Damanhour Journal of Veterinary Sciences 9 (1), (2023) 16–22

Damanhour Journal of Veterinary Sciences

Journal homepage: https://djvs.journals.ekb.eg/

Therapeutic and histopathological effects of probiotic on broiler chickens infected with *Salmonella Typhimurium* and *E. coli* (O78)

Amany Sayed Mawas[1*], Safaa Zakaria [2], Ahmed Ibrahim Ahmed[3], Nabila Osman [3]

[1]Department of Pathology and Clinical Pathology, Faculty of Veterinary Medicine, South Valley University, Qena, Egypt
[2]Reference Laboratory for Veterinary Quality Control on Poultry Production (RLQP), Animal Health Institute, Luxor, Egypt
[3]Department of Poultry and Rabbit Diseases, Faculty of Veterinary Medicine, South Valley University, Qena, Egypt

Abstract

An effective control strategy is needed to avoid the counteract effect of Salmonellosis and Colibacillosis which are considered the major bacterial diseases worldwide that cause heavy economic losses in the poultry industry. This study provides the vital therapeutic role and histopathological view of probiotic treatment on broiler chickens infected with Salmonellosis and Colibacillosis in the liver and intestine. One hundred and fifty- one-day-old SASO broiler chicks of a mixed sex were divided into five groups thirty chicks each, 1st group is a negative one, 2nd group is infected with *E.coli* (O78), 3rd group is infected with *E. coli* (O78) then treated with probiotic, 4th group is infected with *S. typhimurium* and the 5th group is infected with *S. typhimurium* then treated with probiotic through two delivery system (food and water) for 5 consecutive days and the experiment lasted for four weeks. Necrotic and hemorrhagic hepatitis and catarrhal enteritis were the most pathological findings with both pathogens. In probiotic treated groups the previous pathological findings were significantly decreased. In addition, the probiotic treated group showed improvement of body weight gain, feed intake, and lower feed conversion ratio (FCR) compared to the infected groups. From the previous results, we elucidate the commercial feasibility and the prophylactic potency of probiotics in the future of the broiler industry especially because of its constancy during storage, processing, and delivery mechanism.

***Keywords*:** Broilers; *Colibacillosis*; Histopathology; Public health; *Salmonellosis*

***Corresponding Author:** Amany Sayed Mawas.
E-mail address: Amany_Jaber@vet.svu.edu.eg
Department of Pathology and Clinical Pathology, Faculty of Veterinary Medicine, South Valley University, Qena, Egypt

P ISSN: 2636-3003
EISSN: 2636-2996
DOI: 10.5455/djvs.2022.115706.1060
Received: October 20, 2022; Received in revised form: November 03, 2022; Accepted: November 13, 2022
Editor-in-Chief:
Prof Dr/Ali H. El-Far (ali.elfar@damanhour.edu.eg)

1. Introduction

Avian colibacillosis and salmonellosis are the two among the most significant poultry bacterial infections in the world that are associated with a decrease in productivity, high morbidity, and mortality rates as a primary or secondary pathogen causing variable disease manifestations including enteritis and peri-hepatitis (Kabir, 2010). Under certain stress circumstances as immunosuppression, bad hygiene, overcrowding, and unfavorable housing ventilation, *E. coli* turns to be pathogenic instead of being a commensal intestinal organism (Goswami et al., 2004). Some predisposing infectious diseases as Newcastle, Infectious bursal disease (IBD), Mycoplasmosis, IBV infection, and coccidiosis followed by avian colibacillosis resulting in an important economic decrease in the poultry industry. Yolk sac infection, as well as high mortality rate, also can occur due to fecal contamination of the eggs which causes the penetration of the eggshell with *E. coli* from which O78, O1:K1, and O2:K1 are the most common serotypes and are usually resistant to some antibiotics as chloramphenicol, cefradine, and tetracyclines (Rahman et al., 2004).

Contaminated poultry meat causes human food poisoning as it is a source of multiresistant *E. coli* strains that can easily infect humans either directly or through food consumption followed by intestinal colonization as the intestinal tract is the most important reservoir of *E. coli* causing resistance genes to human flora followed by bacterial excretion then widespread contamination (Van den Bogaard et al., 2001).

