Streptococcus pneumoniae, *Haemophilus influenzae*, and *Moraxella catarrhalis*

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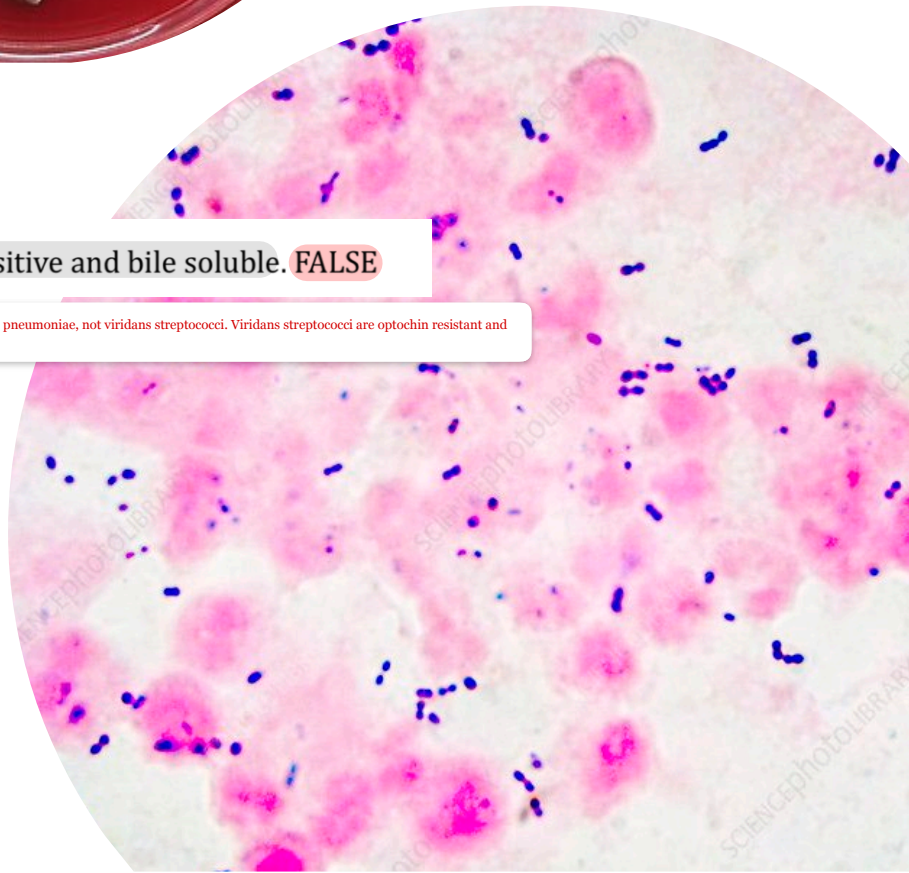
Overview of *Streptococcus pneumoniae*

- **Gram-positive**, lancet-shaped diplococci in pairs, sometimes short chains.
- α-hemolytic on blood agar (greenish halo).
- Optochin sensitive and bile soluble which are the classic identification tests.
- Facultative anaerobe; requires enriched media.
- Over 100 capsular serotypes. The **capsule** is the basis for serotyping and vaccine formulation.

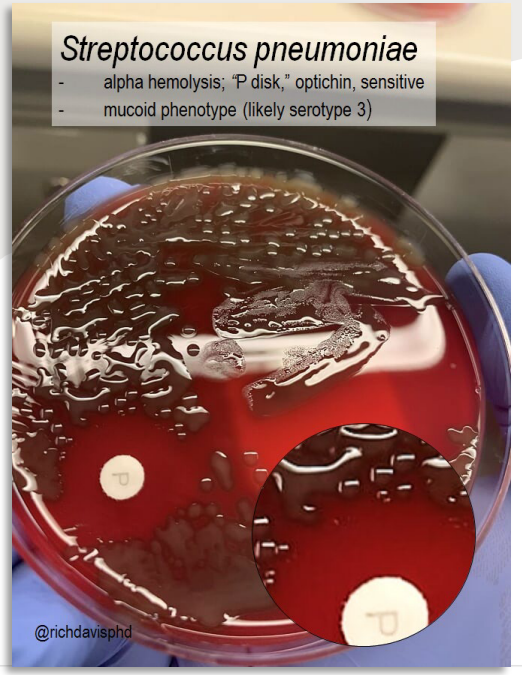


4. Viridans streptococci are optochin sensitive and bile soluble. FALSE

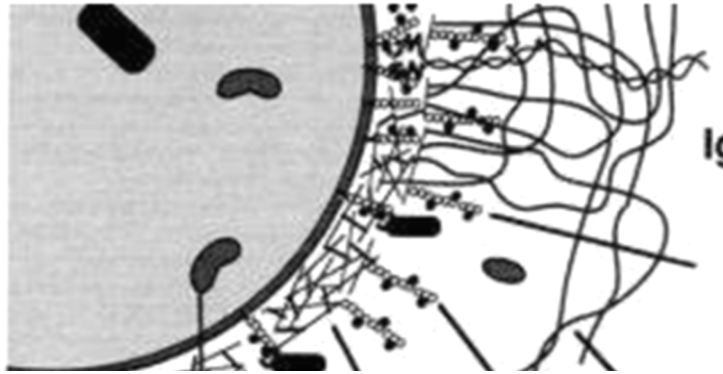
Optochin sensitivity and bile solubility are characteristic of Streptococcus pneumoniae, not viridans streptococci. Viridans streptococci are optochin resistant and not bile soluble.

