Microbiology

Sheets for
Dr. Nader Material
Lecture (2-4)

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Lec 2: Spore Forming G+ Bacilli

Bacillus Species

spore forming rods occurring in chains includes large aerobic or facultatively anaerobic + Saprophytic(meaning they are mostly found where there is decomposed organic matter so they are ubiquitous(prevalent in soil, water, and air)

Feature	Bacillus anthracis	Bacillus cereus
Morphology	Non-motile, non-hemolytic, non-licithenase	Motile, hemolytic, licithenase-positive (a phospholipase c), A 3–4 μ m
Colony	pathognomic appearance, Medusa head (dry, waxy)	Feathery upon blood agar or in exclusive agar as MYP agar.
Diseases	- Cutaneous anthrax (most common) - Inhalational anthrax (Woolsorter's disease) - Gastrointestinal anthrax (rare) - Anthrax meningitis (from bacteremia)	- Food poisoning → Emetic type, Diarrheal type: - Eye infections (endophthalmitis) - Systemic infections in immunocompromised (meningitis, pneumonia ,endocarditis,osteomyelitis
Diagnosis	Culture, clinical features	Clinical + Isolation from food/stool/vomit (≥10^6 CFU/g)
Treatment	Antibiotics for anthrax	Usually self-limited Antibiotics not needed (resistant to penicillins/cephalosporins)
Prevention	Animal vaccine, avoid contact with infected animals/products	Proper food handling, storage, reheating

Some notes on Bacillus cereus

-aerobic or facultatively anaerobic

- العوامل التي تزيد من خطر الإصابة تشمل وجود medical devices (أجهزة طبية داخل الجسم) أو استخدام intravenous drugs (المخدرات الوريدية)، حيث تزداد احتمالية الإصابة بـ catheter-associated septicemia (تسمم الدم المرتبط بالقسطره)

-Enterotoxins are usually produced by bacteria outside the host and therefore cause symptoms soon after ingestion of B. cereus, the spores germinate if left at room temperature and when they come into contact with organic matter or within an insect or animal host.

-B.cereus are inherently resistant to Penicillin and cephalosporins ,while B.anthrax could be sensitive to them that is why the main stream of treatment for gastroenteritis is fluid and electrolytes replacement and no need for antibiotics treatment since they are inherently resistant to penicillin and cephalosporin) (except in cases of salmonella ,shigella, and vibrio cholera where treatment tends to be more specified).

Bacillus subtilis is a model organism in bacteriology, particularly used for studying chromosomal multiplication in bacteria and for its role in antibiotic production as well as probiotic production, but overall it does not seem to cause significant human disease except in immune compromised individuals).

B thuringiensis which is found in insecticides.

Bacillus cereus Food Poisoning

Feature	Emetic Type (Vomiting Type)	Diarrheal Type
Toxin Type	Heat-stable exotoxin (Cerulide)	Heat-labile exotoxin
Associated Food	Fried rice (flash frying), cereals	Meat dishes, sauces, vegetables
Toxins Involved	Cerulide, hemolysins, phospholipases (lecithinase), hemolysin BL (HBL), NHE, cytotoxin K	Same as left
Pathogenesis Mechanism	Ingestion of preformed toxin produced when cooked rice is left at room temp and reheated	Ingestion of spores → germinate in GIT → produce enterotoxins locally
Enterotoxin Site	Preformed in the food	Produced in the intestine or preformed in the food
Incubation Period	0.5–6 hours (short)	6–15 hours (longer)
Symptoms	Vomiting (main), occasionally diarrhea and cramps (rarely), no fever	Diarrhea, abdominal cramps, possibly nausea (vomiting rare), no fever
Duration	Usually over in 24 hours, self-limiting	Usually over in 24 hours, self-limiting
Mechanism of Cerulide	Not elucidated; believed to act as a superantigen	—
Reason for Incubation Time	Toxin is preformed → short incubation	Spore ingested → needs time to germinate and release toxin → longer incubation

- Heat-labile toxin: gets destroyed by heat (like boiling or cooking).
- Heat-stable toxin: resists heat, even when boiled, it still works.

