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Microbiology

Doctor 2017 | Medicine | JU

Sheet

Slides

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DOCTOR

Anas

THIS SHEET WAS WRITTEN ACCORDING TO THE FOLLOWING CRITERIA;

1-THE UNDERLINED (BLACK) WORDS ARE WHAT THE DOCTOR SAID

2-THE UNUNDERLINED (RED) WORDS ARE FROM SLIDES

Note: For a bacteria to be considered as an opportunistic colonizer or a (real) pathogen, there are factors that should be kept in mind; its virulence, the host's immunity and the site of infection. But things aren't always as simple as that.

But in this lecture we will be talking about opportunistic pathogens (they only become pathogens when opportunity allows).

1.PSEUDOMONAS

Motile, straight or slightly curved, gram-negative rods (0.5 to 1.0 × 1.5 to 5.0 μm) typically arranged in pairs

Members of the genus are found in soil, decaying organic matter, vegetation, and water. Also found throughout the hospital environment. And is resistant to many antibiotics and disinfectants.

Pseudomonas (pseudo =mimics , monas =one) aeruginosa (green).

It is commonly found as pairs that appear as one long bacteria. Primarily opportunistic pathogens with certain diseases . It happens when certain abnormalities in the immune barrier . Pseudomonas can be found in adverse environments because it can utilize a wide group of nutrients **for carbon and nitrogen** .

Pseudomonas have certain characteristics that give us evidence that the species where are studying is Pseudomonas ;

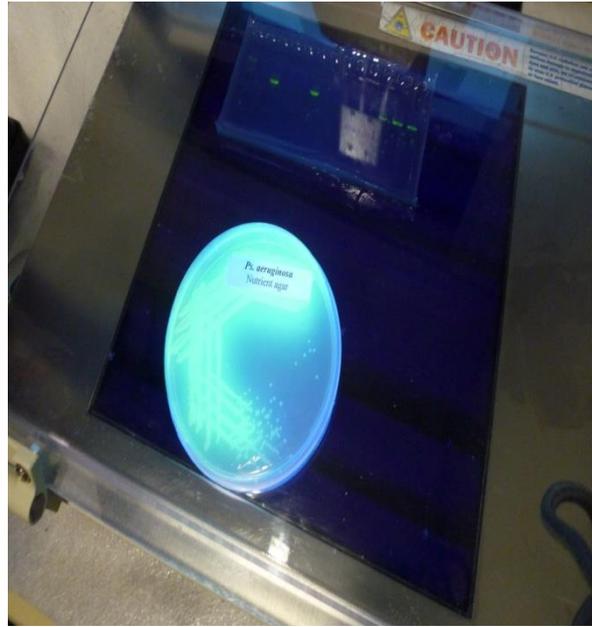


FIGURE 27-5 Colonial morphology of *Pseudomonas aeruginosa*; note the green pigmentation that results from the production of two water-soluble dyes: blue pyocyanin and yellow fluorescein.

1-They form certain dyes (pyocyanin-blue green dye - and fluorescein) which gives green color in colonies and broths.

2-fluorescein will emit light when put under UV LIGHT(certain wavelengths)

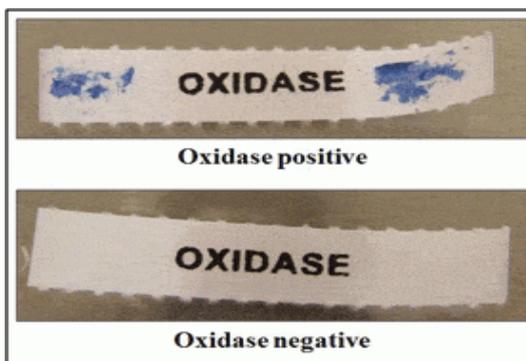
3- The oxidase test(detected in a rapid 5-minute test and specific) which looks for cytochrome C .It is oxidase positive

Very important note mentioned by doctor and in slides :

All bacteria that are oxidase positive are aerobic, and can use oxygen as a terminal electron acceptor in respiration. This does NOT mean that they are strict aerobes. Bacteria that are oxidase-negative may be anaerobic, aerobic, or facultative; the oxidase negative result just means that these organisms do not have the cytochrome c oxidase that oxidizes the test reagent. They may respire using other oxidases in electron transport.

