Defenses

• Respiratory system
• Normal flora
• Protection
Respiratory tract system

- Most common entry point for infections
- Upper tract
  - Mouth, nose, nasal cavity, sinuses, throat, epiglottis, and larynx
- Lower tract
  - Trachea, bronchi, and bronchioles in the lungs
Anatomy of the respiratory tract.

Fig. 21.1 The respiratory tract.
Normal flora

- Commensals
- Limited to the upper tract
- Mostly Gram positive
- Microbial antagonist (competition)
- Immunocompromised individuals are at risk of infection
Protection

- Nasal hair
- Cilia
- Bronchi
- Mucus
- Involuntary responses (coughing, etc.)
- Immune cells
Upper respiratory tract

- Common cold
- Sinusitis
- Ear infections
- Pharyngitis
- Diphtheria (mentioned)
- Influenza (may also involve lower respiratory tract in serious cases)
Common cold

- Viral infection
  - Over 200 viruses are involved
- Rhinitis
- Prevalent among human population
- Prone to secondary bacterial infections
- No vaccine
- No chemotherapeutic agents
- Costly
**Features of rhinitis.**

### CHECKPOINT 21.1 Rhinitis

<table>
<thead>
<tr>
<th>Feature</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Causative Organism(s)</strong></td>
<td>200-plus viruses</td>
</tr>
<tr>
<td><strong>Most Common Modes of Transmission</strong></td>
<td>Indirect contact, droplet contact</td>
</tr>
<tr>
<td><strong>Virulence Factors</strong></td>
<td>Adhesins; most symptoms induced by host response</td>
</tr>
<tr>
<td><strong>Culture/Diagnosis</strong></td>
<td>Not necessary</td>
</tr>
<tr>
<td><strong>Prevention</strong></td>
<td>Hygiene practices</td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>For symptoms only</td>
</tr>
</tbody>
</table>
Sinusitis

- Bacterial infection
- Viral infections
- Rare fungal infection
- Inflammation of the sinuses
- Noninfectious allergies are primary cause of most sinus infections
Features of sinusitis.

### Checkpoint 21.2 Sinusitis

<table>
<thead>
<tr>
<th>Feature</th>
<th>Bacterial Sinusitis</th>
<th>Fungal Sinusitis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Causative Organism(s)</strong></td>
<td>Various bacteria, often mixed infection</td>
<td>Various fungi</td>
</tr>
<tr>
<td><strong>Most Common Modes of Transmission</strong></td>
<td>Endogenous (opportunism)</td>
<td>Introduction by trauma or opportunistic overgrowth</td>
</tr>
<tr>
<td><strong>Virulence Factors</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Culture/Diagnosis</strong></td>
<td>Culture not usually performed; diagnosis based on clinical presentation, occasionally X rays or other imaging technique used</td>
<td>Same</td>
</tr>
<tr>
<td><strong>Prevention</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td>Broad-spectrum antibiotics</td>
<td>Physical removal of fungus; in severe cases antifungals used</td>
</tr>
<tr>
<td><strong>Distinctive Features</strong></td>
<td>Much more common than fungal</td>
<td>Suspect in immunocompromised patients</td>
</tr>
</tbody>
</table>
Ear infection

- Bacterial infection
- Acute otitis media
- Common sequela of rhinitis
- Effusion
- Biofilm bacteria may be associated with chronic otitis media
Bacteria can migrate along the eustachian tube from the upper respiratory tract, and a buildup of mucus and fluids can cause inflammation and effusion.

