# THE INFLUENCE OF DIETARY LYSOZYME ON THE GROWTH PERFORMANCE, BLOOD CONSTITUENTS, INTESTINAL MORPHOLOGY AND RESISTANCE AGAINST *ESCHERICHIA COLI* IN THE BROILER CHICKS

**R.A. Hassan[1*]; Y.Z. Eid[2]; Zeinab, M. Farouk[1], M. El-Gbaly[1] and M. A. M. Mousa[1]**

*[1]Animal Production Research Institute, Agricultural Research Center, Dokki, Giza, Egypt. [2]Department of Poultry Production, Faculty of Agriculture, Kafr El-Sheikh University, Egypt.*

**Corresponding author e-mail address (redaalihasan@yahoo.com)*

***(Received 3/12/2023, accepted 22/12/2023)***

## SUMMARY

One of the main Hazardous reasons the chicken sector suffers significant financial losses is *Escherichia coli* (E. coli). The benefits of lysozyme addition in the diet for preventing avian colibacillosis are not well understood. The purpose of the current study was to compare the effects of lysozyme dietary supplementation with antibiotic treatment in broiler chicks infected with E. coli. Eight groups of 600 one-day-old unsexed Ross chicks were created: The first group (control) was not infected and was given a balanced diet free of additives, the second and third groups were also not infected and were treated with 50 mg and 100 mg of lysozyme, respectively, the fourth group was non-infected and treated with 10 mg avilamycin / kg diet, the fifth group was infected and non-treated, the sixth and seventh group was infected and were treated with 50 mg and 100 mg of lysozyme, respectively, and the eighth group was infected and treated with 10 mg avilamycin / kg diet. The infection by E. coli was given orally to the chicks at days 5 and 16 days of age. The experimental period was 35 days for all examined groups. The E. coli-infected group showed decreased phagocytic activity, dressing percentage, relative weight of thymus and bursa, and serum total antioxidant capacity. The liver enzymes, renal function tests, MDA, relative weights of the liver, heart, and spleen, as well as changes in gut morphology and intestinal microbial counts, were all significantly elevated in the same group. The lysozyme-pretreated infected chick showed improvements in body performance metrics in addition to a noteworthy reduction in the tests for renal and hepatic enzymes. Significant improvements were observed in gut morphology, intestinal microbial counts, antioxidant enzymes, and serum immunological parameters in the treated groups. It is possible to draw the conclusion that adding lysozyme (at 50 and 100 mg/kg) to the diet of broiler chickens improves their immune system, performance, and reduces the pathological lesions caused by E. coli infections.

***Keywords:*** *Escherichia coli, lysozyme, antibiotic, broilers, performance, immunity*

## INTRODUCTION

One of the most significant bacterial infections to affect the poultry production, avian colibacillosis causes significant financial losses in broilers. The disease is measured one of the major causes of mortality and morbidity in broilers that reaching up to 50%, either as a main or as a secondary pathogen. *Escherichia coli* causes avian colibacillosis, which is considered a native commensal occupant of the chicken's intestinal tract and the trachea to a lesser degree. Generally, 10–15% of intestinal E. coli are avian pathogenic E. coli with different virulence factors, which might result in systemic diseases such as perihepatitis, pericarditis, peritonitis and airsaculities (Abo El-magd *et al.*, 2019).

When a bird's defence mechanism fails, infection is promoted and is exacerbated by a number of variables, including poor management, concomitant infections, and immunosuppression. Due to the wide range of avian pathogenic E. coli strains present in the field, there is currently no effective vaccine against colibacillosis. As a result, colibacillosis must be controlled with preventative measures and antibiotic therapy. The most typical method for treating colibacillosis is thought to involve the use of antibiotics.

And this approach may have significant negative consequences on the bird and consumers combined with the development of drug resistance (Hashem *et al.*, 2022).

Lysozyme is one feed component that may eventually replace dietary antibiotics (Zhang *et al.*, 2006). This enzyme separates the glycosidic bond between N-acetylglucosamine and N-acetylmuramic acid in the bacterial peptidoglycan of the cell wall (Phillips, 1996). Commercially, lysozyme can be made from avian egg whites. It frequently appears in a variety of animal tissues and fluids (Grossowicz and Ariel, 1983). According to numerous studies, lysozyme in several organisms plays a protector against bacteria (Ibrahim *et al.*, 1996). Lysozymes have an antibacterial effect that is achieved by their direct inducing the phagocytic activity of macrophages or by bacteriolytic action (Biggar and Sturgess**,** 1977). There are a few number of studies have examined the effects of exogenous lysozyme supplementation on broiler chicks performance and digestive health. The current study aims to understand how these feed additives, like lysozyme, can be used to control E. Coli infections and raise chicken productivity.

## MATERIALS AND METHODS

In accordance with an agreement between the Faculty of Agriculture in Kafr El-Sheikh and Animal Production Research Institute, this research was carried out on a private farm under the administration of the Poultry Department of the Faculty of Agriculture, Kafr El-Sheikh University. In order to conduct this study, 600 ROSS broiler chicks that were one-day old were divided into 8 treatments, each of which had five repetitions and each replicate contained 15 chicks that were housed in a 1.3 × 3.5-meter area that was covered in wood shavings to a depth of 5 cm. The treatments were as follows: The 1$^{st}$ group was (control) non infected and fed balanced diet without additives, 2$^{nd}$ group was non-infected and treated with 50 mg lysozyme/kg diet, 3$^{rd}$ group was non-infected and treated with 100 mg lysozyme/kg diet, 4$^{th}$ group was non-infected and treated with 10 mg avilamycin/kg diet, 5$^{th}$ group was infected and non treated, 6$^{th}$ group was infected and treated with 50 mg lysozyme/kg diet, 7$^{th}$ group was infected and treated with 100 mg lysozyme/kg diet and 8$^{th}$ group was infected and treated with 10 mg avilamycin / kg diet. The infection by E. coli was given orally to the chicks at 4x10$^{10}$ CFU/ml per chick at days 5 and 16 of age. By the third week of age, the birds' incubation temperature of 32 degrees Celsius had been gradually dropped to 26 degrees Celsius and they had been exposed to 23 hours of light. During the course of the experiment, feed was available *ad libitum*.

***Diet:***

According to the NRC (1994) standards, two basal diets were created for the starter stage (1–21 days) and the grower stage (22–35 days). Table 1 displays the components and nutrient makeup of the basal diets as determined by AOAC (2000).

***Lysozyme:***

Egg white lysozyme (activity 20000 U/mg); Beijing Solarbio Science & Technology Co., Ltd., Beijing, China)**.**

***Antibiotic:***

Avilamycin 10%, an antibiotic growth promoter. Produced by Kavilamycin®, Kartal Kimya Sanayive Ticaret AS Gebze-Kocaeli / Turkey.

At the 5$^{th}$ and 16$^{th}$ days of age, each chick received an oral infection with 0.5 ml of E. coli (O111:K58) containing (4x10$^{10}$ CFU/ml). Throughout the trial, unlimited access to feed and water was supplied (35 days).

***E. coli (O111:K58):***

The Animal Health Research Institute's Strains Bank, located in Dokky, Giza, is where the E. coli (O111:K58) strain was acquired. This used strain was isolated from omphalitis chicks.

