

EFFECT OF YEAST (*SACCHAROMYCES CEREVICIAE*) ON QUAIL FED RATION CONTAMINATED WITH OCHRATOXIN A

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Abstract

One hundred and twenty day-old, unsexed Japanese quail chicks were used in this study. The quail chicks were randomly assigned into four groups of 30 birds in each. The four experimental groups were fed on a basal diet, supplemented with 2gm yeast / kg diet , 50µg OA / kg diet, and 2gm yeast / kg diet + 50µg OA /kg diet respectively. The results showed a significant decrease in body weight, serum total proteins, albumin and globulins in a group of quails received ochratoxin A (G 3), compared with the control group (G1) and all treatments. Supplementation of yeast (G2) lied to the improvement in the studied parameters. Also Yeast alleviates the toxic effect of ochratoxin A group four (G 4).

INTRODUCTION

Increasing the productive ability and health of the domestic fowl was and still the primary goal of investigator and producers in the field of the poultry production. The risks associated with the presence of natural toxins or their metabolites are mostly unknown. Since these toxins are of natural origin, most people consider that natural products are safe (El-Barkouky, 2008). They believe that they do not carry risks. However, contamination of human or animal food with natural toxins may result in a number of troubles and even severe diseases. In fact, the natural toxins can be hepatotoxic, haematotoxic, nephrotoxic, immunotoxic, neurotoxic, mutagenic, genotoxic, teratogenic and carcinogenic. They can cause numerous disorders and diseases, which sometimes prove to be fatal in animals or humans (CAST, 2003).

Mycotoxins are toxic substances produced as secondary metabolites of fungi that commonly grow in cereals used in animal feeds and human food (Santin et al., 2002). Mycotoxins represent a diverse group of secondary fungal metabolites, which vary widely differ in their chemical nature and consequently in their mode of action (Sudakin, 2003). From the thousands of known secondary fungal metabolites only a few hundred are referred to as mycotoxins (Riley, 1998). The nature of toxic effects varies depending on different factors including type of mycotoxin, amount and duration of the exposure, animal species, age, health, sex, dietary status and metabolism of the exposed individual (Galvano et al., 2001). Toxic effects of mycotoxin ingestion get worse if other nutritional injuries such as vitamin deficiency, caloric deprivation, malnutrition, and infectious disease status occur (Hussein and

Brasel, 2001). In many cases these mycotoxins can be found in combination in food (Versantvoort et al., 2005) and feed (Garaleviciene et al., 2001) and among these, aflatoxin is the most intensively studied, however, another very important mycotoxin is ochratoxin A (OA) (Sweeney and Dobson, 1998).

Ochratoxins is a general term describing a family of toxic compound consisting of three members known by the trivial name of ochratoxin A, B and C, they are the second major group of mycotoxins to be characterized after the discovery of aflatoxins. Structurally the three toxins differ only very slightly from each other, however, these differences have marked effects on their respective toxic potentials. (Elaroussi et al., 2006). OA is a mycotoxin food and feed-contaminant known to be implicated in a diverse range of toxicological effects in a variety of animal species. It cause kidney and liver tumors in poultry and possibly in humans (Kuiper-Goodman and Scott, 1989, Petzinger and Ziegler, 2000, Biro et al., 2002, Ringot et al., 2006). The most prominent toxic effects of OA being nephrotoxicity. OA was found to be involved in a fatal human chronic kidney disease called balkan endemic nephropathy (BEN), a chronic tubulo-interstitial nephropathy, which is found in the rural population in Bulgaria, Romania and Yugoslavia (Vrabcheva et al., 2000, Pfohl-Leszkowicz et al., 2002).

A live yeast, *Saccharomyces cerevisiae* (SC), was found to alleviate the adverse effects of mycotoxicosis in poultry (Stanley et al., 1993). Sc has shown considerable binding ability with several commonly occurring mycotoxins and is also found more effective as a low-inclusion binder to bind mycooxins present in contaminated poultry feed when compared with other physical or chemical materials (Mahesh and Devegowda, 1996). Furthermore, Sc and cell wall component of Sc have the ability to alleviate the adverse effects of the several combinations of mycotoxins present naturally in feed on productivity and serum biochemical and hematological parameters in poultry (Aravind et al., 2003). Furthermore, The cell wall of yeasts known to be composed of complex polymers of β -glucans, α -mannans, mannoproteins, and a minor amount of chitin, all of which have a number of bioprotective properties. The beneficial effects of Sc were attributed to this cell wall component. This resultant used to remove/bind pathogenic bacteria in the intestine (Fernandez et al., 2002). This increased the intestinal absorption capacity for nutrients) and improved immuno-modulation (Fritts and Waldroup, 2003, Shashidhara and Devegowda, 2003)

MATERIALS AND METHODS

A total number of 120 one day old, unsexed Japanese quail chicks, obtained from the farm of Biological Application Department, Nuclear Research Center at Inshas were used in the present study. The quail chicks were randomly assigned equally to four groups (30 birds per each). Each group contained 3 replicates of 10 birds. Each group was kept in 3 cages with dimensions of 100x 80x 20 cm. The birds were battery reared and provided with water and feed ad Libitum under the same conditions of temperature and light regimen during the experimental period which lasted for 6 weeks of age. Quails basal diet contained 3200Kcal ME/Kg and 24% crude protein. Camphor (Eucalyptus Globules) contained 92.17% dry matter, 37.5% ash as DM basis, 8.1% ether extract as DM bases, 12.1% CP as DM basis and 4412.5 Kcal GE/Kg as DM basis. Four Experimental diets were used as follows in table (1).