Salmonellosis is considered also a wasteful and destructive disease for the poultry rearing system because most of the salmonella strains are possibly pathogenic for humans and animals. Fowl typhoid and pullorum diseases which are caused by *S. gallinarum-pullorum* have the ability to transmit from one generation to the next by the infected eggs so it is associated with foodborne illnesses (Wigley et al., 2001). The liver and spleen are the main sites of the multiplication of these bacteria as they are rich reticuloendothelial organs (Barrow et al., 1994). Diarrhea and gastroenteritis are common clinical signs for avian salmonellosis; making salmonellosis one of the health risk problems to people and the object of the world monitoring programs (Yan et al., 2004). Probiotics which are preventative live microorganisms are fighting non-residual tools against specific pathogens associated with diarrhea as *Salmonella* and *E. coli* as it increases intestinal health when they are ingested through immune activation, degradation of epithelial invasion, adherence and metastasis, and antimicrobial substances production, in addition to its good, advanced role on poultry performance. Colon cancer also can be inhibited by probiotics because of its effective role in the inhibition of intestinal bacterial enzymes which may be involved in the progression of colon carcinogenesis (Rolfe, 2000). In our previous study, nine different Salmonella species (*S. Typhimurium, S. Enteritidis, S. Anatum, S. Kentucky, S. Bargny and S. Molade, S. Newport, S. Ingada and S. Agona*) and 8 *E. coli* serotypes (O78, O1:H7, O91:H21, O128:H2, O2:H6, O26:H11, O55:H7, and O146:H21) were isolated from 300 different pooled broiler chicks with *S. typhimurium*, and *O78* are the most common isolates for *Salmonella* and *E. coli* respectively. Also, we evaluated the effect of 21 antibacterial agents on the different bacterial strains and we found the resistance of *Salmonella* against (Oxytetracycline, Doxycycline, Tetracycline then Enrofloxacin, Sulphamethoxazole) and its sensitivity to (Gentamycin, colistin sulfate, and Ceftiofur). While most *E. coli* isolates were resistant to (Neomycin and Streptomycin) and were sensitive to (Ceftiofur then Colistin sulfate) (Zakaria et al., 2018). The aim of this study was conducted to evaluate the therapeutic and histopathological efficacy of mixed probiotic applications on broiler chickens' performance which were infected with Salmonellosis and colibacillosis as maintenance of intestinal microflora provides important nutritional health benefits which can help in their elimination, decrease potential hazards to the public health and poultry industry progress.

2. Material and Methods

2.1. Experimental Chicks

A total of one hundred and fifty-one-day-old Saso broiler chicks of mixed sex were used for evaluation of the protective value of probiotic mixture against *S. typhimurium* and *E. coli* (O78) challenge. The chicks were taken from a breeder flock free from Salmonellosis and Colibacillosis. Chicks were randomly divided into five equal groups; each group contained 30 birds. All birds were subjected to the ordinary vaccination program against Newcastle using live Hitchner B1 and La Sota vaccine strains at 6 and 17 days of age, respectively, and against Gumboro disease using live intermediate plus strain (228 E) at 14 days of age. All the vaccines were given via eye drop instillation. All birds were fed balanced commercial starter and growing rations (21% and 18% protein respectively) and water was supplied and libitum. The birds were housed in floor-pen and clean well ventilated separate experimental rooms.

2.2. Experimental Design

On the first day, ten chicks were taken randomly, sacrificed, and then examined bacteriologically to prove their freedom from *S. typhimurium* and *E. coli* (O78) infections. Chicks were randomly divided into five equal groups; each group contained 30 chicks as the following (**Table 1**).

2.3. Probiotics

Commercial preparation (Micro- Procell [cheil- Bio.com. LTD]) (Guardizen M) ® mixed probiotics concentrate 5.6g (min. 1×10^{10} cfu) containing Lactobacillus Plantarum, Lactobacillus Acidophilus, Lactobacillus rhamnosus, Lactobacillus bulgaricus, Bifidobacterium bifidum, Streptococcus thermophilus, Enterococcus faecium, Aspergillus oryzae, Candida pintolopesii and lactose, Dextrose 994.4g. It was given through two delivery systems either in the drinking water or in feed at one day of age for 5 consecutive days in a dose of 0.5gm/ 25 liter of the drinking water or 1000g/ton of feed as recommended by the manufacturer.

2.4. Preparation of S. typhimurium and E. coli (O78:80) challenge strains

S. typhimurium and *E. coli* (O78) field strains which were previously isolated from Luxor province and identified serologically in Animal Health Research Institute, Dokki, Cairo, were centrifuged at 3000 r.p.m. for 10 min. Sediment was diluted with sterile buffer saline and adjusted using MacFarland 0.5 tube to contain 10^9 and 3×10^8 colony-forming units (CFUs) /ml for *S. typhimurium* and *E. coli* (O78) respectively (Timms et al., 1990).

2.5. Experimental infection

On the 6^{Th} day of age, each chick in group four was inoculated orally with 1 ml saline suspension containing 10^9 CFUs *S. typhimurium* (Okamoto *et al.*, 2007) and on the 7^{Th} day of age, each chick in group 2 was inoculated orally with 0.5 ml of saline suspension containing 3×10^8 CFUs *E. coli* (O78) (Mahmoud et al., 2007). The period of the experiment extended for 3 weeks after infection.

2.6. Evaluation of the therapeutic effect of Probiotics

Clinical signs, mortalities, and gross lesions: All chicks were kept under daily observation for clinical signs, mortalities, and postmortem lesions.

The Performance parameters: At arrival, the chicks were weighed and then the chicks in each group were subjected to weekly determination of the production parameters that included the body weight (BW), feed intake (FI), and feed conversion ratio (FCR) at the end of each week. These measures were taken till the end of the study (4 weeks of age).

2.7. Evaluation of growth Performance parameters

Bodyweight: The average bodyweight of the chicks in all groups was determined at the start of the experiment at one day old then weekly by weighing the whole birds in each group and then mean weight was calculated.

Body weight gain, feed intake, and feed conversion ratio (FCR): The gain in body weight per week, feed intake and FCR were calculated till the end of the experiment according to Brady measurements of some poultry performance parameters (Brady, 1968).

2.8. Pathological examination

Gross pathology was conducted through post-mortem examination of the chicks which died during the experiment or scarified to observe the gross lesions of the liver and intestine. Histopathology of paraffin embedding technique was used for formalin-fixed tissue processing. Tissue specimens from liver and intestine were collected from sacrificed birds, properly trimmed, washed in tap water, fixed in 10% neutral buffered formalin solution (PH 7.4), passed in ascending grades of alcohols, processed by standard paraffin embedding technique, and were cut (5 µm) thickness by semiautomatic microtome. The sections were placed onto glass slides, dried, and stained with hematoxylin and eosin (H&E) (Bancroft et al., 1996).

