



Prevalence and Pathomorphology of Endoparasites in Indian Peafowl (*Pavo cristatus*) under Different Housing Systems in Odesa Region, Ukraine

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ABSTRACT

Parasitic infections represent a major health concern in ornamental and exotic poultry, adversely affecting the health, productivity, and survival of Indian peafowl (*Pavo cristatus*). The present study aimed to determine the prevalence and species composition of endoparasites in Indian peafowl maintained under free-range and aviary housing systems, and to characterize the gross and pathomorphological lesions associated with histomonosis and cestodosis. The present study was conducted in 2020-2025 on private farms and bioparks in the Odesa Region, Ukraine, and included 90 Indian peafowl, 44 males and 46 females, aged 1-2 years. Fecal samples were collected in all seasons, with 45 peafowl kept under free-range housing and 45 under cage-aviary housing. Fecal samples were analyzed by direct smear, flotation, and the McMaster method. The pathomorphological manifestations of histomonosis were examined through a retrospective analysis of 17 cases in Indian peafowl aged 6 weeks to 16 months. Meanwhile, gross lesions of cestodosis were assessed during postmortem examinations of three naturally infected peafowl. The overall prevalence of endoparasitic infections in Indian peafowl was 51.1% under free-range housing and 66.7% under cage-aviary housing. Under free-range housing, Indian peafowl were most frequently infected with *Histomonas meleagridis* (*H. meleagridis*; 13.3%), *Eimeria* spp. (11.1%), *Heterakis* spp. (8.9%), *Raillietina* spp. (6.7%), *Ascaridia galli* (4.4%), *Davainea proglottina* (4.4%), and *Choanotaenia infundibulum* (2.2%). In cage-aviary housing, the most common parasites were *H. meleagridis* (24.4%), *Eimeria* spp. (15.6%), *Heterakis* spp. (13.3%), *Ascaridia galli* (6.7%), and *Davainea proglottina* (2.2%). A mixed infection of *H. meleagridis* and *Ascaridia galli* was found exclusively in cage-aviary housing (4.4%). No apparent sex-related differences in susceptibility to protozoan infections were observed. The intensity of protozoan and nematode infections was generally higher in Indian peafowl housed in cage-aviaries than in those under free-range housing. Histomonosis was characterized by fibrinonecrotic typhlitis and characteristic necrotizing hepatitis, whereas cestodosis was associated with catarrhal-desquamative enteritis and severe destructive intestinal lesions. The current results indicated that the housing system affected the prevalence and intensity of parasitic infections, as well as the structure of the parasitic community, in Indian peafowl.

Keywords: Cestodosis, Hepatitis, Histomonosis, Indian peafowl, Protozoan infection

INTRODUCTION

The Indian peafowl (*Pavo cristatus* Linnaeus, 1758) is a member of the family *Phasianidae* in the order *Galliformes* (Kushwaha and Kumar, 2016). The natural distribution of the Indian peafowl spans South Asia, including India, Sri Lanka, Myanmar, and parts of the Malay Peninsula. Owing to its remarkable ornamental appeal, the Indian peafowl has been extensively introduced and is frequently maintained in zoological gardens, private breeding facilities, and park collections worldwide (Rajashekara et al., 2019). Captive housing conditions, including high stocking density, restricted movement, and limited opportunities for natural foraging, may increase the risk of gastrointestinal parasite transmission in Indian peafowl (Rodrigues et al., 2020). Furthermore, even with proper veterinary and sanitary management in zoological collections, poultry can be exposed to infectious parasite stages through contaminated feed, water, and soil, as well as the presence of intermediate hosts within aviaries (Dutta et al., 2013; Ombugadu et al., 2019).

Despite their relatively high tolerance to adverse environmental conditions, Indian peafowl remain vulnerable to a broad spectrum of parasitic diseases, among which gastrointestinal endoparasites are of primary importance (Kathiravan et al., 2017). Helminths and protozoa are frequently reported in the gastrointestinal tracts of poultry (Ruhnke et al., 2021). Among helminths, the classes *Nematoda*, *Cestoda*, and *Trematoda* are most commonly reported in domestic and wild poultry. Nematodes are considered the most prevalent and pathogenic group due to their widespread presence and

significant negative impact on host health (McJunkin et al., 2003; Udoh et al., 2014). Unlike nematodes, the diversity of cestode species that infect poultry is relatively limited (Abd El-Ghany, 2022). Nevertheless, several cestode species are of considerable epizootiological importance and may cause significant financial losses (Bogach et al., 2020).

Histomonosis is a major protozoan disease in poultry that can lead to significant economic losses and sometimes result in high-mortality outbreaks (Liebhart et al., 2017). Nematodes and cestodes infect the gastrointestinal tract of poultry, leading to damage to the intestinal mucosa, enteritis, and reduced nutrient absorption. These effects cause growth impairment, reduced productivity, higher risk of other infections, emaciation, and, in cases of severe infestation, mortality in young poultry. Poultry reared on deep litter or in backyard systems show the highest rates of helminth infections compared to those kept in cage housing systems (Basit et al., 2014; Abd El-Ghany, 2022).

Under captive conditions, peafowls exhibit increased susceptibility to endoparasitic infections, largely owing to inappropriate anthelmintic use and inadequate or delayed assessment of health status (Pradeep et al., 2017). In peafowl, endoparasitic infections frequently remain subclinical; however, heavy parasite burdens may result in clinical disease and even mortality (Lemos et al., 2024).

Parasites have multiple adverse effects on the host, including competing for nutrients and vitamins, disrupting digestion and absorption, mechanically damaging the intestinal tract, and releasing toxic metabolites. These effects result in progressive weight loss and worsening physiological health in infected poultry (Kruchynenko, 2021). Parasitic infections are prevalent among wild poultry; however, scientific investigations have predominantly focused on infections that threaten livestock production or public health (Daszak et al., 2000). Therefore, investigating the prevalence and species composition of endoparasites in peafowl maintained under free-range and aviary housing systems is important for improving strategies to prevent and control parasitic infections in ornamental and wild poultry. The present study aimed to investigate the prevalence and species diversity of endoparasites in Indian peafowl and to characterize the gross and pathomorphological lesions caused by histomonosis and cestodosis.

MATERIALS AND METHODS

Ethical approval

The present study was conducted in accordance with the current legislation of Ukraine (Article 26 of the law of Ukraine No. 3447-VI of 21 February 2006, On Protection of Animals from Cruelty), the General Ethical Principles for Animal Experiments (Reznikov, 2003), and International bioethical standards, including the provisions of the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes (Strasbourg, 1986; Simmonds, 2018; Kabene and Baadel, 2019). The bioethics protocol No. 4/25, dated 8 October 8.10.2025, was reviewed and approved by the Bioethics Commission of the National Scientific Center, Institute of Experimental and Clinical Veterinary Medicine, Kharkiv, Ukraine, in accordance with established procedures.

