Bacterial Gastroenteritis

Gastrointestinal Tract Module

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Outlines

- **Gastroenteritis:** definition, clinical presentation, Classification, diagnosis and treatment.
- Shigella
- Salmonella
- Diarrheagenic *E. coli*
- *Clostridioides difficile*

1. Introduction: Definitions

- **Gastroenteritis:** inflammation of the gastrointestinal tract that usually manifests with acute diarrhoea, vomiting, and/or abdominal pain
- **Infectious gastroenteritis:** gastroenteritis caused by pathogens; **most commonly viruses**, but can also be caused by **bacteria**, parasites, and fungi.

1. Introduction: Definitions

- **Diarrhoea:** A condition defined either as the presence of ≥ 3 bowel movements per day, or water content exceeding 75%.
- **Acute diarrhoea** lasts for no longer than 2 weeks and is typically caused by viral or bacterial infection or food poisoning.
- **Chronic diarrhoea** is often caused by underlying gastrointestinal or endocrinologic conditions.

Gastroenteritis: Clinical features

- Common symptoms:
  - Abdominal pain and cramps
  - Diarrhoea (watery, mucoid, or bloody)
  - Vomiting
  - Fever
  - Anorexia
  - Headache, Myalgia
- Evidence of severe dehydration: Tachycardia, Hypotension

Gastroenteritis: Modes of infection

- Foodborne intoxication:
  - bacteria grow and release toxins into the food → toxin ingestion, not necessarily the live bacteria.
  - the toxin is already present and acts immediately → a very short incubation period — often just 1 to 6 hours
- Faecal-oral (bacterial contamination)
  - bacteria contaminate food → microorganisms colonize the gut, multiply, and cause disease.
  - The incubation period is longer — hours to days — and fever is typically present.

Gastroenteritis: Diagnosis

- Clinical diagnosis:
  - Perform a thorough history and physical examination.
  - Evaluate for risk factors for specific pathogens.
  - Evaluate for **clinical features of dehydration** and hypovolemia.

Gastroenteritis: Diagnosis- Laboratory studies

- **General testing:**
  - Generally, only done in severe disease with evidence of dehydration
  - Basic metabolic panel → assesses for acute kidney injury and electrolyte abnormalities
- **Stool testing:** Most patients **do not require stool testing**.
  - Guided by clinical history and findings:
    - Blood or pus in the stool
    - Persistent fever, severe symptoms
    - Prolonged course
    - High-risk patients

Gastroenteritis: Diagnosis- Laboratory studies

- Faecal leukocytes or lactoferrin → inflammatory diarrhoea
- Stool culture and polymerase chain reaction (PCR) panel
- Stool ova and parasites
- *C. difficile* toxin enzyme immunoassay

Gastroenteritis: Treatment

- **Supportive care:**
  - Most infections are self-limiting and only require oral rehydration therapy.
  - Intravenous (IV) fluid hydration may be required for severe disease.
  - Oral and IV solutions should contain replacement electrolytes.
- **Antidiarrheal agents (loperamide):**
  - ↓ Gut motility
  - But, it can delay the excretion of the causative pathogens or toxins

    → **contraindicated** in diarrhoea with fever, bloody or mucoid stool.

Gastroenteritis: Treatment

- Antibiotic therapy:
  - Not routinely used
  - The decision to use antibiotics is often empirical
  - Frequently used antibiotics: Fluoroquinolones, Azithromycin

Gastroenteritis: Classification

	Inflammatory	**Non-inflammatory**
Mechanism of diarrhoea	The bacteria invade the intestinal mucosa or produce cytotoxins that damage the mucosal lining directly.	Adhesion of bacteria to the gut lining, or production of enterotoxins that disrupt normal fluid secretion.
Presentation	Dysentery (blood or mucus) or inflammatory diarrhoea	Watery diarrhoea
Stool findings	Faecal polymorphonuclear leukocytes Increased faecal lactoferrin	No faecal WBCs no increase in faecal lactoferrin

