



Physiological, Histopathological, and Molecular Characterizations of *Escherichia coli* Infection in Commercial Laying Hens in Basrah Governorate, Iraq

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ABSTRACT

Colibacillosis represents one of the most important bacterial diseases affecting poultry production, particularly in commercial laying hens, where it leads to marked economic losses. The present study aimed to evaluate the physiological, histopathological, and molecular characteristics of *Escherichia coli* (*E. coli*) infection in commercial laying hens in Basrah Governorate, Iraq. A total of 250 laying hens aged 35 weeks with clinical signs and postmortem lesions of colibacillosis were examined. Bacterial isolation was performed using conventional culture methods, followed by molecular confirmation using PCR targeting the *16S rRNA* gene. Blood samples were collected for hematological analysis, and tissue samples from the liver and oviduct were processed for histopathological examination. The results showed a clear decrease in red blood cell (RBC) count, white blood cell (WBC) count, hemoglobin (Hb), and hematocrit (Hct) values in infected hens compared with healthy controls. In contrast, mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) were significantly increased in infected hens compared with healthy controls. Histopathological findings showed severe pathological alterations in the liver and oviduct, including vascular congestion, inflammatory cell infiltration, vacuolation, hemorrhage, and necrosis. Molecular analysis confirmed the presence of *E. coli* through successful amplification of the *16S rRNA* gene. The findings of the current study suggest that *E. coli* infection induces marked physiological and tissue-level alterations in laying hens, which may adversely affect their health and productivity, highlighting the need for effective control measures on poultry farms.

Keywords: Colibacillosis, Hematological parameters, Liver, Oviduct, PCR, *16S rRNA*

INTRODUCTION

Colibacillosis is a localized or systemic infection caused by the avian pathogen *Escherichia coli* (*E. coli*) and is one of the most prevalent and economically important bacterial diseases in poultry worldwide (Guabiraba and Schouler, 2020). *Escherichia coli* infection can present in several clinical descriptions, such as acute fatal septicemia, subacute pericarditis, airsacculitis, salpingitis, peritonitis, and cellulitis (Nolan et al., 2020). In laying hens, colibacillosis is often seen as salpingitis and peritonitis, whereas with young chickens, it is often linked with

omphalitis (yolk sac infection) and large head syndrome (Nolan et al., 2015).

Escherichia coli is a facultative anaerobic, rod-shaped, Gram-negative bacterium of the family *Enterobacteriaceae* with an average length of 2-3 µm and a diameter of about 0.5 µm (Nolan et al., 2020). In addition to a clinical manifestation, colibacillosis has a clear economic implication on the poultry sector in terms of mortality rates and decreased productivity in laying cages that can even extend to 70 percent in severely affected flocks (Costantini et al., 2009). Additional

economic losses are associated with reductions in egg production, hatching rates, and meat condemnation during treatment and preventive measures during processing, which are rather expensive (Blyton et al., 2015).

Colibacillosis occurs under the influence of several predisposing factors, such as the presence of unfavorable environmental conditions, co-morbid respiratory infections, immunosuppressive agents, or metabolic stress in poultry, particularly in laying hens (Guabiraba and Schouler, 2020). The disease control strategies usually depend on management and environmental conditions control, including ventilation, chlorination of drinking water, and humidity, combined with antibiotic treatment or vaccination courses (Kamal et al., 2026). Nevertheless, intensive antibiotic use in poultry production, as a disease preventive measure and a growth promoter, has contributed to the development and transmission of multidrug-resistant *E. coli* strains, posing serious health concerns for both animals and humans (Faife et al., 2020).

According to pathogenic potential, *E. coli* isolates can be classified into non-pathogenic intestinal, pathogenic intestinal, and pathogenic extraintestinal groups (Pitout, 2012). Pathogenic strains are also categorized into those causing diarrhea and those responsible for extraintestinal infections in poultry, which are collectively referred to as avian colibacillosis (Tonini da Rocha et al., 2021). Transmission to humans can occur through direct contact with infected chickens or via the consumption of contaminated poultry products, highlighting the zoonotic relevance of this pathogen (Kathayat et al., 2021).

A common clinical manifestation in laying hens is *E. coli* peritonitis syndrome (EPS), which is associated with subacute mortality and significant production losses (Landman and Van Eck, 2015). The avian *E. coli* strains that are pathogenic are associated with various virulence factors, such as toxin production, biofilm formation, hemolysin activity, and increased attachment to host tissues, which promote colonization and systemic dissemination (Guabiraba and Schouler, 2020). Extraintestinal lesions such as pericarditis, airsacculitis, fallopian tubes, synovitis, yolk sac infection, and osteomyelitis are commonly reported in the affected chickens (Dziva and Stevens, 2008). However, despite the recognized importance of avian colibacillosis, limited studies have comprehensively investigated the combined physiological, histopathological, and molecular characteristics of *E. coli* infection in commercial laying hens under local field conditions in Iraq. Therefore, the present study was designed to evaluate the physiological alterations, histopathological lesions, and molecular

identification of *E. coli* isolates in commercial laying hens in Basrah Governorate, Iraq.

