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EFFECT OF PEDIOCOCCUS ACIDILACTI ON ENZYMATIC ANTIOXIDANT, PATHOLOGY AND BROILERS MEAT QUALITY UNDER SALMONELLA INFECTION

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ABSTRACT

One hundred and twenty five three days-old broilers chick were used in this study to evaluate growth performance of *S. enteritidis* colonization, antioxidant, lipid peroxidation, meat quality and histopathological findings of broilers chick supplemented with *Pediococus* and challenged with *Salmonella* infection. The broilers chick were divided into four groups, control group without infection, *Pediococcus* group without infection, group infected with salmonella and *Pediococcus* group with *Salmonella* infection. The results indicated that the use of *Pediococcus acidilactici* was without affect on the growth performance, improved antioxidant, decreased lipid peroxidation, improved quality of meat and decreased histopathological lesion of *Salmonella.*

Key words: *Pediococcus acidilacti, Broilers, Salmonella.*

INTRODUCTION

The vertebrate innate immune system protects the organism by producing reactive oxygen species (ROS) in a process called oxidative burst. These oxygen species are highly reactive and destroy pathogens by damaging their proteins, lipids and DNA. These reactive species are not pathogen-specific and can also damage host tissues if there are not enough protective antioxidants present (Sepp *et al.,* 2012). Also in numerous physiological and pathological states, the systemic amounts of free non-oxygen radicals and reactive oxygen species (ROS) are elevated. Lipid peroxidation is one of the best parameter used for indicating the level of ROS-induced systemic biological damage. One lipid peroxidation end-product, malondialdehyde (MDA), is isolated from urine, blood and tissues and is used as a biomarker for radical-induced damage. Chickens possess both enzymatic and non-enzymatic antioxidant mechanisms of defence that prevent ROS formation or limit their toxic effects. Superoxide dismutase (SOD) and catalase (CAT) are antioxidant enzymes involved in endogenous antioxidant defences against ROS (Georgieva *et al.,* 2006).

Lipids are an important component of meat and contribute to several desirable characteristics of meat and meat products. Lipids are important to enhance the flavour and aroma profile of meat and also increase the tenderness and juiciness of meat. However, it is generally accepted that lipid oxidation is the primary process responsible for quality deterioration of meat during storage (Weber and Antipatis 2001). Membrane lipids are protected against oxidative attack by naturally occurring antioxidants (Jensen *et al.,* 1995). The oxidative status of the feed given to animals has a significant influence on the final meat quality. Meat obtained from animals grown on oxidized feed has a significantly lower oxidative status compared to the animals fed on a feed of good oxidative status (Tesoriere *et al.,* 2002).

Reactive oxygen species (ROS) are implicated in several pathophysiological conditions. ROS are known to attack almost all molecules of the cells including membrane lipid peroxides (Koinarski *et al.,* 2006). Mathew *et al.* (2007) reported a concomitant increase in lipid peroxidation and a drop in antioxidant enzyme activities of SOD, CAT, Gpx and Glutathion-S- transferase in the digestive gland, muscle and haemolymph of P. monodon following infection with white spot syndrome virus (WSSV). Also, Mehta *et al.* (1998) reported a decreased endogenous intestinal protection against ROS in *S. typhimurium*-mediated infection, which could contribute to the pathogenesis of the disease.

Probiotics are harmless bacteria that help the well being of the host animal and contribute, directly or indirectly to protect the host animal against harmful

bacterial pathogens (Gatesoupe, 1999). Oral supplementation of Ped. acidilactici lactic acid bacteria modulates intestinal bacterial communities in on-growing tilapia and also stimulates some aspects of the nonspecific immune response where *Ped. acidilactici* colonized the intestinal tract and significantly affected the intestinal microbial communities (Ferguson *et al.,* 2010). Beneficial microflora promote gut development and health by influencing enterocyte turnover, competing with pathogenic bacteria for nutrients and binding sites, and producing bacteriostatic compounds that limit the growth of pathogenic bacteria (Farthing 2004).

Lactic Acid Bacteria (LAB) have antioxidant activities (Kullisaar *et al.,* 2002) and some studies showed an enhancement of total antioxidant status (TAS) in blood of humans fed LABs (Kullisaar *et al.,* 2003). Treatment of Pediococcus acidlactici-based probiotic on intestinal villus height of broiler chickens increased villus height in duodenum and ileum when compared with control (Taheri *et al.,* 2010). Pediococcus acidilactici enhanced the colonization and adhesion of probiotics on the surface of intestines besides increasing the density, length and width of intestinal villus resulting in better performance and meat quality in broilers (Satheesh *et al.,* 2012).

Aim of the work:-
The present study is conducted to evaluate the effect of *Pediococcus acidi lacticae* on meat quality and villius hights in broilers chicken and to investigate the relationship between infection and antioxidant.

MATERIALS and METHODS

This study was conducted in the animal health research institute – kafrelshikh, Egypt.

Experimental design:
A total of 125 three days –old chicks assumed to be free of salmonella by (15 fecal swabs and organs culture from 5 sacrificed chicks) were obtained from the General Egypt Poultry Organization and classified into 4 groups. The chicks were housed in clean well-ventelated room under sanitary hygienic, good mangement and kept either on a basal ration (as shown in the table 1) which was formulated according to NRC (1994) or on a basal ration with *Pediococcus acidilactici* (probiotic) at a rate of 100mg/kg ration for 35 days for performance and biochemistry and 40 days for meat quality. All groups chicks were vaccinated against ND and IBD at 7 & 12 day respectively. Experimental infection with overnight broth culture of *S. enteritidis* at 2 week of age was done as shown in the table (2)

Bacterial infection:
Experimental infection was done via oral route with 1 ml containing $3x10^8$ of *S. enteritidis* at 15 day –old. This strain was supplied by A.M. Hegazy (Animal Health Research Institute, Kafr El-Sheikh Regional lab).

Measurements:
Growth performance parameters:
Body weight (BW), feed conversion ratio (FCR), feed intake (FI) and mortality rate were recorded weekly.

