

Food Microbiology

The good, the bad and the ugly

- Good-important in food production; provide better taste and texture
- Bad-cause of food borne illness:
 - Infection with live organisms
 - Intoxication with bacterial toxins
- Ugly-cause food spoilage with undesirable changes

Factors affecting microbial growth in food

- Intrinsic: conditions present in food
 - Water availability (aw): amount of water in food (pure water is 1.0)
 - Most food >0.98; most bacteria require >0.90
 - pH: most pathogens not grow at pH<4.5 (except Lactic acid bacteria)
 - Nutrients
- Extrinsic:
 - Storage temperature
 - <0° no growth (water crystallizes)
 - Refrigerator: 4°C to 10°C (enzyme rxns very slow or non-existent)
 - Atmosphere: availability of O₂

Natural Food Protectants

- Antimicrobial chemicals:
 - Egg whites-lysozyme
 - Cranberries-benzoic acid
 - Raw Milk- peroxidase system
- Biological Barriers: rinds, shells, other coverings
 - Whole lemons last longer than slices
 - Some microbes can degrade

Microorganisms in food and beverage production

- Lactic acid fermentation:
 - Glucose→pyruvic acid→lactic acid
 - Fermented milk products (cheese, yogurt)
 - Pickled vegetables (sauerkraut)
- Alcoholic fermentation by yeast:
 - Glucose→pyruvic acid→alcohol + CO₂
 - Wine, beer, distilled spirits
- Yeast for bread production
- Mold growth
 - Soy sauce/Bleu cheese

Food Spoilage

- Undesirable changes
 - Repugnant taste, odor, appearance: usually not harmful but pathogens may also be present
- Common food spoilage microbes
 - Pseudomonas, Erwinia, Acetobacter, Alcaligenes, Lactobacillus, Leuconostoc
 - Fungus
 - Rhizopus, Penicillium
 - Aspergillus: grain and peanuts; produce aflatoxin
 - Claviceps purpurea: grain; ergot poisoning

Food Preservation

- Kill organisms: canning, pasteurization, cooking, irradiation
- Inhibit growth: refrigeration, freeze, dehydration (reduce a w), lower pH, high salt or sugar, chemicals

Food borne intoxication

- **Food borne intoxication:** bacteria grow within the food and produce **toxins**, the toxins are what lead to food poisoning symptoms. Ingestion of toxins
- Examples: *Clostridium botulinum*
Staphylococcus aureus

Exotoxins

- Produced inside bacteria and secreted
 - Enterotoxin, Neurotoxin, Cardiotoxin
- Superantigen: Type I → intense immune response → cytokines → fever, nausea, vomiting, shock (*Staph aureus*)
- Membrane disrupting toxin: Type II → lysis of cells by disrupting membrane
- A-B toxins: Type III has two parts
 - A is active enzyme toxin that inhibits protein synthesis and kills cell
 - B binds to surface so toxin is transported across plasma membrane (*Clostridium*)

Clostridium botulinum : botulism

- General characteristics: gram positive rod, anaerobe, spore former
- Produces a toxin (neurotoxin)
 - Heat sensitive
 - One gram can kill 1 million
- Toxin inhibits the release of acetylcholine from neurons.....what happens next?

Botulism

- **Foods associated:** home canned “low acid” vegetables, honey
- **Symptoms:** 12-72 hours after ingestion vomiting, diarrhea, blurred vision, and descending muscle weakness
- **Treatment:** antitoxin not antibiotics

Staphylococcus aureus: 24 hour Flu?

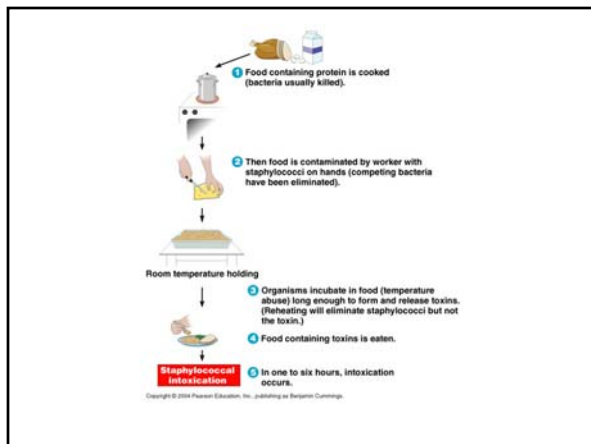
- **General characteristics:** gram positive coccus in clusters, facultative anaerobe, part of normal skin flora
- Produces a toxin
 - Enterotoxin (targets the GI tract)
 - Acts as a Superantigen

