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Evaluation of Lactobacilli and active dry yeast in the prevention and control of quail colibacillosis

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Two experiments were carried out to evaluate the efficacy of the administration of active dry yeast and/or lactobacillus preparation (AVI-BAC), either before or after the infection with antibiotic resistant field strain of *Escherichia coli* O127 (*E. coli* O127) in controlling the severity of infection in quail chicks. The quail chicks of the different experimental groups were infected orally for two successive days with $3x10^7$ CFU of *E. coli* O-127 as an individual dose. The used field strain proved to be highly pathogenic for quails. Probiotics were supplemented in the drinking water for the different treatment groups at a dose level of 0.5 gm/L. The results revealed that the inclusion of lactobacilli or active dry yeast before *E. coli* infection has been highly effective in reducing mortality rate, organ invasion and the number of *E. coli* positive quail chicks. In addition, it decreased the severity of macroscopic and microscopic lesions in different organs in the probiotic treated groups as compared to the infected controls. Lactobacilli preparations were more efficient in controlling the severity of the infection. On the other hand, the administration of yeast and /or lactobacilli after inducing *E. coli* infection reduced the mortality rate and the severity of lesion score in different organs but probiotics failed to protect quail chicks against the infection. It has been proved that the two probiotics have synergistic effect in controlling collibacillosis in quails.

In recent years, a great attention was paid towards quail farming as an alternate to fulfill the increasing demands for the poultry meat.

Colibacillosis is a common systemic infection caused by any of the different serotypes of *Escherichia coli* (Barnes, *et al*., 2003). Quails of all ages are susceptible to diseases caused by *E. coli,* resulting in significant economic losses due to high morbidity, increased medication bills and condemnation of the infected quails (Abou El-Makarem and Ali, 1997; Barnes, *et al*., 2003; Hammouda, 1992; Reddy and Koteeswaran, 1994).

On the other hand, it was found that the misuse of different antibiotics in the treatment of poultry diseases or as growth promoter feed additives resulted in the emergence of resistant strains of bacteria that are difficult, if not impossible, to treat. Thus, many researches were done to find other safe and effective alternate. One of the alternatives, which have been introduced, is the use of probiotics.

Probiotics are heterogeneous group of live preparations that contains microorganisms or microbial metabolites from various sources (Fuller, 1989) and they are used to promote the growth of food animals and to control microbial invasion by the intestinal pathogens.

Our research project was designed to evaluate the efficacy of two commercial probiotic preparations (Active dry yeast and lactobacilli combination) in controlling *E. coli* in quails either as prophylaxis before the infection or as therapy after an established infection**.**

Material and Methods

Quail chicks. One-day-old quail chicks were collected as from a commercial hatchery and litter reared under complete hygienic conditions. The quail chicks were subjected to bacteriological examinations and proved to be free from pathogenic *E.coli*.

***E. coli* field strain.** A chicken strain of *E. coli* was isolated from broiler chicken flocks exhibiting severe septicaemia. Morphological, cultural and biochemical identifications were carried out according to (Halt *et al*., 1996). The field strain was initially identified as *E. coli* and subjected to serological typing using *E. coli* polyvalent and monovalent O antisera (Behring Werke Ag., Marburg-Lahn, Germany). Susceptibility of the isolated *E. coli* field strain (O127) was screened against different antibiotics by disc diffusion

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Table (1): Antibiotic sensitivity test of *E. coli* (field strain O127).

Antibiotic Disc	Disk Conc. (ug)	Diameter of inhibition zone (mm)		Comment
		Standard Inhibition Zone (>/=)	Result	
Amoxycillin (AML)	25	16	0	Resistant
Chloramphenicol (C)	30	21	18	Resistant
Ciprofloxacin (Cip)	5	21	18	Resistant
Colistin sulphate(CT)	25	11	14	Sensitive
Danofloxacin (DFX)	5	21	0	Resistant
Erythromycin (E)	15	15	0	Resistant
Enrofloxacin (ENR)	10	30	14	Resistant
Flumequin (UB)	30	22	0	Resistant
Gentamycin (GM)	10	19	12	Resistant
Norfloxacin (NOR)	10	28	0	Resistant
Oxytetracycline (OT)	30	19	0	Resistant
Trimethoprim (SXT)	1.25	24	0	Resistant

technique adapted according to (Koneman *et al*., 1992) using antibiotic discs collected from Oxoid (Table 1).

Probiotics. Two commercial probiotics used in this study: (i) Active dry yeast (*Saccharomyces cervisiae*) obtained from Holw Elsham Company for Powder and Light Food Industry, Sixth- October City, Giza, Egypt. (ii) AVI-BAC: A commercial probiotic consists of combination of three species of lactobacillus (*Lactobacillus acidophilus, Lactobacillus planterum and Lactobacillus brevis*). AVI- BAC obtained from Sure Pharmaceutica Company, Heliopolis, Cairo, Egypt and produced by Pro- Byn International Inc, Lombard, Illinois 60148 USA. Probiotics were supplemented in the drinking water at a dose level of 0.5gm/L (Gram per liter).