Table 1. Experimental Design:

Group	No of quail	Age of quail/day	OA (50µg/bird/day)	Yeast (2gm/kg ration)
G1	30	1	-	-
G2	30	1	-	+
G3	30	1	+	-
G4	30	1	+	+
Total	120			

Table keys: µg/b/d = microgram / bird / day + = treated at age 20 days - = non treated

Preparation and production of Ochratoxin A (OA):

Ochratoxin A produced by *Aspergillus ochraceus* NRRL3174 culture material was prepared by the National Research Center, Cairo, Egypt. Procedures for OA production, extraction and purification were carried according to Davis et al. (1969) and Nesheim, (1969).

Body weight data was registered weekly by weighing the birds individually every week till the end of the experiment, at 7th weeks of age. At this age, 7th weeks old, two birds of each replicate group (8 birds/ group) were randomly slaughtered.

Blood samples were collected during slaughtering to process measure total proteins (TP) and albumin (Alb), using commercial kits of Bio-Merieux Co. Marcy. L. Eoile chorbouneries. France. Total globulins were calculated by subtracting serum albumin value from serum total proteins.

(1) Total serum proteins were detected according to Dumas, (1974).

(2) Serum albumin were detected according to Drupt, (1976).

- (3) Serum Alpha, Beta, and Gamma globulins were detected according to protein fractions by using cellulose acetate electrophoresis as described by Henry et al., (1974).

Statistical analysis:

According to Sendecore and Cochran (1982), using ANOVA.

RESULTS AND DISCUSSION

1. Effect of Ochratoxin A on Body weight:-

It is clear from Table (2) that adding *saccharomyces cerevisiae* (Sc) to the basal diet resulted in a significant increase in body weight at all times through the duration of experimented periods than control. The same table show that body weight was significantly decreased in group (G 3) throughout the duration of the experiment,

Supplemented OA-treated group 4 in the present study (OA+ Sc group) significantly alleviated ($P < 0.05$) the adverse effects of OA on body weight as compared with OA-fed birds alone (group 3). At the same time, inclusion of Sc alone recorded a significantly higher body weight when compared with control and with all experimental groups.

Results in Table (2) shows that body weight was decreased significantly in OA- treated group (OA group3) when compared to all experimental groups. Such decrease in body weight due to ochratoxicosis in the present study was in agreement with several previous investigations using dietary OA inclusion with rates of 50 -567 ppb (Stoev et al.,2004, El-Barkouky, (2008) , El-Barkouky and Abu-Taleb, (2008), El-Barkouky et al.,(2010) and Garcia et al.,(2003) .

The present findings also agree with Hatab, (2003) and Elaroussi et al. (2006), who attributed the decrease in body weight of OA – fed broilers to decrease in serum T_4 and the increase in serum T_3 . Elbarkouky (2008) and Elbarkouky et al. (2010) found that supplementation of broiler diets with Sc improved the performance parameters affected by OA. They indicated that addition of Sc to broiler diets providing the partial protection against the toxic effects of OA. In this respect, additions of Sc to mycotoxins-free diet provided significant improvements in all performance measurements of birds and did not produce any negative changes (Oguz and Parlat, 2004, Yildiz, et al., 2004 and Elbarkouky et al., 2010).

Table 2. Effect of dietary Ochratoxin A on body weight (in grams) in Quails

	Wk 1	Wk 2	Wk 3	Wk 4	Wk 5	Wk 6	Wk 7
G1(Control)	143±1.7 ^c	178.9± 1.1 ^b	204.9±1.7 ^b	215.0± 3.4 ^b	235.0± 3.2 ^b	248± 3.3 ^b	250.5±2.8 ^b
G2 (Yeast)	154 ±1.9 ^a	188 ±3.2 ^a	211.0 ±2.3 ^a	225.7± 3.0 ^a	247.1± 3.1 ^a	264.4±3.1 ^a	268.0±3.3 ^a
G3(OA)	138.6±0.8 ^c	158.2 ±1.0 ^f	170.7±1.2 ^d	176.9± 2.4 ^c	210.0± 3.1 ^d	224± 5.6 ^d	234.7±3.6 ^d
G4 (OA+Y)	140± 2.4 ^c	171.8 ±1.7 ^c	193.8 ±2.1 ^c	206.0± 2.4 ^c	231.0± 2.7 ^c	240± 2.4 ^c	248±2.3 ^c

a, b Means within row with different superscripts are differ significantly ($P < 0.05$).

(2) Effect of Ochratoxin A on total leucocytic counts (10^6 cu mm) "WBCs"

Results in Table (3) indicated that dietary OA supplementation at a level of ($50\mu\text{g/b/d}$) (group 3) caused a significant reduction in WBC count beginning from the first week until the end of experiment.

At the same table (3) showed that dietary supplementation of Sc (group 2) alone or with OA (group 4) partially and numerically improved WBC counts as compared with the control group.

The toxic effects of OA on WBC counts in quails obtained in this study was in agreement with the results of Patil *et al.* (2005) . The decrease in number of leucocytes was primarily reported to be a reflection of a decrease in lymphocytes, and to a lesser extent monocytes or heterophils, such a lymphocytopaenia may be a sensitive and useful indicator of ochratoxicosis that may possibly occur due to a direct effect on germinal centers of lymphoid tissues and implies alteration of the immune function (ELaroussii *et al.* 2006). Slight anemia and leucocytosis were observed by Stoev *et al.* (2000) in broiler chicks fed contaminated diets with OA at dietary levels of 130, 305 and 790 ppb. In this regard, Hatab (2003) and ELaroussii *et al.* (2006) found that both RBC and WBC counts were decreased significantly to reach about 37and 30% respectively in broiler chicks fed OA-containing ration at dietary concentration of 400 and 800ppb, and the effects of OA was proportional to OA doses. Moreover, El-Barkouky (2008) and El- Barkouky and Abu-Taleb (2008) reported that contamination of broiler diet with lowest concentration (50 and 100ppb) of OA resulted in a significant decrease in RBC and WBC counts at both examined levels. They indicated that the effects of OA were dose – dependent.

