BACTERIAL DISEASES OF THE INTESTINE

ESCHERICHIA COLI

- *Escherichia coli have several properties and affects that result in* disease in animals.
- They make *colonization or adhesion to the mucosa; they cause metabolic dysfunction or death of enterocytes; they affect the local or systemic vasculature;* or they promote *invasion and septicemia.*
- *According to these different properties and affects we can divide into subgroups:*
- **"Enterotoxigenic" *E. coli (ETEC)* "Enteropathogenic" *E. coli (EPEC)*** colonizes the mucosa of the intestine by a mechanism involving adhesion **(so we call "enteroadherent" *E. coli*) *do not*** produce recognized toxins, but are associated with villus atrophy.
- Some produce cytotoxins, categorized as **"Shiga toxin-producing" (STEC) = "verotoxinproducing" (VTEC), or "enterohemorrhagic" (EHEC).**

ESCHERICHIA COLI

1. Enterotoxigenic colibacillosis (LT "heat-Labile" and ST "heat-stable" toxins) (ETEC) cause secretory smallbowel diarrhea stimulated by enterotoxins produced by E. coli colonizing the mucosa of the small intestine.

2. Enteropathogenic colibacillosis (EPEC)

- ✓ Entero-adherent *E.coli* **(villus atrophy + enteritis)**
- ✓ Verotoxin-producing *E.coli* ="*Shiga toxin-producing*" **(diarrhea)**

  Enterohemorrhagic *E.coli* **(hemorrhagic enterocolitis)**

3. Edema disease of Swine
   (Hemolytic *E. coli*)
   Postweaning *E. coli* enteritis
   (Hemolytic *E. coli*)

4. Enteroinvasive *E. coli* [humans and certain other species] (EIEC) can infect mostly humans.

5. Septicemic colibacillosis is a common manifestation of *E. coli*
   Peracute septicemic and endotoxemia
   Subacute
   Chronic
   ] **Joints/meninges/eye**

PREDISPOSITION TO INFECTION

- Reduced transfer or absorption of maternal colostral immunoglobulin
- Intercurrent disease
- Debilitation

❑Enterotoxigenic colibacillosis. (ETEC)

- is one of the **major forms of diarrhea** in neonatal pigs, calves, and lambs, as well as in humans.
- The effects of E.coli depends on two major factor:
  - **ability to colonize** the intestine,
  - **capacity to produce toxins**
- *Colonization and enterotoxin production must occur together for disease to ensue.*
- Enterotoxigenic strains of *E. coli* produce 2 classes of plasmid-encoded proteins—heat-labile toxin (LT) and heatstable toxin (ST)—which act locally in the intestine to alter secretion and absorption of electrolyte and water by enterocytes.
- In contrast to the viruses, *ETEC usually* ***does not cause significant villus atrophy***
- Small clumps, or a continuous layer, of bacteria may be found on the surface of enterocytes on villi.

❑**Enteropathogenic colibacillosis.**

- **Enteropathogenic *E. coli*** (**EPEC**) are those that cause direct damage to the mucosa, through a *characteristic mechanism of attachment to, and effacement of, epithelium.*

- Due to the terms of the **location and shape** of their impact:

✓**Attaching-effacing *E. coli*** (AEEC)

✓**Enterohemorrhagic *E. Coli*** (EHEC)**-Verotoxinproducing *E. coli*** (VTEC)= *"Shiga toxin-producing"*

❑Enteropathogenic colibacillosis

- **Attaching-effacing *E. coli*** (AEEC): AEEC are pathogenic **despite failure to secrete enterotoxins or cytotoxins.**

- *The degree of diarrhea seems* ***related to the extent of bacterial colonization****, which is most consistent in lower small intestine and large bowel.*

- Enterocytes to which bacteria are adherent round up or contract, and exfoliate from the mucosa singly or in clumps, resulting in mild to severe atrophy of villi in the small bowel, and attenuation of surface cells, or microerosions, in the large intestine.

