

# Upper Respiratory Infections

Based on Cowan & Talaro

Microbiology: A Systems  
Approach Chapter 21

# 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.

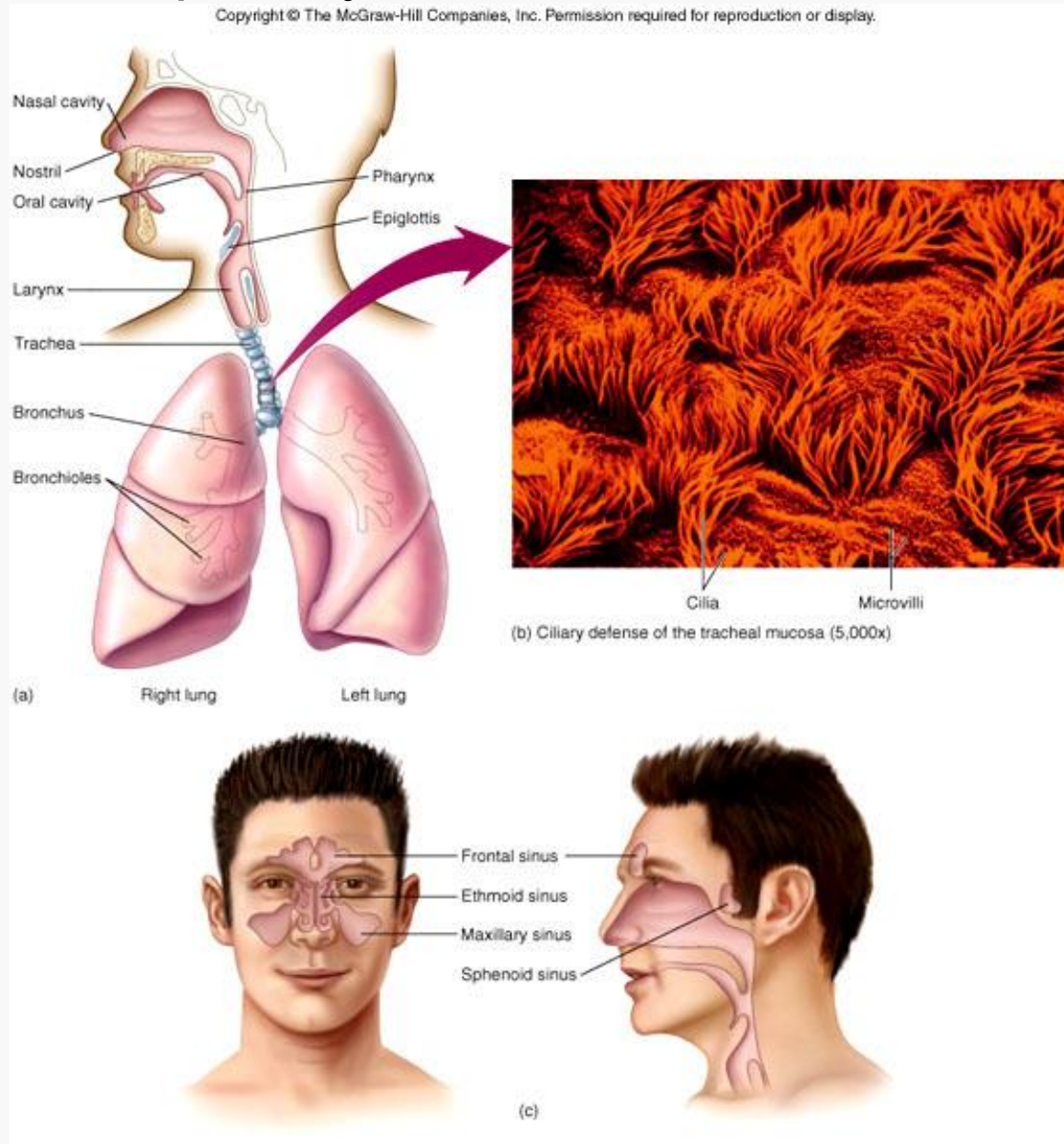


Fig. 21.1 The respiratory tract.

