EFFECTIVENESS OF MYCODOTE* FOR DETOXIFICATION OF AFLATOXIN CONTAMINATED LOCAL CHICKEN DIETS

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Abstract

Two local strians of chickens Gimmizah (G) and Dokki-4 (D_4), at age of 48 weeks were used in this study. Ninety- six individuals of each strain (90 hens + 6 cocks) were distributed randomly into 6 groups for each strain to present three levels of aflatoxin B_1 (AFB_1) 0.0, 2 and 5 ppm. Within each of AFB_1 groups, two levels of mycodote as antitoxin 0.0 and 0.01% were added. Birds of both strains were exposed to toxin and toxin plus mycodote for three weeks (treatment period). Thereafter, they were fed for 4 weeks on free aflatoxin and mycodote diets (recovery period).

Results of overall means were determined and summarized as follows:

1. There was slight increase in the concentration of ALT(Alanine aminotransferase), alkaline phosphates (ALP) and creatinine enzymes for those fed contaminated diets. Plasma total protein, albumin, and globulin concentration showed slight decrease during treatment period.
2-The treated groups fed on AFB_1 without mycodote had significantly higher aflatoxin residue in egg yolk than in albumen. Slight effect lasted till the end of the recovery period for birds.
3-The liver contained the highest levels of aflatoxin residues compared to the spleen, heart, breast, gizzard and thigh tissues.
4-The two local breeds showed different and promoting effect in effecting aflatoxicosis.

However, administration of mycodote ameliorated the negative effect of aflatoxin B1 in the diet. Also, the recovery period for four weeks was not sufficient to bring the birds to normal production activity.

* Mycodote ®: adsorbent agent hydrate sodium, calcium alumionsilicate (HSCAS).

INTRODUTCTION

Aflatoxins are the secondary metabolites which are produced mostly by certain species of *Aspergillus flavus* and *Aspergillus parasiticus* (Peers and Linsell, 1997). These compounds contaminate various food and feed products and they threat animal and human health.Afltoxin has elicited greatest public health concern of all mycotoxins because of its widespread occurrence in several feed grains, especially corn which comprises between 50 to 60% of many poultry diets (Phillips *et al.* 1988). Additionally, liver damage characterized by enlargement and of enzymes asparate aminotransferase (AST) and alkaline phosphates (ALP) in the blood has peen reported in pigs fed 500 ppb aflatoxin (Harvey *et al.* 1990). Harvey *et al.* (1989) reported on protection from changes in serum biochemical measurements that were caused by consumption of diet containing 3 mg of AF_1 per kg diet in growing Leghorn chicks. The highest amounts of AF residues were detected in the liver followed by breast, muscles finally for egg content, in an experiment, where, laying hens received diets containing maize naturally contaminated with 150 and 750 ug / kg aflatoxin B_1 for 3 and 6 weeks, respectivèly (Rizk *et al.,* 1993).

Residues of aflatoxin B_1 had been found in eggs and tissues from hens and broilers fed on aflatoxin contaminated ration by methods capable of detecting aflatoxin (M_1) and (B_1) (Truchsess *et al.*, 1977).

The study was conducted to investigate the effect of AFB_1 with or without Micodote on blood parameters, aflatoxin residues in tissues and egg.

MATERIALS AND METHODS

A total of 192 chicken birds of Gimmizah (G) and Dokki-$_4$ (D_4), local strains, at 48 weeks of age were used. Ninety-six birds of each strain (90+6 cocks) were wing-banded, weighed and randomly distributed into three experimental groups with 2 replicates (15 hens+1 cock). The birds were housed in floor pens. All experimental groups were fed on basal diet for 4 weeks before experimental treated period

The experimental diet was formulated with two levels aflatoxin B_1 (2 and 5mg / kg diet) only and other diets containing 0.1% Mycodote (antitoxin). Hens were fed on the experimental diets for 3 weeks as treatment period, then, they were fed on free aflatoxin diet for 4 weeks as a recovery period, to study the withdrawal time required for bringing back the flock to the normal production. Aflatoxin was produced via