The adverse effects of OA WBC count in quails in the current study may be partially protected by supplementing quail diets with active dried live yeast (Sc). These results are in accordance with those reported by Elbarkouky (2008), who reported that supplementation of broiler diets with Sc improved the negative effects of

OA on WBC counts when incorporated at a dietary concentration of 200 mg / kg diet. Similar results were also reported in Japanese quail chicks by Khalil (2008), who observed that supplemented quail diet with 2 gm Sc / kg diets ameliorated the negative effects of 16.5 ppm OA on WBC count of quail fed this contaminated diets from 20 days until 7 weeks of age. Basmacioglu et al. (2005) reported that addition of esterified glucomannan (EG), a cell wall component of yeast, to an AF (Aflatoxin) - containing diet significantly diminished the adverse effects of AF on RBC and WBC counts in broilers. In this respect, Bejaoui et al. (2004) reported that yeast and their enzymes are capable of detoxifying OA and an adsorption mechanism has been suggested for OA removal.

Table 3. Effect of dietary Ochratoxin A on the total leucocytic counts (10^3 cu mm) in quails

	Wk 1	Wk 2	Wk 3	Wk 4	Wk 5	Wk 6
G1 (Control)	23.24±0.07 ^d	25.56±0.45 ^{cd}	29.38±0.67 ^c	29.83±0.31 ^c	32.3 ±0.75 ^b	35.56± 0.73 ^b
G2 (Yeast)	26.2±0.04 ^b	27.86± 0.16 ^b	31.4±0.72 ^a	32.3±0.55 ^b	32.3±0.75 ^b	40.06±0.55 ^a
G3(OA)	13.2±0.46 ^g	18.56±0.40 ^f	20.77±1.01 ^f	20.2±0.3 ^g	23.83±0.15 ^f	25.53 ±0.61 ^d
G4(OA+Y)	20.07±0.82 ^e	22.96±0.21 ^e	27.81±0.63 ^c	28.41±0.48 ^e	30.2±0.75 ^d	34.83±0.15 ^b

a, b Means within row with different superscripts are differ significantly (P < 0.05).

4. Biochemical parameters:

Tables (4-10) demonstrates that adding yeast to the basal diet (G2) resulted in a significant increase in total protein and globulin at all times throughout the experimental periods. While, (G3) that adding OA to the basal diet showed significant decrease in total protein and globulin. But G4 was increase slightly in quails than control when adding yeast with OA. Tollba, et.al.,(2004) and Sharma, et.al., (2008), showed significant increases in total serum proteins, albumin, Globulin values as the result of feeding yeast in all treatment groups when compared with the control group.

Also table (4 - 10) illustrates the dietary treatment effects by supplementing quail diets with Sc, OA individually or in combination for 6 weeks on serum protein levels (TP, Alb and Glob) of male broiler chicks. The results indicate that OA supplementation to quail diets at 50ug/kgm (group 3) caused significant decreases in serum TP, Alb and globulin levels until 6th weeks compared with the control and group 2. The negative effects of OA on serum blood proteins of broilers observed in the present study are in agreement with the previous investigations. Campbell et al. (1983) and Richardi and Huff (1983) explained that feeding male broiler chicks a contaminated diets with OA at the dose levels of 0.5, 1, 2, 4 and 8 μ g / g feed for

the first 3 weeks of age caused a significant decrease in TP, Alb and Glob at the dose level of 2 μ g / g feed and above. Also, Kubena *et al.* (1989) found that serum TP and Alb levels were significantly depressed in male broiler chicks fed starter diet supplemented with OA at levels of 3, 2.5, and 2 μ g / g feed, respectively for a period from 21 to 26 days. They attributed this depression in blood proteins to a decrease in protein absorption and / or utilization or to the inhibition of protein synthesis by this mycotoxin.

Similar decrease in serum proteins were reported when OA was administered to broiler chicks at 130-790 μ g / Kg or 567 μ g / Kg (Stoev *et al.*, 2000 and Garcia *et al.*, 2003, respectively) and at 0.5 - 4 mg / Kg feed (Huff *et al.*, 1988, Bailey *et al.*, 1989, Singh *et al.*, 1990, Raina *et al.*, 1991, Huff *et al.*, 1992, Gentles *et al.*, 1999, Santin *et al.*, 2002, Kumar *et al.*, 2003 and Sawale *et al.*, 2009). They suggested that the low TP concentrations might be due to a decrease in Alb and Glob levels or to degeneration of endoplasmic reticulum lead to pathological changes in the liver that in turn caused reduction in hepatic protein synthesis.

Sakhare *et al.* (2007) used the same levels of OA (200 ppb) as in the present study and reported that there was a significant decrease in serum proteins (TP, Alb and Glob) at 21 days of age and this reduction was more noticeable at 42 days of age, indicating the effects of OA was time-dependent. They concluded that the reduction in blood proteins induced by OA could be due to pathological changes in liver provoked by OA. Similar results were also reported as OA was fed as supplementation to broilers diets at 50 and 100 ppb (El-Barkouky and Abu-Taleb, 2008) and at 400 and 800 ppb (Hatab, 2003 and Elaroussi *et al.*, 2006)

A change in serum proteins are very sensitive indicators of ochratoxicosis and reflect liver function. Signs of liver disease were further supported by the significant decrease in TP, Alb and Glob of OA treated groups, as OA is known to inhibit hepatic protein synthesis. The low level of albumin and globulin observed in this study in OA treated birds may be due to decreased the synthesis in the liver that suffered hepatic toxicity or may be due to its loss in urine as a result of renal impairment (as reflected by the increased serum uric acid and creatinine).