1. Differences in pneumococcal serotype virulence are capsule-dependent. TRUE

S. pneumoniae virulence factors



- **Polysaccharide capsule:** Anti-phagocytic that inhibits opsonization and complement activation. Serotyping determines invasiveness and disease severity.
- **Pneumolysin:** Causes pores in host cell membranes. Damages respiratory epithelium and triggers inflammation.
- **Autolysin:** Causes bacterial self-lysis, releasing cell wall components that intensify inflammation.
- **IgA protease:** Allows evasion of mucosal IgA



6. Autolysin as a virulence factor for *Streptococcus pneumoniae* acting to evade neutralization by the mucosal IgA. FALSE

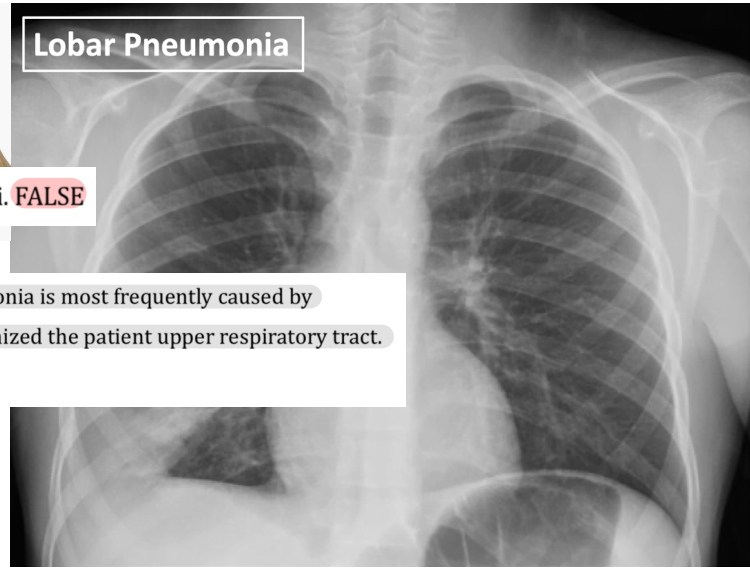
Pathogenesis and clinical manifestations of RTI

pneumococcal infections

- Pathogenesis: Nasopharyngeal colonization triggered by viral URTI, smoking, splenectomy, hypogammaglobulinemia).
- (Endogenous infection)
- Spread to alveoli resulting in intense neutrophilic exudate and **lobar pneumonia**.
- Middle ear → **acute otitis media**.
- Sinuses → **bacterial sinusitis**.
- Clinical features of pneumococcal pneumonia: abrupt fever, chest pain, **rusty sputum**, lobar consolidation on CXR.
- Sinusitis: severe headache, stuffy or runny nose, post-nasal drip.

3. The spleen is critical for defense against pneumococcal bacteremia. TRUE

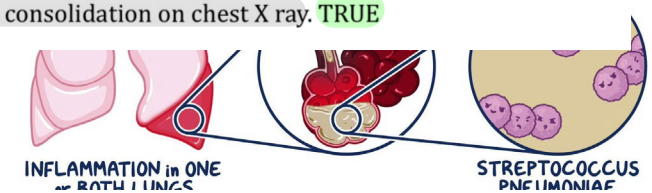
2. Pneumococcal disease usually begins with direct inhalation into the alveoli. FALSE
Spread to the alveoli occurs later, often after viral URTI or impaired host defenses.



20. Pneumococcal community-acquired pneumonia is most frequently caused by endogenous pneumococci that already colonized the patient upper respiratory tract. TRUE

PNEUMONIA

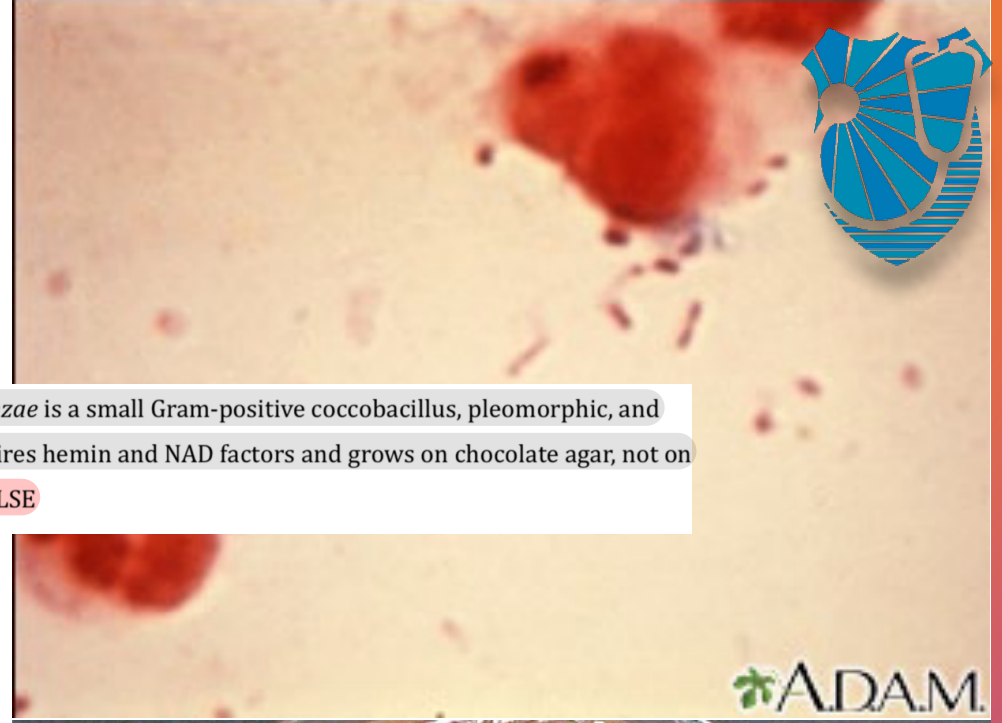
7. The classical clinical features of pneumococcal pneumonia include abrupt fever, chest pain, rusty sputum, and lobar consolidation on chest X ray. TRUE



