

## EFFECTS OF ADDING HUMATE AND HYDRATED SODIUM CALCIUM ALUMINOSILICATE TO CONTAMINATED CHICKEN DIETS ON AFLATOXICOSIS

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### SUMMARY

A total number of 360, one-day old unsexed El-Salam chicks, were divided randomly into six groups of 60 chicks each that represents 6 dietary treatments, each in 3 replicates. Chicks of treatment 1 received the basal diet without supplementation. Chicks of treatment 2 received the basal diet contaminated with 1 ppm aflatoxin B<sub>1</sub>/kg diet (AF-diet). Chicks of treatment 3, 4, 5 and 6 were fed the AF-diet supplemented with 0.1, 0.25, 0.40% humate or 0.50% hydrated sodium calcium aluminosilicate (HSCAS), respectively. The experimental diets were fed from 1 day old to 56<sup>th</sup> day of age (treated period), thereafter, chicks of each group were fed the control diet without supplementation till the end of the 84<sup>th</sup> day of age (57 - 84) as a recovery period.

#### The results showed that:

- 1- Chicks fed aflatoxin contaminated diet (AF-diet) recorded lower body weight, feed intake, lower feed conversion and higher mortality rate than the control diet at the end of the treated period.
- 2- Relative weights of liver, kidney, spleen and proventriculus significantly increased in birds fed aflatoxin contaminated diet (AF-diet).
- 3- Chicks fed diets contaminated with 1 ppm of aflatoxin B<sub>1</sub>/kg diet exhibited lower values of serum total protein, albumin, total lipids, cholesterol and uric acid and higher values of liver enzyme functions.
- 4- The digestion coefficients of nutrients were significantly decreased in aflatoxin fed group.
- 5- Addition of humate at levels of 0.10, 0.25 and 0.40% protected chick body weight by ( 60.77, 78.55 and 80.36% ) , feed intake by ( 52.86, 63.98 and 50.00% ) and feed conversion ratio by (70.30, 89.05 and 95.30% ) , respectively, against 1 ppm AFB<sub>1</sub> at 8 weeks of age . Addition of HSCAS protected chick body weight by ( 73.52% ) , feed intake by ( 53.44 % ) and feed conversion ratio (FCR) by ( 85.95 % ) against 1 ppm AFB<sub>1</sub> at 8 weeks of age . A recovery period, may overcomes some toxic symptoms in chicks.

**Keywords:** Aflatoxin, humate, aluminosilicate, prevention, chicks

### INTRODUCTION

*Aspergillus* species infect economically important crops and forages in the field and during storage, transportation and processing. These fungi produce aflatoxin,

which contaminate food and animal feeds worldwide, causing serious health problems and livestock production losses. Aflatoxin B<sub>1</sub> (AFB<sub>1</sub>) the most toxic of the aflatoxins, is often encountered in food and animal feeds at alarming concentrations around the world (Marquardt, 1996). Unfortunately, discontinuing the feeding of aflatoxin-contaminated grain is not always practical especially when alternate feedstuffs are not readily available or affordable (Ramos *et al.*, 1996b). Several of physical, chemical and biological techniques for mycotoxin decontamination of agricultural commodities have been used, but they have had limited success (Doyle *et al.*, 1982).

One of the most practical approaches is the use of nonnutritive adsorbents, which bind the mycotoxins and inhibit their absorption from the gastrointestinal tract, thus minimizing the toxic effects in livestock and the carryover of these fungal metabolites into animal products (Ramos *et al.*, 1996a). Aluminosilicates, activated charcoal, polymers, such as cholestyramine and polyvinylpyrrolidones, and yeast and yeast products have been extensively studied with promising, but with varying results (Huwig *et al.*, 2001). For an adsorbent to successfully prevent the absorption of aflatoxins from the gastrointestinal tract, it should have a high affinity for aflatoxin resulting in the formation of a strong complex with little risk of dissociation. The adsorbent should also have a high binding capacity, to prevent saturation (Ramos and Hernandez, 1996).

Humic acid form colloid solutions, can protect the digestive tract mucosa from the effects of infectious and toxic agents. Due to their chelate structure, humic acid can bind various toxic agents, thus forming insoluble and non-resorbable complexes (Alvarez-Puebla *et al.*, 2004); that may be shed through the intestines (Kuhnert *et al.*, 1982a,b). Mechanisms of adaptive and bio-regulative qualities of humic acid and their salts are associated with their stimulating effect on immunological responses, detoxicating activity within livers and sulphhydryl-disulphide balance in protein and saccharide metabolism (Santos *et al.*, 2004).

Colloidal characteristics of humic acid and their salts and their ability to form chalets, can significantly modify the toxic effects of a number of xenobiotics and undesirable substances that enter the digestive tract together with feeds and water (Livens, 1991 and Herzig *et al.*, 1994). Their strong affinity to mutagens (Cozzi *et al.*, 1993), pesticides (Negre *et al.*, 2001), mono aromatic and polycyclic aromatic compounds (Kollist-Sugur *et al.*, 2001 and Nanny and Maza, 2001), heavy metals (Madronova *et al.*, 2001), aflatoxin B<sub>1</sub> (Rensburg *et al.*, 2006) and microorganisms (Fein *et al.*, 1999) was described.

The HSCAS binds AFB<sub>1</sub> *in vitro* (Scheideler, 1993). Thus, the efficacy of sorbent materials as HSCAS probably lies in their ability to bind AF in the intestine, rendering the toxin unavailable for absorption (Southern *et al.*, 1994). Ingestion of HSCAS to broilers does not improve skin pigmentation (Brake, 1987). The sorbent additives have raised questions about their effects on minerals and vitamins status, although Chung and Baker (1990) with P, Chung *et al.* (1990) with riboflavin and Southern *et al.* (1994) and Qota (2003) with Ca and P, have reported that HSCAS does not impair the nutrient utilization.

The objective of this study was to evaluate the effects of humate and HSCAS on growth performance, serum constituents and carcass characteristics in local chicks exposed to aflatoxins.

## MATERIALS AND METHODS

The present study was carried out at Sakha Animal Production Research Station, Animal Production Research Institute, Agricultural Research Center.

