Chapter 20 – Medically Important Gram-Negative Bacilli*

*Lecture notes are to be used as a study guide only and *do not* represent the comprehensive information you will need to know for the exams.

[I. Gram-Negative Bacilli & Septic Shock]*

The Gram negative bacilli are a large group of non-spore-forming rod-shaped bacteria, Table 20.1. They are found in a wide number of niches and habitats. Many of them are residents of the large intestine (enteric), some are zoonotic, some live in soil and water. This group comprises true pathogens, like *Salmonella*, and opportunistic pathogens, like Pseudomonads. See also **Systems Profile 20.1.**

20.1 Aerobic Gram-Negative Nonenteric Bacilli

This group is comprised of soil inhabitants and zoonotic pathogens. This group is differentiated by their oxygen requirements and fermentation of lactose. One universal component to all gram-negative rods is the presence of lipopolysaccharide in the outer cell membrane. This is the endotoxin which causes symptoms associated with gram-negative septicemia. Read: **20.1 MAKING CONNECTIONS.**

NOTE THE FOLLOWING FOR UNDERSTANDING:

Septic shock is a pathologic state of low blood pressure (with concomitant reduction of blood flow to vital organs); major symptoms include nausea, tachycardia (racing heart), cold & clammy skin, and weak pulse. Damage to organs can cause respiratory failure, coma, heart failure, and death in hours.

Lipopolysaccharide (LPS) stimulates the release of interleukins, cytokines, and tumor necrosis factor from macrophages. If the infection is only local, this response is normally positive and results in the control of the invading pathogen. However, if the infection is systemic wide sepsis, the massive release of cytokines etc. will lead to fever (since many cytokines are pyrogenic) and intravascular blood coagulation. The end results are circulatory failure, tissue necrosis, hypotension, and shock.

Remember, LPS molecules are "released" by the pathogen only after it dies. Therefore, administering antibiotics can compound the problem rather than solve it. Immune serum globulins are sometimes available to counteract the LPS toxin effect.

[The following notes organizes the various genera based on their oxygen requirements (aerobic vs. anaerobic).]*

[II. Aerobic Rods *Bordetella, Pseudomonas, and Legionella*]*

Pseudomonas: The Pseudomonads

Species of the genus Pseudomonas are highly versatile; easily adapt to a wide range of habitats. They can degrade numerous extracellular proteins by means of proteases. *Pseudomonas aeruginosa* (fig. 20.1) is the primary pathogen from this group and is highly resistant to soaps, quaternary ammonium disinfectants, drugs, drying, and temperature extremes; they can be frequent contaminants of ventilators, intravenous solutions, and anesthesia equipment. *P. aeruginosa* is unlikely to cross first level portals of entry and opportunistically depends on invasive medical procedures or weakened host defenses.

The most common nosocomial infections occur in compromised hosts with severe burns, neoplastic disease, and cystic fibrosis. Unusual characteristics of *P. aeruginosa* infections are a grapelike odor and the noticeable blue-green color that appears in tissue exudates (the bacterium produces a blue-green/greenish-yellow pigment called pyocyanin) (fig. 20.3). Community acquired infections can come from contaminated loofa sponges or other abrasive exfoliating scrubbers (fig. 20.2).

P. aeruginosa will express virulence factors including exotoxins, a phagocytosis-resistant slime layer, and other exoenzymes which degrade host tissues. Lysis of the bacterium releases LPS endotoxins capable of causing septic shock.

* Section titles in brackets, [], are from the original set of notes.

Treatment: 3rd generation cephalosporins, aminoglycosides, carbenicillin, polymyxin, quinolones, and monobactams. A vaccine is being developed for cystic fibrosis patients. See **Pathogen Profile #1** *Pseudomonas aeruginosa*

20.2 Related Gram-Negative Aerobic Rods

This group is comprised of obligate aerobes, but do not ferment sugars. The bacteria in this group are opportunistic pathogens to a compromised human host.

Burkholderia cepacia can be isolated from moist environments. An opportunistic pathogen of the respiratory tract, urinary tract, and skin. Cyctic fibrosis patients are at most risk for *B. cepacia* infections. It mutates rapidly and is highly resistant to a number of antibiotics.