Study design

The prevalence of gastrointestinal parasitic infections in Indian peafowl was investigated during 2020-2025 in private households operating under free-range conditions and in bioparks of the Odesa region, Ukraine, using a cage-aviary housing system. Under the free-range housing system, Indian peafowl were kept together with chickens (*Gallus gallus domesticus*), ducks (*Anas platyrhynchos domesticus*), and turkeys (*Meleagris gallopavo*), sharing common feeding and watering facilities. Indian peafowl had unrestricted access to outdoor areas, where a grain mixture and green forage were provided, while natural foraging for insects, earthworms, and other invertebrates was also possible. The outdoor areas were cleaned of manure twice a year. No anthelmintic treatment had been administered to the Indian peafowl during the six months preceding sample collection. Indian peafowl were kept in fenced wire-mesh aviaries under the cage-aviary system, providing suitable space for free movement.

Sample collection

The study was conducted on 90 Indian peafowl, including 44 males and 46 females aged 1-2 years. Sample collection was conducted throughout 2020-2025 across all seasons. Of the total number of peafowl, 45 were reared under a free-range housing system, whereas the remaining 45 were kept in bioparks under a cage-aviary housing system. For parasitological examination, one fecal sample was collected directly from the rectum of each Indian peafowl using a sterile cotton swab, yielding a total of 90 samples. The collected material was placed into sterile vials containing 10% formalin, labeled with individual identification numbers, transported to the laboratory, and stored at +4°C until analysis. During sample collection, the macroscopic characteristics of the feces, including color, consistency, and the presence of blood or mucus were evaluated (Keatts et al., 2016; Kathiravan et al., 2017). For the pathomorphological examination, necropsies were performed on 20 Indian peafowl from the same group of 90 individuals that died during the study period.

Coprological examinations were performed using standard parasitological methods (Zajac et al., 2021). Fecal samples were thoroughly homogenized with a 50% glycerol-water solution at a 1:1 ratio. A drop of the resulting suspension was placed on a glass slide, covered with a coverslip, and examined under a light microscope (Carl Zeiss, Germany) at 400x magnification. Protozoa and coccidian oocysts were detected using the direct smear technique, followed by microscopic identification based on their morphological characteristics (Hodgson, 1970). Fecal samples were examined for the presence of nematode eggs, and infection intensity was determined using the McMaster technique, which calculates the number of eggs per gram of feces (EPG; Titilincu et al., 2009).

Eggs of the genera *Ascaridia* and *Heterakis* were distinguished by their morphometric and morphological characteristics. Identification was based on egg shape, size, and shell morphology. *Ascaridia* spp. eggs were oval, measuring $75\text{--}80 \times 40\text{--}50 \mu\text{m}$, whereas *Heterakis* spp. eggs were slightly smaller, measuring $63\text{--}75 \times 36\text{--}48 \mu\text{m}$ (Tarbiat et al., 2021). *Histomonas meleagridis* (*H. meleagridis*) was detected by microscopic examination of impression smears prepared from affected organs and fecal smears obtained from Indian peafowl. Impression smears were prepared from grossly altered areas of the liver and ceca, fixed in methanol for 3–5 minutes, and stained using the Romanowsky-Giemsa method. For fecal examination, direct smears were prepared on clean, grease-free glass slides, air-dried, fixed in ethanol for 5–10 minutes, and stained with Romanowsky-Giemsa stain. The stained slides were observed under a light microscope at 630x magnification (90x objective and 7x eyepiece). *Histomonas meleagridis* trophozoites were identified by their characteristic morphology, with blue cytoplasm and a reddish-purple nucleus, enabling clear visualization of the parasite (Liebhart et al., 2017). The oncospheres of cestodes belonging to the genera *Davainea* and *Raillietina* were differentiated by staining the eggs with a 1:10,000 brilliant green solution. Following incubation, the preparations were examined under a light microscope. The oncospheres of *Davainea proglottina* acquired a light green coloration within 3–5 minutes, whereas those of *Raillietina* spp. remained unstained during the same period, enabling reliable differentiation between the two genera (Bogach et al., 2015). *Choanotaenia infundibulum* was identified through morphological examination of adult cestodes found during postmortem analysis of Indian peafowl, following the diagnostic criteria outlined by Permin and Hansen (1998).

Postmortem and helminthological examinations

Postmortem examinations and helminthological investigations were performed on 20 Indian peafowl obtained from the Laboratory of Parasitology at the Odesa Research Station of the National Scientific Center, Institute of Experimental and Clinical Veterinary Medicine, Odesa, Ukraine. Carcasses were diagnosed with histomonosis, mixed ascariasis-histomonosis infection, and cestode infections caused by *Raillietina* spp., *Choanotaenia infundibulum*, and *Davainea proglottina*. Complete postmortem examinations were performed using the standard evisceration technique according to the established avian necropsy procedures. Gross examination included a systematic assessment of the internal organs, with particular emphasis on the digestive tract. The intestinal mucosa was evaluated for the presence and severity of inflammatory, degenerative, hemorrhagic, and necrotic lesions, and helminths present within the intestinal lumen were recovered and recorded (Swayne et al., 2020). Recovered cestodes were collected, rinsed in physiological saline, and identified based on their morphological characteristics. Species identification was based on the total length and width of the strobila, the morphology of the scolex, the shape and size of the proglottids, and the morphological features of mature segments, following established parasitological identification keys (Soulsby, 1982). Based on a combination of morphological characteristics, the parasites were identified as *Raillietina* spp., *Choanotaenia infundibulum*, and *Davainea proglottina*.

A retrospective analysis was conducted on 17 fatal cases of Indian peafowl, aged 6 weeks to 16 months, submitted to the laboratory during the study period to investigate the pathomorphological features of histomonosis. The diagnosis was established through an integrated approach that incorporated epizootiological data, characteristic gross lesions in the ceca and liver, and microscopic examination of impression smears and tissue scrapings from affected organs. Romanowsky-Giemsa staining was used to identify vegetative forms (trophozoites) of *H. meleagridis*. The observed pathological lesions were documented using a digital camera (Canon EOS 90D, Japan). In order to facilitate systematic presentation and demonstrate the progression of the disease, photographs of the ceca and liver were categorized according to lesion severity. The retrospective reconstruction of histomonosis stages was conducted based on peafowls' history, the extent of cecal lesions, and the severity of degenerative and necrotic changes in the liver.

Data analysis

Statistical analysis was performed using Microsoft Excel 2007 and STATA 13 (StataCorp, College Station, TX, USA). Prior to analysis, all data were checked for completeness and accuracy of entry. Descriptive statistics were used to characterize the prevalence of parasitic infections. Results were presented as absolute mean values with 95% confidence intervals (95% CI). Differences in prevalence among the study groups were evaluated using the Pearson chi-

square (χ^2) test. When expected cell frequencies were less than 5, Fisher's exact test was applied. Differences were considered statistically significant at a P-value less than 0.05. Mixed infections were treated as a separate analytical category and were not included in the corresponding single-parasite groups. Owing to the small number of mixed infections (n = 2), no additional statistical analyses beyond comparing prevalence between housing systems were performed.

RESULTS

The overall prevalence of gastrointestinal parasitic infections in Indian peafowl differed according to the housing system (Table 1). The overall prevalence of gastrointestinal parasitic infections in Indian peafowl was 58.9%. Under the free-range housing system, the overall prevalence was 51.1%, whereas it increased to 66.7% under the cage-aviary housing system. Although gastrointestinal parasitic infections were more frequently detected in Indian peafowl maintained under cage-aviary housing, the difference between the two housing systems was not statistically significant ($p > 0.05$). Figures 1A, 1B, and 1C illustrated the most prevalent protozoan parasites of Indian peafowl.