Fig. 21.2 An infected middle ear.
### Checkpoint 21.3 Otitis Media

<table>
<thead>
<tr>
<th>Causative Organism(s)</th>
<th>Streptococcus pneumoniae</th>
<th>Haemophilus influenzae</th>
<th>Other bacteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Most Common Modes of Transmission</td>
<td>Endogenous (may follow upper respiratory tract infection by S. pneumoniae or other microorganisms)</td>
<td>Endogenous (follows upper respiratory tract infection)</td>
<td>Endogenous</td>
</tr>
<tr>
<td>Virulence Factors</td>
<td>Capsule, hemolysin</td>
<td>Capsule, fimbriae</td>
<td>–</td>
</tr>
<tr>
<td>Culture/Diagnosis</td>
<td>Usually relies on clinical symptoms and failure to resolve within 72 hours</td>
<td>Same</td>
<td>Same</td>
</tr>
<tr>
<td>Prevention</td>
<td>Pneumococcal conjugate vaccine (heptavalent)</td>
<td>Hib vaccine</td>
<td>None</td>
</tr>
<tr>
<td>Treatment</td>
<td>Wait for resolution; if needed, amoxicillin (are high rates of resistance) or trimethoprim/sulfamethoxazole</td>
<td>Wait for resolution; if needed, ceftriaxone or ampicillin if isolate is sensitive</td>
<td>Wait for resolution; if needed, a broad-spectrum antibiotic (azithromycin) might be used in absence of etiological diagnosis</td>
</tr>
<tr>
<td>Distinctive Features</td>
<td>–</td>
<td>–</td>
<td>Suspect if fully vaccinated against other two</td>
</tr>
</tbody>
</table>
Pharyngitis

- Bacterial infection
- Viral infection
- *Streptococcus pyogenes* – most serious type
  - Scarlet fever
  - Rheumatic fever
  - Glomerulonephritis
Streptococcus pyogenes

- Group A is virulent
- Streptolysins - toxin (hemolysins)
- Erythrogenic – toxin
- Toxins can act as superantigens
  - Overstimulate T cells
    - Tumor necrosis factor
Scarlet fever

- *S. pyogenes* is infected with a bacteriophage
  - Erythrogenic toxin - rash
- Sandpaper-like rash
  - Neck, chest, elbows, inner thighs
- Children are at risk
Rheumatic fever

- M protein
- Immunological cross-reaction (molecular mimicry)
- Damage heart valves
- Arthritis, nodules over bony surfaces
Glomerulonephritis

- Bacterial antigen-antibody complexes
- Deposit on the glomerulus of the kidney
- Kidney damage
*Streptococcus* infection causing inflammation of the throat and tonsils.

Fig. 21.3 The appearance of the throat in pharyngitis and Tonsilitis.
Group A streptococcal infections can damage the heart valves due to cross-reactions of bacterial-induced antibodies and heart proteins.

Fig. 21.4 The cardiac complications of rheumatic fever.
Influenza

- Viral infection
- Prevalent during the winter season
- Glycoproteins
  - Hemagglutinin (HA)
  - Neuraminidase (N)
- Antigenic drift
- Antigenic shift
The influenza virus is an enveloped virus with two important surface glycoproteins called hemagglutinin and neuraminidase.

Fig. 21.11 Schematic drawing of influenza virus.
Influenza symptoms

- 1-4 day incubation period
- Headache
- Chills
- Dry cough
- Body aches
- Fever
- Stuffy nose
- Sore throat
Influenza complications

- Vulnerable to secondary infections
- Pneumonia
- Possible death
- Prognosis can be poor for young, old, ill or pregnant
Glycoproteins

• Hemagglutinin
  – Specific residues bind to host cell receptors of the respiratory mucosa
  – Different residues from above are recognized by the host immune system (antibodies)
    • Residues are subject to changes (antigenic drift)
  – Agglutination of rbc
Hemagglutinin is a viral glycoprotein that is involved in binding to host cell receptors on the respiratory mucosa.

Fig. 21.12 Schematic drawing of hemagglutinin of influenza Virus.
Glycoproteins

• Neuraminidase (N)
  – Breaks down protective mucous coating
  – Assist in viral budding
  – Keeps viruses from sticking together
  – Participates in host cell fusion
Antigenic shift involves gene exchange, which encode for viral glycoproteins, between different influenza viruses, thereby the new virus is no longer recognized by the human host.

Fig. 21.13 Antigenic shift event.
Features of influenza.

<table>
<thead>
<tr>
<th>CHECKPOINT 21.8 Influenza</th>
</tr>
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<tbody>
<tr>
<td><strong>Causative Organism(s)</strong></td>
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<tr>
<td><strong>Most Common Modes of Transmission</strong></td>
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<td><strong>Virulence Factors</strong></td>
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</tr>
<tr>
<td><strong>Treatment</strong></td>
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</table>
“Bird Flu”/Avian Influenza

- Why the concern?
- What is a pandemic?
- What can we do to protect ourselves?
- What would happen if antigenic shift occurred with this strain and human influenza today?