The Streptococci

• Diverse collection of cocci.
• Gram-positive
• Chains or pairs
• significant pathogens
• Strong fermenters
• Facultative anaerobes
• Non-motile
• Catalase Negative

Classification

16.1 *Streptococcus* species of clinical importance

Phylogenetic group	Species	Lancefield group	Type of haemolysis[a]
Pyogenic group	*Str. pyogenes*	A	β
	Str. agalactiae	B	β
	Str. equisimilis	C	β
Mitis group	*Str. pneumoniae*	O	α
	Str. mitis	O	α
	Str. oralis	Not identified	α
	Str. sanguis	H	α
	Str. gordonii	H	α
Anginosus group	*Str. anginosus*	G, F (and A)	α
	Str. intermedius		α
Salivarius group	*Str. salivarius*	K	None
Bovis group	*Str. bovis*	D	α or none
Mutans group	*Str. mutans*	Not designated	None
	Str. sobrinus	Not designated	None

[a] On horse blood agar.

• The pyogenic group

• The mitis group

• The anginosus group

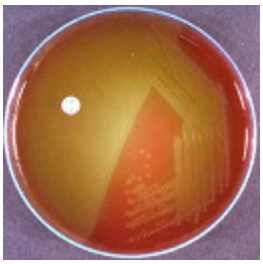
• The salivarius group

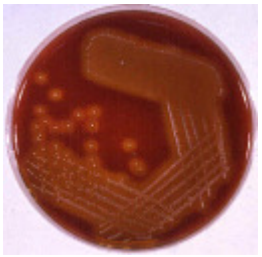
• The bovis group

• The mutans group

Haemolytic activity

• α-haemolysis





• β-haemolysis

Lancefield Grouping

• Serological classification
  – Means of distinguishing PYOGENIC Streps
• Differences in cell wall polysaccharides

Streptococcus pyogenes
• Lancefield Group A
• Most prevalent
• Exclusively human pathogens
• Infection Types
  – Suppurative
  – Toxin related
  – Non-suppurative

Str. pyogenes – suppurative

• **non-invasive**
  – pharyngitis skin infection, impetigo
• **invasive bacteremia**
  – toxic shock-like syndrome
  – "flesh eating" bacteria
• **pyrogenic toxin**

Suppurative
• **Pyrogenic toxin**
  – superantigen
  – T cell mitogen
  – activates immune system

• **Scarlet fever**
  – rash
  – erythrogenic toxin

Non-suppurative

• rheumatic fever
• inflammatory disease
• life threatening
• chronic sequalae
• fever
• heart
• joints
• rheumatic NOT rheumatoid arthritis
• Rheumatic fever
• (penicillin: terminates pharyngitis & decreases carditis)
• Acute glomerulonephritis (immune complex disease of kidney)
• Rheumatic fever –etiology
• M protein (cross-reacts heart myosin; autoimmunity)
• cell wall antigens (poorly digested in vivo persist indefinitely)

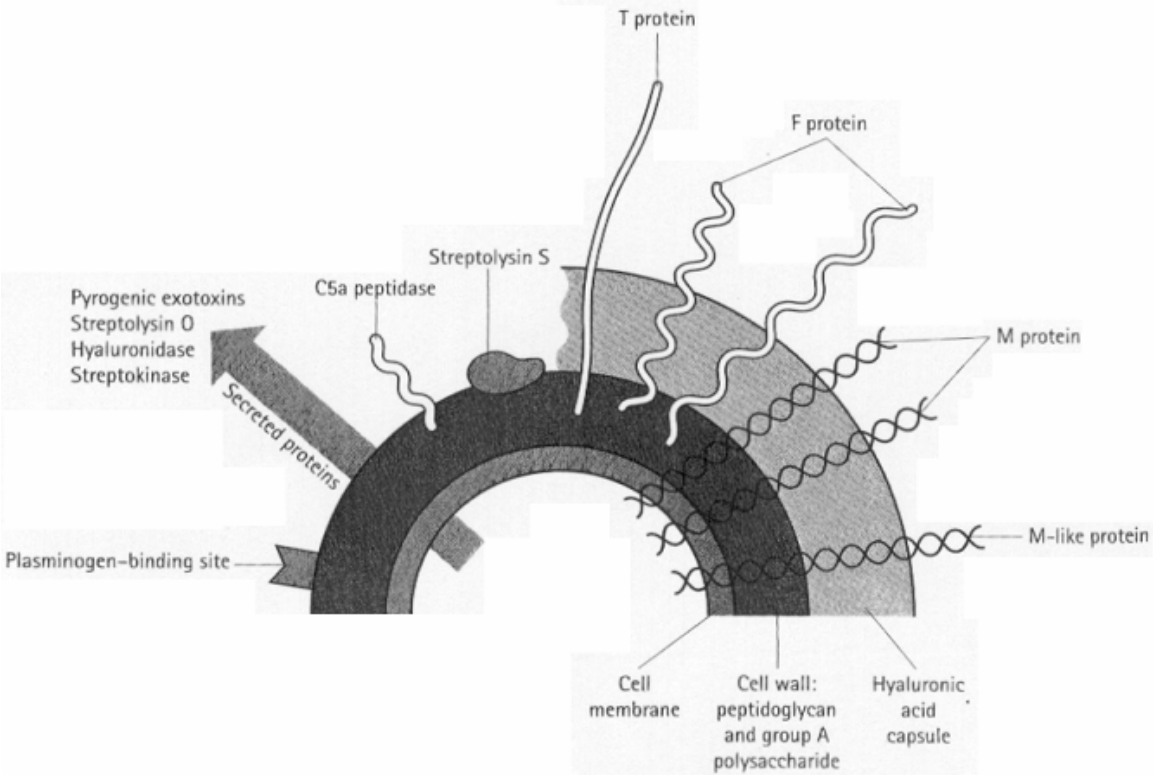
Str. pyogenes: Pathogenesis
• Lancefield Group A