***Preparation of E. coli (O111:K58) inoculum:***

According to Macfaddin (1980), E. coli colonies were cultivated in nutrient broth for 24 hours at 37 °C, and the viable number was adjusted to 4x10$^{10}$ colony-forming units CFU viable organism/ml. Following the procedure outlined by Peighambari *et al.* (2000), chicks were inoculated with 0.5 ml by intranasal route at days 5 and 16 of age.

**Table (1): Composition and calculated analysis of the experimental starter and finisher diets.**

| Ingredient | Starter (%) | Grower (%) |
|---|---|---|
| **Corn grains** | 53.71 | 61.92 |
| **Soybean meal (44%)** | 33.42 | 28.05 |
| **Corn gluten meal (60%)** | 5.22 | 3.20 |
| **Soybean oil** | 3.32 | 2.94 |
| **Limestone** | 1.28 | 1.15 |
| **Dicalcium phosphate** | 1.84 | 1.68 |
| **DL-methionine** | 0.39 | 0.22 |
| **Vitamins and minerals premix*** | 0.30 | 0.30 |
| **L- lysine HCl** | 0.12 | 0.14 |
| **Salt (NaCl)** | 0.40 | 0.40 |
| **Total** | 100 | 100 |
| **Analyzed and calculated composition (NRC, 1994)** | | |
| **Crude protein %** | 23 | 20 |
| **Metabolizable energy (Kcal/kg diet)** | 3094 | 3142 |
| **Methionine %** | 0.80 | 0.58 |
| **Calcium %** | 1.00 | 0.90 |
| **Available phosphorous %** | 0.49 | 0.45 |
| **Lysine %** | 1.25 | 1.11 |

** Composition (per 3 kg): vitamin A 12000000 IU, vitamin D3 2500000 IU, vitamin E 10000 mg, vitamin K3 2000 mg, vitamin B1 1000 mg, vitamin B2 5000 mg, vitamin B6 1500 mg, vitamin B12 10 mg, niacin 30000 mg, biotin 50 mg, folic acid 1000 mg, pantothenic acid 10000 mg, manganese 60000 mg, zinc 50000 mg, iron 30000 mg, copper 4000 mg, iodine 300 mg, selenium 100 mg, and cobalt 100 mg.*

***Measurements:***

Average daily weight gain (ADWG), Average daily feed intake (ADFI), and feed conversion ratio (FCR) were calculated at the end of the experimental period. Throughout the study time, the death rate was noted, and the ratio for each treatment was computed.

Five chicks per treatment were randomly selecting (bird per replicate) during slaughter at 35 d of age. All the organs, comprising the liver, hearts, bursa, spleen, and thymus, were weighed, and then estimated the relative weight to live BW. By dividing the carcass weight plus the edible weight by the live body weight and multiplying by 100, the dressing percentage was calculated.

***Microbial enumerations:***

Sections from the ileum were taken at the conclusion of the experiment from 1 chick per replication (5 chicks from each group) that was acquired during slaughter. The Collins and Lyne (1970) method was used to conduct the E. coli, Clostridium, and Lactobacillus counts.

***Biochemical assays:***

One chick per replicate, or five chicks per group, was used to collect the blood. The blood samples were centrifuged for 15 minutes at 3500 rpm to extract blood plasma, which was then stored at -20 °C for examination of aspartate aminotransferase (AST), alanine aminotransferase (ALT), and cholesterol, creatinine, and uric acid levels, using commercial kits (Diamond Diagnostics, Egypt) and a spectrophotometer. Oxidative capacity parameters, involving activity of total antioxidant capacity (TAC) and thiobarbituric acid reactive substances (TBARS) were analyzed in blood plasma using commercially available kits. The phagocytic activity was detected according to Goddeeris *et al.* (1986).

***Morphological analysis:***

Following the birds' slaughter at the end of experimental period, 5 intestinal sections from each group were obtained in order to calculate the Crypt depth (µm), Villus height (µm), and Villus to crypt ratio. for 48 hours, jejunum samples were treated with 4 percent paraformaldehyde and fixed in paraffin. Hematoxylin and eosin was used to stain tissue samples (5 µm) for morphological investigation. Each segment evaluated nine full villi. The crypt depth was calculated from the bottom of the villus to the lamina propria, and the villus height was calculated from the tip of the villus to the villus-crypt junction.

The villus to crypt ratio was estimated after that. ProgRes CapturePro software and an Olympus optical microscope were utilized for all measurements and evaluations (version 2.7; Jenoptik, Jena, Germany).

***Immune response against Newcastle disease (ND):***

At 35 days of age, antibody titers for the immune response for Newcastle disease virus (NDV) were evaluated in each group (bird per replicate) using the HI test, as recommended by King and Seal (1998).

***Statistical analysis:***

The trial was operated using a completely random design. Duncan's multiple range tests (Duncan,1955) were employed to evaluate the differences in means ($P< 0.05$) and were used in conjunction with The General Linear Model (GLM) approach of SAS (2003).

## RESULTS

***Growth performance:***

Body weight gain and feed intake significantly decreased while FCR significantly damaged in the E. coli-infected group when compared to the control group. Treatments with lysozyme and antibiotics reduce mortality, while improving body weight, weight increase, and FCR throughout the trial period (Table2). The infected non-treated groups demonstrated mortality (10%)**,** while groups that received treatment did not record any mortality.

**Table (2): Effect of experimental treatments on growth performance in broiler chickens during whole the experimental period.**

| Treatments | Growth performance | | | |
|---|---|---|---|---|
| | Daily weight gain, g | Daily feed intake (g/bird/day) | FCR (g feed/g gain) | Mortality % |
| **Control** | 54.00$^{b}$ | 102.80$^{ab}$ | 1.90$^{b}$ | 1.50$^{b}$ |
| **NLyz50** | 55.15$^{ab}$ | 104.57$^{a}$ | 1.89$^{b}$ | 1.50$^{b}$ |
| **NLyz100** | 56.47$^{a}$ | 105.69$^{a}$ | 1.87$^{bc}$ | 1.00$^{b}$ |
| **NANTI** | 56.59$^{a}$ | 104.57$^{a}$ | 1.85$^{bc}$ | 1.00$^{b}$ |
| **INT** | 48.85$^{d}$ | 98.52$^{c}$ | 2.02$^{a}$ | 10.0$^{a}$ |
| **Ilyz50** | 52.00$^{c}$ | 101.55$^{b}$ | 1.95$^{b}$ | 1.50$^{b}$ |
| **Ilyz100** | 54.75$^{ab}$ | 103.66$^{ab}$ | 1.89$^{b}$ | 1.00$^{b}$ |
| **IANTI** | 54.78$^{ab}$ | 103.50$^{ab}$ | 1.88$^{b}$ | 1.00$^{b}$ |
| **SEM** | 0.428 | 0.321 | 0.018 | 0.257 |
| **p-value** | 0.0001 | 0.0001 | 0.0001 | 0.0001 |

*$^{a-e}$Means with different superscripts in a column differ significantly ($P < 0.05$). *SEM =Standard Error of the mean; NLyz50: Non infected treated with lysozyom (50mg). NLyz100: Non infected treated with lysozyom (100mg). NANTI: Non infected treated with antibiotic. INT: Infected with E. coli non treated group. Ilyz50: infected treated with lysozyom (50mg). Ilyz100: infected treated with lysozyom (100mg). IANTI: Infected with E. coli and treated with antibiotic.*

***Microbial enumeration:***

As demonstrated in Table 3, the substantial increases in the ileal Clostridium spp. and E. coli, in addition to the declines in Lactobacillus bacteria counts at 35 days of age, were caused by E. coli infection. The outcomes demonstrated that regardless of the lysozyme dose effect of lysozyme was considerable on the E. coli bacteria, Clostridium spp. and total Lactobacillus bacteria.