Piolet pathogenicity testing of *E. coli* in quails. Ten 4-day-old quail chicks were randomly collected and each was infected subcutaneously with $3x10^7$ CFU of the tested *E. coli* strain (O127). The quails were fed on starter commercial quail feed, supplemented with fresh hygienic drinking water and kept under observation. Signs, mortalities and lesions were recorded. Resiolation of the inoculated field strain (*E. coli* O127) from the internal organs for checking backs its serotype identity was performed.

Experimental design.

Experiment (1):Evaluation of the administration of probiotics pre-infection with *E. coli* (O127). A total of 120 ten-days-old quail chicks were sorted out into six equal treatment groups, reared in separated litter breeding pens. Sanitation and hygiene were considered. Chicks of all groups were fed on a starter commercial quail feed from day old till the end of the experiment. Chicks of groups (1) and (4) received fresh hygienic drinking water throughout the experiment. Chicks of groups (2) and (5) received fresh hygienic drinking water supplemented with 0.5gm/L of active dry yeast (*Saccharomyces cervisiae)* from day old till the end of the experiment. Chicks of groups (3) and (6) received fresh hygienic drinking water supplemented with 0.5gm/L of Lactobacillus preparation (AVI-ABC) from day old till the end of the experiment. At the fourth day of the experiment chicks of groups (4), (5) and (6) were infected orally with $3x10^7$ CFU of *E. coli* (O127) as an individual dose for two successive days. The chicks of all groups were observed daily for the appearance of signs, mortalities and lesion scores which were recorded throughout the experimental observation period.

Ten days post-infection, the quails were sacrificed, lesions were recorded and samples were collected aseptically from the internal organs (heart, lung, liver and kidney) and subjected to bacteriological reisolation attempts and histopathological investigation.

Experiment (2):Evaluation of the administration of probiotics post-infection with *E. coli* (O127). A total of 150 four-day old quail chicks were included in this study. All quails from the different groups were fed on starter commercial quail feed from day old till the end of the experiment. Thirty chicks were reared in a separate pen and received starter commercial quail feed and fresh hygienic drinking water throughout the experiment. The remainder quail chicks (120 Quails) were infected orally with 3×10^7 CFU of O127 field strain of *E. coli* for two successive days as an individual dose. Those quails were fed on starter commercial quail feed thereafter. On the fourth day post-infection the chicks were sorted out into four groups

Table (2): Piolet pathogenicity test of *E. coli* (field strain O127) in quails.

Bacterial field strain	Dose of experimental infection (CFU/CHICK)	Infected quails		Route of inoculation	Mortality	
		No.	Age		No.	%
***E. coli* (O127)**	$6x10^7$	10	4 days old	S/C	10/10	100%

Table (3): Mortality rate of quail chicks infected with *E. coli* (field strain (O127) after probiotic administration (Experiment 1).

Group No.	Treatment	Mortality/days post-infection										Mortality	
		1	2	3	3	4	5	6	7	8	9	10	Total No.	%
1	Blank control	0	0	0	0	0	0	0	0	0	0	0	0/20	0%
2	Lactobacillus only	0	0	0	0	0	0	0	0	0	0	0	0/20	0%
3	Yeast only	0	0	0	0	0	0	0	0	0	0	0	0/20	0%
4	*E. coli* infection	6	5	1	2	1	1	0	0	0	0	0	16/20	80%
5	*E.coli*+ Lactobacillus	2	3	0	0	0	0	0	0	0	0	0	5/20	25%
6	*E. coli* + yeast	2	1	0	0	1	0	0	0	0	0	0	4/20	20%

Table (4): Reisolation rate of *E. coli* (field strain O127) from infected quail chicks administrated different probiotics before the infection.

Group No.	Treatment	Liver		Heart		Lung		Kidney		Positive Chicks	
		No.	%	No.	%	No.	%	No.	%	No.	%
1	**Blank control**	0	0	0	0	0	0	0	0	0	0
2	**Lactobacillus only**	0	0	0	0	0	0	0	0	0	0
3	**Yeast only**	0	0	0	0	0	0	0	0	0	0
4	***E. coli* infection**	4/4	100	4/4	100	4/4	100	4/4	100	4/4	100
5	***E. coli*+ Lactobacillus**	7/15	46.67	5/15	33.33	5/15	33.33	6/15	40	9/15	60
6	***E. coli* + yeast**	8/16	50	7/16	43.75	7/16	43.75	5/16	31.25	11/16	68

(30 Chicks each) as follows:

Group (1): Infected chicks fed commercial feed and supplemented with fresh drinking water till the end of experiment (Positive control).

Group (2): Infected chicks fed commercial feed and supplemented with drinking water containing 0.5gm/L of lactobacilli preparation (AVI-BAC) till the end of experiment.

Group (3): Infected chicks fed commercial feed and supplemented with drinking water containing 0.5gm/L of active dry yeast *(Saccharomyces cervisiae)* till the end of experiment.

