Air-Borne Bacterial Infections

Air-borne disease can spread when an infected person coughs, sneezes, or talks, ejecting nasal and throat secretions into the air. Certain viruses or bacteria take flight and hang in the air or land on other people or surfaces.

When you breathe airborne pathogenic organisms in, they take up residence inside you. You can also pick up germs when you touch an infected surface, and then touch your own eyes, nose, or mouth.

Because these diseases travel in the air, they're hard to control.

Types of airborne diseases: Many diseases are spread through the air, including these:

Tuberculosis (TB)

Mycobacteria

- More than 100 spp.: Medically important mycobacteria include:

1. Those that cause tuberculosis (referred as Mycobacterium tuberculosis complex –consisting of *M tuberculosis*, *M bovis*, *M africanum*, *M microti*, *M canettii*).

2. *M leprae* which cause leprosy or Hansen disease.

3. nontuberculous mycobacteria (NTM) or mycobacteria other than tuberculosis (MOTT) cause disease called Mycobacteriosis (those organism live in soil and water).

- long, slender rods that are nonmotile and do not form spores.
- cell walls are composed of 60 percent lipid, including a unique class of very long–chain (75 to 90 carbons), β-hydroxylated fatty acids (mycolic acids). These complex with a variety of polysaccharides and peptides, creating a waxy cell surface that makes mycobacteria strongly hydrophobic and accounts for their acid-fast staining characteristic. Mycobacteria are also resistant to drying but not to heat or ultraviolet irradiation.
- Some mycobacterium spp. grows on simple media, some are fastidious, and some (Leprae) do not grow at all.

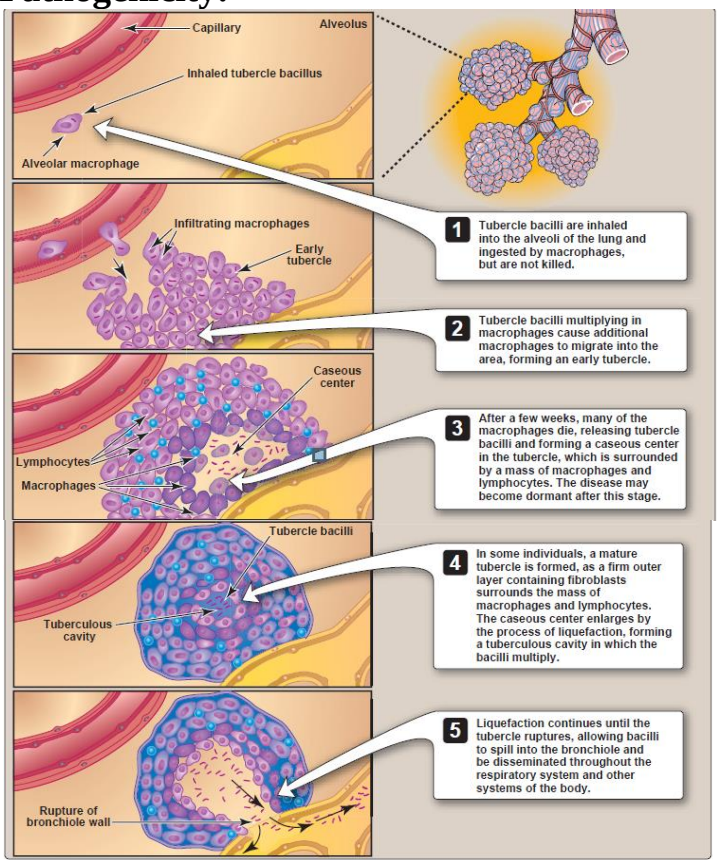
- Mycobacteria are strictly aerobic. Most species grow slowly with generation times of 8 to 24 hours.

Mycobacteria survive and replicate intracellularly. Mycobacterial infections generally result in the formation of slow-growing granulomatous lesions that are responsible for major tissue destruction.

A. **Mycobacterium tuberculosis**

- **Tuberculosis (Pulmonary, Tuberculous meningitis, Miliary tuberculosis, Renal and urogenital tuberculosis, Bone and joint tuberculosis)**
- It is currently estimated that about one third of the world's population is infected with M. tuberculosis (tubercle bacillus), with 30 million people having active disease.
- Patients with active pulmonary tuberculosis shed large numbers of organisms by coughing, creating aerosol droplet nuclei. Because of resistance to desiccation, the organisms can remain viable as droplet nuclei suspended in room air for at least 30 minutes.
- The principal mode of transmission from person-to-person is by inhalation of the aerosol. A single infected person can pass the organism to numerous people in an exposed group, such as a family, classroom, or hospital ward without proper isolation.

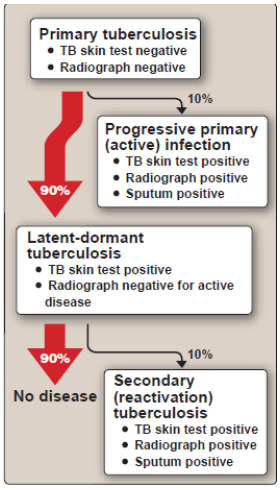
Pathogenicity:

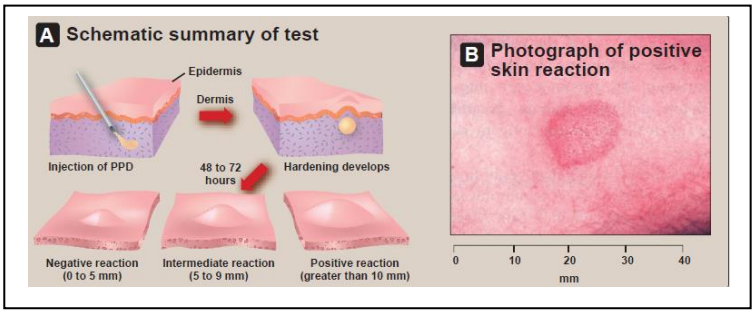


The virulence of M. tuberculosis rests with its ability to survive and grow within host cells. Although the organism produces no demonstrable toxins, when engulfed by macrophages, bacterial sulfolipids inhibit the fusion of phagocytic vesicles with lysosomes.

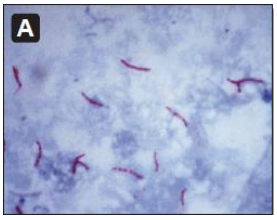
Immunity: M. tuberculosis stimulates both a humoral and a cell mediated immune response. Although circulating antibodies appear, they do not convey resistance to the organism. Instead, cellular immunity (CD4+ T cells) and the accompanying delayed hypersensitivity directed against a number of bacterial protein antigens, develop in the course of infection and contribute to both the pathology of and immunity to the disease.

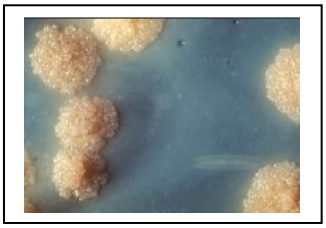
Clinical significance: Primary tuberculosis occurs in a person who has had no previous contact with the organism. For the majority of cases (90-95 percent), the infection becomes arrested, and most people are unaware of this initial encounter. The only evidence of tuberculosis may be a positive tuberculin test. Approximately 10 percent of those with an arrested primary infection develop clinical tuberculosis at some later time in their lives.





