

### Lec. 3 micro summary , by Ghada Alzoubi

#### Enterobacteriaceae

→ Gram **negative bacilli** , habitat is the **intestinal tract** of humans and animals.

→ **all motile** with peritrichous flagella **EXCEPT** for Shigellae, Yersinia and Klebsiella

→ **facultative anaerobes** → **oxidase negative** , **catalase positive**, reduce nitrate to nitrite

→ **LPS**: component of the G-ve bacterial cell wall and is an **endotoxin**

→ **Antigenic Structure of Enterobacteriaceae**:

**O antigens** “ heat-stable” , [with capsule]**K (capsular) antigens** “ heat- labile” ,  
**H (flagellar) antigens**” motile”

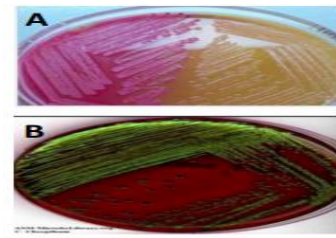
→ **Salmonella** serotype Typhi is called Vi antigen because it’s not only resistant to serum killing but is also a virulence antigen

→ produce **Colicins** “bacteriocins produced by E. coli.”

- 1) **E.Coli** : cause disease by 3 ways : **gain** virulence factors “plasmids/phages” +**opportunistic** infection +**introduced** into a sterile site “ urine +blood”

-Main cause of **UTI** -oxidase **negative** and lactose fermenters

	Lactose fermenters	Non-lactose fermenters
MacConkey (pic A)	Pink	Colorless
EMB (pic B)	Black dots on colony (nucleated) (E. coli specifically forms <b>green sheen colonies</b> )	Colorless



#### E coli-associated diarrheal diseases (cause gastroenteritis):

- A) **Enteropathogenic E coli (EPEC)** : **infantile** diarrhea, **outbreaks** of explosive diarrhea in nurseries.

-**Pathogenesis**: requires two important factors for attachment and effacement “EPEC adherence factor (EAF) +effacement (LEE) “

-**feco-oral** as kids are unhygienic , **watery diarrhea** “contain mucus”

-**self limiting** , but can prolonged or chronic “may need hospitalization”

- can be **cured** by antibiotic treatment

- B) **Enterotoxigenic E coli (ETEC)** : **traveler’s diarrhea**

-**pathogenesis**: : ETEC **colonization factors** [ CFAs ]+**produces toxins**[ ST: cGMP + LT: cAMP ]

-**Both toxins** result in hypersecretion of fluids and electrolytes and poor absorption of sodium→ watery diarrhea.

-**PREVENTION**: **ingestion of bismuth subsalicylate suspension**

C) **Shiga toxin-producing E coli (STEC/EHEC) /VTEC/EHEC:**

- Most common E. coli strain that causes gastroenteritis
- The most common of all the E coli serotypes that produce Shiga toxin is O157:H7
- Toxins: Shiga-like toxin 1 and toxin 2 -STEC is NOT INVASIVE -develop HUS
- clinically : Colonic edema and an initial non-bloody then may developed into bloody diarrhea “contains RBCs, WBCs, some mucous”
- self limited , not invasive= no fever
- Dx: test for shiga toxin “ EIAs” , Sorbitol MacConkey agar plate “EHEC is the only E. coli that doesn’t ferment sorbitol” → it won’t grow on the plate
- Treatment : Antibiotics are contraindicated because they increase the risk for HUS , Anti-motility drugs (reduce motility of colon) and opioids are also contraindicated

D) **Enteroinvasive E coli (EIEC) :** INVASIVE (so it causes bloody diarrhea and fever) , most commonly in children in developing countries and in travelers to these countries , E. coli are lactose fermenters EXCEPT EIEC.

Similar to Shigella, EIEC strains are non-lactose or late lactose fermenters and are nonmotile.

E) **Enteroaggregative E coli (EAEC) :** chronic diarrhea , persistent diarrhea in patients with HIV.

-Some strains of EAEC produce ST (Shiga toxin)-like toxin (EAST). {Others produce a plasmid-encoded enterotoxin that produces cellular damage (hemolysin and enterotoxin)}

-They are characterized by their specific patterns of adherence to human cells. The organisms exhibit a diffuse or “stacked-brick” pattern of adherence to small intestine epithelial cells

- ETEC, EPEC, and EAEC cause non-inflammatory diarrhea.
- EIEC, STEC cause inflammatory diarrhea

**TREATMENT:** cotrimoxazole +No single specific therapy is available. The sulfonamides, ampicillin, cephalosporins, fluoroquinolones, and aminoglycosides

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2) **Shigellae:** facultative anaerobes but grow best aerobically , gram-negative rods , produce shigellosis , humans are the only reservoir

-ferment glucose. They do not ferment lactose, but Shigella sonnei can

-non-motile” no H-antigen” -do NOT produce H<sub>2</sub>S “differentiates them from salmonella”

-developed countries, the most common serotype is S. sonnei “ group D”

-developing countries, the most common serotype is S. flexneri “ group B”

-transmission: Food, Feces, Flies, Fingers

-Group A Shigella Dysenteriae → produces type 1 shiga toxin”most severity, but not common”

-pathogenesis: essential pathologic process is invasion of the mucosal epithelial cells → resulting in later dysentery with blood and pus in stools

-toxins → Endotoxin (LPS) , Shigella Dysenteriae Exotoxin” heat-labile [neurotoxic, cytotoxic and enterotoxic]” -Opioids should be avoided in Shigella dysentery

-Dx: culture “MacConkey or EMB :appear colorless , selective agar : Hektoen enteric agar or Salmonella –Shigella agar) , Serology is NOT used , rectal swab / stool culture”

-treatment : Ciprofloxacin, ampicillin, doxycycline, and trimethoprim–sulfamethoxazole