Other Enterobacteriaceae

Include Klebsiella, Enterobacter, Proteus, and Serratia, which can be found as normal inhabitants of the large intestine.

Enterobacter

Enterobacter species are motile and Lac+. They rarely cause primary disease in humans but frequently colonize hospitalized patients, especially in association with antibiotic treatment, indwelling catheters, and invasive procedures. These organisms may infect burns, wounds, and the respiratory (causing pneumonia) and urinary tracts.

Klebsiella

Klebsiellae are large, nonmotile bacilli that possess capsule. They are Lac+. Klebsiella pneumoniae and Klebsiella oxytoca cause necrotizing lobar pneumonia in individuals compromised by alcoholism, diabetes, or chronic obstructive pulmonary disease.

K. pneumoniae also causes UTI and bacteremia, particularly in hospitalized patients.

Serratia

The species of Serratia that most frequently causes human infection is Serratia marcescens. Serratia can cause extraintestinal infections such as those of the lower respiratory and urinary tracts, especially among hospitalized patients.

Proteus, Providencia, and Morganella

Members of these genera are agents of urinary tract and other extraintestinal infections. Proteus species are relatively common causes of uncomplicated as well as nosocomial UTI. Other extraintestinal infections, such as wound infections, pneumonias, and septicemias,

are associated with compromised patients. Proteus organisms produce urease, which catalyzes the hydrolysis of urea to ammonia. The resulting alkaline environment promotes the precipitation of stones.

Other gram negative rods

The following organisms do share two significant features of structure and physiology.

First, they all have a gram-negative cell envelope and, therefore, contain lipopolysaccharide (LPS), which is a virulence factor.

Second, they grow in the presence of oxygen and, therefore, cause infections at sites where oxygen tension is high (for example, in the lungs, and other vital tissues).

1)organism that are primarily or exclusively pathogens of the human respiratory tract (Haemophilus, Bordetella and Legionella).

2) *Pseudomonas*, an organism that can infect a wide variety of tissues and whose virulence is potentiated by certain immune compromise.
3) those that are primarily pathogens of animals (that is, zoonotic organisms, such as Brucella, Francisella, and Pasteurella, for which humans are accidental hosts).
Yersinia pestis is a member of the family Enterobacteriaceae, but a non-gastrointestinal, gram-negative rod.

HAEMOPHILUS
Haemophilus influenzae—the major human pathogen of this genus—are pleomorphic, ranging from coccobacilli to long, slender filaments.

H. influenzae may produce a **capsule** (six capsular types have been distinguished) or may be **unencapsulated**. The capsule is an important virulence factor. Serious, invasive *H. influenzae* disease is associated particularly with capsular type b (Hib).

H. influenzae is a **normal component of the upper respiratory tract** flora in humans and may also colonize the conjunctiva and genital tract. Humans are the only natural hosts, and colonization begins shortly after birth.

H. influenzae is transmitted by **respiratory droplets**. **IgA protease** produced by this organism degrades secretory IgA, facilitating colonization of the upper respiratory tract mucosa. From this site, *H. influenzae* can enter the bloodstream and disseminate to distant sites.

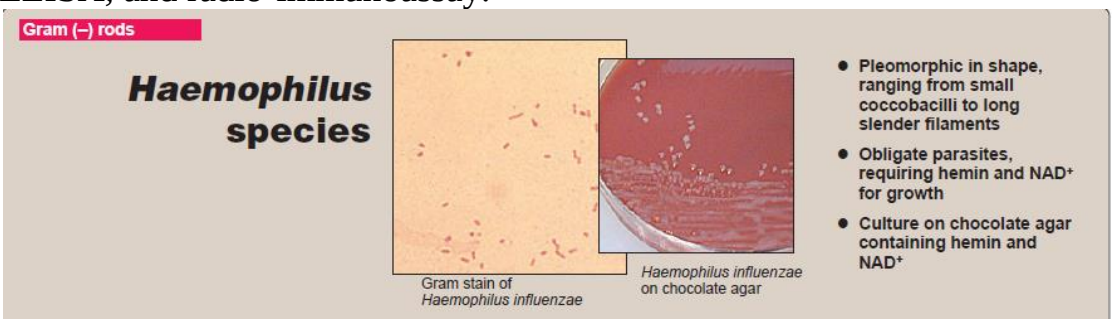
H. influenzae has been a leading cause of bacterial meningitis, primarily in infants and very young children, frequently in conjunction with an episode of otitis media.