Laboratory identification: Diagnosis of active pulmonary tuberculosis includes demonstration of clinical symptoms and abnormal chest radiographs and confirmation by isolation of M. tuberculosis from relevant clinical material.

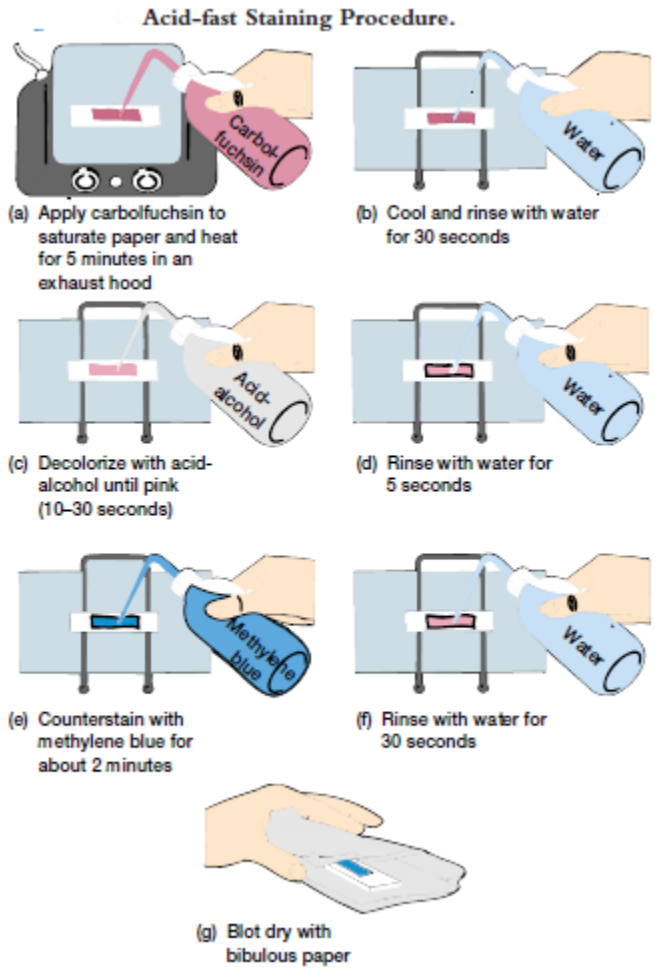




Specimens:
Sputum, not saliva is required to detect AFB. Examination of up to three specimens (at least one as an early morning specimen) may be required to detect the organisms.
In AIDS patients, it is sometimes possible to detect AFB in buffy coat smears prepared from EDTA anticoagulated blood. Cerebrospinal fluid is required to investigate tuberculous meningitis.

Caution: M. tuberculosis is a highly infectious pathogen, therefore handle specimens with care. It is particularly important to minimize the creation of aerosols and to ensure the laboratory is well ventilated. The use of personal respirators should be considered to protect staff working with M. tuberculosis.
Use a hooded Bunsen burner when flaming wire loops. Smears however, can be more easily made using a wooden stick which can be easily discarded and incinerated after use.

AFS:
Concentrating AFB by bleach treating sputum followed by centrifugation, significantly increases the sensitivity of direct microscopy in detecting AFB in sputum. Bleach treated sputum cannot be used for culture. To be detected microscopically, sputum smears need to contain 5000–10 000 AFB/ml which may be found in samples from patients with cavity lesions.
The cell wall lipids make the cell surface hydrophobic, rendering mycobacteria resistant to staining with basic aniline dyes unless they are applied with heat or detergents, or for prolonged periods of time. Once stained, however, mycobacteria resist decolorization with a mixture of 3% hydrochloric acid and 95% ethanol. These properties are described as **acid fastness** or, more properly, acid–alcohol fastness



Growth requirements

Specimens such as sputum, urine, and pus which contain commensals, require decontamination before being cultured for *M. tuberculosis*. For routine purposes, sodium hydroxide, 40 g/l has been found to be an effective decontaminant.

M. tuberculosis will grow aerobically on a protein enriched medium, e.g. Lowenstein Jensen egg medium. The optimal temperature for growth is 35–37 ºC. The organism is slow-growing.

When cultured on Lowenstein Jensen medium at 35–37 ºC, *M. tuberculosis* produces raised, dry, cream (buff) coloured colonies. Visible colonies are usually produced 2–3 weeks after incubation, but
cultures should be incubated for up to 6 weeks before being discarded.

WHO recommendations for chemotherapy of tuberculosis

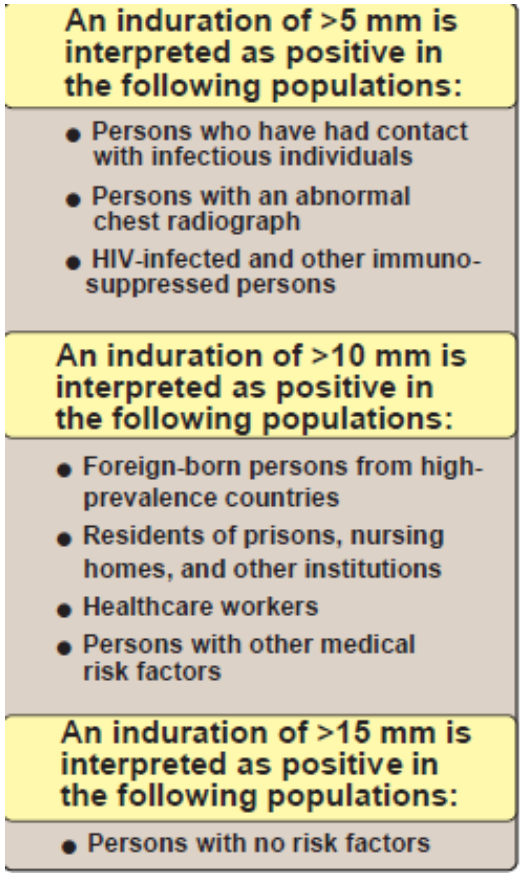
Short course antituberculosis therapy recommended by WHO consists of an initial 2 month intensive treatment with rifampicin, isoniazid and pyrazinamide. Ethambutol or streptomycin is added if resistance to one of the former drugs is common in a given region or if twice or thrice weekly therapy is indicated. This phase is followed by a 4 month continuation

phase of rifampicin and isoniazid. WHO recommends the use of directly observed therapy short course (DOTS) to ensure complete cure and prevent multi-drug resistance.

Prevention:

At present, the bacillus Calmette-Guérin (BCG) vaccine (named for its originators, Calmette and Guérin) is the only available vaccine. It has been used for prophylaxis of tuberculosis in various countries since 1923; administration is usually intradermal. It is a **live** vaccine derived originally from a strain of ***M. bovis*** that was **attenuated by repeated subculture**.
This vaccine, according to most studies, **decreased the highly lethal miliary and meningeal forms of tuberculosis among young children.**

Tuberculin reaction: The tuberculin reaction test is a manifestation of delayed hypersensitivity to protein antigens of M. tuberculosis.
Although such tests can be used to document contact with the tubercle bacillus, they do not confirm that the patient currently has active disease. In the Mantoux test, purified protein derivative (PPD) is prepared from culture filtrates of the organism.
A positive reaction usually develops 4 to 6 weeks after initial contact with the organism. It remains positive for life, although it may wane after some years or in the presence of immunosuppression by medications or disease.



Diphtheria

CORYNEBACTERIA

 small, slender, pleomorphic, gram-positive rods. They are nonmotile and unencapsulated, and they do not form spores.

