Profile: Enterobacteriaceae Family

- Moderate-sized, **non-spore-forming Gram-negative rods.** (only Gram +ve rod can form spore.)
- **Facultative anaerobes.** (Survive O_2 ✓ O_2 ✗)
- **Ubiquitous** in nature and **normal intestinal flora.** → Animals and humans.

Biochemical Baseline

- **Glucose fermenters**
- **Nitrate reducers**
- Catalase positive (+)
- Oxidase negative (-) (* Facultative Bacteria) (H-antigen.)
- Most are **motile** via flagella (exceptions: *Klebsiella*, *Shigella*, *Yersinia*)

Diagnostic Decision Tree (MacConkey Agar)

→ Non-selective as (Blood)

MacConkey Agar Plating → Selective and differential agar

Separate

- **Lactose Fermenters** → *Escherichia coli*, *Klebsiella*, *Enterobacter*
- **Non-Lactose Fermenters** → *Salmonella*, *Shigella*, *Yersinia*, *Proteus*

The Bacterial Toolkit

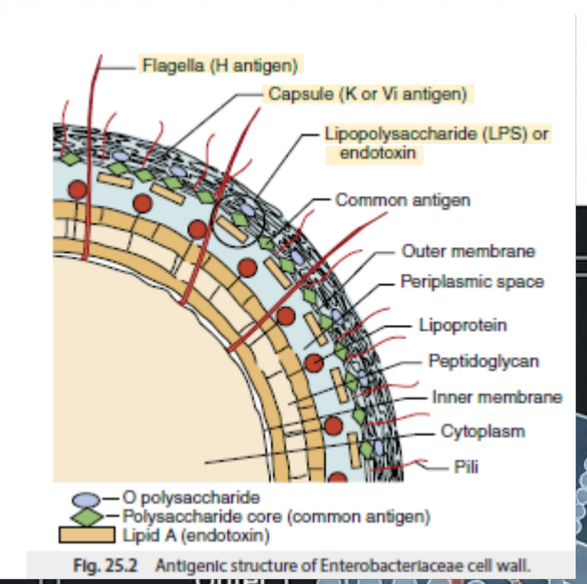


Fig. 25.2 Antigenic structure of Enterobacteriaceae cell wall.

Virulence Toolkit

Endotoxin (Lipid A): Triggers fever, inflammation, shock, and DIC. Shared by aerobic Gram-negative rods.

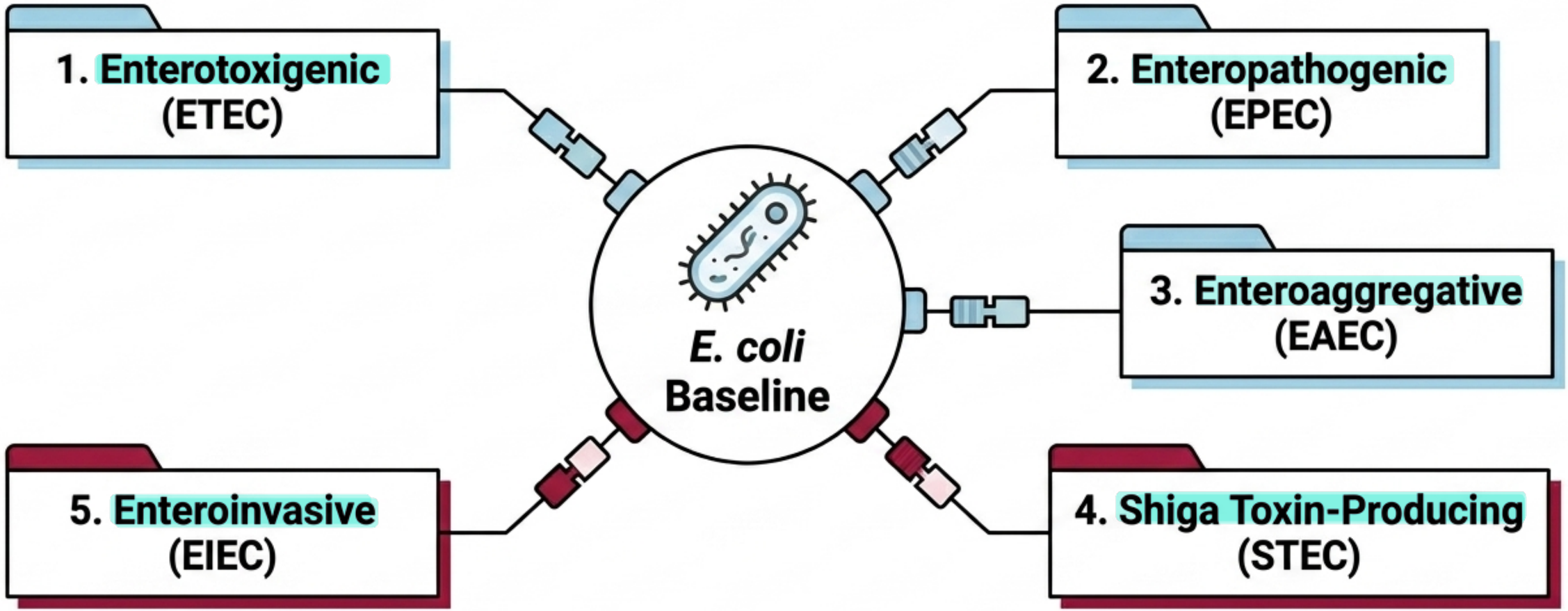
Adhesins: Essential for binding to host epithelial surfaces.

