

Staphylococcus aureus

The name *Staphylococcus* is derived from the Greek word staphyle or “bunch of grapes” because of the characteristic cluster-like appearance of the bacteria under the microscope.

There are 32 species of staphylococci, but only 17 are indigenous to humans.

Staphylococcus aureus is especially prevalent due to its surface proteins, which allow the organism to bind to tissues and foreign bodies coated with collagen, fibronectin, and fibrinogen. This permits the bacteria to adhere to devices such as sutures, catheters, and prosthetic valves. Other medically important staphylococci include *S. epidermidis* and *S. saprophyticus*.

Colonization and Disease

Many newborns and most children and adults are intermittently colonized by *S. aureus* and harbor the organism either in their nasopharynx, occasionally on the skin and clothing, and more rarely in both the vagina and rectum. From these sites, *S. aureus* can contaminate any site on skin or mucous membranes, or other people, by direct transfer or aerosol.

Intact skin and mucous membranes are effective barriers against local invasion. If these barriers are breached, however, by trauma, surgery, or indwelling devices, *S. aureus* may gain access to the underlying tissue and create a local abscess consisting of dead tissue, fibrin, and live or dead white blood cells.

Patients most likely to develop superficial or deep infections are those who are hospitalized or undergoing major surgery, have intravascular lines, indwelling urinary catheters, pressure ulcers, or underlying disease.

At any time, multiplying *S. aureus* can overwhelm local defense mechanisms and invade the lymphatic system or the bloodstream—a serious complication that allows the bacteria to invade other tissues including the heart (endocarditis), lungs (pneumonia), or bone (osteomyelitis).

Resistance

The development of antibiotic-resistant strains of *S. aureus* has paralleled and contributed to the development of modern antibiotics. Penicillin G-resistant *S. aureus* was reported in 1945 – shortly after penicillin was first released for use. Penicillin G resistance was followed by resistance to methicillin and to other penicillins such as oxacillin and cloxacillin. *S. aureus* can produce as many as four different types of enzymes that specifically attack the structure of penicillin-like drugs rendering them ineffective. Methicillin-resistant *S. aureus* (MRSA) is a major therapeutic problem throughout the world.

Although still very rare, *S. aureus* fully resistant to vancomycin, previously an antibiotic of last resort, has been isolated in Japan and the United States. Despite potent antibiotics and hospital infection control strategies, *S. aureus* has remained a major human pathogen.

Socio-Economic Burden of Disease

Nearly 60 percent of hospital-acquired *S. aureus* infections in intensive care units reported to the Centers for Disease Control today are MRSA.

For the patient, the potential impact of MRSA includes increased morbidity and mortality. This impact includes slower response to therapy and elevated risk of therapeutic failure, extra procedures and treatments (such as surgical wound drainage), longer hospital stays, more work absenteeism, delay in return to usual activities, and reduced quality of life. Within hospitals, MRSA may lead to increased cost of infection control, increased laboratory use for surveillance and screening, use of broader spectrum empirical therapy, and longer hospital stays. It may impact hospital waiting times for new admissions and result in the use of costlier therapies and an increased overall staphylococcal infection rate.

Additionally, studies of patients with methicillin-resistant *S. aureus* have reported higher mortality rates, increased morbidity, longer length of hospital stays and higher costs compared with patients with methicillin-sensitive *S. aureus*.

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