БІОЛОГІЯ

BIOLOGY

DOI: 10.18523/2617-4529.2026.9.22-29
UDC 616.98:578.835.2:579.842.11:636.52/.58

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

[1] National University of Kyiv-Mohyla Academy (NaUKMA), 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

COINFECTION OF AVIAN PATHOGENIC *ESCHERICHIA COLI* AND INFECTIOUS BURSAL DISEASE VIRUS IN BROILER CHICKENS

Abstract

Infectious bursal disease virus (IBDV) is the main immunosuppressive agent in the poultry industry that stimulates the development of secondary bacterial infection. However, the avian pathogenic E. coli (APEC) could be a primary agent that induces the development of colibacillosis, nevertheless the absence of immunosuppression. The depletion of bursa after viral replication is crucial for starting bacterial dissemination because of devastation of systemic and local humoral immunity. Therefore, the aim of the work was to investigate the correlation between APEC and IBDV infection in broiler chickens. The bursal atrophy was observed from 21 days with further level depletion from 60 to 90% at the final stage of research. Moreover, APEC strains were isolated from birds at 28–42 days which is correlated with chronical immunosuppression. All E. coli isolates showed resistance to beta-lactams (amoxicillin, amoxiclav), tetracyclines, and trimethoprim, highlighting the risk of multidrug-resistant strains. There were no resistant strains to colistin and florfenicol. The IBDV strains were detected in the bursa of 21, 28 and 35 broilers with Ct level 22.8; 25.8; and 32.2 points, respectively that indicates decreasing the viral load. Based on partial nucleotide sequences of the VP2 gene, the pathogen was identified as very virulent United Kingdom 2019 strain. However, analysis of the samples from 42-day-old broilers revealed that the nucleotide sequence belonged to the vaccine strain.

Therefore, coinfection of APEC and IBDV in broiler chickens enhances immunosuppression, creating conditions for secondary bacterial infections. The susceptibility to colibacillosis correlates with the stage of bursal depletion and may persist even during replication of the vaccine strain of IBDV after primary infection.

Keywords: infectious bursal disease virus (IBDV), Gumboro disease, avian pathogenic *E. coli* (APEC), coinfection, colibacillosis, antimicrobial resistance.

Introduction

Infectious bursal disease virus (IBDV) is a leading immunosuppressive agent which is widely spread all over the world and causes a highly contagious disease in poultry, also known as Gumboro disease. To date, this disease is prevalent in most of the poultry-producing regions of the world [1,2]. The virus primarily targets and destroys immature B lymphocytes in the bursa of Fabricius (BF), leading to severe immunosuppression, which may lead to high mortality rates in susceptible chickens, secondary infections including also gut-associated diseases, which contribute to the economic losses in the poultry industry, and reduced efficacy of vaccination against other pathogens [3,4].

IBDV belongs to the family *Birnaviridae* and the genus *Avibirnavirus*, with a bi-segmented double-stranded RNA genome represented by segments A and B. This virus has two serotypes, namely 1 and 2. Both serotypes of the IBDV can naturally infect chicken, turkey, duck, guinea fowl, ostriches. Although serotype 1 is mostly pathogenic for chickens. This virus can easily mutate and evolve into variant strains despite progressive vaccination strategies. These strains induce subclinical forms characterized by bursal atrophy, weight loss and increase in mortality rate because of systemic colibacillosis. Reemergent infections caused by antigenic variants of IBDV, can be the reason for significant losses and high mortality in chickens due to coinfection with pathogenic strains of *Escherichia coli* [2,5]. Avian colibacillosis, caused particularly by pathogenic variants of *E. coli* (notably so-called APEC strains), is one of the most prevalent infectious diseases affecting domestic, ornamental, and wild birds. This disease is usually associated with various predisposing factors (both infectious and non-infectious). It is widely recognized that infectious bursal disease virus (IBDV) may be one of the most important infectious factors contributing to the development of colibacillosis. Infection of chickens with infectious bursal disease virus (IBDV) resulted in a significant increase in their susceptibility to *E. coli*. Furthermore, it has been demonstrated that lymphocytopenia of the bursa of Fabricius is primarily induced by IBDV. In contrast, a significant depletion of lymphocytes in both the bursa of Fabricius and the thymus can also occur during *E. coli* infection [6].

