Наукові записки НаУКМА
Біологія і екологія 2025;8:45-54

БІОЛОГІЯ

NaUKMA Research Papers
Biology and Ecology 2025;8:45-54

BIOLOGY

DOI: 10.18523/2617-4529.2025.8.45-54
UDC 579.2:579.63/619:636.5

O. Nechypurenko[1, 2], I. Furtat[1], D. Dreval[3], L. Avdeeva[2]

[1] National University of Kyiv-Mohyla Academy, Kyiv, Ukraine

[2] D. K. Zabolotny Institute of Microbiology and Virology of the National Academy of Sciences of Ukraine, Kyiv, Ukraine

[3] LLC "Center for Veterinary Diagnostics", Kyiv, Ukraine

COLIBACILLOSIS IN BROILER CHICKS AND ITS ETIOLOGICAL LINK TO THE BIOLOGICAL CHARACTERISTICS OF *ESCHERICHIA COLI* ISOLATES FROM THE BREEDER FLOCK

Abstract

Pathogenic strains of Escherichia coli, capable of causing avian colibacillosis (APEC), currently pose a significant threat to modern poultry farming, in particular industrial broiler production. Moreover, the origin of colibacillosis in broiler chickens is connected with parental health issues. Therefore, the aim of the work was to investigate the etiological link between colibacillosis in 1-day old chicks and the biological characteristics of E. coli isolates from the breeder flock. A total of 65 pathogenic E. coli isolates were detected from broiler-breeders (n = 14) and 1-day-old chikens (n = 51). This shows a huge impact of collibacillosis on poultry farming at different levels of production. The main lesions were associated with fibrin deposition and omphlaitis formation. The last-mentioned parameter could affect the viability of chicks; nevertheless, the average Pasgar score was identified as 9.7 points. Amomg APEC, the most prevalent were O78 and O18 serotypes, wich were characterized by the absence of β-hemolysis. Moreover, the antibiotic profile of APEC isolated from parenteral and progeny flocks was similar. A high level of resistance to amoxicillin, amoxiclav, doxycycline, tetracycline, and enrofloxacin was found; more than 70% of isolated APEC were invulnerable to the mentioned antimicrobials. The most effective antibiotics were colistin, gentamicin, and florfenicol, to which only 0.0% and 2.0%, 14.3 and 9.8%, 7.1% and 2.0% of isolated APEC from broiler-breeders and chickens, respectively, were resistant. In addition, prolonged treatment of birds led to the formation of multi-resistant strains that affected chickens from the first days of life. Thus, studying the process of APEC infection through an integrated production chain can be useful for taking appropriate measures to prevent early cases of colibacilosis.

Keywords: Avian pathogenic *E. coli* (APEC), antibiotic resistance, colibacilosis.

Introduction

Avian colibacillosis, caused by pathogenic variants of *Escherichia coli* (notably so-called APEC strains), is one of the most prevalent infectious diseases affecting domestic, ornamental, and wild birds. It exhibits a variable course – acute or chronic – accompanied by signs of intoxication. This disease is a major cause of significant economic losses in poultry farming worldwide, due to high mortality rates, reduced egg production, poor weight gain in meat-type birds, ineffective treatment, and compromised immunological responsiveness to vaccinations against various viral infections [1,2].

Colibacillosis can manifest as either localized or systemic infections, which in turn present in various clinical forms, including acute septicemia, subacute pericarditis, chronic respiratory diseases, fibrinous-purulent polyserositis, airsacculitis, and many others. The progression of colibacillosis may affect multiple physiological systems – such as the

respiratory, digestive, reproductive, or musculoskeletal systems – or remain localized, as observed in cases of yolk sac infection, omphalitis, dermatitis, cellulitis, or panophthalmitis. Thus, APEC is an etiological agent of primary infections, the development of which is often associated with suboptimal conditions in large-scale poultry farms, including elevated ammonia levels, high stocking density, temperature fluctuations, and other environmental stressors that negatively impact poultry health [3]. At the same time, they can frequently cause secondary infections that complicate such primary diseases as respiratory viral infections (Newcastle disease, infectious bronchitis, avian influenza) and mycoplasma infections caused by *Mycoplasma gallisepticum* or *Mycoplasma synoviae* [1,2,4].

In general, representatives of the *E. coli* species belong to the commensal microbiota of the lower gastrointestinal tract in mammals and birds, contributing to the maintenance of normal physiological functions of the host organism. At the same time, *E. coli*, being a ubiquitous microorganism, is characterized by remarkable genomic plasticity, as it can enrich its genome through the acquisition of genes whose expression products enable it to adapt to adverse environmental conditions, colonize atypical biotopes, synthesize diverse virulence factors, and produce compounds that contribute to resistance against various classes of antimicrobial agents [5-7]. Notably, it has been established that among the virulence factors involved in the pathogenesis of colibacillosis, F1 fimbriae adhere to the epithelial cells of the pharyngeal and tracheal respiratory tracts of chicks; temperature-sensitive hemagglutinin plays a role in the colonization of the air sacs; the aerobactin iron-acqusition system enables *E. coli* to grow under conditions of low free iron concentration in physiological fluids; and P-fimbriae are critical at later stages of infection, facilitating adhesion to internal organs and providing resistance to phagocytosis. The presence of plasmids in *E. coli* strains may explain the rapid and efficient transfer of virulence factors post-hatching. Moreover, the genetic background can influence the bacterium's ability to acquire, maintain, or express pathogenic traits. It has also been shown that infection of chicks with *E. coli* may occur transovarially from infected hens and/or via a contaminated eggshell surface. Moreover, horizontal transmission through inhalation from infected offspring to other chicks upon contact after hatching is possible [5,6,8,9]. Nevertheless, the number of studies focused on the distribution of pathogenic *E. coli* strains causing avian diseases, as well as on the structure and biological characteristics of the APEC population in broiler production and the transmission pathways from parent stock to chicks, remains rather limited. Thus, the aim of the work was to investigate the etiological link between colibacillosis in 1-day-old chicks and the biological characteristics of *E. coli* isolates from the breeder flock.

