



INFECTIOUS DISEASE

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BACTERIOLOGY

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PARASITOLOGY

VIROLOGY

## BACTERIOLOGY - CHAPTER ELEVEN

# ENTEROBACTERIACEAE, VIBRIO, CAMPYLOBACTER AND HELICOBACTER

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## ENTEROBACTERIACEAE

### General

This group of organisms includes several that cause primary infections of the human gastrointestinal tract. Thus, they are referred to as **enterics** (regardless of whether they cause gut disorders). Bacteria that affect the gastrointestinal tract include certain strains of *Escherichia coli* and *Salmonella*, all 4 species of *Shigella*, and *Yersinia enterocolitica*. The rheumatic disease, Reiter's syndrome (associated with HLA-B27), can result from prior exposure to *Salmonella*, *Shigella*, or *Yersinia*. Other organisms that are not members of the *Enterobacteriaceae*, including *Campylobacter* and *Chlamydia*, are also causative agents of Reiter's syndrome. *Yersinia pestis* (the cause of "plague") will be considered separately with other **zoonotic** organisms.

Members of this family are major causes of opportunistic infection (including septicemia, pneumonia, meningitis and urinary tract infections). Examples of genera that cause opportunistic infections are: *Citrobacter*, *Enterobacter*, *Escherichia*, *Hafnia*, *Morganella*, *Providencia* and *Serratia*. Selection of antibiotic therapy is complex due to the diversity of organisms.

Some of the organisms additionally cause community-acquired disease in otherwise healthy people. *Klebsiella pneumoniae* is often involved in respiratory infections. The organism has a prominent capsule aiding pathogenicity. The commonest community acquired ("ascending") urinary tract infection is caused by *E. coli*. The vast majority of urinary tract infections are ascending, often from fecal contamination. *Proteus* is another common cause of urinary tract infection; the organism produces a **urease** that degrades urea producing an alkaline urine.

Reiter's  
Syndrome

### Isolation and identification of *Enterobacteriaceae*

*Enterobacteriaceae* are Gram-negative facultative anaerobic rods. They lack cytochrome oxidase and are referred to as oxidase-negative. They are often isolated from fecal matter on agar containing lactose and a pH indicator. Colonies that ferment lactose will produce sufficient acid to cause a color shift in the indicator (Figure 1). *Escherichia coli* is a fermenter of lactose, while *Shigella*, *Salmonella* and *Yersinia* are non-fermenters. "Non-pathogenic" strains of *E. coli* (and other lactose-positive enterics) are often present in normal feces. Since they are difficult to differentiate from "pathogenic" *E. coli*, lactose-negative colonies are often the only ones identified in feces. All *Enterobacteriaceae* isolated from other sites (which contain low numbers of bacteria (e.g. urine) or are normally sterile (e.g. blood)) are identified biochemically, for example using the API 20E system. Important serotypes can be differentiated by their O (lipopolysaccharide), H (flagellar) and K (capsular) antigens. However, **serotyping** is generally not performed in the routine clinical laboratory.

### KEY WORDS

Opportunistic Gastroenteritis  
Diarrhea  
Dysentery  
Urinary tract infections  
Lactose positive/negative  
API strip  
Enteropathogenic *E. coli*  
Enterotoxigenic *E. coli*  
Heat stable toxin  
Heat labile toxin  
Enteroinvasive *E. coli*  
Enterohemorrhagic *E. coli*  
Vero toxin (Shiga-like)  
Hemolysin  
Adhesive pili  
*Shigella*  
Bacillary dysentery  
Shiga toxin  
*Salmonella typhi*  
Typhoid  
Vi antigen  
*Salmonella enteritidis*  
(salmonellosis)  
*Salmonella cholerae-suis*  
*Vibrio cholerae*  
Cholera  
Cholera toxin  
*Yersinia enterocolitica*  
*Campylobacter jejuni*  
*Helicobacter pylori*



Figure 1A  
Reactions in TSI agar slants. For more information on this figure, please go [here](#). © Neal R. Chamberlain, Kirksville College of Osteopathic Medicine, Kirksville, MO and The MicrobeLibrary



