List and Characteristics of Enteric Bacteria

The enteric species of bacteria are gram-negative rods inhabit the gastrointestinal tract. Some species are part of the normal flora that incidentally cause illness while some are regularly pathogenic. Physicians need to familiarize themselves with all members of this bacterial group and their treatment strategies.

Secretory (Watery) Diarrhea

*Vibrio* and *Enterobacteriaceae* are two of the common bacterial genus that causes watery diarrhea. The route of infection is often by ingestion of contaminated food and drink. Broadly speaking, they cause diarrhea by attaching to the epithelial lining of the intestinal lumen and secrete a toxin that decreases sodium absorption and increased chlorine secretion. These changes across the cell membrane lead to water leaving the cells, leading to diarrhea.

*Vibrio Cholera*
Morphology & Culture

*V. cholera* is a curved Gram-negative rod that has a single polar flagellum. It is an oxidase positive organism that grows well in thiosulfate-citrate-bile-sucrose (TCBS) medium. *V. cholera* prefers alkaline to acidic environments.

Location

This bacterium is located in coastal estuary waters. Infection occurs when human enter the environment, or the organisms contaminate drinking water or food.

Virulence Factors

There are several virulence factors that make *V. cholera* pathogenic:

- **Choleragen** is the toxin secreted by *V. cholera*. It is composed of one A unit accompanied by five B units. The A unit is the active unit. Once in the cell, the toxin upregulates cAMP leading to secretion of chlorine and limits sodium reabsorption.
- **O antigen** is the most external component of the gram-negative LPS and confirms antigenicity.
- **The H antigen** is part of the subunit of the flagella and is present in this species.
- *V. cholera* has **fimbrie** that helps the bacterium attach to the intestinal lumen and deliver its toxin

Clinical Picture

Patients with cholera present with copious diarrhea. In some cases, it has been reported that patients will lose up to one liter of diarrhea an hour. The diarrhea is described as being **rice-water** in color. Patients will present with dehydration symptoms secondary to this infection. They will have poor skin turgor and diminished capillary refill.

Treatment

Cholera is a self-limiting disease. Patients will eventually expel the bacteria through their diarrhea. However, death from dehydration is a real risk for patients in under developed
countries. Treatment is replenishment of the fluid loss via oral rehydration. Tetracycline antibiotics can be used to shorten the course of exposure.

**Enterotoxigenic *Escherichia coli* (ETEC)**

![Low-temperature electron micrograph of a cluster of E. coli bacteria, magnified 10,000 times.](image)

**Morphology & Culture**

*E. coli* is a Gram-negative rod. It is motile, ferments lactose, and is catalase positive. *E.coli* will grow pink colonies on **MacConkey’s agar** and as metallic, green colonies on **Eosin-Methylene Blue (EMB) agar**.

**Location**

Pathogenic *E. coli* are found exogenously, most likely in the feces of cow manure that contaminates a water supply.

**Virulence Factors**

*E. coli* has many virulence factors that increase its pathogenicity.

- The **heat liable toxin (LT)** can infiltrate the GI epithelium and increase cAMP, causing diarrhea
- The **heat stable toxin (ST)** can also infiltrate the GI epithelium, but will upregulate cGMP to cause secretory diarrhea.
- **O antigen** is the most external component of the Gram-negative LPS and confirms antigenicity.
- **H antigen** is part of the subunit of the flagella and is present in this species.
- **K antigen** is part of the capsule and is present in this species.
- *E. coli* has fimbrie that helps the bacterium attach to the intestinal lumen and deliver its toxin

**Clinical Picture**

ETEC is the number one cause of travelers’ diarrhea in the world. The bacteria use fimbre to attach to the enterocyte and produce a toxin that leads to secretory diarrhea. Similar to *V. cholera*, diarrhea from ETEC is rice-colored. Patients can lose up to twenty liters of
fluid a day with this bacterium.

Treatment

Like the other non-invasive enterics, ETEC is self-limiting and will be dispelled from the body via diarrhea. Treatment involves adequate oral hydration to replace the lost fluid. Tetracyclines can be used in some cases to limit the duration of infection.

Enteropathogenic *Escherichia coli* (EPEC)

Morphology & Culture

*E. coli* is a gram-negative rod. It is motile, ferments lactose and is catalase positive. *E.coli* will grow pink colonies on *MacConkey’s agar* and as metallic, green colonies on *Eosin-Methylene Blue (EMB)* agar.