A total number of 360 one-day old unsexed chicks of El-Salam strain (Nicolas X Mamourah) were wing banded, weighed and randomly divided into 6 treatment groups nearly similar in average body weight. Each treatment consist of three replicates of 20 chicks each. Feed and water were provided *ad libitum*. Chicks were grown in brooders raised wire floors. All chicks were kept under similar managerial hygienic and environmental conditions. Chicks at one-day old were offered the six experimental diets using a basal diet. Basal diets were formulated to cover nutrient requirements (Table 1) according to Egyptian Feed Composition Table (2001). The AFB<sub>1</sub> content of the basal diet showed very little amount of 5 ppb. Chicks were allotted on the following treatments:

- (1) The basal diet without any supplement served as a control diet.
- (2) Basal diet contaminated with aflatoxin B<sub>1</sub> (AFB<sub>1</sub>) with rate 1 ppm / kg diet (AF-diet).
- (3) AF-diet + 0.1% humate
- (4) AF-diet + 0.25% humate.
- (5) AF-diet + 0.40% humate.
- (6) AF-diet + 0.50% hydrated sodium calcium aluminosilicate (HSCAS).

**Table 1. Composition and calculated analysis of the basal diet fed during the experimental period**

<b>Ingredients %</b>	<b>Starter (0 – 8wks)</b>	<b>Finisher (9-12wks)</b>
Yellow corn	64.00	71.44
Soybean meal (44% CP)	32.10	20.00
Wheat bran	00.00	4.69
Di-calcium phosphate	1.80	1.62
Limestone	1.40	1.60
Sodium chloride	0.30	0.35
Vit. min. mixture (premix) <sup>1</sup>	0.30	0.30
DL- Methionine	0.10	--
<b>Total</b>	<b>100</b>	<b>100</b>
<b>Calculated analysis</b>		
Metabolizable energy ( Kcal/Kg)	2860	2900.00
Crude protein %	19.56	15.00
Crude fiber %	3.65	3.62
Crude fat %	2.69	3.20
Calcium %	1.03	1.06
Available phosphate %	0.47	0.43
Lysine %	1.03	0.79
Methionine %	0.41	0.28
Met + cystine %	0.74	0.55

<sup>1</sup> Supplied per kg of diet : Vit. A, 10.000 IU; Vit. D3, 2000IU; Vit. E, 10 mg ; Vit. K3, 1 mg ; Vit. B1, 1 mg ; Vit. B2, 5 mg; Vit. B6, 1.5 mg ; Vit. B12, 10 mcg ; Niacin, 30 mg ; Pantothenic acid, 10 mg ; Folic acid , 1 mg ; Biotin, 50 mcg ; Choline, 260 mg ; Copper, 4 mg ; Iron, 30 mg ; Manganese, 60 mg; Zinc, 50 mg; Iodine, 1.3 mg ; Selenium, 0.1 mg ; Cobalt, 0.1 mg

Aflatoxin was produced via fermentation of rice by *Aspergillus parasiticus* NRRL 2999 as described by Shotwell *et al.* (1966) and modified by West *et al.* (1973). Fermented rice was autoclaved, dried and ground to a fine powder, which was analyzed for its AF content by method of Nabney and Nesbitt (1965) as modified by Wiseman *et al.* (1967). The AFB<sub>1</sub> in the rice powder was extracted by chloroform then incorporated into basal diet and confirmed by HPLC to provide the desired level of 1 mg.

Biofarm® Dry, totally natural humate was purchased from Farmavet International Istanbul-Turkey. Each kg of humate contained 160 mg polymeric polyhydroxy acid (humic, fulvic, ulmic and humatomelanolic acids), 663.3 SiO<sub>2</sub> and other minerals (Mn, 50 mg; Zn, 60 mg; Fe, 60 mg; Cu, 5 mg; Co, 0.2 mg; I, 1 mg; Se, 0.5 mg and Al, Na, K, Mg and P in trace amounts).

Hydrated sodium calcium aluminosilicate (HSCAS) is a chemical compound that containing Silica, 64.70%; Aluminum, 15.50%; Iron, 1.75%; Calcium, 1.26%; Potassium, 1.80%; Sodium, 2.55% and Magnesium, 1.54% and moisture 10.9%, provided from Integrated World Enterprises Co. USA.

Experimental diets were fed for 8 weeks (treatment period) followed by another 4 weeks as a recovery period during which birds were offered the control diet. Parameters investigated included body weights and feed intake of the chicks (measured weekly) and mortality (daily) recorded. At the end of treatment period (8 weeks of age) and 12 weeks of age (the end of recovery period), 3 chicks from each treatment were taken randomly for carcass evaluation. Liver, kidney, spleen, heart, thymus, gizzard and proventriculus were removed and weighed. Organ weights were expressed as percentages of body weight. A total number of 36 samples from meat (as mixture of 50% breast + 50% thigh), as well as from liver tissue were collected for chemical analysis according to the official methods of AOAC (2000). Serum was collected to determine changes in the total protein (TP), albumin (Alb), cholesterol (Chol), total lipids (TL) and uric acid (UA) as well as activity of alanine aminotransferase (ALT), aspartate aminotransferase (AST) and creatine kinase (Cr). Blood constituents were determined by colorimetric methods using commercial kits supplied from Biomerieux (Poains, France). The digestion coefficients of dietary nutrients were examined using 3 birds from each treatment at the end of treatment and recovery periods. Fecal nitrogen was determined according to the method outlined by Jakobsen *et al.* (1960). The proximate analyses of feed and dried excreta were carried out according to AOAC (2000). Liver and meat samples were analyzed for aflatoxin content according to (AOAC, 2000) method.

**Protection percentage:**

For any parameter studied, to compare the anti-aflatoxic effects achieved by the different agents studied, a protection percentage was calculated as follows;

$$\text{Protection percentage} = \frac{X - Y}{Y} \times 100$$

Where, X = change of AF- group (T2) from control (T1),  
and Y = change of any other group ( T3 ,----- T6) from control .

Data were subjected to one way analysis of variance using the General Linear Model of SAS<sup>R</sup> software (SAS, 1998). Before analysis, all percentages were subjected to logarithmic or arcsine values transformation (log<sub>10</sub>x+1) to approximate

normal distribution. Significant differences among treatment means ( $p \leq 0.05$ ) were separated by Duncan's new multiple range test (Duncan, 1955).