Staphylococcus aureus

- **Foods associated:** cream based desserts, custards, potato and egg salads
 - Key is to have a food handler with the organism as part of the normal skin flora
 - Remember a_w coefficient for this organism is low (0.86)
 - Food left at 28°C for 2-4 hours with *S. aureus* will have enough cells grown to cause food poisoning

Staphylococcus aureus

- **Symptoms:** appear 1-6 hours after ingestion and include vomiting (V), diarrhea (D), and intense abdominal pain/cramping(AB), usually no fever -last approximately 24 hours
- **Treatment:** none, supportive care



Food poisoning

- **Food borne infection:** bacteria enter food and grow within the food. Ingestion of organism (may then produce toxins that cause symptoms)
- Examples: *Campylobacter jejuni*, *Salmonella* spp., *Salmonella typhi*, *Shigella* spp., *Vibrio cholerae*, *Vibrio parahaemolyticus*, *Vibrio vulnificus*

Campylobacter jejuni

- **General characteristics:** gram negative curved rod, microaerophile, one or two polar flagella, no capsule
- Special culture to grow: microaerophilic, special media, 42°C incubator
- Part of the normal flora of poultry and dairy cattle
 - unpasteurized milk, undercooked poultry
 - may be found at a concentration of 10⁹cells/gram of chicken

Campylobacter jejuni

- **Incubation period:** 2-5 days
- **Symptoms:** D (which may be bloody), AB, fever (104°C), vomiting not common
 - Last 2-10 days
 - Some cases lead to Guillen-Barre syndrome and Rheumatoid Arthritis weeks after the illness

Campylobacter jejuni

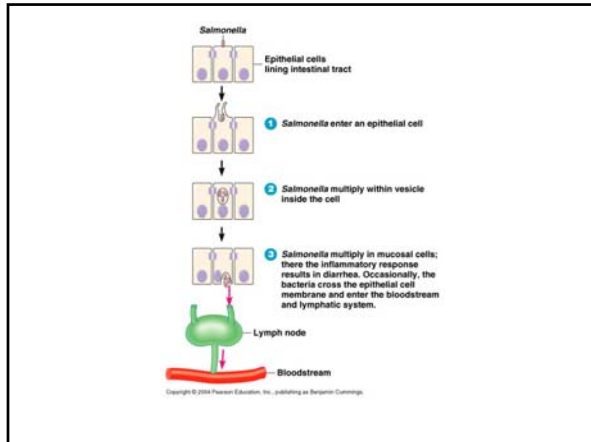
- **Treatment:** none, mostly supportive care
 - some cases require antibiotics (erythromycin, quinolones)
- Avoid undercooked poultry and watch the use cutting boards in food prep

Salmonella spp.

- **General characteristics:** gram negative rod, facultative anaerobe, peritrichous flagella
- Over 2000 closely related serovars (serotypes)
- Part of the normal flora of poultry, reptiles
- Culture: isolate and identify lactose negative, H2S positive

Salmonella

- Incubation time: 12-36 hours
- Symptoms: include D, AB, and a moderate fever
- Full recovery in a few days but may shed the organism for 6 months
- Approx. 2-4 million cases/year, only 40-50,000 are actually reported



Salmonella typhi

- Causes typhoid fever (enteric fever)
- Most Salmonella killed by acid so need to ingest large numbers to survive stomach acid in order to cause infection
- Only found in the feces of other humans
- Some individuals (Typhoid Mary) become carriers (1-3%), usually colonizes gallbladder (free of competition from NF due to bile)

Salmonella typhi

- **Incubation time:** 2 weeks
- **Symptoms:** high fever (104°C), headache, chills for one week
-transition in the second week to D, fever declines

Only 300- 500 cases annually

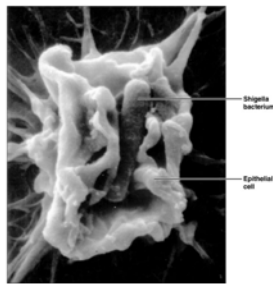
- **Treatment:** antibiotics

- *Salmonella typhimurium* and *enteritidis*:
 - Most common serovars in USA
 - Antibiotics not advised due to increasing resistance (due to widespread addition of antibiotics to animal feed)
 - Not treat gastroenteritis unless invades tissues

Shigella spp.