Even though in different conditions APEC-strains could be a primary or secondary pathogen, these bacteria often cause the death of birds because of acute septicemia, subacute pericarditis, chronic respiratory diseases, fibrinous-purulent polyserositis, airsacculitis, and many other forms. 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 (TSH) plays a role in the colonization of the air sacs; the aerobactin iron-acquisition 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. Moreover, the genetic background can influence the bacterium's ability to acquire, maintain, or express pathogenic traits [7,8]. 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. Also, correlation between Gumboro disease virus and APEC coinfection and severity of lesions is not completely investigated. Therefore, the aim of the work was to investigate the role of avian pathogenic *E. coli* in pathological process during bursal disease virus coinfection in broilers.

Material and methods

The research was conducted on one of the poultry farms of Vinnytsia region, in which previously was isolated very virulent Gumboro disease virus. The broilers were vaccinated regarding standard protocol and immunized in 1 day with immunocomplex vaccine against Gumboro disease.

At the age of 21-, 28-, 35- and 42-days dead birds (n = 9 per group) from one of poultry houses were examined for the presence of lesions in trachea, lungs, air sacs, heart, liver, spleen, kidneys, stomach, small and large intestine, bursa. The degree of lesions was evaluated from 0 to 3 points: 0 – absent, 1 – mild, 2 – moderate and 3 – severe based on the presence of fibrine deposition, hemorrhages, oedema and inflammation [9].

For histological examination, non-frozen samples without signs of autolysis were taken from bursa of broilers (n = 6) of mentioned above age. The size of the samples was 1–2 cm. The selected material was immediately transferred to 10% buffered formalin solution for fixation. After that, samples were cut with blades (thickness of the section was 3–4 mm, width, and length no more than 1.5 cm) considering the peculiarities of the structure and placed in Turboflow plastic cassettes. Then tested samples were processed using automated stations regarding standard protocol [10]. After dehydration and paraffinization of the tissues, the samples were filled with liquid paraffin. At the next stage, pathological material embedded in paraffin were sectioned with slice thickness near 5 μm. Obtained slides were stained with hematoxylin and eosin and sealed in Mounting Medium Gue. The percentage of bursa depletion was further calculated [11].

For IBDV detection the stamps from the affected bursa of dead broilers to FTA cards were made. The collected material was sent to the Hipra Diagnose laboratory for qPCR and further sequencing of VP2 region based on conventional protocol. A reverse transcription-polymerase chain reaction (RT-PCR) test was conducted on all samples, amplifying a specific portion of the IBDV genome (VP2 gene). For positive results, nucleotide sequencing was performed using the Sanger methodology. The obtained sequences were then compared with both IBDV reference strains from Genbank and field strains [12].

The selection of biological material samples for further isolation of bacteria was carried out aseptically from liver, heart and spleen. 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). Isolates of *E. coli* were pathogenic if they were isolated from more than two organs. Bacterial isolates were identified using the API 20E test system (bioMerieux, France), which allows for the determination of the main biochemical diagnostically significant signs, as described in the article [13]. Also, the ability to produce hemolysins was identified.

The susceptibility of *E. coli* isolates to antimicrobials was determined by the Kirby-Bauer disk diffusion method on Mueller-Hinton medium (MHM, Himedia, India) [14]. It was used next antimicrobial compounds (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 (ATCC): *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 of Zabolotny Institute of Microbiology and Virology of the National Academy of Sciences of Ukraine, as described in [15].

Data entry, initial analysis, and figure design were done using Microsoft Office Excel 2010 (Microsoft Corporation, USA) to generate figures and run initial analysis as previously described [13].