Figure 1B Nonlactose fermenter on Hektoen agar which contains bile salts and acid indicators (bromthymol blue and acid fuchsin). The gram-positive bacteria are inhibited so the agar is selective for gram-negative bacteria. The lactose fermenters form orange colonies while the nonfermenters appear green to blue-green. This is especially helpful in distinguishing potential pathogens from normal flora in stool specimens. However, it is difficult to tell the non-fermenters from each other. The organism on this plate could be *Salmonella*, *Proteus*, or *Shigella*. © Pat Johnson, Palm Beach Community College, Lake Worth, Florida and The MicrobeLibrary



Figure 1B Growth of a nonlactose fermenter on MacConkey agar which contains bile salts and crystal violet which inhibit the growth of gram-positive bacteria. The agar also contains lactose and a red dye that differentiates the lactose fermenters from the non-fermenters. Colonies of lactose fermenting bacteria are pink to red while the nonfermenters are colorless or transparent. This agar does not distinguish between the non-lactose fermenters; this growth could indicate several organisms - *Proteus*, *Salmonella* or *Shigella*, for example. In a stool specimen, it would be enough evidence to continue with further identification. © Pat Johnson, Palm Beach Community College, Lake Worth, Florida and The MicrobeLibrary



Figure 1C Growth of gram-negative bacteria that cannot ferment lactose on eosin methylene blue (EMB) agar which contains bile salts and dyes which inhibit growth of gram-positive bacteria. Growth on EMB agar is a useful diagnostic tool to distinguish between lactose fermenters and non-fermenters which will appear colorless. *Salmonella* and *Shigella*, both non-lactose fermenting pathogens, can be distinguished from the more common intestinal flora which ferment lactose. © Pat Johnson, Palm Beach Community College, Lake Worth, Florida and The MicrobeLibrary

## GASTROENTERITIS, DIARRHEA AND DYSENTERY

### *Escherichia coli*

*E. coli* (figure 3) live in the human gut and are usually harmless but some are pathogenic causing diarrhea and other symptoms as a result of ingestion of contaminated food or water.

At the species level, *E. coli* and *Shigella* are indistinguishable. For practical reasons (primarily to avoid confusion), they are not placed in the same genus. Not surprisingly there is a lot of overlap between diseases caused by the two organisms.



Figure 2  
Colorized scanning electron micrograph. Gram-negative *Escherichia coli* bacteria of the strain O157:H7 6836x. CDC



Figure 3 *E. coli* (O157:H7) hemorrhagic type. Gram-negative, enteric, facultatively anaerobic, rod prokaryote. Potentially fatal to humans, contracted when contaminated meat is cooked inadequately. © Dennis Kunkel Microscopy, Inc. Used with permission

1) Enteropathogenic *E. coli* (EPEC). Certain serotypes are commonly found associated with infant diarrhea. The use of gene probes has confirmed these strains as different from other groups listed below. There is a characteristic morphological lesion with destruction of microvilli without invasion of the organism which suggests adhesion is important. Clinically, one observes:

- fever
- diarrhea
- vomiting
- nausea usually with non-bloody stools

2) Enterotoxigenic *E. coli* (ETEC) produce diarrhea resembling cholera but much milder in degree. They also cause "travelers' diarrhea". Two types of plasmid-encoded toxins are produced.

- Heat labile toxins which are similar to cholera toxin (see cholera section below). Adenyl cyclase is activated with production of cyclic AMP and increased secretion of water and ions.
- Heat stable toxins. Guanylate cyclase is activated which inhibits ionic uptake from the gut lumen. Watery diarrhea, fever and nausea result in both cases.

3) Enteroinvasive *E. coli* (EIEC) produce a dysentery (indistinguishable clinically from shigellosis, see bacillary dysentery below).

4) Enterohemorrhagic *E. coli* (EHEC). These are usually serotype O157:H7 (figure 2, 3, 4a). Other kinds of EHEC are sometimes called "non-O157 EHEC". *E. coli* serogroups O26, O111, and O103 are those that most often cause illness in people in the United States. Most non-O157 EHECs cause less severe disease than O157:H7 but a few can cause more severe symptoms. Very often, non-O157 EHECs are not identified and much less is known about them.