Table 1. Prevalence of gastrointestinal parasitic infections in Indian peafowl under different housing systems in Odesa Region, Ukraine during 2020-2025

Gastrointestinal parasites		Overall (n = 90)		Free-range housing (n = 45)		Cage-aviary housing (n = 45)		P-value
		Infected n (%)	95% CI	Infected n (%)	95% CI	Infected n (%)	95% CI	
Protozoa	<i>Eimeria</i> spp.	12 (13.3)	8.0-21.3	5 (11.1)	4.8-23.5	7 (15.6)	7.7-28.8	0.535
	<i>Histomonas meleagridis</i>	17 (18.9)	12.2-28.1	6 (13.3)	6.3-26.2	11 (24.4)	14.2-38.7	0.178
Nematodes	<i>Ascaridia galli</i>	5 (5.6)	2.4-12.4	2 (4.4)	1.2-14.8	3 (6.7)	2.3-17.9	1.000
	<i>Heterakis</i> spp.	10 (11.1)	6.2-19.1	4 (8.9)	3.5-20.7	6 (13.3)	6.3-26.2	0.502
Cestodes	<i>Railletina</i> spp.	3 (3.3)	1.1-9.3	3 (6.7)	2.3-17.9	0 (0.0)	0.0-7.9	0.242
	<i>Choanotaenia infundibulum</i>	1 (1.1)	0.2-6.0	1 (2.2)	0.4-11.6	0 (0.0)	0.0-7.9	1.000
	<i>Davainea proglottina</i>	3 (3.3)	1.1-9.3	2 (4.4)	1.2-14.8	1 (2.2)	0.4-11.6	1.000
Mixed infection	<i>Histomonas meleagridis</i> plus <i>Ascaridia galli</i>	2 (2.2)	0.6-7.7	0 (0.0)	0.0-7.9	2 (4.4)	1.2-14.8	0.494

n: Number of Indian peafowl examined, CI: 95% confidence interval, $p < 0.05$ was considered statistically significant.

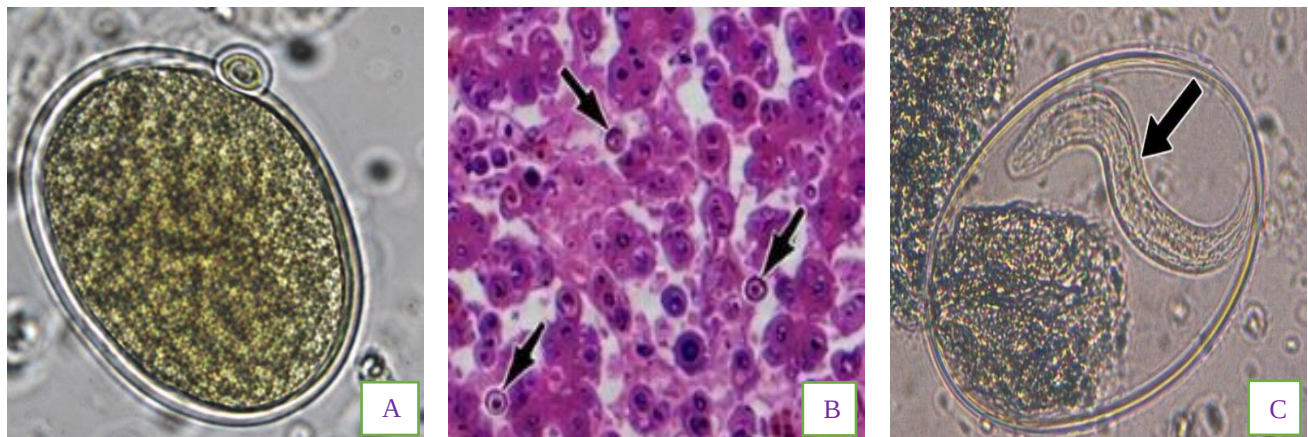


Figure 1. Parasites detected from Indian peafowl in Odesa Region, Ukraine during 2020-2025. A: Unsporulated oocyst of *Eimeria* spp. (400x), B: Trophozoites of *Histomonas meleagridis* (630x), C: *Heterakis* spp. egg containing a larva (400x)

The overall prevalence of protozoan infections was higher in Indian peafowl maintained under the cage-aviary housing system (40.0%) than in those kept under free-range housing (24.4%). *Eimeria* spp. and *H. meleagridis* were the predominant protozoan parasites, accounting for the majority of the detected protozoan infections. Nematode infections were more common in Indian peafowl kept in cage-aviary housing (20.0%) than in those kept in free-range housing (13.3%). This difference was likely attributable to housing-related factors, including higher stocking density and the accumulation of infective stages in the environment. In contrast, cestode infections were more common in Indian peafowl kept in free-range housing, with a prevalence of 13.3%. However, only 2.2% of Indian peafowl maintained under cage-aviary housing were infected. The higher prevalence of cestode infections in free-ranging Indian peafowl was linked to natural intermediate hosts and other potential sources of infection in the outdoor environment.

Mixed parasitic infections were detected only sporadically and were recorded exclusively in a single Indian peafowl maintained under the cage-aviary housing system. Overall, the distribution of gastrointestinal parasites indicated differences in parasite community composition between the two housing systems; however, no statistically significant differences were found between the study groups ($p > 0.05$). In the free-range housing system, there were no significant sex-related differences in the prevalence or types of gastrointestinal parasitic infections in Indian peafowl ($p > 0.05$). Additionally, the parasite species composition was comparable between males and females (Table 2).

The parasite community was mainly composed of protozoan infections, particularly *H. meleagridis* and *Eimeria* spp. Nematode infections from *Ascaridia galli* and *Heterakis* spp. were present in both sexes, indicating no significant differences in prevalence ($p > 0.05$). Cestode infections, including *Raillietina* spp., *Choanotaenia infundibulum*, and *Davainea proglottina*, were detected in both sexes, with no significant sex-related differences in prevalence ($p > 0.05$). No mixed infections were detected among Indian peafowl housed in free-range systems, indicating that single-species infections predominated. Under the cage-aviary housing system, parasitic infections were detected in both male and female peafowl. The parasite species composition was similar between males and females, but female Indian peafowl tended to have a higher prevalence of some parasite species (Table 3).

Under the cage-aviary housing system, the parasite community was dominated by protozoan infections. *Histomonas meleagridis* and *Eimeria* spp. were the most prevalent parasites and were detected more frequently than the other endoparasites identified in the present study.

Nematode infections caused by *Ascaridia galli* and *Heterakis* spp. were detected in both sexes, with comparable prevalence rates ($p > 0.05$). Cestode infections were rare under cage-aviary housing. Only one female Indian peafowl kept under cage-aviary housing was infected with *Davainea proglottina*, and no other cestode species were detected in the cage-aviary group. Mixed infection with *H. meleagridis* and *Ascaridia galli* was observed only under the cage-aviary housing system, suggesting that this housing system may facilitate the simultaneous circulation of multiple parasitic agents. Analysis of parasites related to housing system and sex revealed higher infection intensity in Indian peafowl housed in cage-aviary systems than in free-range systems (Table 4).