*NHE (Non-Hemolytic Enterotoxin)

Clostridium Species – General Info

Feature	Description
Shape & Gram	Gram-positive, spore-forming rods, Spores of clostridia are usually wider than the diameter of the rods in which they are formed.
Oxygen	Anaerobic, few species are aerotolerant such as C.tertium and C.histolyticum both of which can cause gas gangrene but very rarely.
Habitat	Soil, GIT, vagina (C. perfringens)
Motility	Motile (except C. perfringens)
Growth media	Thioglycollate broth, blood agar
Toxins	Powerful exotoxins (e.g., tetanospasmin, botulinum toxin)

Clostridium Species Overview

Species	Major Toxin	Diseases / Efffect	Notes
C. tetani	Tetanospasmin	Tetanus (rigid paralysis)	Blocks inhibitory NTs (GABA & glycine); toxoid vaccine (DTaP), booster every 10 years
C. botulinum	Botulinum toxin	Botulism (flaccid paralysis)	_
C. perfringens	iota, epsilon, edema, Alpha	Gas gangrene, food poisoning, necrotizing fasciitis	Rapid onset necrosis, diarrhea (rarely to form spores in vivo nor on the laboratory media)
C. difficile	Toxin A & Toxin B	CDI (C. difficile infection): mild → antibiotics-associated diarrhea; severe → PMC (pseudomembranous colitis)	#1 causes of nosocomial diarrhea

*All of them cause exotoxin mediated diseases

Clostridium botulinum – Botulinum Toxin

Anaerobic Endospore-forming gram-positive bacilli with subterminal spore location.

Category	Details
Toxin	Botulinum toxin – highly toxic neurotoxin, coded by a prophage.
Lethal Dose	1 µg/kg body weight (≈70 µg for 70 kg person).
Cosmetic Use	Used in very small amounts (nanograms).
Serotypes	A–G (including C1, C2). Most common in humans: A, B, E. F is less likely.
Antitoxin	BIG (Botulism Immune Globulin) – trivalent against types A, B, and E.
Source of Contamination	Spiced, smoked, vacuum-packed, or canned alkaline foods eaten without cooking.
Clue in Food	Bulging/swollen cans (gas production by proteolytic enzymes of C. botulinum).
Mechanism of Action	Absorbed from gut → blood → peripheral synapses → blocks acetylcholine release at NMJ → flaccid paralysis.

*هذا يعني أن الجين المسؤول عن تصنيع هذا السم موجود داخل DNA فيروس بكتيري (phage) دخل إلى البكتيريا وأصبح جزءًا من مادتها الوراثية (prophage). أي أن البكتيريا لا تملك القدرة الأصلية لإنتاج السم، بل اكتسبتها من فيروس بكتيري.

Clostridium botulinum – Types of Botulism

Type	Cause	Notes
Foodborne botulism	Ingestion of preformed toxin in spoiled canned food (intoxication).	Toxin is absorbed through the gut → causes flaccid paralysis. Mechanism of action same as adult botulism.
Wound botulism	Contamination of wounds with spores → spores germinate and produce toxin.	Occurs without ingestion.
Infant botulism	Ingestion of spores, which germinate and produce toxin in the infant's GI tract.	Common in Canada and the US. Honey is the most common vehicle. Doesn't occur in adults due to mature microbiome.
Adult infectious botulism	Ingestion of spores, which germinate in the GI tract and produce toxin.	Similar to infant botulism. Happens in adults with altered gut flora. Mechanism same as foodborne (intoxication).
Inadvertent botulism	Improper use/injection of botulinum toxin in medical or cosmetic settings.	Happens when used in inappropriate doses.

Clostridium botulinum – Other details

Feature	Description
Toxin	Botulinum toxin (most potent exotoxin) Blocks ACh → symmetrical, descending flaccid paralysis usually beginning with cranial nerves then involving motor and autonomic nerves.
Habitat	- Found in soil → may contaminate vegetables grown in/on soil. - Colonizes GI tract of fishes, birds, and mammals.
Diagnosis	- Direct observation is not enough — must confirm toxin production. - Toxin detection: serum, gastric contents, stool, leftover food using ELISA/PCR. - Mouse bioassay (biolethality test) is the gold standard: mouse dies if toxin is present; survives if pre-injected with antitoxin. - Notifiable disease — must be reported to health authorities. - Specimens sent to reference labs, not regular labs.
Prevention	- Proper heating of canned foods to kill spores. - Discard bulged or poorly canned foods. - Boil home-canned foods for >20 minutes before eating. - No honey for infants <1 year (risk of infant botulism).

