**Staphylococcus :**

Staphylococci are Gram-positive cocci 1μm in diameter. They form clumps

**symptoms:**Staphylococci can cause many forms of infection.

(1) *S. aureus* causes superficial skin lesions (boils) and localized abscesses in other sites.

(2) *S. aureus* causes deep-seated infections, such as osteomyelitis and endocarditis and more serious skin infections (furunculosis).

(3) *S. aureus* is a major cause of hospital acquired (nosocomial) infection of surgical wounds and, with *S. epidermidis*, causes infections associated with indwelling medical devices.

(4) *S. aureus* causes food poisoning by releasing enterotoxins into food.

(5) *S aureus* causes toxic shock syndrome by release of super antigens into the blood stream.

(6) *S. saprophiticus* causes urinary tract infections, especially in girls.

**Pathogenesis**

*S aureus* expresses many potential virulence factors.

(1) Surface proteins that promote colonization of host tissues. (2) Factors that probably inhibit phagocytosis (capsule, immunoglobulin binding protein A).

(3) Toxins that damage host tissues and cause disease symptoms. Coagulase-negative staphylococci are normally less virulent and express fewer virulence factors. *S epidermidis* readily colonizes implanted devices

**Transmission :**

Staphylococcal food poisoning is usually caused by food contaminated with staphylococcal bacteria from the skin of someone who handles food.

If the food is not cooked thoroughly, or if it's not kept hot or cold, the bacteria will continue to reproduce when they come in contact with it. The bacteria then produce the toxin (poison) that causes food poisoning

in healthy people, the layers of skin and the immune system usually provide a good defence against a skin infection spreading further into the body

**Diagnosis:**

Diagnosis is based on performing tests with colonies. Tests for clumping factor, coagulase, hemolysins .Commercial latex agglutination tests are available. Identification of *S epidermidis* is confirmed by commercial biotyping kits

**Treatment :**

clindamycin,trimethoprim-sulfamethoxazole (TMP-SMX), rifampin, doxycycline,



***S. aureus, along with S. epidermidis, is responsible for the majority of wound infections***



***Streptococcus pyogenes***:

 (**Group A streptococcus**) is a Gram-positive, nonmotile, non sporeforming coccus that occurs in chains or in pairs of cells.

*Streptococcus pyogenes* is one of the most frequent pathogens of humans. usually in the respiratory tract, without signs of disease. As normal flora, *S. pyogenes* can infect when defenses are compromised or when the organisms are able to penetrate the constitutive defenses. When the bacteria are introduced or transmitted to vulnerable tissues, a variety of types of **suppurative infections**can occur.

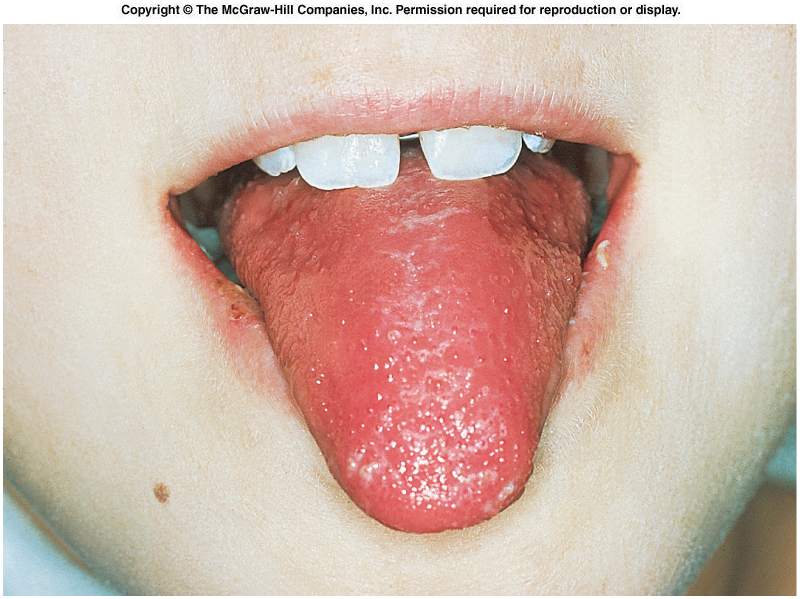
**Symptoms:**

Acute *Streptococcus pyogenes* infections may present as **pharyngitis** (**strepthroat**), **(Strawberry Tongue)**

, **scarletfever** (rash), **impetigo** (infection of the superficial layers of the skin) **Erysipelas** or **cellulitis** (infection of the deep layers of the skin). Invasive, toxigenic infections can result in **necrotizing fasciitis**, **myositis** . Patients may also develop immune-mediated**post-streptococcal sequelae**, such as acute **rheumatic fever** and acute **glomerulonephritis**, following acute infections caused by *Streptococcus pyogenes.*

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**Erysipelas**

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**Strawberry Tongue**

**Pathogenesis:**

Species of *Streptococcus* are classified based on their [hemolytic](http://en.wikipedia.org/wiki/Hemolysis_(microbiology)) properties. Alpha-hemolytic species cause oxidization of iron in hemoglobin molecules within red blood cells, giving it a greenish color on blood agar. Beta-hemolytic species cause complete rupture of red blood cells. On blood agar, this appears as wide areas clear of blood cells surrounding bacterial colonies. Gamma-hemolytic species cause no hemolysis.

Host-pathogen interactions occur due to binding of surface streptococcal ligands to specific receptors on host cells. Attachment of group A streptococci to pharyngeal or dermal epithelial cells is the most important initial step in colonization of the host. Without strong adherence mechanisms, group A streptococci could not attach to host tissues and would be removed by mucous and salivary fluid flow mechanisms and exfoliation of the epithelium. In skin attachment and colonization by group A streptococci, a site of previous damage may be important in overcoming the dermal barrier. Specific adhesion allows competition between normal flora and group A streptococci for tissue sites where normal flora reside. The investigation of adherence determinants of both streptococcal and host cells is vital to the understanding of pathogenic mechanisms in disease and in the development of antiadhesive therapies or vaccines to prevent colonization. Immunization or exposure of humans to microbial adhesions may induce antibodies which concentrate in the mucosal layer and block adherence and colonization at the mucosal epithelium.

**Diagnosis:**

Strep bacteria can be obtained by swabbing the back of the throat, or the infected area with a piece of sterile cotton. A blood sample can also be taken. Microscopic examination of the smear can identify which type of bacteria has been collected. A sample may also be sent to a lab for traditional culturing, which takes from one to two days, because this form of testing is more accurate than the rapid strep test.

**Transmission:**

Transmission via respiratory droplets, hand contact with nasal discharge and skin contact with impetigo lesions are the most important modes of transmission

**Treatment:**

Penicillin and other **antibiotics** are used to treat GAS and other types of strep infection.