- **General characteristics:** gram negative rod facultative anaerobe, lactose negative
 - Only found in the feces of other humans
 - Organisms transmitted by the five F's
 - food, fingers, feces, flies, and fomites
- Shigellosis or bacillary dysentery

Shigella



Shigella

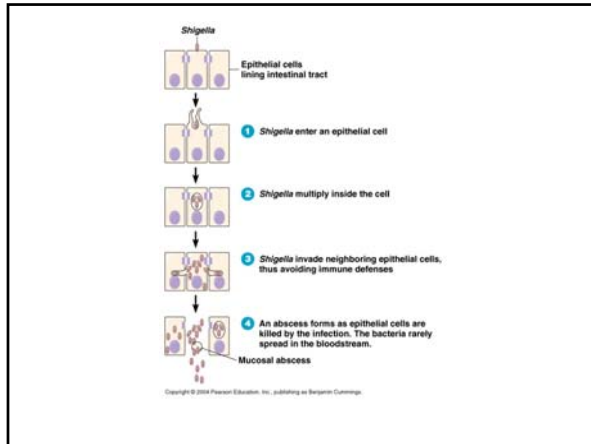
- **Incubation time:** 24-48 hours
 - Only need 10 cells to cause infection; not affected by stomach acid
- **Symptoms:** F, AB, D (may contain blood and mucus)
- See passage of small volume bloody stools (20/day)

Shigella

- *S. sonnei*-most common species in US, responsible; relatively mild; may cause some of traveler's diarrhea
- *S. dysenteriae*-causes more serious infection dysentery
 - due to the production of a Shiga toxin (A-B toxin)
 - in tropical areas-death rate up to 20%

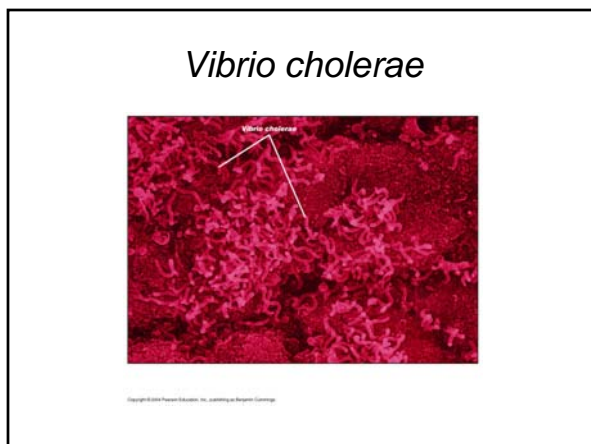
Dysentery

- *Shigella dysenteriae*
 - Virulent strains have plasmids to attach to epi cells of intestine; membrane ruffles around and engulfs bacteria which multiply in cell and produce Shiga toxin which destroys tissue; also has mechanism that allows to spread from cell to cell; sloughed areas of epis leads to intense inflammation, pus, and bleeding
 - Rarely invades blood
 - Toxin strongly associated with hemolytic uremic syndrome; RBCs break in tiny blood vessels leading to anemia and kidney damage



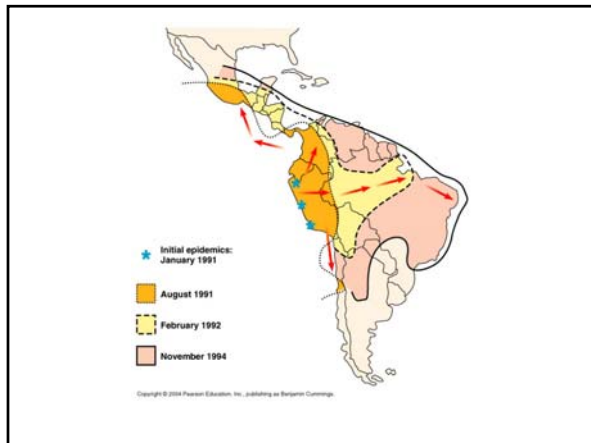
Vibrio cholerae

- Curved gram negative rod, facultative anaerobe, single polar flagella
- Can exist in saltwater for extended periods of time; tolerates high pH and high salt concentrations
- Killed by stomach acid so need large numbers of organisms to cause infection



