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COLIBACILLOSIS IN POULTRY AND PIGS: A BRIEF REVIEW

COLIBACILOZA LA PĂSĂRI ȘI PORCI: O SCURTĂ RECENZIE

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ABSTRACT | REZUMAT

Escherichia coli can be associated with various pathological conditions in farm animals, and syndromes are usually called enteric colibacillosis or systemic colibacillosis (produced by invasive strains).

The aim of this review was the up-dating of *E. coli* infection patterns in poultry and pigs in order to identify knowledge gaps that would require future research. Colibacillosis should remain in the attention of livestock farms, where can cause great economic losses. In poultry and pig farms, particular attention should be paid to the prevention of disease outbreaks. Achieving more effective prevention programs requires more knowledge in internal and external biosecurity measures and vaccination programs, and future research projects could target these objectives.

Keywords: *Escherichia coli*, APEC, edema disease, prevention programs

La animalele de fermă, *Escherichia coli* poate fi asociată cu diferite afecțiuni patologice cunoscute sub numele de enterită colibacilară sau septicemie colibacilară (produsă de tulpini invazive).

Scopul acestei recenzii a fost actualizarea patternului infecțiilor cu *E. coli* la păsări și porci, în vederea identificării lacunelor de cunoștințe care ar necesita cercetări in viitor. Colibaciloza ar trebui să rămână în atenția fermelor de animale, unde poate provoca pierderi economice mari. În fermele de păsări și porci, o atenție deosebită ar trebui acordată prevenirii focarelor de boală. Realizarea unor programe de prevenire mai eficiente necesită mai multe cunoștințe în materie de biosecuritate internă și externă și programe de vaccinare, iar viitoarele proiecte de cercetare ar putea ținti asemenea obiective.

Cuvinte cheie: *Escherichia coli*, APEC, boala edemelor, programe de prevenție

COLIBACILLOSIS IN POULTRY

In poultry, colibacillosis is a localized or systemic infectious disease caused by avian pathogenic *Escherichia coli* which mainly affects broiler chickens.

Aetiology

E.coli is a gram-negative,non-acid-fast, non-spore -forming bacillus, variable in size and shape, with peritrichous flagella, and facultative anaerobic bacterium normally found as a not pathogenic organism in the intestine of poultry.

Some *E. coli* strains acquired virulence factors which led to increased pathogenicity: the virulence genes are clustered in plasmid-borne pathogenicity islands and considered characteristic of the Avian pathogenic *E. coli* (APEC). The infection of weakened birds with commensal *E. coli*; some predisposing conditions are Newcastle disease, mycoplasmas infections, infectious bronchitis, and environmental stress factors. APEC commonly belongs to serogroups O78, O1, O2, O15, and O55. In domestic poultry, infections were frequently isolated serotypes O78:K80, O1:K1, and O2:K1(2- Filali E) (6, 8, 19).

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Epidemiological data

The main source of APEC is carrier chickens which may have in faeces ~10^9 colony forming units/gram (15). Also, APEC can be in the intestinal tract of rodents or other animals from where birds can be infected through water and feed contaminated with rodent faeces. The risk of poultry to develop colibacillosis is related to the following factors: (a) Increasing infection pressure in the environment; (b) Poor hygiene; (c) Overcrowding;(d) Duration of exposure; (e) Virulence of the strain; (f) Breed; (g) Immune status; (h) Damage to the respiratory system; (i) Infection with other infectious agents (e.g., Infectious bronchitis virus, Newcastle disease virus, and *Mycoplasma gallisepticum* infection) (4, 20).

Pathogenetic aspects

The following virulence factors have been involved in the pathogenesis of avian colibacillosis are: (a) F (type 1) and P-fimbriae adhesins; (b) Curli factor; (c) Factors contributing to adhesion; (d) Factors contributing to resistance to immunologic defence; (e) Factors contributing to survival in physiologic fluids; (f) Factors contributing to cytotoxic effects; (g) Factors conferring resistance to serum; (h) Factors conferring resistance to phagocytosis; (i) Aerobactin siderophores; (j) The hemolysing E gene; (k) The *tsh* gene; (l) K1 Capsular antigen; (m) Cytotoxins; (n) Outer mem-

brane proteins; (o) Coligenicity; (p) The heat-labile chick lethal toxin; and (r) Verotoxin-2 like toxin (13, 15). By using the virulence plasmids, *E. coli* can give the following abilities involved in the pathogenesis of avian colibacillosis: (a) Adherence to host structures; (b) Resistance to phagocytosis; (c) Ability to survive the bactericidal action of serum; and (d) Acquiring iron in low-iron environments (18).

