

**Gastrointestinal Tract Module
Bacterial infections
2022-2023**

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Lecture 5

Bacterial infections of GIT

Introduction

Classification

Gastroenteritis/Food poisoning

S. aureus
C. botulinum
C. perfringens
B. Cereus

Watery (secretory) diarrhea

V. cholera
ETEC
EPEC

Cell invasion

Shigella
Salmonella enteritidis
EHEC
EIEC

Antibiotic associated diarrhea

C. difficile

Cell invasion and bacteremia

C. Jejuni
Salmonella typhi

Gastritis and ulcers

H. pylori

Food Poisoning

Staphylococcus aureus

- Food poisoning occurs when consuming a food that is contaminated with bacterial toxin
- Bakery, meat, poultry, egg products, mayonnaise-based salads, cream-filled pastries and cakes, and other dairy products.

Mechanism of intoxication

- Contaminated hands or through coughing or sneezing into foods that are ready to eat.
- The toxin is resistant to gastric pH and heat.



Food Poisoning

Staphylococcus aureus

Clinically:

- Short incubation period of 1-6 hrs
- Mild fever
- Symptoms may last 12 hrs -2 days on average

Treatment

- Usually self limiting
- Rehydration fluids
- Controlling fever (if any)
- Occasionally hospitalization, particularly when infants, elderly or debilitated people are concerned



Food Poisoning

Bacillus cereus

Clinically

- Ingestion of spores in contaminated meat, fish, and vegetables, raw starchy foods such as pasta, potatoes, pastries and noodles)
- 95% of cases are associated with rice dishes (fried Rice Syndrome)
- The diarrhea is caused by in vivo production of a heat-labile enterotoxin
- longer incubation (6-24 hours)
- Watery diarrhea, abdominal cramps
- vomiting (25%)
- Duration of illness ranges from 20-36 hours



Food Poisoning

Bacillus cereus

Control

- By proper cooling and storage of food rice in particular should not be stored for long periods above 10°C.

Treatment

- Oral rehydration
- Intravenous fluid with severe dehydration and vomiting
- Antibiotics are not indicated

(*B. cereus* = Be serious not to give antibiotics)

Diagnosis

By the isolation of *B. cereus* from the implicated food, but such testing is often not done because the illness is relatively harmless and usually self-limiting

