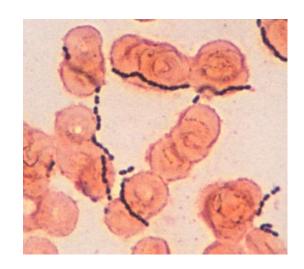
Pyogenic Cocci



Staphylococcus gram-positive



Streptococcus gram-positive



Neisseria gram-negative

Stapylococcus and related organisms

- *S. aureus*: major pathogen for humans, may cause suppuration, abscess formation, scalded skin syndrome, toxic shock syndrome and food poisoning.
- S. epidermidis: may cause infection from prosthetic devices.
- **S.** saprophyticus: may cause urinary tract infections (UTI) in young women.
- S. haemolyticus: endocarditis, UTI, and opportunistic infections.

Micrococcus spp.: opportunistic infections.

Stomatococcus spp.: endocarditis, opportunistic infections.

Alloiococcus otitidis: chronic middle ear infection.

Morphology and Identification

Staphylococci

Nonmotile.

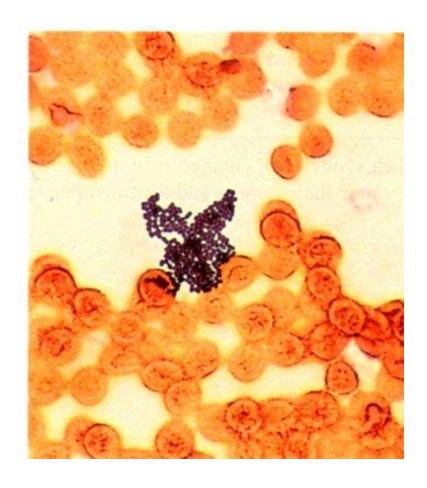
Grow readily on most bacteriological media; facultative anaerobic.

Grow most rapidly at 37 °C, but form carotenoid pigment best at room temperature under aerobic condition on solid medium.

Produce catalase.

Relatively resistant to drying, heat (40°C) and 10% NaCl.

Gram-positive cocci (a bunch of grapes)



Pathogenesis and Immunity

S. aureus can produce diseases both through invasiveness and production of toxins.

Toxins

Cytotoxins

 α -toxin: pore-forming, cytotoxic to many types of cells including muscle cells.

 β -toxin: degrades sphingomyelin and is toxic for many kinds of cells, including human RBCs.

 γ -toxin: bicomponent toxins, pore-forming.

 δ -toxin: has detergent-like activity.

P-V leukocidin: similar to γ -toxin in structure, kills WBCs of many animals and release the lysosomal enzymes. Associated with severe pulmonary and cutaneous infections.

Toxins (continued)

Exfoliative (epidermolytic) toxins: proteases that split desmoglein 1 of the intercellular bridges in epidermis; produced by about 5-10% of *S. aureus*; causes the generalized desquamation of the staphylococcal scalded skin syndrome (SSSS).

Toxic shock syndrome toxin-1 (TSST-1): superantigen, associates with fever, shock, desquamative skin rash of toxic shock syndrome in humans.

Enterotoxins: superantigens, at least 10 (A, B, C1, C2, C3, D, E, G, H, and I) soluble toxins produced by about 50% of *S. aureus*.

Heat-stable (100°C, 30 min.) and resistant to the gastric acid and gut enzymes.

Enterotoxins are produced in carbohydrate and protein foods.

Causing emesis, a characteristic of staphylococcal food poisoning.

Enzymes

Coagulase: bound and free forms. May deposit fibrin on the surface of staphylococci and alter their ingestion by and destruction within the phagocytic cells (associated with invasiveness).

Fibrinolysin (staphylokinase): to dissolve fibrin clot.

Catalase: to remove H_2O_2 .

Hyaluronidase: to facilitate spread of *S. aureus* in tissue.

Lipase: associated with superficial skin infection.

Nuclease: produced only by *S. aureus*.

Penicillinase

Epidemiology

Staphylococci can permanently (coagulase-negative strains) or transiently (*S. aureus*) colonize various areas of the human body, with the anterior nasopharynx as the most common colonization site for *S. aureus* in older children and adults (30% of healthy adults.)

Nasopharyngeal or skin carriers of *S. aureus* are responsible for many hospital infections.

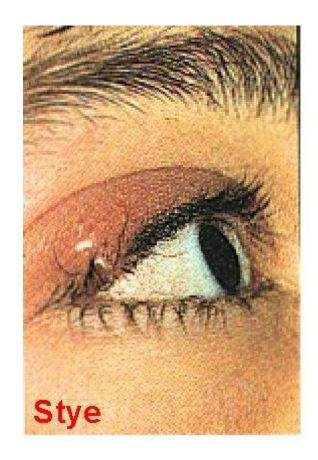
S. aureus can be transmitted through direct personal contact or contact with contaminated fomites.