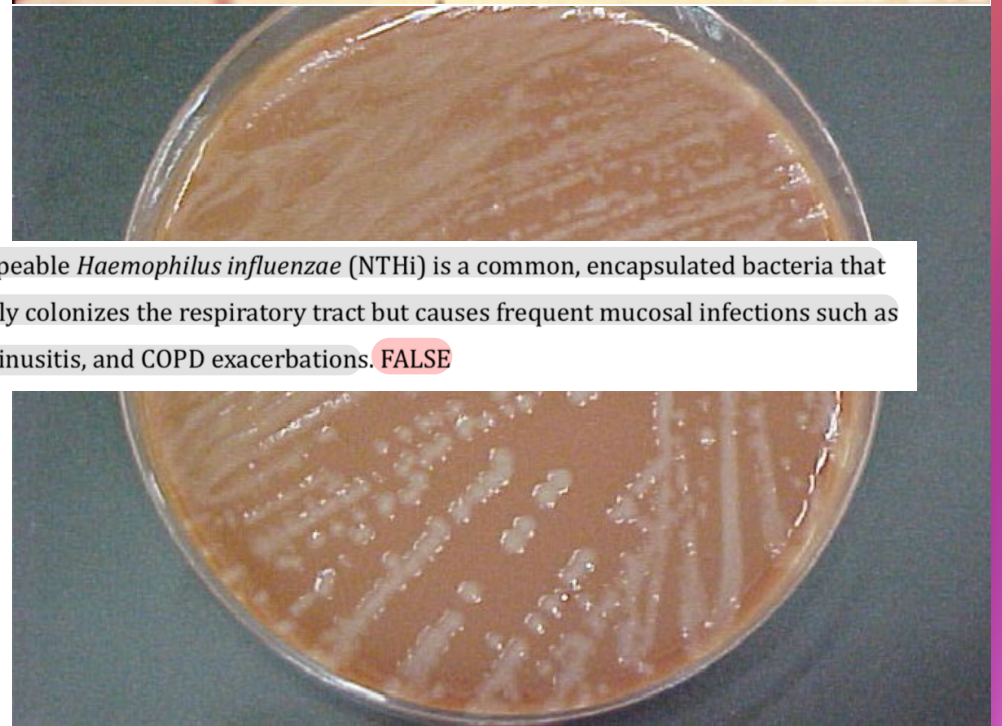
8. The classical clinical features of pneumococcal sinusitis include severe headache, stuffy or runny nose, and post-nasal drip. TRUE

Haemophilus influenzae overview

- Small **Gram-negative** coccobacillus, pleomorphic.
- Fastidious: requires X (hemin) and V (NAD) factors. Grows on chocolate agar, not on plain blood agar.
- Two major groups: Typeable (encapsulated): defined by capsule type (a-f), **Hib most important historically**.
- Non-typeable (NTHi): no capsule; frequent in mucosal respiratory disease.



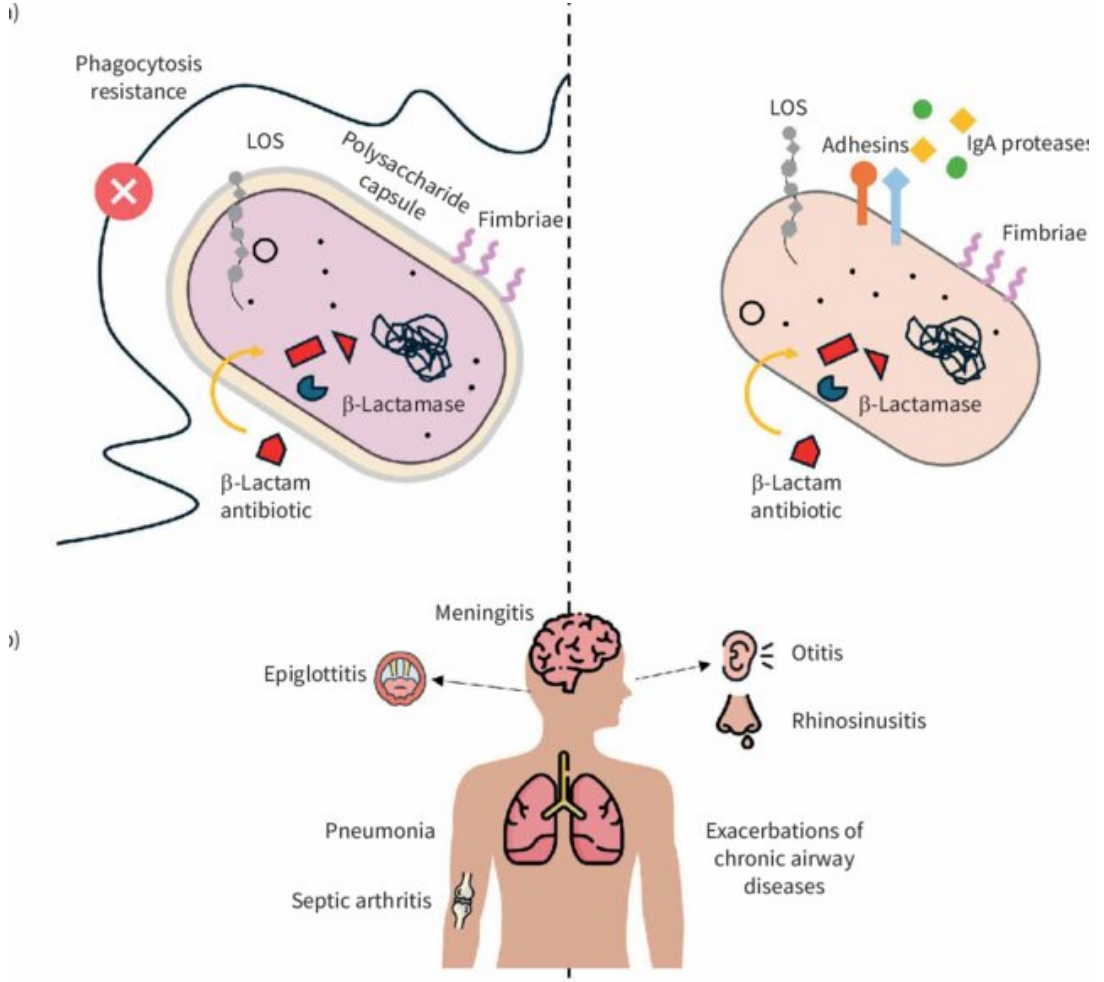
9. *Haemophilus influenzae* is a small Gram-positive coccobacillus, pleomorphic, and fastidious as it requires hemin and NAD factors and grows on chocolate agar, not on plain blood agar. FALSE



10. Non-typeable *Haemophilus influenzae* (NTHi) is a common, encapsulated bacteria that normally colonizes the respiratory tract but causes frequent mucosal infections such as otitis, sinusitis, and COPD exacerbations. FALSE