These organisms can produce a hemorrhagic colitis (characterized by bloody and copious diarrhea with few leukocytes in **afebrile** patients). However, they are taking on increasing importance (figure 4b) with the recognition of outbreaks caused by contaminated hamburger meat. The organisms can

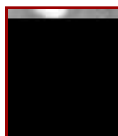


Figure 4A  
Transmission electron micrograph of Escherichia coli O157:H7 CDC/Peggy S. Hayes psh1@cdc.gov



Figure 4B  
Chronology of E. coli O157:H7 infections, an emerging type of foodborne illness. CDC

disseminate into the bloodstream producing systemic hemolytic-uremic syndrome (hemolytic anemia, thrombocytopenia and kidney failure) which is often fatal. Around 5–10% of those who are diagnosed with EHEC infection develop a potentially life-threatening hemolytic uremic syndrome.

Production of Vero toxin (biochemically similar to Shiga toxin - thus also known as "Shiga-like") is highly associated with this group of organisms. The toxin is encoded by a lysogenic phage. Hemolysins (plasmid-encoded) are also important in pathogenesis.

Since these bacteria make Shiga-like toxins, they are often called "Shiga toxin-producing" *E. coli*, or STEC for short. They are also called Verocytotoxic *E. coli* (VTEC); these all refer to the same group of bacteria.

As noted above, there are at least four etiologically distinct diseases. However, in the diagnostic laboratory, the groups are not generally differentiated and treatment is based on symptomatology. Usually, fluid replacement is the primary treatment. Antibiotics are generally not used except in severe disease or disease that has progressed to a systemic stage (e.g. hemolytic-uremia syndrome).

Two major classes of pili are produced by *E. coli*: mannose-sensitive and mannose-resistant pili. The former bind to mannose containing glycoproteins and the latter to cerebrosides on the host epithelium, allowing attachment. This aids in colonization by *E. coli*.

**CASE REPORT**

Multistate Outbreak of *E. coli* O157:H7 Infections Associated with Lebanon Bologna

How does a patient become infected by an EHEC?	How can EHEC infections be prevented?
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**Shigella**

There are about 14,000 reported cases of shigellosis in the United States each year but because many milder cases are not diagnosed, the actual number of infections is thought to be at least twenty times greater. Shigellosis is particularly common and causes recurrent problems in settings with poor hygiene where epidemics can occur. It is diagnosed more often in summer than winter with children between the ages of 2 to 4 years being the most prone to infection. Often the disease is seen in child care facilities and many cases are the result of the spread of the illness in families with small children. *Shigella* pass from an infected person to another as they are present in the diarrheal stools. Stools can be infectious while the patient is sick and for up to two weeks after. Most *Shigella* infections are the result of the bacterium passing from stools or soiled fingers of one person to the mouth of another person. Thus, hygiene is important in containing an outbreak.

In the developing world, shigellosis is far more common and is present in most communities most of the time.

*Shigella* (4 species; *S. flexneri*, *S. boydii*, *S. sonnei*, *S. dysenteriae* (figure 5)) all cause bacillary dysentery or shigellosis, (bloody feces associated with intestinal pain). The organism invades the epithelial lining layer but does not penetrate. Usually within 2 to 3 days, dysentery results from bacteria damaging the epithelial layers lining the intestine, often with release of mucus and blood (found in the feces) and attraction of leukocytes (also found in the feces as "pus"). However, watery diarrhea is frequently observed with no evidence of dysentery. Shiga toxin (chromosomally-encoded), which is **neurotoxic**, **enterotoxic** and **cytotoxic**, plays a role. Its enterotoxicity can make the disease clinically appear as a diarrhea. The toxin inhibits protein synthesis (acting on the 70S ribosome and lysing 28S rRNA). This is primarily a disease of young children occurring by fecal-oral contact. Adults can catch this disease from children, although it can be transmitted by infected adult food handlers who contaminate food. The source in each case is unwashed hands. Man is the only "reservoir".

Managing of dehydration is of primary concern. Indeed, mild diarrhea is often not recognized as shigellosis. Patients with severe dysentery are usually treated with antibiotics (e.g. ampicillin). In contrast to salmonellosis, patients respond to antibiotic therapy and disease duration is diminished.