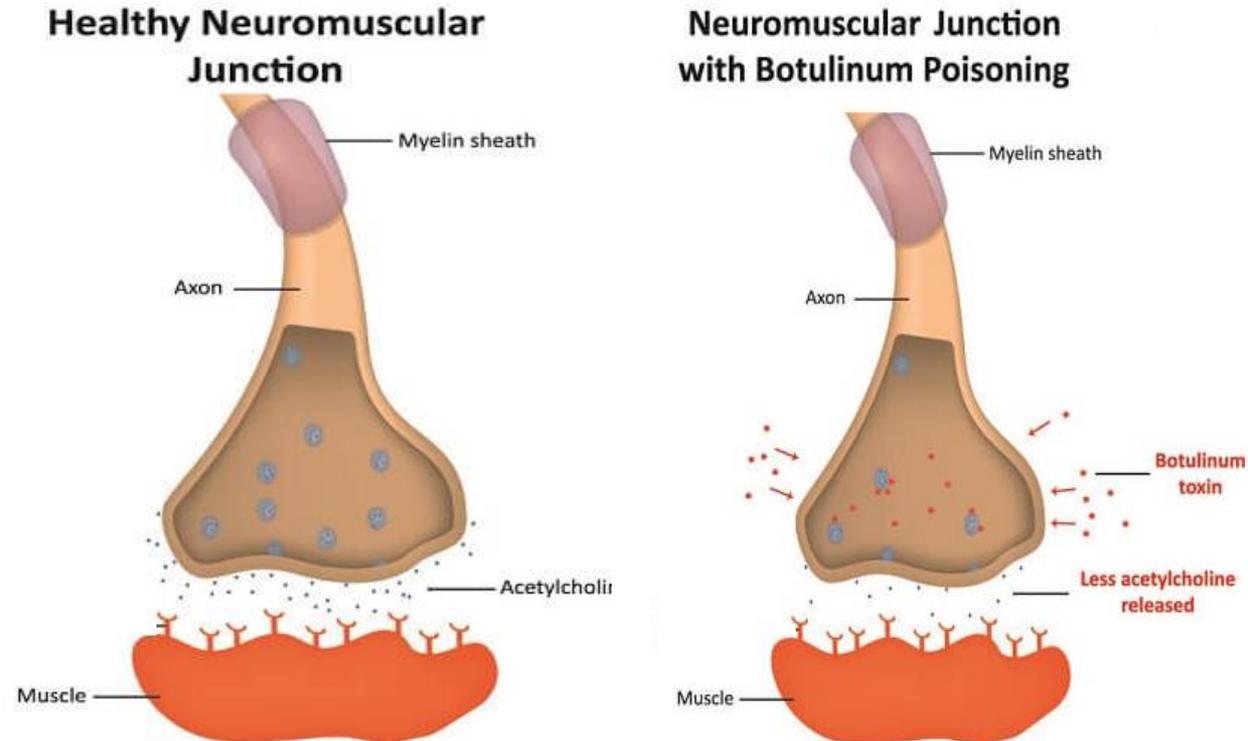
Food Poisoning

Clostridium botulinum (flaccid paralysis)

Botulinium Neurotoxins

- Seven different types: A through G
- Destroyed by boiling

Botulinium toxin (Mode of action)



Food Poisoning

Clinical categories of botulism

1. Foodborne botulism

- Most common from home-canned foods
- Onset : 18 to 36 hours after exposure (range, 6 hours to 8 days)
- Early: nausea, vomiting, weakness, dizziness but no fever
- Late: double vision, difficulty in swallowing, and speaking
- In severe cases, death due to respiratory muscle paralysis

2. Infant botulism

- Contaminated food with spores from varied sources (Honey, food, dust, corn syrup)
 - Baby will develop a weak cry, decreased sucking, floppy head and decreased motor response to stimuli
- Death if not treated