***Jejunal morphological indices:***

The effects of lysozyme inclusion in the feed on the jejunal morphological parameters in broilers with E. coli challenges are shown in Table 3. At 35 days of age, it is evident that E. coli infection significantly decreased the VH:CD ratio and the height of the jejunal villi, although it significantly increased the depth of the jejunal crypt. At the age of 35 days, lysozyme and antibiotic supplements were introduced to the diet, which reduced the adverse effects of the E. coli challenge on the jejunal villi width, crypt depth, villi height, and VH:CD ratio.

**Table (3): Effect of experimental treatments on cecum microbial counts (log CFU $g^{-1}$ ) and gut morphology at 5 week of age.**

| Treatments | Cecum microbial counts (log CFU $g^{-1}$ ) | | | Gut morphology | | |
|---|---|---|---|---|---|---|
| | Lactob. | E.coli | Clostr. | Villus Height (VH) (µm) | Crypt Depth (CD)(µm) | VH:CD ratio |
| **Control** | $1.77^{d}$ | $3.62^{a}$ | $2.43^{b}$ | $1305^{cd}$ | $327^{b}$ | $4.00^{a}$ |
| **NLyz50** | $2.55^{c}$ | $3.05^{b}$ | $2.00^{c}$ | $1410^{b}$ | $354^{b}$ | $3.98^{a}$ |
| **NLyz100** | $3.67^{a}$ | $2.16^{d}$ | $1.08^{e}$ | $1650^{a}$ | $394^{a}$ | $4.20^{a}$ |
| **NANTI** | $3.25^{a}$ | $1.85^{e}$ | $0.89^{f}$ | $1386^{b}$ | $342^{b}$ | $4.05^{a}$ |
| **INT** | $1.52^{e}$ | $3.85^{a}$ | $2.86^{a}$ | $1205^{e}$ | $403^{a}$ | $3.00^{c}$ |
| **Ilyz50** | $2.58^{c}$ | $2.88^{c}$ | $2.55^{b}$ | $1300^{d}$ | $357^{b}$ | $3.65^{b}$ |
| **Ilyz100** | $3.08^{ab}$ | $2.58^{c}$ | $1.75^{d}$ | $1341^{c}$ | $336^{b}$ | $4.00^{a}$ |
| **IANTI** | $2.85^{b}$ | $2.08^{d}$ | $1.09^{e}$ | $1310^{cd}$ | $340^{b}$ | $3.86^{ab}$ |
| **SEM** | 0.225 | 0.246 | 0.225 | 25.86 | 6.271 | 0.079 |
| **p-value** | 0.0001 | 0.0001 | 0.0001 | 0.0001 | 0.001 | 0.0001 |

*$^{a-e}$Means with different superscripts in a column differ significantly (P < 0.05). *SEM =Standard Error of the mean; NLyz50: Non infected treated with lysozyom (50mg). NLyz100: Non infected treated with lysozyom (100mg). NANTI: Non infected treated with antibiotic. INT: Infected with E. coli non treated group. Ilyz50: infected treated with lysozyom (50mg). Ilyz100: infected treated with lysozyom (100mg). IANTI: Infected with E. coli and treated with antibiotic. Lactob: spp. E. coli: Escherichia coli. Clost: Clostridium.*

***Blood biochemical parameters:***

Table 4 summarizes data indicating the impact of dietary treatments on serum uric acid, creatinine, ALT, and AST at 35 days of age. Serum levels of creatinine, uric acid, ALT, and AST in the E. coli-infected group that wasn't treated increased significantly at the end of the experimental stage as compared with the control group. In contrast, lysozyme-treated E. coli infection groups demonstrated a significant diminution in serum creatinine, uric acid, ALT, and AST when compared with the untreated E. coli infection treatment. Regarding antioxidant measures, comparing the E. coli-infected non-treated group to the control treatment, there was a substantial drop in TAC activity and a rise in TBARS concentration. At 35 days of age, the E. coli-infected chicks treated with lysozyme and antibiotics showed a significant drop in TBARS concentration and a rise in TAC activity (4).

**Table (4): Effect of experimental treatments on some blood constituents**

| Treatments | Liver function | | Kidney function | | Oxidative status | | Phagocytic activates | |
|---|---|---|---|---|---|---|---|---|
| | AST U/L | ALT U/L | Uric acid mg/dl | Creat. mg/dl | MDA nmol/ml | TAC mmol/l | Phagocytic (%) | Phagocytic index |
| **Control** | $182^{c}$ | $22.0^{bc}$ | $3.60^{c}$ | $0.260^{b}$ | $1.05^{bc}$ | $1.47^{b}$ | $69.8^{a}$ | $6.08^{ab}$ |
| **NLyz50** | $180^{c}$ | $23.0^{bc}$ | $3.62^{c}$ | $0.280^{b}$ | $1.00^{bc}$ | $1.55^{ab}$ | $70.22^{a}$ | $6.58^{ab}$ |
| **NLyz100** | $181^{c}$ | $21.0^{c}$ | $3.45^{c}$ | $0.260^{b}$ | $0.80^{c}$ | $1.78^{a}$ | $71.05^{a}$ | $7.05^{a}$ |
| **NANTI** | $186^{c}$ | $22.6^{bc}$ | $3.72^{c}$ | $0.270^{b}$ | $1.00^{bc}$ | $1.50^{b}$ | $70.31^{a}$ | $6.09^{ab}$ |
| **INT** | $230^{a}$ | $31.0^{a}$ | $6.50^{a}$ | $0.380^{a}$ | $1.98^{a}$ | $1.07^{c}$ | $38.65^{d}$ | $3.45^{c}$ |
| **Ilyz50** | $215^{ab}$ | $26.1^{b}$ | $5.76^{ab}$ | $0.280^{b}$ | $1.40^{b}$ | $1.36^{b}$ | $50.66^{c}$ | $4.98^{bc}$ |
| **Ilyz100** | $190^{c}$ | $24.0^{bc}$ | $4.05^{c}$ | $0.263^{b}$ | $1.18^{bc}$ | $1.58^{ab}$ | $62.55^{b}$ | $5.76^{ab}$ |
| **IANTI** | $206^{b}$ | $25.0^{bc}$ | $4.80^{bc}$ | $0.290^{b}$ | $1.30^{bc}$ | $1.42^{b}$ | $50.75^{c}$ | $5.00^{bc}$ |
| **SEM** | 3.924 | 0.733 | 0.258 | 0.008 | 0.084 | 0.046 | 2.471 | 0.279 |
| **p-value** | 0.0001 | 0.003 | 0.001 | 0.0001 | 0.003 | 0.001 | 0.0001 | 0.014 |

*$^{a-e}$Means with different superscripts in a column differ significantly (P < 0.05). *SEM =Standard Error of the mean; NLyz50: Non infected treated with lysozyom (50mg). NLyz100: Non infected treated with lysozyom (100mg). NANTI: Non infected treated with antibiotic. INT: Infected with E. coli non treated group. Ilyz50: infected treated with lysozyom (50mg). Ilyz100: infected treated with lysozyom (100mg). IANTI: Infected with E. coli and treated with antibiotic.*

In terms of phagocytic activity, non-infected groups treated with lysozyme and antibiotics considerably outperformed the control group in characterizes of phagocytic percent and phagocytic

index. While they significantly decreased in the E. coli-infected treatment that was not treated compared with the control treatment. When compared with the infected E. coli non-treated group, the E. coli infection groups that received lysozyme and antibiotic treatment showed a significant rise in phagocytic index and phagocytic percent, as shown in Table (4).