2.9. Bacterial count of S. typhimurium

Three chicks from the infected treated and infected non treated groups were taken randomly at 14, 21, and 28 days of age, sacrificed and one gram of cecal contents were aseptically removed and ground in a sterile mortar then placed into sterile tubes containing 9 ml of buffer peptone water than ten-fold serial dilution up to 10^6 was prepared. One ml of each dilution was plated on Xylose Lysine Deoxycholate Agar (XLD agar) and incubated for 24 hours at 37°C and the colony-forming unit (CFU) of S. Typhimurium per gram of cecal content was determined.

2.10. Statistical analysis

One-Way analysis of variance (ANOVA) was used according to Shott Statistical for health professionals (Shott, 1990).

3. Results

3.1. Clinical signs and mortalities

Broiler chicks in the negative control group (G1) appeared normal, without abnormal clinical signs. The infected group with *E. coli* (G2) appeared sleepy and suffered from dullness, depression, and had ruffled feathers, these clinical signs gradually developed to respiratory signs as gasping, sneezing, ralls followed by brown diarrhea, red eye which then became swollen and closed (ophthalmitis); the mortality rate was 10%. Group (4) which was infected with *S. typhimurium* showed depression, ruffled feathers, a tendency to huddle together, and white diarrhea; the mortality rate was 6.6%. Infected and probiotic treated groups (G 3 & G 5) showed fewer clinical signs than infected ones.

3.2. Growth performance

Performance parameters in infected groups (2&4) were lower than the negative control and probiotics treated groups (1, 3, and 5) respectively. Infected *E. coli* group had lower body weight (448g/bird) weekly, feed consumption (1300 g), and increased feed conversion (2.9) rate than *E. coli* infected and probiotic treated group and the negative control group which had body weight (495.66 and 712 g/bird) weekly respectively, increased feed consumption (from 100 to 800g during the four weeks and 1070 g), and lower feed conversion rate (1.6 and 1.5) respectively. As well as in an infected group with *Salmonella*, it has lower body weight (489 g/bird) weekly and feeds consumption (1374 g) in addition to increased feed conversion rate (2.81) than *Salmonella* infected then treated with probiotics and the negative control group which had body weight (596 and 712 g/bird) weekly respectively and feed consumption was (895 and 1070 g) in addition to lowered feed conversion rate (1.5 and 1.5), respectively (**Table 2** and **3**).

Table 1. Showing groups of chicks in experimental design

Groups	No.	Infected & Treated groups
1	30	Negative control (non-infected non-treated chicks).
2	30	Positive control infected with *E. coli* (O78) non treated chicks
3	30	Positive control infected with *E. coli* (O78) treated with probiotics
4	30	Positive control infected with *S. typhimurium* non treated chicks
5	30	Positive control infected with *S. typhimurium* treated with probiotics.

Table 2. Showing the effect of probiotic, *E. coli* (O78) and *S. typhimurium* infection and their combination on mean weight gain

Time	*Mean weight gain g/bird	Groups				
		G1	**G2**	**G3**	**G4**	**G5**
1st Wk.	**Mean**	96.8	86.3	90.3	88.4	93.5
	SE	0.19 ^{A}	0.34 ^{B}	0.08^{C}	0.35 ^{D}	0.02 ^{E}
2nd Wk.	**Mean**	213.1	131.4	150.2	177.3	187.3
	SE	0.33^{A}	0.19^{B}	1.2 ^{C}	0.14^{D}	0.02 ^{E}
3rd Wk.	**Mean**	387.2	266.6	280.7	279.4	289.9
	SE	0.58^{A}	0.18^{B}	0.078^{C}	0.24 ^{D}	1.64^{E}
4th Wk.	**Mean**	712	448.6	495.7	489.1	595.8
	SE	0.27 ^{A}	0.40 ^{B}	0.18^{C}	0.24^{D}	0.37 ^{E}

*Mean with the different standard error (SE) and letters (A, B, C, D, E) means highly significantly different p≤0.001. G1: negative control group, G2: infected group with *E. coli* (O78), G3: infected group with *E. coli* and treated with probiotics, G4: infected group with *S. typhimurium*, G5: infected group with *S. typhimurium* and treated with probiotics. Mean± SE

Table 3. The performance parameters of different experimental groups

Groups	Age/ week	F1, g/bird	BWG	FCR
G1	**1st**	120	95.31	1.25
	2nd	430	298.75	1.43
	3rd	890	540	1.65
	4th	1070	712	1.5
G2	**1st**	97	86.51	1.12
	2nd	351	200.75	1.7
	3rd	930	404	2.3
	4th	1300	448	2.9
G3	**1st**	100	90.33	1.10
	2nd	352	235.8	1.49
	3rd	785	434	1.8
	4th	800	495.66	1.6
G4	**1st**	113	88.66	1.27
	2nd	372	260.33	1.42
	3rd	1006	457.33	2.19
	4th	1374	489	2.81
G5	**1st**	118	93.51	1.26
	2nd	370	269	1.37
	3rd	880	517.33	1.7
	4th	895	596	1.5

FI: feed intake, BWG: body weight gain, FCR: feed conversion rate. G1: negative control group, G2: infected group with *E. coli* (O78), G3: infected group with *E. coli* and treated with probiotics, G4: infected group with *S. typhimurium*, G5: infected group with *S. typhimurium* and treated with probiotics.