Material and methods

In the research, biological material collected during 2023–2024 from one of the productive zones of a poultry farm in Ukraine was used. In cases of increased bird mortality during the production period, samples were taken from birds of various age groups (specifically at 180–190, 230–240, 310–320, and 380–390 days) for subsequent pathological and bacteriological analysis. Importantly, the birds had been vaccinated twice with a live attenuated vaccine derived from *Escherichia coli* strain EC 34195, which carries a mutation in the *aroA* gene. The quality of one-day-old chicks originating from diseased breeder-broilers was assessed based on the criteria including the presence of pathological changes and bacterial infection.

The analysis of the pathological condition of the birds selected for the study was carried out in the laboratory of LLC "Center for Veterinary Diagnostics". For this purpose, was autopsies were performed and lesions were described in particular: trachea, lungs, air sacs, heart, liver, spleen, kidneys, stomach, small and large intestine and yolk sac. The degree of pathological changes was determined depending on the area of the affected organ according to a 0-3-point system. The degree of lesions was valuated from 0 to 3 points: 0 – absent, 1 – mild, 2 – moderate and 3 – severe based on the presence of fibrin deposition, hemorrhages, edema and inflammation [10].

The selection of biological material samples for further isolation of bacteria was carried out aseptically from the internal organs: in broiler parents – from the liver, heart, spleen, and in day-old chicks – from the liver, heart, and yolk sac, respectively. At the first stage of isolation of cultures, the samples were placed in a liquid nutrient medium tryptone soy broth (TSB, Himedia, India) and cultivated at a temperature of 37 °C for 24 hours. After that, the bacteria were sown using the dense lawn technique on MacConkey diagnostic and differential medium (McCM, Himedia, India). Subsequently, the colonies that grew on the surface of the nutrient medium were subcultured using the depletion streak technique on tryptone soy agar (TSA, Himedia, India) for further

identification. Isolates of *E. coli* were considered to be pathogenic if they were isolated from more than two organs [11].

Bacterial isolates were identified using the APi20 E test system (bioMerieux, France) according to the main diagnostically significant features of the *E. coli* species, which allows assessing a number of biochemical indicators: the presence of the enzymes β-galactosidase, tryptophan deaminase, lysine decarboxylase, ornithine decarboxylase, arginine dihydrolase, urease, gelatinase; the ability to ferment glucose, mannitol, inositol, sorbitol, rhamnose, sucrose, melibiose, amygdalin, arabinose; the ability to utilize citrate and form hydrogen sulfide, indole, and acetoin.

The susceptibility of *E. coli* isolates to antimicrobials was determined by the Kirby-Bauer disk diffusion method on Mueller-Hinton medium (MHM, Himedia, India) [12]. The following antimicrobial compounds were used (Oxoid, Holland): spectinomycin, gentamicin, neomycin, fosfomycin, amoxicillin, amoxiclav, doxycycline, oxytetracycline, colistin, florfenicol, flumequine, enrofloxacin, norfloxacin, ciprofloxacin and trimethoprim. The quality control of antimicrobial discs and Mueller-Hinton media was determined using reference strains of the American Type Culture Collection (ATTC): *E. coli* ATCC 25922, *Staphylococcus aureus* ATCC 25923, *Pseudomonas aeruginosa* ATCC 27853 and *Enterococcus faecalis* ATCC 29212, which are maintained in the Ukrainian Collection of Microorganisms at the Zabolotny Institute of Microbiology and Virology of the National Academy of Sciences of Ukraine. Isolated multiresistant pathogenic *E. coli* strains were stored on semi-solid thioglycolate medium at 2 °C for further research [13,14].

Serotyping of the *E. coli* isolates was performed using polyvalent Anti-Coli P (O78, O2, O86, O18) sera (Sifin, Germany). Hemolytic activity of the cultures was determined using blood agar (BA, Himedia, India) and classified as α-, β- and γ-hemolysis according to the severity [15].

The etiological impact of the state of the broiler-breeders on the health of their offspring was determined in accordance with the Global Health Chicks Program for day-old chicks, which involves assessing the viability of chicks according to the Pasgar scale, as well as PCR analysis of samples of biological material from the liver, heart, yolk sac and respiratory tract of chickens, selected on FTA cards for identification of representatives of the species *Mycoplasma galisepticum*, *Mycoplasma synoviae*, *Salmonella* spp. and *E. coli*. PCR was performed in the Hipra Diagnose laboratory (Spain) [1,11].

Data entry, initial analysis, and figure design were done using Microsoft Office Excel 2010 (Microsoft Corporation, New York, NY, USA) to generate figures and run initial analysis as previously described [14]. The chi-square test was calculated using Pearson probability value (p-value) to compare the number of isolates resistant to aminoglycosides, β-lactams, tetracyclines, polypeptides, phenicols, cephalosporins, quinolones and diaminopyrimidines between groups. A p-value less than 0.05 was considered statistically significant [6].

Results and discussion

Poultry farming is a critical source of affordable protein worldwide. However, it faces ongoing threats from various poultry diseases that significantly impact public health, economic stability, and food security [16]. Therefore, the problem of colibacillosis has been and remains to this day one of the main causes of economic losses in poultry farming. Given the globalization of production, pathogenic strains of *E. coli* can not only act as a secondary factor in the pathological condition, but also serve as the main cause of death regardless of the age of the bird [7]. Moreover, there is evidence of vertical transmission of pathogenic *E. coli* strains from the breeders to their offspring, in particular to day-old chicks, as well as the transfer of virulence and resistance genes to commensal microbiota, including *E. coli* [17]. Thus, understanding the transmission routes, risk factors and survival characteristics of the most important pathogens affecting poultry populations, as well as maintaining strict biosecurity, are key points [16].