***Internal organ weights:***

Thymus and bursa weight significantly decreased in infected broilers compared to non-infected broilers (Table 5). However, compared to non-infected broilers, the relative weight of the spleen, heart, and liver was greater in the infected broilers (Table 5). On the other hand, the lysozyme and antibiotic restored the negative effects of infection with E. coli on organ weights.

***Hemagglutination inhibition (HI) test:***

When compared with the control group, the E. coli-infected treatment that was not treated had a considerably lower antibody titer against NDV. When compared with the infected E. coli non-treated group, the E. coli infection groups that received lysozyme and antibiotic treatment showed a significant rise in antibody titer against NDV, as shown in Table (5).

**Table (5): Effect of experimental treatments on carcass traits and lymphoid organs**

| Treatments | Carcass traits % | | | Lymphoid organs weights % | | | Immune response |
|---|---|---|---|---|---|---|---|
| | Dressing | Liver | Heart | Spleen | Bursa | Thymus | NDV |
| **Control** | 70.00$^{a}$ | 2.37$^{c}$ | 0.43$^{b}$ | 0.11$^{c}$ | 0.16$^{a}$ | 0.38$^{a}$ | 3.8$^{c}$ |
| **NLyz50** | 70.50$^{a}$ | 2.36$^{c}$ | 0.42$^{b}$ | 0.12$^{c}$ | 0.15$^{ab}$ | 0.37$^{a}$ | 4.2$^{b}$ |
| **NLyz100** | 71.60$^{a}$ | 2.33$^{c}$ | 0.42$^{b}$ | 0.13$^{c}$ | 0.17$^{a}$ | 0.40$^{a}$ | 5.6$^{a}$ |
| **NANTI** | 70.80$^{a}$ | 2.40$^{c}$ | 0.43$^{b}$ | 0.12$^{c}$ | 0.16$^{a}$ | 0.39$^{a}$ | 5.2$^{a}$ |
| **INT** | 68.55$^{b}$ | 3.16$^{a}$ | 0.50$^{a}$ | 0.22$^{a}$ | 0.11$^{c}$ | 0.30$^{b}$ | 3.0$^{d}$ |
| **Ilyz50** | 69.00$^{ab}$ | 2.65$^{b}$ | 0.42$^{b}$ | 0.16$^{b}$ | 0.16$^{a}$ | 0.35$^{ab}$ | 3.5$^{c}$ |
| **Ilyz100** | 70.05$^{a}$ | 2.51$^{bc}$ | 0.43$^{b}$ | 0.14$^{bc}$ | 0.17$^{a}$ | 0.36$^{ab}$ | 4.8$^{ab}$ |
| **IANTI** | 69.25$^{a}$ | 2.63$^{b}$ | 0.42$^{b}$ | 0.15$^{b}$ | 0.14$^{b}$ | 0.36$^{ab}$ | 4.2$^{b}$ |
| **SEM** | 3.65 | 0.11 | 0.02 | 0.01 | 0.02 | 0.04 | 0.136 |
| **p-value** | 0.015 | 0.002 | 0.045 | 0.025 | 0.005 | 0.041 | 0.004 |

*$^{a-e}$Means with different superscripts in a column differ significantly (P < 0.05). *SEM =Standard Error of the mean; NLyz50: Non infected treated with lysozyom (50mg). NLyz100: Non infected treated with lysozyom (100mg). NANTI: Non infected treated with antibiotic. INT: Infected with E. coli non treated group. Ilyz50: infected treated with lysozyom (50mg). Ilyz100: infected treated with lysozyom (100mg). IANTI: Infected with E. coli and treated with antibiotic.*

## DISCUSSION

***Growth performance:***

According to Remus *et al.* (2014), E. coli serotypes could have been the major and secondary reasons for feed intake rejection, which in turn caused a drop in body weight (Shah et al., 2004). Second, the synthesis of acute phase proteins, proteolysis, and gluconeogenesis might have been triggered by the release of interleukin 6 and C reactive protein in response to pathogen invasions. These subsequently caused the chicks' feed consumption to drop, which in turn caused their growth to slow (Bodell *et al.*, 2009). Third, slow feed transit may have been a result of the corticosterone produced by microbial stress (Hu and Guo, 2008), which may have also been a factor in the increased proteolysis and glucogenesis as well as the decreased feed intake in chicks (Lin *et al.,* 2004). Fourth, malabsorption may have occurred as a result of the chicks' decreased villi height and VH:CD ratio, which may be what caused the decline in ADWG (Table 3). Our findings support those of Huff *et al.* (2002), who exhibited that an E. coli infection dramatically reduced avian body weight. By comparison to the control group, the chicks exposed to E. coli had lower body weights, according to Shah *et al.* (2004). Lysozyme and antibiotic dietary inclusion during trial phases enhanced ADFI and ADWG, which improved FCR levels in their chicks. According to Miles *et al.* (2006), adding virginiamycin to a diet consisting of maize and soy meal encouraged an increase in the body weight of male and female broiler chicks as well as the proportion of absorptive cells per unit length in their intestines. Better nutrition absorption is made possible by this physical change, which supports performance stimulation.

Lysozyme's beneficial effects on growth performance in the present study may be attributable to better gut antioxidant and antibacterial activities as well as improved nutritional digestibility in chick's gut, which may be related to enhanced nutrient absorption in the intestine (Remus *et al.,* 2014). Additionally, the stronger immunological responses provided by the advantageous microbiota may be responsible for the higher growth performance.

Chicks with E. coli O78:K80 infection were treated with antibiotics, which improved FCR levels while also raising DFI and DWG levels. In terms of BWG, FCR, and survival rate, antibiotic therapy has been shown to be beneficial for broiler hens exposed to Clostridium spp (Sen *et al.,* 2012).

Mortality in infected group with E. coli was 10%. This result was in agreement with that published by Shen *et al.* (2002), who said that E. coli causes mortality rates of 8%. No mortality was noted in those that were infected or given Ca Fosfomycin treatment. This finding validates the use of antibiotics to manage E. coli infection.

***Intestinal microbial count:***

The findings exhibited that lysozyme had a significant impact on the overall amount of Clostridium spp, E. coli bacteria and Lactobacillus bacteria irrespective of the lysozyme dosage. Our findings concur with those of Ibrahim *et al.* (2011) who stated that lysozyme plays as protector against bacteria. They added that, the pathogenic bacterial cell wall's peptidoglycan is hydrolyzed by lysozyme as an antibiotic agent. When broilers were gavaged with Clostridium perfringens, Liu *et al.* (2010) discovered that 40 mg lysozyme efficiently decreased the amount of Clostridium pp in the ileum and inhibited intestinal injuries. Lysozyme can successfully aid broiler chickens in coping with Eimeria infection so that coccidiosis is avoided, as demonstrated by Sotirov and Koinarski (2003).