A vaccine against *H. influenzae* type b, administered to infants, has dramatically decreased the frequency of such infections.

Laboratory identification
H. influenzae is fastidious and requires supplementation with hemin, factor X, and nicotinamide adenine dinucleotide (NAD+), factor V. thus, *H. influenza* can be cultured on chocolate agar (lysed blood cells provide these growth factors) but cannot be grown on blood or MacConkey agar.
Isolation from normally sterile sites and fluids, such as blood, CSF, and synovial fluid, is significant, whereas isolation from pharyngeal cultures is inconclusive.

Gram staining of CSF commonly reveals pleomorphic, gram-negative coccobacilli.

Type b capsule may be identified directly in CSF, either by the capsular swelling (quelling) reaction or by immunofluorescent staining. Capsular antigen may be detected in CSF or other body fluids using immunologic tests, such as latex agglutination, countercurrent immunoelectrophoresis, ELISA, and radio-immunoassay.



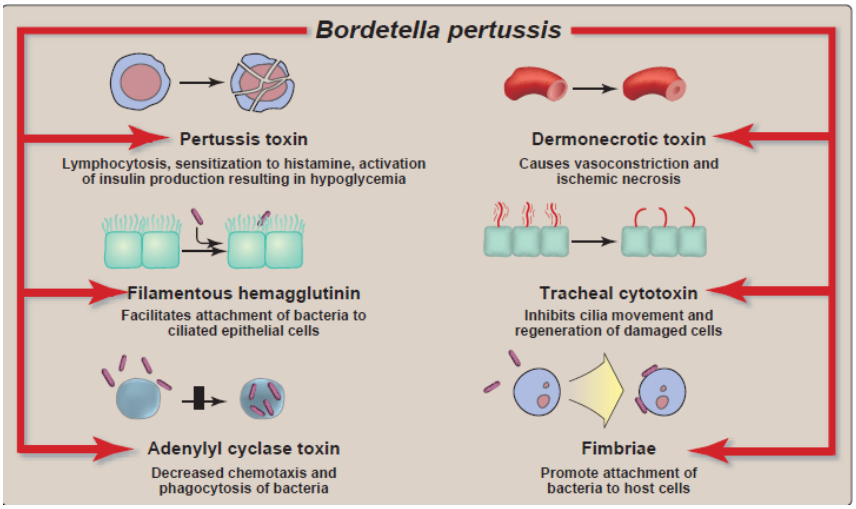
BORDETELLA

Bordetella pertussis (pertussis also known as whooping cough), Which is a highly contagious disease and a significant cause of morbidity and mortality worldwide. Members of the genus Bordetella are **aerobic**. They are small, **encapsulated** coccobacilli that grow singly or in pairs. They can be **serotyped** on the basis of cell-surface molecules including adhesins and fimbriae.

The major mode of **transmission is via droplets spread by coughing**, but the organism survives only briefly outside the human respiratory tract.

Pathogenesis

B. pertussis **binds to ciliated epithelium** in the upper respiratory tract, There, the bacteria **produce a variety of toxins** and other virulence factors that interfere with ciliary activity, eventually causing death of these cells.

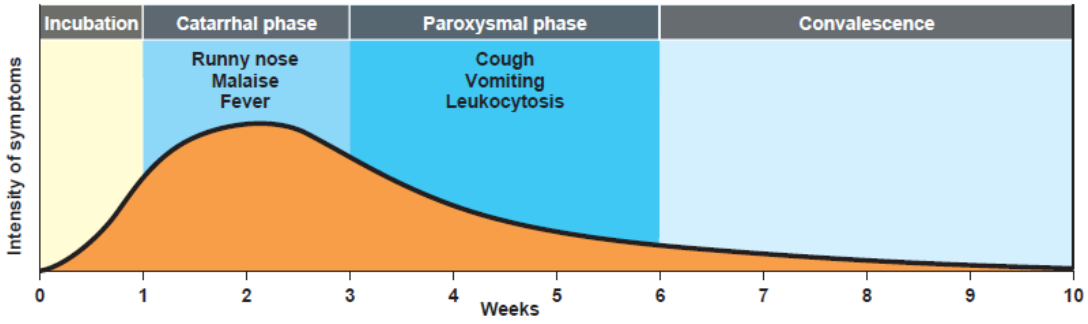


The incubation period: 1 to 3 weeks. The disease can be divided into two phases: catarrhal and paroxysmal.