- Unlike Gillian Barré syndrome which starts in an ascending pattern (× aspirin for children with influenza and fever)

*The presence of the bacteria itself is not enough since it could be non-toxin producing strain, since that toxin is encoded by a prophage

Clinical Findings of Botulism

- **Early gastrointestinal symptoms (start 18–36 hours after eating toxic food):**

Nausea, Vomiting, Abdominal cramps, Diarrhea

- **Early neurological symptoms (in a descending pattern):**

Dry mouth, Blurred Vision, Double vision (diplopia)

- **Later neurological symptoms:**

Difficulty swallowing, Difficulty speaking, in severe cases: respiratory muscle paralysis, which may cause breathing failure

- **Important note:**
  - Motor function is affected, but sensory function stays normal

- **Infant botulism:**
  - Occurs in babies in the first months of life
  - Symptoms: poor feeding, weakness, signs of paralysis (floppy baby appearance)
  - May be a cause of sudden infant death syndrome (SIDS)

Treatment of Botulism

- **Monitor the patient closely:**
  - Watch heart rate and breathing regularly.
  - Be ready to use a ventilator if breathing muscles stop working.
- **Supportive care is essential:**
  - The most important treatment is mechanical ventilation if the patient has severe breathing problems.
- **Antitoxin therapy:**
  - Use Botulism Immune Globulin (BIG A).
  - It works against toxin types A, B, and E (the most common in humans).
  - It should be given early through the vein (IV) along with supportive care.

- **Foodborne botulism:**
  - Often goes away on its own (self-limited), especially if mild.
- **Infant botulism:**
  - Most babies get better with supportive care alone.
  - But giving antitoxin is still recommended to help recovery.
- **Wound botulism:**
  - Needs surgical cleaning (debridement) of the infected wound.

Clostridia that Produce Invasive Infections

- Many different toxin-producing clostridia can cause invasive infections (including myonecrosis and gas gangrene) when introduced into damaged tissue, especially post traffic accidents.
- Approximately 30 species of clostridia may produce such infections, but the most common in invasive disease is C. perfringens (90% of cases).

Clostridium Perfringens

Category	Details
Morphology	- Large Gram-positive, non-motile, spore-forming rods (spores are rare in tissue or on agar).- Anaerobic bacterium.
Distinguishing Features	- Stormy fermentation in milk media.- Double zone of hemolysis on blood agar due to alpha and beta toxins.- Produces lecithinase (alpha toxin).
Reservoir	- Found in soil, human colon, and vagina.
Transmission	- Foodborne (contaminated meat, especially type A).- Traumatic implantation (e.g., soil in wounds).- Type C strains may cause necrotizing enteritis (pigbel) after ingestion of contaminated food.
Epidemiology	- Common in environment and GI tracts.- Spores survive cooking (especially in large meat portions), then multiply during improper cooling/storage.
Pathogenesis	- Spores enter through wounds or GI tract → germinate in low oxidation-reduction potential tissue (anaerobic).- Vegetative cells multiply, ferment carbohydrates in the tissue, and produce gas.- Toxins such as alpha (lecithinase) and theta toxins have lethal, necrotizing, and hemolytic properties.- Some strains also produce CPE (enterotoxin) that causes diarrhea.

*Stormy fermentation: Litmus milk is converted into coagulant and gases in between coagulants.