Cell invasion

Shigella

Salmonella enteritidis

EHEC

EIEC



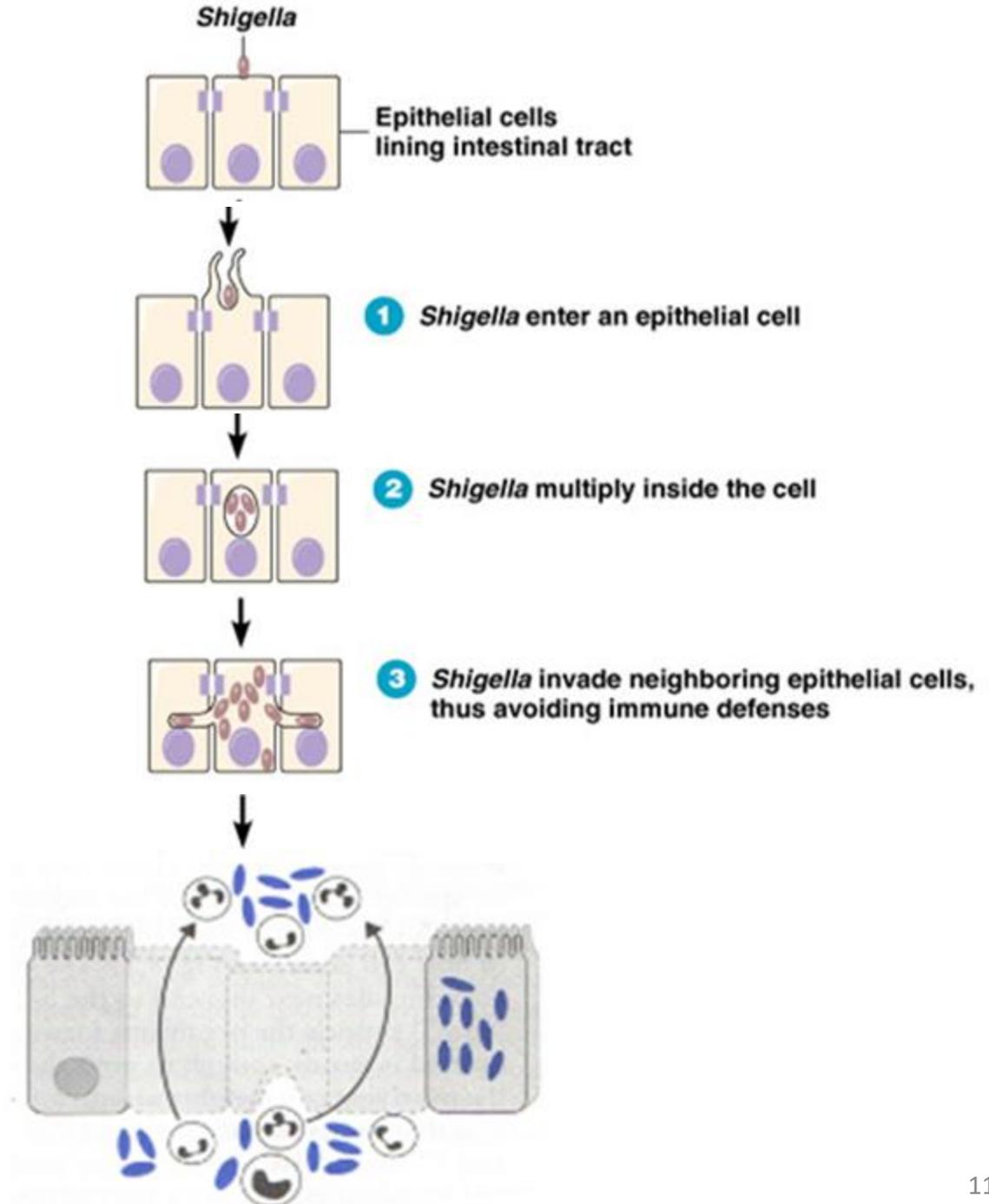
Shigella

Sources of infection

- Fecally contaminated water
- Any food contaminated by a food handler with poor hygiene
- Raw vegetables
- *S. dysenteriae* (Group A, the most pathogenic)

pathogenesis

- Destruction of endothelial cells causing hemorrhage
- Bacteria enter blood are quickly killed by phagocytes



Shigella

Clinically

- The infective dose is between 10-200 organisms
- Incubation of 1-7 days
- Followed by fever, cramping, abdominal pain, and watery diarrhea for 1-3 days
- This may be followed by scant stools with blood, mucous, pus, and tenesmus

Diagnosis

- Dehydration with fast heart rate and low blood pressure
- Abdominal tenderness
- Elevated white blood cell count
- Stool culture
- White and red blood cells in stool

Shigella

Treatment

- Self limiting
- Rehydration
- Antibiotics are usually avoided in mild cases
- Medical treatment should only be used in severe cases or for certain populations with mild symptoms (elderly, immunocompromised, food service industry workers, child care workers)

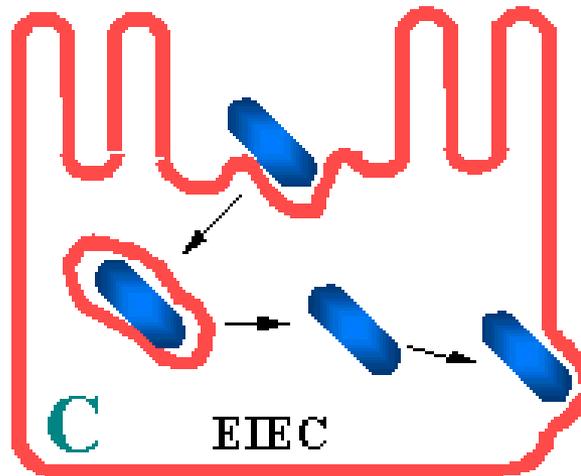
Control

- Proper hand washing after using the bathroom.
- Use properly treated water.
- Cook foods to appropriate temperatures