fermentation of rice by *Aspergillus flavus* NRRL 2999 as described by Shotwell *et al.* (1966). Fermented rice was autoclaved, dried and ground to a fine powder which was analyzed spctrophotometrically for its aflatoxin content by method of Nabeney and Nesbitt (1965). Aflatoxin in the rice powder was extracted by chloroform, then incorporated into the basal diet and confirmed by TLC to provide the desired levels of 2 and 5 mg aflatoxin B_1 / kg diet. At the end of every week, three hens from each treatment of each strain were randomly selected, weighed and slaughtered. Parts of breast, thigh and organs were subjected to chemical analysis for determination of aflatoxin residuels in tissue and organs. Egg (yolk and albumen) were also examined to detect aflatoxin content. Serum concentration of aspartate aminotransferase (AST), alanine aminotransferase (ALP), alkaline phosphatase, creatinine total protein, albumin and glolulin were determined on a clinical chemistry analyzer. Data for all traits were statistically analyzed according to one way analysis of variance design using general linear model (GLM) procedure by computer program of SAS (1985) as the model

Xij =M + Ai + eij. where:

Xij = represents observation, M = overall means

Ai = effect of treatments (diets)

eij = experimental error.

RESULTS AND DISCUSSION

Data presented in Tables 1 and 2 indicated that, at the end of the treatment period, there was a high decrease for AST values by aflatoxin increasing from 2 to 5 ppm, while, there was significantly ($P<0.05$) higher ALT for treated groups fed on aflatoxificated diets with or without mycodote. On the other hand, hens fed aflatoxificated diets with 0.1% mycodote showed a slight improve in plasma AST of birds suffering from aflatoxin 2 ppm in their diet more than those received contaminated diet with 5 ppm aflatoxin B_1. In this respect, Coles (1986) reported that, most increases in plasma (ALT) activity are associated with hepatocellular damage and also, when cellular degeneration or destruction occurred in liver cells. The increase of ALT level during aflatoxicosis may be due to the effect of aflatoxin on the permeability of the liver cell which causes liver cell death (Hess, 1962).

Also, there was a high ($P<0.05$) increase in (ALP) for hens fed on aflatoxicated diets without micodote. The increasing of (ALP) activity could be a result of damage of liver cells and bile duct obstruction due to proliferation of its cells (Newberne and Butler, 1969), while; there was significant ($P<0.05$) increase in plasma creatinine concentration for birds fed aflatoxificated diet (5ppm AFB_1) (Hegazi, 1988).

Results in the same tables revealed that there was significant ($P<0.05$) decrease in plasma total protein and albumin concentration of aflatoxificated groups for G and D_4 hens. On the other hand, there were some improve in plasma total protein and albumin concentration for birds fed detoxificated (0.01% mycodote) diet. A decrease in plasma albumin may have resulted commonlly in association with chronic hepatic disease (Coles, 1986).

The increase and decrease of total globulins content my be due to the metabolism of aflatoxin in liver and the effect of aflatoxin on protein synthesis and RNA production resulting in decreasing albumin and B- globulins, and increased y-gloulins (Osuna and Edds, 1982). It was noticed in this study, that at the end of recovery period, all treated groups fed aflatoxin contaminated diet caused improvement for all blood parameters except ALT, ALP and creatinine, while the groups fed aflatoxin contaminited diet alone (without mycodote) contained effective amount of most blood parameters (AST, ALT, PLP and Creatinin).

Aflatoxin residue in egg components: Data in Tables 3 and 4 indicated that, the birds fed aflatoxificated diets without additives deposited the highest amount of AFB_1 in their egg yolk and albumen while, aflatoxificated diets plus mycodote combination decreased residues of AFB_1 in egg yolk and albumen for G and D4 strain. These results are in agreement with those obtained by Iqpal *et al.* (1983).

Aflatoxin residue in hens tissues: The liver was observed to have the highest level of aflatoxin which, inturn, may cause hepatitis or necrosis or cirrhosis or fatty degeneration, and lead to the hens death, while, the withdrawal of aflatoxin from diet decreased the amount of toxin in tissues (Tables 5 and 6). These results are in a harmony with those obtained by Hegazy and Edris (1991).

Table 1. Effect of antitoxin (Mycodote) as detoxification agent for aflatoxin B_1 on blood picture of Gimmizah hens during treatment (0-3 weeks) and recovery (4-7 weeks) periods.