In this respect, Elbarkouky (2008) found that supplementation of broiler diets with Sc at 200 mg / kg abolished the significant reduction in TP, Alb and Glob as a results of presence of OA at 50 or 100 ppb in broiler diets. Also, Khalil (2008) observed that presence of OA in ration of growing Japanese quails at 16.5 ppm (50 μ g / bird / day) caused significant reduction in TP, Alb and Glob, Supplemented quails diet with Sc at 2gm / kg ameliorated all the negative effects observed by OA. Moreover, Raju and Devegowda (2000) reported significant amelioration in the

multiple mycotoxins-dependent detrimental effects alone or in combination in broilers for 21 and 35 days as a result of the addition of esterified-glucomannan (EG) to broiler contaminated diets. They found that serum proteins content was depressed in broilers fed contaminated with multiple mycotoxins (OA, 2ppm, AF, 0.3 ppm and T-2, 3 ppm) singly or in combination. Inclusion of EG to broiler diets at 1g / kg increased (14.7%) the serum proteins affected by mycotoxins, indicating it has beneficial effect on mycotoxicosis in broiler chickens. The same explanation was also concluded by Swamy et al. (2002) who suggested that an adsorbent consisting of EG at 0.2 % has been to be effective in ameliorating the toxic effects of multiple mycotoxins

Furthermore, Aravind et al. (2003) suggested that dietary supplementation with esterified glucomannan (EG) at 0.05% to broilers is effective in counteracting the in vivo toxic effects of naturally contaminated diets with multiple mycotoxins (aflatoxins 168 ppb, ochratoxin 8.4 ppb, zearalenone 54 ppb and T-2 toxin 32 ppb) on performance, haematology, and serum biochemical measurements of broilers. Biernasiak et al. (2006) reported that the use of microorganisms as adsorbents is a successful strategy for the management of mycotoxins in animal feeds. They concluded that *Sc* had unique OA and aflatoxin decontamination properties. They suggested that mycotoxin decontamination by *Sc* functions as a result of adhesion to the cell wall surface. In addition to the mycotoxin binding capacity of *Sc* there is also a nutritional value with essential amino acids, vitamins and minerals all of which are vital for animal health and performance.

Table 4. Effect of dietary Ochratoxin A on serum concentration of total proteins (g/dl) in Quails

	Wk 1	Wk 2	Wk 3	Wk 4	Wk 5	Wk 6
G1(Control)	4.1±0.1 ^{ab}	4.1±0.07 ^{ab}	4.35±0.05	4.4±0.1 ^a	4.5±0.1 ^b	4.55±0.05 ^c
G2 (Yeast)	4.4± 0.04 ^a	4.4 ± 0.1 ^a	4.55±0.05 ^{ab}	4.75±0.05 ^a	4.84±0.05 ^a	4.9 ± 0.1 ^a
G3(OA)	3.65±0.52 ^c	3.35±0.05 ^c	3.63±0.46 ^{cd}	3.35±0.13 ^d	3.25±0.05 ^c	3.42±0.02 ^f
G4(OA+Y)	3.86±0.05 ^{bc}	3.75±0.04 ^{bc}	3.89±0.03 ^{bc}	4.06±0.12 ^b	4.16±0.21 ^c	4.4 ± 0.1 ^d

a, b Means within row with different superscripts are differ significantly (P < 0.05)

Table 5. Effect of dietary Ochratoxin A on the serum concentration of albumin (gm/dl) in quails

	Wk 1	Wk 2	Wk 3	Wk 4	Wk 5	Wk 6
G1(Control)	1.92±0.07 ^b	1.85±0.06 ^c	1.84±0.04 ^b	2.03±0.15 ^b	1.92±0.07 ^c	1.95±0.04 ^{abc}
G2 (Yeast)	1.77 ± 0.02 ^b	1.81±0.015 ^c	1.83±0.02 ^b	1.85±0.05 ^b	1.84±0.08 ^c	1.89± 0.06 ^{bc}
G3(OA)	2.02 ± 0.1 ^a	1.43±0.057 ^a	2.0 ± 0.37 ^a	1.74±0.05 ^a	1.61±0.152 ^a	1.68 ±0.15 ^a
G4(OA+Y)	1.94 ± 0.06 ^b	1.8 ± 0.11 ^c	1.92±0.07 ^b	1.88±0.08 ^b	1.92±0.09 ^{bc}	1.86± 0.03 ^{ab}

a, b Means within row with different superscripts are differ significantly (P < 0.05)

Table 6. Effect of dietary Ochratoxin A on the serum concentration of globulin (gm/dl) in quails

	Wk 1	Wk 2	Wk 3	Wk 4	Wk 5	Wk 6
G1 (Control)	2.18±0.08 ^{ab}	2.25±0.13 ^{bc}	2.50±0.02 ^{bc}	2.36±0.15 ^{cd}	2.57±0.18 ^{bc}	2.59±0.02 ^b
G2 (Yeast)	2.2 ± 0.06 ^a	2.58±0.12 ^{ab}	2.71±0.03 ^{ab}	2.89± 0.05 ^a	3.00±0.08 ^a	3.00±0.045 ^a
G3(OA)	1.63± 0.54 ^d	1.92± 0.43 ^e	1.63± 0.11 ^e	1.61± 0.03 ^f	1.64± 0.13 ^f	1.74 ± 0.15 ^d
G4(OA+Y)	1.92±0.11 ^{bc}	1.87± 0.07 ^d	1.97± 0.09 ^d	2.18± 0.19 ^d	2.14±0.11 ^d	2.54 ± 0.10 ^b

a, b Means within row with different superscripts are differ significantly (P < 0.05)