 Corynebacterium is a large genus of diverse habitat. Most species are facultative anaerobes, and those associated with humans, including the pathogen *C. diphtheriae*, grow aerobically on standard laboratory media such as blood agar.

 C. diphtheriae is found in the throat and nasopharynx of carriers and in patients with diphtheria.

 Clinical significance: Infection may result in one of two forms of clinical disease, respiratory or cutaneous, or in an asymptomatic carrier state, (a. Upper respiratory tract infection, b. Cutaneous diphtheria)

 Diphtheria is a local infection, of the throat, and the organism is primarily spread by respiratory droplets, usually by convalescent or asymptomatic carriers.

 Diphtheria is caused by the local and systemic effects of a single exotoxin that inhibits eukaryotic protein synthesis. The toxin molecule is a heat-labile polypeptide that is composed of two fragments, A and B. Fragment B binds to susceptible cell membranes and mediates the delivery of fragment A to its target.

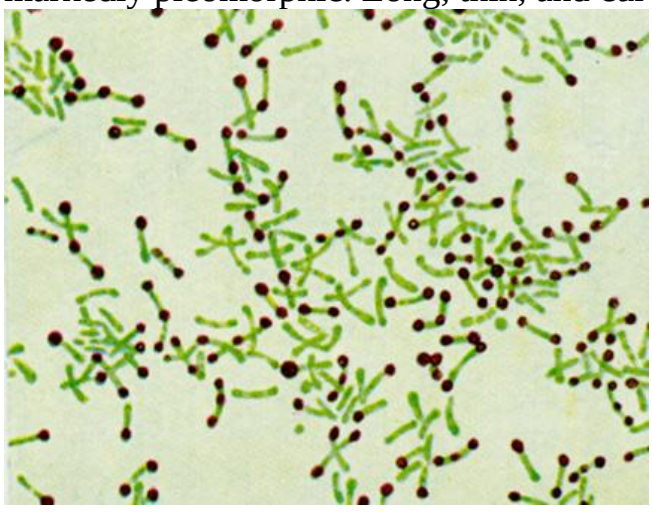
The infection produces a distinctive thick, grayish, adherent exudate (pseudomembrane) that is composed of cell debris from the mucosa and inflammatory products. It coats the throat and may extend into the nasal passages or downward in the respiratory tract, where the exudate sometimes obstructs the airways, even leading to suffocation. As the disease progresses, generalized symptoms occur caused by production and absorption of toxin. Although all human cells are sensitive to diphtheria toxin, the major clinical effects involve the heart and peripheral nerves.
Cutaneous (skin) diphtheria which usually develops when *C. diphtheriae* infects open wounds. Infection of the skin rarely leads to the serious complications associated with diphtheria of the throat.

LABORATORY FEATURES

Specimens: Include throat, and, or nasopharyngeal swabs to confirm a diagnosis of throat diphtheria, and a skin swab if cutaneous diphtheria is suspected

Morphology

diphtheriae is Gram positive but usually stains unevenly and weakly. It is markedly pleomorphic. Long, thin, and curved forms can be seen and



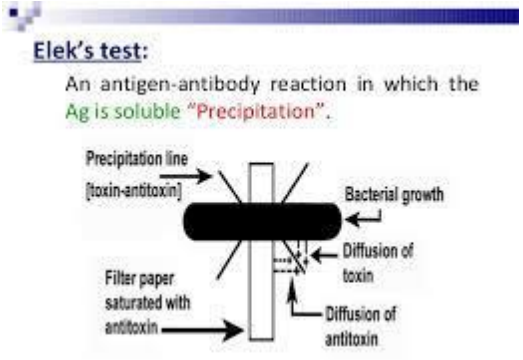
also short rods and rods enlarged at one end (club shaped). They often appear in clusters, joined at angles like Chinese letters Commensal diphtheroids: These are strongly Gram positive and stain uniformly. They are usually short and show little variation in size and form.

Culture

***Tellurite blood agar*:** This medium is widely used as a primary medium for isolating *C. diphtheria* from throat and nasopharyngeal swabs. *C. diphtheriae* reduces tellurite and produces grey or grey-black colonies measuring 0.5–2 mm in diameter after 24–48 h incubation

Toxigenicity (virulence) testing of *C. diphtheriae*

Diphtheria is caused by toxin-producing strains of *C. diphtheriae*. Toxigenicity of *C. diphtheriae* can be tested by the Elek gel precipitation test.



Neisseria spp.

The genus Neisseria consists of gram-negative, aerobic cocci.

Two Neisseria species are pathogenic for humans
Neisseria gonorrhoeae (commonly called gonococcus), the causal agent of gonorrhea and Neisseria meningitidis (commonly called meningococcus), a frequent cause of meningitis.

Gonococci and meningococci are nonmotile diplococcic that cannot be distinguished from each other under the microscope but can be differentiated in the laboratory by sugar-use patterns, and the sites of their primary infections.

Both bacteria are classified as **pyogenic cocci** because infections by these organisms are also characterized by the production of purulent (puslike) material comprised largely of white blood cells.

N. meningitidis: referred to as Meningococcus

Structure

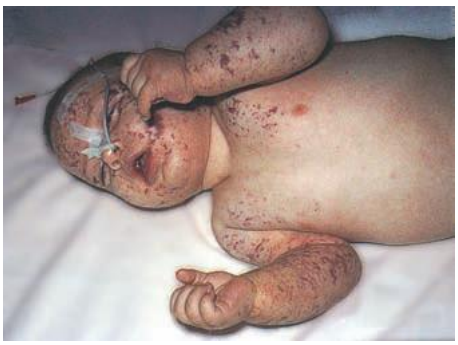
N. meningitidis is a nonmotile, gram-negative diplococcus, shaped like a kidney bean, which always appears in pairs. It is also piliated and the pili allow attachment of the organism to the ***nasopharyngeal mucosa*** where it is harbored both in carriers and in those with meningococcal disease. When meningococcus is isolated from blood or spinal fluid, it is **encapsulated**. The meningococcal polysaccharide capsule is antiphagocytic and, therefore, the most important virulence factor.

Transmission occurs through **inhalation of respiratory droplets** from a carrier or a patient in the early stages of the disease.

Risk factors for disease include recent viral or mycoplasma upper respiratory tract infection, active or passive smoking, and complement deficiency. In susceptible persons, pathogenic strains may invade the bloodstream and cause systemic illness after an incubation period of 2 to 10 days

Pathogenesis

Antiphagocytic properties of capsule aid in the maintenance of infection. LOS is responsible for the toxic effects found in disseminated meningococcal disease. IgA protease cleaves IgA1 helps the pathogens to evade immunoglobulins of this subclass.



Petechial and/or purpuric rash and neck extension characteristic of meningococcal meningitis.

Causes of meningitis (<u>2-18</u> y major cause N.meningitidis , S. pneumonia, H influenza), frequent cause of <u>neonatal meningitis</u> are S. agalactiae, L. monocytogenes, E. coli. Viral meningitis is caused by enteroviruses, herpes simplex viruses.

Laboratory identification

Sedimented CSF and skin lesion aspirates (gram-negative diplococcic association with polymorphonuclear leukocytes.

Carriers can be detected by culturing swabs from the nasopharyngeal region.