• Human pathogen

• Suppurative infections
  – Respiratory tract
  – Soft tissue infections http://www.4um.com/tutorial/icm/softiss.htm
  – Toxin associated infections

Spectrum of infection resembles that of Staph. aureus BUT virulence characteristics DIFFER

Location of virulence associated factors Pathogenesis

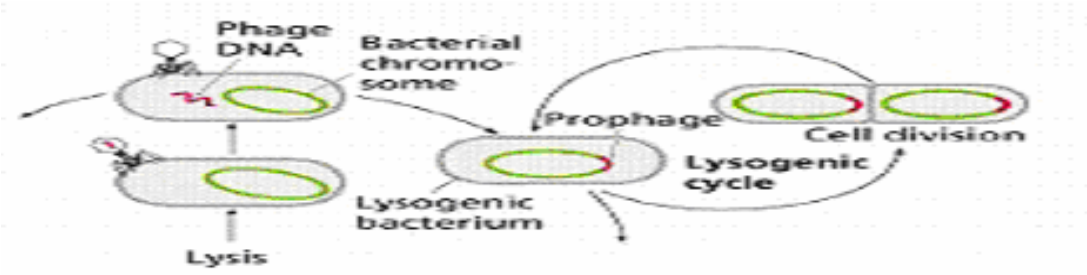


Virulence factors:Pathogenesis

• Any attributes that are conducive to the process of infection
  • adherence
  • evasion of the host immune system and
  • tissue damage

• Variability
• Genetic info transfer via “transduction”

Transduction



Virulence factors: adhesion

- Principal mechanism
  - F protein===fibronectin
- Adherence
- Entry of the organism INTO the cell
  - Lipoteichoic acid
  - M protein

Virulence factors: M protein

- Resistance to phagocytosis
- Fibril
  Cytoplasmic membrane
  Cell wall
- Polymorphism in gene encoding→variability
  >80 forms
- Strains may have :
  - More than 1 type on their surface and
  - M-like
- Bind to host serum proteins

Virulence factors: Capsule

- Hyaluronic acid capsule
  - Some strains
- Severe infections
- Mucoid colonies on blood agar
- Anti-phagocytic effect
  - Variable significance

Streptococcus agalactiae

- Lancefield Group B
- Primary habitat is the human colon; other areas include
  - throat
  - vagina (10→40%)
- Bovine mastitis

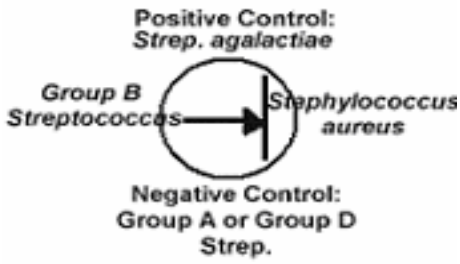
Str. agalactiae: Pathogenesis

- virulence factors

  - haemolysins
  - capsule polysaccharide; (9 different types)
  - C5a peptidase (only the human pathogenic strains)
  - hyaluronidase (not all strains)
  - various surface proteins

CAMP

– Means of lab recognition of Str. Agalactiae



http://gold.aecom.yu.edu/id/micro/camp.htm

***Str. agalactiae*: Features**

• Infections in Neonates
  – Early onset (at or T<12 h of birth)
  – Late onset (T> 7 days but T<3 months)

• Newborn babies become infected in three ways:
  – before birth
  – during birth
  – after birth

Early onset disease

• Risk factors for early onset disease include:

  • Premature delivery
  • Multiple births
  • Premature rupture of membranes before the onset of labour
  • Prolonged rupture of membranes lasting more than 18 hours before delivery
  • Urinary tract infection in mother caused by Group B Strep
  • Lack of antibody in mother against type of Group B Strep carried in birth canal
  • Fever in mother during labour
  • History of previous newborn with Group B Strep disease.