# 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.

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## CHECKPOINT 21.1 Rhinitis


<b>Causative Organism(s)</b>	200-plus viruses
<b>Most Common Modes of Transmission</b>	Indirect contact, droplet contact
<b>Virulence Factors</b>	Adhesins; most symptoms induced by host response
<b>Culture/Diagnosis</b>	Not necessary
<b>Prevention</b>	Hygiene practices
<b>Treatment</b>	For symptoms only

# Sinusitis

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

# Features of sinusitis.

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 CHECKPOINT 21.2 Sinusitis		
Causative Organism(s)	Various bacteria, often mixed infection	Various fungi
Most Common Modes of Transmission	Endogenous (opportunism)	Introduction by trauma <i>or</i> opportunistic overgrowth
Virulence Factors	–	–
Culture/Diagnosis	Culture not usually performed; diagnosis based on clinical presentation, occasionally X rays or other imaging technique used	Same
Prevention	–	–
Treatment	Broad-spectrum antibiotics	Physical removal of fungus; in severe cases antifungals used
Distinctive Features	Much more common than fungal	Suspect in immunocompromised patients

## Checkpoint 21.2 Sinusitis

# 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.

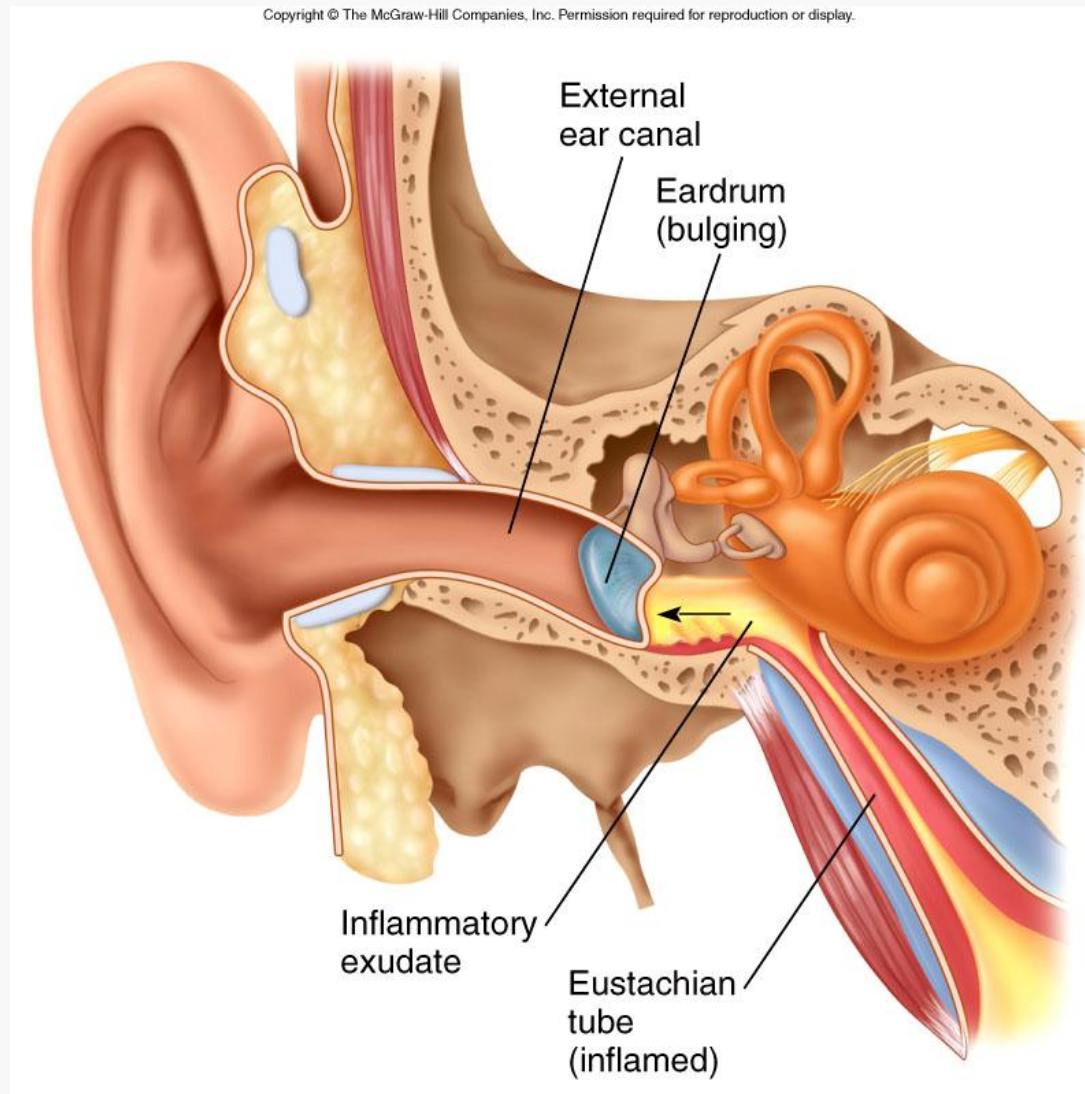


Fig. 21.2 An infected middle ear.

# Features of otitis media.

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## CHECKPOINT 21.3 Otitis Media

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

## Checkpoint 21.3 Otitis media.

# 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.

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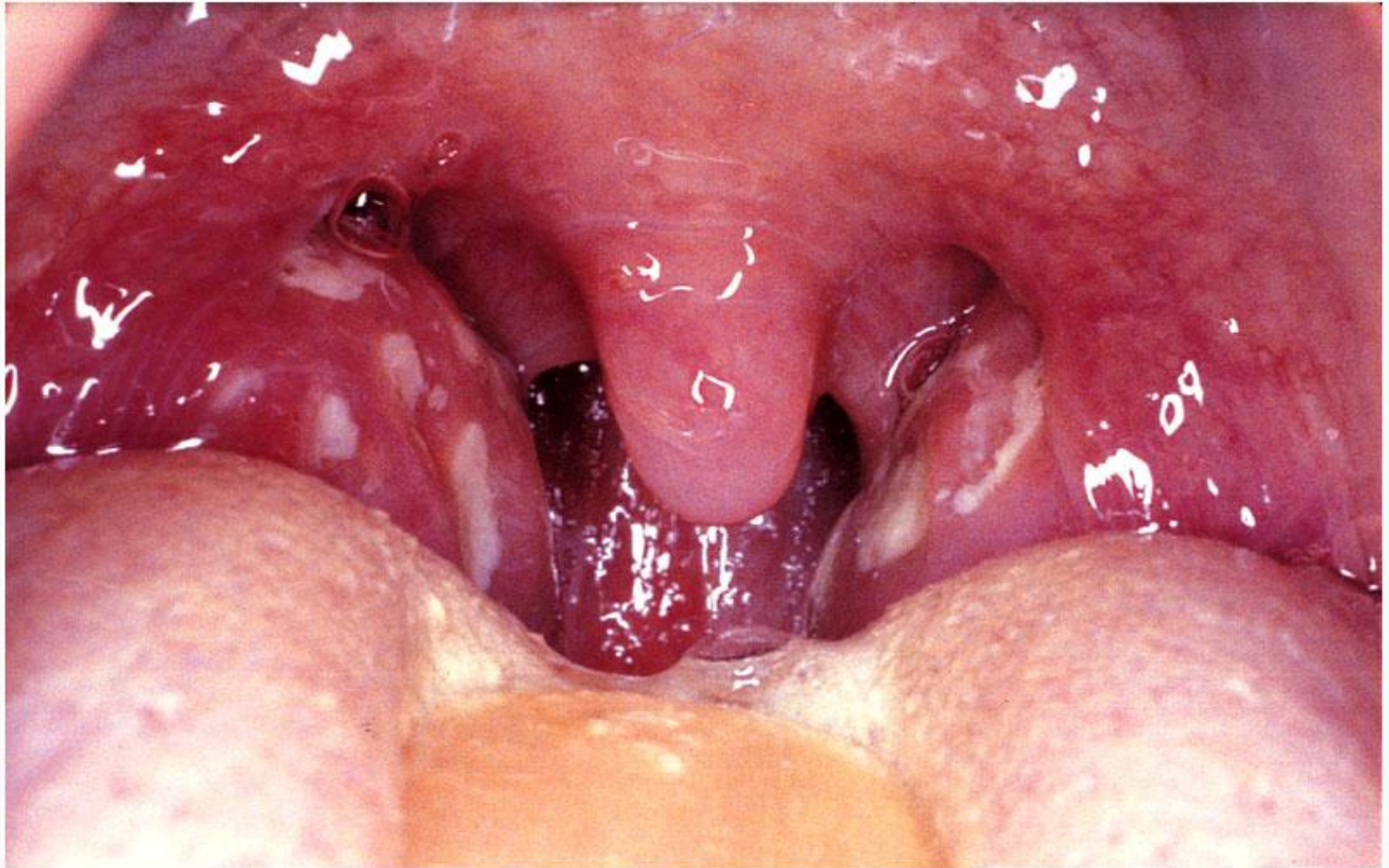