Group (4): Infected chicks fed commercial feed supplemented with drinking water containing 0.5gm/L of lactobacill preparation (AVI-BAC) and 0.5gm/L active dry yeast *(Saccharomyces cervisiae)* till the end of experiment.

The chicks from all groups were observed daily for the development of signs, mortalities and lesions which were recorded throughout the experimental observation period.

Quail chicks were sacrificed ten days post medication and quails were sampled for lesion score. Organs were sampled aseptically for the reisolation of the inoculated field strain and for the histopathological examination.

Bacteriological examination. Re-isolation and identification of the inoculated *E. coli* field strain (O127) from the different internal organs of infected quail chicks were done using MacConkey agar medium and Congo red agar medium according to (Berkhoff and Vinal, 1986).

Histopathological examination. Internal organs that showed lesions or any abnormal changes were collected, then fixed in 10% formol saline solution. The collected samples were dehydrated, cleared and embedded in paraffin wax, and then specimens were sectioned to 4 micron thickness and stained by Harris haematoxylin and eosin (Harris, 1990).

Results and Discussion

The pathogenicity testing of *E. coli* field strain (O127) that used in this research work (Table 2)

Table (5): Mortality rate of quail chicks infected with *E. coli* (field strain (O127) before probiotic administration (Experiment 2).

Group No.	Treatment	Mortality/days post-infection										Mortality	
		1	2	3	4	5	6	7	8	9	10	Total No.	%
I	Blank control	0	0	0	0	0	0	0	0	0	0	0/30	0%
II	*E. coli* infection	5	7	15	0	0	0	0	0	0	0	27/120	22.5%
1	*E. coli* control	-	-	-	5	2	1	4	1	1	0	14/23	60%
2	*E. coli*+ Lactobacillus	-	-	-	4	3	2	2	1	0	0	12/23	52%
3	*E. coli* + yeast	-	-	-	3	0	2	1	1	0	0	7/23	30%
4	*E. coli*+Lactobacillus + yeast	-	-	-	2	2	1	1	0	0	0	6/23	25%

Table (6): Reisolation rate of *E. coli* (field strain O127) from infected quail chicks receiving different probiotics after infection.

Group No.	Treatment	Liver		Heart		Lung		Kidney		Positive Chicks	
		No.	%	No.	%	No.	%	No.	%	No.	%
I	Blank control	0/30	0%	0/30	0%	0/30	0%	0/30	0%	0/30	0%
1	*E. coli* infection	9/9	100	9/9	100	9/9	100	8/9	88.89	9/9	100
2	*E. coli*+ Lactobacillus	10/11	81.81	9/11	81.81	9/11	81.81	8/11	72.72	11/11	100
3	*E. coli* + yeast	15/16	93.38	13/16	81.25	14/16	87.59	13/16	81.25	16/16	100
4	*E. coli* + Lactobacillus+ yeast	14/17	82.35	12/17	70.38	12/17	70.38	10/17	58.82	17/17	100

Table (7): Histopathlogical findings recorded in lungs of quails receiving probiotics before and after infection with *E. coli* (Field strain 0127).

Treatment	* Lesions of the lung									
	Congestion	Haemorrhages	Thrombosis	Emphysema	Oedema	Perivascular oedema	Inflammatory cells Infiltration	Bronchial epithelium hyperplasia	Epithelization	Granulomatous structure
Yeast	++	-	-	-	-	-	-	-	-	-
Lctobacilli	+	-	-	+	-	-	-	-	-	-
E. coli infection	++++	++++	++++	++++	++++	++++	++++	++++	++++	++++
Yeast + *E. coli*	++	++	++	++	++	++	++	++	++	-
E. coli + yeast	+++	+++	++	++	++	++	+++	++	+++	++
Lactobacilli + *E. coli*	+	-	+	+	+	+	+	+	-	-
E. coli + lactobacilli	++	+++	++	+++	++	++	+++	++	+	-
E. coli + Yeast+lactobacillii	++	++	++	-	++	++	++	-	-	+

* Histopathology Score: According to Barnes, *et al*., (2003) and Reddy and Koteeswaran, (1994)
- = No lesion. + = Mild lesion. ++ = Pronounced lesion. +++ = Moderate lesion. ++++ =Severe lesion.

