CLINICOPATHOLOGICAL STUDIES ON DIETARY SACCHAROMYCES CEREVISIAE SUPPLEMENTATION IN BROILERS EXPERIMENTALLY INFECTED WITH E COLI

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ABSTRACT

Two hundred and forty, one day old, chicks were divided into 6 groups and reared for 6 weeks. Group I: control group fed on balanced commercial ration. Group II and III: treated groups fed on balanced commercial ration supplied with 0.5% and 2% S. cerevisiae for 6 weeks respectively. Group IV and V infected at one week age with Escherichia coli strain O78 and fed on balanced commercial ration supplied with 0.5% and 2% S. cerevisiae respectively for 6 weeks. Group VI infected with E. coli at one week age and fed on balanced commercial ration. Growth performance were studied allover the experiment. Some biochemical and immunological parameters were investigated at 4^{th} and 6^{th} week. Also, parts from the heart, liver, kidney, intestine, spleen, thymus and bursa were obtained for histopathological examination.

Our results revealed that elevation AST, creatinine and uric acid, while total protein and albumin decreased in infected non treated group when compared with the control one. Dietary S. cerevisiae supplementation revealed significant improved in all investigated parameters. The immunological parameters, lysozyme, bactericidal activity and HI titer were enhanced in the treated groups fed S. cerevisiae in the infected treated groups compared with the infected non treated group one.

We could conclude that Saccharomyces cerevisiae has prospective effect on the growth performance, non specific and specific immune response in broilers.

INTRODUCTION

Antibiotics have been used as feed additives to improve growth performance and control disease in animals. However, antibiotic use tends to produce antibiotic resistance and residues in animal products. Probiotics have therefore become important as replacement feed additives **(Steiner, 2006)**. *Saccharomyces* with protein digestion and high acidic capacity could prevent antimicrobial-associated

diarrhea. **(Bleichner et al., 1997).** Colisepticemia is responsible for worldwide economic losses to the poultry industry **(Gross, 1991).** Pathogenic *Escherichia coli* isolates affecting poultry commonly belong to certain serogroups, particularly 078, O1, and O2 **(Sojka and Carnaghan, 1961).** With the advent of using yeast cultures as growth promoters in poultry diets, several beneficial effects have been recorded. Dried yeast has been used as a source of mannan oligosaccharides and β-glucans by a number of companies providing antibiotic-replacement products for animal production. β-glucans and oligosaccharides are reported to enhance growth promoters immune response in poultry **(Kemal et al., 2003 and Huff et al., 2006).**

The present study aims to investigate the effect of dietary supplementation of broilers with different doses of *Saccharomyces cerevisiae* on the growth performance, immunomodulatory effect and to investigate the changes on hematological picture, serum biochemistry as well as pathological alterations in broiler chicken experimental infected with *E coli* O78.

MATERIAL AND METHODS

1- Experimental chickens:

Two hundred forty one day old, apparent healthy chicks, Cobb breed were obtained from Ismailia- Masr Poultry Company Serapum City, Egypt. Chickens were reared in litter under standard environmental and hygienic conditions. Chickens were fed on a balanced ration (basal diet) free from antibacterial agents and water ad libitum. All chickens were subjected to the following vaccination schedule, Hitchner at 5^{th} day of age, Gumboro at 14^{th} day of age and Lasota at 18^{th} & 28^{th} day of age.

2- Ration:

Ingredient	Starter ration (0-3 weeks)	Grower ration (4-6weeks)
Yellow corn ground%	63.75	74.00
Soya bean%	27.75	14.75
#Broiler concentrate%	10	10
Bone meal %	1	1
Limestone ground%	0.50	0.25
Common salt	2.50	2.50
Methionine	0.4	2.5
Lysine	---	1.50

Calculated chemical analysis:

Crude protein (C.P.)%	22	18.04
ME/K. calorie/kg diet	3200	3031

#Broiler concentrate: Meat meal 55%, Fish meal 35%, Ca-carbonate 3%.

