University of AL-Anbar /College of Applied Science-Heet
Departments of Ecology
Microbiology

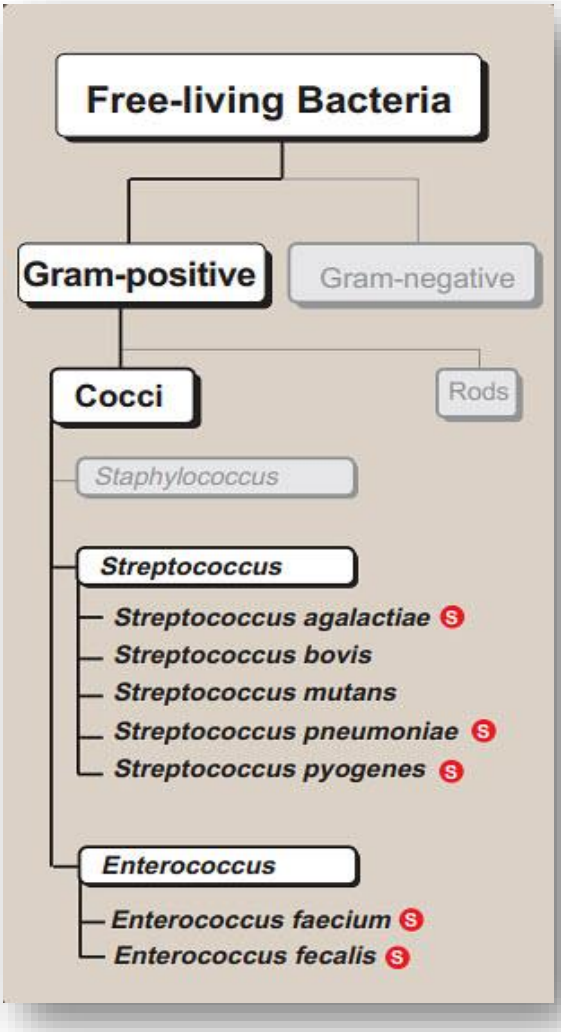
Streptococci, Enterococci, and Related Genes

The streptococci are Gram- positive spherical bacteria that characteristically form pairs or chains during growth. They are widely distributed in nature. Some are members of the normal human flora; others are associated with important human diseases attributable in part to infection by streptococci.

Streptococci and ***staphylococci*** constitute the main groups of medically important **gram-positive cocci**. Streptococci are ***gram-positive***, ***nonmotile,*** and ***catalase-negative***. Clinically important genera include ***Streptococcus*** and ***Enterococcus***. They are ovoid to spherical in shape, and occur as pairs or chains.

Most are ***facultative anaerobes*** (capable of growing in the absence or presence of oxygen), but grow fermentatively even in the presence of oxygen. Because of their complex nutritional requirements, blood-enriched medium is generally used for their isolation.

Diseases caused by this group of organisms include ***acute infections of the throat and skin caused by Group A streptococci***(***Streptococcus pyogenes***); ***female genital tract colonization***, resulting in **neonatal sepsis** caused by **Group B streptococci** (***Streptococcus agalactiae***); ***pneumonia, otitis media, and meningitis*** caused by ***Streptococcus pneumoniae***; and ***endocarditis*** caused by the ***viridans group of streptococci***.



♦. Morphology and Identification

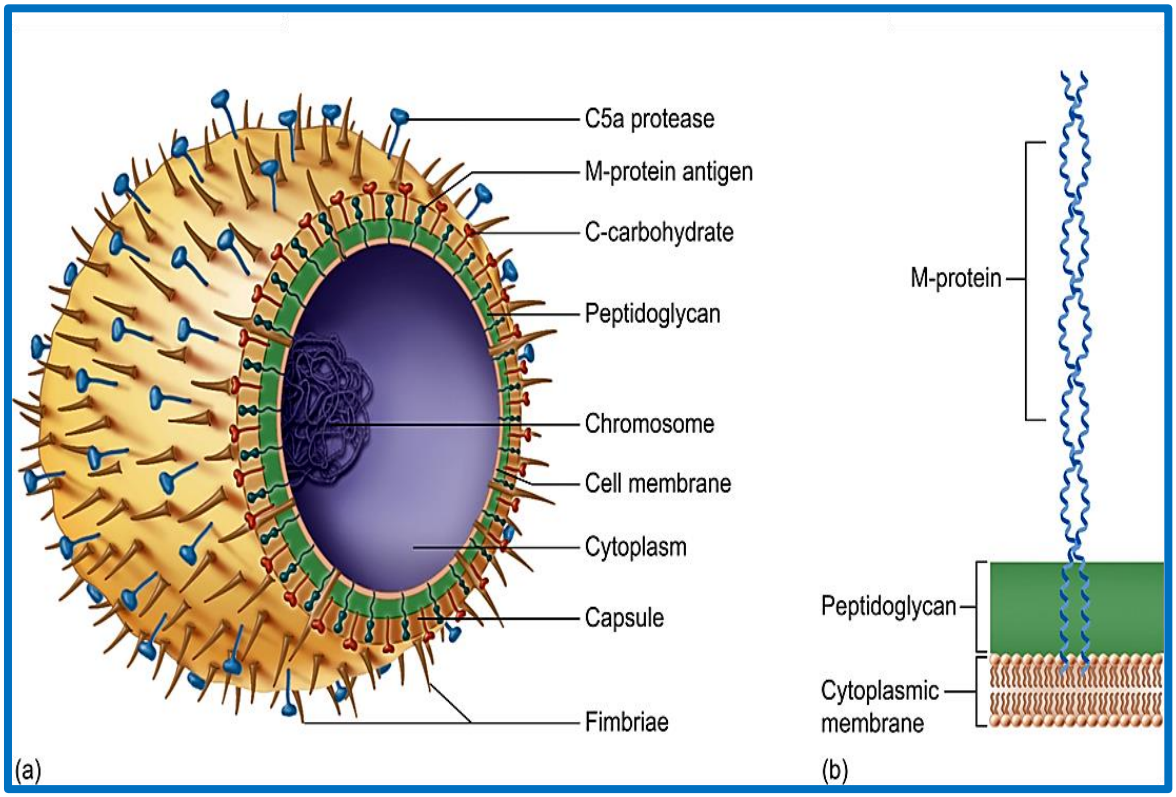
A- Typical organisms

Individual cocci are spherical ore ovoid and are arranged in chains. The cocci divide in a plane perpendicular to the long axis of the chain.The members of the chain often have a striking diplicoccal appearance, and rod-like forms are occasionally seen. The lengths of the chains vary widely and are conditioned by environmental factors.

Most **group A** strains produce capsules composed of hyaluronic acid. The capsules are most noticeable in very young cultures. They impede **phagocytosis**. The hyaluronic acid capsule likely plays a greater role in virulence than is

generally appreciated and together with M protein was believed to be an important factor in the resurgence of rheumatic fever (RF) in the United States in the 1980s and 1990s.The capsule binds to hyaluronic-acid-binding protein, CD44, present on human epithelial cells. Binding induces disruption of intercellular junctions allowing microorganisms to remain extracellular as they penetrate the epithelium. Capsules of other streptococci (eg, ***S. agalactiae and S. pneumoniae***) are different.