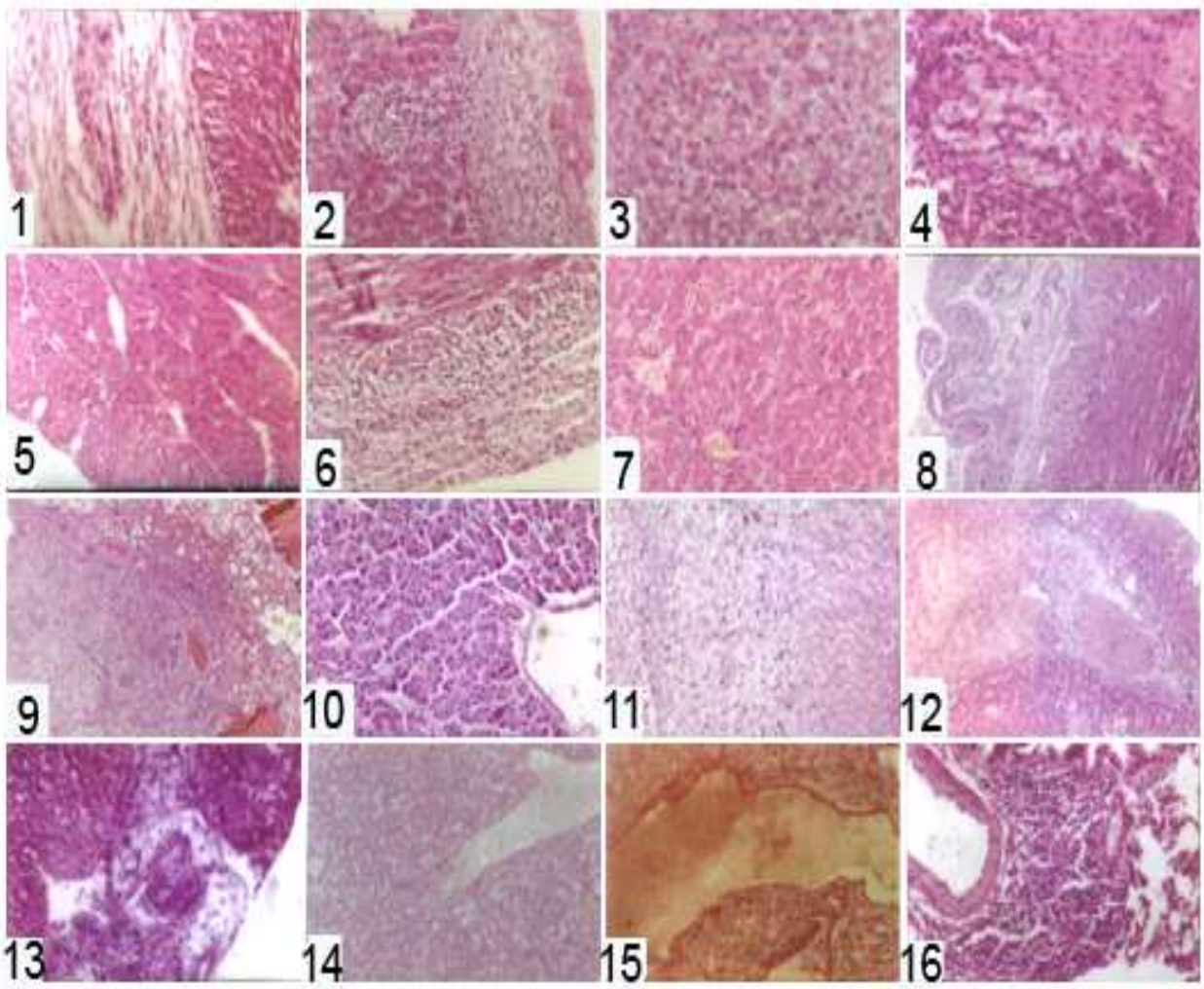

Fig.(1): Heart of experimentally infected quail with *E. coli* showing pericarditis (H&E ×400).
Fig.(2): Liver of experimentally infected quail with *E. coli* showing perihepatitis (H&E ×400).
Fig.(3): Liver of experimentally infected quail with *E. coli* showing degenerative changes of hepatocytes. (H&E×400).
Fig.(4): Lung of experimentally infected quail with *E. coli* showing exudation of proteinacious fluid (H&E ×400).
Fig.(5): Heart of quail given yeast only showing no defined lesions (H&E ×250).
Fig.(6): Heart of quail given yeast then infected with *E. coli* showing mild pericarditis and myocarditis (H&E ×250).
Fig.(7): Liver of quail given yeast then infected with *E. coli* showing congestion (H&E ×250).
Fig.(8): Heart of quail infected with *E. coli* and treated with yeast showing pericarditis (H&E ×250).
Fig.(9): Lung of quail infected with *E.coli,* and treated with yeast showing granulomatous structure (H&E ×250).
Fig.(10): Liver of quail given only lactobacilli showing no defined lesions (H&E ×250).
Fig.(11): Heart of quail infected with *E. coli* and treated with lactobacilli showing pericarditis (H&E ×250).
Fig.(12): Liver of quail infected with *E. coli* and treated with lactobacilli showing coagulative necrosis (H&E ×250).
Fig.(13): Heart of quail infected with *E. coli* and treated with yeast and lactobacilli showing hyperplasia of the pericardium (H&E ×250).
Fig.(14): Liver of quail infected with *E. coli* and treated with yeast and lactobacilli showing thorombosis and microthorombosis (H&E ×100).
Fig.(15): Lung of quail infected with *E. coli* and treated with yeast and lactobacilli showing thorombosis (H&E ×100).
Fig.(16): Lung of quail infected with *E. coli* and treated with yeast and lactobacilli showing inflammatory cells infiltration (H&E ×100).