The following model was used to study the effect of some feed additives (humate or hydrated sodium calcium aluminosilicate with aflatoxin) on parameters investigated as follows:

$$Y_{ij} = \mu + T_i + e_{ij}$$

Where,

$Y_{ij}$  = an observation

$\mu$  = overall mean

$T_i$  = effect of the factor studied ( $i = 1, 2, \dots, \text{and } 6$ )

$e_{ij}$  = residual " random error "

## RESULTS AND DISCUSSION

### **Growth Performance:**

Data presented in Table 2 show the effect of the experimental feed additives on body weight (BW), feed intake (FI) and feed conversion ratio (FCR) during treatment and recovery periods, respectively. Feeding basal diet supplemented with AF significantly ( $P < 0.05$ ) reduced BW, FI and recorded the worst FCR compared to the control group ( $P < 0.05$ ). Feeding the AF-diet decreased BW by 22.66%, FI by 6.83% and resulted in a poorer FCR by 21.92% at 8 weeks of age (end the treatment period). Results herein are consistent with those reported by Kubena *et al.* (1992); Scheideler (1993) and Edrington *et al.* (1997) who mentioned about 6.5-38% reduction in body weight of chicks fed diet containing aflatoxin than the control diet. The reduction in feed intake and poorer feed conversion of chicks may be attributed to the harmful effect of aflatoxin on the liver as well as the negative impact of aflatoxin on the activity of enzymes involved in starch, lipids and nucleic acid digestion in broiler (Osborn and Hamilton, 1981).

The addition of humate levels especially at 0.25 and 0.40% improved BW by (23.02 and 23.56%), FI by (4.69 and 3.66%) and FCR by (16.01 and 17.13%), respectively, compared to chicks fed AF-diet, and alleviate the adverse effect by (78.55 and 80.36%), (63.98 and 50.00%) and (89.05 and 95.30%), respectively, at 8 weeks of age (end of treatment period). These results indicated that 0.25 and 0.40% humate ameliorated the adverse effects of AF on performance parameters (BW, FI and FCR) of local chicks. These results agree with Rensburg *et al.* (2006) who reported that the decrease in BW gain was diminished at 42 d of age by the addition of 3.5 g of oxihumate/ kg of contaminated diet with AF.

The addition of HSCAS protected BW by 73.52%, FI by 53.44% and FCR by 85.95% against 1 ppm AFB1 at 8 weeks of age. These results agree with those obtained by Kubena *et al.* (1990a&b); Scheideler (1993); Hassan (2000) and Qota *et al.* (2005) who reported that adding 0.5% HSCAS improved chick body weight, feed intake and feed conversion ratio compared to chicks fed AF-supplemented diet.

During recovery period, the adverse effects on chicks BW, FI and FCR for all aflatoxin groups were gradually diminished. At the end of recovery period, chicks fed AF-supplemented diet without or with humate and HSCAS consumed more feed than that of the control group. This may be due to compensatory growth after being inhibited by aflatoxicosis during treatment period (Table 2). There were insignificant differences in FCR values among the experimental treatments during recovery period

(8-12 weeks of age). Postmortem investigation indicated that mortalities were due to aflatoxicosis during the 5<sup>th</sup> and 8<sup>th</sup> week of treatment period. Mortality rates averaged 2.0, 20.0, 6.6, 5.5, 5.0 and 8.0% for control, AF-diet, AF+0.1% humate, AF+0.25% humate, AF+0.40 humate and AF +0.5% HSCAS, respectively. The higher mortality rate due to aflatoxicosis was also reported by Kubena *et al.* (1990b); Edrington *et al.* (1997) and Ghazalah *et al.* (1995). In this respect, Rensburg *et al.* (2006) found that adding oxihumate decreased mortality rate compared to broiler chicks fed AF-supplemented diet. Also, Kubena *et al.* (1990a) found that mortality rates decreased from 30 to 10% by adding 0.5% HSCAS to aflatoxic diets. In this study, chicks consumed diet supplemented with 1 ppm/ kg AFB<sub>1</sub> showed a significant reduction in body weight. Also, AF negatively affected feed consumption and FCR values. These adverse effects on performance of birds were noticed from the 2<sup>nd</sup> week until the end of the treatment period which may be due to anorexia, listlessness, inhibition of protein synthesis and lipogenesis (Oguz and Kurtoglu, 2000 and Parlat *et al.*, 2001). Impaired liver functions and utilization of protein, lipid and carbohydrate may have also affected the growth performance and general health (Miazzo *et al.*, 2000 and Ortatlati and Oguz, 2001).

**Table 2. Effect of humate levels and hydrated sodium calcium aluminosilicate (HSCAS) on growth performance of birds fed diet containing 1 ppm AFB<sub>1</sub>/ kg diet at the end of treatment and recovery periods**

Treatment	Live body weight g	Change from control (%)	Feed intake (g)	Change from control (%)	Feed conversion (g feed/ g gain)	Change from control (%)	Mortality (%)
<b>At the end of treatment period (8 wk of age)</b>							
Control	750.00 <sup>a</sup>	0.00	2098.33 <sup>a</sup>	0.00	2.92 <sup>c</sup>	0.00	3.33
AF-diet	580.00 <sup>d</sup>	-22.66	1955.00 <sup>c</sup>	-6.83	3.56 <sup>a</sup>	+21.92	20.0
AF+0.1%humate	683.33 <sup>c</sup>	-8.89	2030.66 <sup>b</sup>	-3.22	3.11 <sup>b</sup>	+6.51	6.66
AF+0.25%humate	713.53 <sup>b</sup>	-4.86	2046.66 <sup>b</sup>	-2.46	2.99 <sup>c</sup>	+2.40	5.00
AF+0.40%humate	716.66 <sup>b</sup>	-4.45	2026.66 <sup>b</sup>	-3.42	2.95 <sup>c</sup>	+1.03	5.00
AF+0.50%HSCAS	705.00 <sup>b</sup>	-6.00	2031.66 <sup>b</sup>	-3.18	3.01 <sup>c</sup>	+3.08	8.33
SEM	13.137	-	10.852	-	0.054	-	-
P-value	0.0001	-	0.0001	-	0.0001	-	-
<b>At the end of recovery period (12 wk of age)</b>							
Control	1206.66 <sup>a</sup>	0.00	1582.00	0.00	3.46	0.00	1.66
AF-diet	1005.00 <sup>d</sup>	-16.71	1566.66	-1.00	3.68	+6.36	10.0
AF+0.1%humate	1135.00 <sup>c</sup>	-5.94	1597.66	+1.00	3.54	+2.31	3.33
AF+0.25%humate	1166.66 <sup>b</sup>	-1.68	1590.00	+0.50	3.52	+1.73	1.66
AF+0.40%humate	1173.33 <sup>b</sup>	-1.12	1586.00	+0.25	3.48	+0.58	-
AF+0.50%HSCAS	1158.33 <sup>bc</sup>	-2.38	1583.33	+0.08	3.50	+1.16	5.00
SEM	15.96	-	4.323	-	0.033	-	-
P-value	0.0001	-	0.497	-	0.444	-	-