Sampling and antioxidant and lipid peroxidation measured:-
Blood was collected through slaughtering. 1 ml of this blood was mixed immediately in Ependorf tubes with EDTA and used for determining of reduced glutathione (Beutler *et al.,* 1963). The rest of blood after coagulation was centrifuged at 3,000 x g for 15 minutes for separation of serum (stored deep freezer until use) for detecting superoxid dismutase (SOD) (Nishikimi *et al.,* 1972), catalase (CAT) (Ashru and Sinha 1971) and Lipid peroxidation (represented in malonaldehyde = MDA) (Beuge and Aust 1978).

S. enteritidis colonization:
Fecal shedding and organ colonization were recorded weekly for three weeks. Confirmation of the results of colonization and shedding was done by *S. enteritidis* antiserum prepared in rabbit as described by Seleim (1999).

Symptoms and P. M lesions:
All groups were kept under observation for symptoms, post mortum (P.M) and mortality along the experimental period.

Chemical analysis of feed and meat: -
The proximate analysis of feed sample moisture, crude protein (CP), ether extracts (EE), crude fiber (CF), total ash content were determined according to A.O.A.C. (1990).

The examined samples of chicken meat were analyzed for determination of moisture, protein, fat, ash and carbohydrate by using the standard method recommended by Association of Official Analytical Chemists "AOAC" (2000) as follow:

1. Determination of moisture %:
A dish was dried in an oven and cooled in the desiccator. Approximately 2 g of sample were weighed into the dish and dried in the oven at 102°C with the lid alongside for 2 hours. The dish was covered with the lid, and transferred to the desiccator and when the dish completely cooled, it was weighed, then heated in the oven half-an-hour and re-weighed. Repeat until successive weights do not differ.

$$\text{Moisture \%} = \frac{\text{Weight lost x 100}}{\text{Weight of sample}}$$

2. Determination of protein %:
The weighed samples were placed in Kjeldahl flask containing mercury oxide (catalyst) and potassium sulphate (increasing boiling point). Concentrated sulphuric acid was added, and the mixture was heated and boiled until the sample was completely digested and yielded ammonia. The flask was cooled, the solidified content (including a sulphide to precipitate the mercury) was added and, the released ammonia was distilled via a condenser into a boric acid solution containing acid-base indicator. The collected ammonia was titrated with standardized hydrochloric acid. So the amount of ammonia present could be detected, and thus the amount of nitrogen can be calculated.

Crude protein % = nitrogen % x 6.25.

3. Determination of fat %:
A weighed sample was dried at 60 °C for 72 hours. The dried sample was exactly weighed and wrapped carefully in a filter paper. Such prepared sample was used in determination of fat %.

Soxhlet flask containing 75 ml ether was placed on the electrical heater; the sample was placed in the extractor which was fixed tightly over the Soxhlet flask. Then, the condenser was fixed over the extractor. When heating occurred ether will be evaporated and raised up through the outside capillary tube to the condenser, where it was re-condensed to liquid again under the effect of water current in the condenser, and down set on the sample dissolving apart of the fat. Ether was accumulated in the extractor until siphoning occurred where the ether returned to the flask again with the dissolved fat. This process was repeated until all fat in the sample will be extracted this take about 6 hours. Repeat weighing the sample after drying to record the loss of weight and fat % was calculated.

4. Determination of ash %:
A weighed sample was dried at 60 °C for 72 hours. In a porcelain crucible of known weight, the known weight of dried sample was placed and the crucible was transferred into muffle furnace at 450°C to free ash from carbon and inorganic matters. The process was continued for about 6 hours then the crucible was cooled, desiccated and weighed. This technique of heating and weighing was repeated several times till reaching 2 equal successive weights. Therefore, the ash % was calculated as follow:

Ash % = difference between crucible with ash and empty crucible.

5. Determination of Carbohydrate %:
Each sample was estimated for its moisture, protein, fat, crude fiber and ash content. Accordingly, carbohydrate % was calculated by the difference from the following equation:

Carbohydrate %= 100- (moisture % + protein % + fat % + crude fiber % + ash %)

5. Determination of pH (Pearson 1984):
In a blender, approximately 10 g of sample were blended in 10ml of distilled water. The homogenate was left at room temperature for 10 minutes with continuous shaking. The pH value was determined by using an electrical pH meter (Bye model 6020, USA).

Histopathological findings of chicken supplemented with pediococus and challenged with salmonella infection:

Necropsy and histopathology
After necropsy, small pieces of liver, intestine and spleen were dehydrated and embedded in paraffin wax by routine methods. They were then sectioned at 3 µm, stained with haematoxylin and eosin (HE), and examined by light microscopy.

Statistical analysis:
The obtained numerical data were statistically analyzed using S.P.S.S., (1997) for one-way analysis of variance. When F- test was significant, least significant difference was calculated according to Duncan (1955).

RESULTS

Symptoms and P. M lesions:
Experimental infection revealed suggestive clinical and gross pathological lesions, in the form of depression which appeared after 48h post infection and was associated with whitish diarrhea unabsorbed yolk sac, distended gall bladder, enlarged congested liver, distended cecum and sometimes cecal core and the appearance of intestinal ulcer (characteristics for ND specifically in the duodenum) were obvious in *salmonella* infected group. All the previously mentioned symptoms and P. M changes were less prominent in group of *pedicoccus* with infection in comparison with the *salmonella* infected group. This may be attributed to the effect of *pedicoccus* sp. No significant variation in mortality as it was 3&2 chicks for each of *salmonella* infected group & group of *pedicoccus* with infection respectively (table 4). Deaths were restricted to 1st 5days pi.

Growth performance:
Table (5) showed the growth performance parameters, where the final body weight was significantly decreased ($P < 0.05$) in groups of *pediococcus* with or without infection compared with the control group without infection. *Salmonella* infected group significantly decreased ($P < 0.05$) in body weight when compared with the control without infection and *pediococcus* groups with or without infection.

The feed conversion ratio in the different groups are presented in table (5). It is clear that the general mean of feed conversion in *Salmonella* infected group increased FCR (1.79±0.13a) versus (1.66±0.04a), (1.63±00.009a) and (1.58±0.1a) in control negative; *pedicoccus- salmonella*; and *pedicoccus* control groups, respectively but not significant ($P < 0.05$).