Table (8) : Histopathlogical findings recorded in the heart and liver of quails receiving probiotics before and after infection with *E. coli* (field strain 0127).

Treatment	* Heart lesion			* Liver lesion					
	Pericarditis	Myocarditis	Perihepatitis	Degenerative changes	Coagulative necrosis	Inflammatory cells	Congestion	Haemorrhage	Thorombpsis
Yeast	* -	-	-	-	-	-	-	-	-
Lactobacilli	-	-	-		-	-	-	-	-
E. coli infection	++++	++++	++++	++++	++++	++++	++++	++++	++++
Yeast + *E. coli*	++	++	++	++	++	++	++	++	++
E. coli + yeast	+++	+++	++	++	++	++++	++++	++++	++++
Lactobacilli + *E. coli*	-	-	+	+	-	++	+	-	+
E. coli + lactobacilli	+	+	+++	++	++	++	++	++	+++
E. coli+ (Yeast+ lactobacilli)	++	++	++	++	++	++	++	++	++

* Histopathology Score: According to Barnes, *et al*., (2003) and Reddy and Koteeswaran (1994)
- = No lesion. + = Mild lesion. ++ = Pronounced lesion. +++ = Moderate lesion. ++++ = Severe lesion.

revealed high susceptibility of quails to *E. coli* isolated from chickens. Hundred percent mortalities occurred within 24 h post-infection indicating that quails might play an important role in the perpetuation and spread of *E. coli* infection in chickens. These results agreed with Reddy and Koteeswaran (1994) who found that there was a relative susceptibility of Japanese quails to serotypes of pathogenic *E. coli* isolated from chickens. They found that seven-day and seven-week-old quails were susceptible to all tested serotypes of chicken *E. coli* as evidenced by more numbers of deaths and quickness of mortality even at lower concentrations. The lesions observed in most of the infected quail chicks were typical to avian colibacillosis. Also, the results shown in Table (2) showed that the used *E. coli* strain (O127) proved to be highly pathogenic for quails. These results agreed with Abou El-Makarem and Ali (1997) who found that *E. coli* serogroup O127 was one of the most predominant *E. coli* (O) serogroups isolated from the lung tissues of living and slaughtered quails suffering from respiratory disorders.

The antibiotic susceptibility testing of the *E. coli* (O127) revealed its sensitivity only to colistin sulphate and its resistance to the other twelve antibiotic discs used (Table 1). This result agreed with Barnes, *et al*., (2003) who reported that strains of *E. coli* from poultry are frequently resistant to one or more drugs especially if they have widely used in poultry industry over a long period or as a result of misused administration as growth promoter feed additives at low concentrations resulting in the development of resistant strains of bacteria. Tables (3) and (5) showed that the administration of the lactobacilli preparation (AVI-BAC) either before or after *E. coli* infection reduced the mortality rate from 80% and 60% in the infected non-treated groups to 25% and 52% in the treated groups respectively. Lactobacilli preparation was also able to reduce the number of *E. coli* positive quail chicks and organ invasion from 100% in the infected non-treated quails to 60% in lactobacillus- treated quail chicks only when administrated before *E. coli* infection (Table 4). On the other hand, lactobacilli failed to protect chicks after the induction of infection (Table 6). Lactobacilli are major producer of lactic acid Fuller, (1997) and Humphrey *et al*., (1993). The mechanism attributed to lactic acid bacteria to produce their protective effect against enteropathogenic bacteria (Juven, *et al*., 1991; Pascual *et al*., 1999) is achieved through the following effects: (a) Reduction of the intestinal colonization by the invasive enteropathogens which is attributed to the decrease in caecal

hydrogen ion concentration, increased lactic acid concentration and increased undissociated volatile fatty acids concentration (Schneitz, *et al*., 1990; Hinton *et al*., 1990; Corrier *et al*., 1991; Hume *et al*., 1992; Vandenberg, 1993; Barnhart *et al*., 1999; Ezz-Eldeen, and Zouelfakar, 2003). (b) Competition with the pathogen for adhesion sites or nutritional sources (Nisbet *et al*., 1993; Bernet *et al*., 1994; Hejliceck *et al*., 1995; Pascual *et al*., 1999). (c) Stimulation of the systemic immune responses (Muir *et al*., 1998; Quéré and Girard, 1999; Huang *et al*., 2004). Regarding the effect of active dry yeast (*Saccharomyces cervisiae*) in controlling *E. coli* infection in quails, Tables (3) and (5) revealed that it was able to reduce the mortality percentages from 80% and 60% in the infected non-treated quail chicks to 20% and 30% in treated ones in the administration before and after *E. coli* infection respectively. The rate of reisolation of *E. coli* from internal organs of infected quail chicks were greatly reduced from 100% in the infected, non-treated groups to 31.25% (from kidneys) and 50% (from liver). In quail chicks administrated yeast before *E. coli* infection with great reduction of the number in positive quail chicks from 100% in infected non-treated quails to 68% in infected, yeast-treated ones. The active dry yeast achieves its protective effect against enteropathogens through different mechanisms including: a) Competition with the pathogenic microorganisms for the adhesion sites and act as pathogen adherent bacteria that enter the gastrointestinal tract before the pathogenic bacteria can attach to the bird intestinal wall. Yeast does not permanently colonize the intestine, so yeast and yeast-bound pathogen should pass out of the bird during excretion thus minimizing bacterial colonization (Oyofo *et al*., 1989a,b ; Bernet *et al*., 1994). b) Inhibition of the production or the action of the bacterial toxins (Czerucka, *et al*., 1994 ; Brandäo *et al*., 1998). c) The yeast polysaccharides are very promising immuno-stimulating agents that increase both humoral and cell mediated immune responses (Jing *et al.,* 1989; Badr and El-Kholy, 2003).