<sup>a, b, c, d</sup> Means in the same column having different litter are significantly different (P<0.05)

#### **Weights of Some Internal Organs:**

The effect of feed additives on alleviation of the negative effect of aflatoxicosis on weights of some internal organs at 8 weeks (end of treatment period) is shown in Table 3. AF-diet group showed significantly higher relative weights of liver (57.40%), kidney (39.25%), spleen (57.40%) and proventriculus (21.62%) compared to the control group. While, relative weights of bursa of fabricus and thymus

significantly decreased by AF (26.80 and 26.66%) compared to the control group, respectively. Relative weights of heart and gizzard were not affected by AF. The increase in liver weight may be due to increasing fat accumulation in liver as a result of interference of aflatoxin with lipid metabolism (Smith and Hamilton, 1970; Qota, 2003 and Hassan, 2005). There were a significant ( $P<0.05$ ) increase in relative weight of kidney, spleen and proventriculus for chicks fed AF-diet compared to the other treatments. These findings are in agreement with those obtained by Kubena *et al.* (1998) and Qota *et al.* (2005). Aflatoxin has been induce nutritional deficiency, resulting in depressed body weight, enlarged liver, spleen and proventriculus (Kubena *et al.*, 1990a and Huff *et al.*, 1992).

**Table 3. Relative weight (%) of some organs at the end of treatment and recovery periods as influenced by dietary AF and the treatment with humate levels and HSCAS**

Treatment	Liver	Spleen	Kidney	Proventri- culus	Gizzard	Heart	Bursa	Thymus
<b>At the end of treatment period (8 wk of age)</b>								
Control	2.236 <sup>d</sup>	0.216 <sup>d</sup>	1.076 <sup>d</sup>	0.370 <sup>c</sup>	2.428	0.432	0.250 <sup>a</sup>	0.450 <sup>a</sup>
AF-diet	3.516 <sup>a</sup>	0.340 <sup>a</sup>	1.490 <sup>a</sup>	0.450 <sup>a</sup>	2.409	0.450	0.183 <sup>c</sup>	0.330 <sup>c</sup>
AF+0.1% humate	2.943 <sup>b</sup>	0.293 <sup>b</sup>	1.376 <sup>ab</sup>	0.406 <sup>b</sup>	2.479	0.450	0.216 <sup>b</sup>	0.373 <sup>bc</sup>
AF+0.25% humate	2.500 <sup>d</sup>	0.254 <sup>c</sup>	1.250 <sup>bc</sup>	0.390 <sup>bc</sup>	2.413	0.460	0.230 <sup>ab</sup>	0.410 <sup>ab</sup>
AF+0.40% humate	2.460 <sup>d</sup>	0.240 <sup>cd</sup>	1.170 <sup>cd</sup>	0.383 <sup>bc</sup>	2.447	0.453	0.240 <sup>ab</sup>	0.420 <sup>a</sup>
AF+0.50% HSCAS	2.763 <sup>bc</sup>	0.256 <sup>c</sup>	1.340 <sup>b</sup>	0.403 <sup>b</sup>	2.435	0.435	0.216 <sup>b</sup>	0.406 <sup>ab</sup>
SEM	0.105	0.010	0.037	0.007	0.012	0.003	0.005	0.010
P-value	0.0001	0.0001	0.0001	0.0001	0.694	0.276	0.001	0.001
<b>At the end of recovery period (12 wk of age)</b>								
Control	2.416 <sup>c</sup>	0.223 <sup>c</sup>	1.106 <sup>c</sup>	0.393	2.440	0.445	0.240	0.440 <sup>a</sup>
AF-diet	3.496 <sup>a</sup>	0.300 <sup>a</sup>	1.346 <sup>a</sup>	0.416	2.410	0.463	0.200	0.370 <sup>c</sup>
AF+0.1% humate	2.910 <sup>b</sup>	0.280 <sup>ab</sup>	1.293 <sup>ab</sup>	0.406	2.478	0.443	0.217	0.396 <sup>bc</sup>
AF+0.25% humate	2.580 <sup>bc</sup>	0.246 <sup>bc</sup>	1.200 <sup>abc</sup>	0.393	2.469	0.446	0.226	0.420 <sup>ab</sup>
AF+0.40% humate	2.520 <sup>c</sup>	0.236 <sup>c</sup>	1.180 <sup>bc</sup>	0.396	2.450	0.450	0.233	0.426 <sup>ab</sup>
AF+0.50% HSCAS	2.723 <sup>bc</sup>	0.250 <sup>bc</sup>	1.293 <sup>ab</sup>	0.410	2.441	0.450	0.220	0.416 <sup>ab</sup>
SEM	0.095	0.007	0.025	0.004	0.0103	0.002	0.005	0.007
P-value	0.0001	0.002	0.024	0.379	0.460	0.204	0.389	0.024

a, b, c, d Means in the same column having different litter are significantly different ( $P<0.05$ )

Supplementation of humate levels or HSCAS decreased the severity of AF on all organs weights. Humate levels (0.1, 0.25 and 0.40%) decreased the severity of aflatoxicosis on organs weight by (44.53, 79.00 and 82.40%), (37.90, 69.36 and 80.64%), (28.56, 57.15 and 76.18%) and (55.00, 75.00 and 83.77%), while HSCAS showed 58.59, 67.74, 35.72 and 58.72% protection for liver, spleen, kidney and proventriculus, respectively, against 1 ppm AFB1 at the end of treated period. These findings agreed with those obtained by Rensburg *et al.* (2006) who reported that

oxihumate showed significant protective effects with respect to liver damage as indicated by an inhibition of liver enlargement. The results of Kubena *et al.* (1990a&b), Huff *et al.* (1992) and Hassan (2000 and 2005) showed that adding HSCAS to aflatoxic diet alleviated the adverse effect of aflatoxicosis on liver damage. Decreasing bursa and thymus glands may be attributed to the depletion of follicular lymphocytes (Abd El-Hamid *et al.*, 1992). Adding the studied additives decreased the severity of aflatoxin diets effects on the bursa and thymus weights. Protection of organs weight was observed by 0.1% humate (49.25, 35.82%), 0.25% humate (70.15, 66.70%), 0.40% humate (85.07, 75.02%) and HSCAS (49.25, 63.35%) for bursa and thymus, respectively, against 1 ppm AFB1/ kg diet at the end of treatment period.