The poultry population was examined for the presence of colibacillosis in one production zone of the farm during the productive period. We identified four age intervals during which an increased mortality rate was observed ranging from 0.6 to 1.3%. The increase in livestock mortality was observed during the period of the greatest psychological stresson the poultry and the influence of possible stress factors, which were not analyzed in this work. In particular, from the 150th day, birds undergo significant hormonal changes, which are associated with the process of laying and fertilization. This leads to a weakening of the immune response through the synthesis of the stress hormone corticosterone, which inhibits the proliferation of cytotoxic lymphocytes and natural killers, thereby disrupting the functioning of the cellular link of immune defense aimed at combating bacterial infection [18].

Twenty-three *E. coli* isolates were detected in broiler parent flocks, 14 of which were considered pathogenic, because they were detected simultaneously in multiple organs, suggesting a systemic infection (Table 1).

To conclude, among the main pathological changes caused by pathogenic isolates of *E. coli*, fibrinous deposits were observed on internal organs, which progressed with age and indicated a chronic stage of the disease. These results coincide with

Table 1

Isolates of *E. coli* detected in broiler-breeders

Features	**Age categories (days)**			
Age	180–190	240–250	310–320	380–390
Number of breeders	6	5	6	7
Number of isolates / pathogenic	5/4	5/3	7/4	6/3

In dead broiler-breeders at 185 days, the main pathological changes were the development of fibrinous peritonitis (3 points). Fibrinous lesions of the liver and heart (1–2 points) were also detected. However, the air sacs were without signs of aerosacculitis, which excludes infection of chickens with *E. coli* by airborne droplets. The spleen was enlarged, hyperemic, which indicated the immune system's reaction to the presence of inflammation. However, no pathological changes were detected in the trachea, lungs, kidneys and stomach. The ovaries were formed without signs of inflammation. In broiler-breeders at 240–250 days, pathological changes were similar to those observed earlier age.

It should be noted that with age, the progression and degree of organ damage increased significantly. Thus, signs of fibrinous pericarditis, perihepatitis, peritonitis and ovaritis (3 points) were found in 100% of the dead birds (Fig. 1). In addition, the liver was of a dense consistency with signs of connective tissue replacement, which could indicate a chronic stage of bacterial infection. Moreover, after 380 days, some cases of coligranulomatosis were detected, a chronic form of old *E. coli* affection.

data presented by Khairullah A. et al. about *E. coli* lesion variety in different age periods of birds [19].

To evaluate the impact of colibacillosis in the breeder flock on broiler offspring, 10 groups of one-day-old chicks of 10 individuals each were examined with an interval of 21 days, which corresponds to the incubation period. As a result of the quality assessment on the Pasgar scale during 10 hatchlings, scores of no less than 9.5 points were obtained, which is considered a fairly high point. And the average values according to the Pasgar scale, considering viability, the condition of the navel, skin of the legs, beak and abdomen, were 9.7 points, which indicates a fairly high mark and good quality. The worst among all the evaluated parameters was the viability of the chicks, as well as the tightness in the thoracic cavity, which was noted in 9.4 and 8.3% of the chicks, respectively. In our opinion, this may be a likely result of the influence of bacterial infection because of egg contamination or vertical transmission of the pathogen (Fig. 2) [20].

During autopsy, 79% of day-old chicks showed a change in the color of the yolk sac from brown to green, as well as signs of omphalitis. According to data from other authors [20] pathogenic strains of

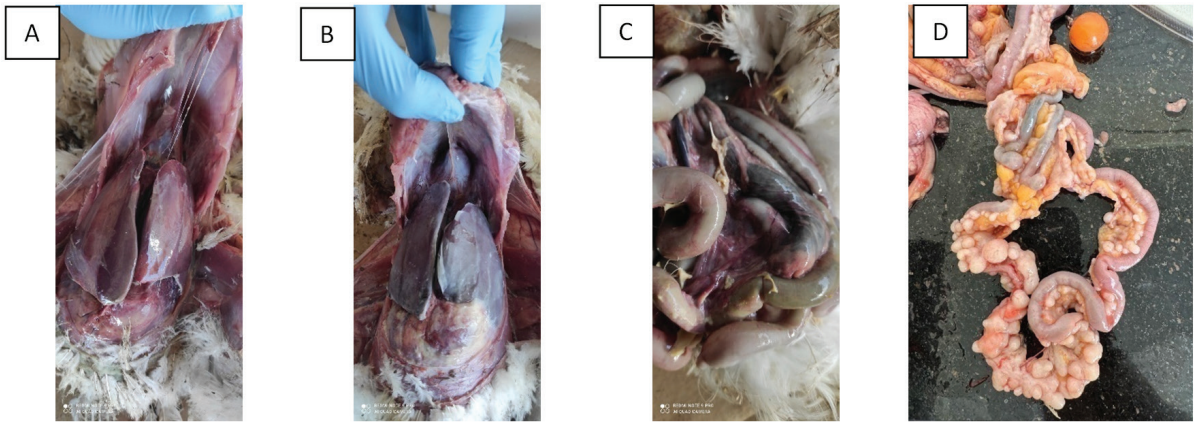

Fig. 1. Lesion that was observed in dead birds: A – development of fibrinous perehepatitis, pericarditis (2 points); B – fibrinous pericarditis and perihepatitis (3 points); C – peretonitis; D – granulomatous formation on the intestine