Burkholderia pseudomallei resides in soil and water of the tropics. It causes melioidosis. It is acquired through a penetrating injury or inhalation. Signs of this organism are skin nodules, fever and muscel aches. Can infect the lungs (fig. 20.4). A chronic infection can lead to speticemia, endotoxic shock, and death.

Stenotrophomonas maltophilia can be part of the fecal microbiota of humans. It appears in the clinical setting as a contaminant of disinfectants and hospital equipment. This bacterium can form biofilms, which protects it from drugs and disinfectants. Found in several tissue and body fluid samples. Seen often in cancer patients and people with indwelling devices.

Brucella and Brucellosis

Brucellosis is a zoonotic infectious agent. There are two (2) speceis of human medical importance – *Brucella abortus* (from cattle) and *Brucella suis* (from pigs). They are both tiny, gram negative coccobacilli. Humans infected experience a severe febrile illness, but not abortion. Brucellosis typically occurs in those people who handle livestock on a daily basis – slaughter houses, veterinarians. In humans *Brucella* enters via broken skin or mucous membranes of the digestive tract, eye, or respiratory tract. An infected human has a fluctuating fever, hence the name undulant fever. Serological tests can be used to diagnose the bacterium in a patient (fig. 20.5).

Francisella tularensis and Tularemia

Francisella tularensis is the causative agent of tularemia, a zoonotic disease of animals endemic the Northern hemisphere. It is associated with outbreaks in rabbits, hence its name rabbit fever. *Francisella tularensis* can be transmitted by animals and ticks. The infectious dose of this bacterium is 10 – 50 cells. It is an intracellular pathogen that “hides” in macrophages. A vaccine is available, and it is recommended to wear protective clothing if exposure is possible.

Bordetella pertussis and Relatives

Pertussis or whooping cough is characterized by a cough with a "whooping" sound (fig. 20.6). Incubation period averages 10 days (5-21 days). Initially it is characterized by cold symptoms lasting 1-2 weeks followed by the coughing. Bouts of coughing last 10-20 seconds.

It is caused by *Bordetella pertussis*, a small G- coccobacillus. It produces a capsule and a toxin called tracheal cytotoxin (a peptide fragment from the cell wall). The toxin causes ciliated cells of the trachea to cease beating rhythmically, causes the ciliated cells to detach from the epithelium, modifies cellular metabolism, and inhibits DNA synthesis. This results in the buildup of mucus which blocks the airways and leads to secondary pulmonary infection. Cough persists for a week after pathogen is eliminated.

Humans are the only reservoir for the organisms, which are transmitted in aerosols while coughing. DTaP vaccination is available. There are 2000 to 4000 cases reported annually in the U.S. with fewer than 10 deaths. In countries lacking routine vaccination and

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in industrialized countries with lax vaccination, pertussis is a major cause of childhood sickness and death. Figure 20.7 for pertussis prevalence.

A new acellular pertussis (aP) vaccine (made only from parts of the organism, rather than the whole organism) has been produced. It produces less severe and less frequent local and systemic reactions than the current whole-cell vaccine (American Journal of Nursing, June 1992).

Treatment: Erythromycin is the drug of choice. It kills the organism, but does not destroy the toxins. Human pertussis immune serum globulin (antitoxin) can offset the effects of the toxins.

See also **Pathogen Profile #2** *Bordetella pertussis*

Alcaligenes faecalis is isolated from feces, sputum, and urine. It is an opportunistic pathogen and can cause pneumonia, septicemia, and meningitis.

Legionella and Legionellosis

Legionella is a novel bacterium unrelated to other strictly aerobic gram-negatives. *L. pneumophila* is the species most often isolated in infections. Bacteria are endocytosed into phagocytes were they evade the immune system (fig. 20.8).

Easily surviving harsh environmental conditions, *L. pneumophila* is widely distributed. Pneumonia resulting from infection has been termed legionellosis or Legionnaires' disease (see chapter 18 notes).

See also **Pathogen Profile #3** *Legionella pneumophilia*

20.3 Identification and Differential Characteristics of Family Enterobacteriaceae

[III. Characteristics of the Family Enterobacteriaceae – Facultative Anaerobic Rods]*

[*Escherichia coli, Klebsiella, Proteus, Enterobacter, Serratia, Citrobacter, Salmonella, Shigella,* and *Yersinia*]*

Enterics are those organisms which occupy the digestive tract of humans and animals. They are small non-spore forming rods. Enteric pathogens are frequent causes of diarrhea illnesses (Read: **20.2 MAKING CONNECTIONS**).