V. cholerae

- Incubation time: several hours to days
- Symptoms: “**rice water stools**”, sudden onset of explosive watery diarrhea (up to 20 liters/day) with vomiting and pain
- Cholera toxin is the key pathogenic feature
 - A-B toxin causes activation on enzyme that causes cells to continuously secrete chloride ions and other electrolytes and H₂O → watery diarrhea



Non-cholera Vibrios

- *V. parahaemolyticus*: halophilic (requires sodium); inhabits coastal salt waters; usually transmitted by raw or undercooked shellfish; less severe gastroenteritis
- *V. vulnificus*: halophilic (requires 1% NaCl); wound infections—minor skin infection in coastal water → rapid spread through tissues → possible amputation
 - Fatal septicemia in 50% of patients with liver disease

Escherichia coli

- Normal bowel flora
- Strains that cause GI disease have virulence factors (coded by plasmids)
 - Enterotoxin production
 - Ability to adhere to small intestine

Diarrhea causing *E. coli*

- Classified according to virulence
 - Enterotoxigenic *E. coli* (ETEC)
 - Enteropathogenic *E. coli* (EPEC)
 - Enterohemorrhagic *E. coli* (EHEC)
 - Enteroinvasive *E. coli* (EIEC)

Enterotoxigenic *E. coli* (ETEC)

- Also known as traveler's diarrhea
- Two Enterotoxins promote the pumping of Cl⁻ and inhibition of NaCl which results in diarrhea
 - Profuse watery stools
- No invasion
- Can develop immunity
- Prevent with bismuth (Pepto-Bismol)

Enteropathogenic *E. coli* (EPEC)

- Attacks the small intestine
- In developing countries accounts for 20% of diarrhea in bottle-fed infants
- Attaches to mucosa of small intestine and causes cell surface changes (loss of microvilli)

Enteroinvasive *E. Coli* (EIEC)

- Invades lining of large intestine causing necrosis, inflammation, and ulceration of large bowel
- Usually seen in children in areas with poor sanitation

Enterohemorrhagic *E. coli* (EHEC)

- Obtain from the consumption of animal products
- Small dose (< 100 bacteria) to infect
- Attacks the colon
- Produces Shiga like toxin and lesion; inflammation and bleeding (hemorrhagic colitis)
- O157:H7 causes bloody diarrhea which may lead to hemolytic uremic syndrome
- Sorbitol negative MacConkeys used to isolate; culture all bloody diarrhea

Listeria monocytogenes

- Gram positive rod, non-spore forming
- Infects GI tract (usually few symptoms), penetrates mucosa and enters blood stream
- Fourth leading cause of meningitis (perhaps 50% fatality rate)
- Pregnant women: crosses placenta and causes abscess in fetal tissue → miscarriage or stillbirth (60 %)
- Can grow at 4°C (refrigerator)
- Outbreaks from coleslaw, unpasteurized milk, soft cheeses, hot dogs

Bacillus cereus

- Spores are ubiquitous in nature; can cause food poisoning; diarrhea and vomiting
- Most commonly opportunistic (post trauma to eye)
- Heating does not kill spore but will kill other competing bacteria
- Rice in Asian restaurant

Yersinia enterocolitica

- Gram negative rod; lactose negative; grows at 4°C
- Inhabits domestic animals (contaminates meat and milk)
- Fever, diarrhea, abdominal pain
- Can invade mucosa and spread to lymphatics (may present as appendicitis)
- Implicated in contamination of transfused blood