Long term consequences of a Shigella infection	Incidence of Shigella infections
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Figure 5. *Shigella dysenteriae* - Gram-negative, enteric, facultatively anaerobic, rod prokaryote; causes bacterial dysentery. This species is most often found in water contaminated with human feces. © Dennis Kunkel Microscopy, Inc. Used with permission

**Salmonella**

It is estimated that *Salmonella* cause more than 1.2 million illnesses each year in the United States, resulting in more than 23,000 hospitalizations and 450 deaths. The overall rate of Salmonellosis is falling in the United States although outbreaks periodically occur (figure 6b).



Figure 6a.

Salmonella - rod prokaryote (dividing); note the flagella. Causes salmonellosis (food poisoning). (x

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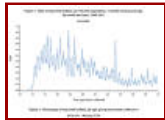


Figure 6b

Rate of reported Salmonella isolates in US per 100,000 population, 3-month moving average by month and year 1968-2011 CDC



Figure 7a

Computer-generated image of five drug-resistant *Salmonella typhi* bacteria based upon scanning electron micrographic image. Note the presence of numerous thin, short fimbriae emanating from the organisms' cell wall, imparting a furry appearance to these bacteria, and the multiple peritrichous flagella.

CDC/Melissa Brower



Figure 7b

Rose spots on the chest of a patient with typhoid fever due to the bacterium *Salmonella typhi*.

CDC/ Armed Forces Institute of Pathology, Charles N. Farmer

*Salmonella* infections most often cause vomiting or diarrhea, sometimes severe. In rare cases, Salmonella illness can lead to severe and life-threatening bloodstream infections.

Based on genetic studies, there is a single species of *Salmonella* (*Salmonella enterica*) (figure 6a). At the other extreme using appropriate antibodies, more than 2000 antigenic "types" have been recognized. There are, however, only a few types that are commonly associated with characteristic human diseases (most simply referred to as *Salmonella enteritidis*, *Salmonella cholerae-suis* and *Salmonella typhi*).

### Salmonellosis

Salmonellosis, the common salmonella infection, is caused by a variety of serotypes (most commonly *S. enteritidis*) and is transmitted from contaminated food (such as poultry and eggs). It does not have a human reservoir and usually presents as a gastroenteritis (nausea, vomiting and non-bloody stools). The disease is usually self-limiting (2 - 5 days). Like *Shigella*, these organisms invade the epithelium and do not produce systemic infection. In uncomplicated cases of salmonellosis, which are the vast majority, antibiotic therapy is not useful. *S. cholerae-suis* (seen much less commonly) causes septicemia after invasion. In this case, antibiotic therapy is required.

### Typhoid

The severest form of salmonella infections, "typhoid" (enteric fever), caused by *Salmonella typhi* (figure 7a), is not often seen in the United States, although it is one of the historical causes of widespread epidemics and still is in the third world. It is estimated that about 5,700 cases occur annually in the United States. Most cases (up to 75%) are acquired while traveling internationally. Typhoid fever affects about 21.5 million persons each year in the developing world.

The organism is transmitted from a human reservoir or in the water supply (if sanitary conditions are poor) or in contaminated food. It initially invades the intestinal epithelium and, during this acute phase, gastrointestinal symptoms are noted. The organisms penetrate (usually within the first week) and pass into the bloodstream where it is disseminated in macrophages. Symptoms of typhoid include a fever up to 103° to 104° F (39° to 40° C). The patient may also feel weak and have stomach pains, headache, and/or loss of appetite. In some cases, patients have a rash of flat, rose-colored spots (figure 7b). Diagnosis of typhoid fever is carried out from stool or blood samples that are tested for the presence of *Salmonella typhi*.

Typical features of a systemic bacterial infection are seen. The septicemia usually is temporary with the organism finally lodging in the gall bladder. Organisms are shed into the intestine for some weeks. At this time, gastroenteritis (including diarrhea) is noted again. The Vi (capsular) antigen plays a role in the pathogenesis of typhoid. A carrier state is common; thus one person (e.g. a food handler) can cause a lot of spread. Antibiotic therapy is essential. Unfortunately, there is increasing resistance to antibiotics, including fluoroquinolones and, as a result there may be increases in case-fatality rates. Epidemics and high endemic disease rates occur in the Central Asian Republics, the Indian subcontinent, and across Asia and the Pacific Islands.