*types of toxins C.difficele produce :iota, epsilon, edema, Alpha

Clinical Syndromes	- Gas gangrene (myonecrosis) → rapid tissue destruction, gas formation, shock.- Food poisoning (type A, CPE toxin) → watery diarrhea.- Pigbel (necrotizing enteritis, type C) in neonates or children after ingesting contaminated food.- Puerperal sepsis (postpartum/abortion).
Clinical Findings	- From a contaminated wound (e.g., compound fracture, postpartum uterus), the infection spreads in 1-3 days, producing crepitation (gas in subcutaneous tissue and muscles), foul-smelling discharge due to carbohydrate fermentation, rapid necrosis, fever, hemolysis, toxemia, shock, and death.- Food poisoning follows ingestion of large numbers of bacteria, with CPE causing diarrhea after 7-30 hours. The illness lasts 1-2 days.
Laboratory Diagnosis	- Gram stain from wound, pus, or tissue shows large G+ rods.- Anaerobic culture on thioglycolate or blood agar.- Stormy fermentation in milk.- Nagler test: The agar is divided into two halves. One half receives the antitoxin (against lecithinase). If C. perfringens is present and toxin-producing, a zone of opalescence appears at the site of antitoxin application.
Sporulation on Agar	- Spores rarely seen in lab culture, unlike other clostridia.

Clostridium Perfringens - Types

Type	Effect/Action
Type A	- Causes food poisoning due to enterotoxin production (CPE).- Commonly associated with gas gangrene and myonecrosis.- Can contaminate meat dishes, leading to diarrhea (7-30 hours after ingestion).
Type C	- Causes pigbel disease (necrotizing enterocolitis) in neonates.- Associated with severe necrosis and intestinal damage.- Can lead to peritonitis and shock if untreated.
Puerperal strains	- Associated with puerperal sepsis (postpartum or after abortion).- Leads to septic shock, often due to the presence of bacteria in the GI tract and vagina.- Can cause significant toxemia and hemolysis.

- **Food poisoning treatment:**
  - Usually self-limited.
  - Treated with symptomatic care: fluids and electrolyte replacement.
- **Contrast with botulism:**
  - Antibiotics are contraindicated in botulism (may increase toxins).
  - Treated with BIG (Botulism Immune Globulin) — a trivalent antitoxin.

Treatment and Prevention of C. perfringens Infections

- **Surgical debridement:**
  - Remove all dead (devitalized) tissue to stop bacterial growth.
- **Antibiotics**:
  - Start immediately, especially penicillin.
  - Often combined with clindamycin for toxin suppression.
- **Hyperbaric oxygen therapy:**
  - May help by reducing toxins and killing anaerobic bacteria.
- **Antitoxins**:
  - Immune globulins are available against toxins.
  - Not reliable as sole treatment—used as support only.

Clostridium difficile

- Most common nosocomial infection.
- Endogenous: non-toxin-producing strains may acquire toxin genes via prophage.
- Exogenous: from unsterile bedding and inanimate objects.
- Spores colonize intestine of 50% of neonates, 4–10% of adults
- Major healthcare-associated infection.
- Risk ↑ with antibiotics like cephalosporins, clindamycin.
- Overgrowth after suppression of normal flora.

Category	Details
Pathogenesis	- Produces Toxin A (enterotoxin): induces cytokine productior and fluid hypersecretion; chemotactic for neutrophils. - Toxin B (cytotoxin): depolymerizes actin, destroys cytoskeleton, targets small G-proteins, leading to enterocyte death. - Adhesin factor and hyaluronidase also contribute. - Leads to plaques, micro-abscesses, and pseudomembranous colitis (PMC).
Diseases	- Antibiotic-associated diarrhea (mild to moderate). - Pseudomembranous colitis (PMC) and fulminant colitis (severe)
Diagnosis	(1) Clinical: ≥3 unformed stools/day for ≥2 days without othe cause. (2) Lab tests: detect toxin A or B by ELISA, latex agglutination, or PCR; culture on selective agar. (3) Colonoscopy: may show pseudomembranes in ~50% of positive cases. - Toxin B tested on human cells to confirm cytotoxicity.
Treatment and Prevention	- Discontinue current antibiotics. - Use narrow-spectrum antibiotics if needed. - Metronidazole: first-line for mild cases. - Vancomycin (oral): for PMC or if metronidazole fails. - Fidaxomicin: FDA-approved alternative. - FMT (fecal microbial transplant): not FDA-approved, used in recurrent cases. - Isolation in nursing homes. - Autoclave bedpans; spores resist common disinfectants.