Enteroinvasive *E. coli* (EIEC)

Pathogenesis

Similar to *Shigella*



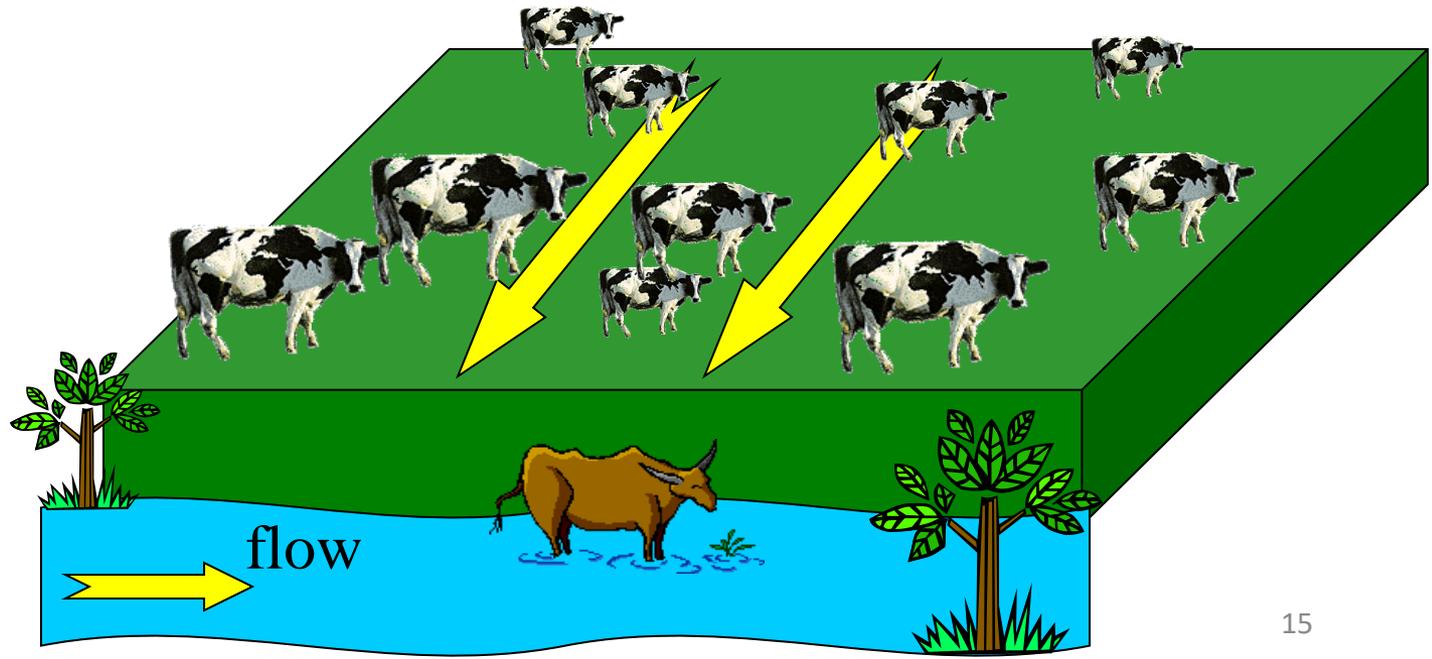
Enterohemorrhagic *E. coli* (EHEC)

Source of EHEC infection

- Consumption of contaminated food water, milk, or by contact with animals, feces and contaminated soil
- Infected hamburger, salami, and sausages served at fast food chains

Characteristics

- Produces Shiga-like toxin (also called verotoxin).