The ration is full-fill the requirements according to NRC (National Research Council).

3- Saccharomyces cerevisiae (Baker's yeast) :

Commercial product of baker's yeast manufactured by AKMAYA CO., Luleburgaz, Kirklareli, Turkey was used as a dietary supplementation to broilers.

4- Escherichia coli strain :

E.coli strain O78 was kindly obtained from Animal Health Research institute, Ismailia.

5- Experimental design :

Two hundred forty, one day old, apparent healthy chickens were classified into equal 6 groups. **Group I:** control non treated group fed on balanced commercial ration free from

antimicrobial agents for 6 weeks. **Group II:** treated group fed on balanced commercial ration supplied with S. cerevisiae in a dose of 0.5% (5 gm S. cerevisiae/ kg ration) for 6 weeks. **Group III:** treated group fed on balanced commercial ration supplied with *S. cerevisiae* in a dose of 2.0% (20 gm S. cerevisiae / kg ration) for 6 weeks. **Group IV:** infected group with E.coli at 1 week of age and fed from 1st day of life on balanced commercial ration supplied with *S. cerevisiae* in a dose of 0.5% (5 gm *S. cerevisiae* / kg ration) for 6 weeks. **Group V:** infected group with *E.coli* at 1 week of age and fed from 1st day of life on balanced commercial ration supplied with *S. cerevisiae* in a dose of 2.0% for 6 weeks. **Group VI:** control infected group with *E.coli* at 1 week of age and fed from 1st day of life on balanced commercial ration free from antimicrobial agents for 6 weeks. Six random samples of serum were taken from all experimental groups at 4th and 6th weeks of the experiment for investigation.

6- Pathogenicity tests:

E. coli strain O78 was known to be pathogenic to chickens, 0.6 ml saline suspension containing $2x10^7$ C.F.U. of *E.coli* (adjusted by plate count technique). One ml of suspension was passed into ten-fold serial dilution in sterile 9 ml distilled water; one ml from each tube was inoculated into 2 plates of nutrient agar and incubated for 24 hrs. Determine the concentration of the plates containing 200 colony growth then the dose of infection was adjusted. **(Macfaddin, 1980).**

7- Experimental Infection:

The method described by **Awaad (1972)** was applied in which the infected chicken inoculated with 0.6 ml saline suspension containing $2x10^7$ C.F.U. of E.coli intra-crop at 7 days of age.

I- Biochemical Parameters:

AST, total protein, albumin, creatinine and uric acid were determined with semi-automatic spectrophotometer (BM-Germany 5010) using commercial test kit (Randox Co. UK) according to enclosed pamphlets. A/G ratio was calculated according to **Kaneko et al., (1997).**

II- Immunological Parameters:

a) Serum lysozyme: Serum lysozyme was determined turbidometric assay by the method of **Parry et al., (1965).**

b) Bactericidal activity: Serum bactericidal activity was done following the procedure of **Kajita et al. (1990).**

c) Haemagglutination inhibition test for Newcastle disease viral antigen: The test is carried out according to king and **Hopkins (1983) and Chauhan and Roy (1996).**

III- Growth Performance Parameters:

a) Body weight:

Each chick was weighed at the beginning of the experiment (one day old) and at the end of every week of 6 weeks of the experiment. Individual live body weight was summed and divided by the number of chickens of each group to obtain the average live body weight/ week. **(Brady 1968).**

b) Body weight gain, Feed consumption (FC) and Feed conversion ratio (FCR):

The gain in body weight per week, FC and FCR were calculated according to **Brady (1968).**

IV- Histopathological studies:

Specimens of heart, liver, kidney, intestine, spleen, thymus and bursa of scarified birds from all groups were fixed in 10 % neutral formalin, embedded in paraffin, sectioned at 5-micron thickness and stained with Haematoxylin and Eosin for histopathological examination **(Bancroff et al., 1990)**.