Note: Diarrhea is a symptom of gastrointestinal disorders. It is characterized by frequent watery stools. Infectious agents which produce diarrhea include viruses, bacteria, and protozoa. Gastritis and enteritis is inflammation of the stomach and small intestine, respectively. Gastroenteritis is an inflammation of both. These inflammations can be caused by diet, toxins, infections, drugs or psychological factors (stress). Enteritis produces profuse, watery discharge induced by toxins, change in ionic balance in epithelium; the mucosa and submucosa may or may not be involved. If involved, it causes inflammation. Colitis is an inflammation of the large intestine. Infections cause mucosal erosion accompanied by abdominal cramps, painful spasmodic contractions of the bowel, inflammatory exudates (pus, blood and leukocytes)- also called dysentery.

Enterics include *Escherichia coli*, *Klebsiella*, *Proteus*, *Enterobacter*, *Serratia*, and *Citrobacter*. These organisms cause diseases other than those involving the intestine. These enterics and *Pseudomonas* account for more than 50% of all nosocomial infections. The genera in this family share several biochemical characteristics (fig. 20.9).

E. coli and other gram-negative normal enteric flora that ferment lactose (within 48 hours) are called coliforms. Noncoliforms are non-lactose-fermenting or slow lactose-fermenting bacteria, including *Salmonella*, *Shigella*, and *Yersinia*. (For reference only - i.e. do not memorize them – see: Figure 20.10 and Table 20.2 shows the protocol for identifying these organisms. Table 20.3 gives the

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results of IMViC series. See also the lists of Coliforms in Normal Microbiota, Noncoliforms in Normal Microbiota, True Pathogen Enterics, True Pathogenic Nonenteric pgs. 614 – 615.

Antigenic Structures and Virulence Factors

Gram-negative enterics have complex surface antigens that are important in their pathogenicity and the basis of immune responses (fig. 20.11). H – flagellar antigen; K – capsule/ fimbrial antigen; O – somatic / cell wall antigen. The O antigen is the most common among the gram negative enterics, it is implicated in endotoxic shock. Most species of gram negative enterics are identified by serotypes. The pathogenesis of enterics is tied to their production of endotoxin, exotoxins, capsules, fimbriae and molecules that attach to host cells. This group also has a propensity of exchange antibiotic resistance genes by mechanisms previously discussed.

20.4 Coliform Organisms and Disease

Escherichia coli: The Most Prevalent Enteric Bacillus

E. coli is a Gram-negative, facultative anaerobic, glucose and lactose-fermenting straight rod. It is oxidase negative, motile by lateral (peritrichous) flagella. It colonizes the intestine, where it is part of the normal flora (mutualism). Of over 150 strains, most are not infectious, however, some have acquired virulence factors through plasmid transfer (or other means).

E. coli can cause urinary tract infections and diarrhea. Enterotoxigenic *E. coli* causes diarrhea brought on by 2 exotoxins (enterotoxins). Onset appears suddenly and causes severe dehydration. It causes Traveler's diarrhea (Montezuma's revenge). Enteroinvasive *E. coli* involves invasion and ulceration of the large intestine mucosa, resulting in an inflammatory disease similar to dysentery. Enteropathogenic strains of *E. coli* are related to a wasting form of infantile diarrhea. The various pathogenic strains can be differentiated on the basis of antigens (H, K, and O serotyping – fig. 20.11).

E. coli O157:H7 an enterohemorrhagic form (having acquired Shiga toxin from Shigella dysenteriae) which caused deaths associated with improperly cooked hamburgers in 1993 and the 2006 outbreak associated with spinach. In about 10% of cases hemolytic uremic syndrome (HUS) can develop when the toxins cause kidney damage and or kidney failure.

Also, remember that normal floral *E. coli* causes 50-80% of urinary tract infections in healthy individuals and up to 90% of nosocomial UTIs.