Fig. 21.3 The appearance of the throat in pharyngitis and Tonsillitis.

Group A streptococcal infections can damage the heart valves due a cross-reactions of bacterial-induced antibodies and heart proteins.

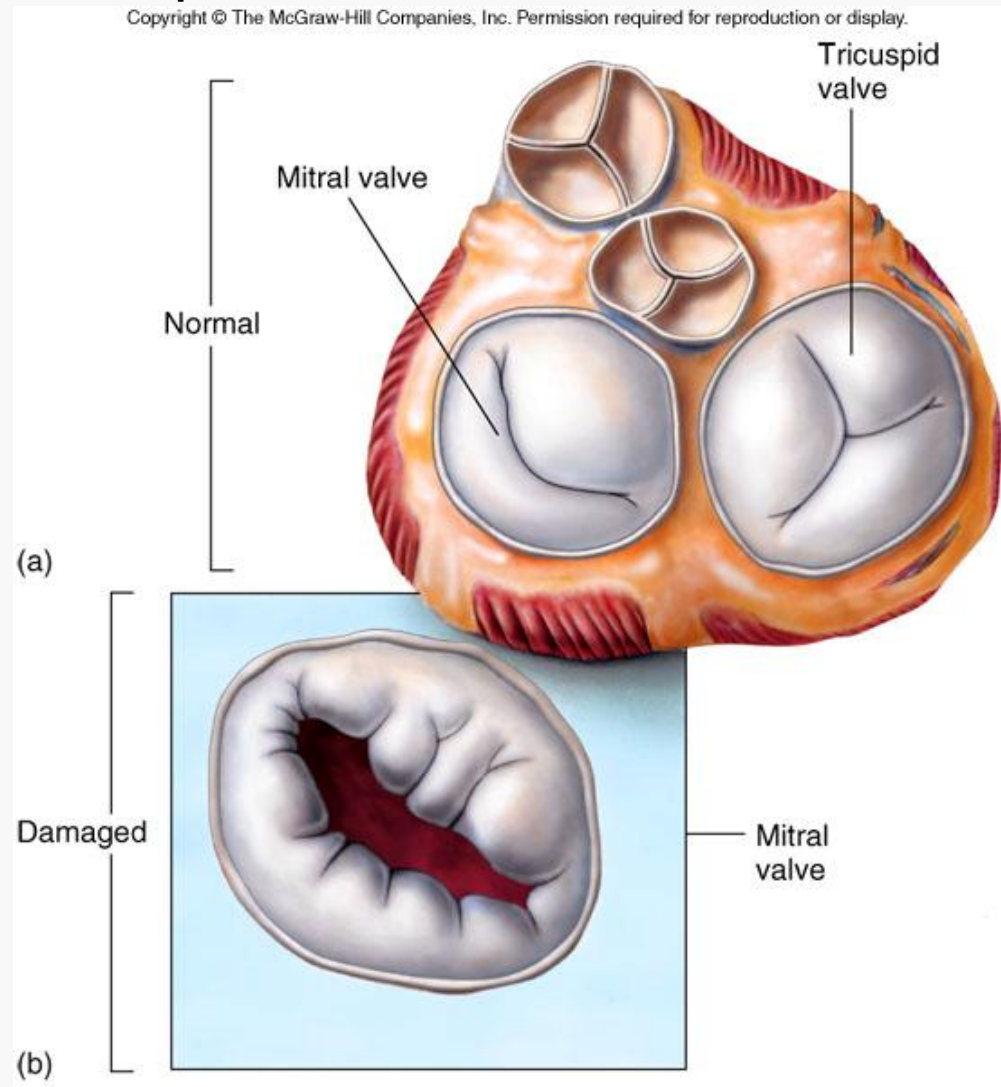