V- Statistical analysis:

Data collected from the haematological and serum biochemical analysis of treated groups of chicks were statistically analyzed in compare to control group using statistical software program (SPSS for Windows, version 15, USA). Differences between means of different groups were carried out using one way ANOVA with Duncan multiple comparison tests.

RESULTS AND DISCUSSION

Regarding the results of biochemical investigation as shown in table (1 and 2), the elevated level of AST seen in the infected non treated group came in agreement with **Hanan (2002) and Fatma (2005)**. This elevation might be due to hepatic injury during the detoxification of E coli bacterial toxin **(Marcel 1994)**. Our pathological results revealed degeneration and atrophy of hepatocytes (fig.1). On the other hand, the reduced level of AST enzyme observed in infected treated groups compared to the infected non treated, might be due to improvement in the physiological condition of the liver and increase in the hepatic metabolic reserve **(Dobicki et al., 2007)**. Our results of non significant changes in the AST in the treated groups fed S. cerevisiae agreed with **Arrieta et al., (2007); Shareef and Al-Dabbagh (2009)**.

Total proteins and albumin were significantly decreased in the infected non treated group indicating hepatic damage because the liver is responsible for the production of a great proportion of plasma protein **(Coles, 1986)**. These results were similar to **Marcel (1994); Mona (1995). Dooley et al., (1988)** reported that the infection with E coli increase the breakdown of plasma protein, increase the renal excretion and impaired protein synthesis as a result of liver disorders caused by colibacillosis. On the other hand, at 4 and 6 weeks age, total proteins and globulin were significantly increased but albumin was non significantly changed in treated and infected treated groups fed S. cerevisiae, the increase in total protein in chickens fed the S. cerevisiae diet may be related to increased production of other serum protein fractions as globulin. These results came in agreement with **Fleicher et al., (2000)**, but disagree with **Abaza et al., (2008)** who reported no significant effect on serum total protein, albumin and globulin.

Concerning the results of renal function tests, showed significant increase in the levels of uric acid and creatinine in the infected non treated group all over the experimental period. **Coles (1986)** who reported the increase in uric acid and creatinine in chickens in case of renal disease or due to bacterial toxins. Also, these results came in agreement with **Dooley et al., (1988); Marcel (1994); Hanan (2002) and Fatma (2005)** reported that the experimental infection with E coli cause elevation of uric acid and this may be attributed to increase in the breakdown of plasma proteins. The pathological results revealed there was congestion of blood vessels and mild focal

interstitial aggregations of mononuclear cell infiltrations consists of macrophages and few lymphocytes (fig. 2). While, the levels of creatinine and uric acid decreased in the infected treated groups at 4 and 6 weeks. These results disagree with **Shareef and Al-Dabbagh (2009)** who found no effect of S. cerevisiae on uric acid level in blood.

Lysozyme is one of the most important factors of innate immunity, possessing antimicrobial action against a wide range of microorganisms due to cationic nature of protein and, in a lesser degree, due to muramidase activity. Our results were significant increase in the treated and infected treated groups, while, significant decrease in the infected non treated group in comparison with the control group. **Cleary et al., (1999) and Bagni et al., (2005)** reported significant increase in serum lysozyme activity post dietary yeast beta-glucan in mice and sea bass. **Truchlinski et al., (2005)** recorded that beta- glucan supplementation in turkey hens increased some indices of unspecific immunity (lysozyme activity). **Gao et al., (2008)** studied the effect of supplemental yeast culture in broiler diets for 42 days on immunomodulatory functions, and found that yeast culture increased serum lysozyme activity. These results came in harmony with the histopathological results, where there was focal hyperplasia of lymphoid organs in the treated groups, but there was lymphoid depletion in the infected non treated group (fig. 5, 6, 9 and 12).

Serum bactericidal activity was significantly increased in treated and infected treated groups, and significantly decreased in infected non treated group in comparison with the control. **Yuming et al., (2003)** reported that dietary β-glucan supplementation increased the macrophage phagocytic activity in broilers. **Yun-CheolHeui et al., (2003)** recorded that β-glucan treatment significantly enhanced phagocytic activity. **Huff et al., (2010)** stated that the numbers and percentages of heterophils in peripheral blood were increased and their oxidative burst activity was stimulated by yeast extracts in turkey poults.