Diagnosis

This is most often caused by serotypes O157:H7

1. This strain of *E. coli* can be differentiated from other strains of *E. coli* by the fact that it does not ferment sorbitol in 48 hours (other strains do)
2. One must confirm that the isolate is *E. coli* O1547:H7 using serological testing
3. Confirm production of the shiga-like toxin by either ELISAs, agglutination, or immunoblotting before reporting out result.
4. Genotyping for shiga toxin gene

Salmonella

General characteristics

- *Salmonella* (like *Shigella*) is never a part of the intestinal flora (always pathogenic)
- Different types of the *Salmonella* bacteria can cause the illness. The two most common pathogens are
 - *S. typhimurium*
 - *S. enteritidis*.

Salmonella

Salmonella associated diseases

Gastroenteritis or salmonellosis (diarrhea)

- Reservoir:
 - Normal flora of domestic animals, especially cattle, chickens, and exotic pets such as turtles
 - Poultry, pork, beef and fish (seafood): if the meat is prepared incorrectly or is infected with the bacteria after preparation
 - Infected eggs, egg products, and milk when not prepared, handled, or refrigerated properly
 - Contaminated fruits and vegetables
- Humans are **infected** when there is contamination of food or water with **animal feces**

Salmonella

Pathogenesis

- The bacteria remains restricted to the intestine: The inflammatory response prevents the spread beyond the GI tract and eventually kills the bacteria.

Manifestations (Gastroenteritis)

- Typically, the episode begins 24 to 48 hours after ingestion
- Diarrhea persists for 3 to 4 days and usually resolves spontaneously within 7 days.
- Fever (39°C) is present in about 50% of the patients.
- The spectrum of disease ranges from a few loose stools to a severe dysentery-like syndrome
- Patients may require hospitalization due to severe dehydration (IV fluids and fever reduction), which is more common among infants and the elderly
- **Prevention: hygiene and proper cooking**

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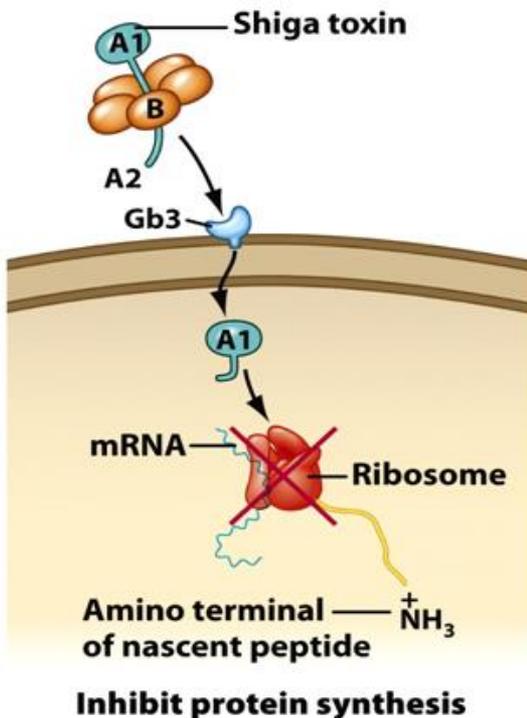
Escherichia coli

Virulence factors

➤ Pili: attachment

➤ Toxins:

1. Shiga-like toxin (Stx)



2. heat-Labile toxin (LT) (inactivation at 60°C for 30 minutes)

- Activation of adenylate cyclase
- Increase CL secretion which follows by Na and H₂O leading to diarrhea

3. heat-Stable toxin

Mediates the inhibition of Na⁺ absorption and stimulates chloride secretion by enterocytes.

Enterotoxigenic *E. coli* (ETEC)

Epidemiology

- ETEC is the leading cause of diarrhea in travelers from developed regions returning from vacations (**travelers diarrhea**).
- Contaminated food and drink with Human or animal wastes.