Virulence factors of *H. influenzae*

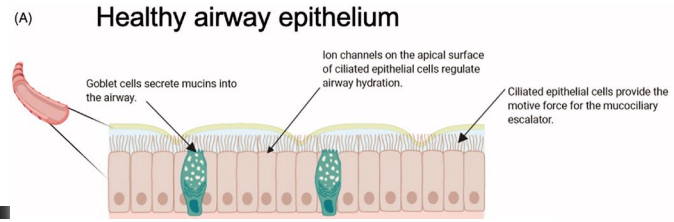
- **PRP capsule:** Anti-phagocytic; major factor in invasive disease (meningitis, epiglottitis).
- **LOS:** Endotoxin; damages ciliated epithelial cells, increasing adherence and invasion.
- **IgA protease:** Helps survive on mucosal surfaces by cleaving secretory IgA.
- **Adhesins:** Promote tight adherence to nasopharyngeal epithelium.
- **Biofilm formation:** Particularly with NTHi in chronic otitis media, sinusitis, and COPD airways.

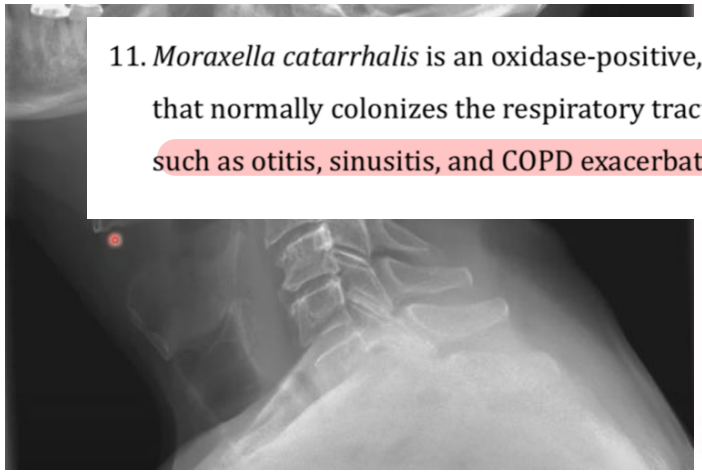


Source: De Angelis A, Marchello M, Tramontano A, Cicchetti M, Nigro M, Simonetta E, Scarano P, Polelli V, Artuso VA, Aliberti S. *Haemophilus influenzae* in bronchiectasis. Eur Respir Rev. 2025 Aug 6;34(177):250007.

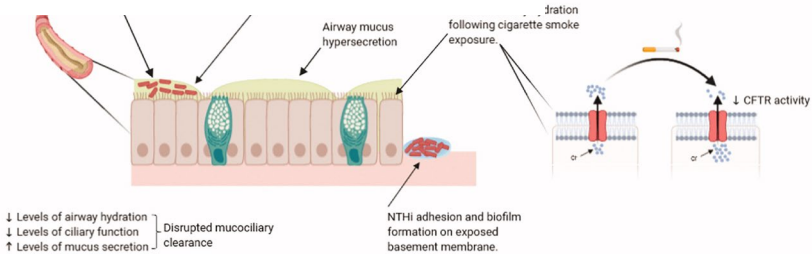
Pathogenesis and clinical manifestations of *H. influenzae*

- Non-typeable *H. influenzae* (NTHi): Common cause of sinusitis and acute otitis media in children.
- COPD exacerbations: chronic colonization of damaged bronchi.
- Hib (encapsulated): **Epiglottitis**. Host risk factors: incomplete vaccination, asplenia, complement deficiencies, chronic lung disease.





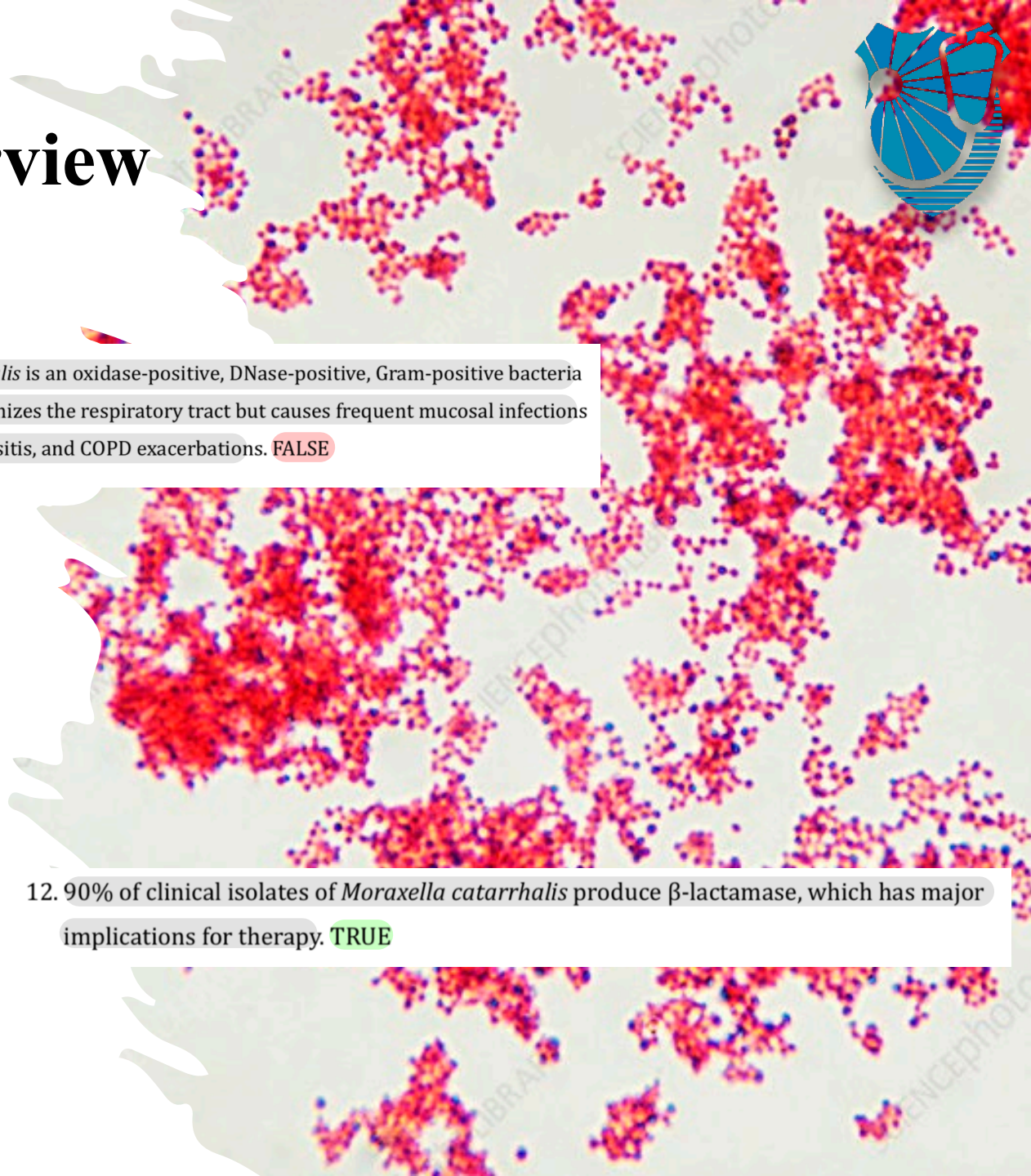
11. *Moraxella catarrhalis* is an oxidase-positive, DNase-positive, Gram-positive bacteria that normally colonizes the respiratory tract but causes frequent mucosal infections such as otitis, sinusitis, and COPD exacerbations. FALSE