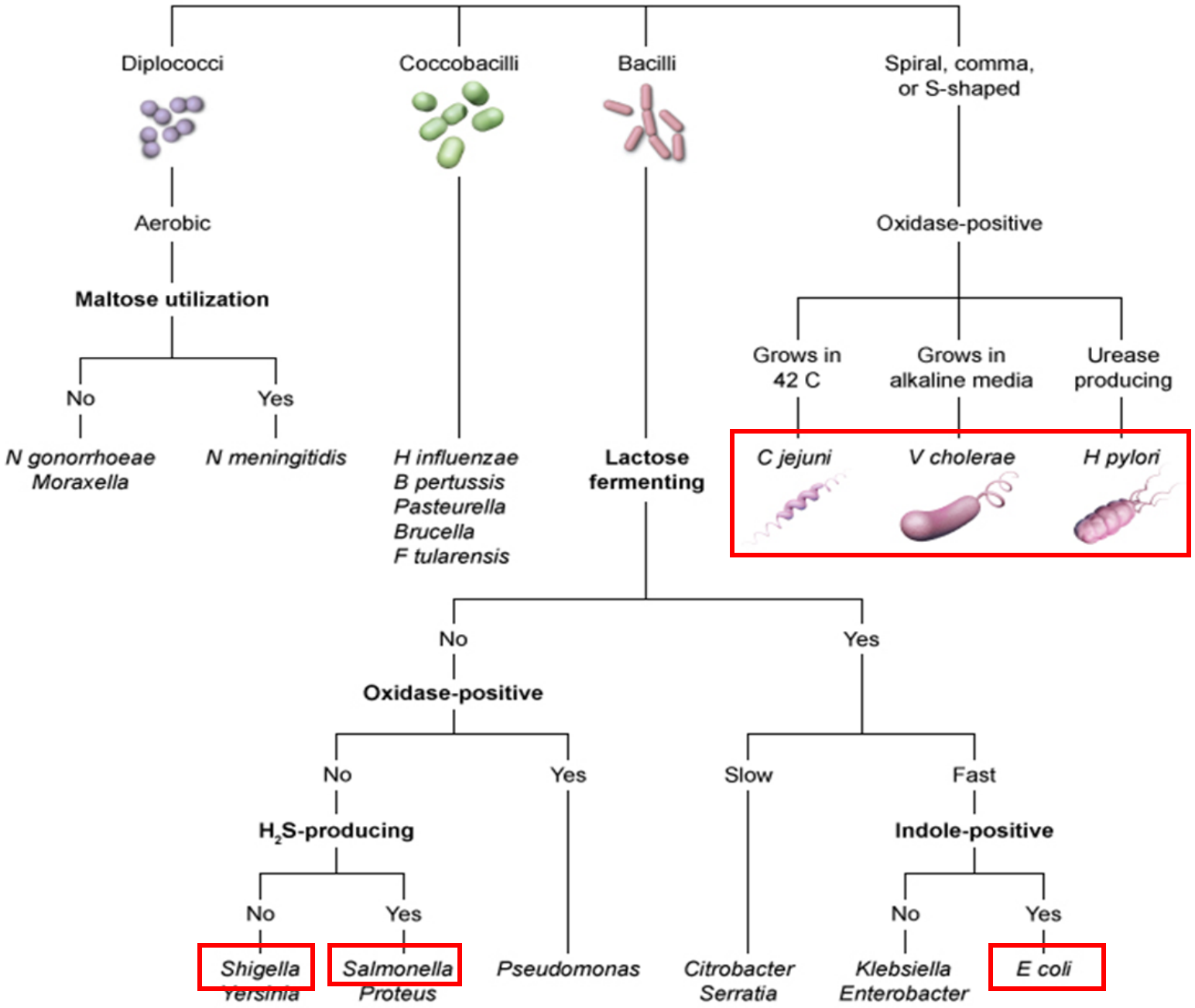
Gastroenteritis: Classification

	Inflammatory	**Non-inflammatory**
Pathogen involved	**Shigella** **Salmonella** *Campylobacter jejuni* **Enterohemorrhagic *E. coli*** **Enteroinvasive *E. coli*** ***Clostridioides difficile***	*Vibrio cholerae* Enterotoxigenic *E. coli* *Clostridium perfringens* *Bacillus cereus* *Staphylococcus aureus* Rotavirus Norovirus Enteric adenoviruses

Enterobacteriaceae (Enteric Bacteria)