Antioxidant and lipid peroxidation measurements:-

Table (6) showed enzymatic antioxidants, GSH and MAD, which presents significant decreased in GSH (1.79 ± 0.01 b), CAT (22.93 ± 1.00c) and SOD (117.21 + 19.88 b) ($P < 0.05$) in *salmonella* infected group without probiotic compared with other groups. Regarding the effect of probiotic, *Pediococcus* group without infection significantly increased ($P < 0.05$) CAT activity (62 ± 2.08 a) compared with the other groups and insignificantly increased ($P < 0.05$) SOD in *Pediococcus* groups with and without infection compared with the control non infected group (161.04 + 23.79 a), insignificantly increased ($P < 0.05$) GSH (3.57 ± 0.18 a) in *Pediococcus* group without infection compared with the control non infected group (3.50 ± 0.15 a) and the *pediococcus* infected one (3.27 ± 0.57 a). SOD

Meanwhile MAD significantly increased ($P < 0.05$) in *salmonella* infected group (10.9 ± 0.92a). Regarding the effect of probiotic, *pediococcus* group without infection (2.9 ± 0.35b) insignificantly decreased MDA ($P < 0.05$) compared with the control non infected group (3.47 ± 0.26b) and the *Pediococcus* infected one (4.1± 0.67b).

Chemical analysis of meat: -

Table (7) showed the chemical composition of broiler meat where the average water content was significantly decreased ($P < 0.05$) in the control group without infection and in the *Pediococcus* group without infection compared with control infected group and *Pediococcus* infected one, where the lower percentage in control group without infection (72.43 ± 0.30b) and the higher percentage in *salmonella* infected group. While, the obtained our results with respect to protein showed significant increase ($P < 0.05$) in protein % in the *Pediococcus* group without infection (21.6 ± 0.38a) compared with the *salmonella* infected group and the *Pediococcus* infected one, and non significant ($P < 0.05$) increased about forth control group without infection (20.73 ± 0.35ab) but without significant. Fat % showed lower % in the groups receiving *Pediococcus* with (2.9 ± 0.19a) or without infection (2.88 ± 0.17a) compared with the control groups with (3.03 ± 0.17a) or without infection (3.38 ± 0.13a) without significance. Regarding to PH in the different groups, table (7) showed that a significant increased in PH in the *salmonella* infected group (5.95 ± 0.04a) compared with the control without infection (5.81± 0.02c) and the *Pediococcus* without infection (5.77 ± 0.02cd) and in the *Pediococcus* infected one (5.86 ± 0.03bc). Comparing between the control group without infection and the *Pediococcus* group without infection, there was insignificant decreased in PH in the *Pediococcus* without infection group. Table (7) showed insignificant decreased in ash % in the *Pediococcus* groups with (1.6 ± 0.19a) or without (1.38 ± 0.18a) infection compared with the control groups with (1.58 ± 0.19a) or without infection (1.55 ± 0.34a). Meanwhile carbohydrate % non significant decreased in the *salmonella* infected group, and the *Pediococcus* without infection compared with the control group without infection but significantly decreased in the *Pediococcus* with infection compared with the control without infection.

S. enteritidis colonization:

S. enteritidis differed in their colonization in different organs. It shows the rates of 60, 40, 33, and 33% for each of intestine, liver, spleen and gall bladder, respectively (table, 8). *S. enteritidis* was capable to colonize different organs with different rates (Table, 8) intestine, 60 vs. 30%, liver, 40 vs.13% , spleen, 33 vs.20% and gallbladder, 33vs.13% in each *salmonella* infected control group and *Pediococcus* vs. *salmonella* group, respectively.

Histopathological findings of chicken supplemented with *Pediococcus* and challenged with salmonella infection:

In group 1(control negative, not received *Pediococcus* or *salmonella* infection), no detectable pathological changes were observed in the examined organs including liver (Fig.1a), intestine (Fig.1b) and spleen (Fig.1c). Group 2 (received Pediococcus without exposed to infection), liver was completely normal and Kuffer's cells granuloma were observed (Fig.2a). Intestine was normal with elongation of the villi (Fig.2b). Spleen showed increase in lymphocytic cells population in the white bulb (Fig.2c).

In group 3 (not received *Pediococcus* and exposed to *salmonella* infection), the liver showed marked congestion (Fig.3a1,2), in addition to hemorrhage (Fig.4a) was observed especially subcapsular in most of the cases. Portal mononuclear cells and heterophils infiltrations was also observed. In addition to, the previously mentioned lesions, focal necrosis of the hepatic cells infiltrated and surrounded with mononuclear cells and heterophils was detected in few cases with hepatic cell regeneration (Fig.5a). The intestine showed enteritis (Fig.3b1,2) in most of cases characterized by shortening, thickening and adhesion of the villi, degeneration and necrosis of the covering epithelium of mucosa together with marked congestion and marked heterophils infiltrations in the lamina propria and submucosa. Goblet cells hyperplasia was observed in some sections. The spleen showed moderate lymphoid depletion and

congestion as well as degeneration and depletion of lymphoid follicles (Fig.3c1,2), in group 4 (received *Pediococcus* and exposed to *Salmonella* infection), the liver in most of the cases was almost completely normal except very mild congestion (Fig.6a) which tended to be moderate in one case and focal mild perivascular mononuclear cells collections which in few cases were admixed with few heterophils were also observed (Fig.7a). Intestine was almost completely normal and elongation of the villi was observed in most of the cases. However, mild degeneration, thickening and necrosis of villi (Fig.4b) as well as moderate congestion and pyre's patches hyperplasia (Fig.5b) were observed in few sections. Spleen showed normal lymphocytic cells population in the white bulb in most of the cases and lymphoid follicles depletion in one case.

Table 1: Ingredients and nutrients (calculated) of 2 the basal experimental diets for the broilers.

Items	Experimental diets	
	Starter	**Grower and finisher**
Ingredient %		
Yellow corn,(8.5%)	53.00	59.50
Soybean meal, (44%)	29.26	29.00
Corn gluten meal, (62%)	9.00	3.427
Sun flower oil	4.50	4.50
Dicalcium phosphate	1.80	1.30
Limestone, ground	1.30	1.30
Lysine	0.08	0.29
DL-Methionine	0.10	0.044
Common salt	0.40	0.40
Choline chloride,50%	0.26	0.20
Trace minerals and vit. premix	0.30	0.30
Values between parentheses are determined crude protein content (N×6.25).		
Nutrient (Calculated)		
	Starter	**Grower and finisher**
Crude protein, %	23.1138	20.0027
ME ,(kcal/kg)	3204.813	3205.0426
Calcium, %	0.99	0.90
Available phosph., %	0.47	0.37
Lysine	1.10	1
Methionine	0.51	0.38

Table 2: Experimental design for the broilers groups.