MATERIALS AND METHODS

Ethical approval

All experimental procedures involving animals were conducted in accordance with the institutional guidelines for the care and use of animals and were approved by the Ethical Committee of the College of Veterinary Medicine, University of Basrah, Iraq.

Sample collection and clinical examination

Samples were collected from five commercial laying hen farms located in Basrah Governorate, Iraq, based on clinical signs and postmortem lesions suggestive of *E. coli* infection. A total of 250 commercial laying hens were included in the current study. The chickens were divided into two groups, consisting of 125 infected hens and 125 healthy control hens, based on clinical signs and postmortem findings. Clinical signs observed in infected hens included conjunctivitis and rales, while postmortem findings revealed enteritis and salpingitis. Approximately 2-3 mL of blood was collected from the wing vein of both healthy and diseased chickens using tubes containing anticoagulants for hematological analysis.

Isolation of *Escherichia coli* and identification

Liver and oviduct tissue samples were inoculated into nutrient broth and incubated at 37°C for 24 hours. Subsequently, subculturing was performed on MacConkey agar and incubated at 37°C for 18-24 hours. Pink lactose-fermenting colonies were further sub-cultured on eosin methylene blue (EMB) agar. Colonies showing a characteristic metallic sheen were selected and inoculated into nutrient broth for further identification (Markey et al., 2013).

Hematological examination

Hematological parameters, such as red blood cell (RBC) count, white blood cell (WBC) count, hemoglobin (Hb), and hematocrit (Hct), were determined using standard hematological procedures as described by Campbell and Dein (1984).

Molecular detection and identification of bacterial isolates

Molecular identification of bacterial isolates was performed by amplification of the 16S ribosomal RNA (16S rRNA) gene using a polymerase chain reaction (PCR) assay. Genomic DNA was extracted using the Promega

Genomic DNA Purification Kit (Promega, USA; Catalog No. M7432) according to the manufacturer's instructions.

PCR amplification was carried out using universal bacterial primers targeting the *16S rRNA* gene, as listed in Table 1. The reaction mixture (50 µL) consisted of 25 µL of master mix, 2 µL of template DNA, 2 µL of each primer, and 19 µL of nuclease-free water. Thermal cycling conditions included an initial denaturation at 96°C for 5 minutes, followed by 35 cycles of denaturation at 95°C for 30 seconds, annealing at 55°C for 30 seconds, and extension at 72°C for 60 seconds, with a final extension at 72°C for 10 minutes. A positive control (*E. coli* ATCC 25922) was included in the PCR assay to ensure the validity of the amplification results. PCR products were separated using a 1.5% agarose gel stained with ethidium bromide, run at 100 V for approximately 45 minutes, and visualized under ultraviolet light using a DNA ladder as a molecular size marker. The expected amplicon size was approximately 1500 bp.

Table 1. Primers used for *16S rRNA* amplification

Primer	Sequence (5'–3')	GC (%)	Tm (°C)
Forward	AGAGTTTGATCCTGGCTCAG	50.0	54.3
Reverse	GGTTAACCTTGTACGACTT	42.1	49.4

Tm: Melting temperature (°C)

Sequence analysis

Positive PCR products were partially sequenced for the *16S rRNA* gene. The obtained nucleotide sequences were cleaned and assembled to generate consensus sequences. Sequence alignment was performed using MEGA 6 software, and similarity analysis was conducted using BLASTn against the NCBI GenBank database to determine the genetic relatedness of the obtained isolates to reference *E. coli* sequences retrieved from GenBank.

Statistical analysis

All data were expressed as mean ± standard error (SE). Statistical analysis was performed using SPSS software (Version 25.0). Differences between the infected and control groups were analyzed using Student's t-test. The value of $p < 0.05$ was considered statistically significant.

RESULTS

Hematological parameters

Hematological analysis showed marked changes in infected laying hens compared with the healthy control

group ($p < 0.05$). Red blood cell (RBC) count, white blood cell (WBC) count, hemoglobin (Hb), and hematocrit (Hct) values were significantly decreased in infected hens compared with the healthy control group ($p < 0.05$). In contrast, mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH) were significantly increased in infected hens compared with the healthy control group ($p < 0.05$). Detailed hematological parameters are presented in Table 2.