Table 2. Prevalence of gastrointestinal parasitic infections in Indian peafowl under free-range housing system according to sex in Odesa Region, Ukraine during 2020-2025

Gastrointestinal parasites	Males (n = 22)		Females (n = 23)		P-value
	Infected n (%)	95% CI	Infected n (%)	95% CI	
<i>Eimeria</i> spp.	2 (9.1)	2.5-28.0	3 (13.0)	4.5-32.1	1.000
<i>Histomonas meleagridis</i>	3 (13.6)	4.8-34.2	3 (13.0)	4.5-32.1	1.000
<i>Ascaridia galli</i>	1 (4.5)	0.8-21.8	1 (4.3)	0.8-21.9	1.000
<i>Heterakis</i> spp.	2 (9.1)	2.5-28.0	2 (8.7)	2.4-27.8	1.000
<i>Raillietina</i> spp.	2 (9.1)	2.5-28.0	1 (4.3)	0.8-21.9	1.000
<i>Choanotaenia infundibulum</i>	1 (4.5)	0.8-21.8	0 (0.0)	0.0-13.8	0.489
<i>Davainea proglottina</i>	1 (4.5)	0.8-21.8	1 (4.3)	0.8-21.9	1.000
<i>Histomonas meleagridis</i> plus <i>Ascaridia galli</i>	0 (0.0)	0.0-13.8	0 (0.0)	0.0-13.8	1.000

n: Number of Indian peafowl examined, CI: 95% confidence interval, $p < 0.05$ was considered statistically significant.

Table 3. Prevalence of gastrointestinal parasitic infections in Indian peafowl under cage-aviary housing according to sex in Odesa Region, Ukraine during 2020-2025

Gastrointestinal parasites	Males (n = 22)		Females (n = 23)		P-value
	Infected n (%)	95% CI	Infected n (%)	95% CI	
<i>Eimeria</i> spp.	3 (13.6)	4.8-34.2	4 (17.4)	7.1-36.8	1.000
<i>Histomonas meleagridis</i>	5 (22.7)	10.0-43.6	6 (26.1)	12.6-45.7	1.000
<i>Ascaridia galli</i>	1 (4.5)	0.8-21.8	2 (8.7)	2.4-27.8	1.000
<i>Heterakis</i> spp.	3 (13.6)	4.8-34.2	3 (13.0)	4.5-32.1	1.000
<i>Raillietina</i> spp.	0 (0.0)	0.0-13.8	0 (0.0)	0.0-13.8	1.000
<i>Choanotaenia infundibulum</i>	0 (0.0)	0.0-13.8	0 (0.0)	0.0-13.8	1.000
<i>Davainea proglottina</i>	0 (0.0)	0.0-13.8	1 (4.3)	0.8-21.9	1.000
<i>Histomonas meleagridis</i> plus <i>Ascaridia galli</i>	1 (4.5)	0.8-21.8	1 (4.3)	0.8-21.9	1.000

n: Number of Indian peafowl examined, CI: 95% confidence interval, $p < 0.05$ was considered statistically significant.

On average, the intensity of parasitic infection was 1.5-2.5-fold higher under the cage-aviary housing system than under free-range housing. Higher parasite infection rates were observed in both males and females, with females generally demonstrating higher rates. Among the protozoa, *Eimeria* spp. and *H. meleagridis* indicated the highest intensity. In the cage-aviary housing system, the levels of *Eimeria* spp. exceeded those observed in free-range housing by more than twofold, whereas *H. meleagridis* demonstrated an approximately twofold increase. The intensity of *Ascaridia galli* and *Heterakis* spp. was comparatively lower than that of the protozoa; however, it was 1.5 to 2.0 times greater in cage-aviary housing than in the free-range housing system. Among the nematodes, the highest values were recorded for *Heterakis* spp. Cestodes were detected at relatively low infection intensity and were mainly found in the free-range housing system, with only a single case of *Davainea proglottina* recorded in a female under cage-aviary housing. Mixed infection of *H. meleagridis* and *Ascaridia galli* was observed only under cage-aviary housing, with higher values in females. Overall, the current results indicated higher parasite levels in Indian peafowl maintained under a cage-aviary housing system than under free-range housing. The present results demonstrated differences in the prevalence, intensity, and species composition of gastrointestinal parasites between the two housing systems. Because *H. meleagridis* was among the most frequently detected parasites, the subsequent stage of the study focused on the pathomorphological changes in the internal organs of Indian peafowl at different stages of histomonosis.

In Indian peafowl, the initial gross lesions of histomonosis appeared as early as 3-5 days after clinical signs began, while severe organ deformation in the final stage typically developed within 12-21 days.

At the early stage of acute histomonosis, which corresponded to 3-5 days after the onset of clinical signs in peafowls, pathological changes were predominantly attributable to *H. meleagridis* infection in the cecal mucosa and the development of an initial inflammatory response. Postmortem examination during the acute stage revealed marked vascular congestion and hyperemia of the mesentery. The loops of the small and large intestines were edematous, and the serosal surface was thickened and moderately hyperemic. In the caudal part of the intestine, early areas of tissue damage exhibiting yellow fibrinous deposits were observed, indicative of acute catarrhal-hemorrhagic or serofibrinous inflammation (Figure 2).

As the disease progressed, approximately 5-7 days after the onset of clinical signs, marked focal thickening of the walls of both ceca was observed. The serosal surface was intensely hyperemic, with a prominent vascular pattern and bluish-red discoloration due to vascular congestion. As a result of exudative inflammation and cellular infiltration of the submucosa, the cecal walls became markedly thickened and firm, while the lumen gradually narrowed due to the accumulation of fibrinonecrotic exudate (Figure 3).

Table 4. Intensity of parasitic infection in Indian peafowl according to housing system and sex in Odesa Region, Ukraine during 2020-2025

Gastrointestinal parasites	Free-range housing (n = 45)		Cage-aviary housing (n = 45)	
	Males (n = 22)	Females (n = 23)	Males (n = 22)	Females (n = 23)
<i>Eimeria</i> spp. (OPG)	320 ± 110 (n = 2)	360 ± 130 (n = 3)	740 ± 230 (n = 3)	820 ± 270 (n = 4)
<i>Histomonas meleagridis</i> (trophozoites per field of view)	6.8 ± 2.5 (n = 3)	7.4 ± 3.0 (n = 3)	14.5 ± 4.8 (n = 5)	18.2 ± 5.9 (n = 6)
<i>Ascaridia galli</i> (EPG)	5.0 (n = 1)	6.0 (n = 1)	9.0 (n = 1)	10.4 ± 4.1 (n = 2)
<i>Heterakis</i> spp. (EPG)	7.5 ± 2.8 (n = 2)	9.2 ± 3.5 (n = 2)	15.1 ± 5.0 (n = 3)	18.2 ± 5.8 (n = 3)
<i>Raillietina</i> spp. (worms/poultry)	2.1 ± 0.8 (n = 2)	3.0 (n = 1)	–	–
<i>Choanotaenia infundibulum</i> (worms/poultry)	6.0 (n = 1)	–	–	–
<i>Davainea proglottina</i> (worms/poultry)	1.5 (n = 1)	2.0 (n = 1)	–	1.0 (n = 1)
<i>Histomonas meleagridis</i> (trophozoites per field of view) plus <i>Ascaridia galli</i> (EPG)	–	–	8.0 7.0	9.0 11.0

n: Number of Indian peafowl examined, OPG: Oocysts per gram of feces, EPG: Eggs per gram of feces. Data are presented as mean ± SD.