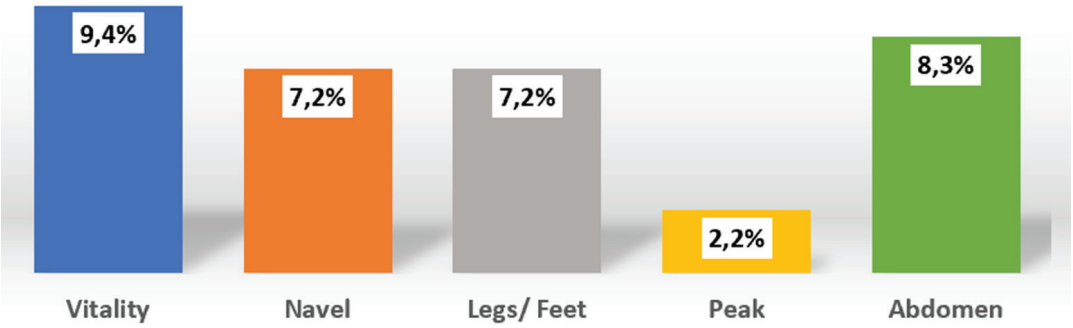

Fig. 2. Number of chick (%) with visual affection regarding Pasgar evaluation

E. coli are the most common cause of omphalitis affecting chicks in the first 7 days of life. In addition, in 6% of cases, signs of fibrinous pericarditis were observed, indicating the presence of a bacterial infection. In most chicks, the liver was icteric without signs of inflammation. The spleen was swollen, the cecum was distended with excess gas, and slight erosions on the cuticle were observed in the stomach, which indicated the development of cuticulitis and is not considered a pathognomonic sign of colibacteriosis.

During the bacteriological study of 1-day-old chickens, 67 isolates of *E. coli* were isolated, of which 51 (i.e., 76%) were considered pathogenic. This indicates a systemic course of bacterial infection and a septic state in chickens. According to the results of PCR, *Salmonella* spp., *Mycoplasma synoviae* and *Mycoplasma galisepticum* were not detected. Instead, a positive result for *E. coli* was recorded in all studied groups, with the Ct value ranging from 19.6 to 35.1 units. It should be noted that in the case when Ct $\leq$ 32.0, the amount of genetic material of bacterial cells is significant and may indicate their virulence, since omphalitis was detected in most of the studied chickens.

Comparing the biochemical features of *E. coli* isolated from broiler-breeders and their progeny, it should be noted their possibility to produce biogenic amines, for instance putrescine and cadaverin that are toxic for microorganisms [21]. Furthermore, these toxins can react with nitrites to form carcinogenic nitrosamines, which cause dilatation of peripheral blood vessels, capillaries, and arteries, thus resulting in hypotension, flushing [22].

Although the majority of *E. coli* cultures isolated from the parent flock (64.6%) and their chickens (57.1%) were lactose negative. However, according to Kaczmarek et al. [23], there was no correlation between virulence and lactose fermentation capacity.

Serotyping is one of the methods used for typing and *E. coli* classification [24]. The majority of pathogenic *E. coli* detected from breeders' flock were classified as serogroups O78 (57.1%, n = 8) and O18 (21.4%, n = 3). Among pathogenic *E. coli* isolated from chickens, representatives of serogroups O78 (49.0%, n = 25) and O18 (19.6%, n = 10) were also

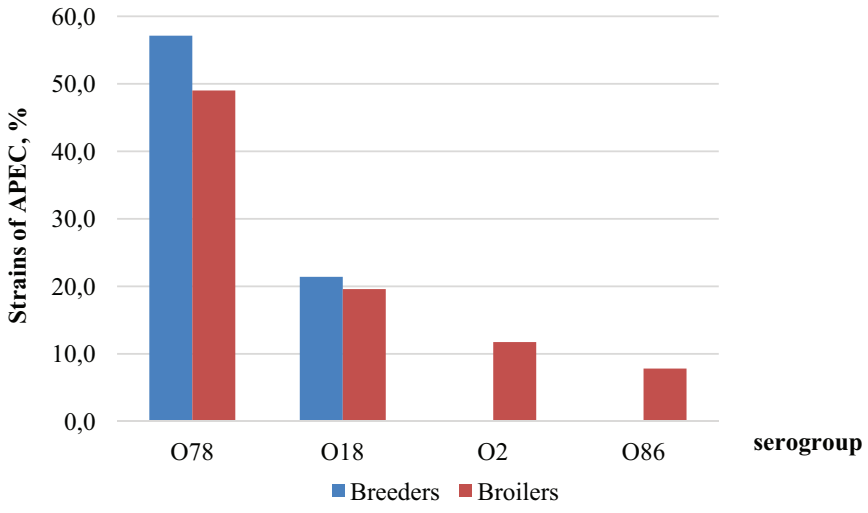

Fig. 3. Prevalence of different serotypes of APEC isolated from breeders and broiler chicks

identified. Also, the variability of serogroups in chickens was wider, it was detected O2 and O86 types, and non-classified isolates, which could be O5, O18, O35, O109, O115 or others, and were not included in diagnostic kit (Fig. 3) [25,26].

However, no correlation between serotype and virulence was detected. Different isolates of any serogroup could acquire several virulence factors. These genes include iron acquisition genes (*iucD*, *iroN*), adhesion genes (*tsh*), toxin genes (*vat*, *hlyF*), serum resistance genes (*iss*), housekeeping proteases (ompT), and ColV operon genes (*cvi/cva*) due to plasmids, phages, pathogenicity islands and others mobile genetic elements [6].

As mentioned above, *E. coli* bacteria generate multiple virulence factors that aid its pathogenicity in extra-intestinal infections. Also, these factors could be transmitted to normal microbiota and influence host-pathogen interactions. In our investigation, none of the pathogenic *E. coli* isolates were characterized by β-haemolysis, and fewer then 7,7% had α-haemolysis. Various studies reported the avian *E. coli* as non-hemolytic and independent of haemolytic activity, which corresponds with our results. However, some reports suggested that *E. coli* has the ability to produce haemolysin and causes the release of iron from erythrocytes which helps in the development of systemic bacterial infection [27,28].

Nevertheless, the use of antibiotics has a major impact on the development of resistance. Antimicrobials are still the main tool for treatment in poultry farming. However, there is evidence that *E. coli* strains can quickly develop the resistance under certain circumstances [29].