Areas at highest risk for severe infections: new born nursery, ICU, operating rooms and cancer chemotherapy wards.

Clinical Diseases

S. aureus

Cutaneous infections (folliculitis, e.g., stye and acne; furuncles; carbuncles; impetigo): usually an intense, localized painful inflammatory reaction that undergoes central suppuration and heals quickly when the pus is drained. Carbuncle patients frequently have systemic signs.



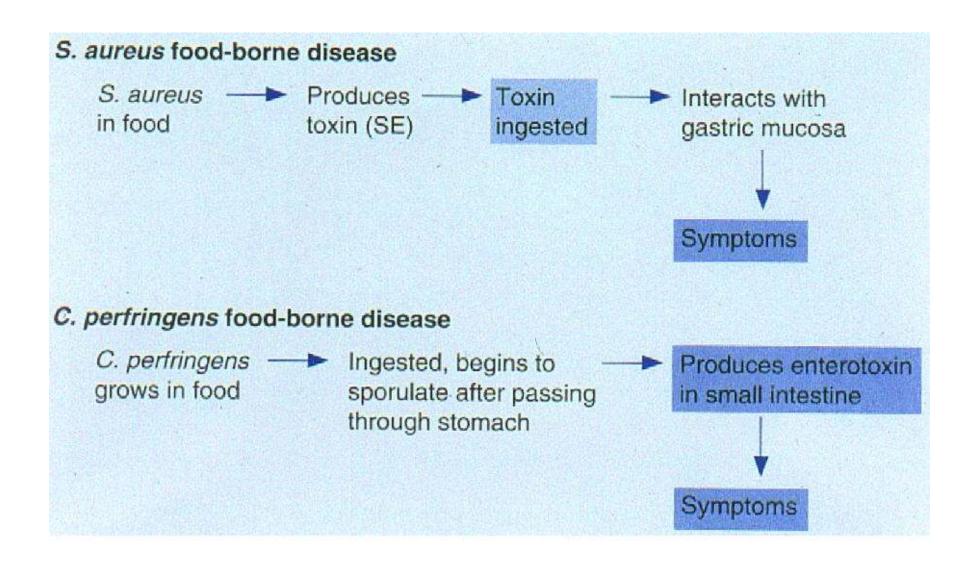
Wound infections: can occur after surgery or trauma, may involve skin, bone (osteomyelitis from an open fracture) or meninges (meningitis from skull fracture.)

Clinical Diseases

Food poisoning: caused by ingestion of preformed enterotoxin in food (meat and carbohydrates). Short incubation (1-8 hr). Violent nausea, vomiting and watery diarrhea; no fever; rapid convalescence.

Staphylococcal enterocolitis occurs in patients who have received broad spectrum antibiotics (antibiotic-associated diarrhea).

Toxic shock syndrome: abrupt onset of high fever, vomiting, diarrhea, myalgia, scarlatini form rash, desquamation of palms and soles, and hypotension with cardiac and renal failure. This disease has occurred in children injected with contaminated vaccine (1928), and young women who used tampons (1980). This may also occur in children or in men with staphylococcal wound infections (half cases are caused by enterotoxin B and, rarely, enterotoxin C.)



Clinical Diseases

S. epidermidis and other coagulase-negative staphylococci

Endocarditis: caused by infection of native (rarely) or prosthetic heart valves.

Catheter and shunt infections: a major medical problem, because catheters and shunts are commonly used in critically ill patients. Slime production that causes biofilm formation prevents the bacteria from antibiotics and inflammatory cells. Persistent bacteremia is generally observed.

Prosthetic joint infections: localized pain and failure of the artificial joint. Systemic signs are not prominent. Reinfection of new joint is increased in such patients.

Urinary tract infections: UTI infection by *S. saprophyticus* occurs mostly to young, sexually active women.

Treatment

Drug resistance of *S. aureus*

Tetracycline are used for long term treatment of acne or furunculosis. Abscess and other closed suppuration lesions are treated by drainage and antibiotics.

Bacteremia, endocarditis, pneumonia and other severe staphylococcal infections: prolonged i.v. therapy with β -lactamase-resistant penicillins (e.g. methicillin, oxacillin, etc.)

Vancomycin is the most effective drug against staphylococci, but its use is restricted in most hospitals.



Pustular impetigo