According to reports, E. coli levels in birds fed antibiotics to stimulate growth have significantly decreased (Rahimi *et al.,* 2011). Salmonella and E. coli load in the digesta are reduced by the antimicrobial agent bacitracin methylene disalicylate, which also drives them toward the mucosal surface (Ghosh *et al.,* 2012).

***Gut morphological:***

Concerning to intestinal morphology**,** E. coli infection significantly diminished the VH:CD ratio and the height of the jejunal villi, although it significantly raised the depth of the jejunal crypt. The findings of Wang *et al.* (2017), who obtained that broilers exposed to E. coli had smaller villi heights and lower VH:CD ratios than control chicks, are in keeping with the current results. Zhang *et al.* (2016) assert that an E. coli K88 challenge changed intestinal structure. Although**,** Miles *et al.* (2006) asserted that an enhancement in intestinal morphology encourages greater feed absorption, this leads to less energy being needed for tissue maintenance, which can then be employed for growth or better nutritional absorption.

Dietary lysozyme supplementation raised the VH:CD ratio and villi height in the jejunum. A larger VH:CD ratio indicates slower tissue turnover, which lowers feed required to make up for villi deteriorate or injury brought on by pathogen-induced irritation (Mahdavi *et al.,* 2010). Lysozyme's positive impact on gut shape may be due to altered intestinal microbiota. Previous research supported the positive impact of lysozyme on gut microflora. Additionally, ingestion of lysozyme milk led to a rise in the number of Bifidobacteriaceae and Lactobacillaceae, both of which have been shown to be advantageous for the development of the digestive tract (Maga *et al.,* 2012). According to findings from earlier studies, adding antibiotics to meals promotes the growth of cells that absorb nutrients in the intestine. In the ileum and jejunum after antibiotic therapy, we observed increased villus height and crypt depth (Xia *et al.,* 2004).

***Blood biochemical parameters:***

The infected non-treated treatment exhibited a statistically significant rise in the serum activity of ALT and AST when compared to the control treatment. These findings support those of Abd-Allah *et al.* (2018). Higher hepatic enzyme activity may arise from the microorganism altering the hepatocytes' membrane permeability, which causes the cell membrane to lose its functional integrity and spill these enzymes into circulation (Gahalain *et al.,* 2011). Comparing the E. coli-treated group to the infected non-treated group, the lysozyme-treated group showed a substantial reduction in serum AST and ALT values. According to the research, lysozyme is an excellent source of antioxidants that can protect cells from free radicals, reduce toxicity, and possibly even safeguard liver health by preventing liver damage.

The fact that uric acid and creatinine levels in the lysozyme-treated groups did not significantly differ from those in the control, healthy group showed that this treatment was safe for the kidneys. Ibrahim and Banaee (2014) claim that using lysozyme on the kidney is safe. According to Abdelhady and El-Abasy (2015), the E. coli-infected non-treated group has higher serum renal function parameters

than the other groups. This difference could be attributed to renal injury, an imbalance in protein metabolism, or an imbalance in amino acid concentration. These findings concur with those of Abd El-Ghany and Ismail (2014) and may be related to the kidney's reaction to the bacteria and their toxin (Abd-Allah *et al.,* 2018). When compared with the infected non-treated treatment, the lysozyme-treated infected group demonstrated a considerably lower level of serum uric acid and serum creatinine.

The infected non-treated group exhibited a noticeably greater level of TBARS and a noticeably lower concentration of TAC as compared to the control group. Zheng *et al.* (2016) found similar outcomes in broiler chickens experimentally infected with lipopolysaccharide (LPS) of E. Coli O55: B5. They observed a drop in GPx activity and an increase in serum MDA. The reason for these outcomes can be attributed to the fact that LPS generates large amounts of reactive oxygen species (ROS), which can cause oxidative damage to cells and tissues by upsetting the delicate balance between the pro-oxidant and antioxidant systems. On the other hand, serum TAC activity significantly decreased in E. coli-infected non-treated chickens, which is exactly in line with the findings of Kumari *et al.* (2020), they found that an E. Coli infection lowered the antioxidant defense system. The lysozyme-treated E. coli-infected groups (groups 6 and 7) demonstrated a significant decline in TBARS and a significant rise in TAC when compared to the E. coli-infected non-treated group (group 5). These results are in harmony with those of Mezbani *et al.* (2019), they discovered that adding lysozyme (100 mg/kg) to broiler diet caused MDA levels to significantly decrease and CAT and GPx activities to significantly rise when compared to control birds.

Furthermore, this investigation showed that the birds treated with lysozyme (50 and 100 mg/kg) and the antibiotic showed a significant increase in both the phagocytic index and phagocytic percentage. These findings are in line with a previous study (Ali *et al.,* 2018), which found that broiler chicks' phagocytic activity and phagocytic index could be increased by adding lysozyme to the feed in comparison to the control group. In contrast, the phagocytic index and phagocytic percentage of the E. coli-challenged birds showed a significant decline after 35 days of age when compared to the control group. These outcomes were consistent with those of an earlier investigation (Abd El-Tawab *et al.,* 2015), which linked these outcomes to pathophysiological effects of bacterial endotoxins and the suppression of antibacterial defense mechanisms. These outcomes could also be linked to the immune system's weariness from an E. coli infection (Lee *et al.,* 2015).

***Internal organ weights:***

A vital part of the chicken immune system is the bursa. Furthermore, the morphological reaction to the immunological state in broilers is represented by the bursal weight, as stated by Manafi *et al.* (2016). The current study's findings were supported by Gottardo *et al.* (2017), who also found that the infected group's thymus and bursa had considerably smaller relative weights. Nevertheless, these results are at odds with those of Manafi *et al.* (2016). The relative weight of the spleens in the infected group increased dramatically on day 35. These results might be connected to the infection, as they were supported by Huff *et al.* (1998) findings in turkeys. However, in the Lysozyme groups, the spleen's relative weight was significantly lower and the thymus and bursa's relative weights were significantly higher. These results agree with those of Sohail *et al.* (2013). Dysfunction of lymphoid organs reduces immunity against bacterial, viral, parasitic, and fungal infections. Because of this, birds are more susceptible to a variety of diseases, which raises mortality and morbidity rates and results in large financial losses (Wan *et al.,* 2017).

The weight of the bursa and thymus significantly increased in non-infected broilers compared to infected broilers (Table 5). The findings supported those made by Sadeghi *et al.* (2013), who asserted that a salmonella exposure diminished the growth of the lymphoid organs. Low bursa weight may be used as an indicator of low immunological activity because the bursa is a significant lymphoid organ in birds and immune tissue weight affects immune cell morphologies, immune cell proliferation, and antibody generation (Ghaderi-Joybari *et al.,* 2014). Immunosuppression in poultry is typically indicated by clinically obvious E. coli infection (Mc Gruder and Moore, 1998). The chickens' immune systems were harmed by E. coli infection, which resulted in lymphocyte diminution in both the thymus and bursa (Nakamura *et al.,* 1990).