After 4 weeks of recovery period, it was noticed that relative weight of organs were more recovered for groups fed aflatoxic diets treated with humate levels and HSCAS compared to AF-diet. On the other hand, heart, gizzard and proventriculus weights were not statistically different among treatments and the control group (Table 3). This finding is in agreement with those of Hegazy and Edris (1991) and Qota *et al.* (2005) who found that after 4 weeks of recovery period, liver, spleen, bursa and thymus of chicks fed AF-diet were not completely recovered.

#### **Chemical Composition of Meat and Liver:**

Data in Table 4 show the chemical composition of meat and liver at 8<sup>th</sup> and 12<sup>th</sup> weeks of age. There was a significant decrease in protein content of meat of aflatoxin treated chicks, while insignificant decrease in fat content and significant ( $P<0.05$ ) increase in moisture content were observed. Liver contents of AF-groups showed significantly ( $P<0.05$ ) higher fat and lower protein and moisture contents compared to those of the control group at the 8<sup>th</sup> week of treatment period. These results confirmed those obtained by Smith and Hamilton (1970); Inova *et al.* (1985) and Abd El-Hamid *et al.* (1995) who reported that aflatoxicosis impaired fat transport, which could be attributed to inhibited RNA synthesis, caused a marked increase in liver fat and as a result of that, its moisture was decreased, while protein content was decreased due to lowering the synthesis of protein in the liver (Abd El-Hamid *et al.* 1992). Adding humate or HSCAS decreased the severity of aflatoxicosis effect on meat and liver component, especially the groups fed aflatoxic diets treated with 0.25 and 0.40% humate. At the end of recovery period, the same alterations of treatments were shown only in contents of liver, while meat protein and moisture of groups fed detoxification additives showed similar value to the control group. Areif (1994) and Genedy *et al.* (1999) found that liver components did not fully recovered after 3 and 4 weeks of withdrawal from aflatoxicosis.

#### **Nutrients Digestibility:**

Addition of AF decreased the digestibility of dry matter, crude protein, crude fiber and ether extract compared to control diet (Table 5). Adding detoxification feed additives to aflatoxin supplemented diets improved digestibility coefficients of EE and CP, while complete recovery was noticed in DM and EE digestibility when humate was supplemented at 0.25 or 0.40%. These results agree with those of Compbell *et al.* (1983) who found that aflatoxin contaminated feed decrease the activities of several enzymes important in the digestion of carbohydrates, protein, lipids and nucleic acids in broiler chicks. Nelson *et al.* (1982) and Johri *et al.* (1996) found a decrease in amino acid and dry matter digestibility and energy utilization in

broiler fed mycotoxins. Bolden and Jensen (1985) stated that nutritional deficiency induced by the aflatoxin could have disrupted the activity of the digestive enzymes and the absorption of essential nutrients.

**Table 4. Chemical analysis (%) of meat and liver at the end of treatment and recovery periods as influenced by dietary AF treated with humate levels and HSCAS**

Treatment	Meat			Liver		
	Moisture	Protein	Fat	Moisture	Protein	Fat
<b>At the end of treatment period (8 wk of age)</b>						
Control	74.00 <sup>b</sup>	76.66 <sup>a</sup>	13.56	80.13 <sup>a</sup>	73.03 <sup>a</sup>	17.36 <sup>c</sup>
AF-diet	79.00 <sup>a</sup>	71.70 <sup>c</sup>	13.00	75.30 <sup>c</sup>	65.33 <sup>d</sup>	26.53 <sup>a</sup>
AF+0.1% humate	76.83 <sup>ab</sup>	74.26 <sup>b</sup>	13.23	77.00 <sup>b</sup>	67.73 <sup>cd</sup>	23.16 <sup>b</sup>
AF+0.25% humate	75.80 <sup>ab</sup>	75.06 <sup>ab</sup>	13.46	77.93 <sup>b</sup>	70.93 <sup>ab</sup>	20.86 <sup>b</sup>
AF+0.40% humate	75.40 <sup>b</sup>	75.23 <sup>ab</sup>	13.53	78.23 <sup>b</sup>	71.00 <sup>ab</sup>	20.16 <sup>bc</sup>
AF+0.50% HSCAS	75.63 <sup>ab</sup>	75.40 <sup>ab</sup>	13.26	77.15 <sup>bc</sup>	69.43 <sup>bc</sup>	21.56 <sup>b</sup>
SEM	0.519	0.429	0.110	0.379	0.669	0.760
P-value	0.087	0.003	0.792	0.0001	0.001	0.001
<b>At the end of recovery period (12 wk of age)</b>						
Control	74.10	77.06	14.10	79.16 <sup>a</sup>	73.63 <sup>a</sup>	18.00 <sup>b</sup>
AF-diet	76.06	75.00	13.56	77.03 <sup>b</sup>	70.63 <sup>c</sup>	22.63 <sup>a</sup>
AF+0.1% humate	75.60	76.10	13.80	77.60 <sup>ab</sup>	71.80 <sup>bc</sup>	20.86 <sup>ab</sup>
AF+0.25% humate	75.00	76.00	14.10	78.00 <sup>ab</sup>	72.36 <sup>b</sup>	19.66 <sup>b</sup>
AF+0.40% humate	74.90	76.26	13.66	77.93 <sup>ab</sup>	72.46 <sup>b</sup>	19.43 <sup>b</sup>
AF+0.50% HSCAS	75.20	76.15	13.66	76.96 <sup>b</sup>	71.76 <sup>bc</sup>	20.00 <sup>ab</sup>
SEM	0.432	0.286	0.089	0.243	0.253	0.457
P-value	0.542	0.893	0.375	0.068	0.002	0.050

<sup>a, b, c, d</sup> Means in the same column having different litter are significantly different (P<0.05)

**Table 5. Digestibility of nutrients (%) at the end of treatment and recovery periods as influenced by dietary AF and treatment with humate levels and HSCAS**