HI titer was significantly increased in the treated and infected treated groups fed 2% S. cerevisiae. **Elizabeth et al., (2003)** found that Sc (0.20%) did not improve the hemagglutination inhibiting antibody titers in birds fed with aflatoxin in diet, however improve the immune response of broilers at challenge with strain velogenic of New castle disease virus (NDV). **Gao et al., (2008)** reported that yeast culture increased antibody titers to Newcastle disease virus, when supplemented for 42 days in broilers diet.

The present results of increased body weight, weight gain, decreased total feed intake and decreased feed conversion ratio in treated groups fed 2% *S. cerevisiae* in comparison with control were disagreed with **Mikulec et al. (1999) and Ladukar et al. (2002)** who reported that probiotic supplementation had no effect on the growth performance of broilers. While, our results came in agreement with **Onifade et al., (1999) and Shareef and Al-Dabbagh (2009)**. Also, **Yalcin et al (1993) and Yadav et al (1994)** found better weight gain and conversion index in broiler when adding yeast in higher percentages (5-20). **Newman (1994), Spring et al., (2000)**

reported that *S. cerevisiae* improved the efficacy of the immune system, improved intestinal lumen health, and increased digestion and absorption of nutrients, which resulted in better performance. These results are proved by the histopathological findings, where *S. cerevisiae* showed normal architecture of epithelial lining the villi (fig. 8). Also, infected treated groups fed 2% *S. cerevisiae* result was increased body weight, weight gain, decreased total feed intake and decreased feed conversion ratio compared to the infected non-treated group. *S. cerevisiae* could act as a growth promotor, because of it is natural improvement of digestibility and absorption of nutrients and controlling infections by enteric pathogens **(Cruickshank 2002)**. The pathological results of the infected treated groups showed mild vacuolar degeneration of intestinal epithelium (fig.11). On the other hand, the infected non treated group showed reduction in the growth, this may be due to toxin production, utilization of nutrients essential to the host or suppression of microbes that synthesize vitamins or other host growth factors **(Celik et al., 2008)**. These results are proved by the histopathological studies, where degeneration, necrosis and sloughing of intestinal epithelium and leukocytic infiltration, in addition to atrophy of intestinal glands in the infected none treated group (fig.4).

From the previous results, we can conclude that *Saccharomyces cerevisiae* added to the chicken ration at 2% has a potentiating on the growth rate, specific and non specific immune responses in broilers.

Table (1): Some biochemical parameters (Mean ± S.E.) in chickens experimentally infected with *E coli* and administrated *Saccharomyces cerevisiae* for 4 weeks.

Group	AST IU/L	T.P gm/dl	Alb. gm/dl	Glob. gm/dl	A/G ratio	Uric acid mg/dl	Creatinine mg/dl
I	163.0d ±2.07	4.00c ±0.18	1.88a ±0.11	2.12b ±0.15	0.89a ±0.08	3.58d ±0.09	0.43ab ±0.05
II	165.1d ±2.67	6.09b ±0.74	1.93a ±0.14	4.16a ±0.67	0.47b ±0.07	3.50d ±0.22	0.27c ±0.02
III	160.1d ±1034	6.62ab ±0.33	2.16a ±0.50	4.46a ±0.29	0.49b ±0.14	3.80d ±0.16	0.25c ±0.02
IV	192.2c ±1.90	6.68a ±0.40	2.00a ±0.45	4.68a ±0.59	0.44b ±0.15	7.34ab ±0.43	0.44ab ±0.07
V	207.1b ±2.16	6.78a ±0.16	2.08a ±0.19	4.70a ±0.31	0.42b ±0.09	5.03c ±0.37	0.35bc ±0.03
VI	247.5a ±3.03	3.22d ±0.43	0.66b ±0.24	2.56b ±0.30	0.26c ±0.09	7.76a ±0.60	0.54a ±0.02

Table (2): Some biochemical parameters (Mean ± S.E.) in chickens experimentally infected with *E coli* and administrated *Saccharomyces cerevisiae* for 6 weeks.