Fig. 21.4 The cardiac complications of rheumatic fever.

# 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.

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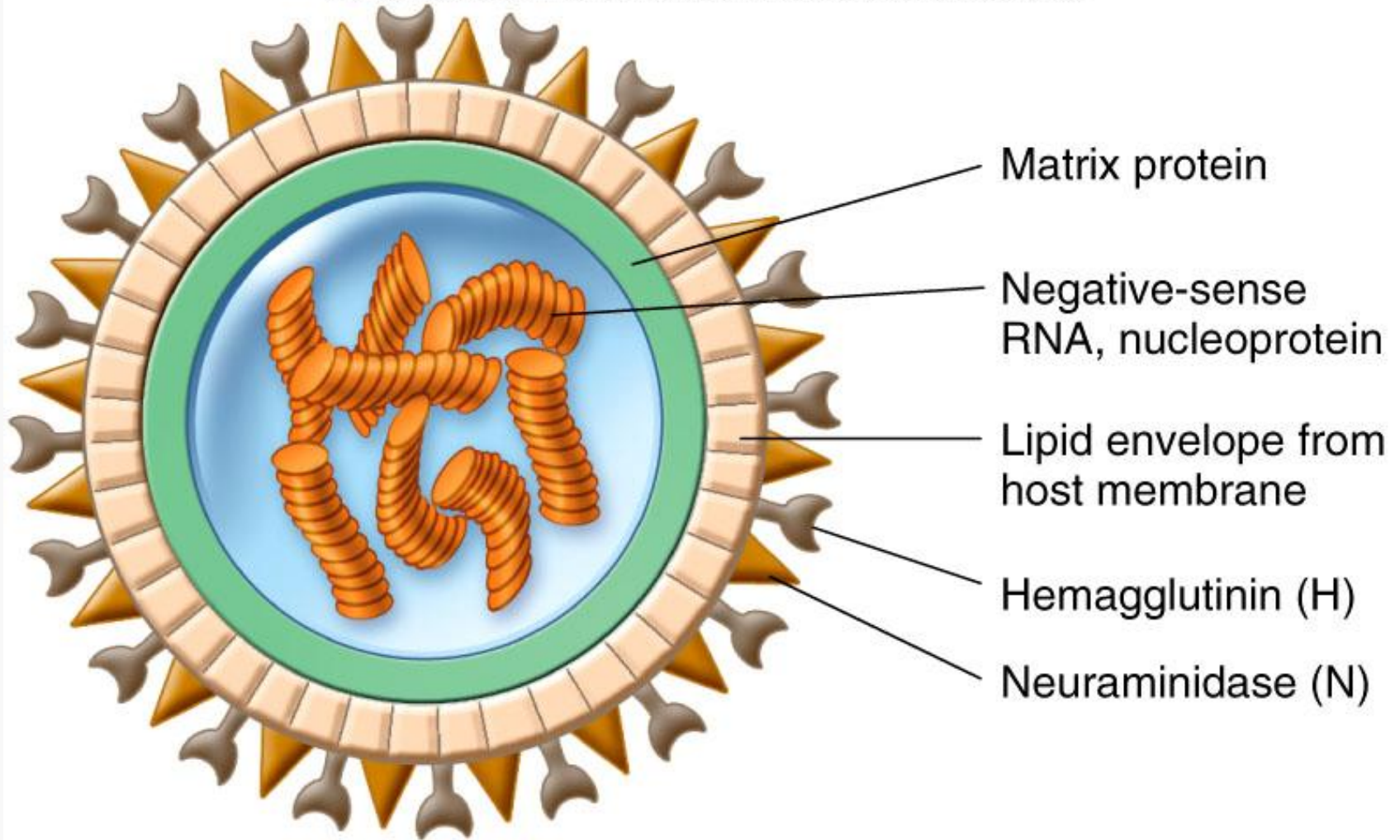