Histopathological changes in the liver and oviduct

Histopathological examination of infected laying hens revealed marked alterations in both liver and oviduct tissues compared with healthy controls. In the liver, severe vascular congestion of central and portal veins was observed, accompanied by inflammatory cell infiltration in hepatic parenchyma and periportal areas (Figure 1A-C). Hepatocellular vacuolation, hemorrhage, and focal necrosis were also evident, indicating degenerative and circulatory disturbances (Figure 1D-F).

In the oviduct, marked pathological changes were observed in different segments, including the magnum, isthmus, and uterus. Lesions included epithelial degeneration, vascular congestion, hemorrhage, inflammatory cell infiltration, tissue vacuolation, and necrosis (Figure 2A-F). In contrast, no pathological alterations were observed in the corresponding tissues of healthy control chickens (Figure 2G-H).

Molecular detection of isolates

Molecular identification of *E. coli* was confirmed by PCR amplification of the *16S rRNA* gene. Agarose gel electrophoresis revealed clear and distinct bands corresponding to the expected product size in all tested isolates (lanes 1-6), confirming positive amplification (Figure 3).

Sequence analysis of positive isolates

Partial sequencing of the *16S rRNA* gene of positive *E. coli* isolates produced high-quality nucleotide sequences. Comparative analysis revealed a high degree of similarity between the obtained sequences and reference strains available in public databases.

A representative isolate showed 96% sequence identity with *E. coli* strain CP14 (Figure 4). Additional comparisons demonstrated 97-98% similarity with strains previously reported from different geographical regions, including Thailand, Colombia, and the Netherlands, indicating close genetic relatedness.

