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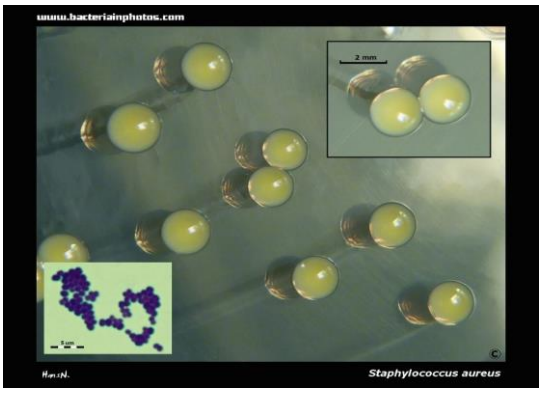
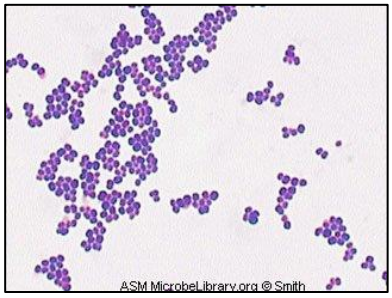
Medical Bacteriology - Lecture 5

Staphylococci



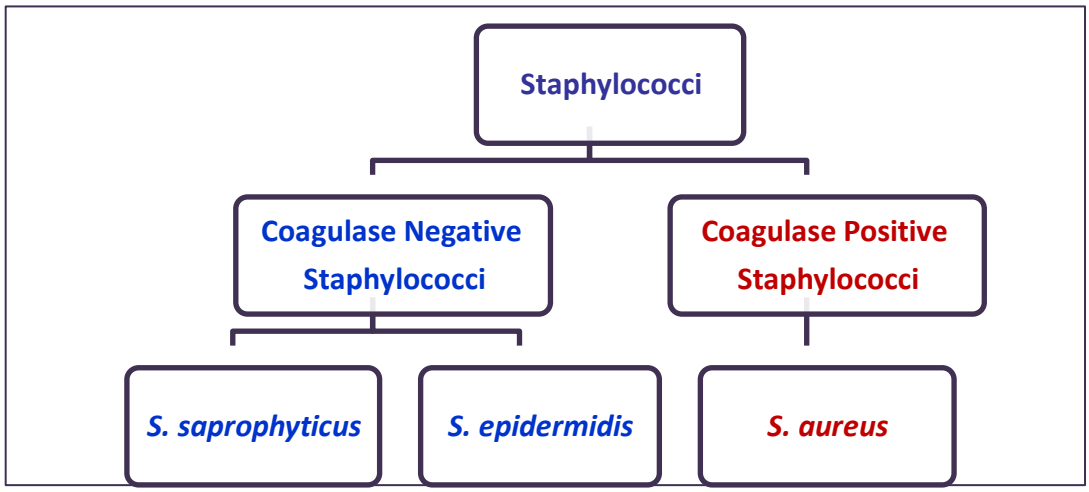


Staphlyococci

Micrococcaceae

Staphylococcus: Pathogenic or commensal

***Micrococcus*:** Free-living saprophytes



General Phenotypic Characteristics of Staphylococci

Staphylococci are

Gram-positive
Cocci, which appears as grape- like clusters when viewed through a microscope.
Catalase-positive & oxidase-negative
Grow at **15 % NaCl** concentrations.
Facultative anaerobes (respiration or fermentation), fermentation of glucose produces mainly lactic acid.
Optimum temperature at 37°C, can grow at a temperature range (15 to 45 c).
Non Fastidious
Non motile
Non spore forming.

Medically Important Staphylococci Species:

Coagulase Positive Staphylococci (CoPS)

- ***S. aureus*** colonizes mainly nasal passage as normal flora, but it may be found in other sites (skin, mucous membranes, oral cavity & gastrointestinal tract)
- Always considered a **potential pathogen**
- **Causes nosocomial infections**

Coagulase Negative Staphlyococci (CoNS)

- are part of normal flora of human skin and mucous membranes
- relatively **low virulence**
- frequently involved in **nosocomial and opportunistic infections**
- Clinically significant infection associated with endocarditis, joint infection, wound infections, bacteremia, Urinary tract infections (UTI).
- ***S. epidermidis*** is an inhabitant of the skin and mucous membranes, mostly **nonpathogenic** & may play a protective role in humans as normal flora.
- In contrast to *S. aureus*, little is known about mechanisms of pathogenesis of *S. epidermidis* infections.
- Adherence and colonization of catheters by *S. epidermidis* is a crucial step in the initiation of foreign body infections.
- The production of **biofilm**, a significant determinant of **virulence** for ***S. epidermidis***.

- ***S. saprophyticus*** is a leading cause of **cystitis in young women**. And shares of urinary tract infection.

Pathogenesis of *S. aureus*

S. aureus causes a variety of **suppurative (pus-forming)** and **toxigenic infections** in humans.

It causes:

- **Superficial skin lesions** such as
  Boils, furuncles, abscess

- **More serious skin infections** such as
  Impetigo (bubble-like swellings that can break and peel away; common in newborns)

Staphylococcal scalded skin syndrome (SSSS) or Ritter's disease (relatively rare); (toxin induces bright red flush, blisters, then desquamation of the epidermis)

- **Serious infections (Deep)** such as
  Pneumonia (infections in the lung), **Osteomyelitis** (Localized infection of bone), **endocarditis**, **meningitis**, skeletal muscle, **urinary tract infections.**

S. aureus is a major cause of **hospital acquired (nosocomial) infections**
Surgical wounds and infections associated with medical devices.