- **Enteropathogenic colibacillosis**
- A distinct subset of EPEC is the **Shiga toxin-producing *E. coli*** (STEC), also known as **enterohemorrhagic *E. coli*** (EHEC).
- In addition to their ability to attach and efface, these strains **produce cytotoxic Shiga toxins.**
- Because of their effect on Vero cells in culture, these *E. coli* are also referred to as **verotoxinproducing *E. coli*** (VTEC).

- **Enteropathogenic colibacillosis**

- In **calves <4 weeks of age,** strains of EHEC have been associated with a syndrome of ***erosive fibrinohemorrhagic enterocolitis.***

- **Microscopically,** in affected small intestine the profile of villi is ragged or markedly scalloped, and they are blunted, moderately *atrophic, or fused.*

❑Enteropathogenic colibacillosis

- **Edema disease and postweaning *E. coli* enteritis.**
- **Edema disease** is a distinct syndrome in pigs, characterized by sudden death, or the development of nervous signs associated with enteric colonization by STEC
- Mortality often approaches 100% of affected animals
- Typically, *edema is variably present in one or more sites.*

- **Postweaning *E. coli* enteritis** (coliform enteritis of weaned pigs) typically occurs during the first week or two following weaning,
- In fatal cases there may be blue-red discoloration of the skin and evidence of dehydration. Deep red *gastric venous infarcts* are present in almost all cases.

❑Septicemic colibacillosis.

- Generalized systemic infection with *E. coli* occurs commonly in ***calves***, and less commonly or sporadically, **especially among young animals** of the other domestic species.
- *Colisepticemia is most commonly a disease of neonates,* and may vary from peracute septicemia and endotoxemia resulting in sudden death, to subacute or chronic disease in which signs are related to sites of bacterial localization, especially in the meninges, joints, and eyes.

THE PORTAL OF ENTRY OF *E. COLI*

- Umbilicus in the neonate
- Upper respiratory tract
- Tonsil
- Intestine
- Nasopharyngeal route

SALMONELLOSIS

S. cholerae- suis	Swine	Enteritis, septicemia
S. typhi	Human	Typhoid fever
S. paratyphi-A	Human	Paratyphoid-A
S. schottmuelleri	Human, animals (rare)	Enteric fever, Paratyphoid-B
S. typhimurium	Rodentia, animals, human	Gastroenteritis, septicemia “food poisoning”
S. enteritidis	Human and other species	Enteritis
S. gallinarum	Avian	Enteritis, septicemia, fowl typhoid
S. give	Cattle	Enteritis
S. pullorum	Chick	Enteritis, septicemia, pullorum disease
S. abortus-equi	Horse	Abortion
S. dublin	Cattle, swine, sheep	Abortion, enteritis, septicemia, osteomyelitis, meningitis
S. anatum	Duck, monkey	Enteritis, septicemia
S. abortus-ovis	Sheep	Abortion

SALMONELLOSIS

- The **pathogenesis of salmonellosis** may be divided into several stages:

1. *entry* of the bacteria into the host and attainment of the enterocyte;
2. attachment to the surface (*colonization*); and
3. *invasion* of enterocytes

- *The ability to attach, invade, and penetrate enterocytes is crucial to virulence,* and the first step in the development of salmonellosis.

SALMONELLOSIS

- A number of known virulence factors contribute to the pathogenesis of salmonellosis, including

✓pili, or

✓fimbriae,

✓lipopolysaccharides.

Motility, associated with the presence of flagella,
Fimbriae (**pilus adhesins**) they may play a role in colonization of the gut.
The main function of LPS may be to facilitate survival in the intestinal mucosa and eventual entry into deeper tissues.

SALMONELLOSIS

- The clinical and pathologic syndromes of salmonellosis typically vary from localized **enterocolitis** to **septicemia**; **abortion** may also occur, with or without obvious systemic disease.
- **Exotoxins** lead to the **degeneration and necrosis of enterocytes**; **endotoxins** lead to **thrombosis of mucosal venules and vascular lesions**.