TABLE 25.2 (continued)		
Disease	Pathogen	Comments
Bacterial Diseases of the Lower Digestive System		
Cholera	<i>Vibrio cholerae</i> O:1 and O:139	Exotoxin causes diarrhea with large loss of water and electrolytes; no invasion of tissue.
<i>Vibrio</i> gastroenteritis Non-O1	<i>V. cholerae</i>	Mild diarrhea
<i>Vibrio parahaemolyticus</i> gastroenteritis	<i>V. parahaemolyticus</i>	Exotoxin causes cholera-like diarrhea, but generally milder.
<i>V. vulnificus</i> gastroenteritis	<i>V. vulnificus</i>	Very dangerous for people suffering from liver disease.
Enterotoxigenic <i>E. coli</i> gastroenteritis	<i>Escherichia coli</i>	Watery diarrhea that resembles mild form of cholera; typical traveler's diarrhea.
Enteroinvasive <i>E. coli</i> gastroenteritis	<i>E. coli</i>	Enterotoxin causes Shigella-like dysentery.
Enterohemorrhagic <i>E. coli</i> gastroenteritis	<i>E. coli</i> O157:H7	Causes hemorrhagic colitis (very bloody stools) and hemolytic uremic syndrome (blood in urine, possible kidney failure).
Campylobacter gastroenteritis	<i>Campylobacter jejuni</i>	Microaerophilic pathogen found in animal intestinal tracts; very common cause of gastroenteritis.
<i>Helicobacter</i> peptic ulcer disease	<i>Helicobacter pylori</i>	Pathogen is adapted to survive in stomach; presence leads to peptic ulcers.

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TABLE 25.2 (continued)		
Disease	Pathogen	Comments
<i>Yersinia</i> gastroenteritis	<i>Yersinia enterocolitica</i>	Pathogen is inhabitant of intestinal tract of animals; grows slowly at refrigerator temperatures. Symptoms are abdominal pain and diarrhea, usually mild. May be confused with appendicitis.
<i>Clostridium perfringens</i> gastroenteritis	<i>Clostridium perfringens</i>	Usually limited to diarrhea.
<i>Bacillus cereus</i> gastroenteritis	<i>Bacillus cereus</i>	May take form of diarrhea or nausea and vomiting; probably caused by different toxins.
Viral Diseases of the Digestive System		
Mumps	Mumps virus	Painful swelling of parotid glands.
Hepatitis A	Hepatitis A virus (HAV)	Mild disease, mostly malaise; often subclinical. Fecal-oral transmission, low mortality rate.
Hepatitis B	Hepatitis B virus (HBV)	Transmitted by blood and other body fluids, including sexual activity. Severe disease likely to cause liver damage; about 10% of cases become chronic.
Hepatitis C	Hepatitis C virus (HCV)	Similar to hepatitis B but much more likely to become chronic.
Hepatitis D	Hepatitis D virus (HDV)	Very severe liver damage with high mortality rate. Must be coinfectd with HBV.
Hepatitis E	Hepatitis E virus (HEV)	Similar to hepatitis A; fecal-oral transmission. Pregnant women may have high mortality rate.
Viral gastroenteritis	Rotavirus, caliciviruses (or Norwalk)	Self-limiting.

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TABLE 25.2 (continued)		
Disease	Pathogen	Comments
Fungal Diseases of the Digestive System		
Ergot poisoning	Mycotoxin produced by <i>Claviceps purpurea</i>	Ingestion causes neurological or circulatory problems.
Aflatoxin poisoning	Mycotoxin produced by <i>Aspergillus flavus</i>	Mycotoxin probably contributes to liver cancer.
Protozoan Diseases of the Digestive System		
Giardiasis	<i>Giardia lamblia</i>	Protozoan adheres to intestinal wall, may inhibit nutritional absorption. Causes diarrhea.
Cryptosporidiosis	<i>Cryptosporidium parvum</i>	Shed in animal feces, protozoan enters water supply; causes self-limiting diarrhea but may be life-threatening if immunosuppressed.
Cyclospora diarrheal infection	<i>Cyclospora cayentanensis</i>	Usually ingested with fruits and vegetables; causes watery diarrhea.
Amoebic dysentery (amoebiasis)	<i>Entamoeba histolytica</i>	Amoeba lyses epithelial cells of intestine, causes abscesses; significant mortality rate.

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