The ***S. pyogenes*** **cell wall** contain proteins (**M,T,R** antigens), carbohydrates (**group- specific**), and peptidoglycans . **Hair – like pili** project through the capsule of group **A streptococci**. The pili consist partly of **M ptotein** and are covered with **lipotechoic acid**. The latter is important in the attachment of streptococci to epithelial cells.



B- Culture

Most streptococci grow in solid media as discoid colonies, usually 1–2 mm in diameter. *S pyogenes* is β-hemolytic; other species have variable hemolytic characteristics.

C- Growth Characteristics

Energy is obtained principally from the utilization of sugars .Growth of streptococci tends to be poor on solid media or in broth unless enriched with blood or tissue fluids. Nutritive requirements vary widely among different species. The human pathogens are most exacting, requiring a variety of growth factors. Growth and hemolysis are aided by incubation in **10**% CO_2.

Most pathogenic hemolytic streptococci grow best at 37°C. Most streptococci are facultative anaerobes and grow under aerobic and anaerobic conditions.

.Classification of streptococci

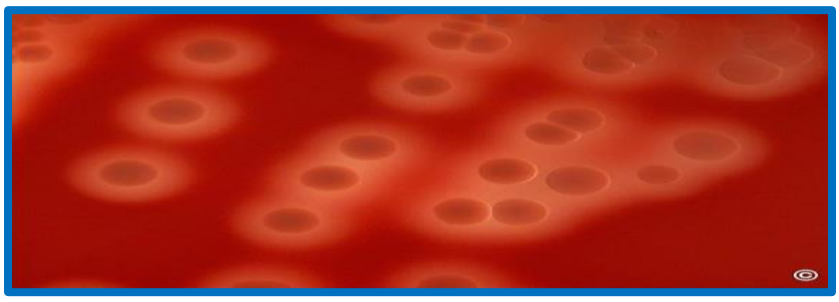
Four different classification systems exist for this important microorganism:

A - Hemolysis

Brown on the basis of red blood cell lysis on blood agar plates, divides the streptococci into:

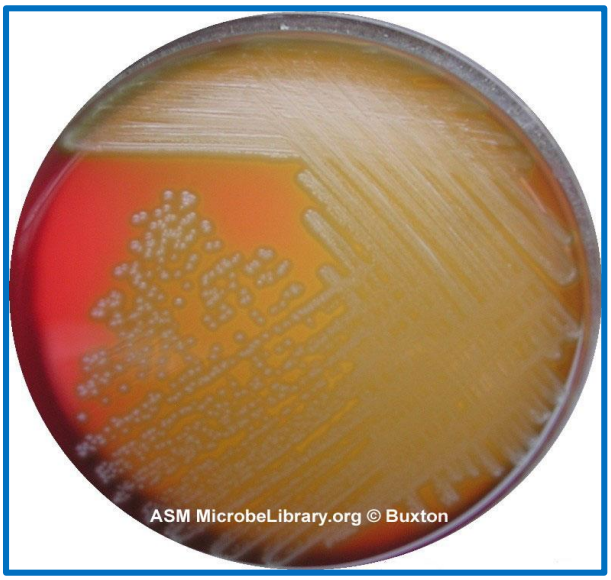
1- Beta hemolytic streptococci (β- Hemolysis)

This type produce wide, clear, translucent zone of complete hemolysis around the colony (complete disruption of erythrocytes with release of hemoglobin),e.g ***Streptococcus pyogenes***.



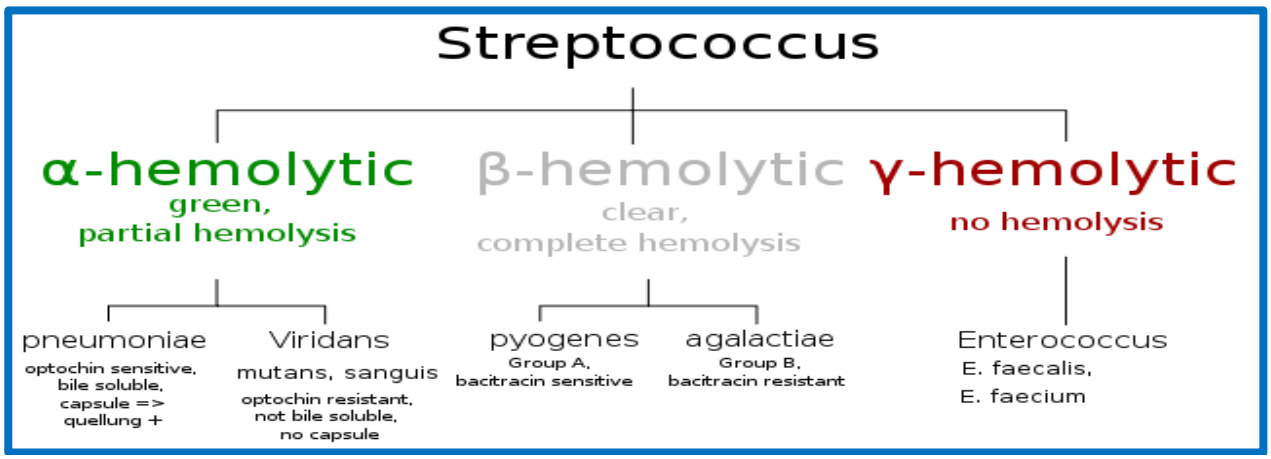
2 - Alpha hemolytic streptococci (α- Hemolysis)

They produce green zone around the colony due to incomplete lysis of erythrocytes with the formation of green pigment, eg. ***Streptococcus pneumoniae***.



3- Non hemolytic streptococci (ϒ- Hemolysis)

No change on the blood agar, e.g. ***Streptococcus bovis***.



B- Serologic Group-Specific Substance (Lancefield Classification)

The most precise and useful classification, has developed from the work of **Rebecca Lancefield**. She extracted serologically reactive material (the carbohydrate group – specific substances) with hot dilute hydrochloric acid and using precipitin techniques (with specific antisera). The serologic specificity of the group specific carbohydrate is determined by an amino sugar.

Streptococci can be placed into groups, referred to as **Lancefield groups**, based on the major cell wall carbohydrates they possess. Commercial agglutination kits are available for streptococcal grouping. At least **18** groups are recognized, designated groups **A** through **H** and **K** through **U**, but not all are equally important as human pathogens.

-The following are worthy of note:

1-Group A includes the important human pathogen ***Streptococcus pyogenes***.

2- Group B contains one species, ***Streptococcus agalactiae***, an inhabitant of the female tract; causes infection in neonates.

3- Group C mainly causes diseases in animals.

4- Group D includes the enterococci (***Streptococcus faecalis***, etc.)

C - Capsular Polysaccharides

The antigenic specificity of the capsular polysaccharides is used to classify ***Strep. pneumoniae*** into **90** types and to type the **group BStreptococci** (***Strep. agalactiae***).

D-Biochemical Reactions

Biochemical tests include **sugar fermentation reactions**, tests for the **presence of enzymes**, and tests for **susceptibility** or **resistance to certain chemical agents**. **Biochemical test** are most often used to classify **streptococci** after the colony growth and hemolytic characteristics have been observed.

Biochemical tests are used for species that typically do not react with the commonly used antibody preparations for the group-specific substances, groups A, B, C, F, and G.

Streptococci of particular medical interest

♦. Streptococcus pyogenes (Group A)

Habitat and transmission

Normal habitat is the human upper respiratory tract and skin; may survive in dust for some time. Spread by airborne droplets and by contact.