Clinical Diseases of E. coli

Mostly common among humans. Pathogenic strains of *E. coli* are frequent agents of infantile diarrhea. The immature, neonatal intestine has no protection against pathogens that enter via unsanitary food or water. Traveler's diarrhea is most often picked up by eating or drinking contaminated food and water. Pepto-Bismol seems to be the most effective OTC to provide relief from the effects of the enterotoxin and provided some antimicrobial effect.

The Role of Escherichia coli in Food Infections

Prevention: avoid contaminated water and raw vegetables. Drink bottled water or boil water. Oral sulfonamides and bismuth subsalicylate (Pepto-Bismol) have been recommended and shown effective against traveler's diarrhea. Hamburger should never be eaten "rare" (rare beef = <150ºF; well done = >170ºF) – want to avoid *E. coli* O157:H7, an enterohemorrhagic bacterium (fig. 20.12). Any *E.coli* which contaminate a slaughterhouse gets mixed into the hamburger and thus if improperly cooked, will survive. Steaks, and other non-ground beef, are usually "safer" to eat rare since bacteria which coat the surface of the meat are killed during cooking. A more severe case is hemolytic uremic syndrome (HUS).

Treatment: drugs include tetracycline, sulfonamides and ampicillin. Electrolytes and fluid replacement where needed.
http://www.cdc.gov/nczved/dfbmd/disease_listing/stec_gi.html
http://www.cdc.gov/travel/diseases.htm

* Section titles in brackets, [], are from the original set of notes.

Miscellaneous Infections

E. coli can invade sites other than the intestine. It can cause UTI – urinary tract infections. Left untreated, it can move up to the bladder and kidneys, especially in women. Other infections include neonatal meningitis, septicemia and wound infections.

E. coli and the Coliform Count

It is used to measure fecal contamination in water, food, and dairy products. The presence of *E. coli* as a fecal contaminant is indicative of other possible pathogens such as Salmonella, viruses and pathogenic protozoans.

Other Coliforms

Other coliforms of clinical importance are opportunists:

Klebsiella pneumoniae can be found in the respiratory tract of normal individuals. Its presence can lead to chronic lung infections. It produces large capsule (Fig. 20.13) which prevents phagocytosis.

Enterobacter can be a cause of UTIs. Can be lethal if in the blood.

Citrobacter inhabits the human colon, can cause an opportunistic UTI and bacteremia in debilitated persons.

Serratia can be found in the intestine. *S. marcescens* produces a red pigment at room temperature (fig. 20.14). It can invade a compromised host, such as alcoholics, where is can cause pneumonia. It is most often seen in burn and wound infections, as well as fatal septicemia and meningitis in immunosuppressed patients.

See also **Pathogen Profile #4** *Escherichia coli*

20.5 Noncoliform Enterics

Opportunists: *Proteus* and Its Relatives

Proteus can be saprobic and commensals of humans. *Proteus* exhibits a characteristic swarming pattern on agar (fig. 20.15). *Proteus* UTI seems to stimulate renal stones.

True Enteric Pathogens: *Salmonella* and *Shigella*

Salmonella (salmonellosis) and *Shigella* (shigelloses) are distinguished by the fact that they have well-developed virulence factors, are primary pathogens, and are NOT part of the normal human flora.

Typhoid Fever and Other Salmonelloses

Typhoid fever is a gastrointestinal infection caused by *Salmonella typhi*. It is characterized by high fever (104ºF), headache, diarrhea, and abdominal tenderness. Incubation period for typhoid fever is 1-3 weeks, average = 2 weeks. Early symptoms: nosebleeds, general weakness, mild headache and malaise followed b abdominal tenderness and rose spots on the abdomen, high fever,

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enlarged spleen and diarrhea (3-7 bowel movements/day). If the bacteria reach the blood, the endotoxin may cause shock (fig. 20.17).

Salmonella is a facultative intracellular parasite of macrophages. Bacteria are phagocytized, but not killed by macrophages. Instead they multiply and escape in large numbers in the bloodstream. Transmitted by fecal-oral route. No vaccine gives full protection.

About 5% of those who recover from typhoid fever shed organisms for one year or more although they are asymptomatic (i.e. feel fine and show no symptoms) – they are carriers.