- **Toxigenic infections such as**
  ***S. aureus* causes food poisoning** by releasing heat stable **enterotoxins** into food.

It can produce **toxic shock syndrome** (leading to shock and organ failure) by release of superantigens into the blood stream.

- **Serious consequences of staphylococcal infections (**Systematic infections)
  occur when the bacteria invade the blood stream. A resulting **septicemia** may be rapidly fatal or **bacteremia**.

S. aureus expresses many potential virulence factors

(1) Surface proteins

Promote colonization of host tissues such as **laminin** and **fibronectin.**

(2) Invasins

Promote bacterial spread in tissues (**leukocidin**, **kinases**, **hyaluronidase).**

(3) DNase

Digests DNA

(4) Lipases

Digest oils; enhances colonization on skin .

(5) Surface factors

Avoidance of host defenses; Inhibit phagocytic engulfment (**capsule**, **Protein A**).

- The majority of clinical isolates of *S aureus* express a surface polysaccharide (**microcapsule)** because it can be visualized only by **electron microscopy**. *S. aureus* strains isolated from infections express high levels of the capsule but rapidly lose it when **cultured in the laboratory**.

- **Protein A:** binds IgG antibody in the wrong orientation (Fc region), which disrupts opsonization and phagocytosis.

(6) Biochemical properties

Enhance survival in phagocytes

Staphyloxanthin; carotenoid pigment which responsible for golden colonies, and it has an antioxidant action that helps bacteria to evade reactive oxygen by the host immune system.

Catalase production.

(7) Immunological disguises

(**Protein A**, **coagulase**)

(8) Membrane-damaging toxins

Lyse eucaryotic cell membranes

Hemolysin;s (α, β, γ, δ) lysis red blood cells.

Leukocidin; (lysis neutrophils and macrophages).

(9) Exotoxins

Damage host tissues or provoke symptoms of disease

- **Staphylococcal enterotoxins**; (**SEA-G)**; food poising (nausea, vomiting, diarrhea).

- **Toxic shock syndrome toxin** (**TSST**); induces fever, vomiting, shock, organ damage.

- **Exfolioative toxins** (**ETs); responsible for Staphylococcal scalded skin syndrome (SSSS);** separates the epidermis from the dermis**.**

- **Panton-Valentine Leukocidin (PVL)** creates pores in the membranes of infected cells. It is associated with severe necrotizing pneumonia in children.

(10) Inherent & acquired **resistance to antimicrobial agents**
(Penicillinase- inactivates penicillin)

Host Defense against Staphylococcal Infections

Phagocytosis Neutrophil is the primary cellular defenses of innate immunity against Staphylococcal infections.

Antibodies are produced which neutralize toxins and promote opsonization.

Staphylococci may be difficult to kill after phagocytic engulfment because they produce catalase which neutralize oxygen and superoxide, which are primary phagocytic killing mechanisms within the phagolysosome.

Treatment

Hospital acquired infection of *S. aureus* is often caused by antibiotic resistant strains (e.g. MRSA) and can be treated with vancomycin or an alternative.
The term **MRSA** refers to **Methicillin resistant *S. aureus*** and related beta-lactam antibiotics (e.g. penicillin, oxacillin, amoxacillin). Some MRSA are resistant to vancomycin (VRSA).
The infections have been treated with combination therapy using sulfa drugs and or rifampin.

(CoNS); produce an enzyme called **beta lactamase** that makes them resistant to methicillin and oxacillin. Vancomycin is the most common antibiotic used to treat infections caused by CoNS-; if they not resistant. Rifampin and gentamicin may be added to prevent antibiotic resistance.

Vaccines

No vaccine is generally available that stimulates active immunity against staphylococcal infections in humans.

Differentiation between Coagulase Positive Staphylococci and Coagulase Negative Staphylococci

	S. aureus	***S. epidermidis***	***S. saprophiticus***
Hemolytic	**+ (most strains)** **Beta hemolytic**	No hemolytic	No hemolytic
Mannitol Fermentation	**Ferment mannitol**	No ferment	No ferment
Coagulase enzyme	**Produce coagulase**	Not produce	Not produce
Pigment production	Usually forms yellow (golden) colony on rich media	Usually has relatively small white colony	Usually small white colonies
DNase production	Produce	none	none
Sensitivity to Novobiocin	Sensitive	Sensitive	**Resistant**
Normal habitat	Nose	Skin	Skin

Review Questions

- What are the important phenotypic characteristics of *Staphylococci?*
- Give 4 examples of *S. aureus* infections?
- Give 2 examples of Coagulase negative Staphylococci infections?
- *S. aureus* expresses many virulence factors, explain?
- Write the full worlds of the following abbreviations: MRSA, CoNS, VRSA, TSST, ETs, SE, PVL?
- You studied Three Species under the Genus *Staphylococcus*. What are they? and how you differentiate between them?
- Coagulase negative Staphylococci are always resistant to methicillin, why? and how can treatment its infection ?
- What is the major virulence factor of *S. epidermidis* ?