4-Pseudomonas (like many other species) can't ferment lactose,so do not change the color of MacCONKEY agar which is used to differentiate it from Enterobacteriaceae(also a gram negative rod) .We can detect the fermentation of lactose by the changing of the color of the pH indicator of the MacConkey agar .

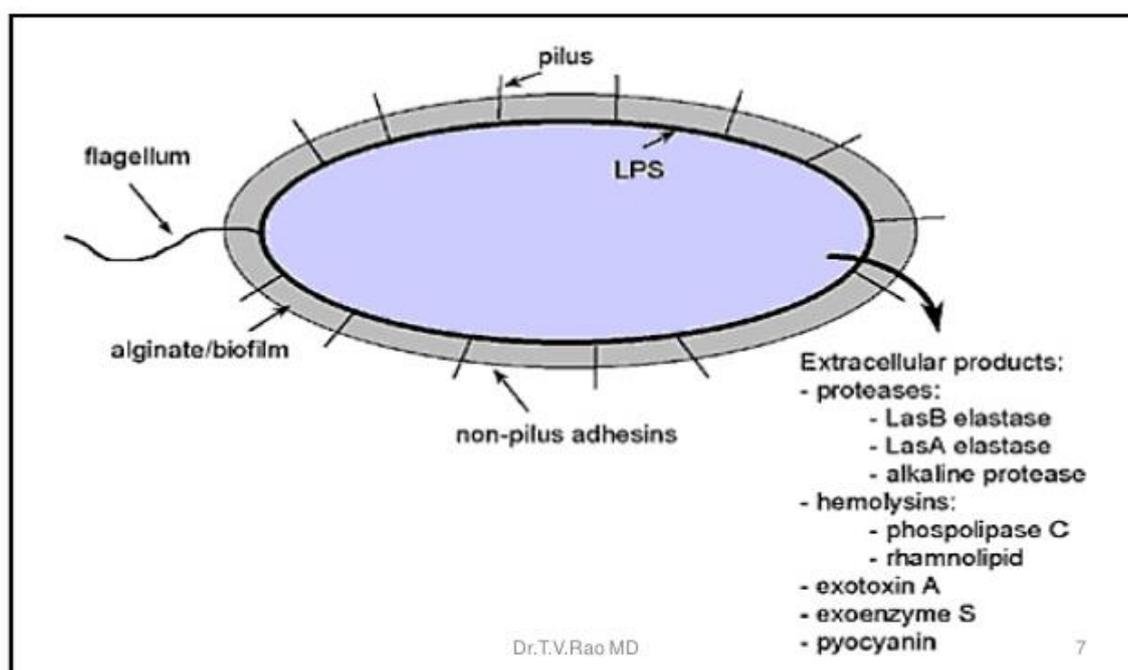
5- The most important thing that differentiate Pseudomonas is that it have a fruity smell (grape-like) .Many other bacteria have weird smells but what is special regarding Pseudomonas is that it's smell is somehow nice.



PATHOGENESIS AND IMMUNITY

Pseudomonas have large arsenal of virulence factors

1- Adhesions like flagella ,pili ,lipopolysachtride (LPS) , and a capsule made of a polysaccharide called alginate which all helps in sticking to tissue.



2- Exotoxins like

A) Exotoxin A which works on disrupting protein synthesis by blocking peptide chain elongation in eukaryotic cells.(diphtherium toxin works also on inhibiting peptide elongation but is more potent than EXOTOXIN A)

B)Elastases that break down the tissue

Two elastases, LasA (serine protease) and LasB (zinc metalloprotease), act synergistically to degrade elastin, related to lung parenchymal damage. Chronic *Pseudomonas* infections are characterized by the formation of antibodies to LasA and LasB.