Treatment	DM	CP	EE	CF
	<b>At the end of treatment period (8 wk of age)</b>			
Control	79.50 <sup>a</sup>	80.33 <sup>a</sup>	76.56 <sup>a</sup>	23.00
AF-diet	74.03 <sup>c</sup>	73.53 <sup>b</sup>	68.93 <sup>c</sup>	22.40
AF+0.1% humate	76.26 <sup>bc</sup>	77.60 <sup>a</sup>	71.96 <sup>b</sup>	23.20
AF+0.25% humate	77.26 <sup>ab</sup>	78.10 <sup>a</sup>	73.26 <sup>b</sup>	23.16
AF+0.40% humate	77.86 <sup>ab</sup>	78.55 <sup>a</sup>	74.00 <sup>b</sup>	22.40
AF+0.50% HSCAS	76.43 <sup>bc</sup>	77.70 <sup>a</sup>	72.93 <sup>b</sup>	23.20
SEM	0.504	0.584	0.622	0.379
P-value	0.015	0.004	0.001	0.982
<b>At the end of recovery period (12 wk of age)</b>				
Control	79.26	80.00 <sup>a</sup>	76.00 <sup>a</sup>	22.00
AF-diet	76.13	76.93 <sup>b</sup>	70.73 <sup>b</sup>	22.03
AF+0.1% humate	77.30	78.56 <sup>ab</sup>	74.30 <sup>a</sup>	22.46
AF+0.25% humate	78.30	78.86 <sup>a</sup>	74.43 <sup>a</sup>	23.16
AF+0.40% humate	78.03	79.26 <sup>a</sup>	74.53 <sup>a</sup>	22.06
AF+0.50% HSCAS	77.80	78.33 <sup>ab</sup>	75.16 <sup>a</sup>	22.13
SEM	0.338	0.301	0.475	0.247
P-value	0.133	0.045	0.006	0.791

<sup>a, b</sup> Means in the same column having different litters are significantly different (P<0.05)

After 4 weeks of recovery period, digestibility of CP and EE was still low in aflatoxin supplemented group. Meanwhile, group fed diets supplemented with humate (0.25 and 0.40%) or HSCAS showed nearly similar nutrient digestibility to that of the control group.

#### **Serum Constituents:**

Data in Table 6 demonstrate the effect of aflatoxin on serum constituents during treatment and recovery periods. The results clearly indicated that at the treatment period, total protein, albumin, total lipids, cholesterol and uric acid were reduced, but the serum enzymatic activity of AST and ALT increased in chicks fed AF-diet. Decreasing serum protein may be due to lower liver synthesis of albumin and globulin as a result of aflatoxicosis. These results are similar to those obtained by Abd El-Hamid *et al.* (1992), while decreasing serum total lipids may be due to the influence of aflatoxin with lipid metabolism (Hamilton *et al.*, 1972). The low serum cholesterol concentration is most likely due to inhibition of cholesterol biosynthesis, with liver involvement, and perhaps a shift of concentration from the blood to the liver (Kubena *et al.*, 1993). The serum enzymes data in the current results showed that the activity of AST, ALT and creatine kinase were increased by aflatoxin. These results supported the hepatotoxic effect of aflatoxin and consequence hepatic cell damage and liver hypofunction (Varley *et al.*, 1980). The kidney function data showed significant ( $P < 0.05$ ) decrease in uric acid and increase in creatine kinase.

**Table 6. Some blood constituents at the end of treatment and recovery periods as influenced by dietary AF and treatment with humate levels and HSCAS**

Treatment	TP g/ 100 ml	Alb g/ 100 ml	TL g/l	Chol g/ 100 ml	AST IU/ L	ALT IU/ L	UA mg/ dl	Cr mg/ dl
<b>At the end of treatment period (8 wk of age)</b>								
Control	4.31 <sup>a</sup>	2.33 <sup>a</sup>	7.23 <sup>a</sup>	175.0 <sup>a</sup>	107.66 <sup>d</sup>	12.38 <sup>c</sup>	7.56 <sup>a</sup>	1.98 <sup>c</sup>
AF-diet	2.21 <sup>d</sup>	1.53 <sup>e</sup>	5.08 <sup>e</sup>	126.66 <sup>c</sup>	167.66 <sup>a</sup>	19.83 <sup>a</sup>	5.33 <sup>d</sup>	2.53 <sup>a</sup>
AF+0.1%humate	2.44 <sup>d</sup>	1.78 <sup>d</sup>	5.69 <sup>d</sup>	149.00 <sup>b</sup>	132.00 <sup>bc</sup>	16.06 <sup>b</sup>	5.83 <sup>cd</sup>	2.45 <sup>a</sup>
AF+0.25%humate	3.40 <sup>b</sup>	2.07 <sup>bc</sup>	6.25 <sup>c</sup>	161.33 <sup>b</sup>	134.33 <sup>b</sup>	14.33 <sup>bc</sup>	6.26 <sup>bc</sup>	2.14 <sup>bc</sup>
AF+0.40%humate	3.62 <sup>b</sup>	2.23 <sup>ab</sup>	6.82 <sup>b</sup>	157.66 <sup>b</sup>	121.33 <sup>c</sup>	14.00 <sup>bc</sup>	6.73 <sup>b</sup>	2.07 <sup>bc</sup>
AF+0.50%HSCAS	3.05 <sup>c</sup>	2.03 <sup>c</sup>	6.44 <sup>c</sup>	155.66 <sup>b</sup>	129.66 <sup>bc</sup>	14.63 <sup>bc</sup>	6.16 <sup>bc</sup>	2.32 <sup>ab</sup>
SEM	0.174	0.068	0.173	3.853	4.568	0.613	0.187	0.058
P-value	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.009
<b>At the end of recovery period (12 wk of age)</b>								
Control	4.40 <sup>a</sup>	2.42 <sup>a</sup>	7.15 <sup>a</sup>	177.33 <sup>a</sup>	109.33 <sup>d</sup>	12.58 <sup>c</sup>	7.63 <sup>a</sup>	2.02 <sup>c</sup>
AF-diet	3.11 <sup>e</sup>	2.07 <sup>c</sup>	5.93 <sup>d</sup>	139.00 <sup>d</sup>	146.00 <sup>a</sup>	18.50 <sup>a</sup>	5.70 <sup>c</sup>	2.40 <sup>a</sup>
AF+0.1%humate	3.38 <sup>de</sup>	2.18 <sup>bc</sup>	5.99 <sup>d</sup>	157.00 <sup>c</sup>	131.00 <sup>b</sup>	15.80 <sup>b</sup>	6.16 <sup>bc</sup>	2.32 <sup>ab</sup>
AF+0.25%humate	3.82 <sup>bc</sup>	2.27 <sup>b</sup>	6.48 <sup>c</sup>	167.33 <sup>b</sup>	132.66 <sup>b</sup>	14.16 <sup>bc</sup>	6.60 <sup>b</sup>	2.11 <sup>c</sup>
AF+0.40%humate	3.93 <sup>b</sup>	2.32 <sup>ab</sup>	7.05 <sup>ab</sup>	164.66 <sup>bc</sup>	119.33 <sup>c</sup>	14.06 <sup>bc</sup>	6.76 <sup>b</sup>	2.02 <sup>c</sup>
AF+0.50%HSCAS	3.62 <sup>cd</sup>	2.19 <sup>bc</sup>	6.78 <sup>bc</sup>	164.00 <sup>bc</sup>	129.00 <sup>b</sup>	14.30 <sup>bc</sup>	6.70 <sup>b</sup>	2.19 <sup>bc</sup>
SEM	0.104	0.031	0.121	3.00	2.958	0.485	0.167	0.040
P-value	0.0001	0.002	0.0001	0.0001	0.0001	0.0001	0.003	0.002