Characteristics

A commensal in the nasopharynx of a minority of healthy adults but more commonly (about 10%) in children. Grows well on blood agar ,with a characteristic halo of **β- haemolysis** . Some strains produce mucoid colonies as result of having a hyaluronic acid capsule. This may contribute to virulence by offering resistance to phagocytosis.

.Antigenic structure

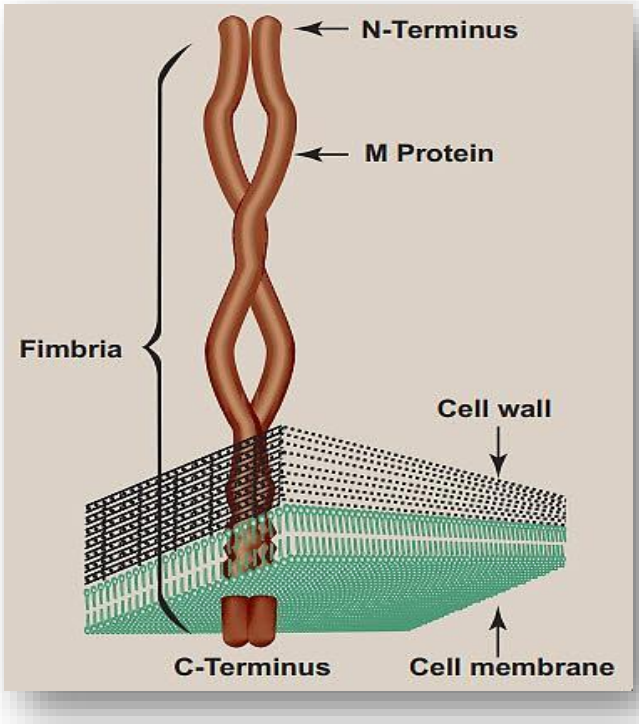
1- Group- specific cell wall antigen

This carbohydrate is contained in the cell wall of many streptococci and forms the basis of serologic grouping (**Lancefield group** '**C**-substance' **A-H, K-U**).

2- M protein

This substance is a major virulence factor of **group A *S. pyogenes***. **M protein** is a filamentous structure anchored to the cell membrane that penetrates and projects from the streptococcal cell wall.

When **M protein** is present, the streptococci are virulent, and the absence of **M type-specific antibodies**, they are able to resist phagocytosis by polymorphonuclear leukocytes. **Group A streptococci** that lack **M protein** are not virulent. Because there are more than 150 types of M protein, a person can have repeated infections with ***S. pyogenes*** of different M types.



M protein is thought to serve as a virulence factor in two ways:

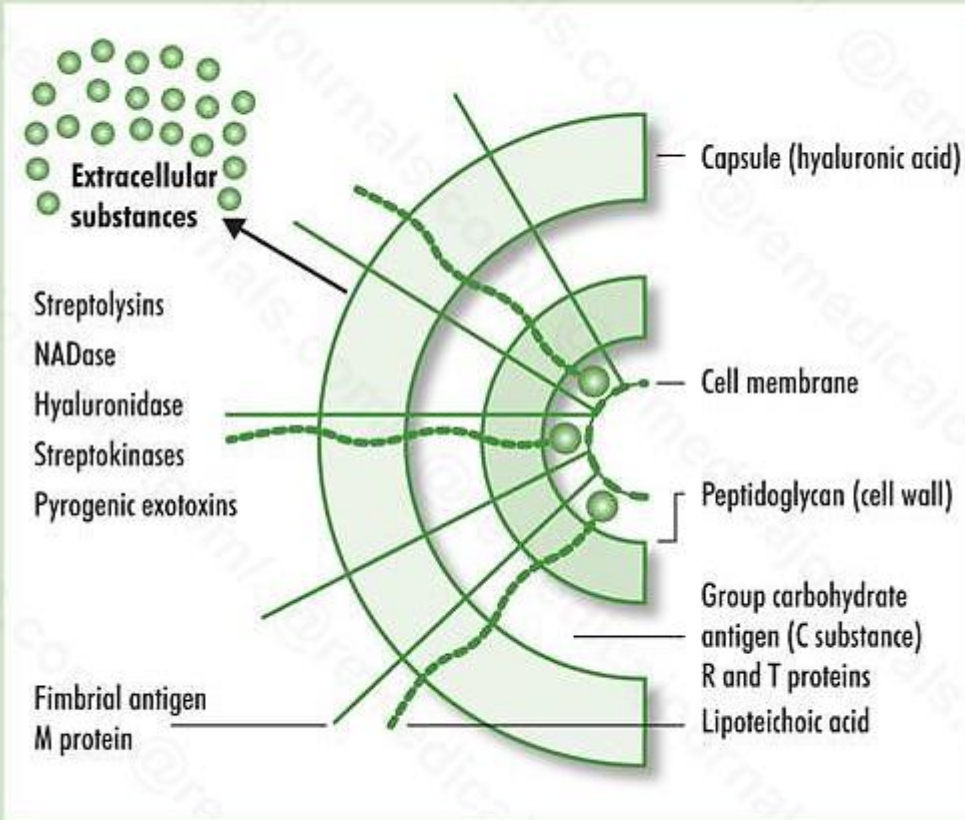
(1) by enabling *S. pyogenes* cells to adhere to surfaces and

(2) by functioning as an antiphagocytic.

3- T substance

This antigen has no relationship to virulence of streptococci. Unlike **M protein**, **T substance** is acid-labile and heat-labile. **T substance** permits differentiation of certain types streptococci by agglutination with specific antisera, while other types share the same **T substance**. Yet another surface antigen has been called **R protein**.

Figure 1. Cell surface structure of *Streptococcus pyogenes* and secreted products that are involved in virulence.



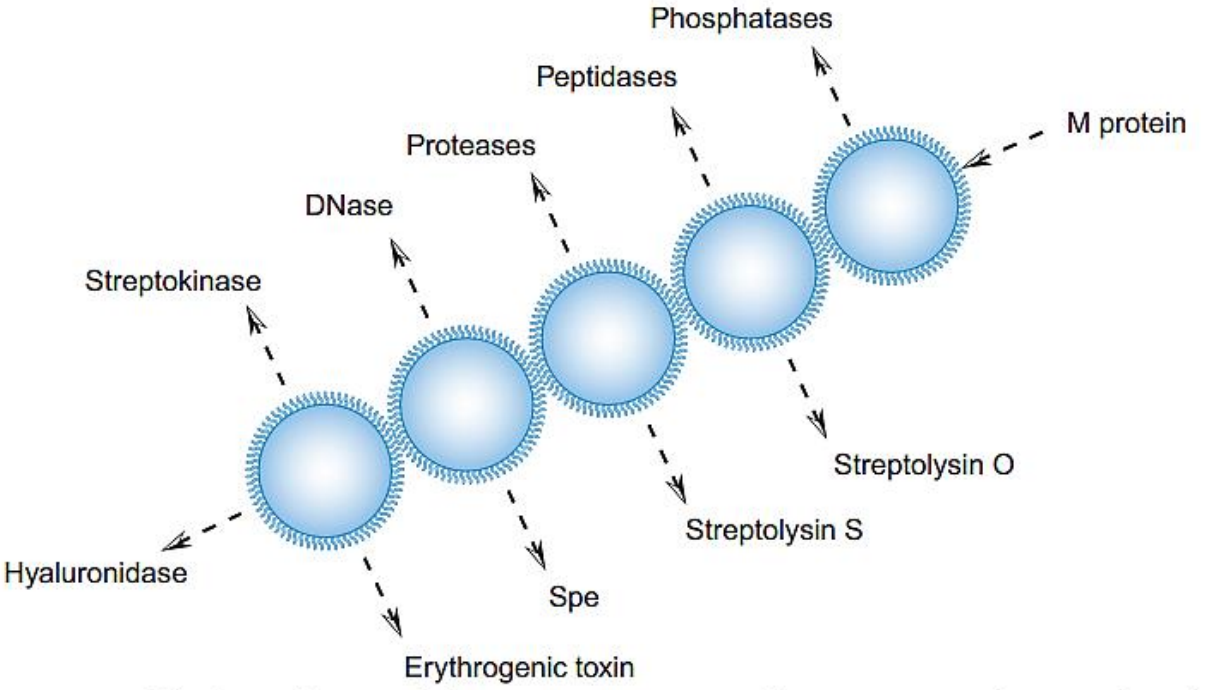
Reproduced with permission from Todar's Online Textbook of Bacteriology [69].