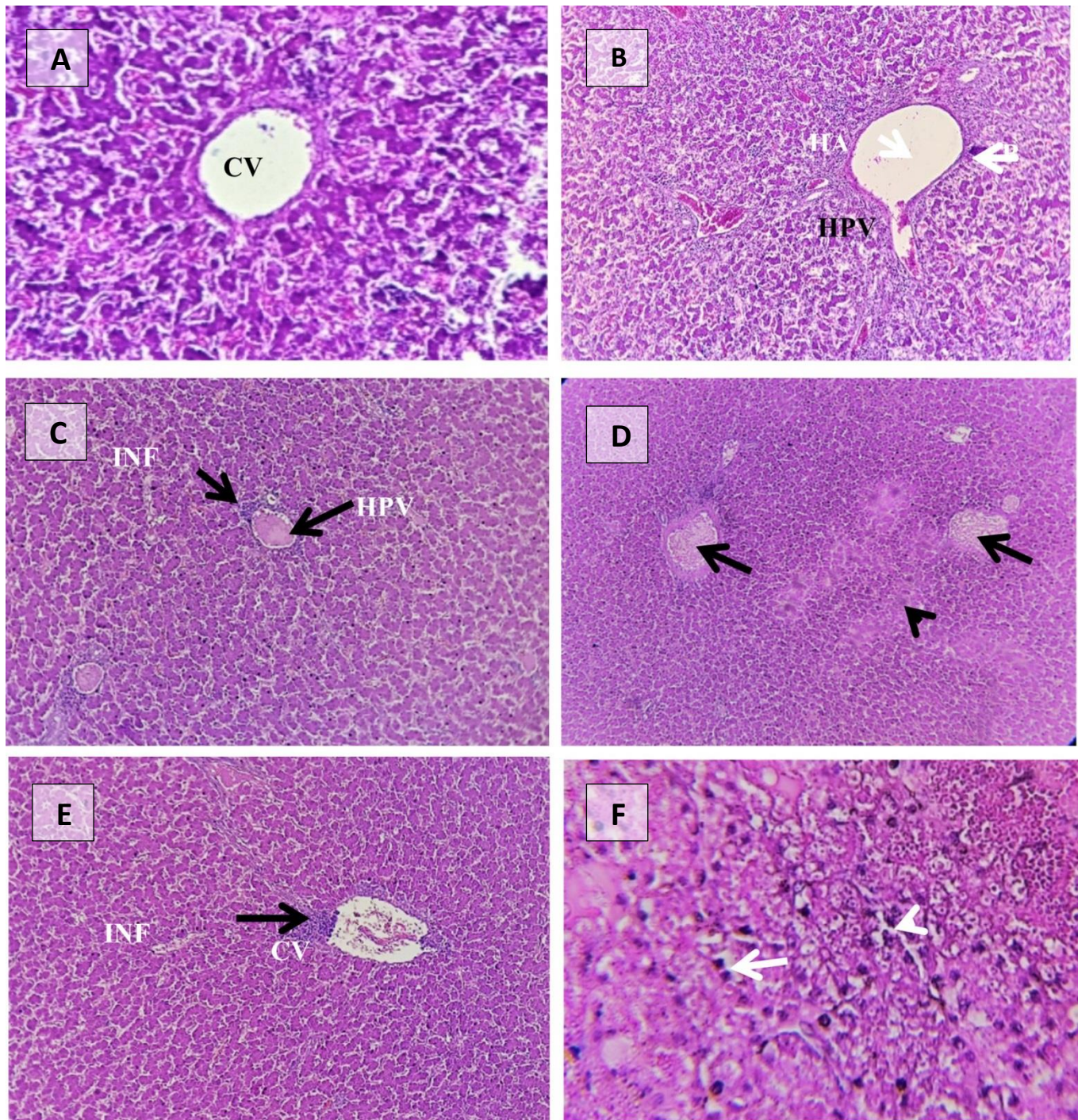
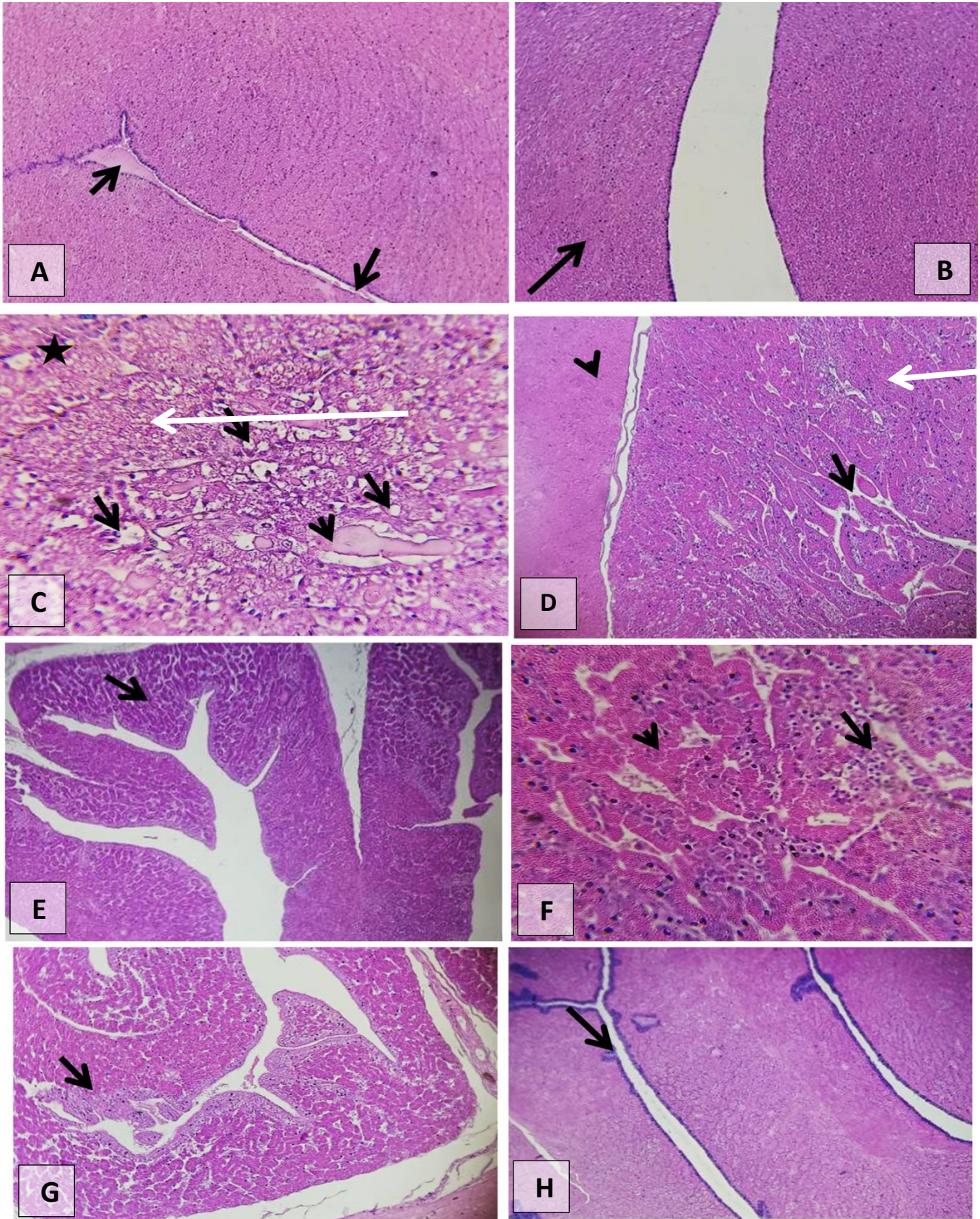