- This large family includes many medically important gram-negative bacteria, it includes *E. coli*, Salmonella, Shigella, Klebsiella, Proteus, Enterobacter.
- Key Characteristics:
  - Morphology: Gram-negative rods (bacilli), non-spore forming
  - Facultative anaerobes
  - Ferment glucose
  - Oxidase negative (key distinguishing feature from other gram-negatives like Pseudomonas)
  - Catalase positive
  - Reduce nitrates to nitrites

Gram-negative bacteria



Inflammatory Gastroenteritis: Shigellosis (bacillary dysentery)

- **Pathogens:** Shigella is Gram-negative non-lactose-fermenting bacilli.
- Shigella is never a part of the intestinal flora (always pathogenic).
- The genus shigella is divided into four serological groups according to (O) antigen: *Shigella dysenteriae, Shigella flexneri, Shigella sonnei, Shigella boydii.*

Inflammatory Gastroenteritis: Shigellosis (bacillary dysentery)

- **Transmission:** Faecal-oral, Foodborne (unpasteurized milk products and raw, unwashed vegetables), Contaminated water
- Shigella is highly contagious and has an extremely low infectious dose — as few as 10 to 100 organisms are enough to cause disease.

Inflammatory Gastroenteritis: Shigellosis (bacillary dysentery)- Pathophysiology

A. Invasion: The main pathogenic process of bacillary dysentery. Leading to necrosis, superficial ulceration & bleeding. There is NO blood invasion .

B. Toxin production:

- Exotoxin (Shiga toxin): This is produced by *Sh. dysenteriae*.
- It acts as enterotoxin causing diarrhoea and as a neurotoxin causing meningism.
- The toxin may damage blood vessels and may cause renal failure seen in haemolytic uraemic syndrome (HUS).

Inflammatory Gastroenteritis: Shigellosis (bacillary dysentery)- Clinical presentation

- Lasts 2–7 days
- Fever
- Abdominal cramping
- Tenesmus (urgency to defecate)
- Inflammatory diarrhoea with mucus, pus, and blood

Inflammatory Gastroenteritis: Shigellosis (bacillary dysentery)- Treatment

- Usually resolves spontaneously
- Rehydration and electrolyte replacement may be needed.
- Antibiotics shorten the duration of symptoms and pathogen shedding In the stool: Fluoroquinolones or Azithromycin
- **Complications**
  - HUS → due to Shiga toxin, often seen in children
  - Acute blood loss → mucosal ulcerations