In our research, it was shown that isolated *E. coli* were most often resistant to amoxicillin (100%) and amoxiclav (98%), the group of beta-lactame antibiotics which inhibit cell wall synthesis. Also, we detected high level of resistance to tetraclines, more than 78,6 and 85,7% of isolated *E. coli* from breeder's flock were non vulnerable to doxycycline and oxytetraciline, respectively. A similar trend was observed in APEC isolated from chickens (Fig. 4).

It should be noted that we observed high resistance to chinolones, for instance, enrofloxacine. There were detected near 78.6% and 72.5% resistance strains of *E. coli* in breeders and chicks, respectively. Such tandnacy could be explained by prophylactic usage of enrofloxacine and amoxicilline every 4 weeks in productive age of broiler breeders. That high level of resistance in chickens could be due to vertical or horyzontal transmission of APEC from parents to progeny flocks. This data coralated with results of Joseph J. et al. [17]. Moreover, ciprofloxacine and norfloxacine characterized by high number of resistant strains isolated from broiler breeders (57.1% and 64.3%) and chickens (51.0% and 47.1%). The effectivity of flumequine, as antimicrobial from the same group, also was low because of crossresistance development [30].

Amynoglycosides are the group of antimicrobials with wide spectrum and possibility to inhibit the proteins synthesis after binding to 30S ribosome [31]. Among amynoglycosides the highest level of resistance was observed to spectymomycin. However, the number of resistant isolates of APEC to gentamycine was the lowest and described as 14.3% and 9.8% from broiler-breeders and chickens,

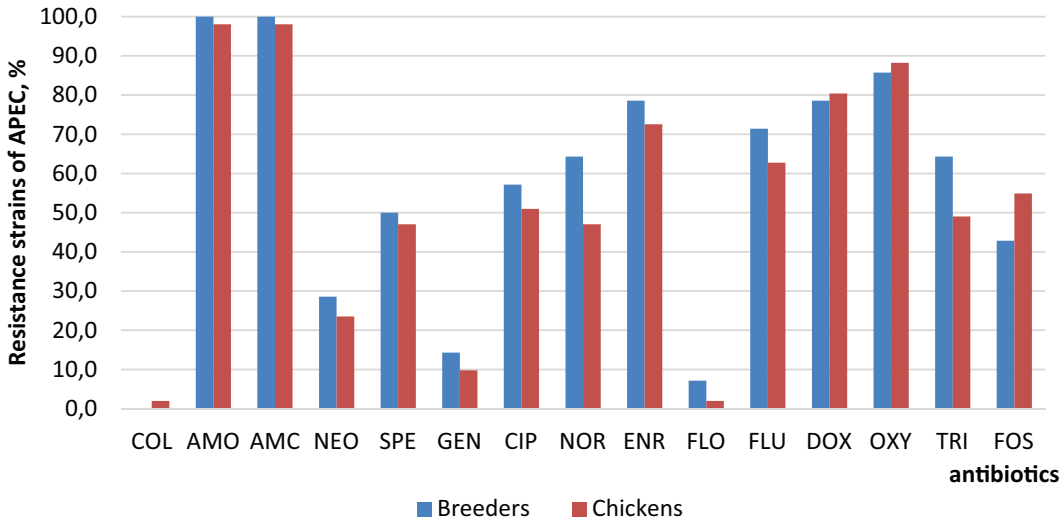

Fig. 4. Number of APEC isolates resistant to antimicrobials: COL – colistin, AMO – amoxicillin, AMC – amoxiclav, NEO – neomycin; SPE – spectinomycin; GEN – gentamycin; CIP – ciprofloxacin; NOR – norfloxacin; ENR – enrofloxacin; FLO – florfenicol; FLU – flumequine; DOX – doxycycline; OXY – oxytetracycline; TRI – trimethoprim; FOS – fosfomycin

respectively. That indicates its high efficiency. It should be noted that amynoglycosides after oral usage is effective only in gastro-intestinal tract, that is why it is necceery to use parenteral route to combat systemic infection (Fig. 4). The resistance profile of isolated APEC to amynoglycosides corresponds to datas described on the work of Nechypurenko O. et al. [14].

Nevertherless progressive adaptation of APEC was the lowest level of resistance detected to colistin and florfenicol, bactericidal antibiotics (Fig. 4). It was observed only one resistant strain in chickens during all investigated time. However, colistin as aminoglycosides after application via drinking water works only in the intestine that excluded the influence on systemic infection. Also, the number of florfenicol resistant *E. coli* strains varied from 7.1% to 2.0% in broiler-breeders and chickens, respectively. That indicates the probable influence of broiler-breeders APEC on florfenicol resistance formation in chickens. Basically, florfenicol is not recommended to use in one day old chickens because of negative impact to the growth and development of chicks, and the body weight and immune organ index. Histopathological examination showed that there was a decrease in the number of lymphocytes in the bursa of Fabricius in the treatment group [32].

Nowaday the usage of trimethoprim during colibacilosis became more often ineffective, for instance it was identified 64.3% and 49.0% resistant strain in parental and progeny flocks. That is why vets began to find new solution such as fosfomycin, originally called phosphonomycin, which is a phosphonic acid derivate. Fosfomycin interferes with the early stages of peptidoglycan production, inhibiting UDP-N-acetylglucosamine enolpyruvyl transferase (MurA) enzyme. MurA enzyme catalyzes the formation of peptidoglycan precursor, N-acetylmuramic acid. The binding FOS to MurA and, thus, the inability to proceed in peptidoglycan formation result in a bactericidal activity of the drug [33]. Despite this fact the number of resistant isolated APEC strains varied from 42.9 to 54.9% in broiler-breeders and 1-day old chickens. Such tandancy could be due to the influence of amynoglycosides to crossprotection development with fosfomycine.