Items	Treatment period (Start to 3 weeks)						Recovery period (4-7 weeks)					
	AF level (ppm)						AF level (ppm)					
	0		2		5		0		2		5	
	Mycodote %						Mycodote %					
	0.0	0.1	0.0	0.1	0.0	0.1	0.0	0.1	0.0	0.1	0.0	0.1
AST*	283.7^{b}	291.5^{a}	235.2^{d}	239.7^{c}	222.5^{e}	231.6^{d}	278.1^{a}	294.7^{a}	249.2^{a}	256.1^{b}	232.0^{c}	247.1^{bc}
ALT*	98.4^{c}	87.5^{c}	118.3^{b}	116.2^{b}	132.7^{a}	126.3^{ab}	102.3^{c}	92.6^{c}	113.7^{cb}	108.8^{b}	130.2^{a}	115.6^{b}
Alkaline-Ph**	33.2^{a}	33.9^{a}	35.4^{b}	37.6^{a}	41.7^{a}	38.1^{a}	25.9^{a}	27.8^{a}	31.5^{a}	30.8^{a}	37.7^{a}	33.8^{a}
Criatinine**	1.47^{a}	1.46^{a}	1.52^{a}	1.50^{a}	1.57^{a}	1.54^{a}	1.50^{a}	1.63^{a}	1.71^{a}	$1..79^{a}$	1.91^{a}	1.82^{a}
Total protein g/100 ml	5.03^{b}	5.10^{a}	4.02^{b}	4.07^{b}	3.4^{b}	3.8^{b}	4.86^{a}	5.17^{a}	4.19^{b}	4.26^{b}	4.04^{b}	4015^{b}
Albumin g/100 ml	2.50^{a}	2.57^{a}	1.99^{b}	2.11^{b}	1.89^{b}	1.95^{b}	2.40^{a}	2.59^{a}	2.12^{b}	2.15^{b}	2.00^{b}	2.10^{b}
Globulin g/100 ml	2.32^{a}	2.36^{ab}	2.12^{b}	2.17^{b}	2.01^{b}	2.03^{b}	2.26^{a}	2.58^{a}	2.07^{a}	2.11^{a}	2.03^{a}	2.05^{a}

a, b, ... = Mean on the same row are differently superscripted are significantly different ($P< 0.05$).

*=unit / ml.

**=unit/dl.

Table 2. Effect of antitoxin (Mycodote) as detoxification agent for aflatoxin B_1 on blood picture of Dokki 4 hens during treatment (0-3 weeks) and recovery (4-7 weeks) periods.

Items	Treatment period (Start to 3 weeks)						Recovery period (4-7 weeks)					
	AF level (ppm)						AF level (ppm)					
	0		2		5		0		2		5	
	Mycodote %						Mycodote %					
	0.0	0.1	0.0	0.1	0.0	0.1	0.0	0.1	0.0	0.1	0.0	0.1
AST*	278.3	290.2^{a}	232.5^{c}	239.3^{c}	220.8^{d}	227.2^{c}	275.4^{a}	286.8^{a}	246.2^{c}	261.3^{b}	23.8^{d}	244.1^{c}
ALT*	91.6^{b}	89.7^{a}	12.2^{a}	117.3^{a}	134.4^{a}	128.6^{a}	97.5^{bc}	92.6^{c}	113.5^{ab}	106.2^{bc}	128.9^{a}	114.7^{ab}
Alkaline-Ph**	32.9^{a}	35.4^{a}	36.7^{a}	37.2^{a}	4.08^{a}	39.9^{a}	32.8^{b}	34.7^{ab}	36.4^{ab}	32.5^{ab}	42.3^{a}	40.7^{a}
Criatinine**	1.38^{b}	1.47^{a}	1.51^{a}	1.52^{a}	1.61^{a}	1.58^{a}	1.37^{b}	1.54^{ab}	1.57^{a}	4.19^{b}	4.28^{b}	4.04^{b}
Total protein g/100 ml	4.99^{a}	5061^{a}	4.11^{b}	4.15^{b}	3.80^{b}	3.91^{b}	4.87^{a}	5.10^{a}	4.12^{b}	4.14^{b}	3.97^{b}	4.06^{b}
Albumin g/100 ml	2.47^{a}	2.66^{a}	2.02^{a}	2.14^{b}	1.86^{b}	1.93^{b}	2.54	2.59^{a}	2.08^{b}	2.09^{b}	1.96^{b}	2.04^{b}
Globulin g/100 ml	2.52^{a}	2.52^{a}	2.09^{a}	2.14^{a}	1.97^{a}	1.98^{a}	2.42^{a}	2.52^{a}	2.04^{a}	2.03^{a}	2.01^{a}	2.02^{a}

a, b, ... = Mean on the same row are differently superscripted are significantly different ($P< 0.05$).
*= unit/ml.
**= unit/ dl.