Treatment: Drug of choice is amoxicillin.
http://www.cdc.gov/ncidod/dbmd/diseaseinfo/typhoidfever_g.htm

Animal Salmonelloses

In general, any bacterial gastroenteritis is an inflammation of the epithelial lining of the stomach and intestines. Symptoms: abdominal pain, nausea, vomiting and diarrhea. *Salmonella* gastroenteritis is characterized by these symptoms. It is also called Salmonella poisoning. It is caused by large number of organisms rather than the toxins. Culprits include *Salmonella enteritidis* and *S. cholerae-suis*; are zoonotic in origin, i.e. they are normal intestinal flora in cattle, poultry, rodents, and reptiles. *Salmonella cholerae-suis* causes septicemia while *Salmonella enteritidis* does not. They enter the gastrointestinal tract through contaminated food. More than 100,000 organisms are needed to cause disease. This contrasts with 1,000-10,000 *S. typhi* to cause typhoid fever. Leaving food in a warm place for more than 4 hours allows bacteria to multiply to an infectious level. Generally self-limiting disease and is not treated. See 20.3 **MAKING CONNECTIONS** for information on avoiding gastrointestinal infections associated with food.

See also **Pathogen Profile #5** *Salmonella typhi, Salmonella enterica*

Shigella *and Bacillary Dysentery*

Bacterial dysentery is caused by *Shigella* species. This condition is also called shigellosis. It is characterized by diarrhea, the urge to defecate but can't (tenesmus), cramps, and mucus and blood in feces. Involves inflammation of the ileum or colon which causes a sloughing of mucosal lining resulting in intestinal ulcerations (fig 20.18).

Shigella is a gram-negative, facultative anaerobic, non-motile, straight rod. Oxidase positive. Three species (*Shigella dysenteriae*, *S. sonnei*, *S. flexneri*) produce an enterotoxin that works like choleragen (see *Vibro* below): sloughing of intestine, loss of water and electrolytes. Less than 200 organisms can cause the disease!

Shigella dysenteriae type I produces a neurotoxin (exotoxin). It causes bleeding and paralysis by inhibiting protein synthesis. Like typhoid fever, it is transmitted by the fecal-oral route. It occurs in low numbers in countries with high standards of hygiene: chlorination of water, maintenance of reservoirs and water pipes, raw sewage treatment, pasteurization of milk. There is no vaccine.

Treatment: Ampicillin. Chloramphenicol and trimethoprim sulfamethoxazole used where there is ampicillin resistance. Most cases are not treated at all. Where treatment is needed, it is fluid replacement and electrolyte therapy.
http://www.cdc.gov/nczved/dfbmd/disease_listing/shigellosis_gi.html

The Enteric* Yersinia *Pathogens

(i.) Enteric Yersinia Pathogens
Enterocolitis caused by *Yersinia enterocolitica*, a G- bacillus. It is more prevalent in Western Europe and Scandinavia than in U.S. Symptoms: fever, diarrhea, abdominal pain. Animals serve as reservoirs. Isolated from food, water and unpasteurized milk.

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Nonenteric *Yersinia pestis* and Plague

(ii) Nonenteric *Yersinia pestis*

Plague or Black Death swept through Europe in 1348, 1349 and sporadically thereafter. This invasion killed an estimated 50% of the population. First pandemic (worldwide epidemic) occurred in the 6th century (AD).

Plague is caused by *Yersinia pestis*, a small G-, straight, nonmotile, coccobacillus (fig. 20.19).

Virulence Factors

Yersinia pestis produces a necrotizing exotoxin that kills phagocytes in the lymph nodes where the bacilli are multiplying. This causes lymph nodes to swell and elicit a fever. From lymph nodes, *Yersinia* can spread to lymph and blood causing bacteremia, fever, delirium, shock and death. The necrotizing exotoxin causes hemorrhaging in various parts of the body. It also produces a coagulase that clots the blood.

The Complex Epidemiology and Life Cycle of Plague

The organism is transmitted by the bite of the rat flea (*Xenopsylla cheopsis*). The bacterium is picked up from an infected animal and is contained in the gut of the rat flea. The flea, upon biting another rodent, regurgitates blood and bacilli into the wound. If the rodent host dies, the flea seeks another host and will take a human host if a rodent is unavailable (fig. 20.20).

THE ANIMAL RESERVOIRS The endemic reservoirs are rodents, such as mice and voles, that harbor the organism. These rodents spread the disease to other mammals, such as, rats, squirrels, and rabbits, then it can infect humans.