Figure 1. Histopathological sections of the liver in 35-week-old laying hens. Healthy control showing normal hepatic architecture and central vein. **A).** Healthy control showing bile duct, hepatic artery, and portal vein (white arrow; **B).** *Escherichia coli* infected hen showing inflammatory cell infiltration near the hepatic portal vein (black arrows), HPV: Hepatic portal vein, INF: Inflammatory cell infiltration, **C).** Infected hen showing congestion of the portal vein and hepatic sinuses (black arrows; **D).** Infected hen showing hepatocellular vacuolation, hemorrhage, and focal necrosis (black arrow) of the central vein; CV: Central vein, INF: Inflammatory cell infiltration, **E).** Infected hen showing congestion of the central vein and hepatic sinusoids (white arrows; **F).** Hematoxylin and eosin (H&E) stain, $\times 100$ (**B-E**), $\times 400$ (**A, F**).



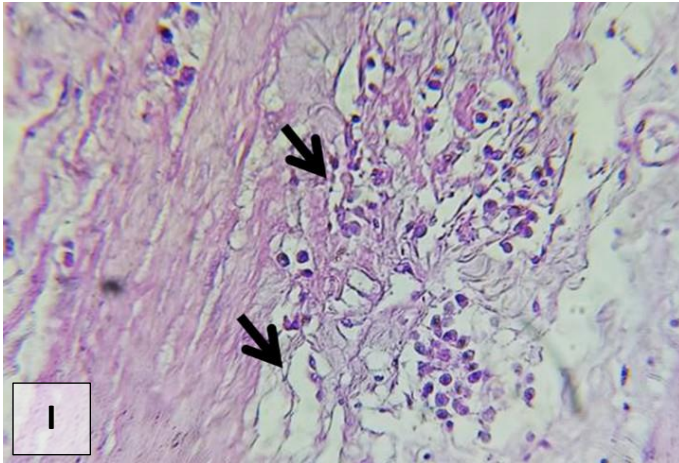


Figure 2. Histopathological sections of the oviduct in 35-week-old laying hens. An infected hen showing marked hemorrhage (black arrows) in the magnum. **A)** Healthy control showing normal magnum and secretory glands (black arrow; **B)** Infected hen showing epithelial vacuolation (black arrows), hemorrhage, and vascular congestion in the magnum; **C)** Infected hen showing necrosis and congestion (black arrow) in the magnum; **D)** Healthy control showing normal isthmus and secretory glands (black arrow; **E)** Infected hen showing necrosis (black arrows), inflammatory cell infiltration, and vascular congestion in the isthmus; **F)** Infected hen showing epithelial hyperplasia (black arrow) and hemorrhage in the isthmus; **G)** Healthy control showing normal uterine (shell gland; black arrow) structure; **H)** Infected hen showing marked hemorrhage, vacuolation, and inflammatory cell infiltration (black arrows) in the uterine (shell gland) serosal layer; **I).** Hematoxylin and eosin (H&E) stain, $\times 100$ (A-E, G-I), $\times 400$ (F).