4- Protein F (fibronectin-binding protein)

Protein F mediates attachment to fibronectin in the pharyngeal epithelium. **M proteins** and lipoteichoic acids also bind to fibronectin.

♦.Toxins and Enzymes

More than 20 extracellular products that are antigenic are elaborated by ***S. pyogenes***, including the following:

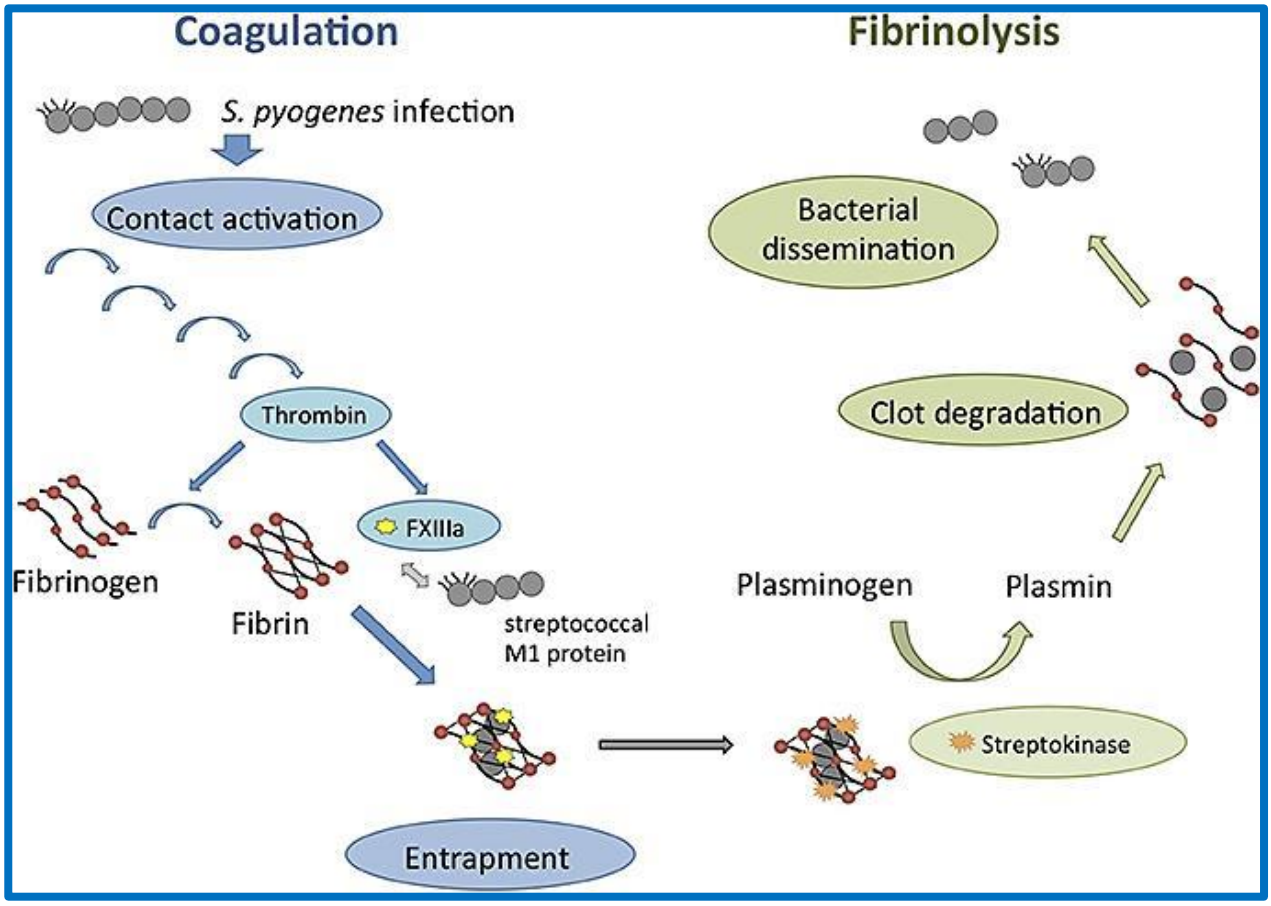


Virulence factors of *Streptococcus pyogenes*. Spe, streptoccocal pyrogenic toxin.

A- Sreptokinase (Fibrinolysin)

Streptokinase is produced by many strains of **group A** β-hemolytic streptococci.It transforms the **plasminogen** of human plasma into **plasmin**, an active proteolytic enzyme that digests **fibrin** and other proteins. This process of digestion may be interfered with by nonspecific serum inhibitors and by a specific antibody, antistreptokinase.

Streptokinase has been given intravenously for treatment of pulmonary emboli and of coronary artery and venous thrombosis.



B-Streptodornase

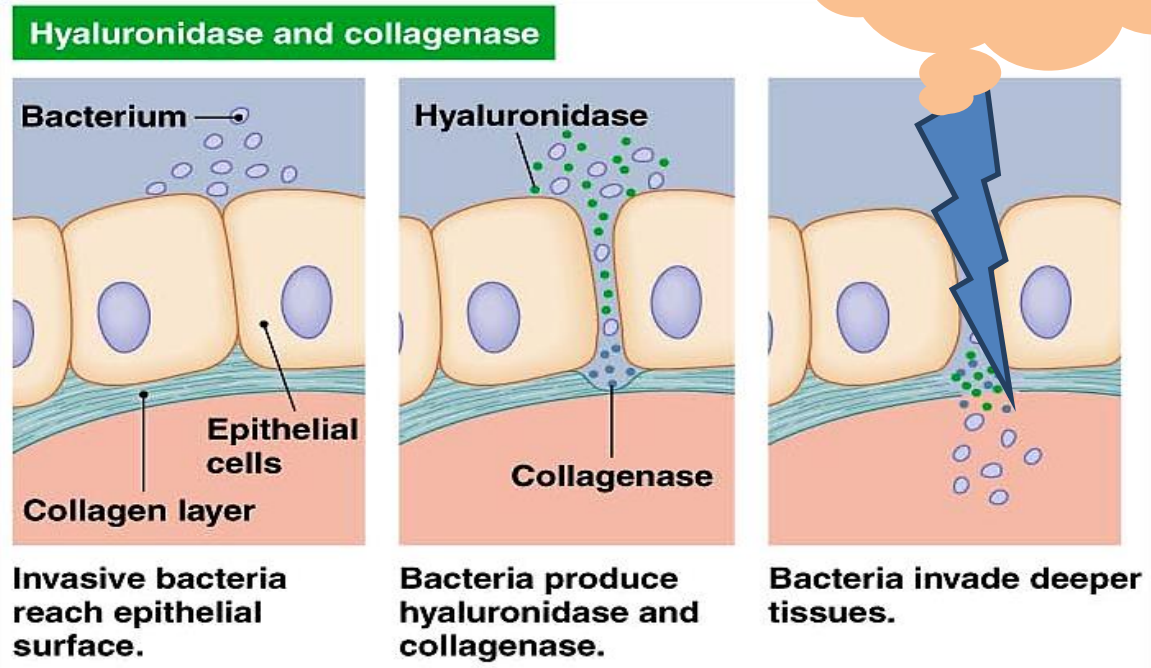
Streptodornase depolymerizes DNA. The enzymatic activity can measured by the decrease in viscosity of known DNA solutions.