1. Catarrhal phase: This phase begins with relatively nonspecific symptoms. Patients in this phase of disease are highly contagious.

2. Paroxysmal phase: With worsening of the cough, the paroxysmal phase begins. The term "whooping cough" derives from the paroxysms of coughing followed by a "whoop" as the patient inspires rapidly. [Note: Whooping may not occur in all patients.]

Following the paroxysmal phase, convalescence requires at least an additional 3 to 4 weeks. During this period, secondary complications, such as infections (for example, otitis media and pneumonia) and central nervous system (CNS) dysfunction (for example, encephalopathy or seizures), may occur. Disease is generally most severe in infants.



Laboratory identification

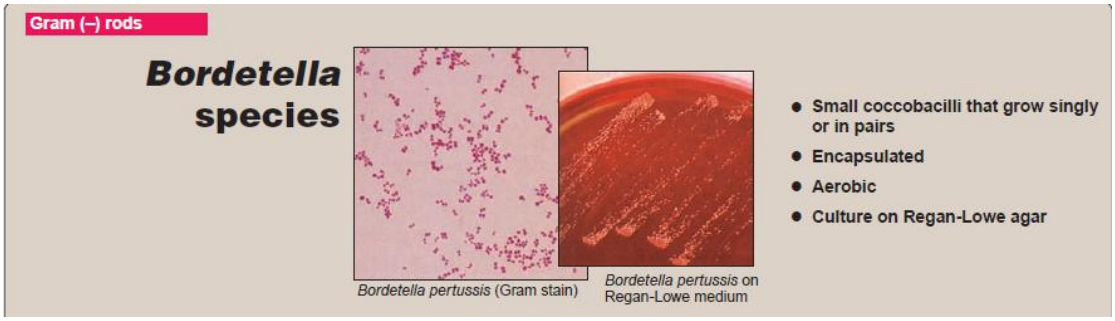
Presumptive diagnosis may be made on clinical grounds once the paroxysmal phase of classic pertussis begins.

Culture of *B. pertussis* on **Bordet-Gengou** media.

More rapid diagnosis may be accomplished using a direct fluorescent antibody test to detect *B. pertussis* in smears of nasopharyngeal specimens.

Serologic tests for antibodies to *B. pertussis* are primarily useful for epidemiologic surveys.

Prevention: Pertussis vaccine is available (DTP)



LEGIONELLA

Legionellaceae are facultative intracellular parasites that cause primarily respiratory tract infections.
In nature, Legionella cells are **unencapsulated**, relatively slender rods, whereas in clinical material, they appear coccobacillary in shape.
Members of the Legionellaceae family are **aerobic and fastidious**, with a particular requirement for **L-cysteine**.

Epidemiology

The Legionellaceae family includes 34 species whose normal habitat is within environmental protozoa and amebae in soil and water, including water in cooling towers and distribution systems.
About 85 to 90 percent of human disease is caused by a single species, Legionella pneumophila.
Most infections result from **inhalation of aerosolized** organisms within amebas or within environmental biofilm but, occasionally, may follow other exposures (for example, swimming in contaminated water).
Both sporadic cases and localized outbreaks may occur.
The organism is **chlorine tolerant** and, thus, survives water treatment procedures. There is no person-to-person spread of the disease.