Inflammatory Gastroenteritis: Salmonellosis

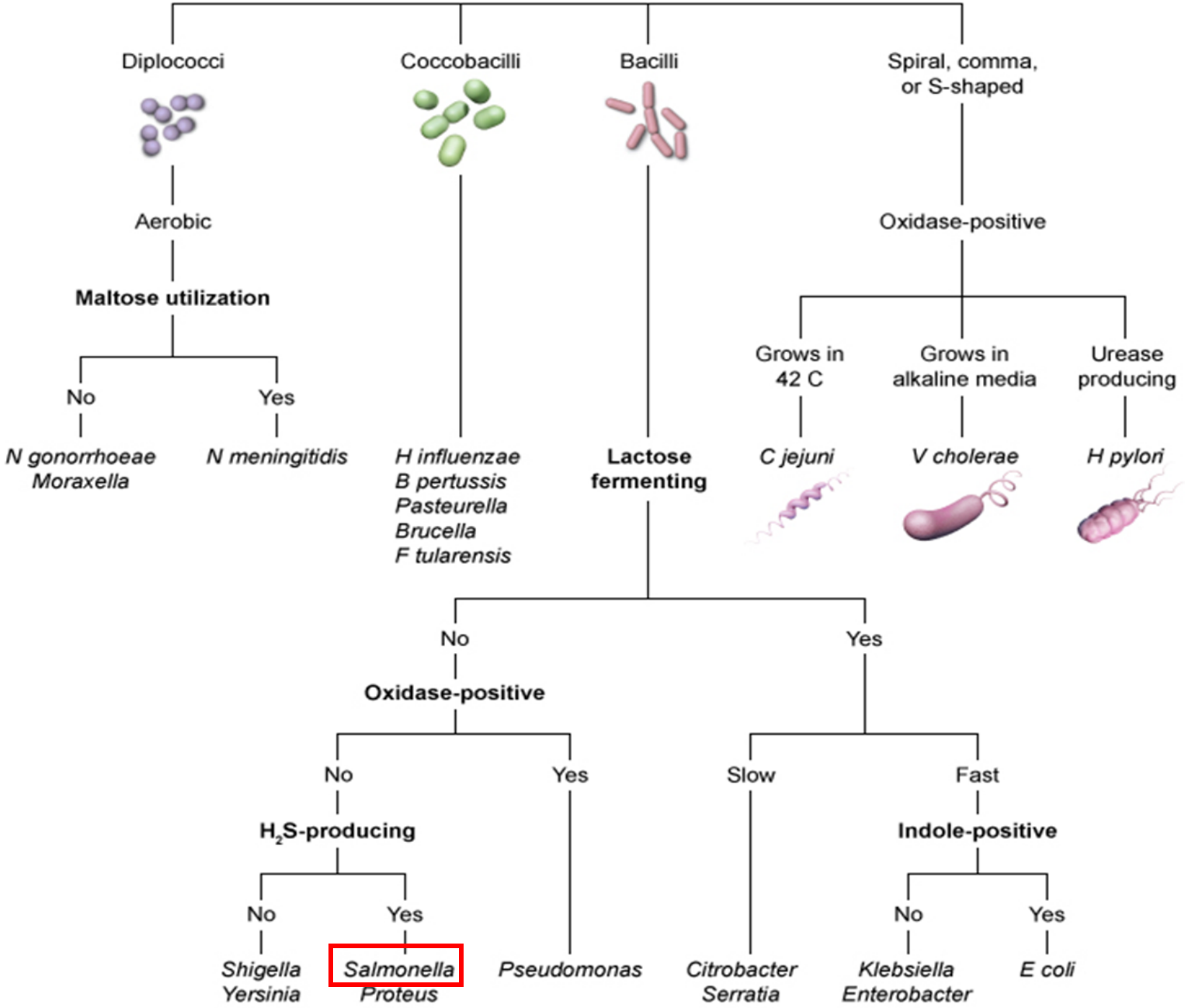
Pathogen: Salmonella are gram-negative, non–lactose fermenting bacteria.

- Produce **hydrogen sulphide** and are **motile** (unlike Shigella)
- Salmonella (like Shigella) is never a part of the intestinal flora **(always pathogenic)**

The main species of medical importance:

- Typhoidal Salmonellae (Causing enteric fever):
  - *Salmonella typhi* .
  - *Salmonella paratyphi* A, B, C.
- Non-typhoidal Salmonellae (Causing enterocolitis):
  - *S. typhimurium* .
  - *S. enteritidis*

Gram-negative bacteria



Inflammatory Gastroenteritis: Salmonellosis

- **Transmission:** foodborne (poultry, raw eggs, and milk).
- **Clinical presentation:**
  - lasts 3–7 days
  - Inflammatory watery diarrhoea (occasionally bloody)
  - Fever, chills
  - Headache
  - Myalgias
  - Severe vomiting
  - Abdominal cramping