Group No.	Diet type	Pedicoccus supplementation*	S.enteritidis infection*
1	Basal diet	-	-
2	"	100mg/kg ration	-
3	"	-	+ve
4	"	100mg/kg ration	+ve

* *Pedicoccus* is a Probiotic (Bactocell) a commercial product each 1gm contains 1x109 CFU. EGAVET, Giza, Egypt

Table 3: Chemical composition of the experimental diets.

	Experimental diets	
	Starter	**Grower and finisher**
Chemical composition *		
Dry matter, %	89.60	89.69
Moisture, %	10.40	10.31
CP, %	21.87	19.16
EE, %	5.64	5.99
Ash , %	6.81	6.89
CF, %	3.02	3.43
NFE, %	52.26	54.22
ME,(Kcal/Kg)	3147.1281	3176.7282

*Analysed values

The used permix (multimix broiler) without choline composed of vitamin A 12000000 ıU, vitamin D3 2200000 ıU, 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, Iron 30000 mg, Manganese 60000 mg, Copper 4000 mg, Zinc 50000 mg, Iodine 1000 mg, Cobalt 100 mg, Selenium 100 mg, Calcium carbonate(CaCo3) carrier to 3000 g.

Table 4: Effect of *pediococcus acidi lactici* supplementation without or with SE infection on mortality rate post infection.

parameters	Supplementation	Without infection	With SE infection
Total No.	Control	25	25
	pediococcus acidi lactici	25	25
Dead No.	Control	0	3
	pediococcus acidi lactici	0	2
Survival ,%	Control	100%	88%
	pediococcus acidi lactici	100%	92%
Mortality ,%	Control	0%	12%
	pediococcus acidi lactici	0%	8%

SE = *Salmonella Entritidis*

Table 5: Effect of *pediococcus acidi lactici* supplementation without or with SE infection on growth performance of broiler chicks.

parameter	Supplementation	Without infection	With SE infection
Initial B.W.	Control	56 ± 1.32 a	56 ± 1.32 a
	pediococcus acidi lactici	53 ± 0.87 a	53 ± 0.87 a
Final B. W.	Control	1439.1 ± 0.64 a	1211.1 ± 0.59c
	pediococcus acidi lactici	1405 ± 0.59 b	1397.3 ± 0.64 b
FCR	Control	1.66±0.04a	1.79±0.13a
	pediococcus acidi lactici	1.58±0.1a	1.63±00.009a

B. W. = body weight FCR = feed conversion ratio

Values are expressed as mean ± standard errors. Means in the same row (a-c) with different letters significantly differ at ($p \leq 0.05$).

Table 6: Effect of *pediococcus acidi lactici* supplementation without or with SE infection on antioxidant of broiler chicks.

Parameter	Supplementation	Without infection	With SE infection
Whole blood GSH (mmol/l)	Control	3.50 ± 0.15 a	1.79 ± 0.01 b
	pediococcus acidi lactici	3.57 ± 0.18 a	3.27 ± 0.57 a
Serum CAT (nm/ml)	Control	31.33 ± 0.68 b	22.93 ± 1.00c
	pediococcus acidi lactici	62 ± 2.08 a	29 ± 1.61 b
Serum SOD	Control	161.04 + 23.79 a	117.21 + 19.88 b
	pediococcus acidi lactici	163.52 + 28.93 a	174.65 + 9.52 a
Serum Lipid peroxide(nm/ml)	Control	3.47 ± 0.26 b	10.9 ± 0.92 a
	pediococcus acidi lactici	2.9 ± 0.35 b	4.1± 0.67 b

GSH= reduced glutathion CAT = catalase SOD=super oxid dismutase
Values are expressed as mean ± standard errors. Means in the same row (a-d) with different letters significantly differ at ($p \leq 0.05$).

Table 7: Effect of pediococcus acidi lactici supplementation without or with SE infection on chemical composition of broiler meat.

parameter	Supplementation	Without infection	With SE infection
Moisture, %	Control	72.43 ± 0.30b	74.18 ± 0.34a
	pediococcus acidi lactici	72.65 ± 0.34b	73.65 ± 0.31a
Protein, %	Control	20.73 ± 0.35ab	19.5 ± 0.33cb
	pediococcus acidi lactici	21.6 ± 0.38a	20.38 ± 0.34b
Fat, %	Control	3.38 ± 0.13a	3.03 ± 0.17a
	pediococcus acidi lactici	2.88 ± 0.17a	2.9 ± 0.19a
Ash, %	Control	1.55 ± 0.34a	1.58 ± 0.19a
	pediococcus acidi lactici	1.38 ± 0.18a	1.6 ± 0.19a
Carbohydrate, %	Control	1 ± 0.11ab	0.8 ± 0.09b
	pediococcus acidi lactici	0.9 ± 0.09b	0.7 ± 0.07cb
PH	Control	5.81 ± 0.02c	5.95 ± 0.04a
	pediococcus acidi lactici	5.77 ± 0.02cd	5.86 ± 0.03bc

Values are expressed as mean ± standard errors. Means in the same row (a-d) with different letters significantly differ at ($p \leq 0.05$).

Table 8: Colonization of S. enteritidis and rate of shedding as judged by intestinal colonization.