Table 7. Effect of dietary Ochratoxin A on the alpha-globulin (g/dl) in quails

	Wk 1	Wk 2	Wk 3	Wk 4	Wk 5	Wk 6
G1 (Control)	0.87 ± 0.035 ^a	0.86 ± 0.01 ^c	0.85 ± 0.01 ^c	0.87±0.01 ^c	0.88±0.01 ^c	0.89 ± 0.01 ^b
G2 (Yeast)	0.72 ± 0.02 ^{ab}	0.64 ± 0.02 ^f	0.66 ± 0.02 ^c	0.57± 0.02 ^f	0.61±0.02 ^c	0.53 ± 0.02 ^a
G3 (OA)	0.77 ± 0.28 ^{ab}	0.96 ± 0.01 ^a	0.92 ± 0.01 ^a	0.93±0.01 ^a	0.89±0.01 ^b	0.97 ± 0.01 ^a
G4 (OA+Y)	0.89 ± 0.03 ^a	0.90± 0.015 ^b	0.88 ± 0.02 ^b	0.89±0.01 ^{bc}	0.87±0.01 ^c	0.86 ± 0.01 ^c

a, b Means within row with different superscripts are differ significantly (P < 0.05)

Table 8. Effect of dietary Ochratoxin A on the beta-globulin (g/dl) in quails

	Wk 1	Wk 2	Wk 3	Wk 4	Wk 5	Wk 6
G1(Control)	0.72± 0.05 ^{cd}	0.69± 0.02 ^c	0.72± 0.02 ^c	0.77± 0.02 ^d	0.78±0.05 ^b	0.75± 0.02 ^{bc}
G2 (Yeast)	1.4± 0.25 ^a	1.06± 0.12 ^a	0.94±0.04 ^b	1.2± 0.03 ^b	1.17±0.04 ^a	1.02±0.04 ^b
G3 (OA)	0.53± 0.22 ^d	0.80± 0.35 ^c	0.6± 0.03 ^d	0.56± 0.01 ^f	0.64±0.02 ^c	0.60±0.03 ^c
G4 (OA+Y)	0.67± 0.03 ^{cd}	0.74± 0.03 ^c	0.713±0.04 ^c	0.76± 0.03 ^d	0.77±0.03 ^b	0.69±0.03 ^{bc}

a, b Means within row with different superscripts are differ significantly (P < 0.05)

Table 9. Effect of dietary Ochratoxin A on the gamma-globulin (g/dl) in quails

	Wk 1	Wk 2	Wk 3	Wk 4	Wk 5	Wk 6
G1 (Control)	0.58±0.01 ^{abc}	0.73±0.10 ^{bc}	0.93±0.01 ^{bc}	0.73±0.13 ^{bc}	0.91±0.14 ^{bc}	0.96±0.01 ^b
G2 (Yeast)	0.54±0.20 ^{abc}	0.88±0.06 ^{ab}	1.11± 0.04 ^a	1.13± 0.01 ^a	1.22± 0.03 ^a	1.45± 0.03 ^a
G3 (OA)	0.33±0.30 ^{cd}	0.17±0.19 ^d	0.11± 0.01 ^e	0.12± 0.1 ^d	0.11± 0.04 ^e	0.17± 0.05 ^d
G4 (OA+Y)	0.37±0.08 ^{bcd}	0.23±0.05 ^d	0.38± 0.07 ^d	0.54± 0.16 ^c	0.49± 0.09 ^d	0.98± 0.08 ^b

a, b Means within row with different superscripts are differ significantly (P < 0.05)

Table 10. Effect of dietary Ochratoxin A on the albumin/globulin ratio in quails

	Wk 1	Wk 2	Wk 3	Wk 4	Wk 5	Wk 6
G1 (Control)	0.88±0.05 ^b	0.81±0.07 ^b	0.73±0.02 ^d	0.86±0.12 ^c	0.75±0.08 ^{cd}	0.75 ± 0.017 ^c
G2 (Yeast)	0.67±0.03 ^b	0.7±0.04 ^b	0.67±0.01 ^d	0.636±0.03 ^c	0.61±0.04 ^d	0.626 ± 0.006 ^c
G3 (OA)	1.96±0.92 ^a	2.46±0.85 ^a	2.29±0.23 ^a	2.03±0.05 ^a	1.85±0.31 ^a	1.68 ± 0.34 ^a
G4 (OA+Y)	1.01±0.09 ^b	1.0±0.10 ^b	0.97±0.08 ^b	0.86±0.11 ^c	0.89±0.08 ^c	0.72 ± 0.028 ^c

a, b Means within row with different superscripts are differ significantly (P < 0.05)

REFERENCES

1. Aravind, K. L., Patil, V. S., Devegowda, G., Umakantha, B. and Ganpule, S. P. 2003. Efficacy of esterified glucomannan to counteract mycotoxicosis in naturally contaminated feed on performance and serum biochemical and hematological parameters in broilers. *Poult. Sci.*, 82 (4): 571-576.
2. Bailey, C. A., Gibson, R. M., Kubena, L. F., Huff, W. E. and Harvey, R. B. 1989. Ochratoxin A and dietary protein: 2- Effects on hematology and various clinical chemistry measurements. *Poult. Sci.*, 68: 1664-1671
3. Basmacioglu, H., Oguz, H., Ergul, M., Col, R. and Birdane Y.O. 2005. Effect of dietary esterified glucomannan on performance, serum biochemistry and haematology in broilers exposed to aflatoxin. *Czech. J. Anim. Sci.*, 50 (1): 31-39.
4. Bejaoui, H., Mathieu, F., Taillandier, P. and Lebrihi, A. 2004. Ochratoxin A removal in synthetic and natural grape juices by selected oenological *Saccharomyces* strains. *J. Appl. Microbiol.*, 97(5): 1038-1044.
5. Biernasiak, J., Piotrowska, M. and Libudzisz, Z. 2006. Detoxification of mycotoxins by probiotic preparation for broiler chickens. *Mycotoxin Research*, 22 (4): 230-235.
6. Biro, K., Solti, L., Barna-Vetro, I., Bago, G., Glavits, R., Szabo, E. and Fink-Gremmels, J. 2002. Tissue distribution of ochratoxin A as determined by HPLC and ELISA and histopathological effects in chickens. *Avian. Pathol.*, 31:141-148.