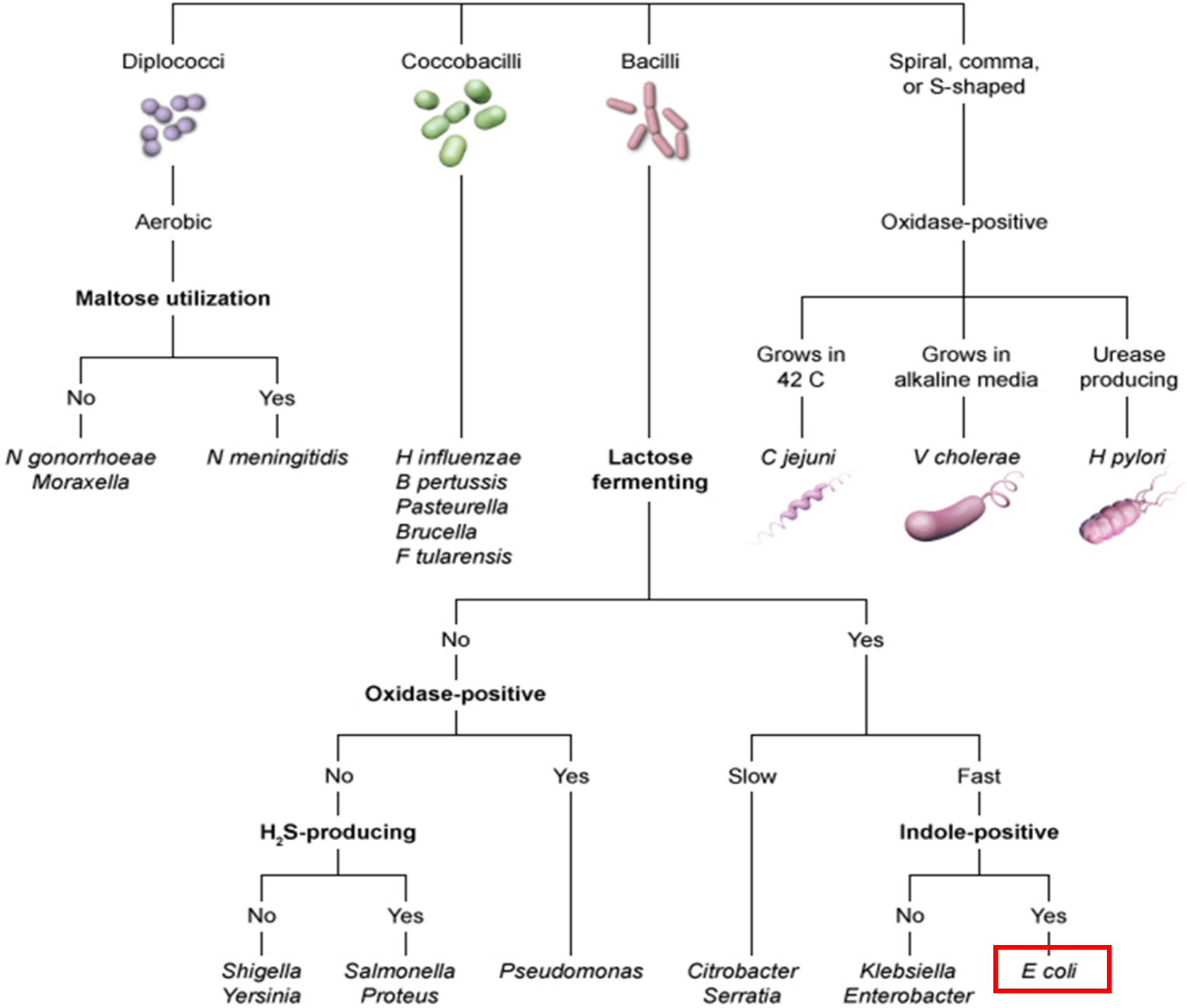
Inflammatory Gastroenteritis: Salmonellosis-Treatment

- Supportive care
- Antibiotic therapy:
  - Not routinely indicated
  - Antibiotic treatment for salmonellosis **prolongs faecal excretion** of the pathogen. Therefore, it is only indicated for severe nontyphoidal Salmonella infections.
  - Preferred regimens Fluoroquinolones: e.g., ciprofloxacin usually given for 7 to 10 days
- **Complications:** Bacteraemia

Inflammatory Gastroenteritis: Diarrheagenic *E. coli*

- *Escherichia coli* (*E. coli*): Gram-negative, rod-shaped, flagellated
- It has three antigens: O (somatic antigen); H (flagellar antigen); and K (capsular or virulence antigen).
- Because there are more than 150 O, 50 H, and 90 K antigens, the various combinations result in more than 1000 antigenic types of *E. coli*.
- Five pathogenic groups of *E. coli* are recognized as causing diarrheal diseases through different mechanisms: ETEC, EIEC, EHEC, EPEC, and Enteroaggregative E .coli (EAEC).

Gram-negative bacteria



Inflammatory Gastroenteritis: Diarrheagenic E.coli- Enterohemorrhagic *E. coli* (EHEC)

- Subset of Shiga toxin-producing *E. coli* (STEC)
- Mainly associated with outbreaks following ingestion of undercooked hamburger at fast food restaurants.
- Enterohemorrhagic *E. coli* results in symptoms via Shiga toxin production:
  - Localized effect: inhibits enterocyte protein synthesis → enterocyte death → inflammation causing bloody diarrhoea
  - Systemic effect: vascular endothelial injury in glomeruli → microthrombi and renal dysfunction haemolytic uremic syndrome (HUS).
    - O157:H7 is the strain most commonly associated with HUS worldwide.