3.3. Histopathology

Recorded postmortem lesions in *E. coli* were swollen, congested liver, and hemorrhagic enteritis accompanied with gas-filled ceca. In the case of Salmonellosis, gross examination revealed discolored congested round border livers with necrotic patches, while intestines were hemorrhagic and swollen and has necrotic debris **(Figure 1).**

Histopathologically, livers of infected chickens with *E. coli* revealed hepatitis and cholangiohepatitis characterized by thrombosis of the central veins with surrounded necrotic hepatocellular parenchyma resulting in widened hepatic stroma including hepatic sinusoids and associated with disorganized hepatic cords. Some areas have complete necrosis and hepatocytes replaced by debris and loss of hepatic cords appearance. Other livers showed fibrotic tissue filled the portal area and infiltrated with inflammatory reaction as well as biliary epithelial cells degeneration and necrosis. Livers that were infected with *E. coli* then treated with probiotics had mild hepatic congestion, fibrosis, and inflammatory reaction **(Figure 2).**

Different enteric lesions were noticed in chickens infected with *E. coli* and others infected with *E. coli* then treated with probiotics. Some intestines showed catarrhal enteritis appeared as hyperactivation of mucous secreting glands and others revealed massively degenerated mucosa and necrotic glands. Hyperplasia, desquamation, and sloughing of the epithelial lining of the intestinal villi in addition to sub-mucosal leukocytic infiltration were also noticeable. In cases infected with *E. coli* then treated with probiotics, intestines had mild atrophied intestinal gland and inflammatory reactions **(Figure 3).**

Livers infected with *Salmonella* revealed congested central veins surrounded by massive areas of hepatic coagulative necrosis characterized by nuclear pyknosis and karyolysis associated with architectural distortion, other livers showed focal mononuclear cells infiltration either between hepatic cells or around the portal areas. Livers infected with *Salmonella* then treated with probiotics showed mild vascular congestion in addition to the mild restoration of hepatic architecture **(Figure 4).**

Intestines infected with *Salmonella* revealed hyperplasia of the epithelial lining of the intestinal villi, desquamation of the epithelium resulting in denatured villi where the lumen filled with necrotic masses and massive mixed leukocytic infiltration in the intestinal lumen in patchy distribution were seen. In cases infected with *Salmonella* then treated with probiotics, mild inflammatory reaction and moderate distribution of necrotic gland were revealed as well as keeping of glands architecture **(Figure 5).**

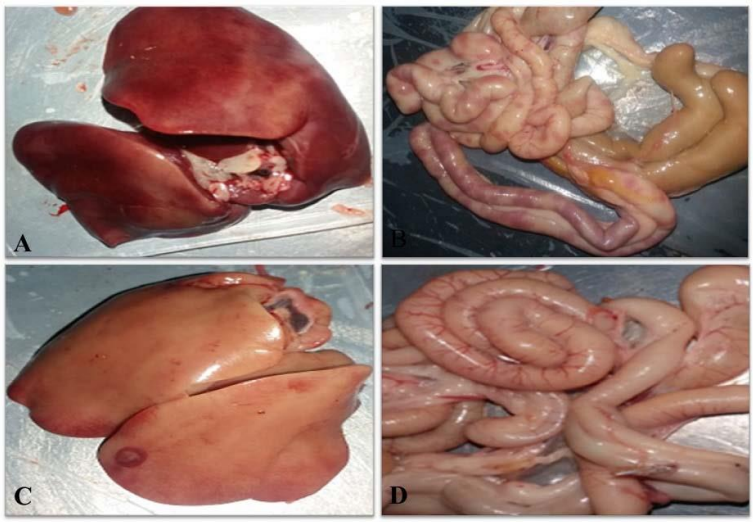

Figure 1. Postmortem examination of chicken infected with *E. coli* on the 7th day of age (G2) showing (A) congested swollen liver and (B) hemorrhagic enteritis with mucosal inflammatory evidence with ballooning of two ceca with gases, while in chicken infected with *S. typhimurium* on the 6th day of age (G4), (C) the liver was enlarged, pale color with necrotic foci and round borders and (D) petechial hemorrhages in the intestinal lumen. (A&B: *E. coli* infection and C&D: *S. typhimurium* infection).