Tables (5, 6) revealed that the addition of both 0.5 gm/L of the lactobacilli preparation (AVI-BAC) and 0.5 gm/L of active dry yeast in the drinking water of quail chicks previously infected with *E. coli*, resulted in reduction of the mortalities and the organ invasion in the treated groups more than did by either preparation when used alone. These results agreed with Fuller, (1995) who reported that probiotic trials could be affected by some factors such as the type of the biotherapeutic agent, methods of probiotic production and administration, the viability of the preparation and the condition of the host and of the gut microbiota (Filho-Lima *et al*., 2000) in addition to the specificity of the protective mechanism could be another factor which could affect the action of the used probiotic. This might explain the synergistic effect of the lactobacilli and active dry yeast in protecting birds thus controlling *E. coli* infection in quails.

Histopathological findings of different lesions in lung (Table 7), heart and liver (Table 8) of quail chicks of different experimental groups revealed that the group infected with *E. coli* only showed pericarditis (Fig. 1) which extended to the parts of the myocardium resulting in myocarditis. There was thickening of the hepatic capsule due to oedema and inflammatory cell infiltration (Fig. 2), degenerative changes of the hepatocytes and vascular and granular degeneration (Fig.3).

Other lesions included pronounced haemorrhages, emphysema,inflammatory cells infiltration, preivascular oedema, thrombosis, epithelization of alveoli, hyperplasia of epithelial lining of secondary and tertiary bronchi with the activation of goblet cells, granuloma and exudation of proteinacious fluid (Fig.4).

In the non-infected groups that given yeast only, no defined lesions were detected in the heart (Fig.5), liver. In groups given yeast before the infection there was mild pericarditis and myocarditis (Fig.6) and congested hepatic blood vessels (Fig.7). In the lung, there were mild lesions of congestion, haemorrhages, emphysema, oedema, inflammatory cells infiltration and hyperplasia of the bronchial epithelium. On the other hand, groups treated with yeast after *E. coli* infection, showed more severe lesions either in the heart (Fig.8), or in the liver and lung (Fig.9) which showed more pronounced granuloma. The non-infected groups given lactobacilli only showed no defined lesions in the heart, liver (Fig. 10) and lung. In the groups receiving lactobacilli before the infection, very mild lesions in heart, liver and lung were observed. When lactobacilli was used as a treatment after the infection, lesions were more pronounced but milder than that when the yeast was used as treatment which appeared as pericarditis (Fig.11) and coagulative necrosis in the liver (Fig. 12). The dual treatment group of both yeast and lactobacillus after *E. coli* infection showed pronounced hyperplasia of

pericardium (Fig.13), pericarditis and myocarditis. The liver showed perihepatitis, degenerative changes in the hepatocytes, inflammatory cells infiltration, coagulative necrosis, congested hepatic blood vessels, haemorrhages, thrombosis and microthrombosis (Fig.14). The lung tissues showed pronounced haemorrhages and thrombosis (Fig.15), infiltration and inflammatory cells (Fig.16), oedema and granulomatous structure. It was noticed that the kidneys of all experimental groups showed no defined lesions. The above mentioned histopathological findings agreed to a great extent with that obtained from the bacteriological examination. In conclusion, both active dry yeast and lactobacilli are effective in controlling the severity of *E. coli* infection in quail chicks especially when given before the infection. Both probiotics have a synergestic effect against the infection with enteropathogenic *E. coli* in quails.

References

Abou El-Makarem, M. R and Ali, A. R. (1997): Bacteriological studies on mycoplasmas and *E. coli* in quails. J. Egypt. Vet. Med. Assoc. 57 (4): 1319-1329.

Badr, J. M. and El-Kholy, **M. M. (2003):** Some studies on yeast-supplemeented ration in salmonella infected layer chicks. J. Egypt. Vet. Med. Assoc. 63 (5): 23-26.

Barnes, H. J.; Vaillancourt, J. J. and Gross, W. B. (2003): Colibacillosis. In The Diseases of Poultry, 11th Ed. Y.M. Saif.; H.G. J.R. Barnes.; Glisson, A.M. Fadly; L.R. McDougald and D.E. Swayne Iowa State University Press, Ames, IA. pp. 631-622.