C)Phospholipases that break down the membranes

Phospholipase C is a heat-labile hemolysin that breaks down lipids and lecithin, facilitating tissue destruction

D)P.agreguisa is very pathogenic because it is resistant to antibiotics and some of this resistance is referred to as intrinsic resistant which is achieved by the slow movement of the antibiotics through the outer membrane pores along with percent of pumps that pump the antibiotic outside of the membrane ,decreasing the antibiotic concentration .We also have acquired resistance that is achieved by horizontal gene transfer .The third type of resistance is adaptive resistance which as the name shows when the bacteria adapts to a new environment.

for example when we add an antibiotic to an agar plate to test sensitivity of a microbial to an antibiotic we are checking intrinsic and acquired resistant and not adaptive resistance and that is one of the reasons why a microbial can be sensitive to an antibiotic on an agar and resistant to the same antibody in the body .Adaptive resistance depends on the environment such as biofilm formation .

Exoenzymes S and T are extracellular toxins facilitating bacterial spread, tissue invasion, and necrosis

Clinical diseases

1-Cystic fibrosis:which is a congenital disease that mutates a certain pump in the cell membrane which causes increased mucous in the airways and damage to the cilia which allows the growth of opportunistic pathogens such as P.aeruginosa

Pulmonary Infections: asymptomatic colonization (in patients with Cystic Fibrosis and other chronic lung diseases) or benign inflammation of the bronchials (tracheobronchitis) to severe necrotizing bronchopneumonia. Previous therapy with broad-spectrum antibiotics and use of mechanical ventilation equipment predispose to infection.

2-Integrity of skin (commonly with burns) : leads to serious infections espically folliculitis (associated with immersion of contaminated water such as in hot tubs)

3 Ear infections (external otitis) such as Swimmers ear : which happens after swimming and is mainly caused by P.AERUGINOSA or happens in moist environments and causes external otitis too.

4- Eye Infections : Occur after initial trauma to the cornea (e.g., abrasion from contact lens)

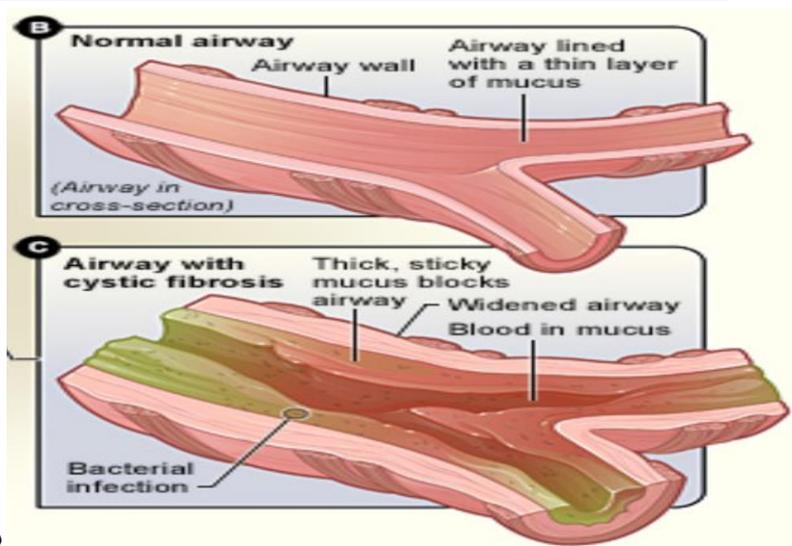
IN burn clinics the main reason for delayed healing is infections and espically P.AERUGINOZA infections and its treatment is complicated because it is resistant to most antibiotics and the patient has compromised defense so we need a combination of antibiotics as a treatment.IF you were asked about diagnosis remember that P.AREUGINOZA is oxidase positive ,green colonies , fruitys smell, green pigments ,lactose non fermentation.

