | • Indwelling devices                             |
| Community-associated (CA)-MRSA   | • Participation in tackle/high collision sports   |
|                                  | • Dormitory or military barracks residence        |
|                                  | • Activity in athletic clubs, locker rooms, and other community settings |
|                                  | • Daycare centers                                |
|                                  | • Correctional facilities                        |
|                                  | • Sharing of personal items                      |
|                                  | • Injection drug use                             |
| Livestock-associated (LA)-MRSA   | • Farm employee                                  |
| *Incomplete or ineffective hygienic practices are a risk factor for all types of MRSA
ic clubs or people within other community settings where individuals come into close contact, including dormitories, daycare centers, barracks, jails, locker rooms, gyms, and other facilities used by sport teams. Those that participate in high impact and collision sports such as football, soccer, rugby, ice hockey, wrestling, and basketball tend to have a higher than average likelihood of contracting CA-MRSA. Other risk factors for a CA-MRSA infection in the US include lifestyle (intravenous drug users are highly susceptible), low socioeconomic status that may prevent proper hygiene, and having other health conditions (HIV, cancer, dialysis). Infection risk for both CA- and HA-MRSA is increased for individuals who carry MRSA or who have had a previous MRSA infection.

CA-MRSA strains can vary from HA-MRSA strains in terms of their virulence factor profiles (9). For example, CA-MRSA strains tend to produce more α-type phenol soluble modulins and α-hemolysin compared to HA-MRSA strains (10). The PVL toxin is more commonly seen with MRSA strains circulating within communities but not within hospital or other healthcare settings (4).

Figure 6

Transmission of MRSA infections. Distinguishing MRSA strains that have originated in hospitals versus communities versus livestock is getting complicated.

Image adapted from Mole 2013 (12).
3. Livestock-associated (LA)-MRSA colonization is prevalent in some livestock species from which they can spread to humans and cause disease. Infections transmitted from animals to humans are called **zoonoses**. During the past 10 years, livestock has become a significant source for human MRSA infections, especially affecting people working with colonized animals and to a lesser extent, members of their households (Figure 6) (12). Livestock-associated bacteria can be a reservoir for antibiotic resistance genes in general. Antibiotics are frequently used to prevent infections in these animals through a practice that is known as metaphylactic therapy. Furthermore, in several countries including the US, antibiotics are incorporated into some animal feeds to promote growth (11).

While MRSA strains tend to circulate among the species to which they are most closely adapted, they can jump species barriers. For example, cats and dogs can become infected with HA-MRSA or CA-MRSA via contact with a colonized or infected owner through a process known as **anthroponosis**. Although humans are more likely to transmit MRSA to their pets rather than to become infected from them, infected or colonized animals can serve as a source of infection for their human contacts.

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Meticillin/Methicillin resistant *Staphylococcus aureus* on Oxoid Brilliance/Spectra M.R.S.A. selective chromogenic agar.
How can MRSA infections be detected, treated, and prevented at the personal and public health levels?

The CDC estimates that two in 100 people are colonized with MRSA in the nose (2). MRSA colonization, however, does not equate to a MRSA infection. An individual with an MRSA infection has signs and symptoms that are dependent upon the area of the body that is infected. For example, skin infections display redness, inflammation, heat, and could become swollen when pus or another liquid accumulates. Because of the appearance of these MRSA skin infections, they may be mistaken for insect and spider bites (Figure 7). Signs of a more serious, internal MRSA infection include fever, chills, confusion, dizziness, muscle aches, swelling of the affected body part, and wounds that will not heal.

When an MRSA infection is present, clinicians will need to assess the antimicrobial susceptibility of the bacterial strain that is causing the infection to determine the appropriate antibiotic therapy to administer to the patient. Determining the antibiotic susceptibility of MRSA requires taking specimens from a particular anatomic site or bodily fluid, culturing bacteria that are present in those specimens, and then testing the ability of those bacteria to grow in the presence of a variety of antibiotic agents. Results from culturing techniques usually take about 24 to 72 hours to obtain (Figure 8) (13). Rapid tests taking only about...