It is necessary to add that during research period we isolated 5 multiresistant (non vulnerable to more than 3 antibiotics) strains of pathogenic *E. coli* for example that can be damaged only by colistin, florfenicol and gemtamicin. All multiresistant APEC were isolated at the final stage of the research that indicated resistance development after prolonged antimicrobial application that also influenced *E. coli* isolated from progeny chickens.

Therefore, further investigation on genetic level dedicated to relationship between APEC isolated from broilers-breeders and their chicken should be done. Preventative measueres agains *E. coli* on parental flocks such as probiotics, live and inactivated vaccine application could play a crucial role in disease control in broiler farming.

Conclusion

It was detected 65 pathogenic *E. coli* isolates from broiler-breeders and 1 day old chikens. That has shown a huge impact of collibacilosis to poultry farming in different levels of production. The main lesions were associated with fibrin deposition and omphlaitis formation. Amomg APEC the most prevalent were O78 and O18 serotypes wich characterized by absence of β-haemolisis. Moreover, the antibiotic profile of APEC isolated from parenteral and progeny flocks was similar. There was found a high leve of resistance to amoxicillin, amoxiclave, doxycycline, tetracycline and enrofloxacine, more than 70% of isolated APEC was unvulnarable. If coliform pathogens are transmitted to broiler chickens, the risk of developing this infection increases and the quality of the chickens deteriorates. In addition, the treatment of birds for a long time leads to the formation of multi-resistant strains that affect chickens from the first days of life. Thus, studying the process of APEC infection through an integrated production chain can be useful for taking the appropriate measures to prevent early cases of coliforms.