	Liver			G.bladder			spleen			Intestine			Total(T)		
S. E + P	+	T	%	+	T	%	+	T	%	+	T	%	+	T	%
S.E	2	15	13	2	15	13	3	15	20	5	15	33	12	60	20
Total	6	15	40	5	15	33	5	15	33	9	15	60	25	60	42
S. E + P	8	30	27	7	30	23	8	30	27	14	30	47	37	120	31

*S.E+P = *S.enteritidis* + *Pedicoccus* **S.E* = *S.enteritidis*

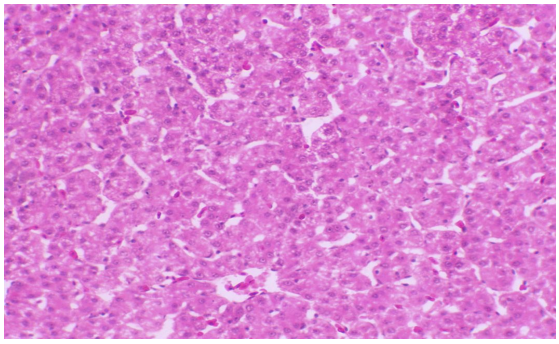

Fig. 1a. G1, the liver showing normal lobular architecture with central vein and radiating hepatic cell cords. H&E. X 200.

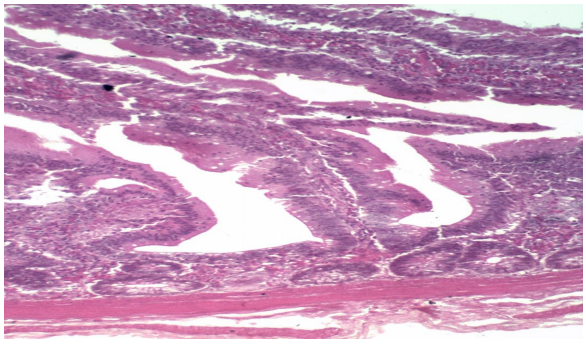

Fig.1b, G1, showing normal intestine. H&E. X 100.

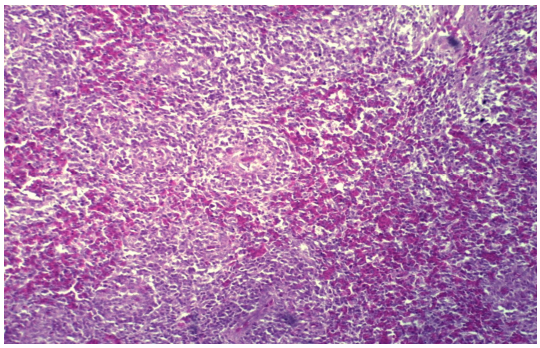

Fig.1c, G1, spleen showing normal lymphocytic cells population in the white bulb H&E. X 100.

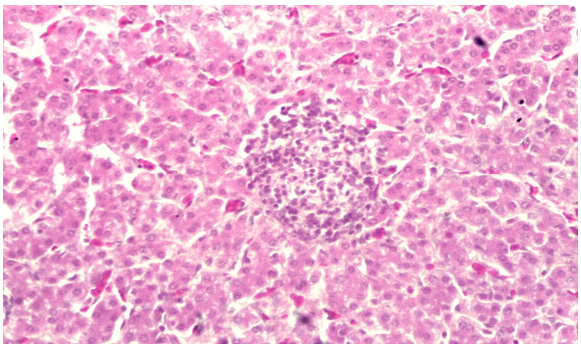

Fig.2a, G2, the liver showing small mononuclear cells collections. H&E. X 200.

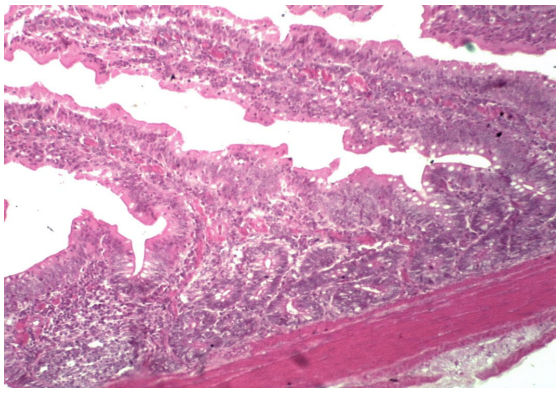

Fig.2b, G2, normal intestine with elongation of the villi. H&E. X 100.

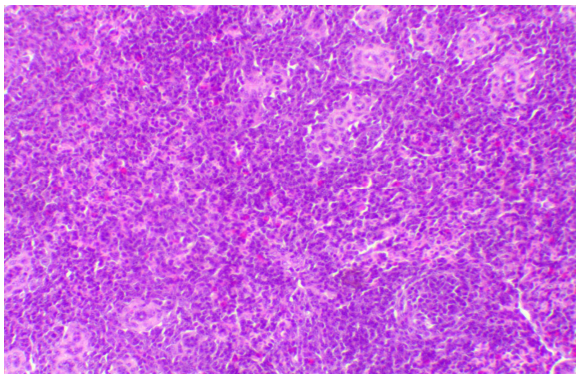

Fig.2c, G2, spleen showing increase in lymphocytic cells population in the white bulb. H&E. X 100.

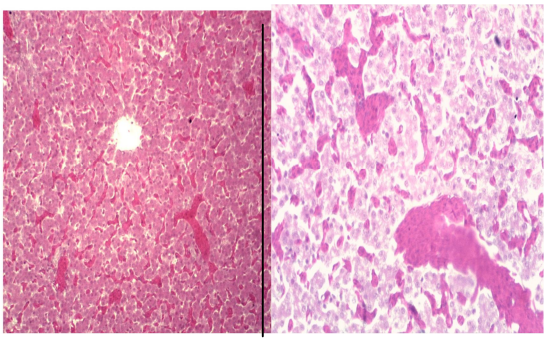

Fig.3a1,2, G3, the liver showing marked congestion. H&E. X 100, H&E. X 200.

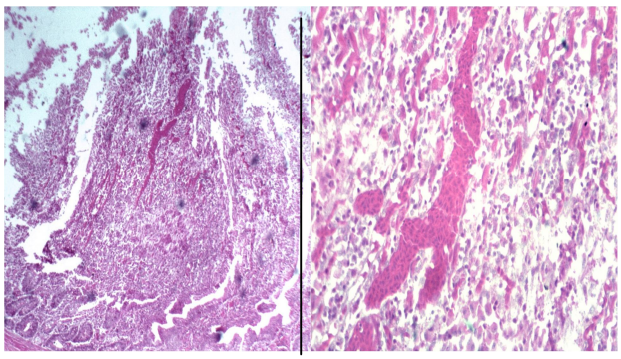

Fig.3b1,2, G3, intestine showing thickening, degeneration and necrosis of the superficial layer of the covering epithelium of mucosa together with marked congestion of the lamina prpria. H&E. X 100, H&E. X 200.