5- Urinary Tract Infections: seen primarily in patients with long-term indwelling urinary catheters.

Remember : The underlying conditions required for most infections are

(1) the presence of the organism in a moist reservoir

(2) compromised host defenses (e.g., cutaneous trauma, elimination of normal microbial flora as a result of antibiotic usage, neutropenia)



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FIGURE 27-3 *Pseudomonas* infection of burn wound.

Pseudomonas aeruginosa

Pulmonary infections: range from mild irritation of the bronchi (tracheo-bronchitis) to necrosis of the lung parenchyma (necrotizing bronchopneumonia)

Primary skin infections: opportunistic infections of existing wounds (e.g., burns) to localized infections of hair follicles (e.g., associated with immersion in contaminated waters such as hot tubs)

Urinary tract infections: opportunistic infections in patients with indwelling urinary catheters and following exposure to broad-spectrum antibiotics (selects for these antibiotic-resistant bacteria)

Ear infections: can range from mild irritation of external ear (“swimmer’s ear”) to invasive destruction of cranial bones adjacent to the infected ear

Eye infections: opportunistic infections of mildly damaged corneas

Bacteremia: dissemination of bacteria from primary infection (e.g., pulmonary) to other organs and tissues; can be characterized by necrotic skin lesions (ecthyma gangrenosum)

THAT'S ALL WITH P.AERUGINIZO

2. Burkholderia

It was first thought to be a kind of PSEUDOMONAS, but new techniques proved that wrong. *Burkholderia* species can colonize a variety of moist environmental surfaces and are opportunistic pathogens

***Burkholderia cepacia* Complex**

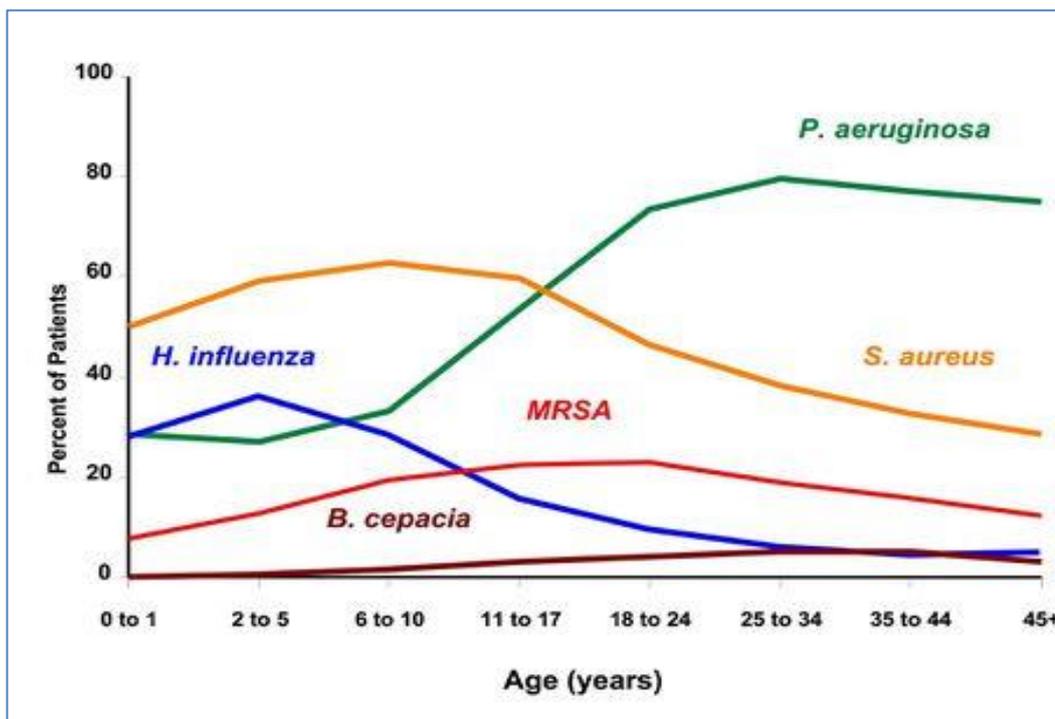
Pulmonary infections: most worrisome infections are in patients with chronic granulomatous disease or cystic fibrosis, in whom infections can progress to significant destruction of pulmonary tissue