Source:Short, B., Carson, S., Devlin, A. C., Reihill, J. A., Crilly, A., MacKay, W., … Martin, S. L. (2021). Non-typeable Haemophilus influenzae chronic colonization in chronic obstructive pulmonary disease (COPD). Critical Reviews in Microbiology, 47(2), 192–205.

Moraxella catarrhalis overview

- **Gram-negative** diplococcus (resembling *Neisseria*)
- **Oxidase-positive**, DNase-positive
- Commensal of URT, especially in young children; colonization decreases with age.
- 90% of clinical isolates produce β-lactamase, which has major implications for therapy.



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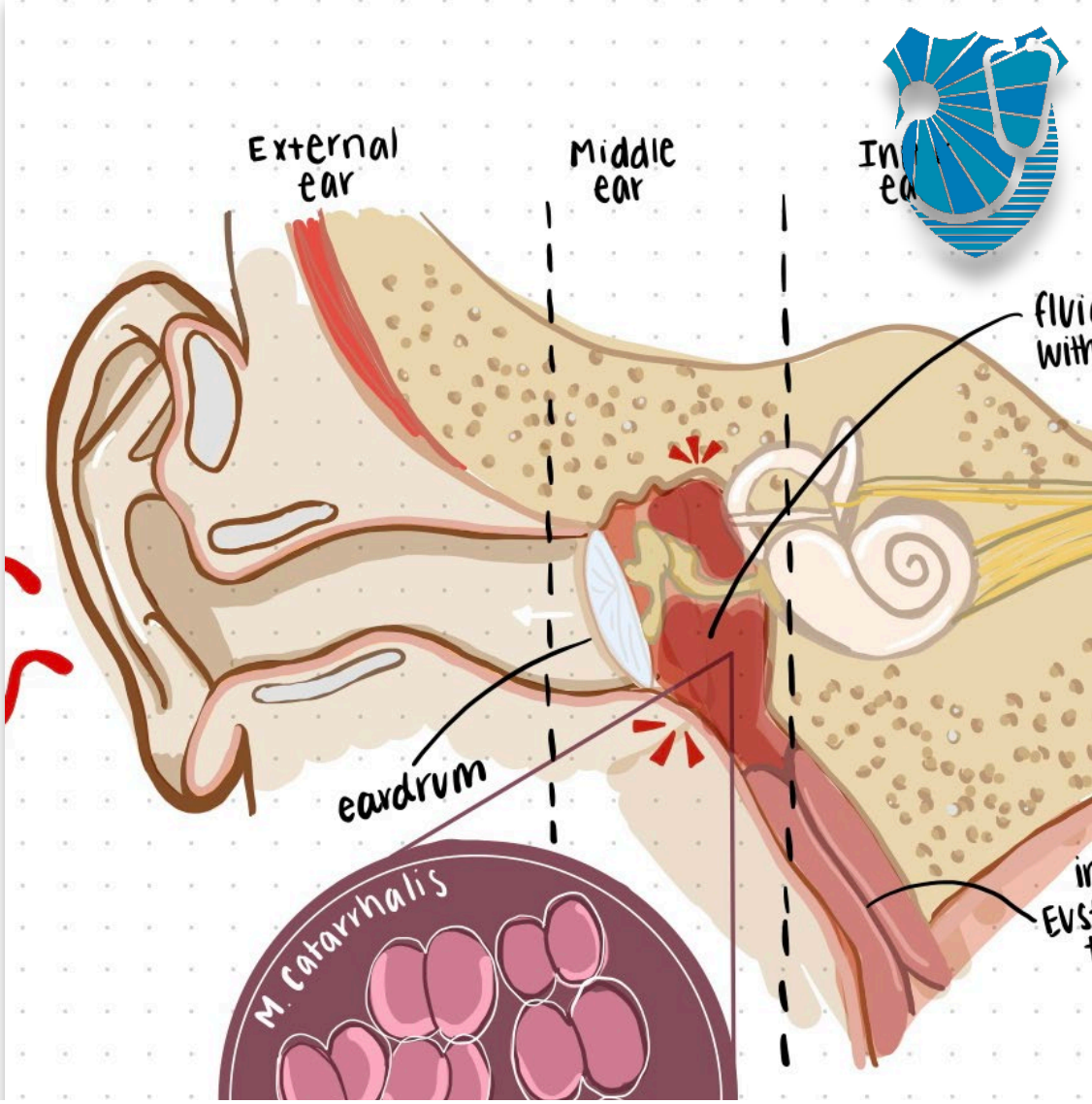
12. 90% of clinical isolates of *Moraxella catarrhalis* produce β-lactamase, which has major implications for therapy. TRUE

Virulence factors and clinical features of *M. catarrhalis*

- **Ubiquitous surface proteins (USP):** Mediate strong **adherence** to epithelial cells and extracellular matrix and mediate complement resistance.
- **Biofilms:** Important in chronic otitis media and sinusitis; shield against immune factors and antibiotics.
- **Endotoxin (LOS):** Stimulates inflammation, contributes to symptoms and tissue damage.
- Clinical outcome: primarily mucosal infections, rarely invasive disease (e.g., otitis media, sinusitis, bronchitis, COPD exacerbations, laryngitis).