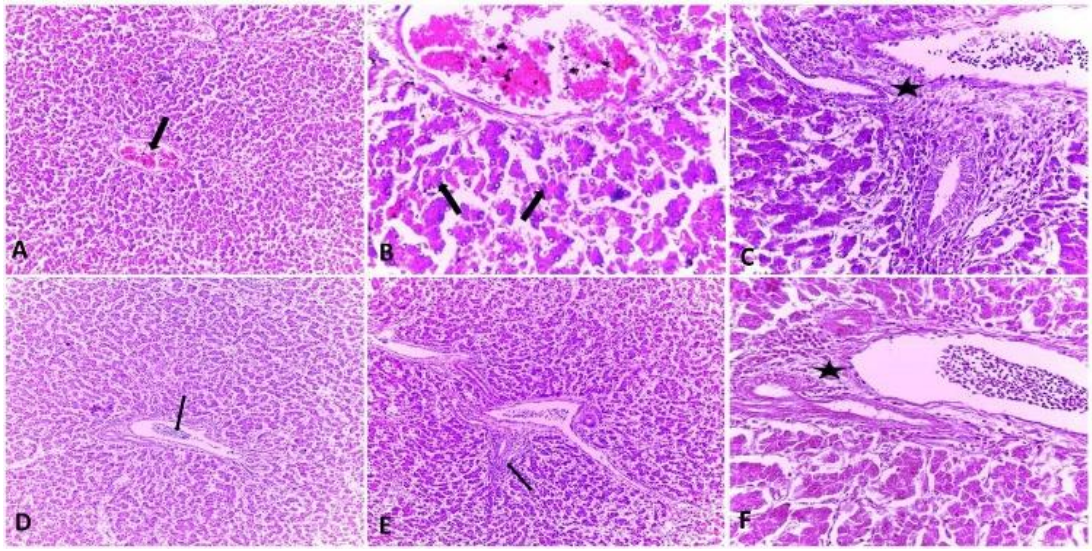

Figure 2. Liver of chicken infected with *E. coli* on the 7th day of age (G2) and liver of chicken infected with *E. coli* then treated with probiotics (G3) showing (A) thrombosis in the central vein (thick arrow), (B) necrotic hepatocellular parenchyma (thick arrows), (C) fibrous tissue in the portal area (asterisk), (D&E&F) mild inflammatory reaction (thin arrows & asterisk). (A&B&C: livers infected with *E. coli*, D&E&F: liver infected with *E. coli* then treated with probiotics). H&E stain, A&D&E: X 100, B&C&F: X 400.

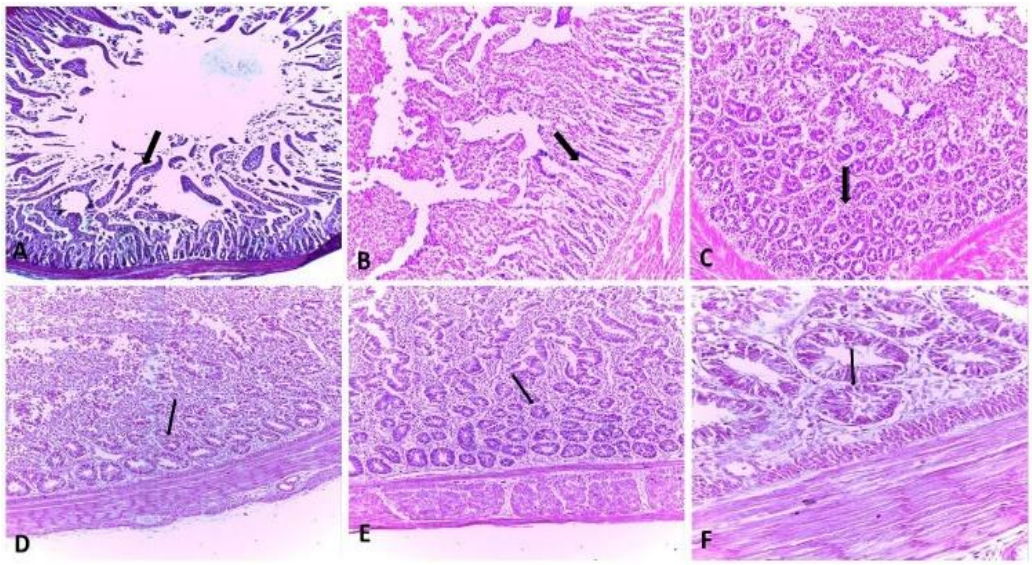

Figure 3. Intestine of chicken infected with *E. coli* on the 7th day of age (G2) and intestine of chicken infected with *E. coli* then treated with probiotics (G3) showing (A&B) necrotic intestinal villi (thick arrows), (C) hyper activating glands (thick arrow), (D&E&F) mild hyper activating glands (thin arrows). (A&B&C: intestine infected with *E. coli*, D&E&F: intestine infected with *E. coli* then treated with probiotics). H&E stain, A&B&C&D&E: X 100, F: X 200.