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**Figure 7**

Appearance of a CA-MRSA skin infection resembling a spider bite. Image retrieved from DeLeo and Chambers 2009 (5).
six hours are recommended for settings such as emergency rooms. In areas of the US where MRSA makes up more than 10% of the local SA infection burden, the Infectious Diseases Society of America (IDSA) recommends using non-β-lactam antibiotics such as tetracycline or sulfa drugs for initial treatment. However, severe MRSA infections involving the heart, bone tissues, or blood may require prolonged treatment with intravenous antibiotics (See 2011 IDSA MRSA treatment guidelines) (14). Ultimately, physicians will need to determine the most suitable treatment for individual patients in their care.

In hospitals, measures such as hand hygiene (hand-washing with soap and water or with an alcohol hand-rub), environmental cleaning and disinfection, use of personal protective equipment such as gowns and gloves (contact precaution), and avoiding use of inappropriate antimicrobial agents should be scrupulously instituted. These measures can be effective against a variety of microbial pathogens in addition to and including MRSA.

MRSA-specific interventions depend upon screening individuals who are carrying this microorganism as the first step. In the future, MRSA carriers should be identified by adopting tailored and risk-based screening programs. Implementing such infection prevention measures can help greatly to avoid spreading MRSA to other patients. Such patients can be decol-
onized—their MRSA removed from them before they are admitted to the hospital or healthcare facility, or before surgery or other procedures are performed. The decision to decolonize individual patients will depend on doctor recommendations, the health condition of each patient, and practices being followed in a particular region or country (14). These measures include treating individuals with an antibacterial body wash and shampoo (chlorhexidine bath) or, in other cases, applying an antimicrobial cream such as mupirocin to the interior of the nose. If decolonization is not possible or not indicated, knowledge of the MRSA colonization helps when initiating the correct antibiotic therapy which is imperative for a successful clinical outcome in severe disease. Similar to HA-MRSA prevention techniques, transmission of CA-MRSA can be impeded by proper hand-washing and not touching or sharing personal items (Figure 9).

As a component of the Emerging Infections Program-Active Bacterial Core surveillance system (EIP-ABCs) at the CDC, invasive (bloodstream, lung, and other serious infections) MRSA infections have been tracked in nine different metropolitan regions of the United States since 2005 (15). All participating medical labs report information about such infections to the CDC, which uses the data to estimate population-based incidence of invasive infections.
MRSA infections across the entire country. Simple skin infections caused by MRSA are not systematically tracked in the US, even if they require hospitalization. If the CDC detects a higher than expected rate of infections or colonizations within a particular population group, the CDC may investigate, although this is not a routine practice. When there is a change in the usual pattern of any type of infection in US hospitals, local hospital infection control practitioners typically investigate.

Quick Facts about SA and MRSA

• Staphylococcus aureus is a bacterium that commonly inhabits the human skin and nose without causing an infection. This organism can become pathogenic if it enters the body, resulting in a range of infections including pimples, boils, impetigo, and abscesses, to more severe and life-threatening illnesses such as lung, heart-valve, bone, and bloodstream infections. Some forms of SA are more aggressive than others due to varying production levels of virulence factors and toxins in combination with different antibiotic resistance mechanisms.

• MRSA or methicillin-resistant Staphylococcus aureus infections are often resistant to numerous additional classes of antibiotics as well as those belonging to this frequently prescribed class, the (beta) β-lactams. The types of infections that MRSA causes are in line with SA (See above). Varying levels of pathogenicity are observed for MRSA strains.

• MRSA can be divided into 3 categories:
  - Healthcare-associated (HA)-MRSA – bacteria contracted in healthcare settings
  - Community-associated (CA)-MRSA – bacteria contracted outside of healthcare-related environments (more common in close-knit communities)
  - Livestock-associated (LA)-MRSA – MRSA in a livestock (most commonly pigs) reservoir transmitted from animals to infect people working with or in close contact with those animals; animals can acquire both HA-MRSA and CA-MRSA strains

  • MRSA is also found on various livestock-associated foods. This can occur due to inappropriate hand hygiene by food handlers in processing plants. However, contaminated food is a very minor route of MRSA transmission to people (16).

• Treatment options for MRSA depend on the type and severity of the infection, and the susceptibility of the organism to antibiotics.

  • Hand-washing and other disinfection and hygienic practices help to decrease the transmission of MRSA across all settings.

Online Resources:
CDC’s MRSA website; www.cdc.gov/hai/mrsa
Prevention Guidelines for MRSA; www.cdc.gov/hicpac/pubs.html
References