Clinical Findings and Lesions

Several localised and systemic APEC infections were described in poultries. The following systemic infections have been described in poultry: colisepticaemia, haemorrhagic septicaemia, and coligranuloma. Colisepticaemia can induce various sequelae as meningitis, encephalitis, pan-ophthalmia, osteomyelitis, and synovitis. The most frequent localised APEC infections are diarrhoeal disease, salpingitis, peritonitis, orchitis, swollen head syndrome, cellulitis, omphalitis, and yolk sac infection (17). Depending on the clinical evolution and the location of the inflammatory process, the lesions produced by APEC can vary from acute to chronic. The most frequent lesions are peritonitis, perihepatitis, swollen liver and spleen, granulomas in liver and spleen, airsacculitis, pericarditis, arthritis, synovitis, enteritis, salpingitis, omphalitis, and cellulitis (3, 18).

Diagnostic options in poultry colibacillosis

- Isolation of *E coli* in pure culture (avian isolates are usually nonhemolytic on sheep blood agar 5%).
- Establishing pathogenicity by (a) detection of plasmid-mediated virulence genes by using multiplex PCR panels; (b) young chicks or poults parenteral inoculation (fatal septicaemia or typical lesions within 3 days); and (c) inoculation of the allantois sac of 12-day-old chicken embryos (encephalomalacia and cranial and skin haemorrhages).
- Molecular finger-printing by: (a)Pulsed field gel electrophoresis (PFGE); (b) Restriction fragment length polymorphism (RFLP); (c) Enterobacterial repetitive intergenic consensus (ERIC-PCR); (d) Repetitive element palindromic PCR (REP-PCR); (e) Random amplification of polymorphic DNA (RA PD–PCR); (f) Ribotyping; (g) Isoenzyme profile (15).

Preventive and control measures

- Good biosecurity measures considering the house sanitation, hygiene in the handling of hatching eggs and hatchery, feed, and water.
- Control of predisposing factors by decrease exposure of birds to APEC and diminishing the impact of stress and predisposition associated with co-infections.
- Vaccination (autogenous or commercial vaccines).
- Treatment with antimicrobial agents is generally not recommended. APEC strains are often resistant to tetracyclines, sulfonamides, aminoglycosides, and β-lactam antibiotics. Restrictions on antimicrobial use in domestic poultry are imposed by international and national regulations and public concern (e.g. fluoroquinolone in the USA) (18, 21).

ENTERIC COLIBACILLOSIS IN PIGS

In pigs, enteric colibacillosis is a common disease caused by enterotoxigenic *E. coli* (ETEC) strains in nursing and weanling pigs.

Aetiology

The common antigenic ETEC types of neonatal colibacillosis are K88, K99, 987P, and F41. The F18 type is commonly involved in postweaning colibacillosis (7).

Epidemiological data

The common affected are young piglets within a few days of birth through well after weaning.

Pathogenetic aspects

The ETEC with specific fimbria or pili will adhere to or colonize the absorptive epithelial cells of the jejunum and ileum. Enterotoxins of the pathogenic strains cause secretion of the fluid and electrolytes into the intestinal lumen, diarrhoea, dehydration, and acidosis (5).

Clinical Findings and Lesions

The main clinical signs associated with enteric colibacillosis in pigs are profuse watery diarrhoea, rapid dehydration, acidosis, and death.

Necropsy examination revealed dehydration, small intestine distention, yellowish, slightly mucoid fluid in small intestine and colon, reddened gastric mucosa (fundic area), rare patchy cutaneous erythema, and several small bacterial rods adhered to the absorptive enterocytes of the intestinal villi (3, 10).

Diagnostic options in colibacillosis in pigs

Because *E. coli* is a common secondary agent, the confirmation must demonstrate the villous colonisation by ETEC K88, K99, 987P, or F41 pilus antigens:

- Histopathologic exam for detection of *E. coli* colonization in the absorptive epithelial cells;
- Immunofluorescence or other immunologic tests for detection of K88, K99, 987P, or F41 pilus antigens in intestinal scrapings;
- Bacteriological diagnostic for isolation of the organism from the small intestine (10, 14).

Preventive and control measures

- Applying the best farm biosecurity measures.
- Reduction of the environmental factors (e.g., dampness, chilling).