<sup>a, b, c, d</sup> Means in the same column having different litter are significantly different ( $P < 0.05$ )

Elevation of creatine kinase in chickens fed aflatoxin supplemented diet is probably a result of muscle trauma (Tietz, 1976). While, the effect of AF on serum uric acid was similar to the effect on serum protein level, this result agreed with those

obtained by Reddy *et al.* (1984) who found a decrease in uric acid percentage during aflatoxicosis.

Feed additives supplementation alleviated the adverse effect of aflatoxicosis on serum constituents during the treatment period. The best results were recorded for group fed diet supplemented with 0.25 or 0.40% humate, which provided more protection than HSCAS (Table 6). This could explain superior performance of these groups. These results are in agreement with those of Rensburg *et al.* (2006) who found that total protein, albumin and creatine phosphokinase were returned to control value, when oxihumate was used with aflatoxin exposed chickens. Addition of HSCAS to the diet containing AF significantly ( $P < 0.05$ ) decreased the negative effect of AF on the serum constituents (Genedy *et al.*, 1999; Hassan, 2000 and Qota *et al.*, 2005).

At the end of recovery period, all groups showed similar serum constituents to the control group except for AST, ALT and creatine kinase of chicks fed AF-diet. These results are in agreement with those reported by Hassan (2000&2005).

#### ***Aflatoxin Residues:***

AFB<sub>1</sub> residue was found in liver and meat tissues of chicks fed aflatoxin diets without or with studied additives at the end of treatment period, while the control group showed no residues of AFB<sub>1</sub> in their tissues (Table 7). These results confirmed those obtained by Trucksess *et al.* (1983); Sova *et al.* (1984) and Hegazy and Edris (1991) who found that chickens fed aflatoxin diets accumulate residues of AFB<sub>1</sub> in their meat and liver tissues. Concentrations of AFB<sub>1</sub> residues were higher in liver than in meat of all aflatoxin groups at the end of treatment period (Table 7). These results agree with those obtained by Abd El-Hamid *et al.* (1995); Abo Sree *et al.* (1999) and Qota *et al.* (2005) who found that liver accumulate more aflatoxin than meats of chicks fed AF- supplemented diets. Adding studied additives to AF-diet decreased residues of AFB<sub>1</sub> in the liver and meat with 0.1% humate by (20.83, 21.43 %), with 0.25% humate by (56.69, 35.21 %), with 0.40% humate by (54.31, 35.71 %) and with HSCAS by (50.00, 33.78 %), respectively, compared to aflatoxin containing diet without additives (Table 7). The efficiency values of humate at levels (0.25 and 0.40%) were higher than HSCAS for diminished aflatoxicosis and decreased AFB<sub>1</sub> residue in liver and meat at the end of treatment and recovery periods. Concentrations of AFB<sub>1</sub> residues in the liver and meat were decreased by 53-71 % after 4 weeks recovery period compared to that of the treatment period (Table 7). Aflatoxin was absent from tissues or presented with low concentrations in breast and leg and removed in two to four weeks after withdrawing the toxic diet ( El-Shaarawi *et al.*, 1983 and Mocco *et al.*, 1988).

*In vitro* experiment showed that oxihumate mixed with poultry feed absorbed AFB<sub>1</sub> with the same efficacy in buffer alone, at pH<sub>3</sub> and pH<sub>7</sub>, suggesting that oxihumate does not bind feed molecules. In this experiment, oxihumate bound 92.0% of the AFB<sub>1</sub> at pH<sub>3</sub> and 82.2% at pH<sub>7</sub>, whereas 92.9% and 87.5% of the AFB<sub>1</sub> were bound at pH<sub>3</sub> and pH<sub>7</sub>, respectively, in the presence of both oxihumate and poultry feed. All oxihumate particles mixed into mycotoxin-contaminated feed should, therefore, be available for the formation of oxihumate-mycotoxin complex. The complex may, however, not be very stable, as it was ruptured to a high degree in the presence of acetonitrile (61%) and acetone (75%), but not chloroform (3%). The failure of chloroform to rupture the complex may be attributed to the high capacity of

oxihumate to hold water in and around its structure with a consequential gel formation, thus preventing the hydrophobic chloroform from penetrating the water layer. The aflatoxin in association with oxihumate is water-insoluble and the chloroform can, therefore, not reach the complexes (Rensburg *et al.*, 2006).

