Ministry of Higher Education & Scientific Research Al Muthanna University College of Veterinary Medicine Branch of Microbiology



Subject: Microbiology II Grade: 3 Lecture : 8

Microbiology II/ ENTEROBACTERIACEAE

Classification of Bacteria

This family has Gram positive and negative Cocci, Bacilli and Rods

Enterobacteriaceae Features

- Commonly present in large intestine
- Non sporing , Non Acid fast, Gram bacilli.
- A complex family of organisms, Some are non pathogenic
- A few are highly Pathogenic, Some commensals turn out to be pathogenic. as

in UTI after catheterization.

Characters of Enterobacteriaceae

- All Enterobacteriaceae
- Gram-negative rods
- Ferment glucose with acid production
- Reduce nitrates into nitrites
- Oxidase negative
- Facultative anaerobic
- Motile except Shigella and Klebsiella

- Non-capsulated except Klebsiella
- Non-fastidious
- Grow on bile containing media (MacConkey agar)

Basic Tests : Basic characters

- Catalase +
- Oxidase –
- Reduce nitrates,
- All are Gram negative and non spore forming.
- Wide diversity / Antigenic heterogeneity

Classification of Enterobacteriaceae

Enterobacteriaceae is divided to

- 1- Lactose fermenters E. coli, Citrobacter, Klebsiella, Enterobacter
- 2- Non-lactose fermenter Salmonella, Shigella Proteus, Yersinia

There are several selective and differential media used to isolate distinguishes between LF & LNF

The most important media are:

- MacConkey agar
- Eosin Methylene Blue (EMB) agar
- Salmonella Shigella (SS) agar
- In addition to Triple Sugar Iron (TSI) agar

Enterobacteriaceae Taxonomical

- Tribe I Escherichia: Genus
 - 1- Escherichia,
 - 2- Edwardsville
 - 3- Citrobacter
 - 4- 4 Salmonella
 - 3- 5 Shigella
- Tribe II; Klebsiella: Genus
 - 1- Klebsiella
 - 2- Enterobacter,
 - 3- Hafnia
 - 4- Serratia

Tribe III ; Proteeae : Genus

- 1- Proteus
- 2- Morganella
- 4- Providencia
- Tribe IV; Erwinieae Genus: Erwinia

Highly Pathogenic Enterobacteriaceae

- Salmonella
- Shigella
- All are Lactose non fermenters,
- Produce colorless colonies on MacConkey medium
- LF also called as Para colons,

Escherichia coli

Escherichia coli is a Gram negative, facultative anaerobic, rod-shaped bacteria. It is a commensal that is found inhabiting the lower intestine of warm blooded animals. A small proportion of E. coli strains are pathogenic.

□ The harmless strains produce vitamin K and prevent colonization of the intestine by pathogenic bacteria.

□ E. coli is classified into serotypes based on cell wall (O), capsular (K), fimbrial (F) and flagellar (H) antigens. Example E. coli O157:H7

Kingdom •Bacteria

Phylum •Proteobacteria

- Class •Gammaproteobacteria
- Order •Enterobacteriales
- Family •Enterobacteriaceae

Genus •Escherichia

Species •Escherichia coli

Pathogenic Strain

- □ Enterohaemorrhagic E. coli (EHEC)
- Enterotoxigenic E. coli (ETEC)
- □ Enteroinvasive E. coli (EIEC)
- □ Enteropathogenic E. coli (EPEC)

Pathogenesis

Enterohaemorrhagic E. coli

□ This is a strain of E. coli that produces cytotoxins that disrupt protein synthesis within host cells.

□ These toxins are also called verocytotoxins or Shiga- like toxins.

□ Enterohaemorrhagic E. coli are pathogenic to humans.

□ They produce verocytotoxins that form attaching and effacing lesions on epithelial cells.

□ Infection occurs via the faecal-oral route.

□ Symptoms range from mild diarrhoea to severe bloody diarrhoea.

□ Complications include haemolytic uremic syndrome (HUS) which can lead to death if untreated.

Common serotype E. coli O157:H7

Enterotoxigenic E. coli

- □ Also known as traveler's diarrhoea.
- □ Infection leads to watery diarrhoea which may last up to a week.
- □ Symptoms include abdominal cramps, sometimes nausea and headache.

□ It establishes itself by adhering to the epithelium of the small intestine via colonization factor antigens (CFA).

□ This is followed by expression of heat stable (ST) or heat labile (LT) enterotoxins.

□ These toxins increase adenylate cyclase> CAMP levels> secretion of chloride ions and water.

Enteroinvasive E. coli

□ Transmitted through faecal-oral route.

□ Following ingestion, organisms invade epithelial cells of the intestine resulting in a mild form of dysentery.

□ Illness is characterized by presence of blood and mucus in stools of infected individuals.

□ Characteristic features of EIEC are their ability to induce entry into epithelial cells and disseminate from cell to cell.

□ EIEC infection can occur through contaminated food or water or through mechanical factors such as flies.

□ The genes required for entry is clustered on a virulence-associated invasion plasmid in EIEC strains.

Enteropathogenic E. coli

□ Following ingestion, organisms adhere to the epithelial cells of the intestine causing watery or bloody diarrhoea.

□ Adherence is mediated by EPEC adherence factor (EAF) and intimin- a nonfimbrial adhesin.

□ EPEC attach to and alter the integrity of the intestine.

□ Bloody diarrhoea is associated with attachment and an acute tissue-destructive process.

□ EPEC do not produce toxins.

□ Their virulence mechanism involves the formation of attaching and effacing lesions followed by interference with host cell signal transduction.

□ This strain is most commonly associated with paediatrics/kids.

Lab Diagnosis

Tests for identification of E. coli:

- MacConkey agar- positive
- Indole- positive
- Methyl red- positive
- Citrate- negative
- TSI (H₂S)- negative
- Lysine decarboxylase- positive
- Motility (36°C)- positive
- Acid/gas production- positive
- Lactose fermenter
- Oxidase- negative

Molecular Methods

Polymerase Chain Reaction

□ It amplifies a specific gene target.

□ The primers used in PCR may detect a characteristic virulence factor as well as other genes.

□ Closely related E. coli have genes encoding the O antigen and this can be exploited to differentiate between different strains.

□ Real-time PCR assays use fluorescence to detect presence/absence of a particular gene.

Treatment

□ Patients, especially healthy adults need no treatment for E. coli infection because it is self-limited.

□ In some studies, it has been noticed that treatment with antibiotics increases the chances of getting HUS.

□ This effect occurs because antibiotics destroys the bacterial cell wall, causing them to release even more toxin.

□ When necessary, treatment includes the replacement of fluids and electrolytes to treat or prevent dehydration.

Reference

1. http://en.wikipedia.org/wiki/Escherichia coli

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3. Stephen A., William J., Elmer K., Gary P., Paul S., Gail W. Koneman's Color Atlas & Textbook of Diagnostic Microbiology. 6th ed. China: Lippincott Williams & Wilkins; 2006.

Dr. Naer Alkaabi