Table 3. Effect of antitoxin (Micodote) as detoxification agent for aflatoxin B_1 on aflatoxin B_1 residue (ug/g)in egg yolk and albumen of Gimmizah hens during treatment (0-3 weeks) and recovery (4-7 weeks) periods.

Treatment		Egg yolk				Albumen			
		Treatment period		Recovery period		Treatment period		Recovery period	
AF Ppm	Mycodote%	1st wk	3rd wk	4th wk	7th wk	1st wk	3rd wk	4th wk	7th wk
0	0.0	-	-	-	-	-	-	-	-
0	0.1	-	-	-	-	-	-	-	-
2	0.0	11.16^{b}	14.18^{c}	3.75^{b}	2.16^{c}	11.167^{b}	7.33^{bc}	1.620^{b}	0.90^{b}
2	0.1	8.92^{c}	9.78^{d}	2.160^{b}	-	8.92^{b}	4.17^{c}	1.255^{b}	-
5	0.0	17.15^{b}	28.04^{a}	10.883^{a}	6.213^{c}	13.153^{b}	16.73^{a}	6.236^{a}	2.20^{a}
5	0.1	14.21^{a}	21.20^{b}	5.563^{b}	3.75^{c}	11.813^{c}	12.09^{b}	2.203^{b}	1.62^{b}

a, b, ... = Mean on the same row are differently superscripted are significantly different ($P< 0.05$).

Table 4. Effect of antitoxin (Mycodote) as detoxification agent for aflatoxin B_1 on aflatoxin B_1 residue (ug/g) in egg yolk and albumen of Dokki 4 hens during treatment (0-3 weeks) and recovery (4-7 weeks) periods.

Treatment		Egg yolk				Albumen			
		Treatment period		Recovery period		Treatment period		Recovery period	
AF Ppm	Mycodote %	1st wk	3rd wk	4th wk	7th wk	1st wk	3rd wk	4th wk	7th wk
0	0.0	-	-	-	-	-	-	-	-
0	0.1	-	-	-	-	-	-	-	-
2	0.0	10.389^{d}	13.89^{c}	3.55^{bc}	0.62	9.071	7.35^{bc}	1.453^{b}	0.21^{b}
2	0.1	9.138^{c}	10.10^{bc}	1.817^{c}	-	4.417	5.19^{c}	0.963^{b}	-
5	0.0	20.003^{b}	26.47^{a}	7.390^{a}	3.52	15.570	15.61^{a}	4.613^{a}	1.63^{a}
5	0.1	15.920^{c}	19.64^{b}	6.100^{ab}	1.81^{b}	11.362	11.19^{b}	2.44^{b}	0.96^{b}

a, b, ... = Mean on the same row are differently superscripted are significantly different ($P< 0.05$).

Table 5. Aflatoxin B_1 residue (ug/g) in organs and glands of Gimmizah hens during treatment (0-3 weeks) and recovery (4-7 weeks) periods.

Items	Treatment period (Start to 3 weeks)						Recovery period (4-7 weeks)					
	AF level (ppm)						AF level (ppm)					
	0		2		5		0		2		5	
	Mycodote %						Mycodote %					
	0.0	0.1	0.0	0.1	0.0	0.1	0.0	0.1	0.0	0.1	0.0	0.1
Liver	0.913^{b}	0.356^{d}	4.990^{bc}	4.237^{c}	6.245^{a}	5.192^{b}	0.513^{d}	0.446^{d}	0.776^{bc}	0.673^{cd}	1.073^{a}	0.881^{b}
Gizzard	0.196^{c}	0.103^{c}	2.23^{b}	1.87^{b}	3.56^{a}	2.59^{b}	0153^{bc}	0.102^{c}	0.516^{ab}	0.467^{ab}	0.813^{a}	0.546^{ab}
Breast	0.091^{b}	0.037^{b}	1.59^{a}	1.89^{a}	2.16^{a}	1.84^{a}	-	-	0.073^{b}	0.030^{b}	0.513^{a}	0.114^{b}
Thigh	-	-	2.0^{ab}	1.55^{b}	2.51^{a}	2.07^{ab}	-	-	0.158^{a}	-	0.660^{a}	0.170^{a}
Heart	-	-	1.77^{b}	1.54^{b}	2.27^{a}	1.99^{ab}	-	-	-	-	0.412	0.337
Spleen	-	-	1.72^{bc}	1.48^{c}	2.19^{a}	1.82^{b}	-	-	$0.16\text{-}2^{bc}$	0.105^{c}	0.493^{a}	0.246^{b}

a, b, ... = Mean on the same row are differently superscripted are significantly different ($P< 0.05$).