Figure 2. Gross pathological changes in the internal organs of an Indian peafowl during the early stage of histomonosis (3-5 days after the onset of clinical signs), reared in Odesa Region, Ukraine. The black arrow indicates the cecum, which shows early pathological changes.



Figure 3. Gross appearance of the ceca with early typhlitis (5-7 days after the onset of clinical signs) in an Indian peafowl, reared in the Odesa Region, Ukraine. The black arrow indicates the pathologically altered cecum with early typhlitis.

At the stage of severe diffuse typhlitis, approximately 7-12 days after the onset of clinical signs, profound destructive changes in the cecal walls were observed. As a result of the intensive progression of the pathological process caused by *H. meleagridis*, the cecal walls became diffusely and irregularly thickened, while the lumen contained large amounts of fibrinonecrotic (caseous) material. Through the thinned and hyperemic serosa, multiple yellowish-gray parts of necrotic lesions were visible. In some areas of the mucosa and muscular layer, erosive and ulcerative lesions were detected, resulting from pathogen-induced tissue destruction, indicating the development of a destructive form of the disease (Figure 4).

In the terminal (chronic) stage of the disease, typically corresponding to 12-15 days of the pathological process, marked organ deformation was observed, along with the formation of large fibrinonecrotic conglomerates and a risk of peritonitis development. At this chronic stage, the ceca of Indian peafowl had lost their normal anatomical structure and appeared as firm, nodular masses. Large nodular areas of yellow and dirty-white coloration were observed on the surface. The cecal lumen was filled with dry, dense caseous (cheesy) fibrinonecrotic masses of layered structure, causing pronounced dilatation of the lumen and stretching of the organ walls (Figure 5).

At the final stage of histomonosis development, approximately 15-21 days after the onset of clinical signs, marked accumulation of fibrinonecrotic masses was observed, which largely replaced the structural components of the cecal wall. In areas with the most extensive caseous deposits, the intestinal wall became markedly thinned and lost its structural integrity. Marked pathological changes created conditions for possible intestinal perforation, leakage of infected contents into the abdominal cavity, and the development of diffuse fibrinous peritonitis (Figure 6).

Parallel to the destructive changes in the ceca, a specific necrotizing hepatitis developed due to the spread of *H. meleagridis* via the portal circulation to the liver. The severity and rate of macroscopic lesion development in the hepatic parenchyma depended on the severity of typhlitis and were characterized by a sequential progression of pathological changes. In the early phase of the widespread invasive process, identified through history and clinical-pathological data indicating initial signs of catarrhal-hemorrhagic typhlitis at 5-7 days into the disease, hepatomegaly and hepatic congestion were observed. The liver exhibited a moderately firm consistency, with slightly rounded lobe margins. Against a dark cherry-red or brown parenchyma background, single small, irregularly distributed, round, grayish-yellow parts were detected, predominantly located subcapsularly (Figure 7).

During the peak of the disease, approximately 7-12 days after clinical signs appear, which corresponds to the stage of diffuse diphtheritic typhlitis, significant destructive and necrotic changes occur in the liver, typical of histomonosis.

Necrotic regions were markedly enlarged and assumed round or disc-shaped forms with clearly defined margins. Crater-like depressions in the central region of the lesions, resulting from coagulative necrosis and subsequent tissue destruction, were observed. The peripheral zone of the lesions remained marginally elevated above the surface of the parenchyma and displayed a yellowish-white or dirty gray coloration (Figure 8).

As the infection progressed to the chronic and terminal stages (after 12-15 days of the pathological process), pronounced dystrophic and necrotic lesions of the liver were observed. The hepatic parenchyma lost its normal color and developed a diffuse clay-brown or dirty-yellow appearance, indicating severe degenerative changes, including fatty and granular degeneration. Numerous necrotic foci enlarged, expanded peripherally, and coalesced to form large fibrinonecrotic conglomerates that extended through a substantial portion of the hepatic lobes (Figure 9).

The hepatic parenchyma was diffusely altered, exhibiting a clay-brown coloration, indicating severe degenerative processes, including fatty and granular degeneration. Numerous necrotic foci expanded in size, coalesced, and formed large conglomerates extending through a substantial thickness of hepatic tissue. In some cases, at the terminal stages of infection, extensive areas of deep tissue destruction were observed, involving the serosal membranes of adjacent organs in the pathological process (Figure 10).

The described extensive and severe lesions of the hepatic parenchyma were associated with marked impairment of liver function, development of pronounced endogenous intoxication, and represented one of the key factors contributing to Indian peafowl mortality.

In addition to cases of monoinfection with histomonosis, mixed parasitic infections were recorded during the study. Postmortem examination of dead juvenile Indian peafowl infected with *H. meleagridis* and *Ascaridia galli* revealed complex destructive and inflammatory changes in the digestive tract. The pathological findings were characterized by a combination of lesions typical of histomonosis and ascariasis, manifested by inflammatory, dystrophic, and necrotic changes in the intestine, accompanied by impaired functional status of the gastrointestinal tract (Figure 11).

In the small intestine, a high intensity of infection with adult *Ascaridia galli* nematodes was observed. Pale yellow, spindle-shaped helminths were observed within the intestinal lumen, occasionally forming dense aggregates. The mucosa of the small intestine in the areas of parasite localization exhibited acute catarrhal-hemorrhagic inflammation, characterized by diffuse hyperemia, edema, thickening of the intestinal folds, and multiple small petechial hemorrhages at sites of mechanical damage caused by the parasites.

Damage to the intestinal mucosa by ascarids contributed to disruption of the intestinal barrier, development of secondary inflammatory processes, and created conditions for the progression of pathological changes, particularly cecal involvement in histomonosis.

Simultaneously, characteristic lesions of the ceca caused by intensive development of *H. meleagridis* were observed. Both cecal pouches were markedly enlarged, with diffusely thickened and firm walls. The serosal surface appeared dull, intensely hyperemic, and exhibited a pronounced vascular pattern. The cecal lumina were dilated and filled with dense fibrinous-caseous (croupous) masses of pale gray or yellowish coloration, which adhered tightly to areas of mucosal damage.

During the necropsy of dead Indian peafowl and examination of the gastrointestinal tract, the small intestine exhibited pronounced diffuse destructive and inflammatory changes associated with parasitism by cestodes of the genera *Raillietina* spp. and *Choanotaenia infundibulum* (Figures 12, 13).