Location

Pathogenic *E. coli* are found exogenously, most likely in the feces of cow manure that contaminates a water supply.

Virulence Factors

Unlike other forms of *E. coli*, EPEC does not have a toxin. The bacterium uses an intimin adhesion to bind to the enterocytes and alter the apical edge of the enterocyte. EPEC does have *O, K, and H antigen*, similar to the other forms of the *Escherichia* family.

Clinical Picture

EPEC is a major cause of watery diarrhea in children in developing countries. The bacteria will be ingested and adhere to the enterocyte and flatten the microvilli of the cell. This will alter the absorption and secretion of sodium and chlorine and cause secretory diarrhea.

Treatment

Like the other non-invasive enterics, EPEC is self-limiting and will be dispelled from the body via diarrhea. Treatment involves adequate oral hydration to replace the lost fluid. Tetracyclines can be used in some cases to limit the duration of infection.

Enteroaggregative *Escherichia coli* (EAEC)

Morphology & Culture

*E. coli* is a gram-negative rod. It is motile, ferments lactose and is catalase positive. *E.coli* will grow pink colonies on *MacConkey’s agar* and as metallic, green colonies on *Eosin-Methylene Blue (EMB)* agar.

Location

Pathogenic *E. coli* are found exogenously, most likely in the feces of cow manure that contaminates a water supply.
contaminates a water supply.

Virulence Factors

EAEC has several virulence factors that cause it to be pathogenic:

- EAEC secretes a heat stable (ST), similar to ETEC that upregulates cGMP which led to chlorine secretion and diarrhea.
- **Fimbriae** aid EAEC in attaching to enterocytes.
- **EAEC** also produces hemolysin, which destroys red blood cells.
- This bacterium is positive for **O, K, and H antigens**.

Clinical Picture

EAEC is a cause of travelers’ diarrhea and can effect immunocompromised patients. EAEC results in both acute and chronic watery diarrhea in HIV and AIDS patients. EAEC uses either hemolysin or ST-like toxin to alter the electrolyte transport mechanisms of the cell, leading to diarrhea.

Treatment

Like the other non-invasive enterics, EAEC is self-limiting and will be dispelled from the body via diarrhea. Treatment involves adequate oral hydration to replace the lost fluid. Tetracyclines can be used in some cases to limit the duration of infection.

Invasive Enteric Bacteria: Bloody Diarrhea and Dysentery

Unlike the bacteria that result in watery diarrhea, the invasive enteric bacteria will invade the enterocytes, causing cellular damage. The invasive bacteria will reach the bloodstream. Dysentery is the inflammation of the intestine that is accompanied by bloody diarrhea. All of these infections need to be treated with **antibiotics** because this enterics are not self-limiting.

*Shigella*
This photomicrograph revealed stool exudates in a patient with shigellosis, which is also known as “Shigella dysentery”, or “Bacterial dysentery.”

Morphology & Culture

*Shigella* are slender gram-negative rods that do not ferment lactose, are oxidase negative, and do not have a flagellum. There are four different species in this family, *(S. dysenteriae, S. flexneri, S. sonni, and S. boydii)* and all exhibit similar characteristics.

Location

Humans are the hosts of this bacteria. It is often transferred via the fecal to oral route. *Shigella* is never considered a normal part of the human flora.

Virulence Factors

There are some virulence factors that make *Shigella* pathogenic:

- The **Shiga toxin** is similar to ST and LT. It has two subunits, A and B. The A subunit is active and once inside the cell, it will inactivate the 60S ribosome, inhibiting protein synthesis, killing the cell.
- The Shigella species gains entry into the enterocytes via induced phagocytosis through the M cells. The bacterium will get phagocytized and escape from the vacuole and release its toxin in the cell.
- Shigella has an **O antigen**.

Clinical Picture

After incubation for one day, patients will begin to report fever, abdominal pain, and watery diarrhea. After a day, the patient will have less frequent stools, but they will contain mucus and blood. Due to the destruction of the cells via the Shiga toxin, patients can also develop an ulcer in the intestine. Symptoms will resolve in two to five day in
adults, but can persist in young children and the elderly. Due to the damaged gut epithelium, patients will have problems with oral rehydration.