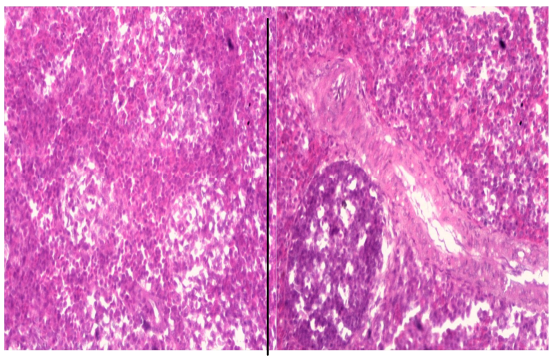

Fig.3c1,2, G3, the spleen showing moderate lymphoid depletion as well as depletion of lymphoid follicles. H&E. X 100, H&E. X 200.

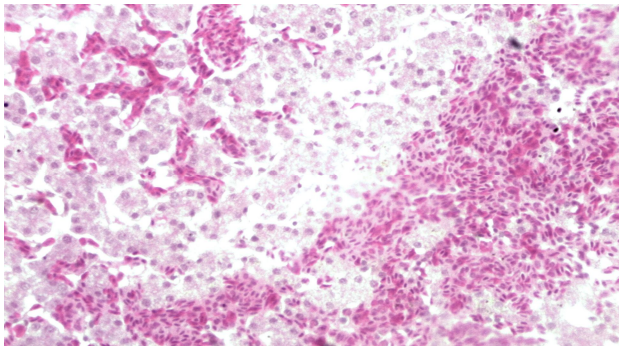

Fig.4a, G3, the liver showing focal hemorrhage. H&E. X 100.

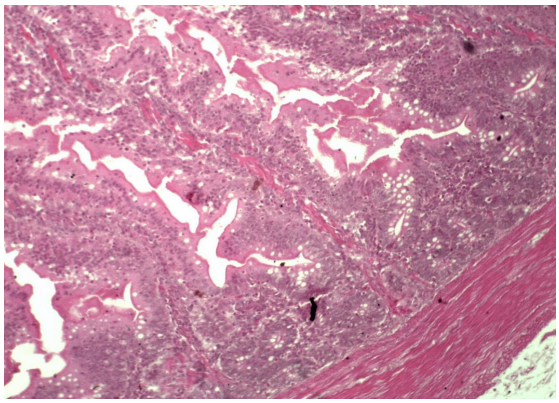

Fig.4b, G4, intestine showing mild degeneration, thickening and necrosis of villi. H&E. X 100.

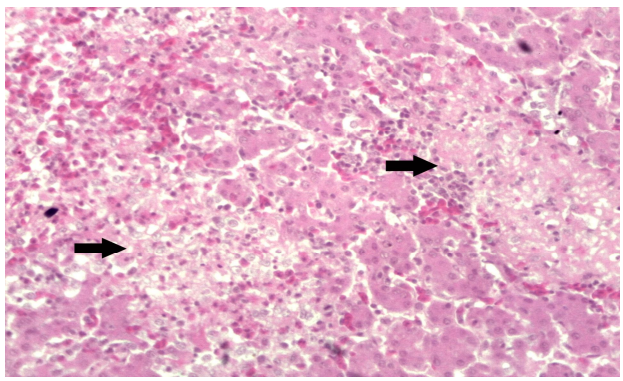

Fig.5a, G3, the liver showing focal hepatic cell necrosis infiltrated and surrounded with mononuclear cells and heterophils (arrow). H&E. X 200.

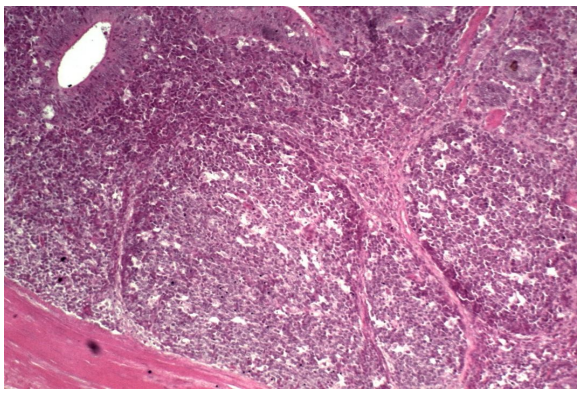

Fig.5b, G4, the intestine showing pyre's patches hyperplasia. H&E. X 100.

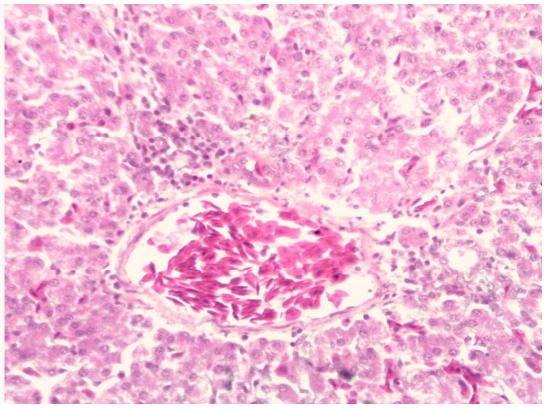

Fig.6a, G4, the liver showing mild congestion of the central vein and mild perivascular mononuclear cells collections. H&E. X 200.

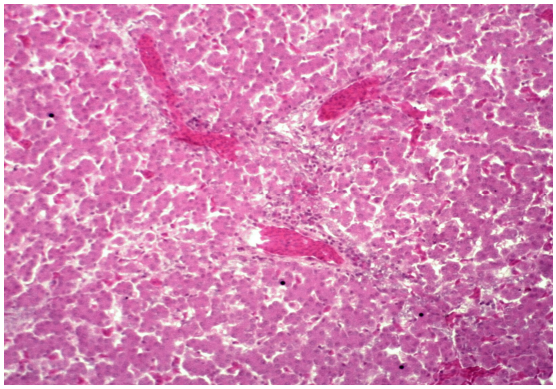

Fig.7a, G4, the liver showing mild perivascular mononuclear cells and heterophils infiltrations and moderate congestion. H&E. X 100.