Opportunistic infections: urinary tract infections in catheterized patients; bacteremia in immunocompromised patients with contaminated intravascular catheters

Burkholderia pseudomallei

Pulmonary infections: can range from asymptomatic colonization to abscess formation

Important pathogens include *B. cepacia* complex, *Burkholderia gladioli*, and *B. CEPACIA* is similar to *Pseudomonas* and causes cystic fibrosis



AS we can see the percent of patients cystic fibrosis across their lifetime, most patients are colonized by *Pseudomonas*

***B. pseudomallei* is of public health importance in endemic areas, particularly in northeast Thailand, Vietnam, and northern Australia, causing melioidosis(found in moist areas)**

Melioidosis is contracted through direct contact with contaminated soil and surface waters. The time between exposure to emergence of symptoms range from one day to many years; generally symptoms appear two to four weeks after exposure. That is approved by US soldiers who showed the disease after years from coming from the war in VIETNAM .

3.ACINETOBACTER

Acinetobacters are strictly aerobic, oxidase-negative, plump gram-negative coccobacilli

Acinetobacters are opportunistic pathogens (that cause infections in the respiratory tract, urinary tract, and wounds; they also cause septicaemia.

A. baumannii is a frequent cause of hospital-acquired pneumonia, especially of late-onset, ventilator-associated pneumonia and if ventilators are contaminated they will give rise to pulmonary disease. Most of our equipments can be contaminated by bacteria and leads to high morbidity and mortality like catheters in bladders and arms.

***Acinetobacter* Species**

Pulmonary infections: opportunistic pathogen in patients receiving respiratory therapy

Wound infections: traumatic (e.g., resulting from military conflicts) and nosocomial wounds

4. Moraxella

M. catarrhalis is a strictly aerobic, oxidase-positive, gram-negative diplococci

The peak rate of colonisation by M. catarrhalis appears to occur around 2 years of age, with a striking difference in colonization rates between children and adults (very high to very low)

Over the last 20 to 30 years, the bacterium has emerged as a genuine pathogen and is now considered an important cause of upper respiratory tract infections in otherwise healthy children and elderly people

M. catarrhalis is an important cause of lower respiratory tract infections, particularly in adults with chronic obstructive pulmonary disease (COPD) (COPD which commonly happens commonly in smokers and damages lungs, decreasing lungs immunity against infections.It is considered a nosocomial pathogen_

It is becoming more spread over the last period mainly because of 2 reasons :

1-It spreads in hospitals and is an important cause of upper respiratory tract infection

2-It is becoming more resistant to antibiotics.