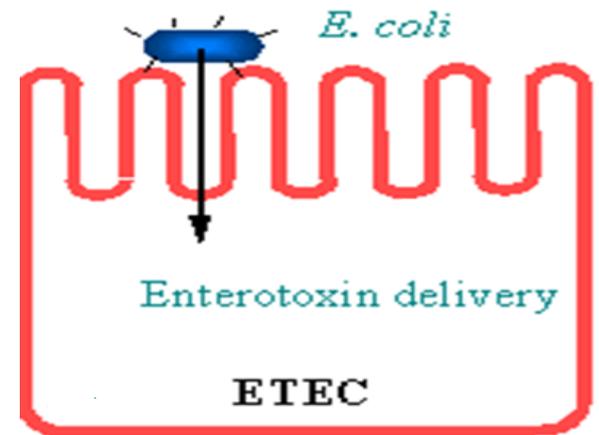
Types of toxins and pathogenesis

- A heat-stable toxin and a heat-labile toxins
- The organism attaches to the intestinal mucosa via colonization factors and then liberates enterotoxin

Entero**T**oxigenic = **T**ravelers

Clinically

- Self-limiting diarrhea
- Recovery within a few days, without specific treatment



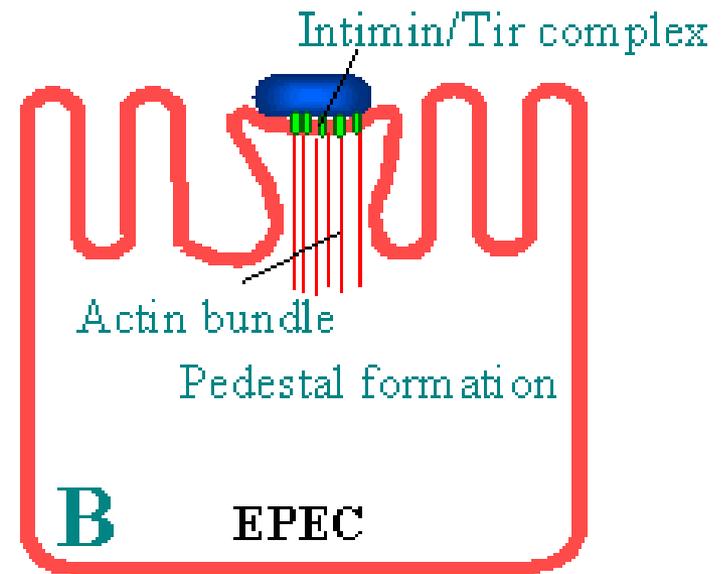
Enteropathogenic E. coli (EPEC)

Epidemiology

- Contaminated drinking water and meat products
- Contact with domestic animals
- Typically occurs in neonates and children ≤ 2 years of age (mostly ≤ 6 months), why?

During hospital outbreaks, EPEC is isolated from asymptomatic carriers including nursing and family members (1% -30%)

(**p**athogenic=**p**ediatrics)



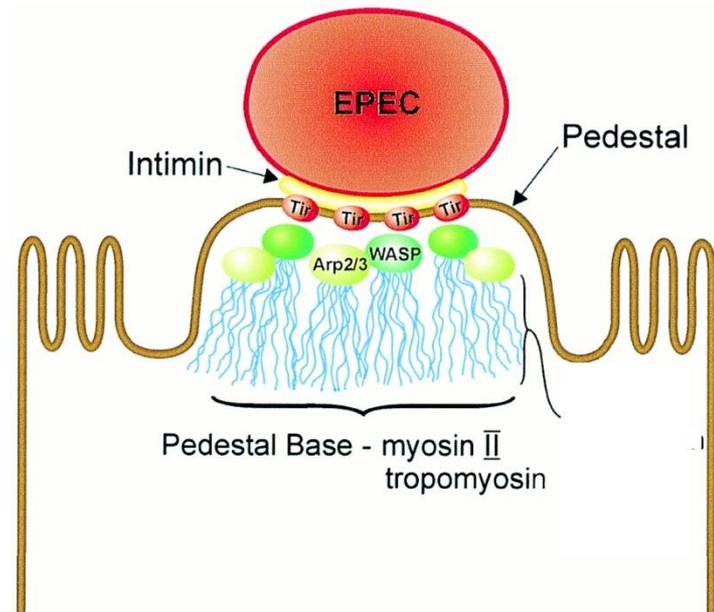
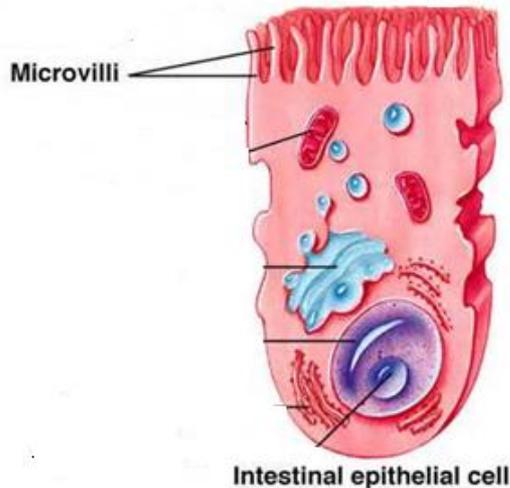
Enteropathogenic E. coli (EPEC)