FLEA VECTORS The principle vector in transmitting the plague bacillus to humans is the flea. The flea ingests a blood meal from an infected animal and can transmit it to humans. Humans can also be infected by handling infected animals, animal skins, or meat.

Pathology of Plague

A pustule may or may not appear at the site of entry. From the bite, the bacilli enter the dermal lymphatics and are transported to the regional lymph nodes, usually the groin or armpit nodes become enlarged and tender and are called buboes, hence bubonic plague (fig 20.20). Incubation lasts 2-8 days, ending abruptly with the onset of fever, chills, headache, nausea, malaise, weakness and extreme tenderness of the bubo. If untreated, fatality rate is 50-75%.

From lymph nodes, the organisms can spread via circulation to the spleen, liver, lungs and sometimes the meninges. This is septicemic plague. Subcutaneous hemorrhagic, blackened lesions that can degenerate into necrosis and gangrene appear, giving the disease the name "Black Death." If untreated, the fatality rate approaches 100%.

Sometimes when it is spread to the lungs it initiates what is called pneumonic plague. Early stage symptoms include fever, heavy cough and a thick mucus stained with blood. This disperses the organism quickly making pneumonic plague highly contagious. If untreated, fatality rate approaches 100%. If septicemic and pneumonic plague are treated, there is a 90-95% survival rate.

Prevention: Eliminating slums and garbage heaps, which support rodent populations. Limiting the numbers of ground squirrels that come in contact with humans. When rodents and ground squirrels are found dead, it is called epizootic plague. Dying rats was an indicator of the presence of plague.

There is a vaccination available for persons at high risk. However, the vaccination only yields short-term immunity.

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Treatment: Rapid diagnosis and administration of streptomycin. Fifteen cases are reported/year with an average of 1 death. *Yersinia pestis* is harbored in the rodent population of 12 western states (including Texas). It was brought to San Francisco around 1906.
http://www.cdc.gov/ncidod/dvbid/plague/index.htm

Oxidase-Positive Nonenteric Pathogens in the Family Pasteurellaceae

[IV. Non-coliform, Non-enteric, oxidase-positive G – pathogens]*

Pasteurella multocida

Pasteurella is a zoonotic pathogen that is of main concern to veterinarians. ***P. multocida*** can be responsible for opportunistic infections in poultry and wild fowl, cattle, household cats and dogs. It can be transmitted to humans by animal bites. People with other health complications are very susceptible.

Haemophilus: The Blood-Loving Bacilli

The genus *Haemophilus* has several species that cause significant pathologies. These are Gram-negative, pleomorphic rods with fastidious growth requirements. *H. influenzae* causes severe bacterial meningitis (untreated cases have a fatality rate of 90%), but is being brought under control due to the Hib vaccine. Fig. 20.22. (Also, see notes on meningitis covered in the chapter 18 lecture notes.)

Haemophilus aegyptius is the etiologic agent of acute communicable conjunctivitis (also known as pinkeye) (fig. 20.23). Infection of the conjunctiva causes hemorrhage that will turn the sclera (normally white) of the eye bright pink/red. Primary occurrence in children where it is spread easily via fingers and shared items. Treatment: Antibiotic eye drops.

Haemophilus ducreyi is the etiologic agent of **chancroid** (soft chancre), a STD, common in the tropics and subtropics. Mostly common in males. It is seen most often in the sexually promiscuous and those with poor hygiene.

BELOW ARE ADDITIONAL NOTES – FYI!!!!!

[V. Other Curviform Gram-Negative Bacteria]*

Vibrio cholerae, *Campylobacter jejuni*, and *Helicobacter pylori*

1. *Vibrio*

Cholera is an acute infection by *Vibrio cholerae*. It is characterized by profuse, watery discharge. Initially diarrhea is brown and then develops to a pale whitish color known as "rice water" because it contains flecks of mucous. It may be accompanied by vomiting.