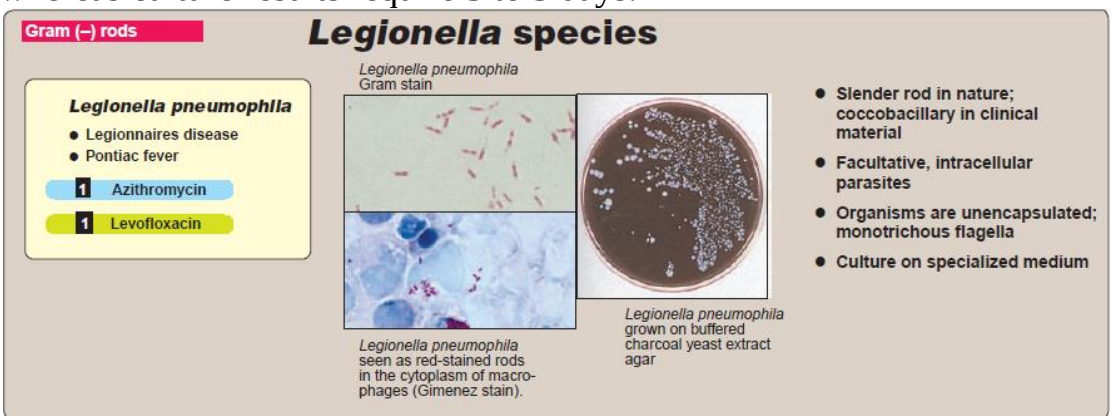
Pathogenesis

The organism gains entry to the upper respiratory tract by aspiration of water containing the organism or by inhalation of a contaminated aerosol. Failure to clear the organisms permits them to reach the lungs. Alveolar macrophages in the lung bed normally constitute an important line of defense for clearing invading organisms. Although the macrophages do phagocytose *L. pneumophila*, the resulting phagosome fails to fuse with a lysosome. Instead, the organisms multiply within the protected environment of the phagosome until the cell ruptures, releasing a new bacteria.

There are two distinctly different presentations: Legionnaires disease (LD atypical, acute lobar pneumonia) and Pontiac fever (influenza-like illness).

The state of the host's cell-mediated immunity plays a critical role in determining which manifestation will occur.
Immunosuppressed patients are more likely to develop severe pneumonia when infected with Legionella while Pontiac fever is almost always seen in otherwise healthy individuals.

Laboratory identification
LD cannot be diagnosed on the basis of clinical presentation or radiologic appearance of lungs.
The organism can be Gram stained, the Gimsa stain is more useful for visualization.
The definitive method of diagnosis involves the culturing of Legionella from respiratory secretions, using **buffered (pH 6.9) charcoal yeast extract enriched with L-cysteine, iron, and α-ketoglutarate**. Visible colonies form in 3 to 5 days.
A urinary antigen test using an enzyme immunoassay is available and has several advantages over culture. For example, the test positivity can persist for days even during administration of antibiotic therapy, making it useful in patients who receive therapy.
Further, the results of the urinary antigen test can be available within hours, whereas culture results require 3 to 5 days.



PSEUDOMONAS

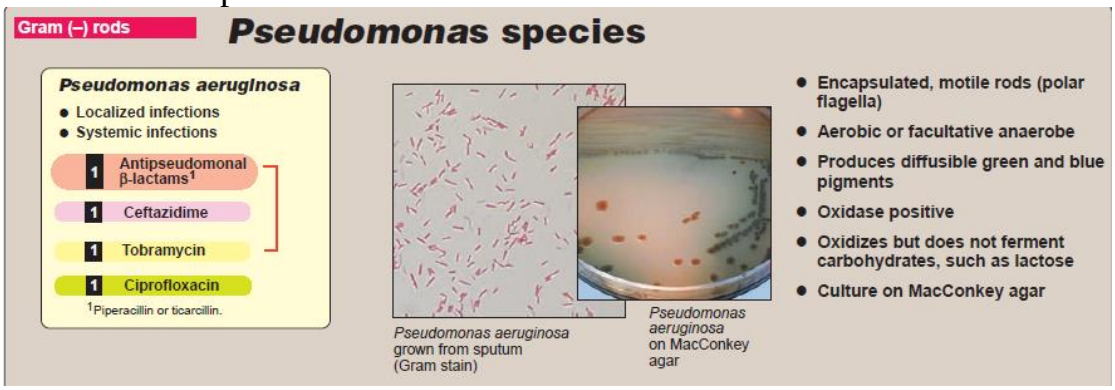
Pseudomonas aeruginosa, the primary human pathogen in the genus Pseudomonas, is widely distributed in nature. It is found in soil, water, plants, and animals. Although it may colonize healthy humans without causing disease, it is also a significant opportunistic pathogen and a major cause of **nosocomial (hospital-acquired) infections**.
P. aeruginosa is regularly a cause of nosocomial pneumonia, nosocomial urinary tract infections, surgical site infections, infections of severe burns, and infections of patients undergoing either chemotherapy for neoplastic disease or antibiotic therapy.