Exotoxins: Enterotoxins and Shiga toxins that drive diarrheal syndromes.

Escherichia coli: The Shape-Shifter

Associated with Gastroenteritis and Extra-intestinal infections.

The Commensal turned Pathogen: *E. coli* is the most common member of the genus *Escherichia*. While normally harmless, disease is dictated by the acquisition of specialized virulence genes (adhesins and exotoxins).



The Clinical Dossier

The *E. coli* Spectrum I: Watery Diarrhea

Watery Diarrhea

ETEC (The Traveler)	EPEC (The Infant Threat)	EAEC (The Persistent Biofilm)
• Acquired via contaminated food/water.	• Important cause of infant diarrhea in developing nations.	• Associated with chronic/ persistent diarrhea in children and immunocompromised.
• Pathogenesis is plasmid-mediated: Heat-Labile toxin (cAMP) and Heat-Stable toxin (cGMP) cause hypersecretion of fluids.	• Pathogenesis: Locus of enterocyte effacement (LEE) pathogenicity island.	• Pathogenesis: Adheres in a **stacked-brick pattern**.
• Presentation: Watery, non-bloody **traveler's diarrhea**. Self-limited. No tissue invasion.	• Creates **Attaching & Effacing (A/E) lesions** that destroy microvilli, causing malabsorption. Not toxin-mediated.	• Forms mucus biofilms causing inflammatory injury and nutritional compromise.

* Resemble mild cholera.
* Fecо-oral.
* Small intestine.
*Similar to cholera toxin.
*Cluster of virulence genes.
and electrolytes.
(Low grade fever)
* Tx
↳ So no blood.
* and growth effect in vulnerable children.

Invasive & Bloody

*Large intestine.

The *E. coli* Spectrum II: Invasive & Bloody

STEC / EHEC (The Toxin Producer)

↳ strain of E.coli produce toxin called Shiga toxins.

↳ Hemorrhagic colitis.

Other names → VTEC → verocytotoxin

Evolution: Evolved from EPEC (has A/E lesions).

Mechanism: Produces **Shiga toxins** (Stx1, Stx2) disrupting protein synthesis.

Classic Presentation: Initial watery diarrhea progressing rapidly to bloody diarrhea and **hemorrhagic colitis**. Little to no fever.

↳ Help to distinguish it from other invasive bacterial dysenteries.

Reservoir/Vector: Cattle; undercooked beef, unpasteurized products, **O157:H7** serotype.

- Vegetables
- person to person
- Contaminated fruit

*Most common serotype.
*Doesn't ferment sorbitol.

EIEC (The Cellular Invader)

Mechanism: Plasmid-mediated **invasion and replication** directly within colonic epithelial cells (resembles Shigella).