Group	AST IU/L	T.P gm/dl	Alb. gm/dl	Glob. gm/dl	A/G ratio	Uric acid mg/dl	Creatinine mg/dl
I	190.3b ±1.38	4.13c ±0.03	1.93a ±0.04	2.20cd ±0.04	0.88a ±0.04	4.50a ±0.17	0.47a ±0.03
II	191.86b ±3.29	4.49bc ±0.09	1.96a ±0.07	2.53bc ±0.11	0.79a ±0.06	4.77a ±0.17	0.38b ±0.02
III	188.8b ±0.54	5.06a ±0.19	2.02a ±0.14	3.04a ±0.14	0.67a ±0.06	4.60a ±0.18	0.31c ±0.02
IV	191.68b ±4.65	4.17c ±0.13	1.90a ±0.13	2.27cd ±0.10	0.85a 0.09	4.78a ±0.39	0.45a ±0.02
V	188.9b ±2.58	4.32bc ±0.07	1.98a ±0.12	2.3bcd ±0.12	0.87a ±0.09	4.76a ±0.32	0.48a ±0.02
VI	246.94a ±2.66	3.63d ±0.11	1.46b ±0.09	2.18d ±0.08	0.67a ±0.05	4.90a ±0.19	0.50a ±0.01

Means with the same letter in the same column are non significant at P<0.05

Table (3): Some immunological parameters (Mean ± S.E.) in chickens experimentally infected with *E coli* and administrated *Saccharomyces cerevisiae* for 4 weeks.

Group	Lysozyme activity µg/ml	Bactericidal activity mm	HI titer
I	0.69c ±0.07	1.68c ±0.25	3.46d ±0.22
II	0.94b ±0.05	2.61b ±0.20	5.41c ±0.24
III	1.68a ±0.06	3.02a ±0.38	8.69a ±0.42
IV	0.74c ±0.07	1.12d ±0.29	3.67d ±0.30
V	0.82c ±0.09	1.75c ±0.34	5.89c ±0.38
VI	0.48d ±0.04	0.72e ±0.16	2.00e ±0.28

Means with the same letter in the same column are non significant at P<0.05

Table (4): Some immunological parameters (Mean ± S.E.) in chickens experimentally infected with *E coli* and administrated *Saccharomyces cerevisiae* for 6 weeks.

Group	Lysozyme activity µg/ml	Bactericidal activity mm	HI titer
I	0.73c ±0.08	1.54c ±0.18	3.96d ±0.29
II	1.14b ±0.10	2.72b ±0.22	5.61c ±0.32
III	1.74a ±0.15	4.25a ±0.25	8.65a ±0.38
IV	0.82c ±0.09	1.71c ±0.21	3.72d ±0.25
V	1.01d ±0.12	2.98b ±0.22	5.82c ±0.41
VI	0.52e ±0.06	0.78d ±0.11	2.18e ±0.14

Means with the same letter in the same column are non significant at P<0.05

Table (5): Some growth performance parameters (Mean ± S.E.) in chicken experimentally infected with *E coli* and administrated *Saccharomyces cerevisiae* for 4 weeks.

Group	Mean body wt/gm	Mean body wt gain, /gm	Mean total feed intake, g/bird	FCR
I	998a ±14.20	650a ±12.15	1260b ±14.21	1.941a ±0.11
II	1025ab ±18.15	664a ±13.10	1251b ±12.28	1.88ab ±0.12
III	1175c ±15.38	767b ±16.42	1298a ±15.28	1.69b ±0.09
IV	828d ±14.15	482c ±12.91	921c ±12.25	1.91a ±0.13
V	901e ±14.95	542d ±13.38	975d ±12.92	1.80ab ±0.11
VI	815d ±10.25	473c ±9.38	901c ±10.34	1.94a ±0.10

Means with the same letter in the same column are non significant at P<0.05

Table (6): Some growth performance parameters (Mean ± S.E.) in chicken experimentally infected with *E coli* and administrated *Saccharomyces cerevisiae* for 6 weeks.