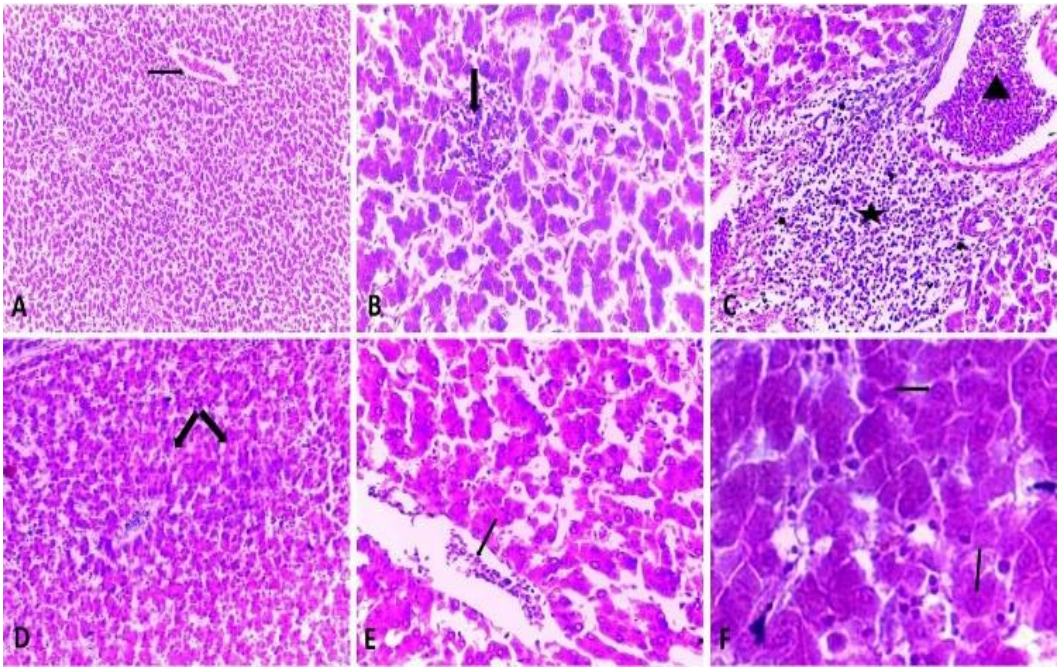

Figure 4. Liver of chicken infected with *S. typhimurium* on the 6th day of age (G4) and liver of chicken infected with *S. typhimurium* then treated with probiotics (G5) showing (A&C) massive hepatic necrosis with congestion in the central vein (thin arrow and triangle), (B) focal inflammatory reaction (thick arrow), (C) periportal inflammatory reaction (asterisk), (D) mild restoration of hepatic architecture (thick arrows), (E) mild vascular congestion (thin arrow), (F) some of the hepatic cells showed nuclear restoration (thin arrows). (A&B&C: livers infected with *S. typhimurium*, D&E&F: liver infected with *S. typhimurium* then treated with probiotics). H&E stain, A&D: X 100, C: X 200, B&E&F: X 400.

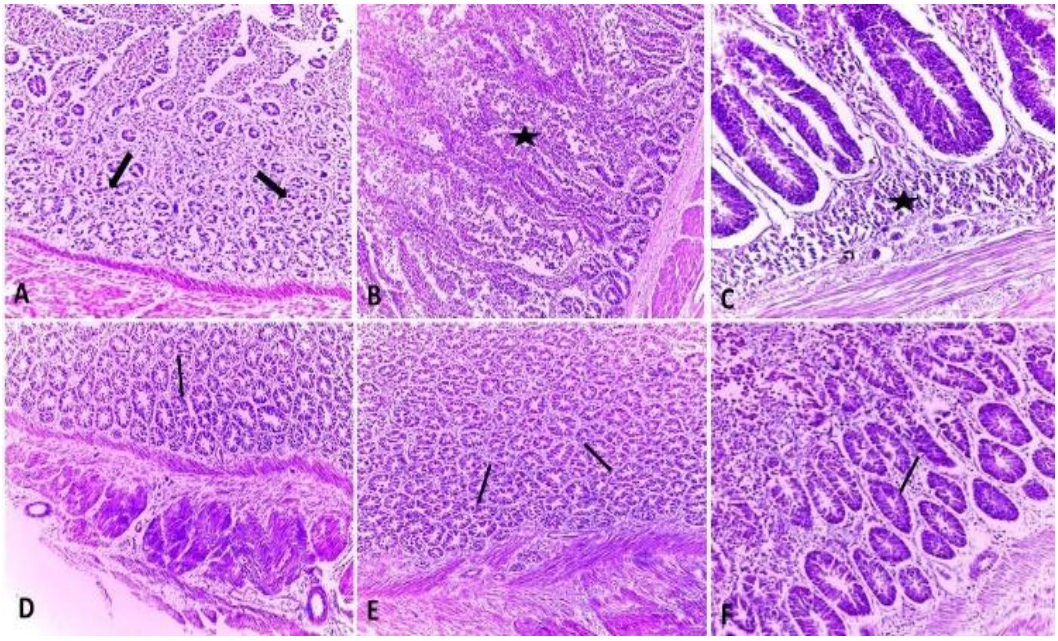

Figure 5. Intestine of chicken infected with *S. typhimurium* on the 6th day of age (G4) and intestine of chicken infected with *S. typhimurium* then treated with probiotics (G5) showing (A) necrotic intestinal glands (thick arrows), (B&C) necrotic intestinal villi with severe inflammatory reaction (asterisks), (D&E&F) moderate gland activation and necrosis, gland architecture is maintained (thin arrows). (A&B&C: intestine infected with salmonella, D&E&F: intestine infected with salmonella then treated with probiotics). H&E stain, A&B&D&E: X 100, F: X 200, C: X 400.

3.4. Bacterial count of S. typhimurium

Cecal count of *S. typhimurium* in the salmonella-infected group (G4) was higher than infected salmonella and probiotic treated group (G5) (**Table 4**).

Table 4. is showing the count of *S. typhimurium* isolated from 1 g of intestinal content

Groups	Bacterial count in Ig of intestinal content post-infection		
	2nd week	3rd week	4th week
G4	4.2×10^9	9.5×10^9	2×10^{11}
G5	8.5×10^6	4.3×10^6	2.7×10^6

G4: infected group with *S. typhimurium*, G5: infected group with *S. typhimurium* and treated with probiotics.