Mixture of **streptodornase** and **streptokinase** are used in "enzymatic debridement". They help to liquefy exudates and facilitate removal of pus and necrotic tissue; antimicrobial drugs thus gain better access, and infected surfaces recover more quickly.

C- Hyaluronidase

Hyaluronidase cleaves hyaluronic acid (an important component of the ground substance of connective tissue) into numerous small fragments, thus converting the gel state of the extracellular matrix to a sol state . The consequence of this reaction is to permit the rapid spread of the infecting microorganisms through the connective tissue spaces (so called **spreading factor**).

Digestion of connective tissues and Invasion of tissues



D- Pyrogenic Exotoxins (Erythrogenic Toxin)

Pyrogenic exotoxins are elaborated by **group A streptococci**. The streptococcal pyrogenic exotoxins have been associated with streptococcal toxic shock syndrome and scarlet fever.

E- Diphosphyridin Nucleotidase

This enzyme is elaborated into the environment by some streptococci. This substance may be related to the organism's ability to kill leukocytes. Proteinases and amylase are produced by some strains.

F-Hemolysins

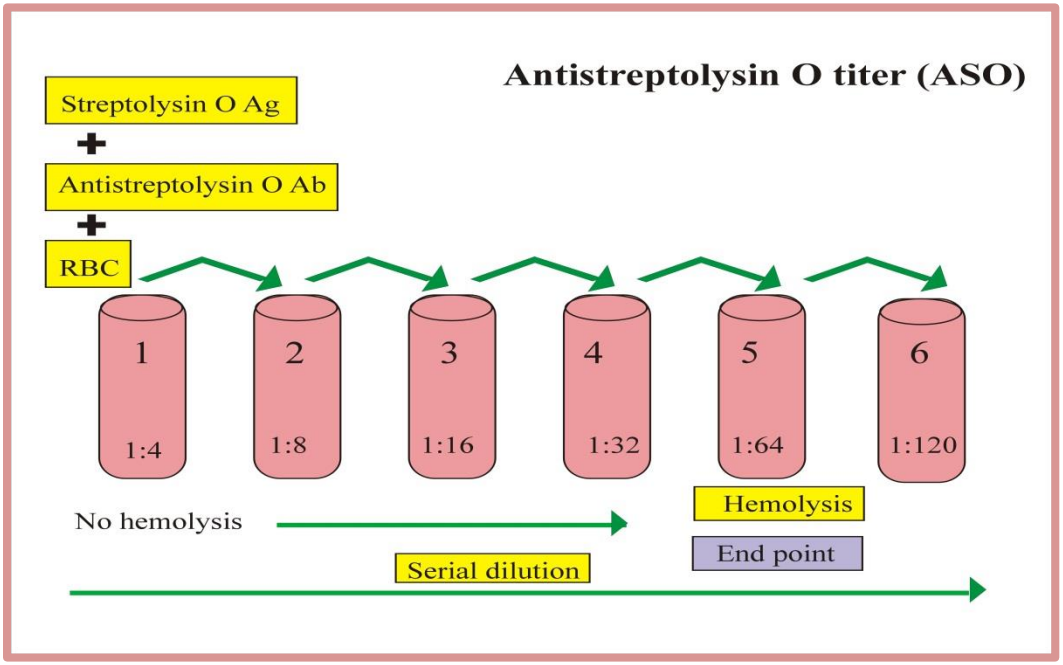
Many streptococci are able to hemolyze red blood cells in vitro in varying degrees.**β-Hemolytic group A *Strep. pyogenes*** elaborates two hemolysins (**streptolysins**) :

(i)- Streptolysin O

Streptolysin O is a protein that is hemolytically active in the reduced state but rapidly inactivated in the presence of oxygen. **Streptolysin O** is responsible for some of the hemolysis seen when growth is in cuts deep into the medium in blood agar plates.

Streptolysin O combines quantitatively with **antistreptolysin O** (**ASO**), an antibody that appears in humans following infection with any streptococci that produce streptolysin O. This antibody blocks hemolysis by streptolysin O.

This phenomenon forms the basis of a quantitative test for the antibody. An **antistreptolysin O (ASO)** serum titer in excess of **160-200 units** is considered abnormally high and suggests either recent infection with streptococci or persistently high antibody levels due to an exaggerated immune response to an earlier exposure in a hypersensitive person.



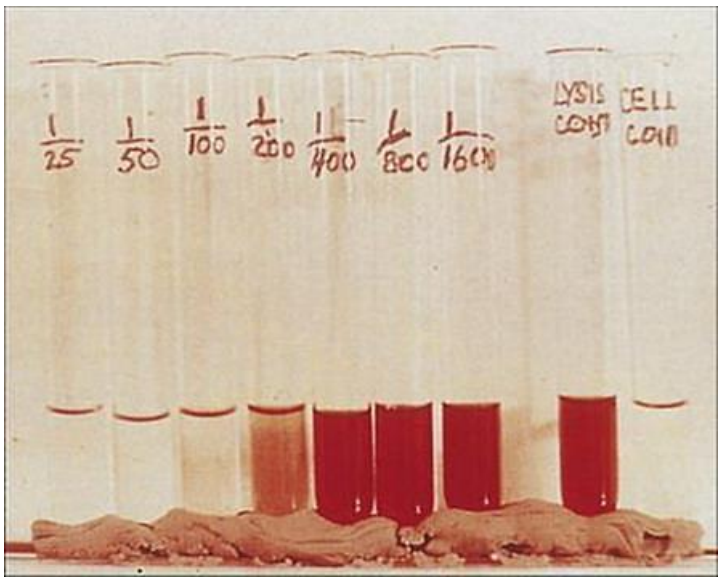


Figure-Illustration of the **anti-streptolysin O (ASO) test** which is now performed primarily by automated instrumentation**. The O-toxin lyses red cells.** Test serum is diluted until the antibodies it contains it no longer inhibit lysis by a standard concentration of toxin**. Positive and negative controls** are included in the test **(right).**