Results and discussion

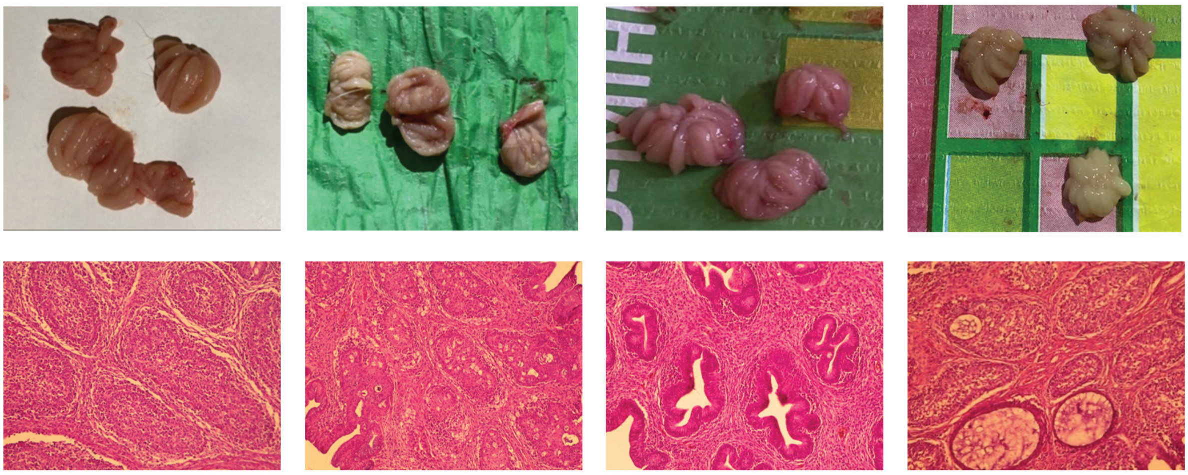
Infectious bursal disease virus (IBDV) causes a highly contagious disease in young chickens and is distributed worldwide. VP2 is the major viral antigen. The VP2 gene contains a hypervariable region (VP2 HRV). Mutations in this region lead to the emergence of antigenically different IBDV strains that nevertheless the vaccination program could induce severe immunosuppression and provoke colibacillosis in layers, broilers and breeders [16]. However, there are no data about APEC and IBDV coinfection stages and its correlation. Thus, research on the influence of immunosuppression on colibacillosis development can play a crucial role in the bacterial infection prevention strategy.

It was detected that the decrease of weight gain of broilers after 14 days of life by 5% compared to the reference Ross 308 points. After necropsy investigation, the presence of bursa atrophy was observed in 21 days. Atrophic lesions in the thymus and spleen were absent. There were no signs of fibrin deposition in heart, liver, and spleen. The respiratory and intestinal systems were without pathological changes. This state of birds could be due to the subclinical form of Gumboro disease [2]. It should be noted that in dead birds after 28 days different levels of fibrin deposition were identified in the mentioned organs that indicates the development of possible systemic bacterial infection. The level of lesion varied from 2 to 3 points because of progressive bacterial infection and organs dysfunction.

Histological examination of bursa explains its atrophy because of the B-lymphocyte depletion, the level of which was near 70%, 80%, 80% and 90% in 21, 28, 35 and 42 days, respectively. Also, there were detected the cystic formation and inflammation of follicular stroma membrane in the age of 35–42 days, which indicates the presence of chronic immunosuppression. There were no signs of hemorrhages (Fig. 1). However, such histological changes could be developed after affection by very virulent or variant strain of IBDV.

Regarding Nagy et al. the damaging of B-cells negatively influenced the formation of secretory immunoglobulins type A, which are important in the prevention of respiratory and intestinal tract colonization with APEC strains [17].

After bacteriological investigation APEC was isolated at the period of 28–42 days. There were only 5 different strains from dead birds (table). The bacteria were isolated from internal organs (spleen, heart and liver) which symbolize the systemic form of colibacillosis (Table 1). Nevertheless, despite their standard biochemical profile, 2 isolates of APEC were lactose negative. However, regarding Kaczmarek et al. [18] there was no correlation between virulence and lactose fermentation capacity.



21-day, depletion 60% 28-day, depletion 80% 35-day, depletion 90% 42-day, depletion 90%

Fig. 1. Lesion evaluation of birds at the age of 21–42 days (staining by hematoxylin and eosin, magnification × 200)

Table 1

Isolates of *E. coli* detected in broilers

Features	Age categories (days)			
Age	21	28	35	42
Number of broilers	6	6	6	6
Number of isolates / pathogenic	0/0	1/1	2/2	2/2

Various studies reported that avian pathogenic *E. coli* could be independent of hemolytic activity, which corresponds with our results. However, some reports suggested that the ability to produce hemolysin causes the release of the iron from erythrocytes which helps in the development of systemic bacterial infection [19].