4. Discussion

Probiotic administration is a beneficial method in introducing a commensal microflora in chicks. In this study, we evaluated the therapeutic and histopathological effect of probiotic treatment on chicks infected with *Salmonella* and *E. coli* which are considered a serious public health risk. Herein probiotics were given to chicks through two methods, food, and water, we found that probiotic treatment improved feed consumption and decreased feed conversion rate. Administration methods of probiotics were through feed and water, both food adequacy and growth capacity were improved by probiotics (Olnood et al., 2015). In our study, postmortem examination indicated congested swollen liver and hemorrhagic enteritis in cases infected with *E. coli* and congested necrotic liver and hemorrhagic necrotic intestines in case of *Salmonella*. The same gross finding was recorded by (Shah et al., 2003) who found that colibacillosis in the liver appeared as grayish-

white fibrin layer and pale necrotic foci, enlarged bronze discolored liver with mottled appearance and necrotic foci were the gross feature of *Salmonella Gallinarum* (El-Sayed et al., 2017). The most prominent hepatic histopathological lesions in both salmonella and *E. coli* infections were diffuse liver necrosis with a pyknotic nucleus and highly acidophilic cytoplasm in addition to focal inflammatory cells infiltration either in hepatic parenchyma or in the portal areas, biliary epithelial cells hyperplasia forming newly formed bile ductules, others showed epithelial degeneration, and some portal blood vessels were congested, and some were thrombotic. Regarding the intestines, catarrhal enteritis, inflammatory cell infiltration, sloughing off the desquamated mucosal epithelium in the intestinal lumen, denatured villi, and necrotic enteritis were the most prominent histopathological features. Hepatic sinusoid dilation, degenerative and necrotic liver changes, and fibrinous perihepatitis were the histopathological lesions in colibacillosis founded by (Gangane et al., 2006). In the case of the intestine, blood vessel congestion, catarrhal enteritis, massive leukocytic infiltration, goblet cell hyperplasia, and shortening of the intestinal villi were the histopathological lesions recorded by (Ghosh et al., 2006; Hooda et al., 2009; Islam et al., 2003; Talha et al., 2001). Livers of chickens infected with *Salmonella* exhibited dilated blood vessels, hepatic cells have degenerative changes and necrosis associated with heterophilic or mononuclear infiltration (Kumari et al., 2013; Nazir et al., 2012), additionally, intestinal mucosa covered with mucinous translucent secretion with necrotic debris and hemorrhages associated with goblet cells hyperplasia with consistent necrotic enteritis (Freitas Neto et al., 2007; Kabir et al., 2005). Some reports revealed the role of probiotics in controlling *Salmonella* strains of poultry via a competitive exclusion mechanism (Garcia et al., 2010; Kabir, 2009). A previous study revealed the role of probiotics in the treatment of necrotic enteritis caused by *Enterobacteria* and *C. perfringens* in the ileum and cecum (Keyburn et al., 2006), others explained the role of the dominant strains L. johnsonii in pathogens control (Cho et al., 2000; La Ragione et al., 2004). Invasive properties and bacterial multiplication within intestinal macrophages are the prominent pathogenicity characters of Salmonellosis causing inflammation in the intestinal mucosa (Humbert and Salvat, 1997), then they spread through the bloodstream and the lymphatic system to the reticuloendothelial tissues like the liver and spleen (Barrow et al., 1994). Probiotic treatment had a significant effect on decreasing the cecal count of *S. typhimurium*. The most important bacterial groups in the intestine were enterobacteria and lactobacilli, and some studies reported that their number was decreased with probiotics administration through the restoration of ileum and cecal microflora equilibrium (Xiao et al., 2014). Otherwise, future studies on the commercial benefits along with cost feasibility are needed for a wide selection on the use of probiotics in the poultry industry.

5. Conclusion

Probiotic inoculation decreased the microbial effect of *Salmonella* and *E. coli* pathogens as it promoted the growth rate and the weight gain, decreased the feed conversion ratio, increased the protection rate against intestinal and hepatic lesions, decreased the mortality rate so it enhanced the immunological and fulfillment prospect of broiler chickens. From the previous results, we elucidated the commercial feasibility and the prophylactic potency of probiotics in the future of the broiler industry especially because of its constancy during storage, processing, and delivery mechanism.

Conflict of interest

The authors report that they have no conflict of interest.

6. References

Bancroft J, Stevens A and Turner D (1996) Theory and practice of histological techniques 4th Ed Churchill Living Stone, New York Edinburgh. *Madrid, Sanfrancisco* 20.

Barrow P, Huggins M and Lovell M (1994) Host specificity of Salmonella infection in chickens and mice is expressed in vivo primarily at the level of the reticuloendothelial system. *Infection and immunity* 62(10): 4602-4610.

Cho J-S, Choi Y-J and Chung D-K (2000) Expression of Clostridium thermocellum endoglucanase gene in Lactobacillus gasseri and Lactobacillus johnsonii and characterization of the genetically modified probiotic lactobacilli. *Current microbiology* 40(4): 257-263.

El-Sayed N, Oda S, Tohamy H, et al. (2017) Pathologic study on the enterohepatic affections in chickens at Alexandria Province, Egypt. *Adv. Anim. Vet. Sci* 5(1): 30-38.

Freitas Neto O, Arroyave W, Alessi A, et al. (2007) Infection of commercial laying hens with Salmonella Gallinarum: clinical, anatomopathological and haematological studies. *Brazilian Journal of Poultry Science* 9(2): 133-141.

