RES-209

BACTERIA CAUSING UPPER RESPIRATORY TRACT INFECTIONS

by

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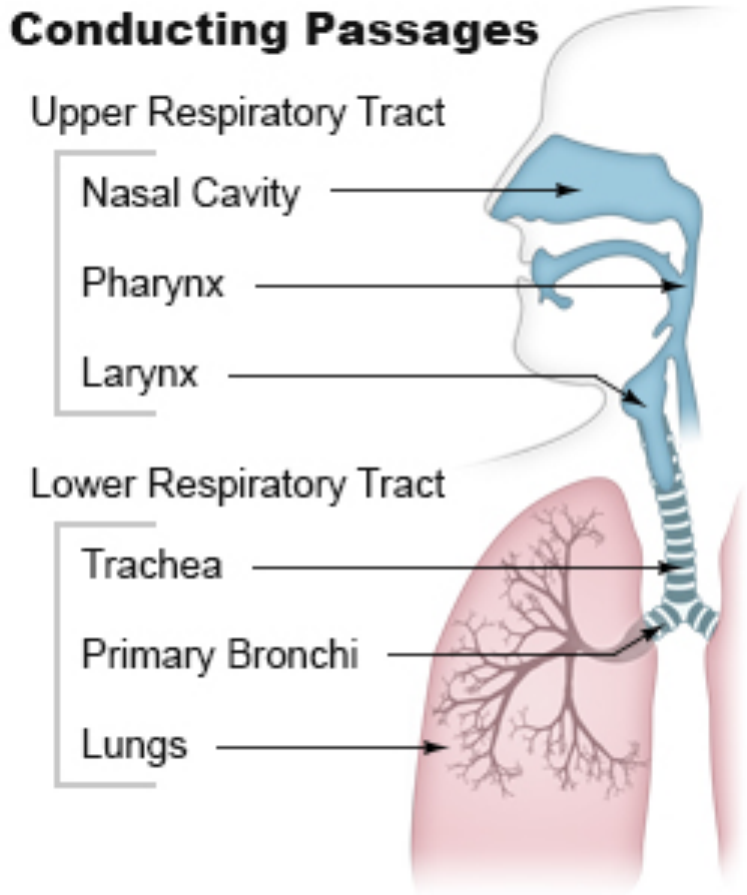
OBJECTIVES

By the end of this session, you will be able to:

- Describe the etiology of the common upper respiratory tract infections caused by bacteria.
- Understand the pathogenesis, lab diagnosis and prevention of infections discussed in the lecture.
- Compare the major characteristics of specific bacterial diseases of the upper respiratory tract

UPPER RESPIRATORY TRACT INFECTIONS

- Common cold (mainly by viruses)
- Pharyngitis
- Tonsillitis
- Epiglottitis
- laryngotracheitis
- Sinusitis
- Otitis Media
- Otitis Externa



Pharyngitis

- Abrupt onset of sore throat, fever, malaise, and headache.
- May be complicated by tonsillar abscesses and tender anterior cervical lymph nodes.

Common bacterial causes:

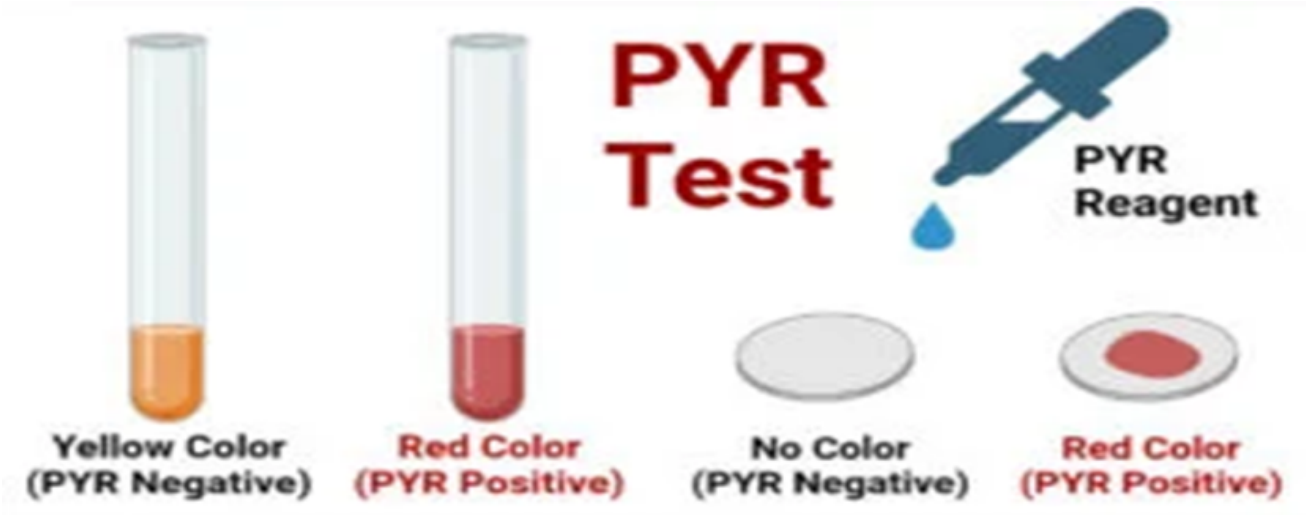
- Group A *streptococcus (Strep. pyogenes*).
- Anaerobic bacteria
- *Corynebacterium diphtheriae*
- *Hemophilus influenzae*