Mechanism of diarrhea

1- Attachment and effacing

- By Intimin and its translocated intimin receptor (Tir)
- Attachment to enterocytes
- Formation of microcolonies
- The net result is the loss of microvilli (effacing)

2- Injection of protein that mediate electrolyte imbalance



Enteropathogenic E. coli (EPEC)

Clinically

- Fever (60%)
- Watery diarrhea that is often severe and can result in dehydration (30%)
- Abdominal distension
- Symptoms usually last for one week

Diagnosis

- Diarrhea, commonly lasting for **as long as two weeks**
- Detection of EPEC pili by specific antibodies or PCR amplification for pili encoded gene (not widely available)

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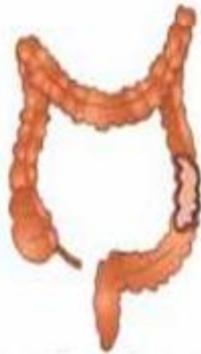
Cell invasion and bacteremia

C. Jejuni
Salmonella typhi

Gastritis and ulcers

H. pylori

Clostridium difficile



The intestines have hundreds of bacteria, many of which have useful functions, such as helping to stimulate the immune system and produce vitamins. When *C. diff* spores exist in the colon, the other bacteria do not allow them to grow and thrive.



www.patedu.com

+ Antibiotic treatment = Diarrhea

Because of *C. difficile* it becomes very difficile (difficult) to give a patient antibiotics

Clostridium difficile

Associated toxins

Pathogenic strains produce two toxins:

- Toxin A is an enterotoxin that causes excessive fluid secretion, but also stimulates an inflammatory response
- Toxin B is a cytotoxin; in tissue culture, it disrupts protein synthesis and causes disorganization of the cytoskeleton

Clostridium difficile

Diagnosis

A. Clinical diagnosis

- Diarrhea occurring ≥ 3 times a day for at least 2 days
- Abdominal cramping, fever, and dehydration
- Peripheral leukocytosis
- Pseudomembranes: The membrane composed of mucus, fibrin, inflammatory cells and cell debris overlying an ulcerated epithelium, is best demonstrated by lower GI endoscopy
- Toxic megacolon (Infrequently)
- Colonic perforation/peritonitis

B. Laboratory identification

- *C. difficile* can be cultured from stools and identified by routine anaerobic procedures
- Real-time PCR and ELISA (demonstrating of toxin)
- Latex agglutination to detect **antigen** in stools

Pseudomembranous Ulcerative Colitis



C. difficile
overgrowth

Clostridium difficile

Treatment

- Stop all non-essential antimicrobial agents.
- Consider anti- *C. difficile* therapy as recommended
- Discontinue all antiperistaltic, stool softeners, laxative medications

Infection Control

- Requires gowns and gloves for room entry before contact and hand washing with antiseptic soap after contact
- Patient Isolation immediately
- Presumptive isolation may be discontinued after patient is without symptoms for ≥ 72 hours or a negative PCR result