Fig. 21.11 Schematic drawing of influenza virus.

# 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.

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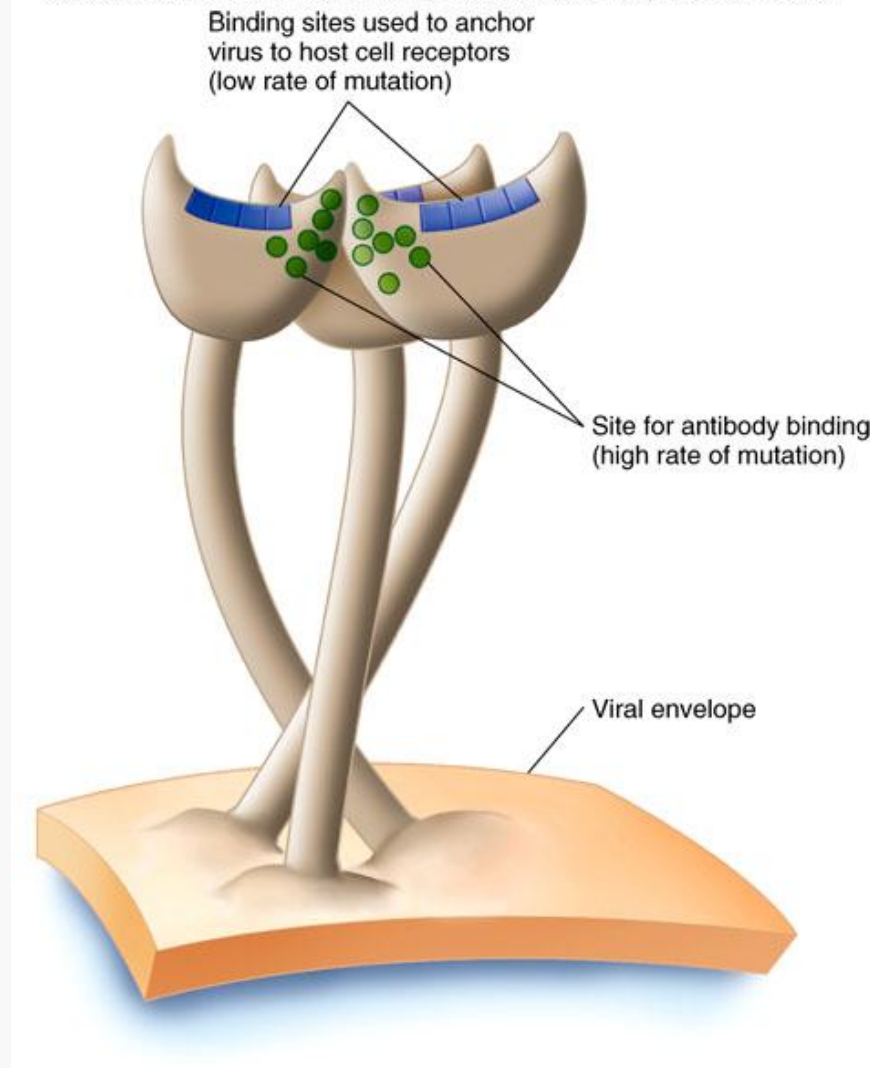