Classic Presentation: Watery diarrhea progressing to a **dysentery** form with fever, cramps, tenesmus, and scant bloody/mucoid stools.

Insight: Shows that not all E. coli diarrhea is toxin-mediated. or watery diarrhea.

Pathogenesis Focus: STEC & HUS

↑urea → renal injury

The most feared complication of STEC infection is hemolytic uremic syndrome

Step 1

Shiga Toxin (Stx) in GI tract disrupts intestinal microvilli.

↓ absorption.

Step 2

Toxin enters bloodstream, targeting vascular endothelium.

Step 3

Direct endothelial injury (inhibition of protein synthesis) triggers inflammation and vascular thrombogenicity.

Step 4

The **HUS Triad.** Organ damage primarily affecting the kidneys and brain.

Microangiopathic hemolytic anemia Break RBCs

Thrombocytopenia ↓platelets

Acute kidney injury

↳ ST x 2

Clinical Warning Banner: Affects 5-10% (especially children <10 and older adults). Mortality is 3-5%.

Crucial Rule: Antibiotics and antimotility agents are generally avoided in STEC! Early IV fluids reduce renal failure risk.

Salmonella spp: The Great Divergence

Salmonella enterica (Gram-negative, facultative anaerobe, survives in macrophages)

Non-Typhoidal *Salmonella*

- Reservoirs: **Animals** and **humans** (poultry, reptiles, livestock).
- Pathology: **Localized intestinal inflammation.**
- Clinical Outcome: **Self-limiting gastroenteritis.** * food borne gastroenteritis.

* Ciprofloxacin → for Susceptible strain.
there's another.

Antibiotic

- High risk patients.
- Severe disease.

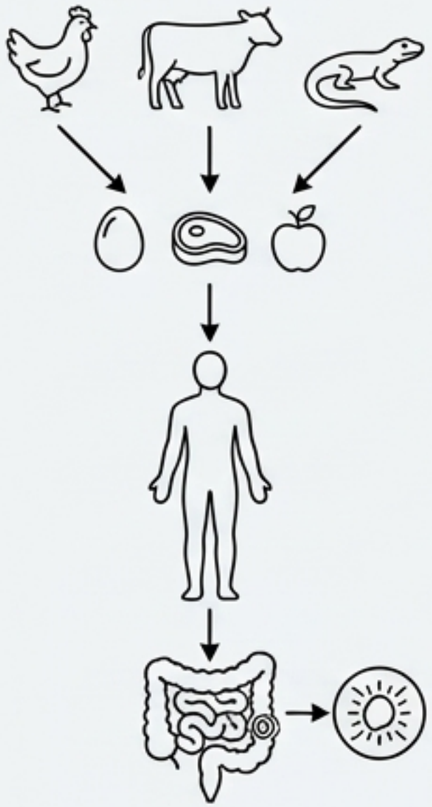
* uncomplicated cases treated with
→ fluids
→ supportive care.

Typhoidal *Salmonella*

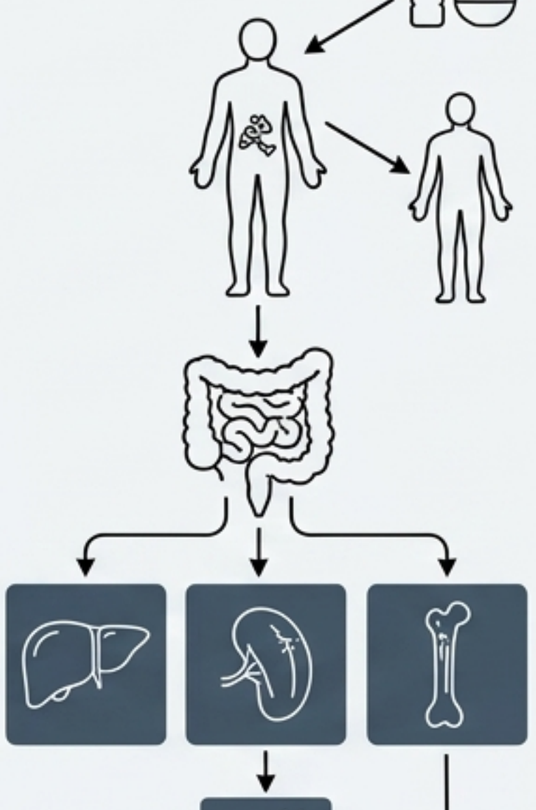
- Reservoirs: Strictly **Humans.**
- Pathology: Adapted for **systemic spread** and **deep-tissue survival.**
- Clinical Outcome: **Enteric** (Typhoid/Paratyphoid) **fever.** (39–40°)
  → systemic febrile illness.