It should be noted that APEC strains were isolated from birds with the highest level of immunosuppression observed after histological evaluation. Therefore, the affection by virulent IBDV strains could induce the development of bacterial infection.

Antimicrobials are still the main tool for treatment in poultry farming, nevertheless, ban tendency in their application. In our research it was shown that all isolated *E. coli* were resistant to amoxicillin and amoxiclav, the group of beta-lactam antibiotics which inhibit the cell wall synthesis. Also, we detected a 100% level of resistance to tetracyclines.

Regarding quinolones it should be noted that strains detected at the age of 42 days were resistant. This situation could be developed because of uncontrolled use of enrofloxacin and ciprofloxacin from the 1 day. There was no effectiveness of flumequine, as antimicrobial from the same group, that can be explained by cross-resistance formation [20].

Aminoglycosides are the group of antimicrobials that are widely used in veterinary field [21]. All isolated APEC were resistant to spectinomycin and active against gentamicin. However, the last-mentioned antimicrobial is effective only in gastro-intestinal tract that exclude its usage during systemic infection. Nevertheless, progressive adaptation of APEC the lowest level of resistance was detected to colistin and florfenicol that is correlated with data obtained by us in previous studies [13]. However, colistin as aminoglycosides after application via drinking water is not adsorbed into the blood.

Veterinarians very often use combined antimicrobials which contain quinolones and trimethoprim. However, regarding Spencer et al. data APEC characterized by high resistance to trimethoprim which could be due to including upregulation of *folA* expression and mutation of the binding pocket, leading to the loss of trimethoprim binding and thus inhibition of activity [22]. In our research all isolated strains were resistant to mentioned-above antimicrobial. Therefore, the application of multicomponent substances could be not effective against APEC and stimulate the formation of multidrug resistant bacteria.

It is known that bacteria usually are a secondary pathogen that finally kill the bird. However, APEC is due to virulence factors (adhesins, invasins, protectins, iron acquisition systems, and toxins) and high level of resistance regarding Kathayat et al. could be a primary cause of the disease in broilers, layers and breeders of different ages [23].

In our case for exclusion of viral load, for instance, the affection by very virulent IBDV was made PCR with further sequencing. As previously was described the histological lesions in bursa were specific for the development of Gumboro immunosuppressive disease. Moreover, the virus was detected in different age periods 21, 28, 35 and 42 days in the level of Ct 22.8; 25.8; 32.2 and 32.5, respectively. The amount of viruses was decreasing within age unlike the level of follicles bursa Fabricii depletion. However, after sequencing of the genetic material from 21–35 days broilers it was detected the very virulent strain of IBDV closely related to the United Kingdom 2019 variant with 95.7–96.1% of homogeneity. And the oldest broilers characterized by the presence of vaccine strain nevertheless severe bursa atrophy and depletion (Fig. 2). Regarding Vishwanatha et al., this strain of very virulent IBDV characterized by the presence of hypervariation region in VP1 segment and induce subclinical form of the disease with decreasing in broiler performance and chronic immunosuppression without massive hemorrhages [24]. That could be the predisposing factor for APEC coinfection that was isolated in 28 days broilers.

The presence of vaccine strain at the bursa Fabricii of 42 days birds was coincided with severe atrophy and APEC infection. That indicates the chronic stage of the disease which progress into bacterial systemic form nevertheless vaccine virus isolation.