7. Campbell, M. L., May, J. D., Huff, W. E. and Doerr, J. A. 1983. Evaluation of immunity of young broiler chickens during simultaneous aflatoxicosis and ochratoxicosis. *Poult. Sci.*, 62: 2138-2144.
8. CAST, Council for Agricultural Science and Technology. 2003. "Mycotoxins: Risks in Plant, Animal and Human Systems". Task Force Report, No. 139. Ames, Iowa, USA, 99 p.
9. Davis, N.D., Searcy, J.W. and Diener, U.L. 1969. production of ochratoxin A by *Aspergillus ochraceus* in a semisynthetic medium, *APPL. Microbiol.*, 17: 742-744.
10. Dumas, B.I. 1974. A biuret colorimetric method for determination of total proteins *Clin. Chem.*, (21) 1159-1166.
11. Drupt, F. 1974. Colorimetric method for determination of albumin. *Phar. Bio. Vol.(9). Tone VIII. 777.*
12. Elaroussi, M. A., Mohamed, F. R., El Barkouky, E. M., Atta, A. M., Abdou A. M. and Hatab, M. H. 2006. Experimental ochratoxicosis in broiler chickens. *Avian Pathol*, 35 (4): 263 – 269.
13. El-Barkouky, E. M. 2008. The role of yeast in improving the performance of male broiler chicken feed ration contaminated with ochratoxin. *Egypt. J. Appl. Sci.*, 23 (2A):13-24.
14. El-Barkouky, E. M. and Abu-Taleb, A. M. 2008. The role of vitamin c in improving the performance of male broiler chickens feed ration contaminated with ochratoxin. *Egypt. J. .Appl. Sci.*, 23 (2A):1-12.
15. El-Barkouky, E. M, Mohamed, F. R., Atta, A. M., Abu-Taleb, A. M., ElMenawey, M. A. and Hatab, M. H. 2010. Effect of *saccharomyces cerevisiae* and vitamin c supplementation on broiler performance subjected to ochratoxin A contamination. *Egypt. Poult. Sci.*, 30 (1): 89-113.
16. Fernandez, F., Hinton, M. and Gils, B. V. 2002. Dietary mannan-oligosaccharides and their effect on chicken caecal microflora in relation to *Salmonella enteritidis* colonization. *Avian. Pathol.*, 31: 49-58.
17. Fritts, C. A. and Waldroup, P. W. 2003. Evaluation of Bio-Mos® mannan oligosaccharide as a replacement for growth promoting antibiotics in diets for turkeys. *Int. J. Poult. Sci.*, 2: 19-22.
18. Garcia, A. R., Avila, E., Rosiles, R. and Petrone, V. M. 2003. Evaluation of two mycotoxin binders to reduce toxicity of broiler diets containing ochratoxin A and T-2 toxin contaminated grain. *Avian. Dis.*, 47 (3): 691-699.
19. Galvano, F., Piva, A., Ritieni, A. and Galvano, G. 2001. Dietary strategies to counteract the effects of mycotoxins: a review. *J. Food. Prot.*, 64:120–131.

20. Garaleviciene, D., Pettersson, H., Augonyte, G., Elwinger, K. and Lindberg, J.E. 2001. Effects of mould and toxin contaminated barley on laying hens performance and health. *Arch Tierernahr*, 55 (1):25-42.
21. Gentles, A., Smith, E. E., Kubena, L. F., Duffus, E., Johnson, P., Thompson, J., Harvey, R. B. and Edrington, T. S. 1999. Toxicological evaluations of cyclopiazonic acid and ochratoxin A in broilers. *Poult. Sci.*, 78 (10):1380-1384.
22. Hanif, N. Q., Muhammad, G., Siddique, M., Khanum, A., Ahmed, T., Gadahai, J. A. and Kaukab, G. 2008. Clinico-pathomorphological, serum biochemical and histological studies in broilers fed ochratoxin A and a toxin deactivator (Mycifix Plus) *Br. Poult. Sci.*, 49 (5): 632-642.
23. Hatab, M. H. 2003. Determination of the toxic effects of ochratoxin A on broiler performance and immune system response by the use of nuclear techniques. M. Sc. Thesis, Fac. Agric., Cairo Univ., Egypt, 336 p.
24. Henry, L.J. , Cannon D.C. and Winkelman, J.W. 1974. Clinical chemistry principles and techniques.434-440, Harper and Row, Hagerstown.
25. Huff, W. E., Kubena, L. F. and Harvey, R. B. 1988. Progression of ochratoxicosis in broiler chickens. *Poult. Sci.*, 67: 1139-1146.
26. Huff, W. E., Kubena, L. F., Harvey, R. B. and Phillips, T. D. (1992). Efficacy of hydrated sodium calcium aluminosilicate to reduce the individual and combined toxicity of aflatoxin and ochratoxin A. *Poult. Sci.*, 71: 64-69.
27. Hussein,H.S and Brasel,J.M. 2001..Toxicity,metabolism,and impact of
28. mycotoxins on humans and animals .*Toxicology*,167(2)101-134.
29. Khalil, M. R. 2008. Effects of Ochratoxin on Immune Response in Quails and Traits for Control. Phd. Thesis, Fac.Vet. Med., Benha Univ,
30. Koynarski, V., Stoev, S., Grozeva, N., Mirtcheva, T., Daskalo, H., Mitev, J. and Mantle, P. 2007. Experimental coccidiosis provoked by *Eimeria acervulina* in chicks simultaneously fed on ochratoxin A contaminated diet. *Res. Vet. Sci.*, 82 (2): 225-231.
31. Kubena, L. F., Harvey, R. B., Huff, W. E., Corrier, D. E., Phillips, T. D. and Rottinghaus, G. E. 1989. Influence of ochratoxin A and T-2 toxin singly and in combination on broiler chickens. *Poult. Sci.*, 68: 867-872.
32. Kuiper-Goodman, T. and Scott, P. M. 1989. Risk assessment of the mycotoxin ochratoxin A. *Biomid. Environ. Sci.*, 2:179-248.
33. Kumar, A., Jindal, N., Shukla, C. L., Pal, Y., Ledoux, D. R. and Rottinghaus, G. E. 2003. Effect of ochratoxin A on *Escherichia coli*-challenged broiler chicks. *Avian Dis.*, 47 (2): 415-424.