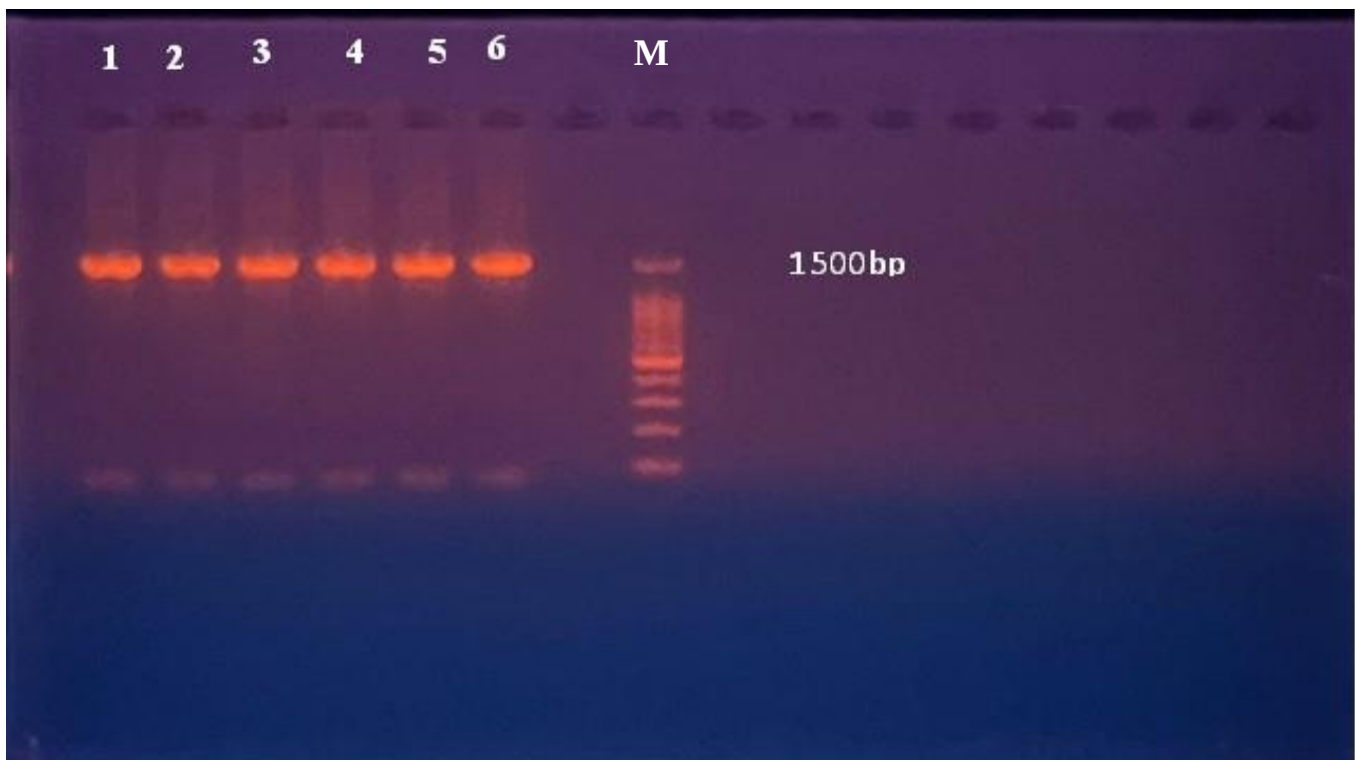


Figure 3. Agarose gel electrophoresis of 16S rRNA gene amplification of *Escherichia coli* isolates (lanes 1-6). **M:** 1500 bp DNA ladder. PCR products were separated on a 1.5% agarose gel and visualized under ultraviolet illumination.

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GGCGGTGTGTACAAGGCCCGGAACGTATTCACCGTGGCATTCTGATCCACGATTACTAG
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CGATTCCGACTTCATGGAGTCGAGTTGCAGACTCCAATCCGGACTACGACGCACTTTATG
.....
AGGTCCGCTTGCTCTCGCGAGGTCGCTTCTCTTTGTATGCGCCATTGTAGCACGTGTGTA
.....
GCCCTGGTCGTAAGGGCCATGATGACTTGACGTCATCCCCACCTTCCTCCAGTTTATCAC
.....
TGGCAGTCTCCTTTGAGTTCCCGGCCGGACCGCTGGCAACAAAGGATAAGGGTTGCGCTC
.....
GTTGCGGGACTTAACCCAACATTTCAACAACAGAGCTGACGACAGCCATGCAGCACCTGT
.....
CTCACAGTTCCC GAAGGCACCAATCCATCTCTGGAAAGTTCGTGGATGTCAAGACCAGG
.....
TAAGGTTCTTCGCGTTGCATCGAATTAAACCACATGCTCCACCGCTTGTGCGGGCCCCG
.....
TCAATTCATTTGAGTTTTAACCTTGC GGCCGTA CTCCCAGGCGGTGAC TTAACGCGTT
.....
AGCTCCGGAAGCCACGCCTCAAGGGCACAACTCCAAGTCGACATCGTTTACGGCGTGGA
.....
CTACCAGGGTATCTAATCCTGTTTGCTCCCCACGCTTTCGCACCTGAGCGTCAGTCTTCG
.....
TCCAGGGGGCCGCCTTCGCCACCGGTATTCCTCCAGATCTCTACGCATTTACCCGCTACA
.....
CCTGGAATTCTACCCCCCTCTACGAGACTCAAGCTTGCCAGTATCAGATGCAGTTCCAG
.....
GTTGAGCCCGGGGATTTACATCTGACTTAACAAACGCGCGCGTGCGCTTACGCCAG
.....
TAATTCCAATTAACGCTTGCA CCTCCGTATTACCGCGGCTGCTGGCACGGAATTAGCCG
.....
GTGCTTCTTCTGCGGGTAACGTCAATGAGCAAAGGT-TTAACTTTACTCCCT-CCTCCCC
.....
GCTAAAA-TACTTTACAACCCGAAAGGC-TTCTTCATACACGCGGCAGGGCTGCTTAAGG
.....
..G..G.....C..-.....A.C...
TTGCGCCCA-TGTGGAGAATTCCCCCTGCTGCCTCC-GCA-GAAGAT-GGAAAGAGGCT
.....
C.....T...C.A.....C...A...-
..C..G.C.G..T. AAATT-CCCgggggggggggggAcccc-
ccAAACAGAAAGGGGTGGTGC CG--GG-GGA
TC..T.....TT..T...ACT...A..T...CCA..T...
GCGTTccccccc-cAA-CTAATCCA TTTGGG--CATCCAATGGAGAAGG
.....
C.....T..A.....-.....GC.....A..A...