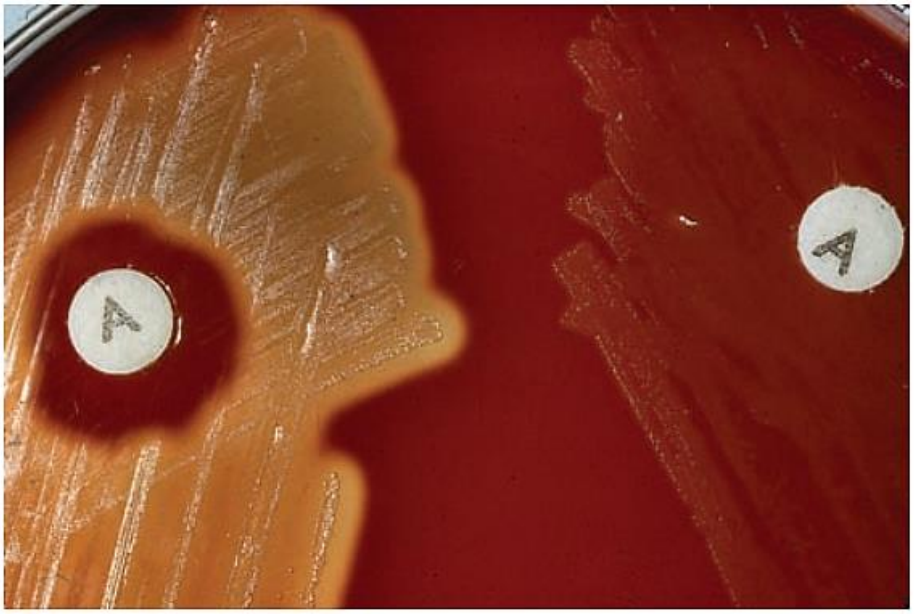
(ii)- Streptolysin S

Streptolysin is the agent responsible for the hemolytic zones around streptococcal colonizes growing on the surface of blood agar plates. It is elaborated in the presence of serum, hence the name **streptolysin S**.

Note : Not all these products are produced by every strain ; the combined action of enzymes and toxins contribute to the pathogenicity.

Culture and identification

Culture on blood agar yields characteristic **β-haemolytic** colonies (lysis of blood due to **streptolysins O** and **S**) . A Gram – Strained smear shows Gram positive cocci in chains ; which are well developed in liquid rather than in solid media . The isolate can be presumptively identified as ***Steptococcus pyogenes*** if it is sensitive to **bacitracin**.



Bacitracin sensitivity test (also known as the A-disk test). *Streptococcus pyogenes*, to the left, is susceptible to bacitracin, whereas *Streptococcus agalactiae*, to the right, is resistant. (From Winn WC Jr, et al. Koneman's Color Atlas and Textbook of Diagnostic Microbiology. 6th Ed. Philadelphia: Lippincott Williams & Wilkins, 2006.)

If rheumatic fever is suspected , then testing the patient`s **antistreptolysin O** (**ASO**) antibody titer will demonstrate previous exposure to ***Steptococcus pyogenes***.

Pathogenicity

Steptococcus pyogenes cause a number of infections ; the most notable are :

- Tonsillitis and pharyngitis
- Peritonsillar abscess (now rare)
- Scarlet fever
- Mastoiditis and sinusitis
- Otitis media (middle – ear infection)
- Wound infections leading to cellulitis and lymphangitis.
- Impetigo (a skin infection).

- **Invasive group A streptococcal disease**: Common during the first half of the century, invasive group A streptococcal (GAS) disease became rare until its resurgence during the past decade. Patients may have a deep local invasion either without necrosis (cellulitis) or with it (necrotizing fasciitis/myositis) [Note: The latter disease led to the term "flesh-eating bacteria].Invasive GAS disease often spreads rapidly, even in otherwise healthy individuals, leading to bacteremia and sepsis. Symptoms may include a toxic shock–like syndrome, fever, hypotension, multiorgan involvement, a sunburn-like rash, or a combination of these symptoms.
- **Streptococcal toxic shock syndrome (Toxic Shock–Like Syndrome [TSLS]):** This syndrome is defined as isolation of group A β-hemolytic streptococci from blood or another normally sterile body site in the presence of shock and multiorgan failure. The syndrome is mediated by the production of streptococcal pyrogenic exotoxins that function as superantigens causing massive, nonspecific T-cell activation and cytokine release. Patients may initially present with flulike symptoms, followed shortly by necrotizing soft tissue infection, shock, acute respiratory distress syndrome, and renal failure. Treatment must be prompt and includes antistreptococcal antibiotics, usually consisting of high-dose penicillin G plus clindamycin.

Complications (Sequelae)

After an episode of infection some patients develop complications, such as rheumatic fever , glomerulonephritis and erythema , which may have long – lasting effects . Note that :

- **In cellulitis** , hyaluronidase (spreading factor) mediates the subcutaneous spresd of infection.
- Erythrogenic toxin causes the **rash** of scarlet fever.
- Post –Streptococcal infection, manifesting as:

A- rheumatic fever : This autoimmune disease occurs 2 to 3 weeks after the initiation of pharyngitis. It is caused by cross-reactions between antigens of the heart and joint tissues, and the streptococcal antigen (especially the M protein epitopes). It is characterized by fever, rash, carditis, and arthritis.

B- A cute glomerulonephritis is caused by immune complexes bound to glomeruli.

Treatment and prevention

S. pyogenes has not developed resistance to **penicillin**, so **penicillin** remains the drug of choice to treat strep throat and most other *S. pyogenes* infections. Some strains have developed resistance to **erythromycin** and many strains are resistant to **tetracycline**. No vaccine available.

In a penicillin allergic patient, a macrolide such as **clarithromycin** or **azithromycin** is the preferred drug. **Penicillin G** plus **clindamycin** are used in treating necrotizing fasciitis and in streptococcal toxic shock syndrome. **Clindamycin** is added to penicillin to inhibit protein (i.e., toxin) synthesis so that a huge amount of toxin is not released abruptly from rapidly dying bacteria.

♦. *Streptococcus agalactiae* (Group β)

Increasingly recognized as a human pathogen, especially as a cause of neonatal meningitis and sepsis.

Habitat and Transmission

The human vagina , and sometimes anorectal carriage. Babies acquire infection from colonized mother during nursing .Common in cattle and cause bovine mastitis .

Characteristics

Gram – positive cocci in chains. Catalase-negative organisms.

Culture and identification

Gram – stained smear and culture which yield β- haemolytic colonies on blood agar ; colonies on blood agar are generally larger colonies and less hemolysis than group A *Steptococcus pyogenes*. Lancefield group determined by antiserum against cell wall polysaccharide.

Virulence factors

Encapsulated strains of GBS are resistant to phagocytosis by white blood cells. Other potential virulence factors include hemolysin, CAMP factor, peptidase, and hyaluronidase.

Pathogenicity

Cause neonatal meningitis and septicemia; also associated with septic abortion and gynecological sepsis .

Treatment

Penicillin and ampicillin are the antibiotics of choice ; **Erythromycin** in patients hypersensitive to penicillin.

In life-threatening infections, an aminoglycoside can be added to the regimen. [Note: Pregnant carriers should be treated with ampicillin during labor if risk factors such as premature rupture of membranes or prolonged labor are present]. Intrapartum prophylaxis of group B streptococcal carriers and administration of antibiotics to their newborns reduce neonatal group B streptococcal sepsis by as much as 90 percent.

♦. Enterococci

These belong to lancefield **group D** and include ***Enterococcus faecalis*** and other less important species.