DISCUSSION

Symptoms and P. M lesions:

All clinical and gross pathological lesions in salmonella infected group were close with Gast and Benson (1995). The mortality rate was lower than that recorded by other workers and this may be explained by the fact that older birds were considerably less susceptible to the lethal effects of *Salmonella paratyphoid* and may experience intestinal colonization and even systemic dissemination without significant morbidity or mortality or the paratyphoid bacteria are not host specific and produced mortality only in young chicks (Gordon 1977).

Growth performance:

The significant ($P < 0.05$) decreased in the final body weight in groups of *Pediococcus* with or without infection compared with the control group without infection was supported by the statement of (Djezzar *et al.,* 2013), who reported that inclusion of *Pediococcus acidilactici* in the diet of broilers didnot improve the body weight, which disagreed with Tollba *et al.* (2007) where *Pediococcus* improved body weight. Kabir *et al.* (2004) observed an improvement in body weight when used other probiotic. Meanwhile *Salmonella* infected group significantly ($P < 0.05$) decreased in the body weight when compared with the control without infection and *the Pediococcus* groups with or without infection. This finding agreed with Azza *et al.* (2012). The infected group by *salmonella* significantly decreased body weight compared with the non infected one and *the Pediococcus* group. The feed conversion ratio in *Salmonella* infected group insignificantly increased. Johri (2004) did not observe any positive effect on the FCR of the chickens when *streptococcus lactis* was incorporated in the feed. Meanwhile Satheesh *et al.* (2012) found a significant change in FCR. Taheri *et al.* (2010) showed a beneficial effects of dietary inclusion of *pediococcus acidilactici*-based probiotic. The treated birds had improved BW and FCR when compared with the control.

Antioxidant and lipid peroxidation measurements:-

The main antioxidant enzymes that constitute the first line of antioxidant enzymatic defenses include

superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx). SOD catalyzes dismutation of superoxide radicals to hydrogen-peroxide and oxygen CAT catalyzes the breakdown of hydrogen-peroxide to water and molecular oxygen. GPx is a selenium-dependent enzyme, which decomposes peroxides using the peptide glutathione (GSH) as its cosubstrate (Halliwell 2006). Meanwhile concentration of MDA in tissues and urine are generally used as a biomarker for radical-induced damage and endogenous lipid peroxidation (Wang *et al.*, 2008).

The results of enzymatic antioxidants, GSH and MAD correlated with meat quality, histopathology and PM lesions. Significant decreased in GSH, CAT and SOD in *salmonella* infected group without probiotic, may be due to infection. This finding disagred with Koinarski *et al.* (2006), where CAT activity was increased in the infected chicks with *E. acervulina* and agree with Koinarski *et al.* (2005). They clearly showed that the chicks infected with *E. acervulina* were under oxidative stress, which was manifested primary via alterations of antioxidant enzyme activities of SOD and CAT, the reduction of some non-enzymatic antioxidants (vitamins A and C) and increased plasma MDA concentration. The increased CAT activity indicated the highly induced capacity to scavenge hydrogen peroxide produced in the red blood cells in response to oxidative stress due to the infection. This situation may reflect the persistent oxidative stress. The increased CAT activity may be a compensatory mechanism to get rid of excess of peroxides (Halliwell and Chirico 1993). Koinarski *et al.* (2006) said that SOD activity was significantly decreased in blood samples from the infected chicks but blood MDA was significantly increased in the infected chicks vs the healthy ones.

Regarding the effect of probiotic, *Pediococcus* group without infection significantly increased CAT activity compared with the other groups and insignificantly increased SOD in the *Pediococcus* groups with and without infection compared with the control non infected group, insignificantly increased GSH in the *Pediococcus* group without infection compared with the control non infected group and *the Pediococcus* infected one. This finding may be due to probiotic addition Castex *et al.* (2010) showed that the infected shrimps with *Vibrio nigripulchritudo* previously fed with the probiotic enriched diet *(Pediococcus acidilactici)* maintained the levels of their antioxidant defenses compared to the control over the 72-hour challenge period. Indeed, TAS and SOD, Gpx and CAT activities in the digestive gland did not decrease after infection as observed in the control group. Lactic Acid Bacteria (LAB) have antioxidant activities (Kullisaar *et al.,* 2002). Some studies showed an enhancement of total antioxidant status (TAS) in blood of humans fed LABs (Kullisaar *et al.,* 2003).

MAD significantly ($P < 0.05$) increased in the *salmonella* infected group. This may be attributed to *salmonella* infection. This finding agree with Koinarski *et al.* (2006) where one of the main blood lipid peroxidation products (MDA) "a marker of radical-induced damage" was statistically significantly ($P < 0.05$) increased in the *E. acervulina* infected chicks. Increased concentrations of lipid peroxidation endproducts have been used as indicators of ROS-derived damage in biological systems (Halliwell and Chirico 1993). Mathew *et al.* (2007) reported a concomitant increase in lipid peroxidation and a drop in antioxidant enzyme activities of SOD, CAT, Gpx and Glutathion-S- transferase in the digestive gland, muscle and haemolymph of *P. monodon* following infection with white spot syndrome virus (WSSV). The mucosal pathology of *Salmonella typhimurium* infection may in part be due to the excessive production of reactive oxygen species (ROS) (Mehta *et al.,* 1998).

Pediococcus group without infection insignificantly decreased MDA compared with the control non infected group and *the Pediococcus* infected one, this finding may be due to *Pediococcus acidilactici*. This finding agree with Castex *et al.* (2010) where MDA and carbonyl protein were significantly ($P < 0.05$) lower in the probiotic group compared to the infected control at 24 h. post infection of *Litopenaeus stylirostris* under infection with *Vibrio nigripulchritudo*.

Chemical analysis of meat: -

Meat can be defined as the product that results from the continuous changes that occur in muscle after the death of the animal (Castillo *et al.,* 2013). Poultry meat is particularly high in quantities of valuable protein, essential amino acids, fat, essential fatty acids, vitamins and minerals, which come from high quality concentrated food and therefore plays an important role in human nutrition (Givens 2005). Water, proteins and fat are major constituents of the meat and their qualitative and quantitative relationship determines the quality, in the other words, nutritional value of meat (Ivanovic *et al.,* 2012).