Inflammatory Gastroenteritis: Diarrheagenic E.coli- Enterohemorrhagic *E. coli* (EHEC)

- Haemolytic–uremic syndrome (HUS) is a life-threatening complication which occurs when Shiga toxin enters the bloodstream. This syndrome consists of haemolytic anaemia, thrombocytopenia, and acute renal failure.

- **Treatment:**
  - Provide management for STEC-positive illness to prevent and monitor for HUS.
  - Avoid anti-peristaltic agents since they increase the risk of systemic complications.
  - **Antibiotic therapy is contraindicated** → antibiotic treatment **triggers the** release of **MORE Shiga toxin**

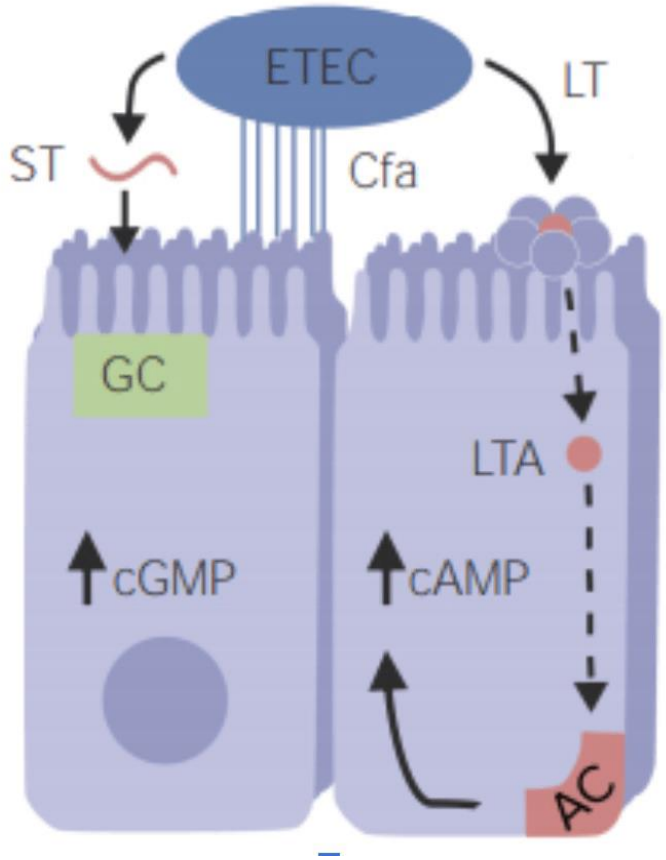
Inflammatory Gastroenteritis: Diarrheagenic *E.coli*- Enteroinvasive *E. coli* (EIEC)

- **Pathophysiology**: EIEC invades an intestinal cell, multiplies intracellularly, and extends into the adjacent intestinal cells. Inflammatory response → necrosis (can produce ulceration) → dysentery.
- **Clinical presentation**: Symptoms similar to shigellosis
  - Watery to bloody diarrhoea, possibly with mucus (dysentery)
  - Fever, chills, malaise
  - Abdominal cramps
  - Possibly vomiting

Non-Inflammatory Gastroenteritis: Diarrheagenic *E.coli*- Enterotoxigenic *E. coli* (ETEC)

- These strains are common cause of Traveler's diarrhoea. A major cause of diarrhoea among children in developing countries.
- Pathophysiology: ETEC produces two types of enterotoxins:
  - Heat-labile enterotoxin: activation of adenylate cyclase → ↑ cAMP levels → ↑ chloride secretion → water efflux into the intestinal lumen → secretory diarrhoea
  - Heat-stable enterotoxin: activation of guanylate cyclase → ↑ cGMP levels → ↓ NaCl reabsorption → water efflux into the intestinal lumen → secretory diarrhoea
  - No invasion of the intestinal mucosa and no inflammation

Non-Inflammatory Gastroenteritis: Diarrheagenic E.coli- Enterotoxigenic *E. coli* (ETEC)



Non-Inflammatory Gastroenteritis: Diarrheagenic E.coli- Enterotoxigenic *E. coli* (ETEC)