Most people in the United States are not vaccinated against typhoid but those traveling to a country where typhoid is common, should consider being vaccinated against typhoid. There are two vaccines available: Ty21a, taken orally in a capsule and ViCPS, taken by injection. Both need boosters after a number of years.

[More on typhoid](#)



Figure 7c

*Yersinia enterocolitica* - Gram-negative, facultatively anaerobic, rod prokaryote (dividing). This bacterium releases a toxin that causes enteritis with pain resembling appendicitis. © Dennis Kunkel Microscopy, Inc. Used with permission

### Yersinia enterocolitica

*Yersinia enterocolitica* (figure 7c) infection (Yersiniosis) is a major cause of gastroenteritis (the main clinical symptom) in Scandinavia and elsewhere and is seen in the United States. The organisms are invasive (usually without systemic spread). Typically the infection is characterized by diarrhea, fever and abdominal pain.

*Y. enterocolitica* infections are seen most often in young children in whom symptoms include:

- fever
- abdominal pain
- diarrhea, which is often bloody.

These symptoms usually develop 4 to 7 days after infection and can last for one to three weeks or more. In older children and adults predominant symptoms include:

- right-sided abdominal pain (this may lead to confusion with appendicitis)
- fever

In a few cases, complications including skin rash, joint pains, or **bacteremia** can occur.

Uncomplicated cases of diarrhea due to *Y. enterocolitica* usually resolve without antibiotics but in more severe or complicated infections the use of antibiotics such as aminoglycosides, doxycycline, trimethoprim-sulfamethoxazole, or fluoroquinolones is recommended.

*Y. enterocolitica* can be transmitted by fecal contamination of water or milk by domestic animals or from eating meat products. It is best isolated by "cold" enrichment: when refrigerated this organism survives while others do not.

### ***Yersinia pseudotuberculosis***

A similar, but less severe, disease is caused by *Y. pseudotuberculosis*. The disease is characterized by

- fever
- acute abdominal pain due to **mesenteric lymphadenitis** that mimics appendicitis.

Secondary symptoms include

- **erythema nodosum**
- **reactive arthritis** (Reiter's Syndrome)

Outbreaks have been reported in Canada, Japan, Finland and Russia (among others). Only in a few of the outbreaks has the vector or source of the infection been identified. Unwashed vegetables including iceberg lettuce and carrots have been implicated by epidemiologic investigations as a source of infection; however, the source of the contamination has not been identified.

## **VIBRIO SPECIES**

Several species of vibrio are known to cause human disease and there are an estimated 80,000 illnesses, 500 hospitalizations and 100 deaths each year in the United States.

### ***Vibrio cholerae***

These are Gram-negative rods. They are comma shaped, facultative anaerobes which are oxidase positive. The most important vibrio, *Vibrio cholerae* (figure 8), is the causative agent of cholera. It has simple nutritional requirements and is readily cultivated. *V. cholerae* is found in the feces of an infected individual and ends up in the water supply if sewage is untreated. The organism is thus transmitted by drinking contaminated water. The organism survives in fresh water and, like other vibrios, in salt water. Food, after water contamination, is another means of transmission. Thus, it is primarily a disease of the third world. In the United States, it is observed in the occasional international traveler (especially to parts of Africa, Southeast Asia, or Haiti), although it is sometimes seen after ingestion of seafood. Once in the gut, the organism adheres to the epithelium of the intestine without penetration. Adhesion to the microvilli is thus important in pathogenesis. Cholera toxin is then secreted.

Cholera (cholera toxin) is chromosomally encoded and contains two types of subunit (A and B). The B subunit binds to **gangliosides** on epithelial cell surfaces allowing internalization of the A subunit. B subunits may provide a hydrophobic channel through which A penetrates. The A subunit catalyses ADP-ribosylation of a regulator complex which in turn activates adenylate cyclase present in the cell membrane of the epithelium of the gut. The overproduction of cyclic AMP in turn stimulates massive secretion of ions and water into the lumen. Dehydration and death (without treatment) result. Thus, fluid replacement is the major component of treatment. Antibiotic therapy (including tetracycline) is additionally used. Vaccination is only partially effective and not generally recommended. It is most commonly used by international travelers.