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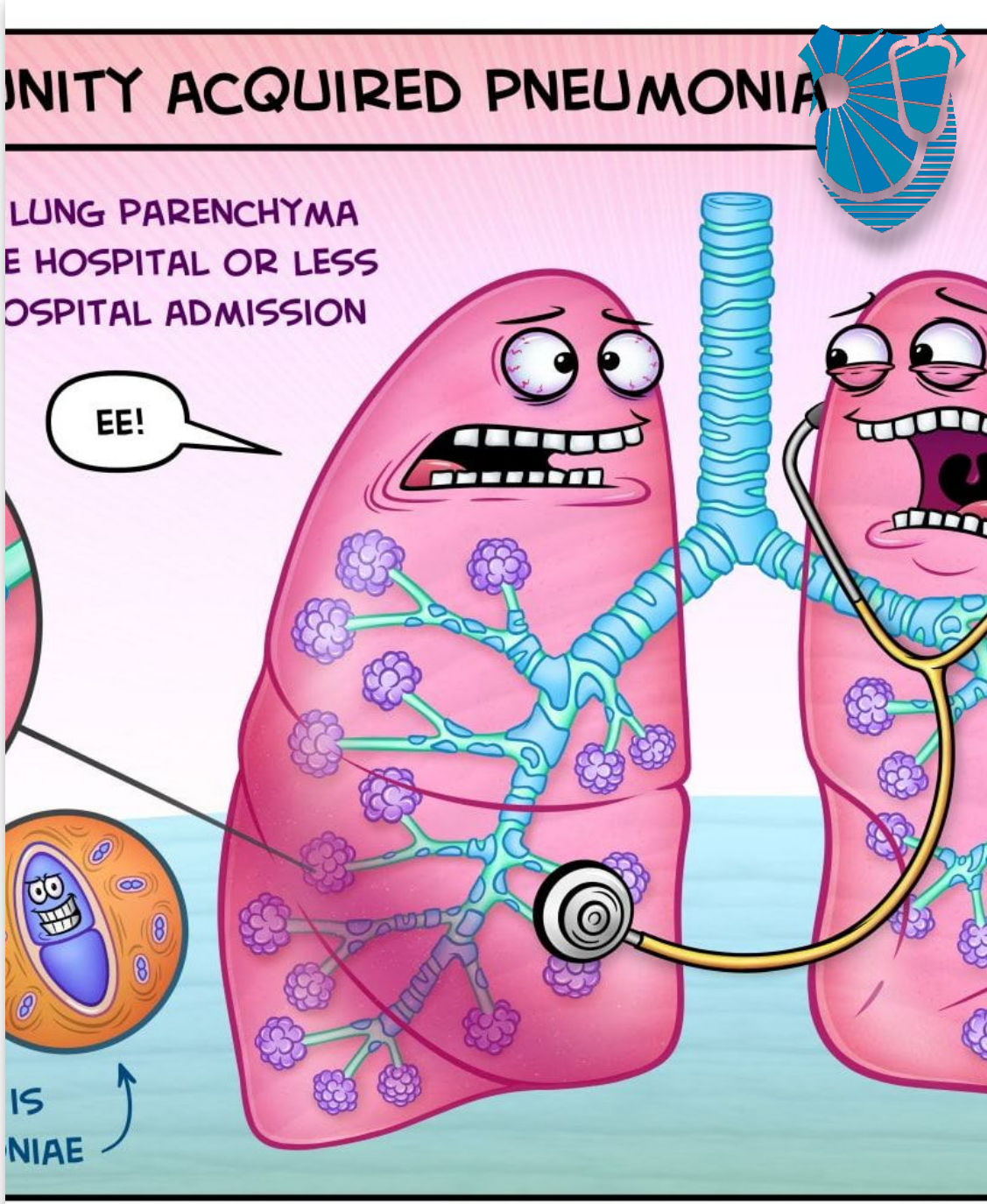
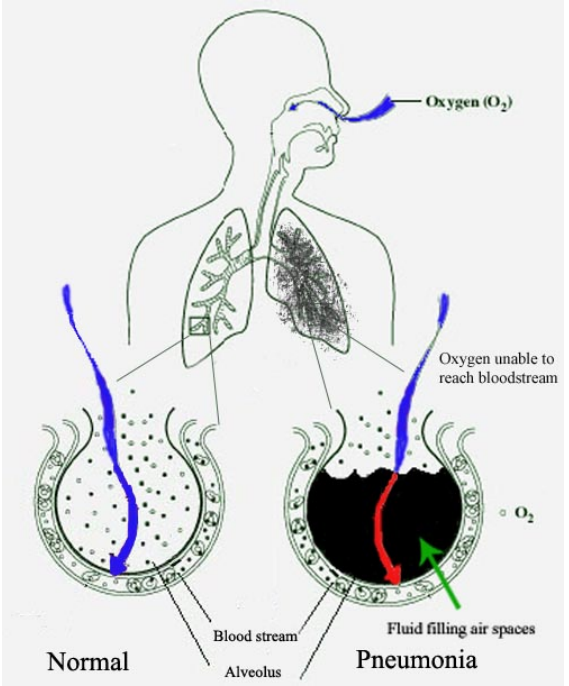
Clinical syndromes