34. Mahesh, B. K. and Devegowda, G. 1996. Ability of aflatoxin binders to bind aflatoxin in contaminated poultry feeds – an in vitro study. In: Proceeding of the 20th World's Poultry Congress, New Delhi, India. 4: 296-303.
35. Nesheim, S. 1969. Isolation and purification of ochratoxin A and B and preparation of their methyl and ethyle esters. *J.O.A.C.*, 52 : 975-979.
36. Oguz, H. and Parlat, S. S. 2004. Effects of dietary mannanoligosaccharide on performance of Japanese quail affected by aflatoxicosis *South African Journal of Animal Science*, 34 (3):144-148.
37. Patil, R. D., Scholar, Ph.D., Degloorkar, N. M., Moregaonkar, S. D. and Kulkarni, G. B. 2005. Ameliorative efficacy of Bantox® in induced ochratoxicosis in broilers: A haemato-biochemical study. *Indian. J. Vet. Pathol.*, 29 (2): 23-30.
38. Petzinger, E. and Ziegler, K. 2000. Ochratoxin A from a toxicological perspective. *J. Vet. Pharmacol.*, 23 (2): 91-98.
39. Pfohl-Leszkowicz, A., Petkova-Bocharova, T., Chemozemsky, I. N and Castegnaro, M. 2002. Balkan endemic nephropathy and associated urinary tract tumours: a review on aetiological causes and the potential role of mycotoxins. *Food. Addit. Contam.*, 19(3):282-302.
40. Prior, M. G., O'Neil, J. B. and Sisodia, C. S. 1980. Effects of ochratoxin A on growth response and residues in broilers. *Poult. Sci.*, 59: 1254-1257.
41. Raina, J. S., Roy, K. S. and Singh, B. 1991. Biochemical and histochemical studies in experimental mycotoxicosis in chicks. *Indian. J. Anim. Sci.*, 62: 1276-1281.
42. Raju, M. V. L. N. and Devegowda, G. 2000. Influence of esterified- glucomannan on performance and organ morphology, serum biochemistry and haematology in broilers exposed to individual and combined mycotoxicosis (aflatoxin, ochratoxin and T-2 toxin). *Br. Poult. Sci.*, 41: 640-650.
43. Richardi, J. C and Huff, W. E. 1983. Effect of acute ochratoxicosis on blood pressure and heart rate of broiler chickens. *Poult. Sci.*, 62: 2164-2168.
44. Riley R.T. 1998. Mechanistic interactions of mycotoxins: theoretical considerations, in: Sinha K.K., Bhatnagar D. (Eds.), *Mycotoxins in Agriculture and Food Safety*, Marcel Dekker Inc., pp. 227-253.
45. Ringot, D., Chango, A., Schneider, Y. J. and Larondelle, Y. 2006. Toxicokinetics and toxicodynamics of ochratoxin A, an update. *Chemico-Biological Interactions*, 159: 18-46.
46. Sakhare, P. S., Harme, S. D., Kalorey, D. R., Warke, S. R., Bhandarkar, A. G. and Kurkure, N. V. 2007. Effect of Toxiroak® polyherbal feed supplement during induced aflatoxicosis, ochratoxicosis and combined mycotoxicoses in broilers. *Veterinarski Arhiv*, 77 (2): 129-146.

47. Santin, E., Maiorka, A., Macari, M., Grecco, M., Sanchez, J. C., Okada, T.M. and Myasaka, A. M. 2001b. Performance and intestinal mucosa development of broiler chickens fed diets containing *Saccharomyces cerevisiae* cell wall. *J. Appl. Poult. Res.*, 10: 236-244.
48. Santin, E., Maiorka, A., Krabbe, E. L., Paulillo, A. C. and Alessi, A. C. 2002. Effect of hydrated sodium calcium aluminosilicates on the prevention of the toxic effects of ochratoxin. *J. Appl. Poult. Res.*, 11: 22-28.
49. Sawale, G. K., Gosh, R. C., Ravikanth, K., Maini, S. and Rekhe, D. S. 2009. Experimental mycotoxicosis in layer induced by ochratoxin A and its amelioration with herbomineral toxin binder 'toxiroak'. *Int. J. Poult. Sci.*, 8 (8): 798-803.
50. Sendecor, G.W. and Cochran, W.G. 1982. *Statistical methods* 7th ED., Iowa state. Ames, Loea, USA.
51. Shashidhara, R. G. and Devegowda, G. 2003. Effect of dietary mannan oligosaccharide on breeder production traits and immunity. *Poult. Sci.*, 82:1319-1325.
52. Singh, G. S. P., Chauhan, H. V. S., Jha, G. J. and Singh, K. K. 1990. Immunosuppression due to chronic ochratoxicosis in broiler chicks. *J. Comp. Pathol.*, 103: 399-410.
53. Stanley, V. G., Ojo, R., Woldesenbet, S., Hutchinson, D. H. and Kubena, L. F. 1993. The use of *Saccharomyces cerevisiae* to suppress the effects of aflatoxicosis in broiler chicks. *Poult. Sci.*, 72(10): 1867-1872.
54. Stoev, S. D., Anguelov, G., Ivanov, I. and Pavlov, D. 2000. Influence of ochratoxin A and an extract of artichoke on the vaccinal immunity and health in broiler chicks. *Exp. Toxicol. Pathol.*, 52 (1) :43-55.
55. Stoev, S. D., Djuvinov, D., Mirtcheva, T., Pavlov, D. and Mantle P. 2002. Studies on some feed additives giving partial protection against ochratoxin A toxicity in chicks. *Toxicol. Lett.*, 5, 135 (1-2): 33-50.
56. Stoev, S. D., Steanov, M., Denev, S., Radic, B., Domijan, A. M. and Peraica, M. (2004). Experimental mycotoxicosis in chickens induced by ochratoxin A and penicillic acid and intervention with natural plant extracts. *Vet. Res. Com.*, 28: 727-746.
57. Sudakin, D. L. 2003. Trichothecenes in the environment: relevance to human health. *Toxicol. Lett.* 143: 97-107.
58. Swamy, H. V. L. N., Smith T. K., MacDonald, E. J., Boermans, H. J. and Squires, E. J. 2002. Effects of feeding a blend of grains naturally contaminated with *Fusarium* mycotoxins on swine performance, brain regional neurochemistry, and serum