Culture: on chocolate agar (if sample taken from blood and CSF which they are sterile) with increased CO2.
Note: Thayer-Martin medium is required for samples obtained from a skin lesion or nasopharyngeal swab, to eliminate contaminating organisms.

oxidase-positive.
sugar utilization tests: N. meningitidis utilizes both glucose and maltose,

CSF elevated protein, decreased glucose (partly resulting from its consumption as a bacterial nutrient), and many neutrophils.
The presence of an infecting organism or of antigenic capsular substance confirms the diagnosis.

Prevention

A conjugate meningococcal vaccine **is available**

Pharyngitis:

A variety of bacteria can cause infection in the pharynx. A classic infection is strep throat. Caused by Streptococcus pyogenes, Contains M proteins which inhibits phagocytosis, Produces pyrogenic toxins which cause the symptoms seen with pharyngitis
Group A streptococci can cause abscesses on the tonsils.
S. pyogenes can cause scarlet fever and toxic shock syndrome.

Scarlet fever:

Caused by Group A streptococci. Usually seen in children under age of 18 years, Symptoms usually begin with appearance of a rash.
Symptoms can also include: sore throat with yellow or white papules

Streptococcus pyogenes (**Group A streptococcus**) is a Gram-positive, nonmotile, nonsporeforming coccus that occurs in chains or in pairs of cells. Individual cells are round-to-ovoid cocci, 0.6-1.0 micrometer in diameter (Figure 1). Streptococci divide in one plane and thus occur in pairs or (especially in liquid media or clinical material) in chains of varying lengths. The metabolism of *S. pyogenes* is fermentative; the organism is a catalase-negative aerotolerant anaerobe (facultative anaerobe), and requires enriched medium containing blood in order to grow. Group A streptococci typically have a capsule composed of hyaluronic acid and exhibit beta (clear) hemolysis on blood agar.

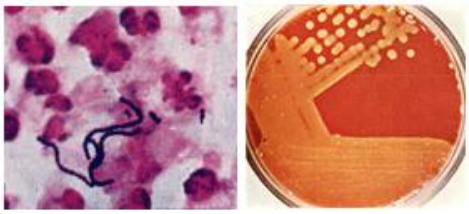


Figure 1. *Streptococcus pyogenes.* Left. Gram stain of *Streptococcus pyogenes* in a clinical specimen. Right. Colonies of *Streptococcus pyogenes* on blood agar exhibiting beta (clear) hemolysis.

Streptococcus pyogenes is one of the most frequent pathogens of humans. It is estimated that between 5-15% of normal individuals harbor the bacterium, usually in the respiratory tract, without signs of disease. As normal flora, *S. pyogenes* can infect when defenses are compromised or when the organisms are able to penetrate the constitutive defenses. When the bacteria are introduced or transmitted to vulnerable tissues, a variety of types of **suppurative infections** can occur.

Today, the pathogen is of major concern because of the occasional cases of rapidly progressive disease and because of the small risk of serious sequelae in untreated infections. These diseases remain a major worldwide health concern, and effort is being directed toward clarifying the risk and mechanisms of these sequelae and identifying rheumatogenic and nephritogenic strains of streptococci.

Acute *Streptococcus pyogenes* infections may present as **pharyngitis** (**strep throat**), **scarlet fever** (rash), **impetigo** (infection of the superficial layers of the skin) or **cellulitis** (infection of the deep layers of the skin). Invasive, toxigenic infections can result in **necrotizing fasciitis**, **myositis** and **streptococcal toxic shock syndrome**. Patients may also develop immune-mediated **post-streptococcal sequelae**, such as acute **rheumatic fever** and acute **glomerulonephritis**, following acute infections caused by *Streptococcus pyogenes.*

Streptococcus pyogenes produces a wide array of **virulence factors** and a very large number of diseases. Virulence factors of Group A streptococci include: (1) **M protein**, fibronectin-binding protein (**Protein F**) and **lipoteichoic acid** for adherence; (2) **hyaluronic acid capsule** as an immunological disguise and to inhibit phagocytosis; **M-protein** to inhibit phagocytosis (3) **invasins** suchas **streptokinase**, **streptodornase** (DNase B), **hyaluronidase**, and **streptolysins**; (4) exotoxins, such as **pyrogenic (erythrogenic) toxin** which causes the rash of **scarlet fever** and systemic**toxic shock syndrome**.

Classification of Streptococci

Hemolysis on blood agar

Beta -hemolysis is associated with complete lysis of red cells surrounding the colony

Alpha-hemolysis is a partial or "green" hemolysis associated with reduction of red cell hemoglobin.

Nonhemolytic colonies have been termed gamma-hemolytic.

Hemolysis is affected by the species and age of red cells, as well as by other properties of the base medium. **Group A streptococci are nearly always beta-hemolytic**; related Group B can manifest alpha, beta or gamma hemolysis. Most strains of *S. pneumoniae* are alpha-hemolytic but can cause ß-hemolysis during anaerobic incubation. Most of the oral streptococci and enterococci are non hemolytic. The property of hemolysis is not very reliable for the absolute identification of streptococci, but it is widely used in rapid screens for identification of *S. pyogenes* and *S. pneumoniae*.

Antigenic types

The cell surface structure of Group A streptococci is among the most studied of any bacteria (Figure 2). The cell wall is composed of repeating units of N-acetylglucosamine and N-acetylmuramic acid, the standard peptidoglycan. Historically, the definitive identification of streptococci has rested on the serologic reactivity of "cell wall" polysaccharide antigens as originally described by Rebecca Lancefield. **Eighteen group-specific antigens (Lancefield groups) were established**. The Group A polysaccharide is a polymer of N-acetylglucosamine and rhamnose. Some group antigens are shared by more than one species. This polysaccharide is also called the **C substance** or **group carbohydrate antigen**.

Pathogenesis

Streptococcus pyogenes owes its major success as a pathogen to its ability to colonize and rapidly multiply and spread in its host while evading phagocytosis and confusing the immune system.