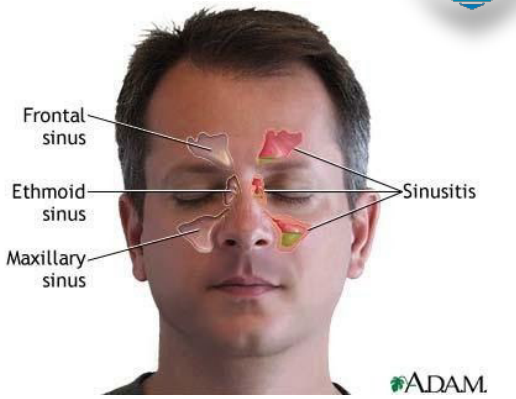
- Acute otitis media: caused by *S. pneumoniae*, NTHi, *M. catarrhalis*. Viral URTI → Eustachian tube dysfunction → middle ear effusion → secondary bacterial infection.
- Acute bacterial sinusitis: caused by *S. pneumoniae,* NTHi, and *M. catarrhalis*. Sinus ostial obstruction with impaired mucociliary clearance and bacterial overgrowth
- Community-acquired pneumonia (CAP): *S. pneumoniae* (lobar pneumonia). *H. influenzae* and *Moraxella* also implicated, especially in smokers/COPD.
- COPD exacerbations (cough, dyspnea, and increased sputum): Major three causes: NTHi, *M. catarrhalis*, and *S. pneumoniae*.
- Bronchitis and laryngitis: Mainly viral but *H. influenzae* and *M. catarrhalis* may become involved especially in patients with underlying chronic lung disease



Clinical diagnosis

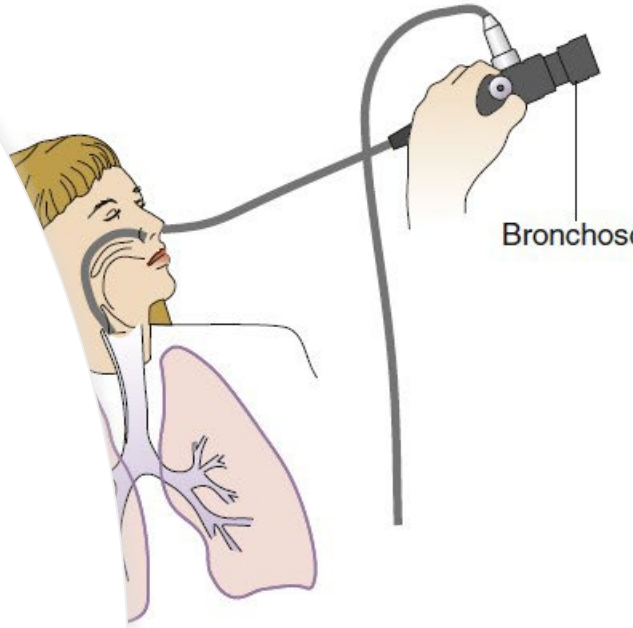
- AOM: Ear pain (otalgia), erythematous tympanic membrane, and fever.
- Bacterial sinusitis: Persistent symptoms without improvement, fever, purulent nasal discharge, and facial pain.
- Community-acquired pneumonia (CAP): Cough, fever, dyspnea, pleuritic chest pain (a sharp, stabbing pain in the chest that worsens with deep breaths, coughing, or sneezing, caused by inflammation of the pleura).