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Figure 4. Sequence alignment of the partial 16S rRNA gene of a representative *Escherichia coli* isolate from the present study with *Escherichia coli* strain CP14, showing 96% sequence similarity.

DISCUSSION

The present study provided a detailed assessment of the physiological, histopathological, and molecular alterations associated with *E. coli* infection in commercial laying hens under field conditions. The combined physiological, histopathological, and molecular findings indicated that colibacillosis is a systemic disease that markedly affects both hematological parameters and tissue integrity.

The amplification and partial sequencing of the 16S *rRNA* gene showed that the isolates identified in the current study showed high genetic similarity with respect to reference *E. coli* strains deposited in the public repositories, including isolates reported in Thailand, Colombia, and the Netherlands, with a sequence similarity value of between 96% and 98% (Nolan et al., 2020). The close genetic relationship between poultry-related and human-related strains suggests the possibility of epidemiological overlap of the animal, environmental, and human reservoirs, which points to the possible zoonotic implications of *E. coli* strains apparent in commercial layer flocks, especially those in eggs that are meant to be consumed by humans. The high sequence similarity observed between the isolates in the current study and previously reported strains from Thailand, Colombia, and the Netherlands highlights the potential epidemiological linkage and zoonotic significance of *E. coli*. The results of the present study suggested that poultry may serve as a reservoir for strains with the ability to cross species barriers, raising concerns regarding public health and food safety (Kathayat et al., 2021).

The present study confirmed colibacillosis based on clinical manifestations and gross lesions, such as salpingitis and enteritis, in addition to conventional PCR of the 16S *rRNA* gene. A clear reduction in hematological parameters was observed, including RBC, WBC, hemoglobin, and hematocrit of the infected hens relative to the healthy controls. The findings of the current study are consistent with previous reports indicating that systemic *E. coli* infection may induce inflammatory and hematological disturbances in infected chickens (Barnes et al., 2008; Nolan et al., 2013). Hematopoietic stem cells differentiate to the final stages of blood cells in the primary bone marrow site, and recovery of *E. coli* in bone marrow after systemic infection has been reported, which could, in part, explain the observed leukopenia or anemia (Landman et al., 2013). The observed reduction in RBC count, hemoglobin, and hematocrit values in infected hens may be due to impaired hematopoiesis, which is likely linked to

systemic bacterial infection and inflammatory responses (Peng et al., 2021). Additionally, the increase in MCV and MCH values indicates macrocytic alterations, which may reflect compensatory mechanisms or disruption in erythrocyte maturation. These hematological disturbances are consistent with the pathological impact of endotoxins produced by *E. coli*, which are known to induce oxidative stress and damage hematopoietic tissues (Surai et al., 2019).

In addition to the hematological changes, *E. coli* infection leads to inflammatory and degenerative processes in various organs due to the release of endotoxins and vascular damage (Kamal et al., 2026). The effects of endotoxins have been known to result in hepatic inflammation, hepatocellular necrosis, and circulatory disturbances in poultry, and renal inflammation and tubular degeneration in infected chickens have also been reported (Dutta et al., 2013; Akanbi et al., 2022). Erythropoietin production by the kidney plays a critical role in erythrocyte regulation; therefore, renal damage may contribute to the decrease in erythrocyte indices observed in infected hens (Landman et al., 2013). In the current study, the histopathological changes in the liver and oviduct are likely related to the systemic dissemination of *E. coli* and the subsequent inflammatory response. Vascular congestion and hemorrhage are indicative of circulatory disturbances, while cellular infiltration and necrosis show tissue damage mediated by bacterial toxins and host immune responses (Kamal et al., 2026). Thus, findings of the present study support the view that colibacillosis involves multi-organ pathology resulting from septicemic spread.