Barnhart, E. T.; Caldwell, D. J.; Crouch, M C.; Byrd, J. A.; Corrier, D. E. and Hargis, B. M.(1999): Effect of lactose
administration in drinking water prior to and during feed withdrawal on salmonella recovery from broiler crops and caeca. Poult. Sci. 78: 211-214.

Berkhoff, H. A. and Vinal, A. C. (1986): Congo red medium to distinguish between invasive and non-invasive *Escherichia coli* pathogenic for poultry. Avian Dis., 30 (1): 117-121.

Bernet, M.F.; Brassart, D.; Neeser, J. R. and Servin, A. L. (1994): *Lactobacillus acidophilius* LA1 binds to human intestinal cell lines and inhibits cell attachment and cell invasion by enterovirulent bacteria. Gut 35: 483-489.

Brandäo, R.L.; Castro, I. M. and Bambirra, E. A. (1998): Intracellular signal triggered by cholera toxin in *Saccharomyces boulardii* and *Saccharomyces cervisiae.* Appl. Env. Microbiol. 64: 564-568.

Corrier, D. E., Hargis, B.; Hinton, A. Jr.; Lindsay, D.; Caldwell, D.; Manning, J. and Deloach, J. R. (1991): Effect of anaerobic caecal microflora and dietary lactose on colonization resistance of layer chicks to invasive *Salmonella enteritidis.* Avian Dis., 35:337-343.

Czerucka, D.; Roux, I. and Rampal, P. (1994): *Saccharomyces boulardii* inhibits secretory gut-mediated adenosine 3, 5-cyclic monophosphate induction in intestinal cells. Gastroenterol., 106: 65-72.

Ezz-Eldeen, N. A. and Zouelfakar S. A. (2003): Role of lactic acid and/or garlic in controlling colonization and shedding of *Salmonella typhimurium* in chicks. J. Egypt Vet. Med. Assoc., 63: 75-86.

Filho-Lima, J. V. M.; Viera, E. C., and Nicoli, J. B. (2000): Antagonistic effect of *Lactobacillus acidophilus, Saccharomyces boulardii* and *Escherichia coli* combinations against experimental infections with *Shigella flexneri* and *Salmonella enteritids* subsp. *Typhimurium* in gnotobiotic mice. J. Appl. Microbiol., 88: 365-370.

Fuller, R. (1989): Probiotics in man and animals. J. Appl. Bacteriol. 66: 365-378.

Fuller, R. (1995): Probiotics: Their development and use. In. The Probiotics: Prospects of use in opportunistic infections. Eds., R. Fuller; P.J. Heidt; V. Rusch and Van, D. der Waajj, pp. 1-8. Herborn: Institute For Microecology.

Fuller, R. (1997): The importance of lactobacilli in maintaining normal microbial balance in the crop. Br. Poult. Sci. 18: 85-94.

Halt, J. G.; Krieg, N. R.; Sneath, P. H. A. and Williams, S. T. (1996): Bergey's Manual of Determinative Bacteriology, 9th Ed. Williams and Wilkins, Baltimore, Maryland, USA.

Hammouda, A. M. (1992): Studies on the bacterial causes of early quail mortalities. M.V.Sc. Thesis, Bacteriology, Fac. Vet. Med., Zagazig Univ., Zagazig, Egypt.

Harris, H. F. (1990): The rapid conversion of haematorytinin to haematinins staining reaction. J. Appl. Micros. Lab. Meth. 3: 777.

Hejliceck, K.; Soukupova, A. and Moltasova, M. (1995): Salmonellosis in 1-day old chicks caused by *Salmonella enteritidis*. Veterinarya 45: 16-18.

Hinton, A. Jr.; Corrier, D. E.; Spates G. E.; Norman, J. O.; , R. L.; Beier, R. C. and Deloach, J. R. (1990): Biological control of Salmonella typhimurium in young chickens. Avian Dis. 34: 626-633.

Huang, M. K.; Choi, Y. J.; Houde, R.; Lee, J. W.; Lee, B. and Zhao, X. (2004): Effect of Lactobacilli and acidophilic fungus on the production performance and immune responses in broiler chickens. Poult. Sci. 83:788-795.

Hume, M. E., Kubena, L. F., Beier, R. C.; Hinton, A. Jr., Corrier, D. E. and Deloach, J. R. (1992): Fermentation of [14C] lactose in broiler chicks by cecal anaerobes . Poult. Sci. 71: 1464-1470.

Humphrey, T. J.; Baskerville, A.; Whitehead, A.; Rowe, B. and Henley, A. (1993): Influence of feeding patterns on the artificial infection of laying hens with *Salmonella enteritidis* phage type 4. Vet. Rec., 132: 407- 409.

Jing,W.J.; Shiying,W.G., Hua, W.Y.; Bong,W.K.; Liang, L.B.; Min,W. and Fanxiaoping, X.P. (1989): Effect of yeast polysaccharide on HI antibody level of Newcastle disease and T-cell profileration. Chin. J. Vet. Med. 24 (2): 15-16.