(A, B, C)

A Tale of Two Syndromes



	Non-Typhoidal Gastroenteritis	Enteric (Typhoid) Fever
Organisms	S. Typhimurium, S. Enteritidis	* Don't cause disease in non-human hosts. S. Typhi, S. Paratyphi
Transmission	Contaminated poultry/eggs/reptiles, high infectious dose	Human feces/water (infected food handlers), very low infectious dose so person—person is common.
Pathogenesis	Invades M cells over Peyer patches, causing localized inflammatory diarrhea	Engulfed by macrophages, systemic transport to liver/spleen/bone marrow. Can establish chronic gallbladder carriage. → can survive there.
Clinical Presentation	Rapid onset (hours/days), non-bloody diarrhea, self-limiting	Delayed onset (10-14 days), systemic sustained high fever (39-40°C), headache, rose-colored spots. → Enteric fever
Primary Diagnosis	Stool culture	Blood culture (early phase) Stool culture (+) later.



Clinical Dossier

Bloody diarrhea syndrome

Shigella: The Low-Dose Invader

* Antibiotic reduce//shorten transmission/illness.
* Antimotility should be Avoided As it can worsen invasive diarrhea.

Pathogen Profile

* Shiga toxin especially from S. dysenteriae contribute to Severe disease and HUS.

Profile: *Shigella* spp. (*S. dysenteriae, S. flexneri, S. boydii, S. sonnei*). Nonmotile.

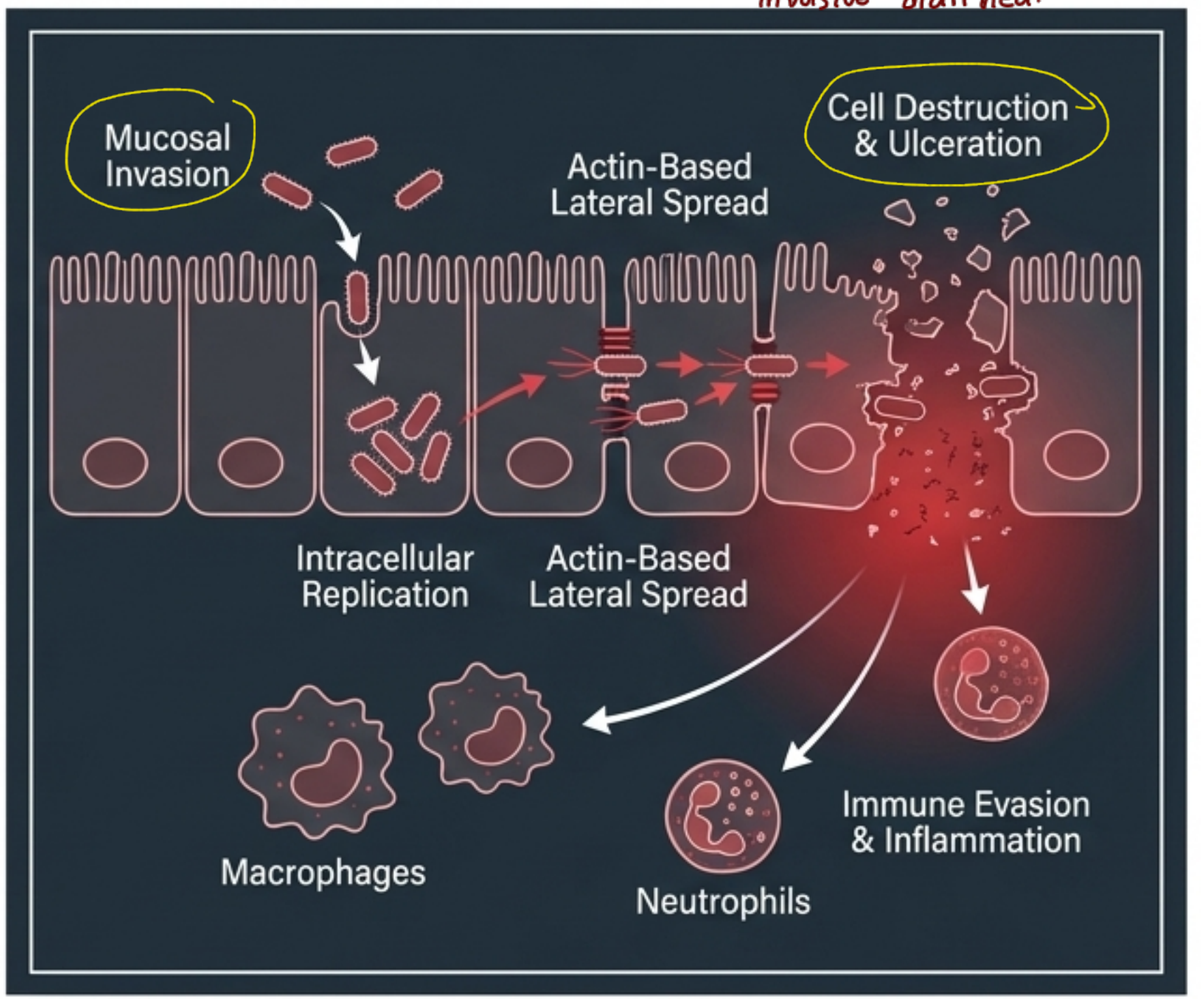
* Feco-oral.