Bacterial invasion induces an acute inflammatory reaction that is also linked to the overproduction of reactive oxygen and nitrogen species (ROS and RNS), therefore leading to oxidative stress and the ensuing damage to cells (Surai et al., 2019). The imbalance between ROS and RNS production and the antioxidant defense system leads to lipid peroxidation, protein oxidation, and DNA damage (Pizzino et al., 2017; Surai et al., 2019). Moreover, the action of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF- α) and interleukin-1 beta (IL-1 β), can stimulate the hypothalamic-pituitary-adrenal axis and raise the level of plasma corticosterone, thus worsening stress and inflammatory reactions in infected chickens (Abbas et al., 2020).

The histopathological examination of the current study demonstrated marked pathological alterations in the

liver and oviduct, including vascular congestion, hemorrhage, inflammatory cell invasion, vacuolation, and focal necrosis. These findings coincide with the ones reported by [Akanbi et al. \(2022\)](#), who identified hepatic inflammation, liver necrosis, and oviductal rupture in laying hens infected with *E. coli*. Systemic spread of bacteria via the blood vessels has been put forward as a major pathological process in the multi-organ involvement in colibacillosis ([Kabir, 2010](#)). The same findings, such as congestion in the central vein, sinusoidal dilation, and cellular periportal infiltration in the liver, have been observed in the infected broiler chicken ([Akanbi et al., 2022](#)).

Avian pathogenic *Escherichia coli* (APEC) infection in laying hens leads to marked reproductive tract infections. The fallopian tube and infundibulum inflammation affects the normal egg capture and transport, resulting in peritonitis and egg formation impairment ([Nolan et al., 2020](#)). Infection of such structures during the pre-oviposition stage has been linked to low egg production and impaired reproduction ([Mehaisen et al., 2016](#)). The results of the current study indicated that the damage to reproductive tissues caused by *E. coli* not only influences the flock productivity but also enhances the risk of bacterial eggs spreading to human food chain.

Overall, hematological, histopathological, and molecular results of the present study support the idea that *E. coli* infection in commercial laying hens is a systemic disease with clinical implications to the chicken, its production performance, and human health. Overall, the findings of the present study emphasize the multifactorial impact of *E. coli* infection on poultry health, integrating hematological dysfunction, tissue damage, and genetic relatedness of circulating strains. The results of the present study highlight the importance of implementing effective control strategies, including improved biosecurity measures, rational use of antibiotics, and the development of targeted vaccination programs to mitigate the impact of colibacillosis in commercial poultry production.

CONCLUSION

Escherichia coli infection in commercial laying hens was associated with significant hematological alterations and severe histopathological lesions, particularly in the liver and oviduct. The observed reductions in RBC, WBC, hemoglobin, and hematocrit, along with increased MCV and MCH, suggest impaired hematopoietic function and systemic infection. Histopathological findings confirmed

extensive tissue damage characterized by vascular congestion, inflammation, and necrosis. These findings emphasize the detrimental impact of colibacillosis on poultry health and productivity and highlight the importance of implementing effective control and prevention strategies in commercial poultry systems.

A limitation of this study is the reliance on *16S rRNA* gene analysis without further molecular characterization of virulence-associated genes. Future studies are recommended to investigate specific virulence factors and antimicrobial resistance profiles of *E. coli* isolates to further understand their pathogenic potential.

DECLARATIONS

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Authors' contributions

Salma Saeed Abbas, Rajaa Abd Alzahra Ali, and Muhammadtaher Abdulrazaq Abdulrasol conceived and designed the study. Alaa Ismail Saood, Waleed Majeed Seger, and Harith Abdulla Najem carried out the field work and data collection. Isam Azeez Khaleefah and Muhammadtaher Abdulrazaq Abdulrasol performed formal analysis and molecular investigations. Salma Saeed Abbas, Isam Azeez Khaleefah, and Muhammadtaher Abdulrazaq Abdulrasol developed the methodology. Muhammadtaher Abdulrazaq Abdulrasol drafted the manuscript. Salma Saeed Abbas, Rajaa Abd Alzahra Ali, Isam Azeez Khaleefah, and Muhammadtaher Abdulrazaq Abdulrasol edited and approved the final manuscript. All authors have read and approved the final version of the manuscript before publication in the present journal.

Availability of the data and materials

The datasets generated and/or analyzed during the current study are included in this published article and are available from the corresponding author upon reasonable request.

Competing interests

The authors declare that they have no competing interests.

Ethical considerations

Ethical issues, including plagiarism, consent to publish, misconduct, data fabrication and/or falsification, double publication and/or submission, and redundancy, have been checked by all the authors. The authors used AI-assisted tools (ChatGPT, OpenAI) for language editing of the manuscript. The authors accept all responsibility for the originality of scientific content, data interpretation, writing, and conclusions in the present study.

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