Late onset disease

• Bacteraemia alone or in conjunction with other conditions

  – purulent meningitis
  – pneumonia
  – septic arthritis
  – osteomyelitis
  – conjunctivitis
  – sinusitis
  – otitis media
  – endocarditis
  – peritonitis

Infection in the adult

• Pregnancy / recent post partum
  – Ascending spread
  – Abortion, chorioamnionitis,post partum sepsis,
  – other infections

e.g. pneumonia in the post partum period

• Non pregnant adults
  – sepsis, pneumonia, soft tissue infections, and UTI
  – complicated by bacteraemia.
• Elderly

Str. Suis *(Group R streptococcus)*

• septicaemia and meningitis in pigs.
• occasionally infect people
  * Through contaminated pork or infected pigs, and
  * cause septicaemia, meningitis, and respiratory tract infections.

Streptococcus pneumoniae

• ***pneumococcus***
  – oropharyngeal flora of 5-70% of the population
  – highest isolation rate in children during the winter months.
• diplococcus
• an important pathogen
  – polysaccharide capsule
• genetically very flexible

Streptococcus pneumoniae:*Pathogenesis*

• Capsule
http://www1.indstate.edu/thcme/micro/s-pneum2.html

• Antiphagocytic
http://www.medschool.lsumc.edu/Micr/COURSES/DMIP/opson.htm
http://medtech.cls.msu.edu/ISL/immunology/opsonize.htm

• IgA1 protease
• Pneumolysin
  – Neutrophil chemotaxis
  – Phagocytosis and the respiratory burst
    http://www.cellsalive.com/nbt.htm
• Autolysin

Clinical features: pneumonia

• Predisposing conditions
• Person-to-person spread is uncommon.

• Possible causes include:
  – Aspiration into the lower respiratory tract
  – Terminal events
  – Immune deficiencies

The Commensal Streptococci: Viridians Streptococci

• Viridans group dominant resident oral flora
  – Mitis
  – Salivarius

• Inhibit colonization of many pathogens
  – Bacteriocins
  – H_2O_2

Viridans streptococci

• ill defined group of species
• α- haemolysis on blood agar,
• may have a variety or no Lancefield antigens.
• The most common are Str. sanguis, mitis, mutans and salivarius.
• Most are commensals of the mouth and upper respiratory tract.

Bacteriocins

• http://www.biochem.ucl.ac.uk/bsm/PLASMID/Bacteriocins.htm
• Agents
• Encoded in the genetic material carried by plasmids
• There purpose is to of kill or inhibit closely related species or even different strains of the same species
• Specific
• Survival in an overpopulated environment

Mitis group

• *Str. mitis*, *Str. oralis*, *Str. sanguis* and *Str. Gordonii*

• Colonize tooth surfaces & mucosal membranes

• May enter the bloodstream

• In healthy individuals

  →cleared from circulation within 1 hour

• Not so with other predisposing factors present

  →post-streptococcal rheumatic fever

• Where the primary cause of infective endocarditis

Mutans group

• *Str. mutans* and *Str. sobrinus*
  – Exclusively colonize tooth enamel
  – Only after eruption
• Lactic acid producers
• may cause subacute bacterial endocarditis.

Anginosus Group

• *Str. anginosus* , *Str. intermedius* & others
• regular commensals
  – tooth surfaces
  – gingival crevices.
• isolated from abscesses and other purulent opportunistic infections

Bovis Group

• Str. Bovis
• Various animal species
• Human gut
• Occasionally causes
  – Bacteraemia
  – Subacute endocarditis
• Associated with colon carcinoma

Enterococcus

• natural habitat in the human intestines
  – **E. faecalis**
  – **E. faecium**
• Associated diseases
  – Urinary tract infection (in hospitals, sporadic outbreaks)
  – Infective endocarditis
  – Biliary tract infections
  – Suppurative abdominal lesions
  – Peritonitis
• Poor prognosis