ENTEROTOXEMIA (CLOSTRIDIUM PERFRINGENS)

- Most of the important enteric clostridial diseases occur in herbivores and are caused by 1 of the 5 toxigenic types of ***Clostridium perfringens*** or by ***Clostridium difficile.***
- Enteritis in dogs is associated with ***C. perfringens*** and ***C. difficile*****.**
- ***Clostridium piliforme*** causes Tyzzer's disease, characterized by multifocal necrotic hepatitis and occasionally enteritis, colitis and myocarditis, in many animal species.
- ***C. chauvoei*** may affect the tongue causing blackleglike glossitis. Enteritis produced by *C. chauvoei* has also been described in an outbreak in heifers.
- ***C. septicum*** causes clostridial abomasitis (braxy) in sheep and calves, discussed in the earlier section, Stomach and abomasum.
- ***C. botulinum*** causes botulism in horses, cattle, and several other species by ingestion of preformed toxins

- *Classification of* Clostridium perfringens *into* ***five toxinotypes*** *based on the presence of genes for* ***four major exotoxins*** *(alpha, beta, epsilon, and iota)*

C. Perfringens type	Toxin gene			
	Alpha	Beta	Epsilon	Iota
A	+	–	–	–
B	+	+	+	–
C	+	+	–	–
D	+	–	+	–
E	+	–	–	+

+, toxin gene present; –, toxin gene not present.

ENTEROTOXEMIA (CLOSTRIDIUM PERFRINGENS)

- The **alpha toxin** is a lecithinase that acts on cell membranes, producing hemolysis and necrosis of cells.
- The **beta toxin** is a pore-forming toxin that induces intestinal necrosis and occasionally a variety of neurologic effects through a yet unknown mechanism.
- The **epsilon toxin** is produced as a relatively inactive prototoxin that is activated by enzymatic digestion. Shows its effects especially in the brain and kidneys.
- The **iota toxin** activates with proteolytic enzymes and increases capillary permeability.

❑ *Clostridium perfringens* type A

- Is the toxinotype *most commonly found in the environment and in the intestine of clinically healthy animals*.
- Its major toxin is the *alpha toxin*, also produce enterotoxin
- This is one of several clostridia that produce *gas gangrene* in humans and animals.
- Enterocolitis in horses
- Diarrhea in pigs and cattles
- A very rare disease of **lambs,** characterized by *acute intravascular hemolysis*, known as *yellow lamb disease*

❑ *Clostridium perfringens* type B

Lamb:

- Typical **gross lesions**
- The characteristic lesion is *extensive necrohemorrhagic enteritis.*
- In cases with more severe and deeply penetrating mucosal ulcerations can be seen
- Also there can be Peritonitis with red fibrin strands on the local mesentery and intestinal adhesions
- The disease in **calves caused by type B *C. perfringens closely***

resembles that in lambs with a course of 2-4 days characterized by weakness and dysentery.

❑ *Clostridium perfringens* type C

Sheep:

- In adult sheep, C. perfringens type C causes **"struck,"** a disease of grazed animals➔ STRUCK DISEASE
- Death usually occurs suddenly with terminal convulsions
- abdominal pain.
- Grossly and microscopically;

small intestinal ulceration may be prominent and the mucosal necrosis is deeper.

❑ *Clostridium perfringens* type C

- Type C disease can apparently also occur, albeit rarely, in **feedlot cattle.**
- The condition is similar to "struck."
- Animals are found either dead or moribund, and congestion and hemorrhage of the gastrointestinal tract are prominent.
- The jejunal and ileal content are bloody with fibrin clots and necrotic debris.

❑ *Clostridium perfringens* type D

- ***Clostridium perfringens* type D.** Enterotoxemia ("*pulpy kidney*" *disease*, "*overeating*" *disease*) caused by *Clostridium perfringens* type D is an important disease of *sheep and goats.*
- The rarely observed subacute and chronic forms of the disease in sheep have been called *focal symmetrical encephalomalacia*
- the animal is found dead without clinical signs being observed or after a short period of acute neurological and respiratory signs, including convulsions and tachypnea, and often bawling as from severe pain.

❑ *Clostridium perfringens* type D

- **Grossly,** in sheep dead of acute enterotoxemia, the carcass is usually well nourished.
- There is pericardial, thoracic, and abdominal fluid with straw-colored that clots on exposure to air
- congestion and edema of the lungs
- hemorrhage beneath the endocardium of the left and occasionally right ventricles of the heart.
- There may be hemorrhages beneath other serous membranes such as the epicardium, and parietal peritoneum are characteristic.
- Sometimes the liver is congested and the spleen enlarged and pulpy.
- There **is no gastrointestinal inflammation visible at autopsy**
- **The so-called "pulpy kidney" may be present, because of accelerated autolysis.**

❑ *Clostridium perfringens* type E

- Causes disease in the intestines in calves and rabbits.
- Sudden deaths occurred in calves.
- Congestion and ulcers and hemorrhagic enteritis occur in the abomasum.

ENTERIC CLOSTRIDIAL INFECTIONS (SUMMARY)

- *Clostridium perfringens* type A
  - ✓ Gas gangrene
  - ✓ Colitis in horses
  - ✓ Diarrhea in pigs and calves
  - ✗ Acute intravascular hemolysis in calves and lambs (known as yellow lamb disease)

- *Clostridium perfringens* Type B
  - ✓Lamb dysentery
  - ✓Dysentery in calves and foals
- *Clostridium perfringens* Type C
  - ✓"STRUCK" in adult sheep
  - ✓Enterotoxemia in feedlot cattle
  - ✓Enterotoxemia in lambs, calves, pigs and foals
- *Clostridium perfringens* Type D
  - ✓Enterotoxemia- "Pulpy kidney" disease in sheep and goats
- *Clostridium perfringens* Type E
  - ✓Enteritis in calves and rabbits

PARATUBERCULOSIS (JOHNE'S DISEASE)

- ***Mycobacterium avium* subsp. *Paratuberculosis.***
- *Most common in domestic ruminants.*
- Incubation period is protracted and irregular. Cattle **are 2 years of age** or older.
- **Persistent diarrhea**, progressive weight loss, debilitation, and eventually death.
- **Malabsorption** and filtration secretion caused by the inflamed small intestinal mucosa overloads the capacity of the colon to resorb electrolytes and fluid.
- Major lesions are confined to the **ileum, colon and lymph nodes,** the infection is generalized.

Gross lesions :

- Lesions are usually best developed in the **lower ileum and upper large intestine.**
- **Diffuse thickening of the mucosa**
- Mucosal thickening is due to **accumulation** of predominantly **macrophages, as well as edema fluid**, in the mucosa and submucosa.
- The ileocecal and mesenteric **lymph nodes** are **enlarged**, pale, and edematous.
- Lymphangitis is common, and the **lymphatic vessels** can often be traced as **thickened cords** from the intestinal serosa through the mesentery to the mesenteric nodes.

Thickened mucosal folds that can also be seen from the serosal surface in the jejunum of a cow with Johne's disease.

Microscopic lesions:

- **Transmural granulomatous enteritis and lymphangitis**.
- **Macrophages** in the lamina propria, submucosa, muscular layers or the serosa of the intestine.
- **Epithelioid macrophages and Langhans-type** multinucleated giant cells.
- **Granulomatous lymphangitis** is one of the most consistent changes, and inflammatory cells can be observed along the lacteal vessels of villi, or in the submucosa.

Jubb-kennedy-palmer pathology of domestic animals

R.EQUI ENTEROCOLITIS OF FOALS

Rhodococcus (Corynebacterium) equi
(opportunistic intracellular pathogen ***found in soil***)
1-2 months of age, spring-summer
Suppurative bronchopneumonia
Ulcerative colitis

MYCOTIC DISEASES OF THE INTESTINE

INTESTINAL PHYCOMYCOSIS and ASPERGILLOSIS

- ✓ *Aspergillus* spp. → ASPERGILLOSIS
- ✓ *Zygomycetes*
  - *Absidia*
  - *Mucor* → MUCORMYCOSIS
  - *Rhizopus*
- ✓ *Oomycete* — *Pythium* spp. → PYTHIOSIS " = OOMYCOSIS"
- ✓ *Entomophthoracetes* — *Basidiobolus*, *Conidiobolus* → ENTOMOPHTHOROMYCOSIS

CANDIDIASIS

- ✓ *Candida albicans*
- ✓ *Candida tropicalis*

INTESTINAL HISTOPLASMOSIS

- ✓ *Histoplasma capsulatum*

PARASITIC DISEASES OF THE INTESTINE

- **ECHINOCOCCOSIS (HYDATIDOSIS)**

✓ *Echinococcus granulosus*

- *E. granulosus granulosus* (Intermediate host ruminants and humans)
- E. granulosus equinus

✓ *E. multilocularis*

✓ *E. oligarthus*

✓ *E. vogeli*

HYDATID CYSTS

- Hydatid cysts are usually spherical, turgid and fluid-filled.
- The lining of fertile cysts is studded with small granular brood capsules which contain protoscolices; "hydatid sand" comprised of free brood capsules and protoscolices is in the fluid.
- *Hydatid cysts occur most commonly in the liver and lung.*

EQUINE STRONGYLOSIS

- Members of the family Strongylidae are common nematode parasites of the cecum and colon in horses.
- *Strongylus vulgaris*
- *Strongylus edentatus*
- *Strongylus equinus*

IMPORTANCE

- Larval forms cause *endoarteritis* in the mesenteric circulation.
- Forming nodules ~5-8 mm in diameter
- Necrotic debris, neutrophils, some eosinophils and macrophages
- Obstructive thrombotic lesions
- Aneurisma

PROTOZOAL ENTERITIS

- GIARDIASIS
  - ✓ *Giardia duodenalis*
- AMOEBIASIS
  - ✓ *Entamoeba histolytica*
- BALANTIDIASIS
  - ✓ *Balantidium coli*

- **COCCIDIOSIS**
  - ✓**Cattle:** *Eimeria zuernii /E. bovis / E.ellipsoidalis / E. auburnensis*
  - ✓**Sheep :** *E. ovinoidalis/E.ahsata*/E.bakuensis**
  - ✓**Goats:** *E. ninakohlyakimovae / E. christienseni* / E. arloingi* / E. caprina*
  - ✓**Horses :** *E. leuckarti*
  - ✓**Swine :** *E. scabra / E. debliecki / E. spinosa*
  - ✓**Dogs :** *Isospora canis / I. burrowski / I. neorivolta / I. ohioensis*
  - ✓**Cats:** *I. felis / I. rivolta*

- CRYPTOSPORIDIOSIS
  - *Cryptosporidium muris / C. parvum*
- TOXOPLASMOSIS
  - *Toxoplasma gondii*
- NEOSPOROSIS
  - *Neospora caninum*
- HAMMONDIASIS
  - *Hammondia hammondi*

SARCOCYSTIS

- **Final host (dogs)**
  - ✓*Sarcocystis cruzi (S. bovicanis)*
  - ✓*S. tenella (S. ovicanis)*
  - ✓*S. capracanis*
  - ✓*S. meischeriana (S. porcicanis)*
  - ✓*S. bertrami*
  - ✓*S. equicanis*
  - ✓*S. faeri*

- **Final host (cats)**
  - ✓*S.hirsuta (S. bovifelis)*
  - ✓*S. gigantea (S. ovifelis)*
  - ✓*S. porcifelis*
- **Final host (humans)**
  - ✓*S. bovihominis*
  - ✓*S. porcihominis*

Final hosts: Dogs, cats, wild carnivores and humans

Intermediate host: Ruminants, swine and horses.