References

1. Wilczyński J, Stępień-Pyśniak D, Wystalska D, Wernicki A. Molecular and Serological Characteristics of Avian Pathogenic *Escherichia coli* Isolated from Various Clinical Cases of Poultry Colibacillosis in Poland. Animals. 2022;12(9):1090. doi:10.3390/ani12091090
2. Chenouf NS, Messaï CR, Carvalho I, Álvarez-Gómez T, Silva V, Zitouni A, Hakem A, Poeta P, Torres C. Serogrouping and Molecular Characterization of ESBL-Producing Avian Pathogenic *Escherichia coli* from Broilers and Turkeys with Colibacillosis in Algeria. Antibiotics. 2025;14(9):356. doi:10.3390/antibiotics14040356
3. Pilati GVT, Salles GBC, Savi BP, Dahmer M, Muniz EC, FilhoVB, Elois MA, Souza DFSM, Fongaro G. Isolation and Characterization of *Escherichia coli* from Brazilian Broilers. Microorganisms. 2024;12:1463. doi:10.3390/microorganisms12071463
4. Apostolakos I, Laconi A, Mughini-Gras L, Yapicier ÖS, Piccirillo A. Occurrence of Colibacillosis in Broilers and Its Relationship with Avian Pathogenic *Escherichia coli* (APEC) Population Structure and Molecular Characteristics. Front. Vet. Sci. 2021;8:737720. doi:10.3389/fvets.2021.737720
5. Denamur E, Clermont O, Bonacorsi S, Gordon D. The population genetics of pathogenic *Escherichiacoli*. Nature Reviews Microbiology. 2021;19:37–54. doi:10.1038/s41579-020-0416-x
6. Johar A, Al-Thani N, Al-Hadidi SH, Dlissi E, Mahmoud MH, Eltai NO. Antibiotic Resistance and Virulence Gene Patterns Associated with Avian Pathogenic *Escherichia coli* (APEC) from Broiler Chickens in Qatar. *Antibiotics*. 2021;10(5):564. doi:10.3390/antibiotics10050564
7. Hu J, Afayibo DJ, Zhang B, Zhu H, Yao L, Guo W, Wang X, Wang Z, Wang D, Peng H, Tian M, Qi J, Wang S. Characteristics, pathogenic mechanism, zoonotic potential, drug resistance, and prevention of avian pathogenic *Escherichia coli* (APEC). Front. Microbiol. 2022;13. doi:10.3389/fmicb.2022.1049391
8. Kathayat D, Lokesh D, Ranjit S, Rajashekara G. Avian Pathogenic *Escherichia coli* (APEC): An Overview of Virulence and Pathogenesis Factors, Zoonotic Potential, and Control Strategies. Pathogens. 2021;10(4):467. doi:10.3390/pathogens10040467
9. Giovanardi D, Campagnari E, Ruffoni LS, Pesente P, Ortali G, Furlattini V. Avian pathogenic *Escherichia coli* transmission from broiler breeders to their progeny in an integrated poultry production chain. Avian Pathology. 2005;34(4):313-8. doi:10.1080/03079450500179046
10. Hussein EA, Hair-Bejo M, Adamu L, Omar AR, Arshad SS, Awad EA, Aini I. Scoring system for lesions induced by different strains of Newcastle Disease virus in chicken. Vet. Med. Int. 2018. doi:10.1155/2018/9296520
11. Afayibo DJ, Zhu H, Zhang B, Yao L, Abdelgawad HA, Tian M, Qi J, Liu Y, Wang S. Isolation, molecular characterization, and antibiotic resistance of Avian Pathogenic *Escherichia coli* in Eastern China. Vet Sci. 2022;9(7):319. doi:10.3390/vetsci9070319
12. Fritsche TR, McDermott PF, Shryock TR, Walker RD. Agar Dilution and Disk Diffusion Susceptibility Testing of *Campylobacter spp*. J Clin Microbiol. 2007;45:2758-59. doi:10.1128/jcm.00569-07
13. Kohanski MA, Dwyer DJ, Collins JJ. How antibiotics kill bacteria: from targets to networks. Nature Reviews Microbiology. 2010;8:423-35. doi:10.1038/nrmicro2333
14. Nechypurenko O, Avdeeva L, Dreval D, Sobko I. Avian Pathogenic *Escherichia coli* and its Antibiotic Resistance. Mikrobiolohichnyi Zhurnal. 2024;86(5):61-74. doi:10.15407/microbiolj86.05.061
15. Khac VH, Holoda E, Pilipcinec E, Blanco M, Blanco JE, Mora A, Dahbi G, López C, González EA, Blanco J. Serotypes, virulence genes, and PFGE profiles of *Escherichia coli* isolated from pigs with postweaning diarrhoea in Slovakia. BMC Vet Res. 2006;2:10. doi:10.1186/1746-6148-2-10
16. Kovács L, Domaföldi G, Bertram P-C, Farkas M, Könyves LP. Biosecurity Implications, Transmission Routes and Modes of Economically Important Diseases in Domestic Fowl and Turkey. Veterinary Sciences. 2025;12(4):391. doi:10.3390/vetsci12040391
17. Joseph J, Zhang L, Adhikari P, Evans JD, Ramachandran R. Avian Pathogenic *Escherichia coli* (APEC) in Broiler Breeders: An Overview. Pathogens. 2023;12(11):1280. doi:10.3390/pathogens12111280
18. Rosales AG. Managing Stress in Broiler Breeders: A Review. Journal of Applied Poultry Research. 1994;3(2):199-207. doi:10.1093/japr/3.2.199.
19. Khairullah AR, Afnani DA, Riwu KHP, Widodo A, Yanestria SM, Moses IB, Effendi MH, Ramandinianto SC, Wibowo S, Fauziah I, Kusala MKJ, Fauzia KA, Furqoni AH, Raissa R. Avian pathogenic Escherichia coli: Epidemiology, virulence and pathogenesis, diagnosis, pathophysiology, transmission, vaccination, and control. Veterinary World. 2024;17(12):2747-62. doi:10.14202/vetworld.2024.2747-2762
20. Steffen RPB, Queiroz B, Silva AT, Rocha VGP, Franco LS, Barbosa FB, Knöbl T. Avian pathogenic *Escherichia coli* (APEC) isolated from chicks and embryos in the hatchery. Braz J Microbiol. 2025. doi:10.1007/s42770-025-01686-x
21. Akinwande FO, Jimon SO. Evaluation of biogenic amines producing potential of bacteria isolated with proteinous food. Malaysian Journal of Microbiology. 2020;16(1):1-6. doi:10.21161/mjm.180131
22. Turna SN, Chung R, Mcintyre L. A review of biogenic amines in fermented foods: Occurrence and health effects. Heliyon. 2024. doi:10.1016/j.heliyon.2024.e24501
23. Kaczmarek A, Skowron K, Budzyńska A, Grudlewska K, Gospodarek-Komkowska E. Virulence genes and antimicrobial susceptibility of lactose-negative and lactose-positive strains of *Escherichia coli* isolated from pregnant women and neonates. Folia Microbiol. 2017;62:363-71. doi:10.1007/s12223-017-0506-y
24. Alber A, Morris KM, Bryson KJ, Sutton KM, Monson MS, Chintoan-Uta C, Borowska D, Lamont SJ, Schouler C, Kaiser P, Stevens MP, Vervelde L. Avian Pathogenic *Escherichia coli* (APEC) strain-dependent immunomodulation of respiratory granulocytes and mononuclear phagocytes in CSF1R-reporter transgenic chickens. Front. Immunol. 2020;10. doi:10.3389/fimmu.2019.03055
25. Mehat JW, van Vliet AHM, La Ragione RM. The Avian Pathogenic *Escherichia coli* (APEC) pathotype is comprised of multiple distinct, independent genotypes. Avian Pathology. 2021;50(5):402-16. doi:10.1080/03079457.2021.1915960
26. Joensen KG, Tetzschner AM, Iguchi A, Aarestrup FM, Scheutz F. Rapid and Easy *in silico* Serotyping of *Escherichia coli* Isolates by Use of Whole-Genome Sequencing Data. J Clin Microbiol. 2015;3(8):2410-26. doi:10.1128/JCM.00008-15
27. Saha O, M Hoque N, Kibria I, Rahaman M, Sultana M, Hossain AM. Multidrug-Resistant Avian Pathogenic *Escherichia coli* Strains and Association of Their Virulence Genes in Bangladesh. Microorganisms. 2020;8(8):1135. doi:10.3390/microorganisms8081135
28. Singh A, Chhabra D, Gangil R, Sharda R, Sikrodia R, Audarya S. Studies of Virulence Factors of Avian Pathogenic *Escherichia coli* in Avian Colibacillosis by *in vitro* Method and PCR. Indian Journal of Animal Research. 2023;57(4):499-504. doi:10.18805/IJAR.B-4329
29. Lucio C, Hansen P, Griebeler J, Kipper D, Lunge V. Virulence and Antimicrobial Resistance of Avian Pathogenic *Escherichia coli* (APEC) Isolates from Poultry in Brazil. Poultry. 2025;4(1):10. doi:10.3390/poultry4010010
30. Swinkels A, Fischer E, Korving L, Christodoulou R, Wagenaar J, Zomer A. Flumequine, a fluoroquinolone in disguise. NPJ Antimicrob Resist. 2024;2(1):28. doi:10.1038/s44259-024-00044-5
31. Tsai A, Uemura S, Johansson M, Puglisi E, Marshall A, Aitken C, Korlach J, Ehrenberg M, Puglisi J. The impact of aminoglycosides on the dynamics of translation elongation. Cell Rep. 2013;3(2):497-508. doi:10.1016/j.celrep.2013.01.027
32. Meng F, Liu K, Shen Y, Li P, Wang W, Zhao Y, Liu S. Florfenicol can inhibit chick growth and lead to immunosuppression. Journal of Integrative Agriculture. 2025;24 (1):261-71. doi:10.1016/j.jia.2023.11.040
33. Marchetti V, Hrabak J, Bitar I. Fosfomycin resistance mechanisms in Enterobacterales: an increasing threat. Cell. Infect. Microbiol. 2023;13. doi:10.3389/fcimb.2023.1178547