Gangane G, Kulkarni G and Yeotikar P (2006) Studies on experimental coli bacillosis in chicks. *Indian veterinary journal* 83(10): 1118-1119.

Garcia KO, Santana AM, Freitas Neto OC, et al. (2010) Experimental infection of commercial layers using a Salmonella enterica sorovar Gallinarum strain: blood serum components and histopathological changes. *Brazilian Journal of Veterinary Pathology* 3(2): 111-117.

Ghosh R, Hirpurkar S and Suryawanshi P (2006) Concurrent colibacillosis and infectious bursal disease in broiler chicks. *Indian veterinary journal* 83(9): 1019-1020.

Goswami P, Chakraborty A, Das R, et al. (2004) Isolation and identification of Escherichia coli from poultry from field cases. *Indian veterinary journal* 81(8): 951-952.

Hooda A, SK M, PRUTHI A, et al. (2009) Studies on poultry mortality with special reference to gastro-intestinal tract disorders. *Haryana Vet* 48: 103-104.

Humbert F and Salvat G (1997) The risk of transmission of salmonellae in poultry farming: detection and prevention in Europe. *Revue scientifique et technique (International Office of Epizootics)* 16(1): 83-90.

Islam M, Das B, Hossain K, et al. (2003) A study on the occurrence of poultry diseases in Sylhet region of Bangladesh. *International Journal of Poultry Science* 2(5): 354-356.

Kabir S (2009) The role of probiotics in the poultry industry. *International Journal of Molecular Sciences* 10(8): 3531-3546.

Kabir S (2010) Avian colibacillosis and salmonellosis: a closer look at epidemiology, pathogenesis, diagnosis, control and public health concerns. *International journal of environmental research and public health* 7(1): 89-114.

Kabir S, Rahman M, Rahman M, et al. (2005) Viability of probiotics in balancing intestinal flora and effecting histological changes of crop and caecal tissues of broilers. *Biotechnology* 4(4): 325-330.

Keyburn AL, Sheedy SA, Ford ME, et al. (2006) Alpha-toxin of Clostridium perfringens is not an essential virulence factor in necrotic enteritis in chickens. *Infection and immunity* 74(11): 6496-6500.

Kumari D, Mishra S and Lather D (2013) Pathomicrobial studies on Salmonella Gallinarum infection in broiler chickens. *Veterinary world* 6(10): 725.

La Ragione R, Narbad A, Gasson M, et al. (2004) In vivo characterization of Lactobacillus johnsonii FI9785 for use as a defined competitive exclusion agent against bacterial pathogens in poultry. *Letters in Applied Microbiology* 38(3): 197-205.

Mahmoud A, Abozyed A and Saleh M (2007) The role of E. coli infection on infectious bursal disease affected broiler flock at north Sinai governorate. *SCVMJ* 2: 69-82.

Nazir S, Kamil SA, Darzi MM, et al. (2012) Pathology of spontaneously occurring salmonellosis in commercial broiler chickens of Kashmir Valley.

Olnood CG, Beski SS, Iji PA, et al. (2015) Delivery routes for probiotics: Effects on broiler performance, intestinal morphology and gut microflora. *Animal Nutrition* 1(3): 192-202.

Rahman M, Samad M, Rahman M, et al. (2004) In vitro antibiotic sensitivity and therapeutic efficacy of experimental salmonellosis, colibacillosis and pasteurellosis in broiler chickens. *Bangladesh Journal of Veterinary Medicine* 2(2): 99-102.

Rolfe RD (2000) The role of probiotic cultures in the control of gastrointestinal health. *The Journal of nutrition* 130(2): 396S-402S.

Shah Q, Soomro N and Tunio S (2003) Coli septicemia in broiler: prevalence and pathology. *Online J. Biol. Sci* 3(3): 287-290.

Shott S (1990) *Statistics for health professionals.* Saunders.

Talha A, Hossain M, Chowdhury E, et al. (2001) Poultry diseases occurring in Mymensingh district of Bangladesh. *The Bangladesh Veterinarian* 18(1): 20-23.

Timms L, Marshall R and Breslin M (1990) Laboratory assessment of protection given by an experimental Salmonella enteritidis PT4 inactivated, adjuvant vaccine. *The Veterinary Record* 127(25-26): 611-614.

Van den Bogaard A, London N, Driessen C, et al. (2001) Antibiotic resistance of faecal Escherichia coli in poultry, poultry farmers and poultry slaughterers. *Journal of Antimicrobial Chemotherapy* 47(6): 763-771.

Wigley P, Berchieri Jr A, Page K, et al. (2001) Salmonella enterica serovar Pullorum persists in splenic macrophages and in the reproductive tract during persistent, disease-free carriage in chickens. *Infection and*

immunity 69(12): 7873-7879.

Xiao J, Zhang Y and Yang Z (2014) Lactic acid bacteria in health and disease. *Lactic Acid Bacteria*. Springer, pp.303-374.

Yan SS, Pendrak ML, Abela-Ridder B, et al. (2004) An overview of Salmonella typing: public health perspectives. *Clinical and Applied Immunology Reviews* 4(3): 189-204.

ZAKARIA S, AHMED A, OSMAN N, et al. (2018) ANTIMICROBIAL SUSCEPTIBILITY TO SALMONELLA AND E. COLI ISOLATES ORIGINATED FROM BROILER CHICKENS IN LUXOR GOVERNORATE. *Assiut Veterinary Medical Journal* 64(157): 163-172.