The chemical composition of broiler meat showed the average water content was significantly decreased in the control group without infection and in *Pediococcus* group without infection compared with control infected group and *Pediococcus* infected one. This result agreed with Ivanovic *et al.* (2012) where addition of probiotic increased water content in drumsticks of broiler but disagreed with Sazedul *et al.* (2010). While protein significantly increased in *Pediococcus* group without infection compared with *salmonella* infected group and *Pediococcus* infected one, and increased unsignificantly control group without infection. This findings agreed with finding of Ivanovic *et al.* (2012). Analyzing the chemical

composition of breast meat they found that the total protein content was statistically significantly higher ($P < 0.05$) after the addition of probiotics. Fat % showed lower % in groups receiving Pediococcus with or without infection compared with control groups with or without infection without significance. This findings agreed with Ignatova *et al.* (2009) that the probiotic decreased fat in breast meat of experimental group compared to control group. The supplementation of probiotics (*Lactobacillus acidophilus* and *Streptococcus faecium*) to broiler diets was reported (Mahajan *et al.*, 2000) to increase moisture, protein, ash, water-holding capacity (WHC), emulsion capacity and stability in broiler meats. Some authors reported advantages of probiotic administration on meat quality (Corrêa *et al.,* 2000; Vargas et al. 2002), whereas others did not observe improvement when probiotics were used (Owings *et al.,* 1990; Quadros *et al.,* 2001). Probiotics were reported to prevent colonization gut by pathogens like *Escherichia coli* and *Salmonella* (Juven *et al.,* 1991). Probiotics may help in minimizing stress and improving meat quality regimen of broilers. Also supplementation of probiotics in broiler ration improved the meat quality both at prefreezing and postfreezing storage (Kabir 2009).

Significant increased in PH in *salmonella* infected group compared with control without infection and *Pediococcus* without infection and in *Pediococcus* infected one, were reported with insignificant decreased in PH in *Pediococcus* without infection group compared with control group without infection. These findings disagreed with Aksu *et al.* (2005) where the use of probiotic *Saccharomyces cerevisiae* (4×10^8 cfu/g) in broiler diets increased pH values, and the highest pH values occurred in the 0.2% group. Pelicano *et al.* (2005) measured pH 5 h after the slaughter and found no statistical significant difference between meat samples from chickens that did not receive probiotics. Insignificant decreased in ash % in the *Pediococcus* groups with or without infection compared with the control groups with or without infection were recorded. This finding agree with Ignatova *et al.* (2009). Carbohydrate % insignificant decreased in *salmonella* infected group, and *Pediococcus* group without infection compared with control without infection but significantly ($P < 0.05$) decreased in *Pediococcus* with infection compared with control without infection. This finding may be due to *Pediococcus* and/or infection. Some studies have demonstrated that meat shelf-life and quality can be improved by natural antioxidants added in the preslaughter stages, incorporating natural antioxidants in animal diets. Thus, among the positive effects of natural antioxidants on meat characteristics were retarding lipid oxidation, color loss, and microbial growth (Velasco and Williams 2011).

S. enteritidis colonization:

The colonization of *S. enteritidis* differed in different organs. Similar observations were reported by Barrow (1991) and Gorham *et al.* (1991). Several experiments had demonstrated that prevention of *Salmonella* colonization in chickens can be achieved by many treatments. Probiotics was one of them (Johannsen *et al.,* 2004). This was true as in the present work *S. enteritidis* was capable to colonize different organs with different rates. This could be supported by the findings of Tollba *et al.* (2007) and Jamila *et al.* (2011) who reported that probiotics, suppressed the counts of pathogenic intestinal bacteria and decreased colonization of *salmonella*.

Histopathological findings of chicken supplemented with *Pediococcus* and challenged with salmonella infection:

The finding in control group without infection and *Pediococcus* without infection agreed with Taheri *et al.* (2010). The effect of *Pediococcus acidilactici*-based probiotic on intestinal villus height of broiler chickens was increased villus height in duodenum and ileum when compared with control. *Pediococcus acidilactici* enhanced the colonization and adhesion of probiotics on the surface of intestines besides increasing the density, length and width of intestinal villus resulting in better performance and meat quality in broilers (Satheesh *et al.,* 2012).

Microscopic changes in liver which included Congestion and haemorrhages, isolated necrotic foci in hepatic parenchyma along with infiltration of leucocytes predominantly mononuclear cells and heterophils were also reported by Freitas *et al.* (2007) and Garcia *et al.* (2010). Depletion of lymphocytes and focal necrotic changes were also reported by Mohammadi *et al.* (1976) and Freitas *et al.* (2007). Prasanna *et al.* (2001) also reported a congestion of mucosal vessels, marked goblet cell hyperplasia, mild to moderate infiltration of heterophils and mononuclear cells in the lamina propria of the villi of intestines.

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تأثير البيدوكوكس أسيدلاكتيسي على مضادات الأكسدة الإنزيمية ، الباثولوجي وجودة لحوم بداري التسمين تحت العدوى بالسالمونيلا

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استخدمت مائة وخمسة وعشرين كتكوت من دجاج اللحم يبلغ من العمر ثلاثة أيام في هذه الدراسة لتقييم أداء النمو، والاستعمار بالسالمونيلا انتريتيدس ، ومضادات الأكسدة، وبيروكسيد الدهون، وجودة اللحوم والنتائج الهستوباثولوجية للدجاج. قسيمت الكتاكيت إلى أربع مجموعات، ومجموعة ضابطة بدون عدوى أو البيدوكوكس ، ومجموعة البيدوكوكس بدون عدوى، ومجموعة العدوى بالسالمونيلا بدون البيدوكوكس، ومجموعة البيدوكوكس المعدية بالسالمونيلا. أشارت النتائج إلى أن استخدام البيدوكوكس اسيدى لاكتسى لم يؤثر على أداء النمو، وأدى إلى تحسين مضادات الأكسدة ، وانخفاض بيروكسيد الدهون ، وتحسين جودة اللحوم وتقليل الاثار الهستوباثولوجية الضارة للسالمونيلا.