Streptococcus pyogenes (Group A *Streptococcus*; GAS)

Distinguishing Features

- β hemolytic
- Bacitracin sensitive
- Pyrrolidonyl arylamidase (PYR) positive

Presumptive identification of certain groups of bacteria based on the activity of the enzyme pyrolidonyl arylamidase



Pathogenesis

1. **M-protein:** antiphagocytic, associated with post-streptococcal acute glomerulonephritis nephritis and rheumatic fever.

2. **Streptolysin O:** hemolysin/cytolysin.

3. **Spreading Factors:**

• Streptokinase: breaks down fibrin clot.

• Streptococcal DNAse: liquefies pus causing extension of lesion.

• Hyaluronidase: hydrolyzes the ground substances of connective tissues.

4. Exotoxins A–C (pyrogenic and erythrogenic exotoxins):

Cause fever and rash of scarlet fever; act as superantigens

Reservoir: human throat; skin

Transmission: direct contact and respiratory droplets.

Diseases:

Acute Suppurative Group A Streptococcal Infections*

Diseases	Symptoms
Pharyngitis	Abrupt onset of sore throat, fever, malaise, and headache; tonsillar abscesses and tender anterior cervical lymph nodes
Scarlet fever	Above followed by a blanching **"sandpaper" rash** (palms and soles are usually spared), circumoral pallor, **strawberry tongue**, and nausea/vomiting
Pyoderma/impetigo	Pyogenic skin infection (honey-crusted lesions)

Nonsuppurative Sequelae to Group A Streptococcal Infections

Disease	Sequelae of	Mechanisms/Symptoms
Rheumatic fever	Pharyngitis with group A strep	Antibodies to heart tissue/2 weeks post pharyngitis, fever, joint inflammation, carditis, erythema marginatum (chorea later)type II hypersensitivity
Acute glomerulonephritis (AGN)	Pharyngitis or skin infection	Immune complexes bound to glomeruli/pulmonary edema and hypertension, “smoky” urine (type III hypersensitivity)

Corynebacterium diphtheriae

Distinguishing Features

• club-shaped gram-positive bacilli arranged in V or L shapes.

Gray-to-black colonies on blood tellurite agar.

• Contain volutin granules (metachromatic granules).

• Pathogenic strains are lysogenized by β-prophage carrying genes for the toxin

Production.