Treatment

Ciprofloxacin, azithromycin, and TMP-SMX can be used to treat the bacteria and shorten the duration of symptoms. Patients will also need oral and intravenous hydration to replace the lost fluid via diarrhea.

Enterohemorrhagic *Escherichia coli* (EHEC)

Morphology & Culture

*Escherichia coli O157:H7, cell with flagella, transmission electron microscopy, pseudoreplica technique.*

*E. coli* is a gram-negative rod. It is motile, ferments lactose and is catalase positive. *E. coli* will grow pink colonies on MacConkey’s agar and as metallic, green colonies on Eosin-Methylene Blue (EMB) agar.

Location

Pathogenic *E. coli* are found exogenously, most likely in the feces of cow manure that contaminates a water supply.

Virulence Factors

There are several virulence factors that cause EHEC to become pathogenic:

- EHEC secretes a Shiga-like toxin that destroys the cell via the 60S ribosome destruction similar to *Shigella*.
- O antigen is the most external component of the gram-negative LPS and confirms antigenicity.
- H antigen is part of the subunit of the flagella and is present in this species.
- K antigen is part of the capsule and is present in this species.
EHEC has **fimbrie** that helps the bacterium attach to the intestinal lumen and deliver its toxin.

**Clinical Picture**

Patients with EHEC will complain of nonfebrile, bloody diarrhea and have hemorrhagic colitis. EHEC is also associated with **hemolytic uremic syndrome (HUS)**, which is caused by the E. coli strain O157: H7. This strain is often found in undercooked beef. Patients will have **acute renal failure, microangiopathic hemolytic anemia, and thrombocytopenia**.

**Treatment**

Treatment for EHEC involves oral hydration to replace the lost fluids. Tetracycline, cephalosporins, and aminoglycosides can be used. EHEC is becoming multi-drug resistant and only susceptible antibiotics should be used.

**Salmonella**

![Color-enhanced scanning electron micrograph showing Salmonella Typhimurium (red) invading cultured human cells.](image)

**Morphology & Culture**

*Salmonella* are gram-negative rods of variable length with flagella. This bacterium **does not ferment lactose**, is **oxidase negative**, but **produces H₂S**. *Salmonella* will grow on regular agar plates.
Location

*Salmonella* is common to the gut flora in many animals and is transmitted to humans via fecal to oral route. It is commonly found in turtles, undercooked chicken, and eggs.

Virulence Factors

There are many virulence factors that cause *Salmonella* to be pathogenic:

- *Salmonella* carry the **H and O antigens**.
- There is a **Vi antigen** that surrounds the O antigen and makes the bacterium harder to phagocytize.

Clinical Picture

Infection with *Salmonella* can result in four different outcomes: **typhoid fever, carrier state, bacteremia, and enterocolitis**.

Typhoid fever (also called enteric fever) is caused by infection of the *typhi* serotype of *Salmonella*. The bacteria enter the bloodstream and disseminate into different organs and lymph tissue, where they can live inside the macrophages and replicate. One to three weeks after infection, patients complain of fever, headache, and myalgias. They will also have characteristic **rose spots** on the abdomen. Patients will also suffer from hepatosplenomegaly.

Bacteremia is associated with the serotype *choleraesuis* which can disseminate into the **lungs** or bones, but **never the intestine**. Symptoms will be related to the infected organ system. Patients with sickle cell anemia are at increased risk for osteomyelitis with this bacterium.

Enterocolitis is the most common outcome from a *Salmonella* infection. Patients will exhibit diarrhea two days after exposure with abdominal pain over the terminal ilium and fevers. Diarrhea will have leukocytes and occasionally red blood cells. Diarrhea resolves in two days.

*Salmonella* can enter the gallbladder where it can sequester itself. The patient will be asymptomatic, but will pass bacteria with every bowel movement and can infect others with improper hand washing.

Treatment

Patients with enterocolitis will need to replenish their fluid loss with oral rehydration and the bacterial episode will self-resolve. For patients with invasive *Salmonella* infections, antibiotic treatment with ampicillin, TMP-SMX, or a third generation cephalosporin will treat the infection.

References


Gladwin, M., Trattler, B. Clinical Microbiology made Ridiculously Simple. 5th ed. Miami: