E.Coli Infections (Colibacillosis)

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Economic significance

It causes economic losses in poultry industry, including:

- Increased mortality
- Regression in performance parameters
- Increased discard rates at the slaughterhouse
- Poor carcass quality
- Higher treatment costs
- Decreased hatching efficiency
- Chick quality problems

Human Health Importance

Poultry origin *E.coli* infections pose a potential risk for human health

- Antibiotic resistance
- Transfer of virulence genes/plasmids to other enteric bacteria
- Colonization of human-associated strains (*E.coli* O157:H7 serotype) in chicken intestines
- *E.coli* strains producing Shiga toxin (STEC) can cause food poisoning
  - Serotype O1:K1, O2:K1 and O18:K1 can cause septicemia in newborns

Etiology

- Gram-negative
- Rod (bacillus) shaped
- Non–spore-forming
- Oxidase-negative, facultative anaerobe
- Generally motile
- Grow at 18–44 °C
- Some of them are encapsulated
- *E. coli* strains causing infection in poultry are called **avian pathogenic *E. coli* (APEC)**

Antigenic structure

1. O (somatic) Antigen: Lipopolysaccharide (LPS) component located in the cell wall; the antigenic portion of LPS. It is heat-stable and **resistant to boiling**
2. H (flagellar) Antigen: Protein antigen associated with different types of flagellin; heat-labile but may **resist temperatures up to 100 °C**
3. K (Capsular) Antigen: Composed of polymeric acids containing approximately 2% reducing sugars; located on the cell surface and **associated with virulence**. It can be removed by **heating at 100 °C for 1 hour**
4. F (pilus) Antigen: Involved in bacterial attachment to host cells. Expression varies depending on environmental conditions and can be classified as mannose-sensitive or mannose-resistant

Typing method

Serotyping (Ewing scheme)

- 167 O
- 74 K
- 53 H
- 17 F antigens
- According to antigens;
  - **Common O serogroups: O1, O2, O35, O78**
  - **Others: O8, O15, O18, O115, O116, O132**
  - **Common serotypes associated with pathogenicity: O1:K1, O2:K1, O78:K80, O1:K1:H5**

Molecular typing

- RAPD, RFLP, PFGE

Virulence characteristics

Adhesins

- **Type 1 fimbriae (F1):** Involved in adherence to epithelial cells
- **P fimbriae:** Mediate adherence, especially in urinary tract infections
- **Sex pilus:** Involved in conjugation (DNA transfer between cells)
- **Intimin:** An important non-fimbrial adhesin associated with attaching and effacing *E. coli* (AEEC); involved in diarrhea pathogenesis

Toxins

- **Enterotoxins**
  - **Stable toxin (ST):** Heat-stable, resistant to 100 °C.
  - **Labile toxin (LT, cytotoxin):** Heat-labile, inactivated at 60 °C
  - **Shiga toxin (Stx):** Potent cytotoxin that inhibits protein synthesis; responsible for hemorrhagic colitis and hemolytic uremic syndrome
  - **Cytotoxins:** Cause host cell damage and tissue necrosis; contribute to systemic infection
  - **Hemolysins:** Pore-forming toxins that lyse erythrocytes and other host cells, facilitating nutrient release and tissue invasion

Virulence characteristics-2

Aerobactin: A siderophore involved in iron acquisition; contributes to virulence in extraintestinal *E. coli* strains
- APEC (Avian Pathogenic *E. coli*)
- UPEC (Uropathogenic *E. coli*)
- NMEC (Neonatal Meningitis *E. coli*)
- SEPEC (Septicemic *E. coli*)

Enterobactin: A high-affinity catecholate siderophore that scavenges iron from host proteins; commonly produced by commensal and pathogenic *E. coli.*

Yersiniabactin: A siderophore system associated with pathogenicity islands; enhances survival in iron-limited host environments and contributes to systemic infection

Bacteriocins: Antibacterial proteins produced by *E. coli* strains that inhibit closely related bacteria
- Colicins (A-V): Specific types of bacteriocins produced by *E. coli*

Bacteriophages: Viruses that infect bacteria; can mediate horizontal gene transfer and carry virulence genes (e.g., Shiga toxin genes in STEC)

Intestinal Pathotypes of *E.coli*

- **Enteropathogenic *E. coli* (EPEC)** : Causes diarrhea by attaching and effacing intestinal epithelial cells; associated with intimin (*eae* gene)
- **Enterotoxigenic *E. coli* (ETEC):** Produces heat-labile (LT) and/or heat-stable (ST) enterotoxins, leading to secretory diarrhea
- **Enteroinvasive *E. coli* (EIEC):** Invades intestinal epithelial cells, similar to *Shigella* spp., causing dysentery-like illness
- **Enterohemorrhagic *E. coli* (EHEC/STEC):** Produces Shiga toxins (Stx1, Stx2), causing hemorrhagic colitis and hemolytic uremic syndrome (HUS)
- **Enteroaggregative *E. coli* (EAEC):** Adheres to intestinal cells in a “stacked brick” pattern via aggregative adherence fimbriae (AAF); produces toxins such as EAST1 and Pet, leading to persistent diarrhea, especially in children
- **Diffusely Adherent *E. coli* (DAEC):** Exhibits diffuse adherence to epithelial cells; associated with chronic diarrhea in children and urinary tract infections in adults
- **Adherent-Invasive *E. coli* (AIEC):** Invades intestinal epithelial cells and survives within macrophages; associated with chronic intestinal inflammation, particularly in Crohn’s disease

Extraintestinal Pathogenic *E.coli*

- **Avian Pathogenic *E. coli* (APEC):** Causes colibacillosis in poultry; associated with adhesins, siderophores (aerobactin, yersiniabactin), and serum resistance genes
- **Uropathogenic *E. coli* (UPEC):** Causes urinary tract infections in humans and animals; possesses P fimbriae, hemolysin, and aerobactin systems
- **Neonatal Meningitis *E. coli* (NMEC):** Causes meningitis and septicemia in newborns; commonly associated with the K1 capsule and invasion of the blood-brain barrier
- **Septicemic *E. coli* (SEPEC):** Associated with bacteremia and sepsis in both humans and animals; exhibits multiple virulence factors for systemic invasion
- **Edema Disease *E. coli* (EDEC):** Causes edema disease in pigs; produces Shiga toxin 2e (Stx2e) and F18 fimbriae.

Vitality

E. coli are sensitive to physical and chemical factors.

- They are inactivated within 2–30 minutes at temperatures between 60–70°C
- Survival times increase under cold environmental conditions

Litter

- A 90% reduction occurs within 1-2 days at >25°C, while survival can last 6–22 weeks at 4°C
- High humidity increases survival time

Growth and survival are inhibited under pH conditions below 4.5 or above 9

Epidemiology

- Hosts: Chicken, turkey, goose, duck and other poultry
- Colibacillosis usually occurs in young chicks
- Predisposing factors include poor management, concurrent bacterial or viral infections, and stress
- *E. coli* is a normal inhabitant of the intestinal flora but it can also be found in
  - Respiratory tract
  - On skin and feathers
  - In environmental resources (litter, dust, and water)

Epidemiology

- 10^4-10^7 cfu/g in intestines,
- 10^8 cfu/g in feathers
- 10^6 cfu/g per litter

➢ Colonization of the digestive tract of poultry begins at hatching, and *E. coli* populations increase rapidly thereafter

➢ Both pathogenic and non-pathogenic *E. coli* strains can be isolated from the intestinal tract of healthy birds

➢ Contamination of eggshells with *E. coli* may occur through several routes:
  - ➢ Passage through the cloaca,
  - ➢ Contaminated laying nests and litter,
  - ➢ **Vertical transmission via eggs through the oviduct (fallopian tube)**

Approximately 0.5–0.6% of eggs may contain *E. coli.*

Such contamination can result in embryonic death, yolk sac infection (omphalitis), and high first-week chick mortality

Epidemiology

Horizontal transmission occurs through;

- direct contact between birds
- indirect contact via contaminated water, feed, litter, or equipment

The bacteria can enter the host through;

- digestive tract,
- respiratory tract,
- eyes, or skin wounds

Clinical signs and necropsy

Systemic forms (colisepticemia): It is a generalized *E. coli* infection that occurs when bacteria enter the bloodstream and spread to internal organs, leading to septicemia and multiple organ lesions in poultry.

In the acute phase, no clinical signs may be observed. As the infection progresses, necropsy findings may include enlargement and dark discoloration of the liver and spleen, arthritis, myocarditis, peritonitis, and cloudiness and thickening of the air sacs.

- Respiratory colisepticemia
- Colisepticemia of enteric origin
- Acute colisepticemia in layers and breeders

Clinical signs and necropsy

Localized forms : Localized forms of colibacillosis are characterized by infections confined to specific organs or tissues

- **Omphalitis (yolk sac infection):** Inflammation of the navel and yolk sac in newly hatched chicks, often resulting from egg contamination with *E. coli*. Chicks may die during incubation. Infected animals show swollen, tense, moist, and reddened abdominal regions. Growth and development are retarded. At necropsy, a yellow, foul-smelling, unabsorbed yolk sac, hemorrhagic areas on the intestinal surface, and peritonitis are observed.
- **Synovitis:** In infected animals, swelling, pain, heat, tension, edema, lameness in the joints and decreased egg production are observed. At necropsy, a mucoid and sometimes caseous exudate accumulation is seen in the joints.
- **Airsacculitis:** *E. coli* strains usually act as secondary pathogens to infections such as ND, IB, ILT, SHS, and CRD. At necropsy, the air sacs appear opaque and thickened. Septicemia may develop.
- **Salpingitis/peritonitis:** The most characteristic findings are obstruction of the oviduct, excessive fat deposition, displacement of the yolk or egg into the abdominal cavity, accumulation of eggs within the oviduct, the presence of hardened eggs, and an appearance of “cooked egg” in the abdominal cavity.
- **Enteritis:** There may be no specific findings to identify the infection. At necropsy, the intestinal wall is thickened and edematous. An enteritis picture is observed, and the intestinal contents are usually greenish in color.

Hjarre Disease

- It is a chronic disease characterized by the formation of granulomas in the liver, intestines, and mesentery of chickens and turkeys.
- No specific clinical symptoms are observed for diagnosis.
- At necropsy, granulomas of various sizes resembling tuberculous lesions are seen in the intestines and liver

Diagnosis

1. Clinical and necropsy findings
2. Laboratory examinations
3. Typing

   Molecular typing

Treatment

According to clinical findings, appropriate antibiotics should be selected based on antibiotic resistance

Protection and control

General precautions

- Monitoring of maintenance, management, and serotypes in breeder flocks
- Proper management of hatching eggs
- Appropriate egg storage conditions
- Control of incubation parameters
- Incubation hygiene and sanitation
- Disinfection and preparation of flocks
- Good management, care, and feeding practices
- Monitoring of water quality and proper drinker management
- Effective rodent control
- Control of microbiota in breeders, hatcheries, and broiler flocks
- Monitoring interactions between microorganisms
- Elimination of social and environmental stress factors

Protection and control

Other precautions- Vaccinations

Inactive vaccines

- Homologous strains
- Combination of dominant strains

Live vaccines

- Natural strains
- Mutant strains

Result

Presence/distribution of *E.coli* infections within the management system

Evaluation of clinical findings

Determination of the pathogenesis of the infection

Serotype distribution

Determination of antibiotic resistance profiles

Organization and monitoring of control programs

INFECTIOUS CORYZA

Etiology

Haemophilus paragallinarum

Avibacterium paragallinarum

- 3 sero-group: A, B, C

Sensitive to enviromental conditions

Epidemiology

- A highly contagious, acute upper respiratory tract infection
- The natural host of the agent is chickens
- Transmission occurs via the respiratory and digestive tracts
- Chickens of all ages are susceptible to the disease
- The disease is more severe in laying or high-production chickens
- It spreads very rapidly within the flock
- High morbidity (60–100%). Low mortality (1–10%), unless complicated by secondary infections
- Decreased growth performance is observed in affected birds
- The infection causes significant production losses in laying flocks
- It is prevalent in many countries and endemic in certain regions
- Chronically infected birds play an important role in the transmission of the disease
- The incidence is higher during autumn and winter
- **No evidence of vertical transmission**

Epidemiology

- The incubation period is quite short (24–48 hours)
- The agent can infect most birds in a susceptible flock within 72 hours
- The disease usually affects the entire flock for about 2–3 weeks; however, this period may be prolonged in the presence of secondary factors, particularly Mycoplasma infections
- The pathogen can survive for approximately 50 days in active sinus exudate
- Concurrent infections (such as *Mycoplasma* or Infectious Bronchitis virus) increase the severity of the disease

Clinical Findings and Macroscopic Lesions-1

- Decreased feed and water intake in affected birds
- Nasal discharge, facial edema, and conjunctivitis; feathers may appear ruffled in roosters
- Loud respiratory sounds when the lower respiratory tract is involved
- Arthritis and septicemia may occur in severe cases
- Diarrhea has also been reported in some cases
- Pneumonia and airsacculitis are rare
- A 10–40% decrease in egg production may be observed in laying birds
- In cases complicated with other bacteria and chronic infections, a foul odor can be noticed in the poultry house. Especially when *Mycoplasma* infections occur concurrently with *Avibacterium paragallinarum* infections, head swelling can be mistaken for *Avian metapneumovirus* (AmPV) infection

Necropsy Findings

- Acute catarrhal inflammation occurs in the mucous membranes of the nasal cavities and sinuses
- Catarrhal conjunctivitis and subcutaneous edema on the face and wattles are observed
- Pneumonia and airsacculitis are rarely seen in the disease

Morbidity and Mortality

Depending on the virulence of the microorganism, uncomplicated infectious coryza usually shows low mortality and high morbidity

Mortality rates and clinical symptoms vary with the age of the birds and the route of infection

Poor flock conditions, parasitic infections, and malnutrition may complicate the disease course and prolong the infection

In general, a marked increase in mortality is observed when the disease is complicated with other infections such as fowlpox, infectious bronchitis (IB), infectious laryngotracheitis (ILT), chronic respiratory disease (CRD), or *Pasteurella* infections.

Diagnosis

Isolation and identification

- Isolation of the bacteria is difficult
- It is possible from samples collected during the acute stage of infection
- The best material for isolation is sinus exudate

Serological tests

- Agglutination
- AGP
- HI
- ELISA

Diagnosis

Molecular tests

- PCR
- PCR-RFLP

Infection complicated with the disease

CRD

Chronic Chicken Cholera

Chicken Pox

AmPV (SHS)

Deficiency of A vitamin

The disease is seen usually mixed infection. Therefore, the mortaility rate can be changeable.

Protection and Control

General precautions

- Chicks and pullets from infected flocks should not be introduced
- Removal of infected carriers
- Separation of old and foster flocks
- Houses should be left empty for at least two weeks after depopulation.

Disinfection

- The bacteria are susceptible to most disinfectants

Biosecurity measures

Treatment

Erythromycin

Sulfonamides

Quinolones

Tetracyclines

Vaccines

Inactivated vaccines are used

Vaccines should be prepared in chicken embryos, broth cultures and in cell cultures at least 10^8 cfu

Losses after complications can be reduced with vaccinations

Protection is provided for 9-12 months after vaccination

Protection is serovar-specific, provided only against the serovar included in the vaccine

Live and attenuated vaccines are also available

In recent years, there are studies about recombinant vaccines

Vaccines

Licensed vaccines used in Turkey

Vaccine 1. Serotype A, Serotype B, Serotype C

Vaccine 2. Serotype A, Serotype C

In Vaccination program

- 1st vaccination in 10-12th week
- 2nd vaccination in 16th week