Reservoir: throat and nasopharynx

Transmission: respiratory droplets

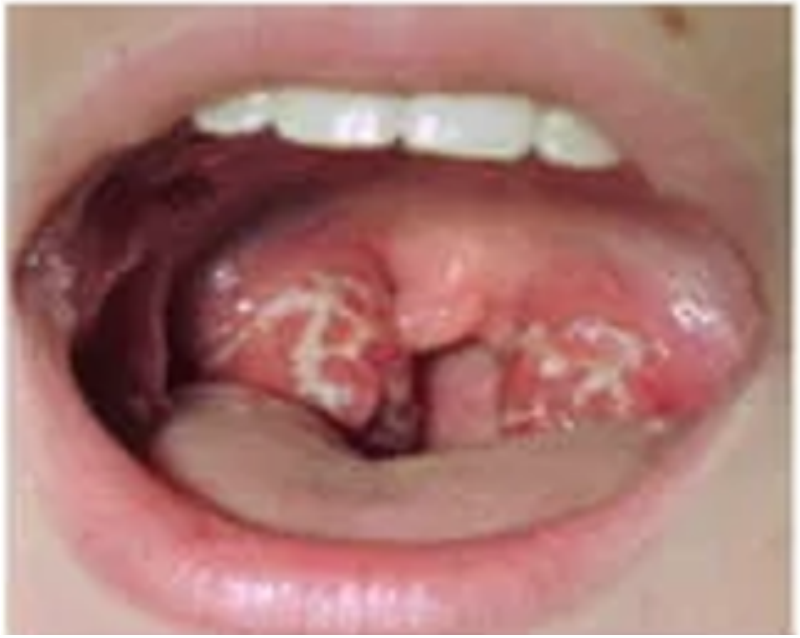
Pathogenesis

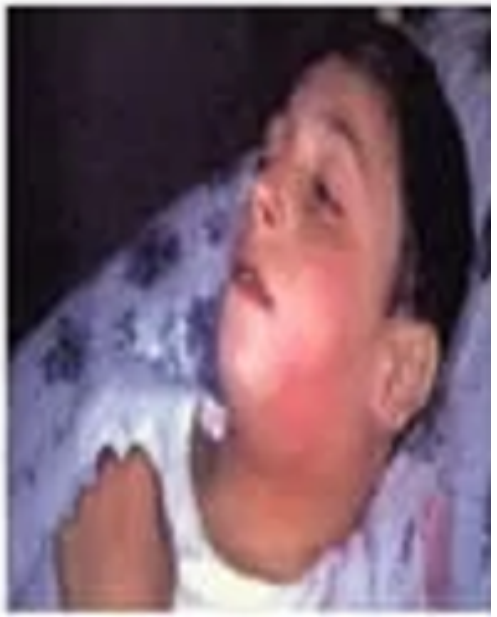
- Organism is not invasive. However, it colonizes epithelium of oropharynx or skin (in cutaneous diphtheria).
- Disease manifestations occur due to Diphtheria toxin (A-B component) → inhibits protein synthesis by adding ADP-ribose to eEF-2 causing cell death.
- Dirty gray pseudo-membrane (made up of dead cells and fibrin exudate, bacterial pigment) is formed in oropharynx.
- Extension of pseudo-membrane into larynx/trachea causes airway obstruction.
- toxin also causes heart, kidney and nerve damage.

Disease: diphtheria

sore throat with pseudo-membrane and bull neck.

Potential respiratory obstruction, cardiac dysfunction and renal failure.





Diagnosis

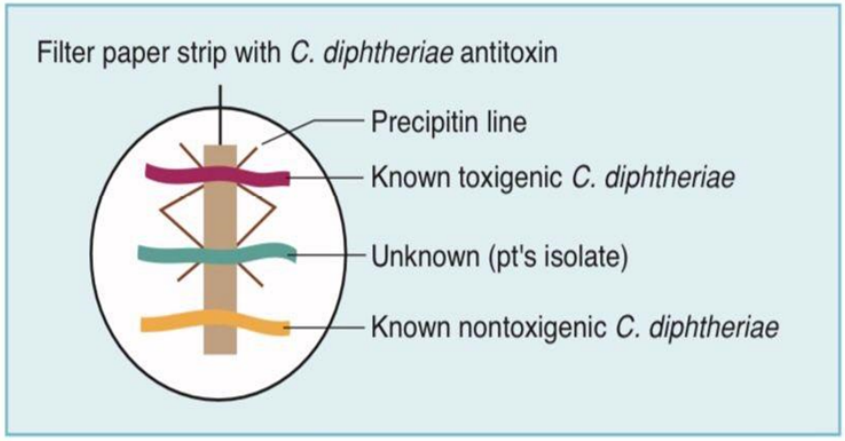
Elek test to document toxin production (ELISA for toxin is now gold standard)

- If the strain is pathogenic, Toxin is produced diffuses away from growth
- Antitoxin diffuses away from strip of filter paper.
- Precipitin lines form at zone of equivalence

Prevention:

toxoid vaccine (formaldehyde-modified toxin is still immunogenic but with no toxicity), part of DTaP, DTP, or Td

Boosters: 10-year intervals



Elek Test

Hemophilus influenzae

Distinguishing Features

• Encapsulated, gram-negative bacilli; 95% of invasive disease caused by capsular type b

• Requires growth factors X (hemin) and V (NAD) for growth on nutrient or blood agar

• Chocolate agar provides both X and V factors

• Grows near *S. aureus* on blood agar "satellite" phenomenon.

Reservoir: human nasopharynx

Pathogenesis

- Polysaccharide capsule is the most important virulence factor
- Capsule is important in:
- diagnosis by antigen screening in CSF (e.g. by latex particle agglutination)
- serotyping of isolates.
- IgA protease is a mucosal colonizing factor.

Diagnosis:

- Blood or throat culture on chocolate agar
- Capsular antigen detection.
- PCR

Prevention

- Conjugate capsular polysaccharide-protein vaccine

Bordetella pertussis

B. pertussis causes whooping cough (pertussis).

Distinguishing Features:

small gram-negative aerobic encapsulated bacilli.

Reservoir: human

Transmission: respiratory droplets

Pathogenesis

1. Attachment of *B. pertussis* to nasopharyngeal ciliated epithelial cells is via filamentous hemagglutinins.

2. Production of toxins that cause respiratory epithelium damage:

- Tracheal cytotoxin: interferes with ciliary action; kills ciliated cells
- Endotoxin
- Pertussis toxin (A and B component toxin):
- interferes with transfer of signals from cell surface to intracellular mediator system.
- It causes lymphocytosis, islet-activation leading to hypoglycemia, blocking

Diagnosis

- Bordet-Gengou media is used: either direct cough plates or nasopharyngeal cultures.
- Direct immunofluorescence (DFA) on nasopharyngeal smear
- PCR and serologic tests.

Prevention:

- DTaP vaccine (acellular pertussis: filamentous hemagglutinin plus pertussis toxoid).
- immunity wanes 5–7 years.

Recall question

A 6 –year- old girl is brought to a doctor with fever and sore throat. On physical examination the child anxious, ill appearing and reveals a gray pseudo-membrane involving pharyngeal wall. The mother states that the child has not received any immunization. Which one of the following virulence factors is involved in the pathogenesis of this disease?

A-Capsule.

B-Endotoxin

C-Exotoxin

D-Hemolysins


Thank you