The Transmission Threat: Humans are the only reservoir. It survives gastric acidity exceptionally well, resulting in an **extremely low infectious dose**. High person-to-person spread in childcare centers and institutions.

Pathogenesis: Invades colonic mucosa -> Replicates in host cell cytoplasm -> Spreads cell-to-cell -> Causes severe mucosal ulceration and intense inflammation.

*Shigellosis ↘

Clinical Dysentery Syndrome: 1-3 day incubation. Watery diarrhea rapidly progresses to **frequent, small-volume bloody diarrhea with mucus**. Cardinal features: high fever, abdominal pain, and **tenesmus** (constant feeling of needing to pass stool). Stool is packed with neutrophils and erythrocytes.

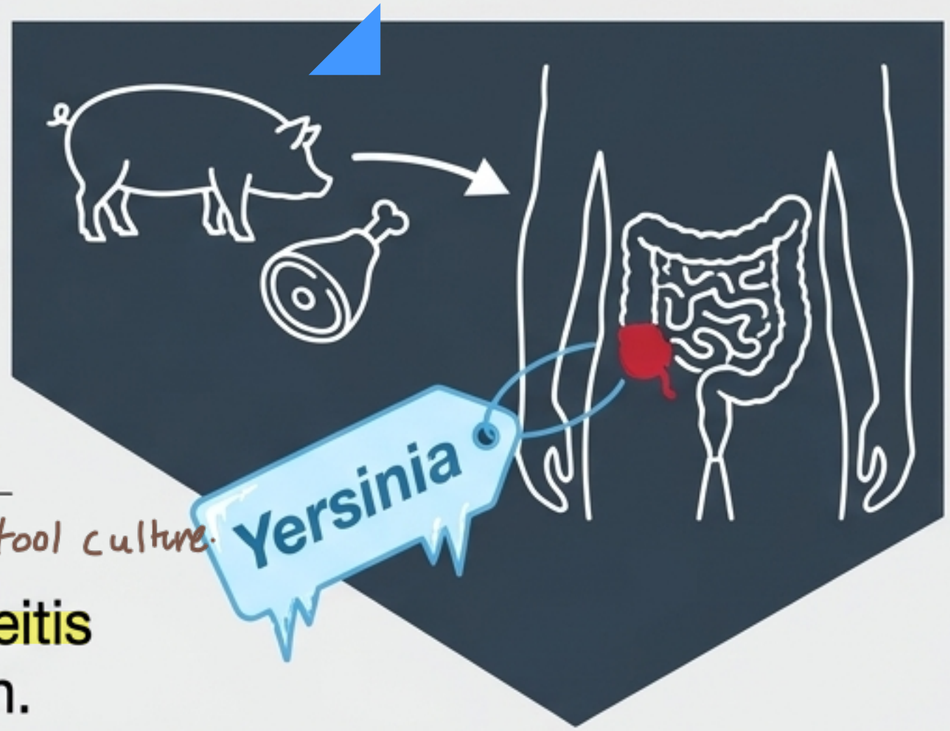


Yersinia: The Cold-Climate Mimic

Yersinia pseudotuberculosis &

Profile: *Yersinia enterocolitica*

Zoonotic pathogen associated with contaminated water and food (especially pork products).



The Cold-Climate Trait:

Uniquely capable of growing at refrigerator temperatures (critical food safety anomaly).

Pathogenesis: may need special culture condition // Stool culture

Invades intestinal lymphoid tissue causing terminal ileitis and mesenteric lymphadenitis. 1 to 10 day incubation.

The Clinical Mimic:

In older children and adults, it causes severe right lower quadrant pain and fever. Because of this specific localization, it clinically mimics acute appendicitis (**pseudoappendicitis**).

Most infections are self-limited, but severe or invasive disease may require antimicrobial therapy.

Cronobacter sakazakii: The Neonatal Threat

MMWR
Morbidity and Mortality Weekly Report

- **Profile:** *Cronobacter sakazakii* (Formerly *Enterobacter sakazakii*).

- **The Environmental Niche:** Naturally found in the environment with a unique capacity to survive in low-moisture, dry foods.

- **The Primary Vector: Powdered infant formula/milk,** herbal teas, and starches.