Cholera is usually an acute, diarrheal illness. It is often mild or asymptomatic, but sometimes it can be more severe. About 5-10% of infected patients develop severe cholera, the early symptoms of which include (CDC):

- profuse watery diarrhea, sometimes described as "rice-water stools,"
- vomiting
- rapid heart rate
- loss of skin elasticity
- dry mucous membranes
- low blood pressure

- thirst
- muscle cramps
- restlessness or irritability

This can lead to:

- acute renal failure
- severe electrolyte imbalances
- coma

If untreated, severe dehydration can rapidly lead to shock and death.

As noted above, diarrhea from people with cholera contains large amounts of infectious bacteria that can contaminate the environment (such as water supplies or food) and infect others, if ingested, thereby spreading the disease. Improved sanitary conditions can prevent the spread of cholera. Washing hands after touching anything that might be contaminated and properly disposing of contaminated items and human waste is essential.

In severe cases of cholera, CDC recommends:

- Oral or intravenous hydration is the mainstay of cholera treatment
- In conjunction with hydration, treatment with antibiotics is recommended for severely ill patients. It is particularly recommended for patients who are severely or moderately dehydrated and continue to pass a large volume of stool during rehydration treatment. Antibiotic treatment is also recommended for all patients who are hospitalized.
- Antibiotic choices should be informed by local antibiotic susceptibility patterns. In most countries, Doxycycline is recommended as first-line treatment for adults, while azithromycin is recommended as first-line treatment for children and pregnant women. During an epidemic or outbreak, antibiotic susceptibility should be monitored through regular testing of sample isolates from various geographic areas.
- None of the guidelines recommend antibiotics as prophylaxis for cholera prevention, and all emphasize that antibiotics should be used in conjunction with aggressive hydration.
- Education of health care workers, assurance of adequate supplies, and monitoring of practices are all important for appropriate dispensation of antibiotics.

### ***Vibrio parahaemolyticus***

*Vibrio parahaemolyticus* (figure 9) is the agent that causes vibriosis and is usually transmitted by ingestion of raw seafood (especially oysters). An estimated 4,500 cases of vibriosis occur each year in the United States. The organism lives in brackish saltwater and causes gastrointestinal illness in humans. *Vibrio parahaemolyticus* inhabits coastal waters in the United States and Canada and is present in higher concentrations during summer; it grows best in high concentrations of salt (i.e. it is halophilic). A non-bloody diarrhea is observed but it is not as severe as cholera.

There was an increase in *Vibrio parahaemolyticus* illnesses associated with consumption of shellfish from several Atlantic coast harvest areas in the United States in 2013 (figure 9).

The symptoms of vibriosis are (CDC):

- watery diarrhea
- frequently with abdominal cramping
- nausea
- vomiting
- fever and chills

Usually these symptoms occur within 24 hours of ingestion of the bacterium. The disease is usually self-limiting and lasts 3 days. Severe disease is rare and occurs more commonly in persons with weakened immune systems. *V. parahaemolyticus* can also cause an infection of the skin when an open wound is exposed to warm seawater.

Diagnosis is by isolation of the bacterium from cultures of stool, wound, or blood. For isolation from stool, the use of a selective medium that has thiosulfate, citrate, bile salts, and sucrose (TCBS agar) is recommended by CDC.

Usually treatment is not necessary and there is no evidence that antibiotic treatment decreases the severity or the length of the illness. The patient should be encouraged to drink plenty of water to replace fluids lost through diarrhea. In severe or prolonged illnesses, antibiotics such as tetracycline or ciprofloxacin can be used.

### ***Vibrio vulnificus***

*Vibrio vulnificus* (figure 9d) is another salt loving (halophilic) bacterium that causes cases of vibriosis and again disease is caused by eating contaminated raw seafood or exposure of an open wound to contaminated sea water. In the latter case, infection can lead to skin breakdown and ulceration. Between 1988 and 2006, CDC received reports of more than nine hundred *V. vulnificus* infections from the Gulf Coast states, where most cases occur.