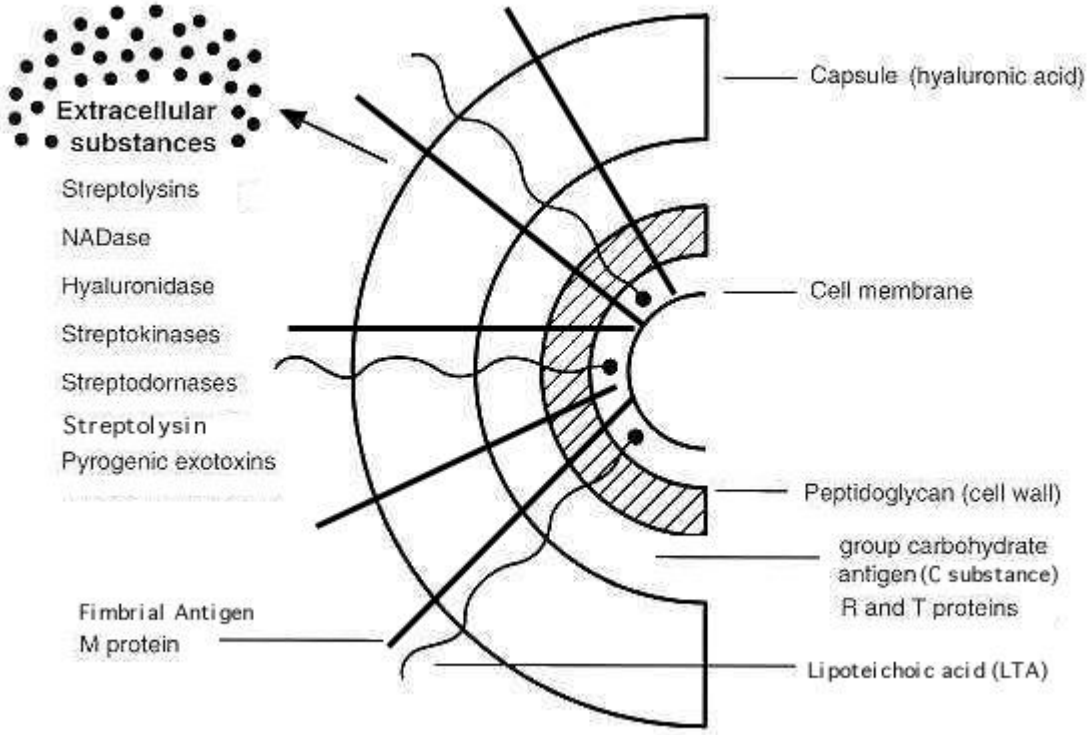


Figure 2. Cell surface structure of *Streptococcus pyogenes* and secreted products involved in virulence.

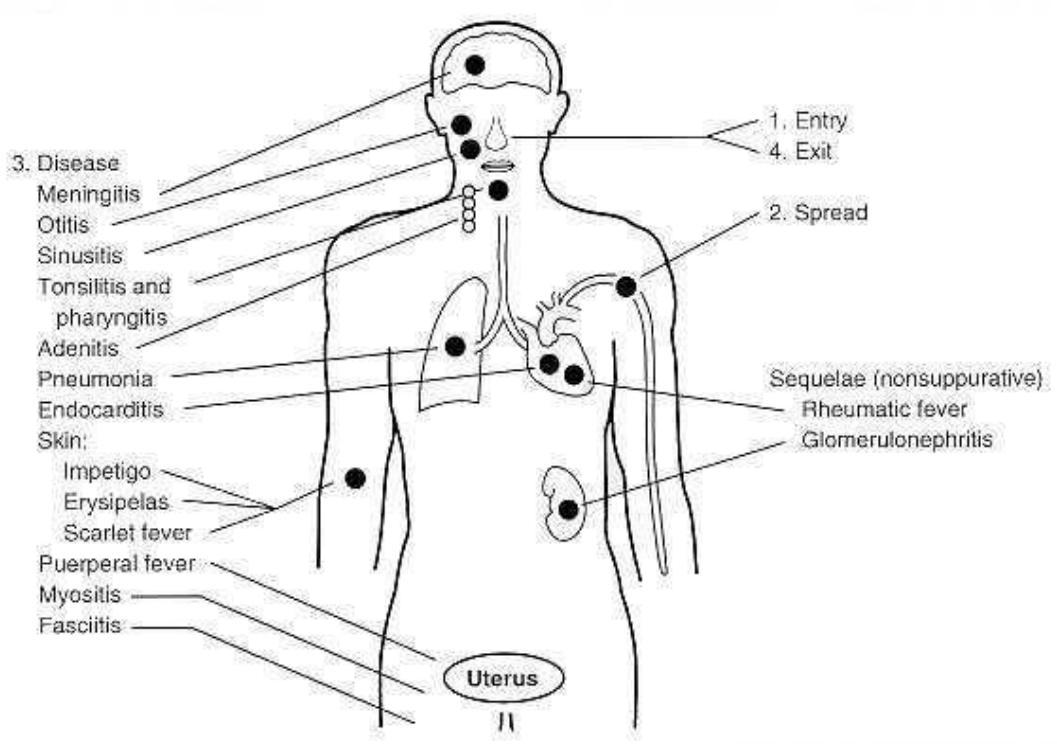


FIGURE 3. Pathogenesis of *Streptococcus pyogenes* infections. Adapted from Baron's Medical Microbiology Chapter 13, Streptococcus by Maria Jevitz Patterson.

S. pneumoniae

Streptococcus pneumoniae (**pneumococcus)** is a normal inhabitant of the human URT, can cause pneumonia (usually of the lobar type), paranasal sinusitis and otitis media, or meningitis. It also causes osteomyelitis, septic arthritis, endocarditis, peritonitis, cellulitis and brain abscesses.
Streptococcus pneumoniae cells are Gram-positive, lancet-shaped cocci. Usually, they are seen as pairs of cocci (diplococci), but they may also occur singly and in short chains. aerotolerant anaerobe, alpha hemolytic. non spore former, and nonmotile. Like other streptococci, they lack catalase and ferment glucose to lactic acid. Unlike other streptococci, they do not display an M protein, and their cell wall composition is characteristic both in terms of their peptidoglycan and their teichoic acid.

Bacterial Determinants of Virulence

Pili
The initial event in invasive pneumococcal disease is the attachment of encapsulated pneumococci to epithelial cells in the upper respiratory tract.

Capsule
The bacterial capsule interferes with phagocytosis by leukocytes by interference with binding of complement C3b to the cell surface.

Autolysin
Streptococcus pneumoniae is a very fragile bacterium and contains within itself the enzymatic ability to disrupt and to disintegrate the cells. The enzyme responsible is called an **autolysin.** The physiological role of this autolysin is to cause the culture to undergo a characteristic autolysis that kills the entire culture when grown to stationary phase. Virtually all clinical isolates of pneumococci harbor this autolysin and undergo lysis usually beginning between 18-24 hours after initiation of growth under optimal conditions.

Hemolysins
Two hemolysins have been described, the most potent of which is pneumolysin. **Pneumolysin** is a 53kDa protein that can cause lysis of host cells and activate complement. It is stored intracellularly and is released upon lysis of pneumococci. Pneumolysin binds to cholesterol and thus can indiscriminately bind to all cells without restriction to a receptor.

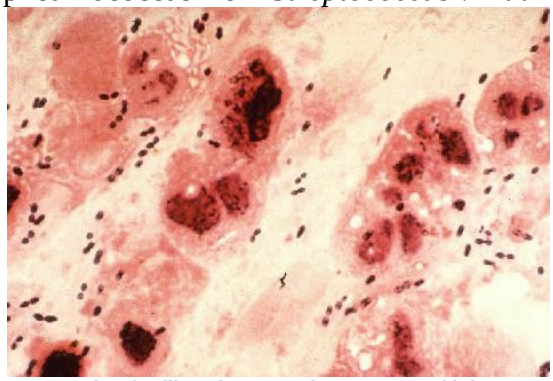
Hydrogen peroxide
H_2O_2 produced by the pneumococcus causes damage to host cells (e.g. can cause apoptosis in neuronal cells during meningitis) and has bactericidal effects against competing bacteria such as *Staphylococcus aureus*.

Neuraminidase and IgA protease
These exoenzymes produced by the bacteria have a presumptive role in virulence as they do in other pathogens.

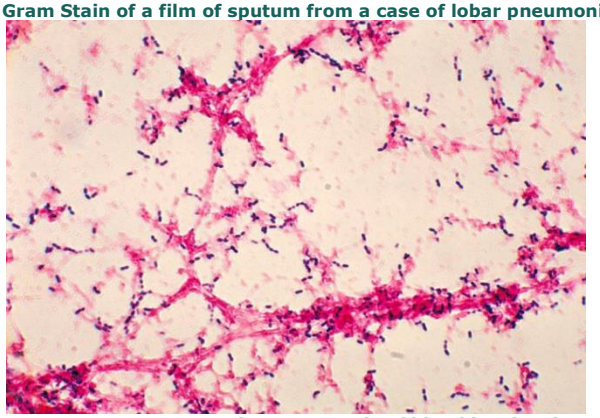
Identification

The minimum criteria for identification and distinction of pneumococci from other *Streptococci* are bile or optochin sensitivity, Gram-positive staining, and hemolytic activity.