- chemistry and the efficacy of a polymeric glucomannan mycotoxin adsorbent. *J. Anim. Sci.*, 80: 3257-3267.
59. Sweeney, M. J. and Dobson, A. D. W. 1998. Mycotoxin production by *Aspergillus*, *Fusarium* and *Penicillium* species. *Int. J. Food. Microbiol.* 43: 141-158
60. Verma, J., Johri, T. S., Swain, B. K. and Ameena, S. 2004. Effect of graded levels of aflatoxin, ochratoxin and their combinations on the performance and immune response of broilers. *Br. Poult. Sci.*, 45 (4) : 512 – 518.
61. Versantvoort, C. H., Oomen, A. G., Van de kamp, E., Rompelberg, C. J. and Sips A. J. 2005. Applicability of an in vitro digestion model in assessing the bioaccessibility of mycotoxins from food. *Food. Chem. Toxicol.*, 43(1): 31- 40.
62. Vrabcheva, T., Usleber, E., Dietrich, R. and Martlbauer, E. 2000. Co-occurrence of ochratoxin A and citrinin in cereals from Bulgarian villages with a history of Balkan endemic nephropathy. *J Agric. Food. Chem.* 48 (6):2483-2488.
63. Watts, C. M., Chen, Y. C., Ledoux, D. R., Broomhead, J. N. Bermudez, A. J. and Rottingus, G. E. 2003. Effects of multiple mycotoxins and a hydrated sodium calcium aluminosilicate in poultry. *Int. J. Poult. Sci.*, 2 (6): 372-378.
64. Yildiz, A. O., Parlat, S. S. and Yildirim, I. 2004. Effect of dietary addition of live yeast (*Saccharomyces cerevisiae*) on some performance parameters of adult Japanese quail (*Coturnix coturnix japonica*) induced by aflatoxicosis. *Revue. Med. Vet.*, 155 (1): 38-41.

مدى تأثير اضافة خميرة (ساكارومييسيس سيرفيسى) غير النشطة على السمان المغذى على علائق ملوثة بسموم الاوكراتوكسين

مصطفى ربيع عبد النبي خليل ، حامد عبد المجيد الامام شلى ، احمد محمد عبد الجليل الشافعى

معهد بحوث صحة الحيوان - معمل فرعى المنصورة

- ١- استهدف إجراء هذا البحث دراسة تأثير إضافة الخميرة لتنشيط سموم الاوكراتوكسين لعلائق السمان اليابانى على النمو والمناعة وبعض مكونات الدم
- ٢ - تم استخدام ١٢٠ طائر من السمان اليابانى عمر يوم وقسمت عشوائياً إلى اربع مجاميع في كل مجموعة ٣٠ طائر .
- المجموعة الأولى : مجموعة ضابطة تم تغذيتها على العليقة الاساسيه بدون اضافات.
- المجموعة الثانية : تم تغذيتها على العليقة الاساسيه مع اضافة ٢ جرام خميرة لكل كيلوجرام.
- المجموعة الثالثة : تم تغذيتها على العليقة الاساسيه مع اضافة سموم الاوكراتوكسين ٥٠ ميكروجرام لكل كيلوجرام.
- المجموعة الرابعة : تم تغذيتها على العليقة الاساسيه مع اضافة سموم الاوكراتوكسين ٥٠ ميكروجرام + خميره ٢ جرام لكل كيلوجرام.
- ٣- وتم تسجيل وزن الجسم اسبوعياً حتى نهاية التجربة.
- ٤- في الأسبوع السادس تم ذبح ستة طيور سمان من كل مجموعة وتم جمع الدم لعد كرات الدم البيضاء وتقدير بروتين الدم والاليومين و الجلوبيولين.
- ٥- دلت النتائج المتحصّل عليها أنّ إضافة الخميرة في هذه الدراسة كان مؤثراً وأعطى زيادة معنوية في وزن الجسم وكذلك عد كرات الدم البيضاء وتقدير بروتين الدم و الجلوبيولين. أما المجموعة الثالثة المغذاة على سموم الاوكراتوكسين فقط كانت النتائج عكس المجموعة الثانية (مجموعة الخميرة). اما المجموعةالرابعة المغذاة على الخميرة والاوكراتوكسين فقد لوحظ اضافة الخميرة لت الى تحسين الضرر الناتج من سموم الاوكراتوكسين.
- أثبتت الدراسة أيضاً أن إضافة الخميرة تؤدي إلى وقف وتنشيط التأثيرات السامة لهذا السم الفطرى على الأداء الانتاجى و المناعى وعلى مقاييس الدم والمقاييس الكيميائية .