- Use wire-mesh flooring (10).
- Vaccination of gestating sows:
- Fimbriae-based ETEC vaccine (bacterin) with fimbrial antigen F4 (K88), F5(K99), F6(987P), and F41;
- Heat labile enterotoxin based on the B subunit (LT_B) of the toxin;
- Recombinant DNA vaccines (16).
- Treatment with antimicrobial agents must consider bacterial antibiotic sensitivity testing. Restoration of fluid and electrolyte balance (5, 10).

EDEMA DISEASE

The edema disease is a condition of the pigs in the weaning period produced by a vascular very powerful toxin (Stx2e) of *E. coli* K88 (F4) or F18 serotypes inducing sudden death, oedema, and/or nervous signs.

Aetiology

Haemolytic *E. coli* that produces F18 pili and Shiga toxin 2e (Stx2e) has two major antigenic variants: (a) F18ab - typical for edema disease strains, and (b) F18ac - described mainly in enterotoxigenic *E coli*.

The F18ab antigenic variants of *E. coli* commonly belong in the serotypes: O138:K81:NM, O139:K12:H1, O141:K85ab:H4, O141:K85ac:H4, and O147 strains carrying the H4, H14 or H17 flagella (1, 9).

Epidemiological data

The disease affects weaners and is not present in sows, lactating piglets, and growers. The disease appears 1 to 4 weeks after weaning. The peak of disease is recorded at 10 days after weaning and classically involves the healthiest animals. Some pigs are genetically resistant to infection because they are carrying a specific mutation in a gene required for the expression of the receptors. The main source of infection is carrier sows that contaminate the environment. Usually, the susceptible pigs are infected by the environment. Morbidity is 30%-40% with 90% mortality (2, 3, 11, 22).

Pathogenetic aspects

After ingestion of edema disease *E. coli* serotypes, the F18 pili will support bacteria in attach and colonise the intestinal epithelial cells which expressed the specific receptor. Expression of the F18 pili receptors is age-related. The Stx2e toxin damages the small blood vessel walls (damage to small arteries and arterioles) and produces fluid accumulation or edema in the eyelids, stomach, small intestine, colon, and brain. The clinical signs are linked with the organ or tissues where the damaged blood vessels are located (e.g., in the brain will produce characteristic nervous symptoms associated with malacia in the brain stem and basal ganglia). At the time clinical signs become visible, the toxin production in the gut is high and the treatment of sick pigs is usually unsuccessful (2, 12).

Clinical Findings and Lesions

The disease may evolve with sudden death without clinical signs. In acute forms, pigs stop eating, develop face or eyelid edema, ataxia, paralysis, recumbency, paddling, and running movements. Animals can die or survive often with neurologic deficits. At necropsy, edema can be seen subcutaneously, in stomach submucosa (mainly in the glandular cardiac region), and mesocolon. Sometimes edema is accompanied by haemorrhage, peritoneal and pleural serous fluid, and fibrin strands in the peritoneal cavity (3, 12).

Diagnostic options in edema disease

Isolation and characterization of the *E. coli* are performed by bacterial culture of the small intestine from a pig who did not receive antimicrobial treatment, followed by a demonstration of the haemolytic *E. coli* presence, and detection of the virulence factors by genotyping (the genes for the F18 pili and Stx2e) and serotyping (2, 12).

Preventive and control measures

- Applying the best farm biosecurity measures, including the purchase of breeding stock from negative-edema disease herds, use the all-in/all-out system, and good sanitation.
- Minimization of the environmental stresses (e.g., prevention of the high-temperature variation);
- Good management of feed by using oats, zinc oxide, plasma protein, and acidifiers (organic or inorganic acids), decreasing the protein concentration in the feed, and controlling the amount of feed consumed after weaning (small quantities of feed, 3-6 times/day).
- Blocking the intestinal *E. coli* receptors by competitive exclusion with nonvirulent agents.
- Raising pigs genetically resistant to edema disease (pigs without receptors for F18 and/or F4 (K88) pili).
- Oral administration of milk, plasma protein, or egg powder with specific antibodies.
- Oral vaccination with live or killed cultures that contain F18 or F4 (K88) pili.
- In contaminated farms, complete depopulation and disinfection can conduct to the eradication of disease (2, 11).

CONCLUSIONS

Colibacillosis should remain in the attention of livestock farms, where can cause great economic losses. In poultry and pig farms, particular attention should

be paid to the prevention of disease outbreaks.

Achieving more effective prevention programs requires more knowledge in internal and external biosecurity measures and vaccination programs, and future research projects could target these objectives. Also, the use of antibiotics should be done with caution because it is not a long-term control solution and antimicrobial resistance prevention must be considered.

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