• Hypervirulent, hypertoxin producing strains now recognised (e.g.
ribotype 027, 078). Causes more severe diseases, with high recurrence and relapse rate.

Lec 3: Enterobacteriaceae (Pt.1)

General Characteristics of Enterobacteriaceae

Feature	Description
Morphology	large, heterogeneous ,Gram-negative rods, Non–spore-forming
Habitat	Intestinal tract of humans and animals
Clinical Importance	Common causes of bacteremia, gastroenteritis, UTIs
Oxygen Requirement	Facultative anaerobes (grow with or without oxygen)
Fermentation	Glucose fermenters (often with gas production)
Catalase Test	Catalase-positive
Oxidase Test	Oxidase-negative (except Plesiomonas)
Nitrate Reduction	Reduce nitrate to nitrite
Motility	Most are motile with peritrichous flagella (except Shigella and Klebsiella); Yersinia is motile at 25°C but not at 37°C

*that's why they are called coliforms and has the prefix entero- in their family name.

Feature	Description
Growth Media	peptone or meat extract media, Grow well on MacConkey and EMB agar; ferment lactose (used for differentiation)
ATP Production	Facultative anaerobes are ordinarily aerobes (respiration generates more ATP), but in the absence of oxygen, they go anaerobic (fermentation; with less ATP)
DNA Content	39–59% G + C
Virulence Factors	Gained via transmissible plasmids, bacteriophages, pathogenicity islands
Normal Flora vs. Pathogenic	Some enteric organisms, such as Escherichia coli, are part of the normal microbiota and incidentally cause disease, but others, the salmonellae and shigella, are regularly pathogenic for humans.
Examples	E. coli, Klebsiella, Shigella, Salmonella, Proteus, Yersinia, Enterobacter, Providencia, Citrobacter,Acinetobacter, Serratia etc.

Although some genera are commensal within the human GI tract, when they gain a new virulence feature or spread to a different anatomical location, they can establish a disease, such example is when E.coli gain a new virulence factor and cause gastroenteritis or gain access to the urinary tract and establish a UTI.

- As they are all G- bacteria, their cell wall has lipopolysaccharides, the innermost layer is composed of lipid A which is considered an endotoxin, the outermost one contain the somatic O antigen, while in the middle they all posses a common core polysaccharide called enterobacterial common antigen (ECA).

Antigenic Structure of Enterobacteriaceae

Antigen Type	Description
O Antigen	Somatic LPS antigen; heat-stable; detected by agglutination tests; stimulates IgM response
K Antigen	Capsular polysaccharide (Surrounds O or H antigens) , heat-labile, antiphagocytic; detected by capsular swelling test with specific antisera , present in some E. coli, Klebsiella
H Antigen	Flagellar protein antigen; found in motile bacteria; agglutinated by anti-H antibodies; stimulates IgG response
Vi Antigen	Special K antigen in Salmonella Typhi/Paratyphi; enhances virulence and has antiphagocytic activity
Fimbriae & Sex Pili	Involved in adhesion and conjugation; encoded chromosomally or via bacteriophages
Colicins (Bacteriocins)	Proteins produced by E. coli to inhibit competing bacteria

لما نحكي إنهم "agglutinate with anti-H antibodies" معناها إنهم بتكتلوا أو يتلازنوا لما ينحط عليهم أجسام مضادة خاصة فيهم، وهذا بيساعد في التشخيص أو التصنيف

E. coli–Associated Diarrheal Diseases

- E. coli is part of the normal intestinal microbiota (also found in small numbers in the respiratory and genital tracts).
- Different types of E. coli cause disease in different ways, based on their virulence factors (at least 5 types).

- **Major causes of disease:**

Gastrointeritis, 20-30% of sepsis cases, 80% of UTIs .

- **Neonatal meningitis and sepsis:**
  - Infection is exogenous (from mother's vaginal canal) at birth.
  - Or endogenous (from own flora gaining virulence or spreading to new site).

- **Virulence factors (like adhesion and toxins)** are carried on plasmids or introduced by bacteriophages.
- **Lab characteristics:**
  - Oxidase negative
  - Lactose fermenter
  - Produces green sheen colonies on EMB agar

E. coli – Laboratory Diagnostic Tests

- Key identifying feature: Lactose fermentation helps distinguish E. coli from non-lactose fermenters like Salmonella and Shigella.