The relative liver and heart weights of infected broilers were higher in the current study than those of non-infected broilers. Similar conclusions were reached by Abalaka *et al.* (2017), who found that broiler hens with E. coli infections had larger livers. Furthermore, Rocha *et al.* (2013) found that broiler chicks exposed to *Salmonella typhimurium* had an increased liver relative weight. The enlarged relative weight of the liver is thought to be due to the necrotic lesions and inflammation caused by E. coli infections (Abalaka *et al.,* 2017). This supports research by Fernandez *et al.* (2002), who found that broiler chicks exposed to E. coli O78:K80 had increased relative weights of the liver, heart, and air sacs.

Similar to this, adding lysozyme to the diet seems to increase the relative carcass weights while decreasing the weight of the liver**,** pancreas, and heart. In line with our findings, Sarica *et al.* (2005) found no differences in the liver and pancreatic weight of broiler chicks fed antibiotic-added diets.

***HI titer:***

The stress of infection and diarrhoea, which may alter the acid-base balance, may be to blame for the decrease in HI titer in the E. coli-infected groups (Sil *et al.,* 2002). Clinically**,** clear Immunosuppression in birds is frequently indicated by E. coli infection (Mc Gruder and Moore, 1998). E. coli infection compromised the chickens' immune systems and reduced the number of lymphocytes in their thymus and bursa (Nakamura *et al.,* 1990). Both innate and adaptive host immune responses are influenced by immune-regulating peptides known as cytokines (Lee *et al.,* 2015). Sadeyen *et al.* (2014) reported that compared to control birds, birds exposed to the E. coli-O78-H9 strain exhibited increased levels of IL-1β, IFNγ, and IL-10. This could be as a result of phagocytic cells in the innate immune system being stimulated by an E. Coli infection, which results in the release of IL-10. Lysozyme-fed groups had higher antibody titers against NDV vaccination, which may indicate that the bird's immune system and antioxidant defences had improved. These results back up those by Fritz *et al.* (2009). Regarding the antibiotic, Perez (2014) corroborated this conclusion by pointing out that fosfomycin had an immunomodulatory effect and encouraged phagocytosis.

## CONCLUSIONS

The results of this study showed that the experimental infection of chicks with E. coli caused notable alterations in the gut's shape, as well as in antioxidant, metabolic, immunological, and immune markers. It's interesting to note that giving non-infected bird group 100 mg/kg of lysozyme increased immune response, enhanced bodily function, and increased poultry production efficiency. Based on the information provided above, lysozyme (100 mg/kg) may be a promising supplement to prevent E. coli infection by improving growth performance and returning the aforementioned parameters to values that are near normal.

## REFERENCES

Abalaka, S. E., Sani, N. A., Idoko, I. S., Tenuche, O. Z., Oyelowo, F. O., Ejeh, S. A. and Enem, S. I. (2017). Pathological changes associated with an outbreak of colibacillosis in a commercial broiler flock. Sokoto Journal of Veterinary Sciences, 15, 95–102.

Abd-Allah, N. A., Isamail, S. A., Hassan, W. M. and Kaser, A. N. (2018). Antibacterial Activity of Ceftiofur Sodium and Garlic in Escherichia coli Infected Chickens Zagazig Vet. J., 43, pp. 81-97

Abd El-Ghany, W.A., and Ismail, M. (2014). Tackling experimental colisepticaemia in broiler chickens using phytobiotic essential oils and antibiotic alone or in combination. Iran. J. Vet. Res. 15, 110–115.

Abdelhady, D.H. and El-Abasy, M.A. (2015). Effect of prebiotic and probiotic on growth, immuno-hematological responses and biochemical parameters of infected rabbits with *Pasteurella multocida*. Benha Veterinary Medical Journal, 28(2):40–51.

Abo El-magd, E. E., Sallam, k., Abd El-Ghany, S. M. and Ramadan, H. H. (2019). Prevalence of Escherichia coli in chicken carcasses from Mansoura, Egypt. Mansoura Veterinary Medical Journal 20:141-44.

Abd El- Tawab, A. A., Ahmed, M. A., Soad A. Nasef, Fatma, I. El- Hofya and Nehal, M. Nabil. (2015). Molecular studies on antimicrobial resistance genes in salmonella isolated from poultry flocks in Egypt. Benha Veterinary Medical Journal, Vol. 28, NO. 2:176-187.

Ali, I. A., El Nabarawy, E.A., Salah, A.B. and Hassan, A.A. (2018). Effect of Apramycin On Pathological, Hematological and Biochemical Changes in Turkey Infected with Coli-Bacillosis. Zagazig Vet. J., 41, 124-136

AOAC, 2000. Official method of Analytical Chemists. 16th Ed. Arlington, VA, USA.

Biggar, W. D. and Sturgess, J. M. (1977). Role of lysozyme in the microbicidal activity of rat alveolar macrophages. Infect. Immun. 16: 974-982.

Bodell, P. W., Kodesh, E., Haddad, F., Zaldivar, F. P., Cooper, D. M., and Adams, G. R. (2009). Skeletal muscle growth in young rats is inhibited by chronic exposure to IL-6 but preserved by concurrent voluntary endurance exercise. Journal of Applied Physiology, 106 (2), 443-453.

Collins, C.H. and Lyne, P. M. (1970). Microbiological Methods 3rd ed. London: Hodder Headline Group (1970).

Duncan, D.B. (1955). Multiple range and multiple F-Test. Biometrics, 11:1-42.

Fernandez, A., Lara, C., Loste, A. and Marca, M.C. (2002). Efficacy of calcium fosfomycin for the treatment of experimental infection of broiler chickens with *Escherichia coli* O78:K80. Veterinary Research Communications. 26:427–436.

Fritz. J., Ikegami, M. and Weaver, T. (2009). Lysozyme ameliorates oxidant-induced lung injury. Am J Respir Crit Care. 179: A4005.

Gahalain, W., Wells, J. and Maxwell, C. (2014). Lysozyme as an alternative to antibiotics improves performance in nursery pigs during an indirect immune challenge. J Anim Sci. 92:4927–34.

Ghaderi-Joybari, M.G., Sadeghi, A.A., Jouzani, G.S., Chamani, M. and Aminafshr, M. (2014). Immune tissue development in pathogen challenged broiler chicks fed diet supplemented with probiotic (Bacillus subtilis). International journal of Biosciences, 5(12):197-203.

Ghosh, T. K., Haldar, S., Bedford, M. R., Muthusami, N. and Samanta, I. (2012). Assessment of yeast cell wall as replacements for antibiotic growth promoters in broiler diets: effects on performance, intestinal histo-morphology and humoral immune responses. Journal of Animal Physiology and Animal Nutrition 96:275-284.

Goddeeris, B. M., Boldwin, C. L., Ole Moiyoio and Morrison, W. L. (1986): Improved method for purification and depletion of monocytes from bovine peripheral blood mononuclear cells. Functional evaluation of monocytes in response to Lectins. Imm. Meth. J., 89, 2, 165-173.

Gottardo Elisangela T., Álvaro M., Burin Junior, Bruna V., Lemke Alexandra M., Silva, Cassiano L., Busatta Pasa, Jovanir I. and Muller Fernandes. (2017). Immune response in Eimeria sp. and E. coli challenged broilers supplemented with amino acids. Austral J Vet Sci 49, 175-184

Grossowicz, N. and Ariel, M. (1983). Methods for determination of lysozyme activity. Methods Biochem. Anal. 29: 435–446.