Juven, B. J.; Meinersman, R. J.; and Stern, M. J. (1991): Antagonistic effects of lactobacilli and pediococci to control intestinal colonization by human enteropathogens in live poultry. J. Appl. Bacteriol. 70: 95-103.

Koneman, E. W., Allen, S. D.; Schrechen, W. M.; Berrjer, P. C. and Winn, W. J. (1992): Color Atlas and Textbook of Diagnostic Microbiology, 4th Ed. J. B. Lippencott Co., Philadalphia, USA.

Muir, W. I.; Bryden, W. L. and Husband, A. J. (1998): Evaluation of the efficacy of intraperitoneal immunization in reducing *Salmonella typhimurium* infection in chickens. Poult. Sci. 77: 1874-1883.

Nisbet, D. J.; Corrier, D. E.; Scanlan, C. M. ; Hollister, A. G. ; Beier, R. C. and Deloach, J. R. (1993): Effect of defined continuous-flow derived bacterial culture and dietary lactose on salmonella colonization in broiler chickens. Avian Dis. 37: 1017-1025.

Oyofo, B. A.; Deloach, J. R.; Corrier, D. L. ; Norman, J. O.; Ziprin, R. L. and Mollenhauer, H. H. (1989a): Prevention of *Salmonella typhimurium* colonization of broilers with d-mannose. Poult. Sci. 68: 1357-1360..

Oyofo, B. A.; Droleskey, R. E.; Norman, J. O.; Mollenhauer, H. H.; Ziprin, R. L.; Corrier, D. L. and Deloach, J. R. (1989b): Inhibition by mannose of in-vivo colonization of chicken small intestine by salmonella. Poult. Sci. 68; 1351- 1356.

Pascual, M.; Hugas, M.; Badiola, J. I.; Monfort, J. M. and Carriga, M. (1999): Lactobacillus salivarius CTC2197 prevents *Salmonella enteritidis* colonization in chickens. Appl. Environ. Microbiol. 65: 4981-4986.

Quéré, P. and Girard, F. (1999): Systemic adjuvant effect of cholera toxin in the chicken. Vet. Immunol. Immunopathol. 70: 135-141.

Reddy, Y. K. and Koteeswaran, A. (1994): Studies on experimental *Escherichia coli* infection in Japanese quails. Ind. Vet. J. 71: 959-963.

Schneitz, C. M.; Hakkinen, L.; Nuotio, E. and Mead, G. (1990): Droplet application for protecting chicks against salmonella colonization by competitive exclusion. Vet. Rec., 126: 510-517.

Vandenberg, P.A. (1993): Lactic acid bacteria, their metabolic products and interference with microbial growth. FEMS Micobiol. Rev. 12: 221-238.

تقييم مستحضرات اللاكتوباسيلاس والخميرة النشطة الجافة فى السيطرة على عدوى الإيشريشيا كولاى فى كتاكيت السمان

أجريت تجربتان لتقييم فعالية مستحضرات البروبيوتيك (الخلائق الإحيائية) وهى مستحضر الخميرة النشطة الجافة ومستحضر مخلوط اللاكتوباسيلاس فى وقاية وعلاج كتاكيت السمان من العدوى ببكتريا الإيشريشيا كولاى المجموعة الجسمية O127 المقاومة للمضادات البكتيرية المتداولة بحقل الدواجن. المجموعات التجريبية لكتاكيت السمان تم عدوتها إصطناعياً عن طريق الفم وليومين متتاليين بمعلق بكتريا الإيشريشيا كولاى O127 بجرعة ٣ × ١٠ ^٧ و.م.م. لكل كتكوت وتلك العترة ثبت إمراضيتها العالية لكتاكيت السمان، تم إضافة المستحضرات الدوائية للبروبيوتيك (الخلائق الإحيائية) فى ماء الشرب بمعدل نصف جرام لكل لتر ماء شرب فى مجموعات معاملات مختلفة. أثبتت النتائج أن إضافة اللاكتوباسيلاس أو الخميرة النشطة الجافة قبل حدوث العدوى بالإيشريشياكولاى كانت لها فعالية مرتفعة فى خفض معدلات النفوق وغزو الأعضاء الداخلية بالبكتريا. وأيضاً فى خفض حدة الإصابة بالأعضاء الداخلية سواء الآفات التشريحية أو التغيرات المجهرية وهذا عند مقارنتها بالمجموعة المصابة التى لم يقدم لها هذا العلاج. إعطاء مستحضرات البروبيوتيك (الخلائق الإحيائية) بعد حدوث إصابة يساعد فى خفض معدلات النفوق وشدة إصابة الأعضاء الداخلية ولكن بدرجة أقل من إعطائه كوقائى نظراً لأنه لم يحمى كتاكيت السمان من حدوث العدوى. الإستخدام المزدوج لمستحضرات اللاكتوباسيلاس والخميرة النشطة الجافة كان له تأثير تعاضدى أفضل من إستخدام أيهما بمفرده.