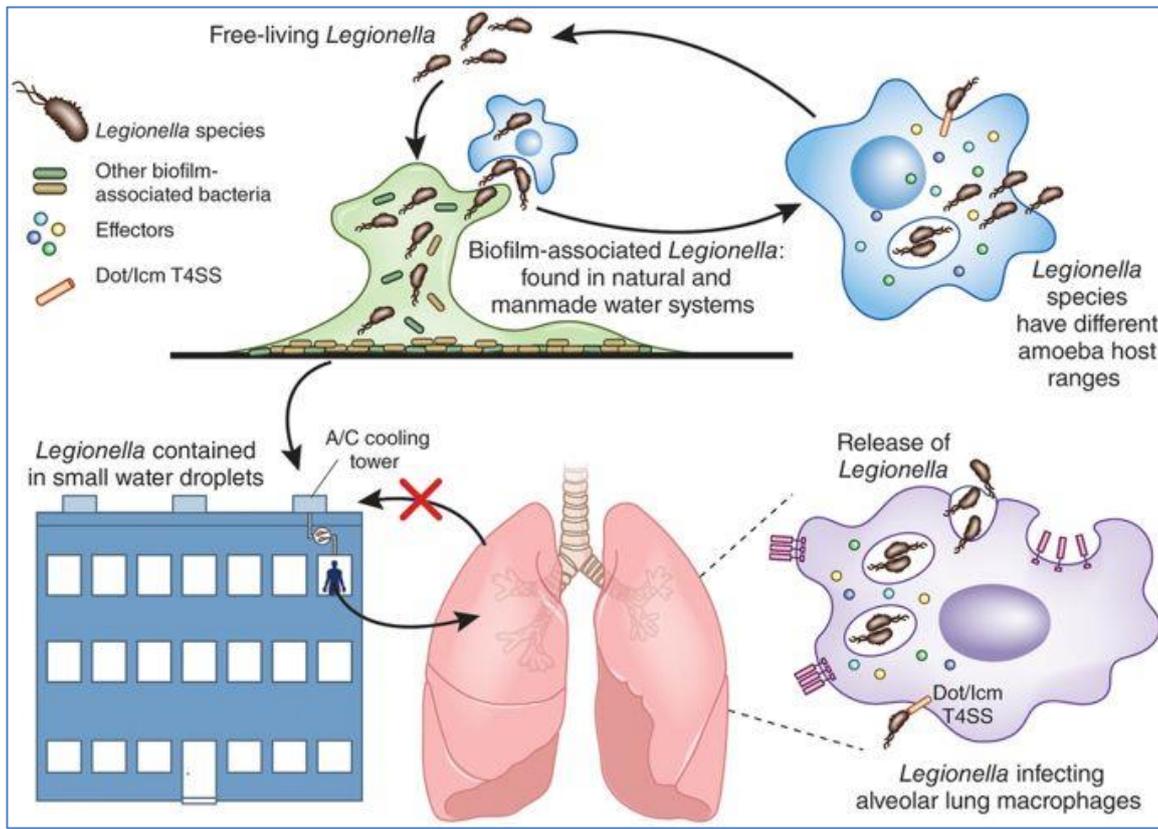
5. Legionella

Slender, pleomorphic, gram-negative rods. Legionellae are obligatively aerobic and nutritionally fastidious (They require media supplemented with cysteine). They are hard to isolate.

Legionella acquired its name after an outbreak of a then-unknown "mystery disease" that sickened 221 people, causing 34 deaths, at a convention of the American Legion. After scientists looked why the legion which went to the convention developed the disease, they found that this microbe is growing within the central air conditioning cooling towers. It can grow in water, sometimes intracellularly, forming biofilm and persists in the tank or goes inside amoeba in the tank and grows inside it and replicates. Then through the air condition to the patients by inhalation. It can also be found in shower heads, water misters whirlpool spas).

Human infections are most commonly associated with exposure to contaminated aerosols

Legionellae are facultative intracellular bacteria (infect and replicate in macrophages and amoeba). Cytokines released by the infected macrophages stimulate a robust inflammatory response that is characteristic of infections with *Legionella*.



The water containing the microbe will spread through the AC and will be inhaled to the lungs ,where the lungs try to get rid of it but legionella is able to survive causing disease.

L. pneumophila is the cause of 90% of all Legionella infections, affecting the lungs and present in one of two forms :

(1) an influenza-like illness (referred to as Pontiac fever (self-limited, febrile illness)) and

(2) a severe form of pneumonia (i.e., legionnaires disease).