- **The Clinical Threat:** Devastating cause of neonatal gastrointestinal and systemic infection. Presents as sepsis, meningitis, or necrotizing enterocolitis.

- **Vulnerable Populations:** Premature infants, low-birth-weight infants, and immunocompromised neonates are at extreme risk. Prevention relies entirely on strict preparation and storage protocols in neonatal units.

The Clinical Diagnostic Matrix

Pathogen	Vector	Buzzword	Mechanism
ETEC	Water/Travel	**Watery**, non-bloody	LT/ST toxins, cAMP/cGMP
EPEC	Fecal-oral	Infant **watery**	A/E lesions, LEE
EAEC	Fecal-oral	**Persistent diarrhea**	Stacked-brick biofilm
STEC	Cattle/Beef	Hemorrhagic colitis, HUS	Shiga toxin, O157:H7
Non-Typhoidal Salmonella	Poultry/Reptiles	Inflammatory **diarrhea**	MacConkey non-fermenter
Typhoidal Salmonella	Human handlers	**Rose spots**, systemic fever	Macrophage survival, gallbladder carriage
Shigella	Daycare/Human	**Dysentery, Tenesmus**	Low infectious dose, cell-to-cell spread
Yersinia	Pork/Cold foods	**Pseudoappendicitis**	Refrigerator growth

Treatment & Prevention Paradigms

Check (Do) / Cross (Don't)	Cross (Don't)	Check (Do)
The Antibiotic Paradox	**The Antimotility Rule**	**Public Health & Prevention**
• **DO NOT USE:** For **STEC** (increases the risk of hemolytic uremic syndrome) or uncomplicated non-typhoidal **Salmonella** gastroenteritis. • **DO USE:** For severe **Shigellosis, Typhoid/Enteric Fever**, or invasive infections (First-line: **Ciprofloxacin, Azithromycin, Ceftriaxone**). Susceptibility testing is mandatory due to rising resistance.	• **AVOID:** Antimotility agents (like **loperamide**) should be universally avoided in **invasive or bloody diarrheas** (**Shigella, STEC**) as they retain toxins and pathogens in the gut, worsening the disease.	• Safe **food handling** (cooking poultry/eggs thoroughly, avoiding cross-contamination). • Safe **water and sanitation** infrastructure. • Identification of **chronic typhoid carriers**. • **Vaccination** for travelers to **typhoid-endemic regions**.