Habitat and Transmission

Normal intestinal inhabitants.

Culture and identification

Most strains non-haemolytic, grow readily on bile-containing media, e.g. MacConkey medium. Their ability to grow in 6.5% sodium chloride, which is seven times the concentration found in normal tissue fluids.

Pathogenicity

Most commonly cause acute infections of the urinary tract, and sometimes wound infections, especially after intestinal operation.

Treatment

Antimicrobial susceptibility testing is very important for enterococci.

♦. Viridans Streptococci

This is mixed group of streptococci with variable characteristics. Hence the nomenclature of this group is in a constant state of flux. Typically they are α-hemolytic, but they may be nonhemolytic. These streptococci principally live in the oropharynx. Oral streptococci can be divided into four main 'species group' as follows:

1- ***Steptococcus mutans*** **group**

2- ***Steptococcus salivarius*** **group**

3- ***Steptococcus milleri*** **group**

4- ***Steptococcus oralis*** **group**

Each of the above groups comprise a number of species.

Habitat and Transmission

The viridans group of streptococci are common inhabitants of the oral cavity and comprise roughly one-quarter of the total cultivable flora from supragingival and gingival plaque and one-half of the isolates from the tongue and saliva. They are vertically transmitted from mother to child.

Infection endocarditis caused by the viridans group is generally a result of their entry into the bloodstream during intraoral surgical procedures (e.g. tooth extraction), and sometimes even during tooth brushing.

Culture and identification

Gram-positive cocci in chains; α-hemolytic; catalase negative. Growth not inhibited by bile or optochine (ethylhydrocupreinhydrochloride), in contrast to pneumococci. Commercially available kits are highly useful in laboratory identification of these organisms (e.g. API kit system).

Pathogenicity

The major agent of dental caries is *Steptococcus mutans* . They have a characteristic ability to produce voluminous amounts of sticky, extracellular polysaccharides in the presence of dietary carbohydrates, these help tenacious binding of the organisms to enamel and to each other.

Steptococcus mutans may act as an opportunistic pathogen under a number of circumstances. Breakdown of the dental enamel from the acidic fermentation products in the development of the carious lesion results in invasion of dentin by the microorganism and eventually in pulpal infection. *Steptococcus mutans* has been isolated from the root canals of such teeth but at a low incidence. It has been demonstrated experimentally that *Steptococcus mutans* can destroy periapical bone when it is inoculated into the dental pulp and that the same organism is isolated from the blood as late as 21 days after the inoculation.

They are also important agents of infective endocarditis. Usually bacteria released during dental procedures settle on damaged heart valves, causing infective endocarditis.

A not on *Steptococcus mutans*

Steptococcus mutans gained notoriety in the 1960s when it was demonstrated that caries could be experimentally induced and transmitted in animals which were orally inoculated with the organism. The name 'mutans' results from its frequent transition from coccal phase to coccobacillary phase.

They are currently seven distinct species of human and animal mutans streptococci (***Steptococcus cricetus, Steptococcus downei, Steptococcus ferus, Steptococcus macacae, Steptococcus mutans***, ***Steptococcus rattus,*** and ***Steptococcus sobrinus***) the term ***Steptococcus mutans*** is limited to human isolates belonging to three serotypes (***c,e*** and ***f***).

Treatment

Prophylactic therapy against **endocarditis** resulting from *Steptococcus mutans* probably is most effective with the use of **Ampicillin** combined with **Gentamicin**. The organism is also sensitive to **Tetracycline**, **Vancomycin**, **Erythromycin**, and **Chloramphenicol**, but it is resistant to **Cephalosporins** and **Streptomycin**.

♦. Streptococcus Pneumoniae (Pneumococcus)

The pneumococci (strep. Pneumoniae) are gram-positive diplococci often lancet-shape or arranged in chains, possessing a capsule of polysaccharide that permits typing with specific antisera. Pneumococci are differentiated from many species of oral streptococci by being sensitive to optochin and soluble in bile.

Pneumococci are normal inhabitants of the upper respiratory tract of 5-40% humans. S. pneumoniae is the most common cause of community acquired pneumonia and adult bacterial meningitis and is an important cause of otitis media, sinusitis and mastoiditis. The risk of disease is highest among young children (Figure), older adults, smokers, and persons with certain chronic illnesses.

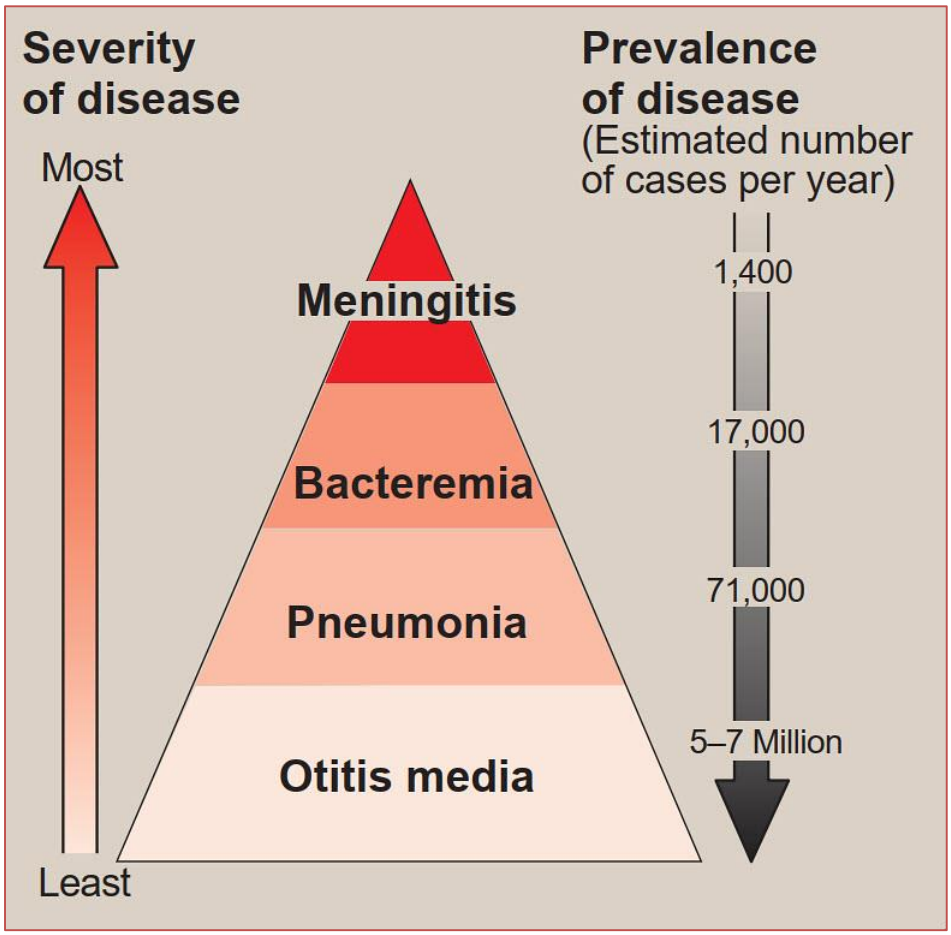


Figure Comparison of severity and prevalence of some pneumococcal infections in children in the United States.

Culture

Pneumococci form a small round colony, at central plateau with an elevated rim. Pneumococci are alph –hemolytic on blood agar.Growth is enhanced by 5-10% CO_2 .