P. aeruginosa is **motile** (it has polar flagella) and aerobic or facultative.
P. aeruginosa does **not ferment carbohydrates** but can utilize alternate electron acceptors, such as nitrate, in anaerobic respiration.
Nutritional requirements are minimal, and the organism can grow on a wide variety of organic substrates. In fact, P. aeruginosa can even grow in laboratory water baths, hot tubs, intravenous (IV) tubing, and other water-containing vessels. **This explains why the organism is responsible for so many nosocomial infections.**

Pathogenesis
P. aeruginosa disease begins with attachment to and colonization of host tissue. Pili on the bacteria mediate adherence, and mucoid strains predominate in patients with cystic fibrosis (CF).
The mucoid capsule is composed of a repeating polymer of mannuronic and glucuronic acids called **alginate**.
Alginate expression confers resistance to phagocytosis and clearing in the CF lung.
Host tissue damage facilitates adherence and colonization.
P. aeruginosa produces numerous toxins and extracellular products that promote local invasion and dissemination of the organism (causes both localized and systemic illness)

Individuals most at risk include those with impaired immune defenses.

Laboratory identification
P. aeruginosa can be isolated by plating on a variety of media, both nonselective (for example, blood agar) and moderately selective (forexample, MacConkey agar).
Identification is based on the results of biochemical and other diagnostic tests.
P. aeruginosa typically produces a blue-green pigment called pyocyanin and is oxidase positive.



BRUCELLA

Brucellosis (undulant fever) is a zoonosis (a disease of animals that may be transmitted to humans under natural conditions). Different species of Brucella are each associated with particular animal species: Brucella abortus (cattle), Brucella melitensis (goats and sheep), Brucella suis (swine), Brucella canis (dogs), and Brucella ovis (sheep).

The brucellae are aerobic, facultatively intracellular parasites that can survive and multiply within host phagocytes.

Cells of the genus Brucella are unencapsulated, small coccobacilli arranged singly or in pairs. LPS is the major virulence factor as well as the major cell wall antigen.

Brucellosis is a chronic, lifelong infection in animals. Organisms localize in reproductive organs (male and female) and are shed in large numbers in milk, urine, the placenta and other tissues discharged during delivery or spontaneous abortion.

The primary manifestations of infection in animals are sterility and abortion.

Transmission to humans characteristically occurs as a result of either direct contact with infected animal tissue or ingestion of unpasteurized milk or milk products.

Pathogenesis

Brucellae typically enter the body through cuts and abrasions in the skin or through the gastrointestinal (GI) tract. Drugs that decrease gastric acidity may increase the likelihood of transmission via the GI route.

Inhalation of infected aerosols can also lead to disease among abattoir workers.

Once the organisms gain entry, they are transported via the lymphatic system to the regional lymph nodes, where they multiply.

The organisms are then carried by the blood to organs that are involved in the reticuloendothelial system, including the liver, spleen, kidneys, bone marrow, and other lymph nodes.

The incubation period for Brucella infections ranges from 5 days to several months but typically lasts several weeks.

Symptoms are nonspecific and flulike (malaise, fever, sweats, anorexia, GI symptoms, headache, and back pains) and may also include depression.

Untreated, patients may develop an undulating pattern of fever (temperatures repeatedly rise then fall, hence the name “undulant fever,” the traditional name for brucellosis).

Laboratory identification

Because the nonspecific symptoms may not point to a diagnosis of brucellosis, a detailed history is often crucial, including the patient's occupation, exposure to animals, travel to countries where brucella infection is prevalent, and ingestion of potentially contaminated foods.
The organism can be cultured from blood and other body fluids or from tissue specimens. Multiple blood specimens should be cultured. For plated materials, colonies may appear in 4 to 5 days, whereas longer times are required for blood cultures, and these are routinely examined for up to 1 month before being declared negative.