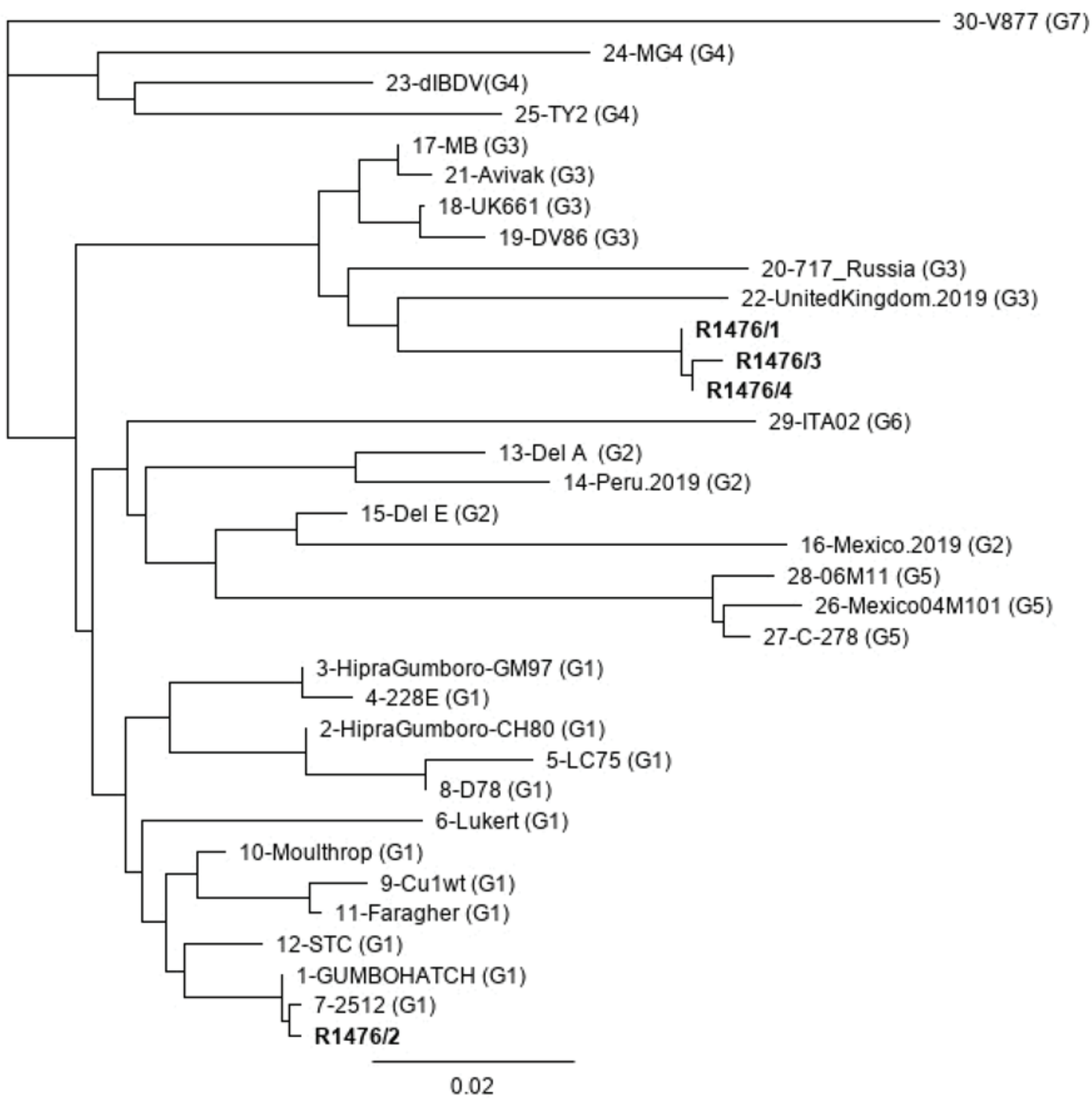


Fig. 2. Phylogenetic tree of field
and reference IBDV strains based on partial nucleotide sequences of the VP2 gene

Conclusion

Coinfection with avian pathogenic *E. coli* was observed mainly between 28–42 days of age, coinciding with peak immunosuppression. Pathogenic strains were isolated from internal organs (heart, liver, spleen), indicating systemic colibacillosis. All *E. coli* isolates showed resistance to beta-lactams (amoxicillin, amoxiclav), tetracyclines, and trimethoprim, highlighting the risk of multidrug-resistant strains. Resistance to quinolones (enrofloxacin, ciprofloxacin, flumequine) was already detected at 42 days, suggesting rapid cross-resistance development under uncontrolled use. However, the lowest resistance levels were found against colistin and florfenicol. The findings confirm that IBDV (United Kingdom 2019 strain) acts as a key predisposing factor for systemic APEC infection, and the combination of immunosuppression with antimicrobial resistance poses a major challenge for poultry health management. Finally, in 42 days there was no isolation of a very virulent IBDV strain, yet APEC strains were detected, indicating that bacterial infection can persist and progress even in the absence of active viral replication.