I	Mean body Wt /gm	Mean body wt gain, /gm	Mean total feed intake, kg/bird	FCR
II	1908a ±14.55	910a ±13.14	1825a ±15.21	2.01a ±0.12
III	1955a ±15.25	930ab ±15.32	1775b ±24.24	1.91ac ±0.09
IV	2176c ±13.58	1001c ±11.98	1725b ±28.94	1.72bc ±0.10
V	1671d ±13.81	843d ±10.52	1625d ±15.45	1.93ac ±0.12
VI	1775e ±15.05	874e ±10.01	1605d ±13.81	1.84c ±0.09
I	1650d ±11.82	835d ±9.82	1691c ±15.96	2.03a ±0.14

Means with the same letter in the same column are non significant at P<0.05

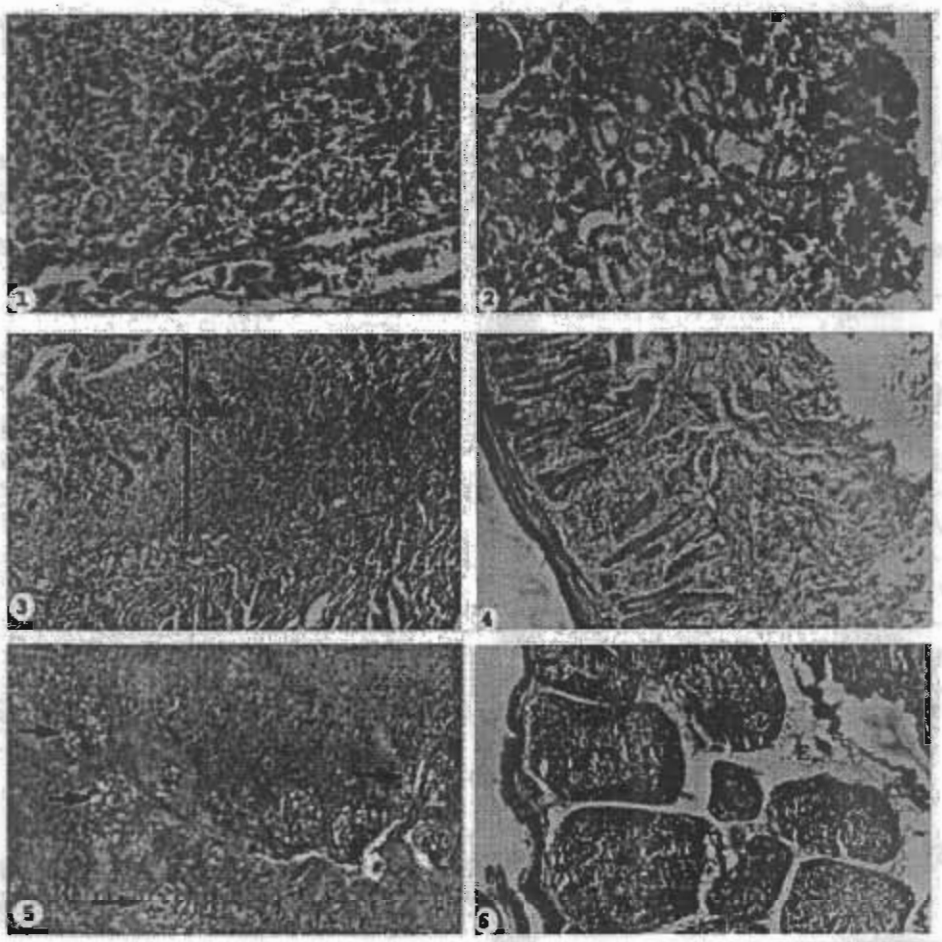

Fig.(1): liver, infected group (*E coli* only) at 6 weeks, showing congestion (C) of hepatic blood vessels, degeneration and necrosis of hepatocytes along with focal hepatitis characterized by aggregation of macrophages, lymphocytes and few heterophil. H&E. X 40.