Infection by *V. vulnificus* can cause, in non-immunocompromised people:

- vomiting
- diarrhea
- abdominal pain

In immunocompromised people, especially those with chronic liver disease, *V. vulnificus* can infect the bloodstream (bacteremia), resulting in a severe disease that can be life-threatening. It is characterized by:

- fever and chills
- decreased blood pressure (septic shock)
- blistering skin lesions

About half of *V. vulnificus* bloodstream infections result in death.

Diagnosis is by stool, wound, or blood cultures.

If *V. vulnificus* is suspected, treatment should be initiated immediately because antibiotics improve survival with aggressive treatment of the wound site; debridement of infected necrotic tissue or amputation of the infected limb is sometimes necessary. Doxycycline and cephalosporin are indicated or a fluoroquinolone such as levofloxacin, ciprofloxacin or gatifloxacin. Children, in whom doxycycline and fluoroquinolones are contraindicated, can be treated with trimethoprim-sulfamethoxazole plus an aminoglycoside.



Figure 8a

*Vibrio cholerae*. Leifson flagella stain (digitally colorized). CDC/Dr. William A. Clark



Figure 8b

*Vibrio cholerae* - Gram-negative, facultatively anaerobic, curved (vibrio-shaped), rod prokaryote; causes Asiatic cholera. © Dennis Kunkel Microscopy, Inc. Used with permission



Figure 9a

*Vibrio parahaemolyticus* - halophilic, facultative anaerobic, rod bacterium that causes a food-borne illness known as seafood poisoning. Usually transmitted through eating raw or undercooked seafood such as oysters. Less commonly, this organism can cause an infection in the skin when an open wound is exposed to warm seawater. © Dennis Kunkel Microscopy, Inc. Used with permission



Figure 9b

*Vibrio parahaemolyticus* - halophilic, facultative anaerobic, rod bacterium that causes a food-borne illness known as seafood poisoning. Usually transmitted through eating raw or undercooked seafood such as oysters. Less commonly, this organism can cause an infection in the skin when an open wound is exposed to warm seawater. © Dennis Kunkel Microscopy, Inc. Used with permission



Figure 9c

Increase in *Vibrio parahaemolyticus* illnesses associated with consumption of shellfish from several Atlantic coast harvest areas, United States, 2013



Figure 9d

Scanning electron micrograph of *Vibrio vulnificus* bacteria; Mag. 13184x CDC

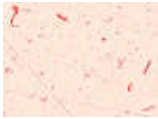


Figure 10a.

Campylobacter fetus. Leifson flagella stain (digitally colored). CDC/Dr. William A. Clark

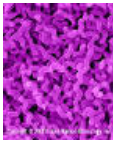


Figure 10b

*Campylobacter jejuni* is an enteric, curved-rod prokaryote (bacterium). It is the bacterium that causes campylobacteriosis, one of the most common bacterial causes of diarrheal illness in the United States. It is a relatively fragile bacterium that is easily killed by cold or hot temperatures. Birds are carriers due to their body temperature being just right to host the bacteria. Improper handling of raw poultry or undercooked fowl is usually the source of infection in humans. © Dennis Kunkel Microscopy, Inc. Used with permission

## CAMPYLOBACTER AND HELICOBACTER

These two groups of Gram-negative organisms are both curved or spiral shaped and are genetically related.

### *Campylobacter jejuni*

Campylobacteriosis is one of the commonest bacterial disease causing diarrhea in the United States. There are approximately 14 cases each year per 100,000 population. However, many cases are not diagnosed and it is estimated that there are over 1.3 million cases annually. Infections occur much more frequently in the summer than in winter and the disease occurs in infants and young adults more often than in older people. It is more often seen in males than females. Campylobacteriosis is rarely fatal but there are approximately 76 deaths in the United States among persons with *Campylobacter* infections each year.

The most common of the *Campylobacter* (figure 10) causing human disease are *C. jejuni*. The organism infects the intestinal tract of several animal species (including cattle and sheep) and is a major cause of cause of abortions. It is transmitted to man in milk and meat products. Watery diarrhea predominates but dysentery is common. The organism is invasive but generally less so than *Shigella*. Malaise, fever and abdominal pain are other disease features. Bacteremia is observed in a small minority of cases.