On blood agar, colonies characteristically produce a zone of alpha (green) hemolysis, which differentiates *S. pneumonia* from the group A (beta hemolytic) streptococcus, but not from commensal alpha hemolytic (viridans) streptococci which are co-inhabitants of the URT. Special tests such as inulin fermentation, bile solubility, and optochin (an antibiotic) sensitivity must be routinely employed to differentiate the pneumococcus from *Streptococcus viridans.*



Gram Stain of a film of sputum from a case of lobar pneumonia.CDC



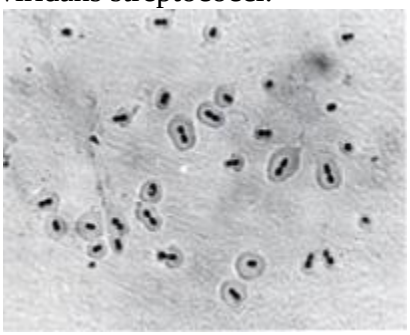
***Streptococcus pneumoniae Gram-stain* of blood broth culture. CDC.**

***Streptococcus pneumoniae* A mucoid strain on blood agar showing alpha hemolysis (green zone surrounding colonies). Note the zone of inhibition around a filter paper disc impregnated with optochin. Viridans streptococci are not inhibited by optochin.**

Note: Under anaerobic conditions they switch to beta hemolysis caused by an oxygen-labile hemolysin.

The **quellung reaction** (swelling reaction) forms the basis of serotyping and relies on the swelling of the capsule upon binding of homologous antibody. The test consists of mixing a loopful of colony with equal quantity of specific antiserum and then

examining microscopically at 1000X for capsular swelling. Although generally highly specific, cross-reactivity has been observed between capsular types 2 and 5, 3 and 8, 7 and 18, 13 and 30, and with *E. coli, Klebsiella, H. influenzae* Type b, and certain viridans streptococci.



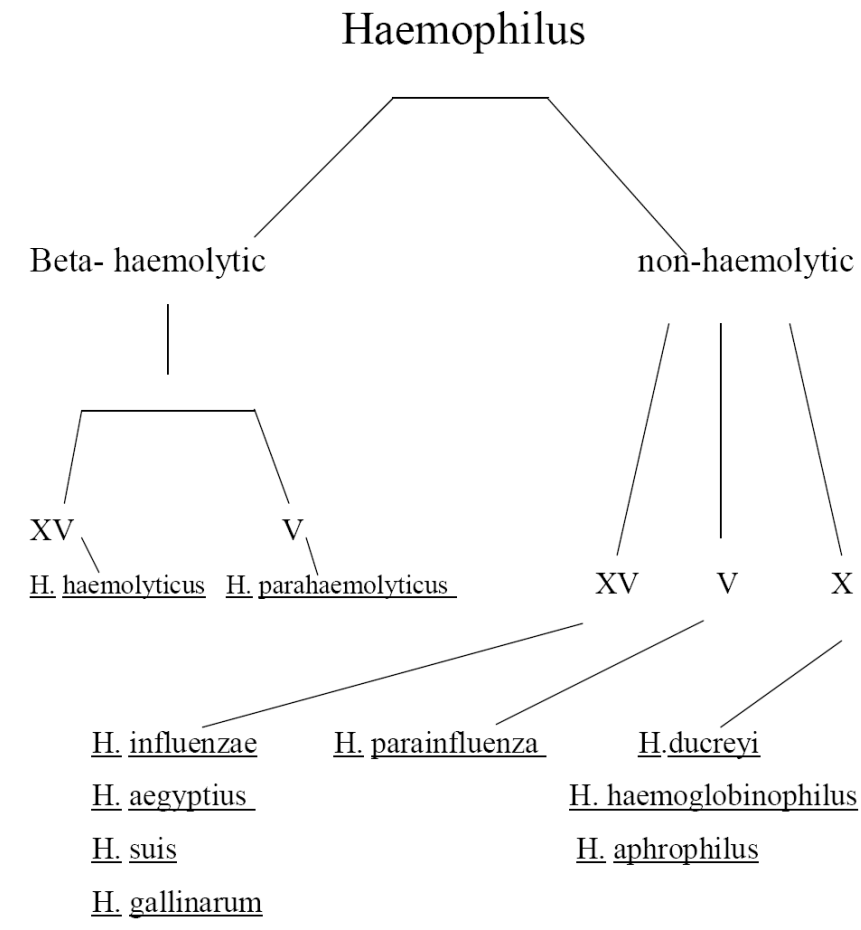
Vaccines

The current Vaccines based on a 23-subgroup of highly prevalent types have been formulated.

Haemophilus

Haemophilus **(**Haemo= blood , philus= loving)

Small gram –ve rods called coccobacilli, sometime are pleomorphic, facultative anaerobic which grow on enriched media, non motile, non spore forming. Require X and V factors for their growth. *Haemophilus* can be divided into



Pathogenesis

Naturally-acquired disease caused by *H. influenzae* seems to occur in humans only. In infants and young children (under 5 years of age), *H. influenzae* type b causes **bacteremia** and acute bacterial **meningitis**. Occasionally, it causes **epiglottitis** (obstructive laryngitis), **cellulitis**, **osteomyelitis**, and **joint infections**. Nontypable *H. influenzae* causes ear infections (**otitis media**) and **sinusitis** in children, and is associated with **respiratory tract infections** (pneumonia) in infants, children and adults.

Seven serotypes of the bacterium have been identified on the basis of capsular polysaccharides. *H. influenzae* type b is the most important serotype involved in meningitis.

Disease caused by *H. influenzae* usually begins in the upper respiratory tract as nasopharyngitis and may be followed by sinusitis and otitis, possibly leading to pneumonia. In severe cases, bacteremia may occur, which frequently results in joint infections or meningitis.

The pathogenesis of *H. influenzae* infections is not completely understood, although the presence of the **type b polysaccharide capsule** is known to be the major factor in virulence. Encapsulated organisms can penetrate the epithelium of the nasopharynx and invade the blood capillaries directly. Their capsule allows them to resist phagocytosis and complement-mediated lysis in the nonimmune host. Nontypable (non encapsulated) strains are less invasive, but they are apparently able to induce an inflammatory response that causes disease. Outbreaks of *H. influenzae* type b infection may occur in nurseries and child care centers, and prophylactic administration of antibiotics is warranted. Vaccination with type b polysaccharide (in the form of Hib conjugate vaccines) is effective in preventing infection, and several vaccines are now available for routine use.