Table 6. Aflatoxin B_1 residue (ug/g) in organs and glands of Dokki 4 hens during treatment (0-3 weeks) and recovery (4-7 weeks) periods.

Items	Treatment period (Start to 3 weeks)						Recovery period (4-7 weeks)					
	AF level (ppm)						AF level (ppm)					
	0		2		5		0		2		5	
	Mycodote %						Mycodote %					
	0.0	0.1	0.0	0.1	0.0	0.1	0.0	0.1	0.0	0.1	0.0	0.1
Liver	0.872^{d}	0.673^{d}	4.68^{b}	4.03^{c}	6.43^{a}	5.217^{b}	0.532^{d}	0.522^{d}	0.833^{b}	0.770^{dc}	0.123^{a}	0.823^{b}
Gizzard	0.243^{d}	0.130^{d}	1.98^{bc}	1.56^{c}	3.82^{a}	2.41^{b}	0.283^{b}	0.130^{c}	0.416^{b}	0.383^{b}	0.813^{b}	0580^{b}
Breast	0.106^{c}	0.053^{c}	1.62^{ab}	1.42^{b}	2.01^{a}	1.67^{ab}	-	-	0.041^{b}	-	0.360^{a}	0.113^{b}
Thigh	-	-	1.56^{b}	1.99^{ab}	2.67^{a}	2.10^{ab}	-	-	-	-	0.390	0.337
Heart	-	-	1.66^{b}	1.61^{b}	2.36^{a}	1.83^{b}	-	-	-	-	0.219	-
Spleen	-	-	1.56^{b}	1.60^{b}	2.23^{a}	1.78^{b}	-	-	0.112^{b}	0.74^{b}	0.416^{a}	0.217^{b}

a, b, ... = Mean on the same row are differently superscripted are significantly different ($P< 0.05$).

REFERENCES

1. Coles E.H. 1986. Veterinary Clinical Pathology 2nd edit. W.B. Saunders Company Philadelphia, London, Toronto, p. 210-215.

2. Harvey R.B.,L.F. Kubena, T.D. Philips, W.E. Huff and D.E. Corrier. 1989. Preventation of aflatoxosis by addition of hydreated Sodium Calcium Aluminosilicate to the diet of growing berrows. Amer. J. Vet. Res., 50: 416-420.

3. Harvey R.B., L.F. Kubana, W.E. Huff, D.E. Corrier, G.E. Rottinghaus and T.D. Philips. 1990. Effect of treatment of growing swine with aflatoxin and T-2 Toxin. Amer. J. Vet. Res., 51 : 1688.

4. Hegazy S.M. 1988. Treatment of aflatoxin contaminated rations and possibility of its safety use. Thesis, Ph.D., Fac. Vet. Med., Cairo Univ.

5. Hegazy S.M. and A.B.M. Edris. 1991. Aflatoxin deposition and clearance from tissues of broiler chickens fed a contaminated diet. Zagazig Vet. J., 19(4): 958.

6. Hess B. 1962. Enyzme in Blutplasma, George thieme Verlag, Stuttgart, 71-90.

7. Iqbal O.K., P.V. Rao and S.J. Reddy. 1983. Does response relationship of experimentally Induced aflatoxicosis in commercial Layer Indian J. Anim. Sci., 53 (11): 1277-1282.

8. Nabney J. and B.F. Nesbitt. 1965. A spectrophoto-meteric method of determining the aflatoxins. Analyst, 90: 155-160.

9. Newberne P.M. and W.H. Butler. 1969. Acute and chronic effects of aflatoxin on the liver of domestic and laboratory animals: A review. Cancer Res., 29: 236-250.

10. Osuna O., and G.T. Edds. 1982. Toxicology of aflatoxin B_1, warfarin and cadmium in young pigs. Clinical chemistry and blood-coagulation. Am. J. Vet. Res., 243: 1387-1394.

11. Peers F.G. and C.A. Linsell. 1977. Dietary aflatoxins and human primary liver cancer. Annls. Nutr. Aliment., 31: 1005-1009.

12. Phillips T.D., L.F. Kubena. R.B. Harvey, D.S. Taylor and N.D Heideldaugh. 1988. Hydrated Sodium Calcium Aluminosilicate. A high affinity sorbent for aflatoxin. Poult. Sci., 67: 243-247.