References

1. Li L, Kubasova T, Rychlik I, Hoerr FJ, Rautenschlein S. Infectious bursal disease virus infection leads to changes in the gut associated-lymphoid tissue and the microbiota composition. PLoS ONE. 2018;13(2):e0192066. doi: 10.1371/journal.pone.0192066
2. Dey S, Pathak DC, Ramamurthy N, Maity HK, Chellappa MM. Infectious bursal disease virus in chickens: prevalence, impact, and management strategies. Vet Med (Auckl). 2019;5(10):85-97. doi: 10.2147/VMRR.S185159
3. Orakpoghenor O, Oladele SB, Abdu PA. Infectious Bursal Disease: Transmission, Pathogenesis, Pathology and Control – An Overview. World's Poultry Science Journal. 2020;76(1):1-12. doi: 10.1080/00439339.2020.1716652
4. Hoerr FJ. Clinical aspects of immunosuppression in poultry. Avian Dis. 2010;54(1):2-15. doi: 10.1637/8909-043009-Review.1
5. Wang W, Wu J, Jiang N, Liang Q, Liu R, Fu Q, Fu G, Wei T, Wan C, Cheng L, et al. Advances in Infectious Bursal Disease Virus Vaccines – a Review. Microorganisms. 2025;13:2801. doi: 10.3390/microorganisms13122801
6. Srinivasan P, Rao GV, Titus VTK. Pathology of *Escherichia coli* and concurrent infection of IBDV with *Escherichia coli* in chicken. Indian Journal of Animal Sciences. 2002;72:967-70.
7. Nawaz S, Wang Z, Zhang Y, Jia Y, Jiang W, Chen Z, Yin H, Huang C, Han X. Avian pathogenic *Escherichia coli* (APEC): current insights and future challenges. Poultry Science. 2024;103(12):104359. doi: 10.1016/j.psj.2024.104359
8. Kamal O, Kneuper H, Cogan T, Woodward MJ. Avian Pathogenic *Escherichia coli*: Advances in Pathogenesis, Diagnosis, and Control. Veterinary Sciences. 2026;13(1),19. doi: 10.3390/vetsci13010019
9. 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;9296520:9. doi: 10.1155/2018/9296520
10. Cazaban C, Swart WBF, Rietema RMW, Wit JJD, Palya V, Gardin Y. Field Assessment of an Immune-Complex Infectious Bursal Disease Vaccine in Chicks Born to Non-Hyperimmunized Broiler Breeders. J Vet Sci Ani Husb. 2018;6(3):302. doi: 10.15744/2348-9790.6.302
11. Aguanta BN, Fuller AL, Milfort MC, Williams SM, Romdhane R, Aggrey S. Histologic Effects of Concurrent Heat Stress and Coccidial Infection on the Lymphoid Tissues of Broiler Chickens. Avian Diseases. 2018;62(4):345-50. doi: 10.1637/11907-052818-Reg.1
12. Sanger F, Nicklen S, Coulson AR. DNA sequencing with chain-terminating inhibitors. Proc Natl Acad Sci USA. 1977;74(12):5463-7. doi: 10.1073/pnas.74.12.5463
13. Nechypurenko O, Furtat I, Dreval D, Avdeeva L. Colibacillosis in broiler chicks and its etiological link to the biological characteristics of *Escherichia coli* isolates from the breeder flock. NaUKMA Research Papers. Biology and Ecology. 2025;8:45-54. doi: 10.18523/2617-4529.2025.8.45-54
14. 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
15. 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
16. Pastyria AS, Budzanivska IG, Polischuk VP. Characterization of vaccine and field IBDV strains in Ukraine for proper vaccine selection for disease prevention. Biopolymers and Cell. 2018;34(1):24-31. doi: 10.7124/bc.00096D
17. Nagy N, Busalt F, Halasy V, Kohn M, Schmieder S, Fejszak N, Kaspers B, Härtle S. In and Out of the Bursa–The Role of CXCR4 in Chicken B Cell Development. Front Immunol. 2020;11:1468. doi: 10.3389/fimmu.2020.01468
18. 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
19. 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
20. 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
21. 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
22. Spencer A, Jurasitritthikun S, Subedi D. Characterization of antibiotic resistance and virulence in avian pathogenic *Escherichia coli* (APEC) from Australia. Sci Rep. 2024;14(1):22176. doi: 10.1038/s41598-024-73043-3
23. Kathayat D, Lokesh D, Ranjan 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
24. Vishwanatha S, Shridevi R, Bhat JA, Desai GS, Reddy PS, Tirumurugaan KG. Molecular characterization of very virulent infectious bursal disease virus (vvIBDV) from southern India: Evidence of genetic reassortment. Infect Genet Evol. 2018;62:208-215. doi: 10.1016/j.meegid.2018.04.018