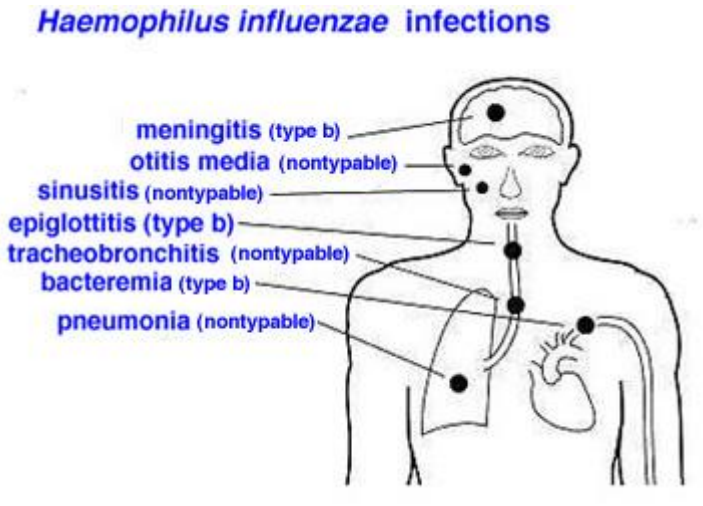


Figure 3. Tissues infected by type b and nontypable strains of *Haemophilus influenzae.*

Virulence

H. influenzae does not produce any demonstrable exotoxins The direct role of

endotoxin in meningitis or bacteremia is unclear, although the Gram-negative bacterium's outer membrane **lipooligosaccharide (LOS)** is thought to play a role in inflammation associated with otitis media. All virulent strains produce **neuraminidase** and an **IgA protease**, but the role of these extracellular enzymes in invasion is unproven. **Fimbriae** increase the adherence of bacteria to human mucosal cells *in vitro*, and they are required for successful colonization of the nasopharynx. The Anton antigen, as defined in red blood cells, appears to be the receptor.

Immunity

The age incidence of *H. influenzae* meningitis is inversely proportional to the titer of bactericidal antibody in the blood, whether passively acquired from the mother or actively formed (see Figure below). Without artificial immunization, in children aged 2 months to 3 years, antibody levels are minimal; thereafter antibody levels increase and the disease becomes much less common. From this curve, it is obvious that artificial active immunization should begin at 2 months of age, when nearly all passive immunity has waned, and the child enters a vulnerable non immune period of life.

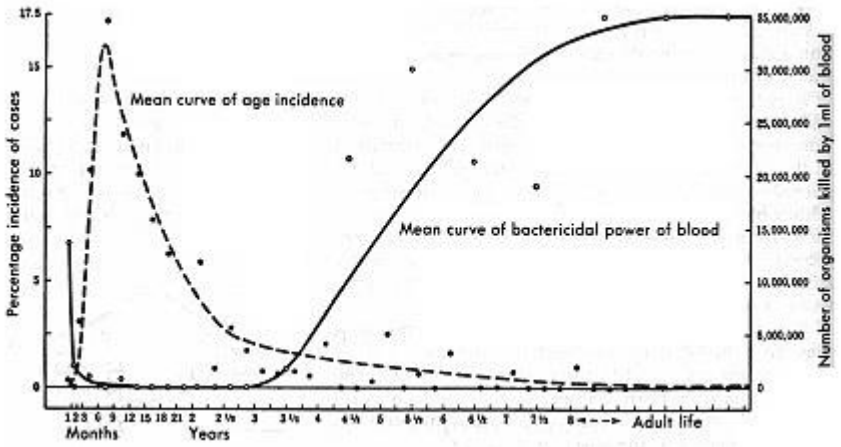


Figure. Relation of the age incidence of bacterial meningitis caused by *Haemophilus influenzae* to bactericidal antibody titers in the blood (data pre 1985).

BACTERIAL INFECTIONS OF THE LOWER RESPIRATORY TRACT

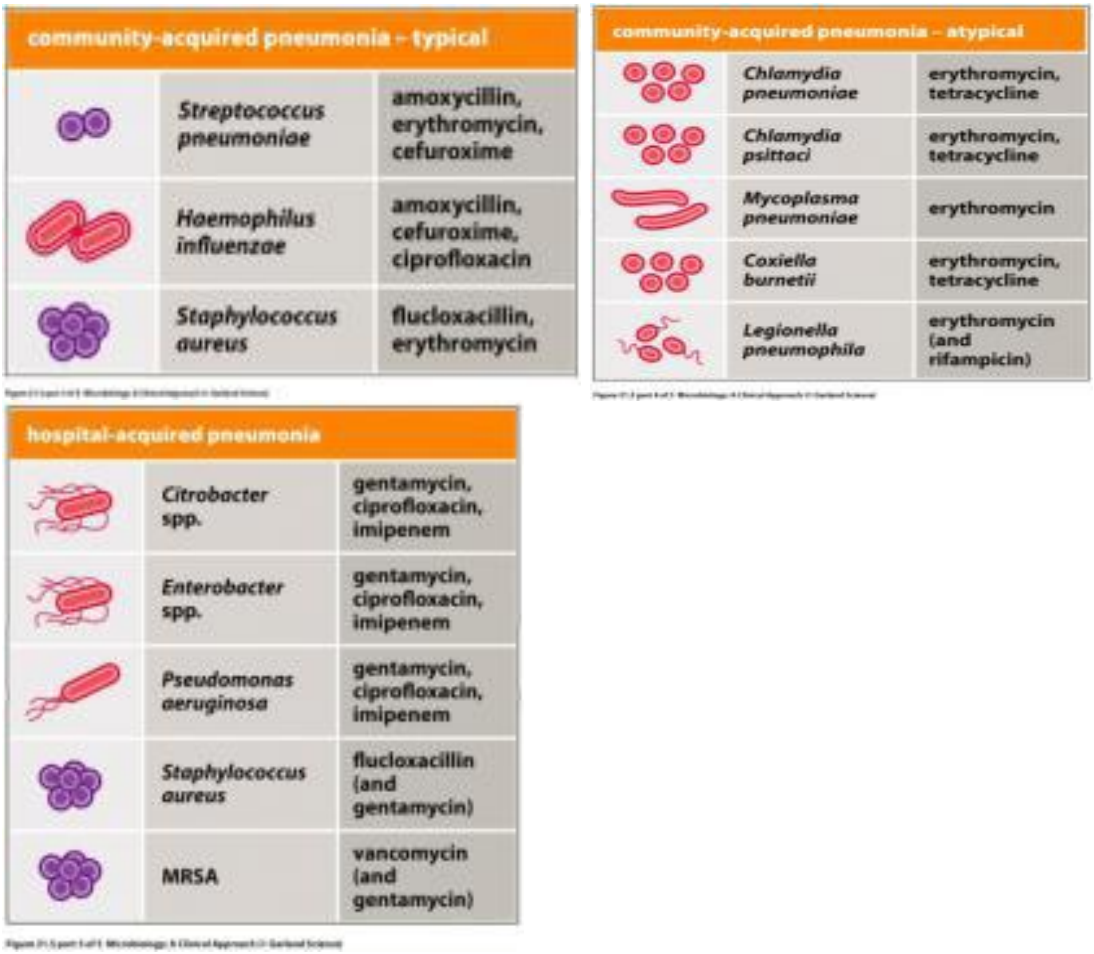
Pneumonia is a disease of the lung that is caused by a variety of bacteria including *Streptococcus, Staphylococcus, Pseudomonas, Haemophilus, Chlamydia and Mycoplasma,* several viruses, and certain fungi and protozoans. The disease may be divided into two forms, bronchial pneumonia and lobar pneumonia. Bronchial pneumonia is most prevalent in infants, young children and aged adults. It is caused by various bacteria, including *Streptococcus pneumoniae.* Bronchial pneumonia involves the alveoli contiguous to the larger bronchioles of the bronchial tree. Lobar pneumonia is more prone to occur in younger adults. A majority (more than 80%) of the cases of lobar pneumonia are caused by *Streptococcus pneumoniae.* Lobar pneumonia involves all of a single lobe of the lungs (although more than one lobe may be involved), wherein the entire area of involvement tends to become a consolidated mass, in contrast to the spongy texture of normal lung tissue.