The medium most commonly used for the isolation of legionellae is buffered charcoal yeast extract (BCYE) agar(almost specific to recognize Legionella)

Legionnaires disease is characteristically more severe and, if untreated, promptly causes considerable morbidity

6. BORDETELLA

Bordetella is an extremely small (0.2 to 0.5 × 1 μm), fastidious, strictly aerobic, gram-negative coccobacillus. Even under ideal conditions, recovery of *B. pertussis* in culture is difficult. Pertussis is a human disease with no other recognized animal or environmental reservoir

Pertussis is primarily a toxin-mediated disease. Pertussis toxin (A-B toxin) inactivates the protein that controls adenylate cyclase activity, leading to an increase in cyclic adenosine monophosphate (cAMP) levels and a subsequent increase in respiratory secretions and mucus production.

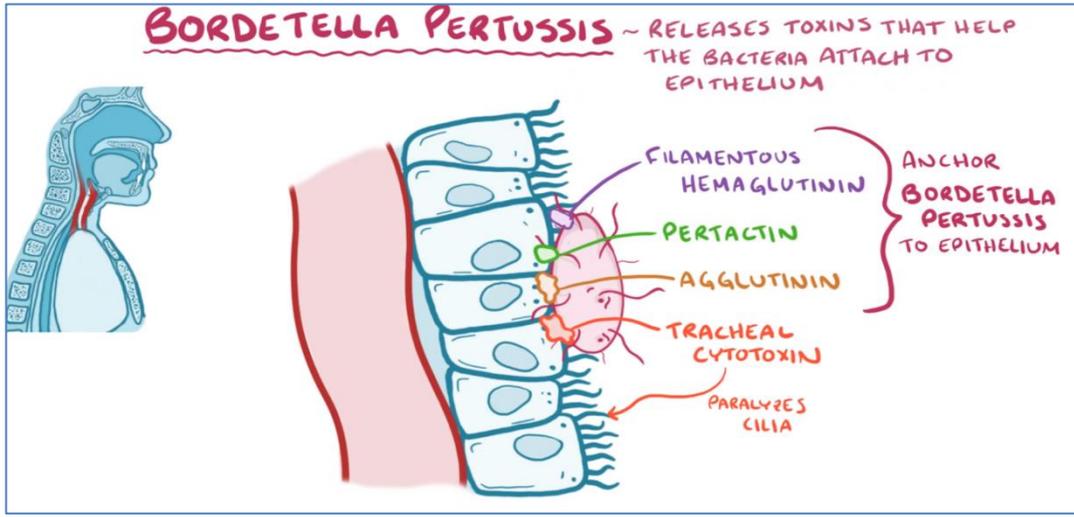
Bordetella pertussis, the agent responsible for pertussis or whooping cough. Transmitted mainly by respiratory droplets.

Nowadays we have vaccines for pertussis (as well as diphtheria and tetanus) . Sometimes even if vaccinated ,you may get the disease because of weak immunity. Common in kids .

The cough it presents is unique because it is continuous and usually comes as paroxysm (which end up with exhaustion and vomiting) and is stopped by the baby taking a breath .

The paroxysm differentiates it from other upper respiratory tract infections. Sometimes there is a recovery phase after the paroxysm .

The reason of the paroxysm is that the bacteria stick to the cilia of the epithelial lining of the lung and then starts producing toxins that damage the mucosa and pertussis toxin will increase the secretion of mucous and damage (paralyze) the cilia and cause inflammation of the respiratory tract, which interferes with the clearing of pulmonary secretions. so we cough to get this mucous out.



	Incubation	Catarrhal	Paroxysmal	Convalescent
Duration	7-10 days	1-2 weeks	2-4 weeks	3-4 weeks (or longer)
Symptoms	None	Rhinorrhea, malaise, fever, sneezing, anorexia	Repetitive cough with whoops, vomiting, leukocytosis	Diminished paroxysmal cough, development of secondary complications (pneumonia, seizures, encephalopathy)
Bacterial culture				

FIGURE 29-3 Clinical presentation of *Bordetella pertussis* disease.



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