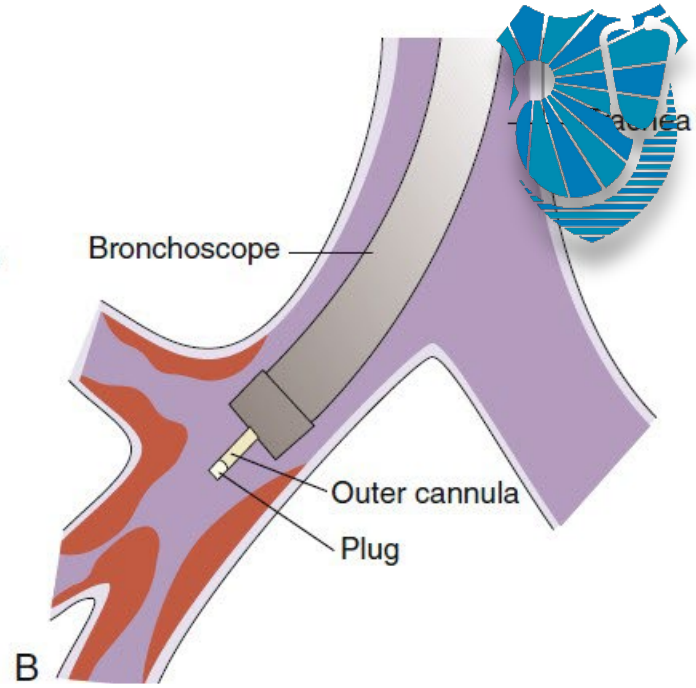


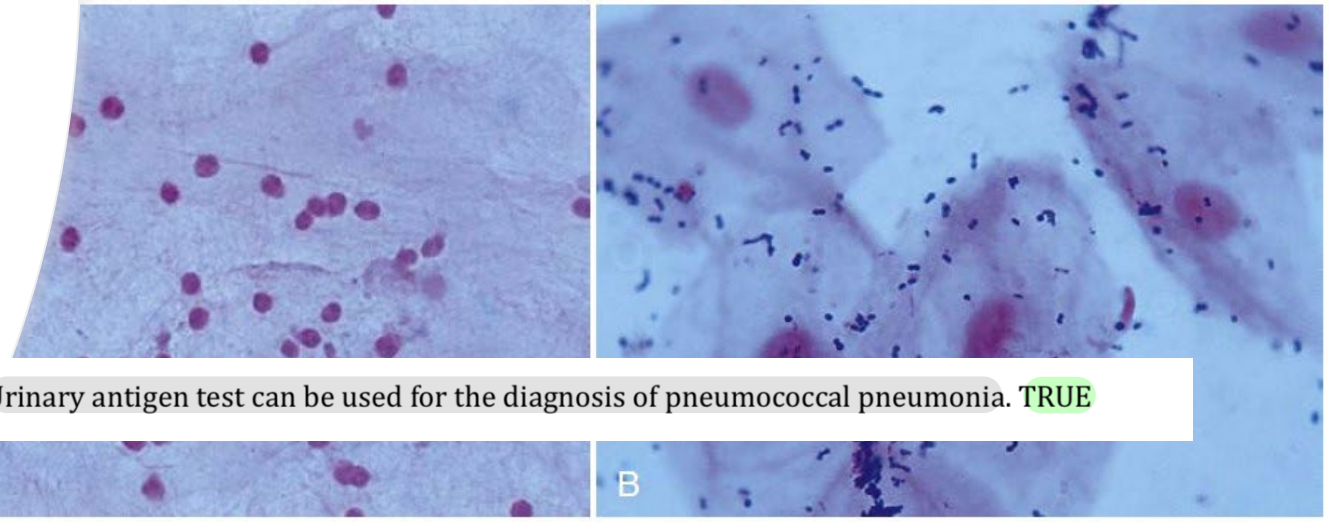


Lab diagnosis

- **Direct microscopy** (Gram stain) of sputum, BAL, middle ear or sinus aspirate:
- *S. pneumoniae*: Gram-positive lancet diplococci. *H. influenzae*: small Gram-negative coccobacilli. *M. catarrhalis*: Gram-negative diplococci.
- **Culture:**
- *S. pneumoniae*: α-hemolytic, optochin sensitive, bile soluble; *H. influenzae*: chocolate agar; *M. catarrhalis*: grows well on standard media; oxidase-positive, DNase-positive.
- **Antigen detection/molecular tests:** Urinary antigen test for pneumococcal polysaccharide in pneumonia. PCR for *H. influenzae* and *M. catarrhalis*.







13. Urinary antigen test can be used for the diagnosis of pneumococcal pneumonia. TRUE

B

14. Multiplex PCR can be used to identify the underlying cause of community-acquired pneumonia. TRUE

phonuclear leukocytes, indicating an inadequate specimen for routine sputum culture.

Antimicrobial resistance mechanisms

- *S. pneumoniae:* Altered PBPs → reduced affinity to penicillin and other β-lactams. Macrolide resistance via methylation of ribosomal RNA (*erm*) or efflux pumps (*mef*).
- *H. influenzae*: β-lactamase production (*TEM*, *ROB*) → ampicillin resistance. BLNAR strains (Beta-Lactamase-Nonproducing Ampicillin-Resistant) due to altered PBPs.
- *M. catarrhalis*: Most have β-lactamase activity, inactivating penicillin/ampicillin.

15. The management of pneumococcal community-acquired pneumonia is based on a high-dose amoxicillin. FALSE

Management of pneumococcal CAP is not based solely on high-dose amoxicillin. Treatment depends on disease severity, resistance patterns, comorbidities, and local epidemiology.

What is an ANTIBIOGRAM?

- Otitis media/sinusitis: First-line: high-dose amoxicillin (for pneumococcus). If β-lactamase producers likely (*H. influenzae, M. catarrhalis*) → amoxicillin-clavulanate.
- Community-acquired pneumonia (CAP) (non-severe, no comorbidities): Amoxicillin or amoxicillin-clavulanate ± macrolide depending on local resistance. In higher risk or hospitalized: ceftriaxone + macrolide, or fluoroquinolone (depending on guidelines).
- COPD exacerbations (with purulent sputum): Amoxicillin-clavulanate, doxycycline, or macrolide guided by local resistance patterns. COPD=Chronic obstructive pulmonary disease.

16. The management of community-acquired pneumonia is always based on the interpretation of the minimal inhibitory concentration, the local epidemiology of causative strains of bacteria, and the patient factors such as allergy, severity, and comorbidities. TRUE

- **You always interpret the minimal inhibitory concentration (MIC) + local epidemiology + patient factors (allergy, severity, comorbidities).**

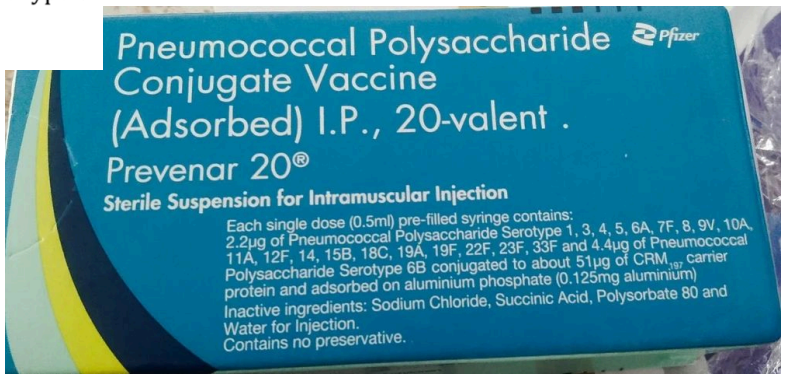
Prevention

- Conjugate Vaccines: Pneumococcus and Hib.
- Reduce carriage of vaccine serotypes and herd protection.
- Marked decline in invasive pneumococcal disease (bacteremia, meningitis).
- Nearly eliminated Hib meningitis and epiglottitis where coverage is high.
- No approved vaccine yet for *M. catarrhalis*, but its role in AOM and COPD makes it an attractive future target.

19. Pneumococcal conjugate vaccine is an immunizing agent used to prevent infection caused by all types of pneumococci and provide her immunity. FALSE

Pneumococcal conjugate vaccines protect against selected serotypes only, not all pneumococcal types.

18. The Hib vaccine protects against serious infections from *Haemophilus influenzae* type b preventing meningitis and epiglottitis. TRUE



17. Vaccination against *Streptococcus pneumoniae, Haemophilus influenzae,* and *Moraxella catarrhalis* are licensed to protect from encapsulated strains only. FALSE

There is no licensed vaccine for Moraxella catarrhalis, and pneumococcal vaccines do not cover all encapsulated strains.

Role of antibiotic stewardship (ASP)

- **Excess and inappropriate antibiotic use drives:**
  - Penicillin-non-susceptible pneumococci.
  - β-lactamase–producing *H. influenzae* and *M. catarrhalis*.
- **Key stewardship principles:**
  - Distinguish viral vs bacterial URTIs; avoid antibiotics in purely viral illnesses.
  - Use narrowest effective spectrum for shortest effective duration.
  - Vaccination and ASP are effective to reduce disease and resistance.

Thank You!
Wishing you all the best!