Нечипуренко О. О.[1, 2], Фуртат І. М.[1], Древаль Д. В.[3], Авдєєва Л. В.[2]

[1] Національний університет «Києво-Могилянська академія» (НаУКМА), Київ, Україна

[2] Інститут мікробіології і вірусології ім. Д. К. Заболотного НАН України, Київ, Україна

[3] ТОВ «Центр ветеринарної діагностики», Київ, Україна

КОЛІБАКТЕРІОЗ У КУРЧАТ-БРОЙЛЕРІВ ТА ЙОГО ЕТІОЛОГІЧНИЙ ЗВ'ЯЗОК ІЗ БІОЛОГІЧНИМИ ХАРАКТЕРИСТИКАМИ ВИДІЛЕНИХ ШТАМІВ *ESCHERICHIA COLI* З ПЛЕМІННОГО ПОГОЛІВ'Я

Мета дослідження – з'ясувати наявність етіологічного зв'язку між колібактеріозом в одноденних курчат та біологічними характеристикамии ізолятів *E. coli*, які було виділено від птахів із племінного стада. **Методи.** Дослідження проводили на одній з птахофабрик із закритим циклом вирощування протягом усього продуктивного періоду. Для оцінювання патологічних змін здійснювали розтин загиблих курат та батьків-бройлерів і визначали ступінь їхнього ураження. Ідентифікацію бактерій проводили за допомогою тест-системи Api20E, з подальшим серотипуванням ідентифікованих ізолятів та оцінюванням їхньої гемолітичної активності. Якість і життєздатність добових курчат визначали за протоколом Global Hatchery Health Programme. Чутливість виділених ізолятів *E. coli* до антибіотиків визначали методом Кірбі – Бауера. **Результати**. Від бройлерів-плідників (n = 14) та одноденних курчат (n = 51) було виділено 65 патогенних ізолятів *E. coli*. Це засвідчило значний вплив колібактеріозу на птахівництво на різних рівнях виробництва. Основні ураження були пов'язані з відкладенням фібрину та утворенням омфлаїту. Останній міг вплинути на життєздатність курчат, проте середній бал за шкалою Пасгара становив 9,7. Серед досліджених патогенних пташиних кишкових паличок (Avian pathogenic *Escherichia coli*, APEC) найпоширенішими були серотипи O78 та O18, які характеризувалися відсутністю β-гемолізу. Ба більше, антибіотичний профіль APEC, виділених із парентеральних та потомствених стад, був подібним. У досліджених ізолятів було виявлено високий рівень резистентності до амоксициліну, амоксиклаву, доксицикліну, тетрацикліну та енрофлоксацину. Понад 70 % виділених APEC були невразливими до згаданих антимікробних препаратів. Найефективнішими антибіотиками виявилися колістин, гентаміцин та флорфенікол, стійкість до яких спостерігалася лише 0,0 % та 2,0 %; 14,3 та 9,8 %; 7,1 % та 2,0 % виділених APEC від бройлерних племінних порід та курчат відповідно. **Висновки**. Отже, у цьому дослідженні встановлено безпосередній зв'язок між ізолятами APEC, виділеними від племінної птиці та курчат-бройлерів. Крім того, показано, що тривале лікування птахів призводило до формування мультирезистентних штамів кишкової палички, які вражали курчат із перших днів життя. Вивчення процесу інфікування патогенними *E. coli* через інтегрований виробничий ланцюг може бути корисним для вжиття відповідних заходів щодо запобігання раннім випадкам колібактеріозу.

Ключові слова: патогенна пташина кишкова паличка (APEC), стійкість до антибіотиків, колібактеріоз.

ARTICLE HISTORY. Submitted 26 June 2025. Accepted 30 June 2025. Published 18 August 2025

Відомості про авторів
Authors Information

Нечипуренко Олексій Олександрович – кандидат біологічних наук, старший викладач кафедри біології факультету природничих наук Національного університету «Києво-Могилянська академія» (НаУКМА), докторант відділу антибіотиків Інституту мікробіології і вірусології ім. Д. К. Заболотного НАН України, Київ, Україна

Nechypurenko Oleksii – Candidate of Biological Sciences, Senior Lecturer at the Department of Biology of National University of Kyiv-Mohyla Academy (NaUKMA), PostDoc Program at Antibiotic Department of D. K. Zabolotny Institute of Microbiology and Virology of the National Academy of Sciences of Ukraine, Kyiv, Ukraine

https://orcid.org/0009-0008-5803-6723

ne4upura@ukr.net

Фуртат Ірина Михайлівна – кандидат біологічних наук, доцент кафедри біології факультету природничих наук Національного університету «Києво-Могилянська академія» (НаУКМА), Київ, Україна

Furtat Iryna – Candidate of Biological Sciences, Associate Professor at the Biology Department of National University of Kyiv-Mohyla Academy (NaUKMA), Kyiv, Ukraine

https://orcid.org/0000-0003-0681-2889

furtat@ukma.edu.ua

Древаль Денис Вікторович – магістр з ветеринарної медицини, завідувач відділу патанатомії та бактеріології ТОВ «Центр ветеринарної діагностики», Київ, Україна

Dreval Denis – Master in Veterinary Medicine, Head of Pathology and Bacteriology Department of Center for Veterinary Diagnostics, Kyiv, Ukraine

https://orcid.org/0009-0008-0416-9619

dreval_denis@ukr.net

Авдєєва Лілія Василівна – доктор медичних наук, професор, завідувачка відділу антибіотиків Інституту мікробіології і вірусології ім. Д. К. Заболотного НАН України, Київ, Україна

Avdeeva Liliya – Doctor of Science in Medicine, Professor, Head of Antibiotics Department, D. K. Zabolotny Institute of Microbiology and Virology of the National Academy of Sciences of Ukraine, Kyiv, Ukraine

https://orcid.org/0000-0002-8458-444X

avdeeva_liliya@imv.ukr.net