Hashem, M.A., Neamat-Allah, A. N., Hammza, H. E. and Abou-Elnaga H. M. (2022). Impact of dietary supplementation with Echinacea purpurea on growth performance, immunological, biochemical, and pathological finding in broiler chickens infected by pathogenic E. coli Tropical Animal Health Prod., 52 , pp. 1599-1607

Hu, X. F. and Guo Y. M. (2008). Corticosterone administration alters small intestinal morphology and function of broiler chickens. Asian-Australas J. Anim. Sci., 21 1773 -1778

Huff, G. R., Huff, W.E., Balog, J. M. and Rath N.C. (1998). The effects of dexamethasone immunosuppression on turkey osteomyelitis complex in an experimental Escherichia coli respiratory infection Poultry Sci., 77, pp. 654-661

Huff, W. E., Huff G. R., Rath N. C., Balog J. M., Xie H., Moore P. A. and Donaghue A.M. (2002). Prevention of *Escherichia coli* infection in broiler chickens with a bacteriophage (SPRO2) Poult. Sci., 81:437–441.

Ibrahim, A.T.A. and Banaee, M. (2014). Ameliorative effect of lycopene and vitamin E on some haematological and biochemical parameters of *Oreochromis Niloticus* against diazinon toxicity. J MedCrave Advance in Plants Agriculture Research. 1: 1-9.

Ibrahim, H.R., Imazato, K. and Ono, H. (2011). Human lysozyme possesses novel antimicrobial peptides within its N-terminal domain that target bacterial respiration. J Agric Food Chem. 59:10336–45.

Ibrahim, H. R., Higashiguchi, S., Koketsu, M., Juneja, L. R., Kim, M., Yamanoto, T., Sugimoto, Y. and Aoki, T. (1996). Partially unfolded lysozyme at neutral pH agglutinates and kills Gram-negative and Gram-positive bacteria through membrane damage mechanism. J. Agric. Food Chem. 44: 3799-3806.

King, D. J. and Seal, B.S. (1998). "Biological and Molecular characterization of Newcastle disease virus (NDV) field isolates with comparisons of reference NDV strains and pathogenicity chicken or embryo passage of selected isolates." Avian Dis., 42:507-516.

Kumari, Mamta, Rajendar P. Gupta, Deepika Lather, Preeti Bagri (2020). Ameliorating effect of Withania somnifera root extract in Escherichia coli-infected broilers. Poultry science vol. 99,4.

Lee, K. W., Kim, D. K., Lillehoj, H. S., Jang, S. I. and Lee, S. H. (2015): Immune modulation by Bacillus subtilis –based direct-fed microbials in commercial broiler chickens. Animal Feed Science and Technology 200, 76–85.

Lin, H., Decuypere E. and Buyse. J. (2004). Oxidative stress induced by corticosterone administration in broiler chickens (Gallus gallus domesticus): 2. Short-term effect. Comp. Physiol. B Biochem. Mol. Biol. 139:745-751.

Liu, D., Guo, Y., Wang, Z. and Yuan, J. (2010). Exogenous lysozyme influence Clostridium perfringens colonization and intestinal barrier function in broiler chickens. Avian Pathol. 39: 17-24.

Maga, E. A., Desai, P. T., Weimer, B. C., Dao, N., Kultz, D. and Murray, J. D. (2012). Consumption of lysozyme-rich milk can alter microbial fecal populations. Appl Environ Microbiol., 78:6153–60.

Mahdavi, G., Darius, S., Smith, S. R. and Ritchie, S. J. (2006). In vitro inhibitory effect of hen egg white lysozyme on Clostridium perfringens type A associated with broiler necrotic enteritis and its α-toxin production. Lett. Appl. Microbiol. 42:138–143.

Manafi, M., Hedayati, M., Khalaji, S. and Kamely, M. (2016). Assessment of a natural, non-antibiotic blend on performance, blood biochemistry, intestinal microflora, and morphology of broilers challenged with Escherichia coli. *Revista Brasileira de Zootecnia*, 45, pp. 745-754

Macfaddin, T. F. (1980): Biochemocal tests for identification of medical bacteriology. 2nd Ed. Williams and Wilkins Company, Baltimore, U.S.A.

Mc Gruder, E.D. and Moore, G.M. (1998). Use of lipopolysaccharide (Lps) as a positive control for the evaluation of immunocompetentiating drugs candidates in experimental avian colibacillosis models. Research of Vet. Sci., 66: .33-37.

Mezbani, T., Masani, K., Sato, C., Hiki, M., Usui, M., Baba, K., Ozawa, M., Harada, K., Aoki, H. and Sawada, T. (2019). Phylogenetic groups and cephalosporin resistance genes of Escherichia coli from diseased food-producing animals in Japan. Acta Veterinaria Scandvica, 53-52.

Miles, R.D., Butcher, G.D., Henry, P.R. and Littell, R.C. (2006). Effect of antibiotic growth promoters on broiler performance, intestinal growth parameters, and quantitative morphology. Poultry Science 85, 476-485.

Nakamura, K., Yuasa, N., Abe, H. and Narita, M. (1990). Effect of Infectious bursal disease virus on infections produced by Escherichia coli of high and low virulence in chickens. Avian Pathol., 19: 713- 721.

NRC. (1994). Nutrient Requirements of Poultry. Washington, D.C.: National Academies Press; 1994.

Peighambari, S. M., Julian, R.J. and Gyles, C. L. (2000): Experimental Escherichia coli respiratory infection in broilers. Avian Diseases 44, 759-769.

Perez, D.S., Tapia, M.O. and Soraci, A.L. (2014). Fosfomycin: uses potentialities in veterinary medicine, open vet. J., 4(1): 26-43

Phillips, D.C. (1996). The three-dimensional structure of an enzyme molecule. Scientific American, 215, 78-79.

Rahimi, S., Teymori Zadeh, Z., Torshizi, K., Omidbaigi, R. and Rokni, H. (2011). Effect of the three herbal extracts on growth performance, immune system, blood factors and intestinal selected bacterial population in broiler chickens. Journal of Agricultural Science and Technology 13:527-553.

Remus, A., Hauschild, L., Andretta, I., Kipper, M., Lehnen, C. R. and Sakomura, N.K. (2014). A meta-analysis of the feed intake and growth performance of broiler chickens challenged by bacteria. Poult. Sci. 2014, 93, 1149–1158.

Rocha, T. M., Andrade, M. A., Gonzales, E., Stringhini, J. H., Santana, E. S., Pôrto, R. N. and Minafra-Rezende, C. S. (2013). Liver function and bacteriology of organs in broiler inoculated with nalidixic acid-resistant *Salmonella* Typhimurium and treated with organic acids. Italian Journal of Animal Science, 12: e55.

SAS Institute. (2003). SAS User's Guide: Statistics. Version 9.03. SAS Inst. Inc., Cary, NC.

Sadeghi, A.A., mohammadi, A., Shawrang, P. and Aminafshar, M. (2013). Immune responses to dietary inclusion of prebiotic-based mannanoligosaccharide and β-glucan in broiler chicks challenged with Salmonella Enteritidis. Turk. J. Vet. Anim. Sci., 37: 206-213.