MacConkey Agar

- **Selective & differential medium**
- **Contents:**
  - Bile salts + crystal violet → inhibit Gram-positive bacteria
  - Lactose → fermentable sugar
  - Neutral red dye → turns pink/red in acidic pH (from lactose fermentation)
- **Result:**
  - Lactose fermenters (e.g., E. coli) → pink/red colonies
  - Non-lactose fermenters (e.g., Shigella, Salmonella) → colorless colonies

Eosin Methylene Blue (EMB) Agar

- Also used to detect lactose fermentation
- **Results:**
  - Lactose fermenters → colonies with dark center and metallic green sheen (e.g., E. coli)
  - Non-lactose fermenters → colorless or transparent colonies

Comparison of E. coli Pathotypes

Feature	EPEC (Enteropathogenic)	ETEC (Enterotoxigenic)	STEC/EHEC (Shiga toxin-producing)
Affected Site	Small bowel	Small bowel	Large bowel
Major Population Affected	Infants (especially bottle-fed)	Travelers, infants in developing countries	Children, all ages with contaminated food
Transmission	Person-to-person	Contaminated food/water	Undercooked beef, raw vegetables, apple cider
Attachment Factors	Bundle-forming pilus (EAF plasmid), LEE pathogenicity island	Colonization factors (CFA, plasmid-encoded)	Intimin (for adherence), no invasion
Toxins	None	LT (heat-labile), ST (heat-stable)	Shiga-like toxin 1 & 2
Toxin Mechanism	Effacement of microvilli	LT → ↑cAMP, ST → ↑cGMP → fluid secretion	Inhibits 60S ribosome → inhibits protein synthesis
Diarrhea Type	Watery, mucus, no blood/WBC	Watery diarrhea	Bloody diarrhea, can lead to HUS
Additional Features	Self-limited; antibiotics may shorten duration	Antibodies protect from LT; ST not immunogenic	Associated with HUS (hemolytic uremic syndrome), O157:H7 serotype
Use of Antibiotics	May help if chronic	May help	Contraindicated – can worsen HUS

Lec 4: Enterobacteriaceae (Pt.2)

General Characteristics of Shigella

Feature	Description
Shape	Slender gram-negative rods; coccobacillary in young cultures
Oxygen Requirement	Facultative anaerobes (grow best aerobically)
Colony Morphology	Convex, circular, transparent, ~2 mm in 24h
Motility	Non-motile
Lactose Fermentation	Only Shigella sonnei ferments lactose (slowly); others do not
H2S Production	Negative
EMB Agar	Colorless colonies

Epidemiology

Feature	Description
Natural Habitat	Human and primate intestinal tract
Transmission	Feco-oral (person-to-person, food, water, flies, door handles, toilet seats)
Age Group Affected	Mostly children under 5; also MSM, institutionalized adults, and household contacts
Infective Dose	Very low (10–1000 organisms)
Common Species (Developed)	Shigella sonnei
Common Species (Developing)	Shigella flexneri
Asymptomatic Carriers	Possible in endemic areas and recovered individuals

Species Classification

Group	Species	Serotypes	Severity	Notes
A	Shigella dysenteriae	12	Most severe	Produces potent Shiga toxin (type 1)
B	Shigella flexneri	8	Mild	Common in developing countries
C	Shigella boydii	18	Mild to severe	
D	Shigella sonnei	1	Intermediate	Only lactose fermenter (slow)

Pathogenesis

Stage	Description
Invasion	Through M cells → apoptosis → lateral spread via actin polymerization
Site of Infection	Colon and terminal ileum
Ulcers	Superficial, limited to mucosa, cause pseudomembrane formation
Blood Invasion	Rare; unlike Salmonella, blood cultures are typically negative
Complications	Dehydration (children/elderly), HUS, reactive arthritis (Reiter's syndrome)

Toxins:
1.Endotoxin (LPS) Produced by All Shigella species,
Action: Bowel irritation after autolysis

2. Shiga exotoxin produced by S. dysenteriae type 1,
Action: Neurotoxic, cytotoxic, enterotoxic; causes watery diarrhea and CNS effects