In the longitudinal section of the intestine, a high intensity of cestode invasion was observed. The parasites were long, ribbon-like strobilae of pale cream or whitish-yellow coloration, predominantly localized in the middle third of the small intestine, where they occasionally formed dense, intertwined aggregates. A large number of helminths led to pronounced narrowing of the intestinal lumen and, in some areas, partial obstruction of the intestinal tube. The intestinal mucosa at the sites of parasite localization demonstrated marked catarrhal-desquamative enteritis, characterized by hyperemia, edema, thickening, and flattening of the mucosal folds. The surface was covered with a substantial layer of thick, translucent to turbid mucus, caused by mechanical irritation and tissue damage from the parasites' attachment structures. In infections caused by *Choanotaenia infundibulum*, the strobila exhibited characteristic morphological features, including triangular or bell-shaped proglottids, with the posterior part of each segment wider than the anterior and partially overlapping the subsequent segment, resulting in an irregular, wavy outline. The parasites were mainly localized in the anterior segments of the small intestine and induced focal inflammatory and destructive changes in the mucosa. At the sites of helminth attachment to the intestinal mucosa, thick mucus exudate, vascular hyperemia, focal petechial hemorrhages, and superficial necrosis of the intestinal villi were observed. Pathomorphological examination revealed extensive damage to the intestinal villi, mucosal hyperemia and edema, and catarrhal-desquamative enteritis. During necropsy, macroscopic changes consistent with Davaineosis-related toxic duodenitis were noted in the anterior part of the small intestine (duodenum). A large number of small tapeworms identified as *Davainea proglottina* were found in the lumen of the opened intestine (Figure 14).

The parasites were characterized by a short strobila and were mainly localized on the mucosal surface and within its deeper layers. In the sites of parasite localization, pronounced pathomorphological changes of the intestinal wall were observed. The mucosa was diffusely edematous and hyperemic, exhibiting signs of catarrhal-desquamative inflammation. Attachment of the parasites via the scolex and mechanical damage caused by hooks led to injury of the epithelial lining and villi, accompanied by small focal hemorrhages and necrobiotic tissue changes.

On gross examination, these changes appeared as grayish and dark brown discoloration due to the accumulation of mucous exudate, desquamated epithelium, and blood admixtures. Pathomorphological examination of the duodenum in Indian peafowl infected with *Davainea proglottina* revealed disruption of the intestinal epithelium, mucosal hyperemia, edema, and focal destructive lesions.



Figure 4. Diffuse necrotizing typhlitis at the peak of infection (7-12 days after the onset of clinical signs) in an Indian peafowl reared in Odesa Region, Ukraine. The black arrow indicates the area of extensive necrotic damage to the cecal wall.

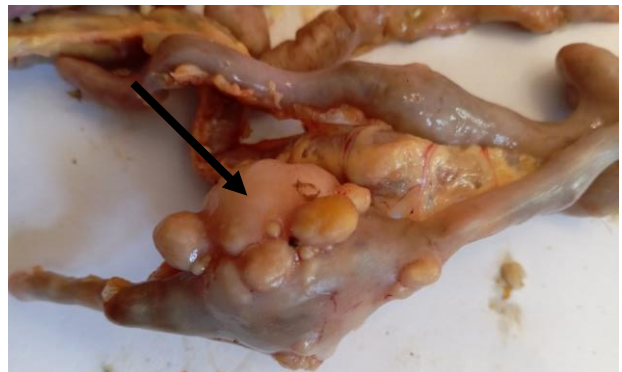


Figure 6. Extensive fibrinonecrotic process (15-21 days after the onset of clinical signs) in the ceca of an Indian peafowl reared in Odesa Region, Ukraine. The black arrow indicates marked thickening and nodular alteration of the cecal wall.

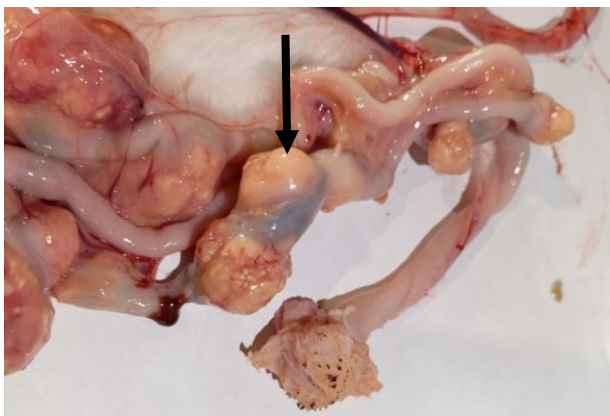


Figure 5. Chronic stage of histomonosis (12-15 days after the onset of clinical signs) in an Indian peafowl with deformation of the ceca, reared in Odesa Region, Ukraine. The black arrow indicates the inflamed and pathologically altered cecum.

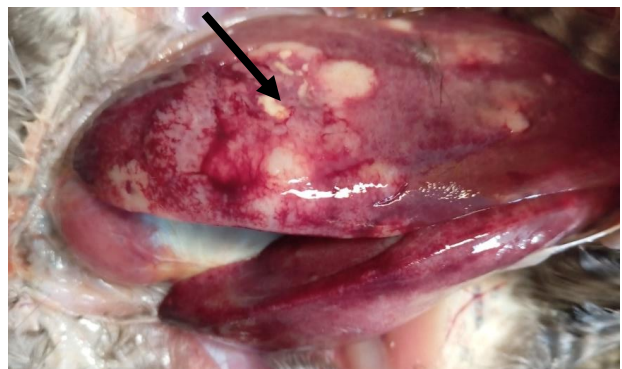


Figure 7. Gross lesions in the liver of an Indian peafowl at the early stage of histomonosis (5-7 days after the onset of clinical signs), reared in Odesa Region, Ukraine. The black arrow indicates multifocal necrotic lesions in the hepatic parenchyma.



Figure 8. Gross appearance of specific necrotizing hepatitis in Indian peafowl at the peak of infection (7-12 days after the onset of clinical signs), reared in Odesa Region, Ukraine. The black arrow indicates multifocal necrotic lesions in the hepatic parenchyma.

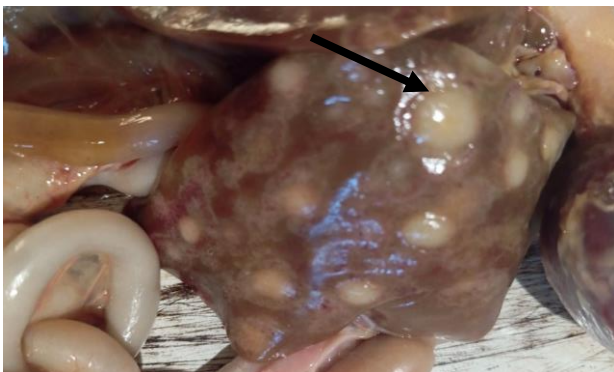


Figure 9. Progressive multifocal dystrophic and necrotizing lesions in the liver of an Indian peafowl at the advanced stage of the pathological process (12-15 days after the onset of clinical signs), reared in Odesa Region, Ukraine. The black arrow indicates extensive multifocal necrotic lesions in the hepatic parenchyma.

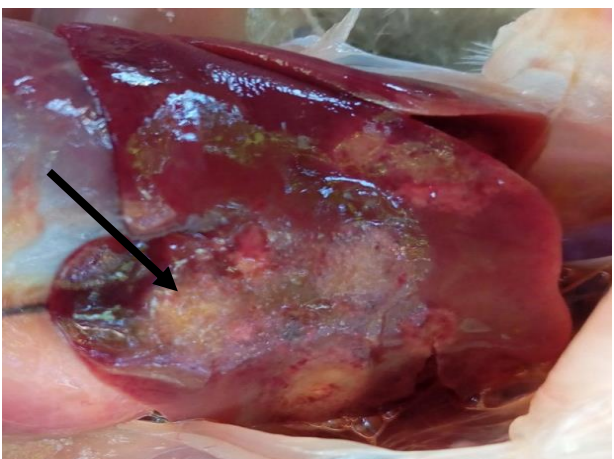


Figure 10. Deep focal necrosis of the hepatic parenchyma at the terminal stage of histomonosis (15-21 days after the onset of clinical signs) in an Indian peafowl reared in Odesa Region, Ukraine. The black arrow indicates extensive multifocal necrotic lesions involving large areas of the hepatic parenchyma.