Fig.(2): kidney, infected group (*E coli* only) at 6 weeks, showing congestion of blood vessels and mild focal interstitial aggregations of mononuclear cell infiltrations consists of macrophages and few lymphocytes. H&E. X 40.

Fig.(3): Heart, infected group (*E coli* only) at 4 and 6 weeks, showing thickening of pericardium due to fibrinous exudation, congestion of blood vessels and infiltration with macrophages, and small number of lymphocytes and plasma cells H&E. X 10.

Fig.(4): Intestine, infected group (*E coli* only) at 6 weeks, showing degeneration, necrosis and sloughing of intestinal epithelium and leukocytic infiltration (L). H&E. X 10.

Fig.(5): Spleen, infected group (*E coli* only) showing lymphoid depletion in the white pulp and peri-arteriolar sheathes. H&E. X 10

Fig.(6): Bursa, infected group (*E coli* only) showing mild to moderate lymphoid depletion of lymphoid follicles (arrows) and inter-follicular edema(E). H&E. X 40.

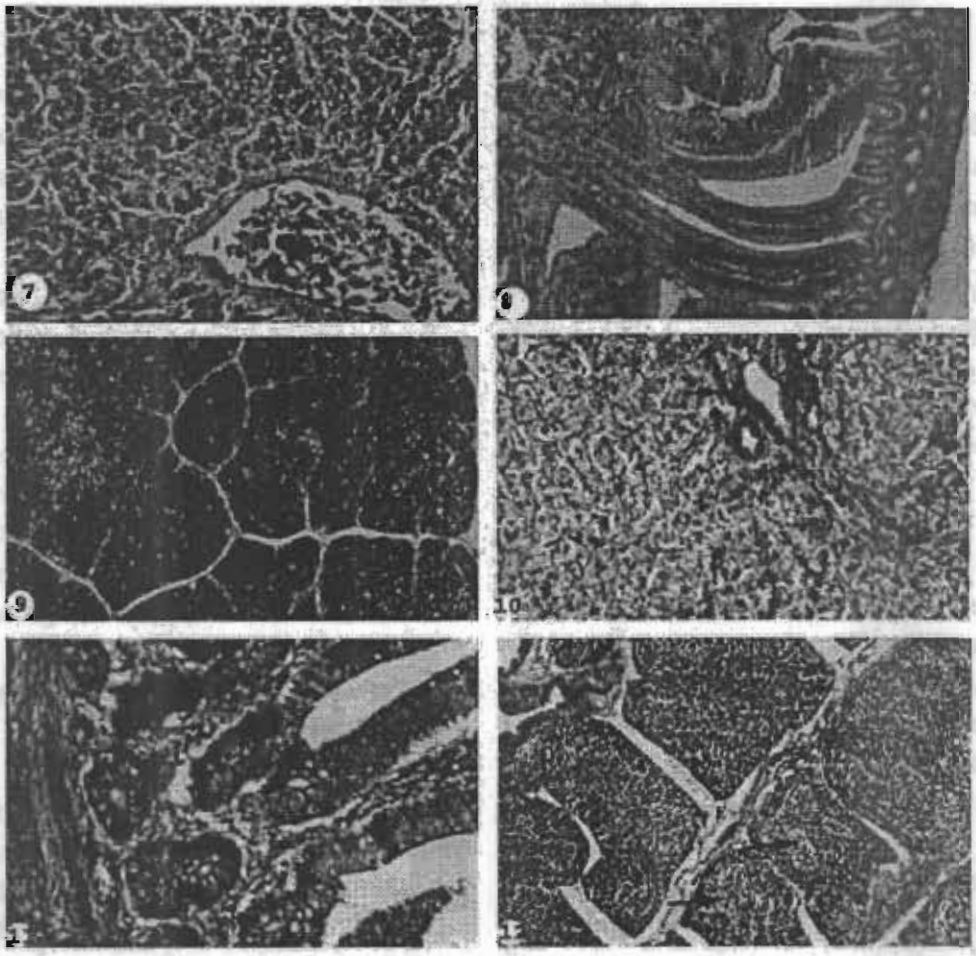

Fig.(7): Liver, treated group (*S. cerevisiae* only) at 6 weeks, showing mild vacuolar degeneration and congestion of central veins and hepatic sinousoids. H&E. X 40.