Fig. 21.12 Schematic drawing of hemagglutinin of influenza Virus.

# 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.

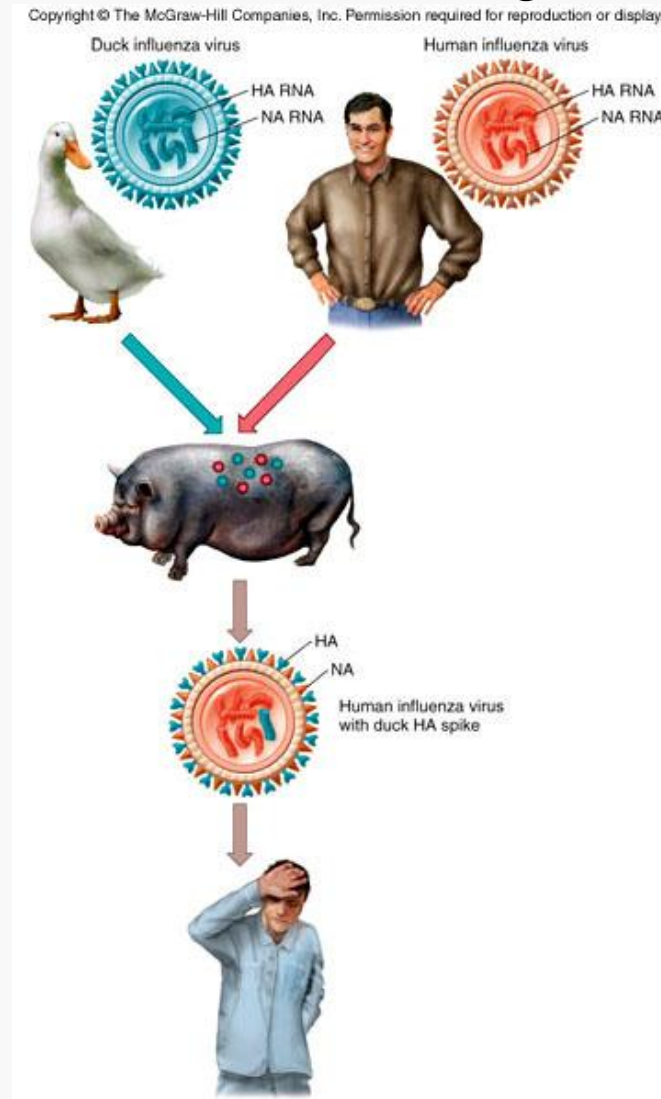


Fig. 21.13 Antigenic shift event.

# Features of influenza.

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## CHECKPOINT 21.8 Influenza

**Causative Organism(s)**

Influenza A, B, and C viruses

**Most Common Modes of Transmission**

Droplet contact, direct contact, some indirect contact

**Virulence Factors**

Glycoprotein spikes, overall ability to change genetically

**Culture/Diagnosis**

Viral culture (3–10 days) or rapid antigen-based tests

**Prevention**

Killed injected vaccine or inhaled live attenuated vaccine—taken annually

**Treatment**

Amantadine, rimantadine, zanamivir, or oseltamivir

# “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?