Quellung Reaction

When pneumococci of a certain type are mixed with specific antipolysaccharide serum of the same type-or with polyvalent antiserum –on a microscope slide, the capsule swells markedly. This reaction is useful for rapid identification and for typing of the organisms, either in sputum or in cultures.

The polyvalent antiserum, which contains antibody to the 84 type (omni serum), is a good reagent for rapid microscopic determination of whether pneumo-cocci are present in fresh sputum.

Pathogenesis

A-Production of Disease

Pneumococci produce disease through their ability to multiply in the tissues. They produce no toxin of significance. The virulence of the organism is a function of its capsule, which prevents or delays ingestion by phagocytes.

A serum that contains antibodies against the type specific polysaccharide protects against infection.

B- Loss of Natural Resistance

Since 40-70% of humans are at some time carries of virulent pneumococci, the normal respiratory mucosa must possess great natural resistance to the pneumococcus. Among the factors that probably lower this resistance and thus predispose to pneumococcal infection are the Following:

1. Abnormalities of the respiratory tract

Viral and other infections that damage surface cells; abnormal accumulations of mucus (e.g. allergy), which protect pneumococci from phagocytosis.

2. Alcohol or Drug intoxication

Which depresses phagocytic activity, depresses the cough reflex, and facilitates aspiration of foreign material.

3. Abnormal circulatory dynamics (e.g. pulmonary congestion, heart failure).

4. Other mechanisms

Malnutrition, general debility, sickle cell anemia, hyposplenism, nephrosis, or complement deficiency.

Treatment

S. pneumoniae isolates were highly sensitive to penicillin G, the initial agent of choice, until the late 1980s. Since then, the incidence of penicillin resistance has been increasing worldwide. Most resistant strains remain sensitive to third generation cephalosporins (such as cefotaxime or ceftriaxone), and all are still sensitive to vancomycin. These antibiotics are therefore the agents of choice for invasive infections by penicillin-resistant strains of *S. pneumonia*.

♦. Peptostreptococcus and related Genera

These streptococci grow only under anaerobic or microaerophilic conditions and variably produce hemolysins. They are part of the normal microbiota of the mouth, upper respiratory tract, bowel, and female genital tract.

They often participate with many other bacterial species in mixed anaerobic infections. Such infections may occur in wounds in the breast, in postpartum endometritis, after rupture of an abdominal viscus, in the brain, or in chronic suppuration of the lung. The pus usually has a foul odor.

Table. Characteristics of Medically Important Streptococci.

Name	Group-Specific Substance[1]	Hemolysis[2]	Habitat	Important Laboratory Criteria	Common and Important Diseases
Streptococcus pyogenes	A	Beta	Throat, skin	Large colonies (> 0.5 mm), PYR[3] test positive, inhibited by bacitracin	Pharyngitis, impetigo, rheumatic fever, glomerulonephritis
Streptococcus agalactiae	B	Beta	Female genital tract	Hippurate hydrolysis, CAMP-positive[4]	Neonatal sepsis and meningitis
Streptococcus dysgalactiae subspecies *equisimilis;* others	C, G	Beta (human) infections), alpha, none	Throat	Large (> 0.5 mm) colonies	Pharyngitis, pyogenic infections similar to group A streptococci
Enterococcus faecalis (and other enterococci)	D	None, alpha	Colon	Growth in presence of bile, hydrolyze esculin, growth in 6.5% NaCl, PYR-positive	Abdominal abscess, urinary tract infection, endocarditis
Streptococcus bovis (non-enterococcus)	D	None	Colon	Growth in presence of bile, hydrolyze esculin, no growth in 6.5% NaCl, degrades starch	Endocarditis, common blood isolate in colon cancer
Streptococcus anginosus group (*S anginosus, S intermedius, S constellatus, S milleri* group)	F (A, C, G) and untypable	Alpha, beta, none	Throat, colon, female genital tract	Small (< 0.5 mm) colony variants of beta-hemolytic species. Group A are bacitracin-resistant and PYR-negative. Carbohydrate fermentation patterns	Pyogenic infections, including brain abscesses
Viridans streptococci (many species)	Usually not typed or untypable	Alpha, none	Mouth, throat, colon, female genital tract	Optochin-resistant. Colonies not soluble in bile. Carbohydrate fermentation patterns	Dental caries *(S mutans),* endocarditis, abscesses (with many other bacterial species)
Streptococcus pneumoniae	None	Alpha	Throat	Susceptible to optochin. Colonies soluble in bile, quellung reaction-positive	Pneumonia, meningitis, endocarditis
Peptostreptococcus (many species)	None	None, alpha	Mouth, colon, female genital tract	Obligate anaerobes	Abscesses (with multiple other bacterial species)

[1]Lancefield classification.

[2]Hemolysis observed on 5% sheep blood agar after overnight incubation.

[3]Hydrolysis of L-pyrrolidonyl-2-naphthylamide ("PYR").

[4]Christie, Atkins, Munch-Peterson test.

Streptococcus pyogenes
(group A, β-hemolytic)

- Acute pharyngitis or pharyngotonsillitis
- Acute rheumatic fever
- Erysipelas
- Puerperal sepsis
- Invasive group A streptococcal disease

1 Penicillin G[1,2]

2 Clarithromycin[3]
2 Azithromycin[3]

[1] *S. pyogenes* has not acquired resistance to penicillin G.
[2] Clindamycin may be added to pencillin G for soft tissue infection such as necrotizing fasciitis.
[3] For penicillin-allergic patient.

Streptococcus agalactiae
(group B, β-hemolytic)

- Meningitis and septicemia in neonates
- Endometritis
- Septicemia or pneumonia in individuals with impaired immune systems
- Diabetic foot infections

1 Penicillin G[4]
2 An aminoglycoside[5]

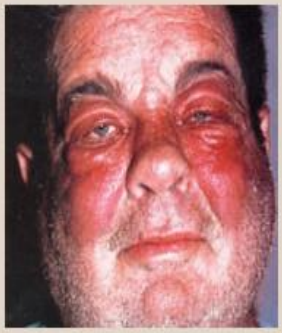
[4] All isolates remain sensitive to penicillin G and ampicillin.
[5] In life-threatening infections, an amino-glycoside can be added to the regimen.

Streptococcus pneumoniae
(α-hemolytic)

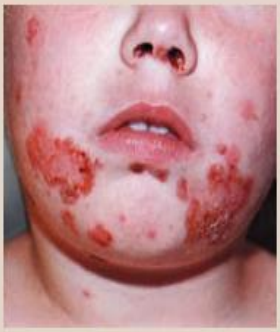
- Acute bacterial pneumonia
- Otitis media
- Meningitis

1 Penicillin G[6]
1 Cefotaxime
1 Ceftriaxone
2 Vancomycin[7]

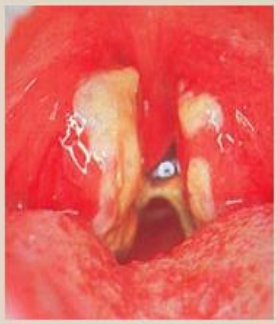
[6] Penicillin G has been the drug of choice, but resistant strains are regularly seen.
[7] Most resistant strains remain sensitive to vancomycin.



Facial erysipelas



Impetigo



Streptococcal pharyngitis

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