Sadeyen, J.R., Kaiser, P., Stevens, M. P. and Dziva, F. (2014): Analysis of immune responses induced by avian pathogenic Escherichia coli infection in turkeys and their association with resistance to homologous re-challenge. Veterinary Research 45(1), 19.

Sarica, S., Ciftci, A., Demir, E., Kilinc, K. and Yildirim, Y. (2005). Use of an antibiotic growth promoter and two herbal natural feed additives with and without exogenous enzymes in wheat based broiler diets. S. Afr. J. Anim. Sci., 35: 61-72.

Sen, S., Ingale, S., Kim, Y., Kim, J., Kim, K., Lohakare, J., Kim, E., Kim, H., Ryu, M. and Kwon, I. (2012). Effect of supplementation of Bacillus subtilis LS 1-2 to broiler diets on growth performance, nutrient retention, caecal microbiology and small intestinal morphology. Res. Vet. Sci. 93, 264–268.

Shah, M.S.D., Khan, S.A., Aslam, A., Rabbani, M., Khan, K.A. and Rai M.F. (2004). Effect of experimental yolk sac infection with Escherichia coli on immune status of broiler chicks. Pakistan Veterinary Journal, 24: 125-128.

Shen, J., Hu, D., Wu, X. and Coats, J. R. (2002). Bioavailability and pharmacokinetics of florfenicol in broiler chickens. J Vet Pharmacol Ther., 26:337–341.

Sil, G., Das, P., Islam, M. and Rahman, M. (2002). Management and disease problems of cockrels in some farms of Mymensingh, Bangladesh. Int. J. Poult. Sci., 1:102–105.

Sohail, M., Ijaz, A., Younus, M., Shabbir, M., Kamran, Z., Ahmad, S., Anwar, H., Yousaf, M., Ashraf, K. and Shahzad, A. (2013). Effect of supplementation of mannan oligosaccharide and probiotic on growth performance, relative weights of viscera, and population of selected intestinal bacteria in cyclic heat-stressed broilers. J. Appl. Poult. Res., 22:485–491.

Sotirov, L. and Koinarski, V. (2003). Lysozyme and complement activities in broiler chickens with coccidiosis. Revue Méd. Vét. 154: 780-784.

Wan, A., Kim, K., Lohakare, J., Kim, E., Kim, H., Ryu, M. and Kwon, I. (2017). Therapeutic potential of different commercially available synbiotic on acetaminophen-induced uremic rats Clin. Exp. Nephrol., 19, pp. 168-177

Wang, S., Peng, Q., Jia, H.M., Zeng, X.F., Zhu, J.L., Hou, C.L., Liu, X.T., Yang, F.J. and Qiao, S.Y. (2017). Prevention of Escherichia coli infection in broiler chickens with Lactobacillus plantarum B1. Poultry Sci. 96, 2576–2586.

Xia, M.S., Hu, C.H. and Xu, Z. R. 2004. Effects of copper-bearing montmorillonite on growth performance, digestive enzyme activities, and intestinal microflora and morphology of male broilers. Poultry Science 83, 1868-1875.

Zhang, L., Zhang, L., Zhan, X., Zeng, X., Zhou, L., Cao, G., Chen, A. and Yang, C. (2016). Effects of dietary supplementation of probiotic, Clostridium butyricum, on growth performance, immune response, intestinal barrier function, and digestive enzyme activity in broiler chickens challenged with Escherichia coli K88. J Anim Sci Biotechnol 7:3

Zheng, X. C., Wu, Q. J., Song, Z. H., Zhang, H., Zhang, J. F., Zhang, L. L., Zhang, T. Y., Wang, C. and Wang, T. (2016): Effects of Oridonin on growth performance and oxidative stress in broilers challenged with lipopolysaccharide. Poultry Science, 1–9.

# تأثير الليزوزيم الغذائي على أداء النمو ومكونات الدم وشكل الأمعاء ومقاومة بكتيريا الإشريكية القولونية في كتاكيت اللحم

**رضا على حسن[1]*، يحيى زكريا عيد[2]، زينب محمد فاروق[1]، محمود الجبالي[1]و محمد عبد العظيم محمد موسى[1]**

***[1]معهد بحوث الإنتاج الحيواني، مركز البحوث الزراعية، الدقي، الجيزة، مصر.***

***[2]قسم إنتاج الدواجن، كلية الزراعة، جامعة كفر الشيخ، مصر.***

****عنوان البريد الإلكتروني (redaalihasan@yahoo.com)***

تعتبر الإشريكية القولونية أحد الأسباب الرئيسية لتكبد قطاع الدجاج خسائر مالية كبيرة، وإضافة الليزوزيم في النظام الغذائي للوقاية من داء العصيات القولونية لدى الطيور ليست مفهومة جيدًا. لذلك كان الغرض من الدراسة الحالية هو مقارنة تأثير الليزوزيم كأحد المكملات الغذائية مع العلاج بالمضادات الحيوية في كتاكيت اللحم المصابة بالإشريكية القولونية. تم إنشاء ثماني مجموعات مكونة من 600 فرخ روس غير مجنس عمر يوم واحد: المجموعة الأولى (الكنترول) لم تكن مصابة وتم إعطاؤها غذاء متوازن خالي من الإضافات، كما لم تصاب المجموعتان الثانية والثالثة وتم علاجها بـ 50 ملجم و 100 مجم من الليزوزيم على التوالي، المجموعة الرابعة كانت غير مصابة وعولجت بـ 10 ملجم من الأفيلامايسين / كجم علف، المجموعة الخامسة أصيبت بعدوى الإشريكية القولونية ولم تعالج، المجموعة السادسة والسابعة أصيبت وعولجت بـ 50 ملجم و 100 ملجم من الليزوزيم على التوالي، أما المجموعة الثامنة فقد أصيبت وعولجت بجرعة 10 ملجم من الأفيلامايسين/كغم علف. تم إعطاء العدوى بالإشريكية القولونية عن طريق الفم للكتاكيت عند عمر 5 و 16 يوم. وكانت الفترة التجريبية 35 يوما لجميع المجموعات المدروسة. أظهرت المجموعة المصابة بالإشريكية القولونية انخفاضًا في نشاط البلعمة، ونسبة التضميد، والوزن النسبي للغدة الصعترية والجراب، والقدرة الكلية لمضادات الأكسدة في الدم. كانت إنزيمات الكبد، واختبارات وظائف الكلى، ونواتج اكسده الليبدات، والأوزان النسبية للكبد والقلب والطحال، بالإضافة إلى التغيرات في شكل الأمعاء وتعداد الميكروبات المعوية، مرتفعة بشكل ملحوظ في نفس المجموعة. أظهر الكتاكيت المصابه والمعالجه بالليزوزيم تحسينات في مقاييس أداء الجسم بالإضافة إلى انخفاض ملحوظ في اختبارات إنزيمات الكلى والكبد. لاحظنا تحسينات كبيرة في مورفولوجيا الأمعاء، وتعداد الميكروبات المعوية، والإنزيمات المضادة للأكسدة، والمعلمات المناعية في الدم في المجموعات المعالجة بالليزوزيم. من الممكن أن نستنتج أن إضافة الليزوزيم (بجرعة 50 و 100 ملجم/كجم) إلى غذاء الدجاج التسمين يحسن جهاز المناعة والأداء ويقلل من الآفات المرضية التي تسببها عدوى الإشريكية القولونية.