Figure 11. Gross lesions of the gastrointestinal tract in an Indian peafowl with mixed histomonosis-ascariasis invasion reared in Odesa Region, Ukraine. The black arrows indicate pathological changes in the intestine associated with histomonosis and the presence of *Ascaridia galli*.

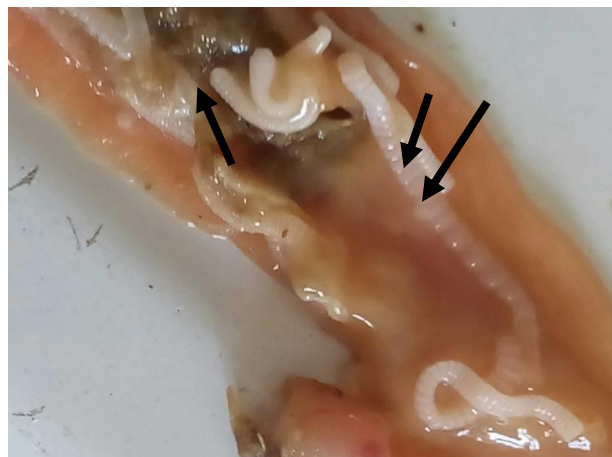


Figure 12. Gross lesions in the intestinal lumen of an Indian peafowl during heavy infection with *Raillietina* spp. in Odesa Region, Ukraine. The black arrow indicates a cestode in the intestinal lumen.



Figure 13. Gross lesions of the small intestine and general appearance of cestode strobilae during heavy invasion by *Choanotaenia infundibulum* in an Indian peafowl reared in Odesa Region, Ukraine. The black arrow indicates cestodes of *Choanotaenia infundibulum* in the intestinal lumen.

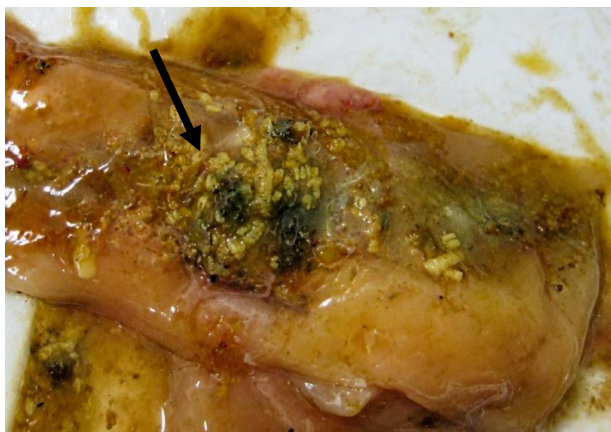


Figure 14. Gross lesions of the duodenum and presence of *Davainea proglottina* in the mucous exudate of an Indian peafowl reared in Odesa Region, Ukraine. The black arrow indicates *Davainea proglottina* cestodes in the mucous exudate.

DISCUSSION

According to previous studies, a wide spectrum of gastrointestinal parasites has been reported in Indian peafowl, including *Hymenolepis* spp., *Davainea proglottina*, *Strongyloides* spp., *Heterakis gallinarum*, *Ascaridia galli*, *Capillaria columbae*, *Acuaria spiralis*, and protozoan parasites such as *Cryptosporidium meleagridis* and *Eimeria* spp. (Kathiravan et al., 2017). Most gastrointestinal parasites are localized in the intestine, where they reproduce, leading to environmental contamination of the same or other poultry, either directly through fecal contamination or via intermediate hosts (Salavati et al., 2023).

When poultry are kept in aviaries, even with proper sanitary management, the observed level of invasion may be linked to contamination of soil, feed, and water with parasite eggs, the introduction of pathogens by free-living wild poultry, as well as factors such as high stocking density, stress, immunosuppression, and concurrent diseases. Under these circumstances, even low-pathogenic parasites may induce notable clinical manifestations (Carrera-Játiva et al., 2020).

In free-range Indian peafowl that interacted with other domestic poultry, the detected level of invasion may result from direct interspecies contact, as well as from shared grazing and feeding areas, which enabled parasitic agents to circulate among different bird types (Penczykowski et al., 2016). Among parasitic diseases, protozoan infections, particularly coccidiosis, were reported as predominant in peafowl worldwide (Zhang et al., 2022). Although parasites typically cause little or no clinical impact in healthy free-living individuals, parasitic infections constitute a major health concern in captive poultry, particularly under high-density housing systems (Jaiswal et al., 2013).

The current results demonstrated a high prevalence of gastrointestinal parasitosis in Indian peafowl, regardless of the housing system. The overall prevalence was 51.1% in free-range housing and 66.7% in cage-aviary housing, indicating a tendency toward higher parasite levels under confined conditions. According to Carrera-Játiva et al. (2018), under zoo conditions with strictly managed sanitation and daily cleaning of artificial enclosures, the prevalence of gastrointestinal parasites was 31.0% in captive poultry and 65.5% in free-ranging poultry.

Protozoan infections predominated in free-range housing, whereas cestode infections were more frequent in cage-aviary housing. The highest prevalence rates were recorded for histomonosis, particularly under cage-aviary housing (24.4%), while coccidiosis was detected in 11.1-15.6% of Indian peafowl. Mixed infections were rare and occurred only sporadically. The obtained prevalence rates were consistent with coprological monitoring data from different geographic regions. In India, the prevalence of endoparasitoses in peafowl ranged from 46.6% to 66.7%, with *Capillaria* (34.0-58.33%), *Ascaridia* (16.66-29.0%), and *Heterakis* (13.4%) as the dominant parasites. Protozoa such as *Eimeria* spp. (12.5-25.0%) and *Giardia* spp. (7.31%) were also recorded. Mixed infections were detected in 34.0% of Indian peafowl in India (Farooq et al., 2020; Manjunatha et al., 2023). In Brazil, nematode invasions in the form of mono- and mixed infections were identified in 66.6% of examined peafowl, with *Ascaridia* eggs being the most frequently detected (Teixeira et al., 2012).

In Nigeria, the highest prevalence among parasites was recorded for *Ascaridia galli* (100%), while *Raillietina* sp. were detected in 28.6% of examined peafowl (Bekeh et al., 2021). The highest overall prevalence (70.0%) was reported in peafowl from Pakistan, where *Eimeria* spp. dominated (66.33%), while the prevalence of *Hymenolepis* spp., *Ascaris* spp., *Ascaridia* spp., *Strongyloides* spp., and *Heterakis* spp. ranged from 3.33% to 10.0% (Naz, 2021).