Fig.(8): Intestine, treated group (*S. cerevisiae* only) showing normal histological architecture of epithelial cell lining the villi. H&E. X 10.

Fig.(9): Thymus, treated group (*S. cerevisiae* only), the 2% dose showing focal hyperplasia along with mild congestion of blood vessels. H&E. X10.

Fig.(10): Liver, infected treated group (fed *S. cerevisiae* and infected with E.coli) at 6 weeks, showing vacuolar degeneration of hepatocytes (V) and mild hyperplasia of portal area (arrows). H&E. X 10.

Fig.(11): Intestine, infected treated group (fed *S. cerevisiae* and infected with E.coli) at 4 weeks, showing mild vacuolar degeneration of intestinal epithelium. H&E. X 40.

Fig.(12): Bursa, infected treated group (fed *S. cerevisiae* and infected with E.coli) at different ages, all doses showed slight, mild focal depletion of lymphoid follicles with mild inter-follicular edema. H&E. X 10.

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الملخص العربى

دراسات باثولوچية إكلينيكية على إضافة الخمائر للعلائق فى بدارى التسمين المصابة بالميكروب القولونى

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تضمنت الدراسة مائتان وأربعون من الدجاج بعمر يوم واحد وقسموا إلى 6 مجموعات كالآتى :

المجموعة الأولى : المجموعة الضابطة، المجموعة الثانية والثالثة : مجموعة معالجة بإضافة سكروميسيز سيرفيسيا (خميرة الخبز الجافة) فى العلف بنسبة 0,5 و 2%(5 جرام و 20 جرام / كجم علف) لمدة 6 أسابيع، المجموعة الرابعة والخامسة : عولجت بسكروميسيز سيرفيسيا فى العلف بنسبة 0.5 و 2% وأصيب بالميكروب القولونى عند عمر 7 أيام ، المجموعة السادسة : مجموعة مصابة بعترة الميكروب القولونى.

وقد تم أخذ ستة عينات عشوائية لتجميع الدم، المصل وأنسجة بعض الأعضاء الداخلية (الكبد، القلب، الكلى، الأمعاء، الطحال، البيرسا ، الغدد الثيماوية) من جميع المجموعات عند عمر 4 ، 6 أسابيع لإجراء بعض القياسات الكيماوية والفحوصات المناعية وكذلك الفحص النسيجى وقد أسفرت الدراسة عن النتائج التالية :

زيادة فى إنزيم أسبرتيت أمينوتر أنسفريز ونقص فى البروتين الكلى والزلال فى المجموعة المصابة، بينما وجد نقص فى الكرياتينين وحمض البوليك فى المجموعات التى عولجت بالخميرة وأصيبت بالميكروب.

وأظهرت النتائج المناعية فى المصل زيادة فى الليزوزيم والقدرة على قتل البكتريا والأجسام المضادة لڤيروس النيوكاسلب فى المجموعات المعالجة بالخميرة، بينما وجد نقص فى هذه القياسات بالنسبة للمجموعات المصابة. كما وجد زيادة فى وزن الطيور ومعدل التحويل الغذائى وقلة كمية العلف المستهلكة فى المجموعة المعالجة بسكر وميسيزسيرفيسيا 2% وأيضاً المجموعة المصابة والتى عولجت بسكروميسيزسيرفيسيا 2%.

مماسبق نستنتج أن خميرة سكروميسيز يمكن أن تستخدم كمحفز حيوى للنمو ومنشط للمناعج فى بدارى التسمين.