**Table 7. Effect of humate levels and hydrated sodium calcium aluminosilicate (HSCAS) on the AFB<sub>1</sub> residues (ng/g.) in liver and meat of birds fed diet containing 1 ppm AFB<sub>1</sub>**

Treatment	Treatment period		Recovery period	
	Liver	Muscles	Liver	Muscles
Control	0.00*	0.00	0.00	0.00
AF-diet	80.00 <sup>a</sup>	14.00 <sup>a</sup>	28.00 <sup>a</sup>	6.47
AF+0.1%humate	63.33 <sup>b</sup>	11.00 <sup>ab</sup>	18.00 <sup>b</sup>	3.78
AF+0.25%humate	34.65 <sup>c</sup>	9.07 <sup>b</sup>	16.00 <sup>b</sup>	2.55
AF+0.40%humate	36.55 <sup>c</sup>	9.00 <sup>b</sup>	12.55 <sup>b</sup>	3.00
AF+0.50%HSCAS	40.00 <sup>c</sup>	9.26 <sup>b</sup>	16.30 <sup>b</sup>	3.28
SEM	5.051	0.665	1.554	0.526
P-value	0.0001	0.046	0.001	0.109

\* No detecting of aflatoxin B<sub>1</sub>

<sup>a, b, c, d</sup> Means in the same column having different litter are significantly different (P<0.05)

The *in vitro* experiments carried out with radio labeled aflatoxin B<sub>1</sub> (Phillips *et al.*, 1988) demonstrated that of 38 different absorbents tested (a variety of aluminas, zeolites, silicas, phyllosilicates and chemically modified phyllosilicates), HSCAS was the best aflatoxin B<sub>1</sub>-absorbing compound as it was able to form the most stable complex with this mycotoxin (with adsorption of more than 8% of aflatoxin B<sub>1</sub> present in the medium). The complex was also demonstrated to be stable in water at pH<sub>2, 7 and 10</sub> and at temperatures of 25 and 37°C. The stability of the HSCAS-aflatoxin B<sub>1</sub> sorption complex was evaluated by extraction with an elutropic series of solvents. As less than 10% of aflatoxin B<sub>1</sub> was extracted, they suggested that the mechanism implicated in this process could be chemisorption with the formation of strong bonds between the molecules, but later on, a more specific mechanisms was proposed (Phillips *et al.*, 1990b and Sarr *et al.*, 1990). This mechanism was the formation of a complex by the β-carbonyl system of the aflatoxin with uncoordinated edge site aluminum ions HSCAS.

In the present study, results indicated that adding HSCAS to chicks diet as a decontamination method to sorb aflatoxin selectively during the digestive process, was less effective compared to humate (0.25 and 0.40%). Oxihumate showed a high affinity for AFB<sub>1</sub>, zearalenone, ochratoxin A, ergosine, ergotamine, ergocormine, ergocryptine and ergocristine, but did not bind to vomitoxin. The binding capacity of Mycosorb (HSCAS) to AFB<sub>1</sub> at PH<sub>3</sub> proved to be considerably less than that of oxihumate (Rensburg *et al.*, 2006).

These results clearly demonstrated that the growth performance (BW, FI and FCR), weights of some internal organs, blood constituents, nutrients digestibility and immune response of local chicks were negatively affected by feeding AFB<sub>1</sub> for 8 weeks. Meanwhile, addition of humate levels and HSCAS to AF-containing diet significantly (P<0.05) recovered and ameliorated the adverse effects of AF on performance especially, humate at 0.25 and 0.40% which resulted in the best mean.

These positive effects may have a safe and practical decontamination procedure and contribute to a solution of AF problem in poultry.

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## تأثير الهيموتات و سليكات الصوديوم والكالسيوم والألومنيوم على سمية الأفلاتوكسين فى علائق الكتاكيت المحلية

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أستخدم عدد ٣٦٠ كنبوت غير مجنس عمر يوم من السلالة المستنبطة السلام و قسمت الكتاكيت عشوائيا فى ٦ معاملات كل منها فى ثلاث مكررات . تغذت المعاملة الأولى على عليقة أساسية بدون إضافات والمعاملة الثانية على العليقة الأساسية الملوثة بمعدل ١ جزء فى المليون أفلاتوكسين ب ١ / كيلو جرام علف والمعاملات الثالثة والرابعة والخامسة والسادسة تم تغذيتها على عليقة ملوثة بالأفلاتوكسين مضاف إليها ٠.١ ، ٠.٢٥ ، ٠.٤ ، ٠.٥ % هيموتات أو ٠.٥ % سليكات الصوديوم والكالسيوم والألومنيوم على الترتيب واستمرت التجربة من عمر يوم حتى ٨ أسابيع ( كفترة معاملة ) وبعد ذلك تم تغذية الكتاكيت على عليقة خالية من الأفلاتوكسين والإضافات فى الفترة من ٨ إلى ١٢ أسبوع ( كفترة استشفاء ) .

أظهرت النتائج ما يلى :

- ١- أدت التغذية على العلف الملوث بالأفلاتوكسين بدون إضافات الى انخفاض وزن الجسم والعلف المستهلك وتدهور الكفاءة الغذائية وزادت نسبة النفوق بالمقارنة بمجموعة الكنترول فى نهاية فترة المعاملة
  - ٢- زاد الوزن النسبى لكل من الكبد ، الكلية ، الطحال والمعدة الغدية معنويا فى الكتاكيت التى تغذت على عليقة ملوثة بالأفلاتوكسين فقط .
  - ٣- انخفضت قيم البروتين الكلى ، الألبومين ، الليبيدات الكلية ، الكوليستيرول وحمض اليوريك وارتفعت قيم انزيمات وظائف الكبد فى سيرم دم الكتاكيت التى تغذت على عليقة ملوثة بالأفلاتوكسين فقط .
  - ٤- انخفضت معاملات هضم المركبات الغذائية فى المجموعة التى تغذت على عليقة ملوثة بالأفلاتوكسين فقط .
  - ٥- أدى استخدام الهيموتات ( ٠.٠ ، ٠.٢٥ ، ٠.٤ % ) الى حماية معدل النمو بمعدل ( ٦٠.٧٧ ، ٧٨.٥٥ ، ٨٠.٣٦ % ) والعلف المستهلك بمعدل ( ٥٢.٨٦ ، ٦٣.٩٨ ، ٥٠.٠٠ % ) والكفاءة الغذائية بمعدل ( ٧٠.٣٠ ، ٨٩.٠٥ ، ٩٥.٣٠ % ) على التوالى بينما ادى اضافة سليكات الصوديوم والكالسيوم والألومنيوم الى حماية معدل النمو بمعدل ( ٧٣.٥٢ % ) والعلف المستهلك بمعدل ( ٥٣.٤٤ % ) والكفاءة الغذائية بمعدل ( ٨٥.٩٥ % ) ضد تلوث الغذاء بمعدل ١ جزء فى المليون أفلاتوكسين ب ١ / كيلو جرام علف .
- ومن هنا يتضح بأن إضافة ٠.٢٥ أو ٠.٤٠ % هيموتات هو الأكفأ بالمقارنة بسليكات الصوديوم والكالسيوم والألومنيوم لإزالة سمية الأفلاتوكسين فى علف الكتاكيت النامية ولهذا يعتبر الهيموتات عامل هام لتخفيف سمية الأفلاتوكسن .