Previous studies indicated that Indian peafowl exhibited greater susceptibility to *H. meleagridis* infection compared to chickens and pheasants. Conversely, the clinical and pathological manifestations of naturally occurring histomonosis were similar to those observed in chickens and turkeys (Lund and Chute, 1972; Clarke et al., 2017). Particular attention

was given to the prevalence of *H. meleagridis*, as histomonosis is regarded as one of the most severe parasitic diseases affecting poultry (Hauck et al., 2013). The identification of histomonad trophozoites in 13.3% of peafowl housed in free-range systems and 24.4% in cage-aviary settings indicated active circulation of the pathogen within the studied populations.

It is well established that *Heterakis* spp. play an important role in maintaining the epizootic cycle, functioning as reservoirs for *H. meleagridis* and facilitating the long-term survival and dissemination of the pathogen within poultry populations (Beer et al., 2022). In the present study, *Heterakis* spp. were identified in 8.9-13.3% of peafowl, supporting the possibility of a sustained natural cycle of histomonosis infection.

Clinical signs of histomonosis typically appear 7-14 days after infection and include depression, reduced feed intake, impaired coordination, ruffled plumage, and changes in fecal consistency and coloration (Hauck et al., 2013). One of the most characteristic signs is the appearance of yellow-gray or sulfur-yellow droppings, traditionally regarded as a typical clinical manifestation of blackhead disease (Spencer, 2010; Majidi et al., 2025).

Pathomorphological changes in histomonosis in galliform poultry were characterized by progressive diphtheritic-necrotizing typhlitis and specific necrotizing hepatitis, with the formation of characteristic round necrotic parts in the liver (Clarke et al., 2017; Michelazzo et al., 2017). Similar lesions were observed in turkeys by Grafl et al. (2015), who considered necrotic lesions of the ceca and liver to be pathognomonic for histomonosis. According to Grabensteiner et al. (2006), hepatic lesions may range from portal heterophilic granulomas to multifocal or coalescing necrotic foci with different severity. The pathomorphological changes observed in the present study fully corresponded to classical descriptions of histomoniasis. The current results indicated pathological progression, from initial catarrhal-hemorrhagic lesions in the ceca to the formation of large caseonecrotic conglomerates and multiple necrotic foci in the liver, consistent with the current understanding of the pathogenesis of histomonosis in galliform Indian peafowl.

Similar lesions were reported by De Sousa et al. (2021) during a postmortem examination of a three-month-old male peafowl kept in mixed-species poultry housing. The Indian peafowl exhibited severe bilateral diffuse necrotizing typhlitis and multifocal coalescing necrotizing hepatitis. Histological analysis of the ceca revealed transmural necrotizing inflammation of the intestinal wall, with many trophozoites identified as *H. meleagridis*, further confirming the pathological changes noted in the present study.

Nematodoses were represented by *Ascaridia galli* and *Heterakis* spp., which were more frequently recorded under cage-aviary housing. The intensity of invasion by these parasites was higher than in peafowl under free-range housing. Similar results were reported by Titilincu et al. (2009), who observed a high prevalence of ascariasis and heterakidosis in captive peafowl. The mixed infection of *H. meleagridis* and *Ascaridia galli* observed in the present study was notable, as it caused severe intestinal pathology. The presence of catarrhal-hemorrhagic enteritis combined with croupous necrotizing typhlitis led to more extensive damage to the intestinal wall than single infections, suggesting that co-infection may synergistically worsen parasitic conditions.

Cestodoses were predominantly detected in peafowl under free-range housing, where *Raillietina* spp., *Choanotaenia infundibulum*, and *Davainea proglottina* were recorded. The overall prevalence of cestode infections was lower than that of protozoan and nematode infections; however, their presence indicated the involvement of intermediate hosts in maintaining the epizootic cycle. The higher infection level in free-range Indian peafowl was likely associated with access to insects, mollusks, and other invertebrates acting as intermediate hosts for cestodes. A similar relationship between housing system and cestode prevalence has been reported by Dutta et al. (2013) and Abd El-Ghany (2022).

The intensity of parasitic infection was 1.5-2.5-fold higher in Indian peafowl maintained under cage-aviary housing than in those kept under free-range housing. The most pronounced differences were observed in *Eimeria* spp., *H. meleagridis*, and *Heterakis* spp., thereby indicating the critical influence of housing conditions in determining the level of parasitic infection in peafowl. Ombugadu et al. (2019) documented a notable prevalence of gastrointestinal parasites in poultry maintained in zoological gardens, with infection rates reaching up to 100% in some areas, and identified five parasite taxa, including coccidia, *Balantidium* spp., *Capillaria* spp., *Ascaris* spp., and *Strongyloides* spp. Among these parasites, *Capillaria* spp. was the most prevalent (14.1%), while mixed infections were observed in 10.1% of poultry cases. Infection intensity ranged from 63.0 ± 16.67 to 101.98 ± 10.36 EPG for nematodes and from 297.89 ± 20.41 to 332.47 ± 16.67 oocysts per gram for coccidia. Increased stocking density and accumulation of infective parasite stages in the environment may facilitate parasite transmission among poultry (Heckendorn et al., 2009).

The current results aligned with previous findings, as both the prevalence and severity of parasitic infections were elevated in the cage-aviary housing system, indicating an increased risk of parasite accumulation and transmission under these conditions. Thus, the present study confirmed that peafowl were susceptible to a wide range of protozoan, nematode, and cestode infections. *Histomonas meleagridis*, *Eimeria* spp., and *Heterakis* spp. were frequently observed in parasitic pathology, while the housing system notably influenced parasite abundance.

CONCLUSION

The present study demonstrated that the housing system influenced the prevalence and intensity of parasitic infections in Indian peafowl, with higher parasite levels observed under the cage-aviary system. Histomonosis was identified as one of the most clinically significant parasitic infections, characterized by necrotizing lesions of the ceca and liver, while cestode infections were associated with catarrhal-desquamative enteritis and impaired intestinal function. A limitation of the study was the inability to determine the exact time course of pathological changes in naturally occurring histomonosis cases, due to the retrospective nature of the analysis. Further studies involving larger populations and molecular characterization of parasite species are recommended to improve understanding of parasite transmission dynamics and develop effective control strategies in Indian peafowl.

DECLARATIONS

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Authors' contributions

Mykola Bogach conceptualized the study and participated in parasitological and postmortem examinations, data analysis, and manuscript revision. Olena Bohach participated in sample collection, laboratory investigations, statistical analysis, and manuscript preparation. Olha Piven contributed to sample collection and parasitological examinations. Liudmyla Perotska participated in data analysis and scientific revision of the manuscript. Liliya Roman participated in postmortem examinations and documentation of pathomorphological findings. Denys Bohach contributed to laboratory investigations and data organization. All authors reviewed and approved the final edition of the manuscript for publication.

Availability of data and materials

All relevant data generated during the study are included in this published article and are available upon reasonable request from the corresponding author.

Competing interests

The authors declared no conflicts of interest.

Ethical considerations

This manuscript is original, has not been previously published, and has not been submitted simultaneously to any other scientific journal. The authors confirmed that the manuscript has been screened for academic integrity using plagiarism-detection software and that the reported findings are based exclusively on their own original scientific research. The authors confirmed that no artificial intelligence tools were used in the preparation or writing of the manuscript. The authors take full responsibility for the accuracy, originality, and integrity of the final content and affirmed that they are solely responsible for the originality of the manuscript throughout the submission, revision, and publication processes.

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