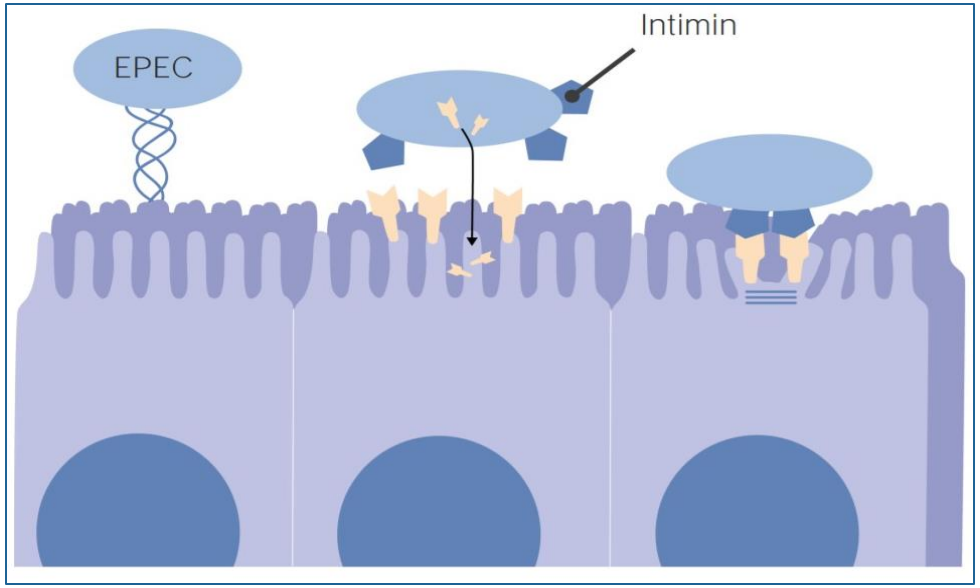
Clinical presentation: Symptoms last 3–4 days.

- Watery diarrhoea
- Abdominal cramping
- Nausea, possibly vomiting
- Fever
- Decreased appetite

Non-Inflammatory Gastroenteritis: Diarrheagenic E.coli- Enteropathogenic *E. coli* (EPEC)

- **Pathophysiology:** EPEC blocks absorption by attaching to the apical surfaces of the intestinal epithelium, causing the villi to flatten.
- No toxin production is involved.

- EPEC uses intimin adhesion molecules to adhere to the intestinal cells.
- Binding causes **brush border degeneration and loss of microvilli**. The characteristic effect of attachment and effacement is the primary cause of diarrhoea.



Inflammatory Gastroenteritis: *Clostridioides difficile*

- Clostridioides difficile (C. difficile; formerly known as Clostridium difficile) is a gram-positive bacillus.
- Gram-positive bacillus, **obligate anaerobe**
- Forms environmentally-resistant spores (capable of withstanding heat, antibiotics, and acid)
- Most common cause of **antibiotic-induced colitis** (especially associated with clindamycin)

Inflammatory Gastroenteritis: *Clostridioides difficile*- Pathophysiology

- The use of broad-spectrum antibiotics (e.g., clindamycin, cephalosporins, fluoroquinolones) disrupt the normal balance of intestinal microbiota, allowing overgrowth of *C. difficile*, which is normally suppressed.
- Spores germinate into functional bacilli in the colon and produces two major toxins:
  - Toxin A (Enterotoxin): it Targets brush border enzymes, alters fluid secretion → causing watery diarrhoea.
  - Toxin B (10 times more potent): Potent cytotoxin that damages colon epithelial cells directly. It causes pseudomembranous colitis

Inflammatory Gastroenteritis: *Clostridioides difficile*- Clinical presentation

- Foul-smelling non-bloody diarrhoea
- Cramping abdominal pain
- Fever, nausea, and vomiting
- Dehydration
- Fulminant colitis: acute abdominal distention and pain, signs of sepsis (hypotension, tachycardia, change in mental status).

Inflammatory Gastroenteritis: *Clostridioides difficile*- Treatment

- Stop all non-essential antimicrobial agents.
- Antibiotic treatment is indicated in all symptomatic patients with CDI and should be guided by the severity of CDI.
- First-line: Oral fidaxomicin or oral vancomycin
- Faecal microbiota transplantation may be indicated in recurrent CDI, severe CDI, or fulminant CDI refractory to antibiotic therapy.

Complications

Toxic megacolon: a life-threatening, acute dilation of the colon associated with systemic toxicity.

Peritonitis, perforation, abscess formation, sepsis

Thank you