13. Rizk R.E., N.A. El-Sayed, G.A. Abd Allah and S.A. El-Deeb. 1993. The residue of low dietary aflatoxin B_1 and its effect on productivity and reproductivity of local chicken strains. Egypt. Poult. Sci., 13: 301-306.

14. SAS Institute. 1985. SAS/Stat User's Guide Release 6-8. ed., SAS Institute Inc., Garry NC U.S.A.

15. Shotwell O.L., C.W. Hesseltine, R.D. Stubblefield and W.G. Sorenson. 1966. Production of aflatoxin on rice. Appl. Microbiol., 14: 425-428.

16. Trucksess M.W., L. Stoloff, W.A. Pons, J.A.F. Cucullu, L.S. Lee and A.O. Franz Jr. 1977. Thin Layer chromatograghic determination of aflatoxin B_1 in eggs. J. Assoc. Off. Anal. Chem., 60: 795-798.

مدى فاعلية الميكودوت فى تثبيط سموم الأفلاتوكسين الملوثة لعلائق الدجاج المحلى

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اســتخدم فى الدراسة عدد ١٩٢ طائراً من سلالتى الجميزة ودقى ٤ عمر ٤٨ أسبوعاً ٩٦ طائراً مــن كــل سلالة (٩٠ دجاجة + ٦ ديوك) وتم تقسيم الطيور إلي ثلاثة مجاميع . تمت التغذية فيها على عليقة تحتوى على أفلاتوكسين بمستوى صفر ، ٢، ٥ جزء فى المليون ، وداخل كل مجموعة تــم اضافة ميكودوت كمضاد لسموم الفطريات بمستوى صفر ، ٠،٠١ % وأستغرقت التجربة ٧ أسابيع مقسمة على فترتين :

الأولى : لمدة ٣ أسابيع (فترة المعاملة).

الثانــية : لمدة ٤ أسابيع تمت التغذية فيها على علائق خالية من الأفلاتوكسين والميكودوت (فترة الأستشفاء)

وأظهرت الدراسة النتائج التالية:

كيمــياء الــدم: – لو حظ انخفاض فى معظم محتويات الدم AST والبروتينات الكلية والألبومين والجلوبيوليــن وزيادة فى إنزيم ALT والفوسفاتيز القاعدى والكرياتنين للمجاميع المغذاة على علائــق ملوثة بالأفلاتوكسينات وكان التأثير أكثر فى المجاميع المغذاة على مستويات مرتفعة من الأفلاتوكسين (٥ جزء فى المليون) بالمقارنة بالمستوى المنخفض (٢ جزء فى المليون).

الأثر المتبقى من الأفلاتوكسين فى البيض: وجد أثر متبقى للأفلاتوكسين فى مكونات البيض لكل من الصفار والبياض وذلك فى المجاميع المغذاة على علائق ملوثة بمستويات مرتفعة من الأفلاتوكسين (٥ جــزء فى المليون/كجم عليقة) بدون إضافة مضاد وكان الأثر المتبقى لهذه السموم فى الصفار أعلى مــنه فى البياض– استمر هذا التأثير ولكن بدرجة أقل فى نهاية فترة الاستشفاء فى المجاميع المغذاة على مستويات مرتفعة من الأفلاتوكسين بدون إضافات.

الأثر المتبقى للسموم فى الأنسجة والأعضاء: لوحظ احتجاز جزء من الأفلاتوكسينات فى الأعضاء وكان أكثرها تأثرا هو الكبد ومن بعده الطحال والقلب والصدر والعضلات ولكن بدرجة أقل، أدى

إضافة مضاد السموم (الميكودوت)إلى انخفاض السموم المحتجزة بالأنسجة والأعضاء وكان أقل انخفاض لها فى فترة الاستشفاء.

نستخلص من هذه الدراسة أن استخدام الميكودوت بنسبة ٠,١% كمضاد للسموم الفطرية كان له تأثيره الفعال فى تقليل الأثار السلبية لمستوى ٢ و ٥ جزء فى المليون أفلاتوكسين ب١ عند تواجده فى علائق الدجاج البياض من السلالات المحلية (الجميزة ودقى ٤).

كما أن فترة سحب السم الفطرى من علائق الدجاج البياض والتي بلغت ٤ أسابيع (الاستشفاء) كانت كافية لعودة الطيور بدرجة كبيرة لأدائها الإنتاجي الطبيعى.