(Bacterial pneumonia, Chlamydial pneumonia, Mycoplasma pneumonia, Tuberculosis, Pertussis, Inhalation anthrax, Legionella pneumonia (Legionnaire's disease), Q fever.

BACTERIAL PNEUMONIA One of the most serious lower respiratory tract infections. Can be divided into two types: Community-acquired, Nosocomial. Each type can be caused by a variety of organisms.

Nosocomial pneumonia: Occurs approximately 48 hours after admission to hospital, Usually associated with Staphylococcus aureus, Also caused by Gram-negative bacteria, Particularly difficult to deal with if pathogen is resistant to antibiotics

Community-acquired pneumonia: Usually presents as a lobar pneumonia, Accompanied by fever, chest pain, and production of purulent sputum, Most common pathogen is Streptococcus pneumoniae



community-acquired pneumonia – typical		
	Streptococcus pneumoniae	amoxycillin, erythromycin, cefuroxime
	Haemophilus influenzae	amoxycillin, cefuroxime, ciprofloxacin
	Staphylococcus aureus	flucloxacillin, erythromycin

community-acquired pneumonia – atypical		
	Chlamydia pneumoniae	erythromycin, tetracycline
	Chlamydia psittaci	erythromycin, tetracycline
	Mycoplasma pneumoniae	erythromycin
	Coxiella burnetii	erythromycin, tetracycline
	Legionella pneumophila	erythromycin (and rifampicin)

hospital-acquired pneumonia		
	Citrobacter spp.	gentamycin, ciprofloxacin, imipenem
	Enterobacter spp.	gentamycin, ciprofloxacin, imipenem
	Pseudomonas aeruginosa	gentamycin, ciprofloxacin, imipenem
	Staphylococcus aureus	flucloxacillin (and gentamycin)
	MRSA	vancomycin (and gentamycin)

CHLAMYDIAL PNEUMONIA Caused by Chlamydia pneumonia: Found throughout the world, Responsible for 10% of pneumonia cases, Infection occurs throughout the year, Spread by person-to-person contact, More infections in the elderly, Can cause both community-acquired and nosocomial infections

Culture is highly specific but is technically demanding, expensive, has a long turnaround time and its sensitivity is highly dependent on transport conditions. Antigen detection tests such as enzyme immunoassay and direct fluorescent antibody assay, and molecular detection methods such as the polymerase chain reaction as say, may provide a rapid diagnosis without the requirement for stringent transport conditions.

MYCOPLASMA PNEUMONIA Mild form of pneumonia, Accounts for about 10% of all pneumonias, referred to as walking pneumonia, No need for hospitalization, Most common age for infections between 5 and 15 years. Causes approximately 30% of all teenage pneumonias.

PERTUSSIS (WHOOPING COUGH) Spread by airborne droplets from patients in the early stages. Highly contagious, Infects 80-100% of exposed susceptible individuals. Spreads rapidly in schools, hospitals, offices, and homes – just about anywhere.

Infection has three stages: Catarrhal stage – 1-2 weeks↵ Persistent perfuse and mucoid rhinorrhea (runny nose), May have sneezing, malaise, and anorexia, Most communicable during this stage.
Paroxysmal stage- Persistent coughing Up to 50 times a day for 2 to 4 weeks, Characteristic whooping sound is heard. Patient's trying to catch his/her breath, Apnea may follow the coughing, especially in infants. Significant increase in lymphocytes.

Convalescent stage Frequency and severity of coughing and other symptoms gradually decrease.
DX: culture on bordet-Gengou media, direct fluorescent antibody test, PCR.

INHALATION ANTHRAX Produces a fulminate pneumonia: Comes on suddenly with great severity Leads to respiratory failure and death Anthrax primarily a disease of herbivores↵ Acquired from spores found in pastures If spores are inhaled, anthrax can occur in the respiratory tract. The causative agent is Bacillus anthracis. Gram-positive rod, Spore-forming, Spores germinate in human tissues. Antiphagocytic properties of the capsule aid its survival and growth in large numbers.
Pathogenesis results from the powerful exotoxin produced. Symptoms of pulmonary anthrax are: 1-5 days of nonspecific malaise, mild fever, nonproductive cough. Progressive respiratory distress and cyanosis. Rapid and massive spread to the central nervous system and bloodstream is followed by death.
DX: visualization of the large, G+ve rods in blood, confirmed by aerobic cultures on blood agar, PCR.

LEGIONELLA PNEUMONIA (LEGIONNAIRES' DISEASE) Caused by Legionella pneumophila, Gram-negative rod, Cannot be stained or grown using normal techniques.

Legionella is ubiquitous in fresh water. Lives within Acanthamoeba organisms, these are infectious reservoirs. Transmitted to humans as a humidified aerosol, Person-to-person transmission has never been seen. Legionella is a facultative intracellular parasite. Aggressively attacks the lungs, produces a necrotizing multifocal pneumonia, involves alveoli and terminal bronchioles, the inflammatory response produces an exudate containing: Fibrin, polymorphonuclear leukocytes, and red blood cells. Organisms inhaled enter the alveoli. Infect alveolar macrophages, produce an endocytic vesicle, continue replication, prevent fusion of the vesicle with lysosomes, Infected macrophages show a coiled morphology.

Symptoms

Airborne diseases usually result in one or more of the following symptoms:

- inflammation of your nose, throat, sinuses, or lungs
- coughing
- sneezing
- congestion
- runny nose
- sore throat
- swollen glands
- headache
- body aches
- loss of appetite
- fever
- fatigue

Whooping cough gets its name from its main symptom, a severe hacking cough, which is usually followed by a forceful intake of air.

Symptoms of TB vary depend on which organs or body systems are affected and may include coughing up sputum or blood.

Diphtheria can cause marked swelling in your neck. This can make it difficult to breathe and swallow.

Complications from airborne diseases are more likely to affect the very young, the very old, and people with a compromised immune system.

Incidence

Airborne diseases happen all around the world and affect virtually everyone.

They spread easily in close quarters, such as schools and nursing homes. Large outbreaks tend to occur under crowded conditions and in places where hygiene and sanitation systems are poor.

Incidence is lower in countries where vaccines are widely available and affordable.

What you can do to prevent spreading an airborne disease

Although it’s impossible to completely avoid airborne pathogens, there are some things you can do to lower your chances of getting sick:

- Avoid close contact with people who have active symptoms of disease.
- Stay home when you are sick. Don’t let vulnerable people come in close contact with you.
- If you must be around others, wear a face mask to prevent spreading or breathing in germs.
- Cover your mouth when you cough or sneeze. Use a tissue to cut down on the possibility of transmitting germs on your hands.
- Wash your hands thoroughly (at least 20 seconds) and often, especially after sneezing or coughing.
- Avoid touching your face or other people with unwashed hands.

Vaccines can reduce your chances of getting some airborne diseases. Vaccines also lower the risk for others in the community. Airborne diseases that have vaccines include:

- diphtheria
- TB: not generally recommended in the United States
- whooping cough