Vibrio cholerae is G-, motile by polar flagella, curved or comma-shaped rod (fig. 21.10). Oxidase positive, facultative anaerobe. The cells adhere to the microvilli of epithelial cells where they multiply and produce a toxin called cholera toxin. The toxin induces excessive fluid loss from the intestinal cells, up to 1 Liter/hour in extreme cases (fig. 21.11). The tremendous fluid loss and loss of electrolytes decreases blood volume and can lead to hypovolemic shock (i.e., shock from loss of blood volume). Hypotension, tachycardia, cyanosis and collapse from shock occurs within 18-24 hours. If the disease goes untreated, death is in less than 48 hours. There is approximately 55% mortality rate.

The organism enters the human host through contaminated food or water. Infected individuals shed the pathogen into sewage, which contaminates water supplies. Spread of the disease is by the fecal-oral route. Carriers harbor the organism in their gall bladders, just as Typhoid Mary did with typhoid.

A vaccine does exist for frequent travelers.

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Treatment: Fluid and electrolyte replacement. Read: Insight 21.2. Tetracycline reduces the numbers of organisms in the intestine and helps eliminate carrier situation.
http://www.cdc.gov/nczved/dfbmd/disease_listing/cholera_gi.html

Vibrio species and seafood

Vibrio parahaemolyticus and *V. vulnificus* are salt-tolerant species that are often the leading cause of food-poisoning associated with eating raw or undercooked seafood (especially raw oysters). Both cause severe gastroenteritis. Incubation period of about 24 hours followed by profuse watery diarrhea, nausea, vomiting, and abdominal cramps. Treatment requires fluid and electrolyte replacement.

NOTE: Some species of *Vibro* are capable of causing a necrotizing fasciitis like *S. pyogenes* (see Ch. 18). These bacteria will infect small cuts and abrasions while a person is swimming in brackish water (for instance Galveston Bay). The symptoms and signs are identical and the condition will spread very quickly. Immediate medical attention is necessary.

2. *Campylobacter jejuni*

Campylobacter jejuni is now considered one of the most important causes of bacterial gastroenteritis worldwide. Primary pathogen transmitted through contaminated beverages and food, especially water, milk, meat, and especially chicken. According to some estimates, approximately 80% of chicken bought in U.S. grocery stores is contaminated with either C. jejuni or Salmonella.

C. jejuni is Gram negative, monotrichous flagella, curved rod, microaerophilic and grows best at 40-42ºC (fig. 21.12). *C. jejuni* adhere to the mucosa of the small intestine where they burrow and multiply.

Signs and symptoms develop after 1 to 7 days post-ingestion: Fever and malaise followed by diarrhea (may be bloody) and severe cramping. Lasts less than 1 week and is self-limiting. Electrolyte and fluid replacement necessary.

3. *Helicobacter pylori*

Although it was first isolated in 1979 and implicated in causing gastritis, *Helicobacter pylori* has been accepted in recent years as an etiologic agent of stomach ulcers. Ulcer formation is linked to other factors including overproduction of stomach acid (genetic cause or stress). However, *H. pylori* is now recognized as a very strong contributing factor.

Occurring in ~25% of the adult population (60% of people 60 and over), *H. pylori* seems to be acquired early in life and carried asymptomatically until its activities begin to damage the digestive mucosa. (Fig. 21.13)

Treatment: Newest recommended therapy for stomach ulcers is 2-4 weeks of clarithromycin to eliminate the bacteria and Zantac™ to inhibit the formation of stomach acid.
An enterotoxin is any toxin produced by a pathogenic organism which acts on the large or small intestine.
Note: Typhoid fever is so named only because the symptoms resemble typhus. Typhus is a Rickettsia disease (see chapter 21).
The most famous was Mary Mallon - "Typhoid Mary" - who worked as a cook in the early 1900s and spread the infection to hundreds of people before she was forced into a quarantine facility.
It is estimated that one out of every three chickens is contaminated with Salmonella.
The Komodo dragon's bacteria count is so high that people visiting a zoo who touched the rail of a dragon's cage contracted *Salmonella*!
Do not confuse bacterial dysentery with amebic dysentery, which is caused by the protozoan *Entamoeba histolytica* and *Giardia lamblia.*
NOTE: This is a zoonotic infection, i.e. an infection acquired from another animal. The rat flea would be considered a biological vector (Ch. 13).
Boubon is Greek for groin
Vibrio, *Campylobacter*, and *H. pylori* are covered in the book's chapter 21; but presented here with the other gram-negative bacilli.

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