Clinical Features

Stage	Symptoms
Incubation	1–2 days
Early Symptoms	Fever, abdominal pain, watery diarrhea (toxin-related)
Later Symptoms	Bloody diarrhea with mucus and tenesmus (invasion-related)
Resolution	Usually self-limited in 2–5 days in adults
Severe Cases	Dehydration, seizures, acidosis, coma in children
Carriers	Some may become chronic carriers

Diagnosis

Test	Description
Microscopy	Fecal WBCs and RBCs, mucus flecks
Culture	MacConkey/EMB: colorless colonies; selective media (Hektoen, SS agar)
Serology	Not useful for diagnosis

Treatment

Approach	Details
First-line Antibiotics	Ciprofloxacin, ampicillin, doxycycline, TMP-SMX
Use of Antibiotics	For young children, elderly, immunocompromised, or household contacts
Antimicrobial Testing	Always perform due to resistance
Self-Limiting	Many cases resolve without antibiotics
Avoid	Opioids (may mask bloody diarrhea and serious conditions)

Prevention and Control

Measure	Description
Host	Humans and primates only
Immunity	IgA in gut limits reinfection; no lasting protection
Main Control Methods	Sanitation, isolation, disinfection, carrier detection, fly control
Transmission Summary	“Food, fingers, feces, and flies”

يُؤخِّرَهَا اللهُ عَلَيْك
لِيُفَاجِئَك بالذِّي هُو
أَحَبُّ إِلَيْك

Salmonella Morphology and General Characteristics

Characteristic	Details
Morphology	Gram-negative, rod-shaped, motile bacteria.
Flagella	Motile, with peritrichous flagella.
Oxygen Requirement	Facultative anaerobe.
Biochemical Properties	Lactose-negative, H_2S-producing, oxidase-negative.
Serotyping	Based on O (somatic) and H (flagellar) antigens.
Special Media	MacConkey agar, XLD agar (produces red colonies with black centers).
Growth Temperature	37°C optimal, can grow between 5°C and 45°C.

Salmonella Pathogenesis and Virulence Factors

Virulence Factor	Function
Invasins	Proteins that allow Salmonella to invade epithelial cells.
Type III Secretion System (T3SS)	Injects bacterial proteins into host cells to manipulate host defenses.
Endotoxin	Lipopolysaccharide (LPS) in the outer membrane, causing inflammation and septic shock.
Survival in Macrophages	Ability to survive and multiply inside macrophages, promoting systemic infection.
Adhesins	Allow attachment to the intestinal mucosa.

Clinical Syndromes, Diagnosis, and Treatment

Clinical Syndrome	Symptoms	Diagnosis	Treatment
Typhoid Fever (Enteric Fever)	Fever, headache, abdominal pain, constipation, rose spots, malaise.	Blood culture, stool culture, Widal test.	Antibiotics: Ciprofloxacin, ceftriaxone.
Gastroenteritis	Diarrhea (often bloody), vomiting, abdominal cramps, fever.	Stool culture (PCR and serotyping).	Rehydration, antibiotics in severe cases.
Bacteremia	Fever, chills, organ-specific symptoms (e.g., osteomyelitis, endocarditis).	Blood culture, PCR.	Antibiotics: Ciprofloxacin, ceftriaxone.
Carrier State	Asymptomatic but shedding bacteria in stool for prolonged periods.	Stool culture for prolonged period.	Antibiotics for prolonged carriers if symptomatic.

Salmonella Transmission, Prevention, and Control

Mode of Transmission	Details
Fecal-Oral	Ingestion of contaminated food or water, direct contact with infected individuals.
Zoonotic Transmission	Contact with infected animals (especially reptiles, poultry, and cattle).
Person-to-Person	Through contaminated hands, food, or surfaces.

Prevention	Details
Vaccination	Vaccination for typhoid fever (Vi polysaccharide vaccine).
Food Safety	Proper cooking, handling, and storage of food, especially poultry and eggs.
Hygiene	Handwashing, especially after handling animals or food, and before eating.
Water Safety	Ensure proper treatment of drinking water to eliminate pathogens.

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