*Campylobacter* infection is diagnosed when a culture of a stool specimen yields the bacterium. The organism is **microaerophilic** and grows best at 42°C. It is frequently isolated under these conditions using selective media. It can be treated with antibiotics but is usually a self-limiting disease. Patients should drink extra fluids as long as the diarrhea lasts. Antibiotics are only used to treat patients with severe disease or those at high risk for severe disease. These include patients with immune systems severely weakened from medications or other illnesses. Azithromycin and fluoroquinolones (e.g., ciprofloxacin) are commonly used for treatment of these infections, but resistance to fluoroquinolones is common.

Campylobacteriosis can sometimes have long-term sequelae. These include:

- arthritis.
- Guillain-Barré syndrome. This is a rare disease that affects the nerves of the body beginning several weeks after the diarrheal illness and results from an attack on body's nervous system by the immune system and can result in temporary paralysis for several weeks. It requires intensive medical care. It is estimated that about one in every 1,000 *Campylobacter* illnesses leads to Guillain-Barré syndrome. As many as 40% of Guillain-Barré syndrome cases in the United States may result from campylobacteriosis.

Guillain-Barré  
Syndrome

### *Helicobacter pylori*



*Helicobacter pylori* (figure 11) has been accepted in the last few years as the major cause of stomach ulcers. The organism chronically lives in and on the stomach mucosa of man. Culture is the preferred method of diagnosis but may miss a number of cases. The organism characteristically produces a urease which generates ammonia and carbon dioxide. This aids in detecting and identifying the isolated organism. Urease is produced in such large amounts that it can be directly detected in mucosa sampled after endoscopy. Alternatively,  $^{13}\text{C}$  or  $^{14}\text{C}$  labeled  $\text{CO}_2$  is detected in the breath after feeding labeled urea. Production of ammonia is a factor in pathogenesis (in locally neutralizing stomach acid). Antibiotic therapy eliminates the organism, peptic ulcers heal and relapses are generally avoided.

## CONCLUSION

Sanitary measures protect the water supply, avoiding contamination with sewage. This is the primary reason that epidemics with life-threatening pathogens (e.g. cholera and typhoid) are rarely seen in western countries but are commonly seen in the third world. Other less severe diseases (e.g. salmonellosis, EHEC) are still common from eating contaminated animal products, which has been less well controlled. *Shigella*, which has a human host, would be even more difficult to eradicate. Vaccination is rarely used and, indeed, is an expensive way to go compared to sewage treatment. In severe diarrhea, fluid replacement is essential. Antibiotic therapy is used in severe local infection and always in systemic disease.



Figure 10c

*Campylobacter jejuni* - Gram-negative, enteric, curved (vibrio-shaped), rod prokaryote. Found in the gastrointestinal tract of humans and animals, it can travel to the oral cavity and genitourinary tract. Causes gastroenteritis, especially in infants. © Dennis Kunkel Microscopy, Inc. Used with permission

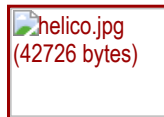


Figure 11a

*Helicobacter pylori* electron micrographs; fastidious microaerophile; typical helical shape shown in EM; causative agent of chronic gastritis, peptic ulcers and gastric cancer. Image can be used to describe the helical morphology of the organism. Average size: 1micron by 2-5 microns. Organism is in log phase of growth. © Cindy R. DeLoney, Loyola University of Chicago, Chicago, Illinois and [The MicrobeLibrary](#)

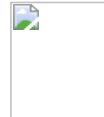


Figure 11b

*Helicobacter pylori* - Gram-negative, spiral to pleomorphic, spiral rod prokaryote. It can move by means of tiny flagella at the end of the cell. There are many strains of *H. pylori* which are distinguished by the human disease with which they cause. *H. pylori* infection is the main cause of chronic superficial gastritis and it is associated with both gastric and duodenal ulcers. It lives in the interface between the surface of gastric epithelial cells (the lining of the stomach). It often clusters at the junctions of epithelial cells. © Dennis Kunkel Microscopy, Inc. Used with permission



Figure 11c

*Helicobacter pylori* - Gram-negative, spiral to pleomorphic, spiral rod prokaryote. © Dennis Kunkel Microscopy, Inc. Used with permission



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