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 та вірусології ім. Д. К. Заболотного Національної академії наук України, Київ, Україна

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

КОІНФЕКЦІЯ ПАТОГЕННИМИ ШТАМАМИ *ESCHERICHIA COLI* ТА ВІРУСОМ ХВОРОБИ ГАМБОРО В БРОЙЛЕРІВ

Мета. Дослідити роль патогенної для птахів *E. coli* в патологічному процесі під час коінфекції вірусом бурсальної хвороби в бройлерів. **Методи.** Дослідження проводили на одній з птахофабрик із закритим циклом вирощування протягом 42 днів. Для оцінювання патологічних змін проводили розтин загиблих курчат віком 21, 28, 35 і 42 дні та визначали ступінь ураження бурси на підставі результатів гістологічного дослідження. Ідентифікацію бактерій здійснювали із застосуванням API 20E тест-системи, з оцінюванням гемолізу. Чутливість виділених ізолятів *E. coli* до антибіотиків визначали за методом Кірбі – Бауера. Виділення та ідентифікацію вірусу хвороби Гамборо здійснювали за допомогою ЗТ-ПЛР та подальшого секвенування утворених фрагментів. **Результати**. Атрофія бурси спостерігалася з 21-го дня з подальшим зниженням рівня з 60 до 90 % на завершальному етапі дослідження. Крім того, штами APEC було виділено у птахів на 28–42-й день, що корелює з хронічною імуносупресією. Усі досліджені ізоляти *E. coli* виявилися резистентними до бета-лактамів (амоксицилін, амоксиклав), тетрациклінів і триметоприму, що підкреслює ризик розвитку штамів із множинною лікарською стійкістю. Стійкість до хінолонів (енрофлоксацин, ципрофлоксацин, флумеквін) було виявлено вже через 42 дні, що свідчить про швидкий розвиток перехресної резистентності за умови неконтрольованого застосування. Втім, найнижчі рівні стійкості спостерігали щодо колістину та флорфеніколу. Штами IBDV було виявлено в бурсі 21, 28 та 35 бройлерів з рівнем Ct 22,8; 25,8 та 32,2 бала відповідно, що свідчить про зниження вірусного навантаження. За результатами визначення часткових нуклеотидних послідовностей гена VP2 збудник був ідентифікований як дуже вірулентний вірус, а саме штам United Kingdom 2019. Однак аналіз зразків від 42-денних бройлерів засвідчив, що нуклеотидна послідовність належить до вакцинного штаму. **Висновки.** Коінфекція APEC та IBDV у бройлерних курчат посилює імуносупресію, створюючи умови для вторинних бактеріальних інфекцій. Схильність до колібактеріозу корелює зі стадією виснаження бурсальної сумки та може зберігатися навіть під час реплікації вакцинного штаму IBDV після первинного інфікування.

Ключові слова: вірус інфекційної бурсальної хвороби (IBDV), хвороба Гамборо, патогенна кишкова паличка птахів (APEC), коінфекція, колібактеріоз, антимікробна резистентність.

Submitted 14.04.2026

Accepted 18.04.2026

Published 28.05.2026

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

Oleksii Nechypurenko – Candidate of Biological Sciences, Senior Lecturer at the Department of Biology of the 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

Iryna Furtat – Candidate of Biological Sciences, Associate Professor at the Biology Department of the 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

Denis Dreval – 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

Liliya Avdeeva – 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