

CAPABILITY OF MANNAN-OLIGOSACCHARIDE (BIO-MOS(R)), ORGANIC SELENIUM AND HYDRATED SODIUM CALCIUM ALUMINOSILICATE TO DETOXYIFY AFLATOXICOSIS FOR GROWING LOCAL CHICKENS.

2- Lymphoid organs, Immune response and residues in tissues.

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Abstract: A total number of 270 unsexed El-Salam chickens at two-weeks of age were used in this study. Chickens were randomly assigned for six treatments (45 bird each). Chickens of the 1st group served as control . Chickens of the 2nd group was received the basal diet contaminated with 1 mg Aflatoxin B₁ (AFB₁)/ kg diet . The 3rd , 4th , 5th and 6th groups were received AFB₁ – diet supplemented with 1g Bio-Mos^(R), 0.5 mg selenomethionine (Se), (1g Bio-Mos + 0.5 mg Se/kg diet) or 0.25% Hydrated sodium calcium aluminosilicate (HSCAS), respectively, for 6 weeks treatment period , After 6 weeks of treatment, chickens were allowed without any treatment for other 4 weeks as recovery period.

Main results obtained can be summarized as follows:-

1- Bursa of fabricius and thymus glands relative weights, hemoglobin (Hb), concentration Red blood cell (RBCs), white blood cell (WBC's) and packed cell volume (PCV%) were significantly decreased in chickens fed diet with AFB₁.

2- The geometric mean of HI antibody response to Newcastle disease vaccines (NDV) did not differ significantly between the AFB₁-diet and control diet during the treatment period .

3-High values of AFB₁ residue was found in liver tissues than that in meat .

4-Bio-MOS and /or Se or HSCAS to aflatoxin contaminated diets decreased residues of AFB₁ in liver and meat compared with control diet. After 4 weeks recovery period, the level of AFB₁ residue was decreased by 57- 88 % in liver for all aflatoxin groups compared with treatment period, while AFB₁ was absent from meat at the end of recovery period .

5- Chickens fed diet contaminated with AFB₁ showed sever signs of low viability, anorexia, perosis, atoxia, acattered feathering and nasal haemorrhage. However, these signs were decreased in chicks when Bio-Mos, Se or HSCAS was supplemented and further improvement was occurred due to a combination of Bio-Mos plus Se. Also, most of these sings were recovered during the 4th week of the withdrawal period especially for chicks fed AFB₁ plus Bio-Mos plus Se.

Bio- Mos , Se or HSCAS decreased aflatoxicosis of chickens fed diet contaminated with AFB₁ especially , when Bio – Mos and Se was combined during the exposure period and helped to fasten during the recovery period .

INTRODUCTION

Aflatoxins (AF) are secondary toxic metabolites produced by certain fungi belonging to the genus *Aspergillus* and can occur as natural contaminants of poultry feeds. AF may cause serious economic losses in the poultry industry because it prevents animals from reaching their optimum body weight gain and reproduction, (Oguz and Kurtoglu 2000). Aflatoxicosis in poultry also causes listlessness, anorexia with lowered growth rate; poor feed utilization, decreased egg production and increased mortality (Miazzo et al., 2000). Additionally, anemia (Oguz et al., 2000), reduction of immune function (Oguz et al., 2003), hepatotoxicosis and hemorrhage (Ortatatli and Oguz 2001) are associated with aflatoxicosis. Aflatoxin may be inhibit the glutathione peroxidase activity as well as decrease the ability to scavenge oxygen free radicals directly or indirectly, which stimulates lipid peroxidation due to an increase of free radical generation (Hoehler and Marquardt, 1996). The most common organ in which aflatoxin residue can be detected very early in the liver in rate 0.1% of the amount of toxin consumed (Refai, 1988).

Recent biotechnological progress has opened new avenues for tackling this problem. Live yeast (*Saccharomyces cerevisiae*: SCE), initially used as a performance promoter in the early 1990, was found to have beneficial effect on weight gain and immune response in broilers exposed to AF (Stanley et al., 1993). Recent studies made with SCE also showed significant improvements in aflatoxicosis cases in quail chicks (Parlat et al., 2001, Yildirim and Parlat, 2003). The beneficial effects of SCE have been later attributed to Mannan oligosaccharide (MOS) drived from cells wall of SCE. The researchers discovered and extracted MOS and used for removing pathogenic bacteria from the intestine (Fernandez et al., 2002) and immuno- modulation (Hooge, 2004) in poultry . MOS also showed considerably high binding ability (80% to 97%) with AF (Mahesh

and Devegowda 1996) and it has been preferred for detoxification of AF in poultry species. The studies performed by MOS (0.5 and 1g / kg) with different concentrations of AF (0.05 to 1 mg / Kg) in broilers (Raju and Devegowda 2000, and Santin et al., 2003) showed that MOS partially and / or completely reversed the adverse effect of AF on performance, biochemistry, hematology and immune response of birds .

Adding HSCAS to chicks diets in the present study is one of the decontamination methods to sorb aflatoxin selectively during the digestive process, which rendered most of the aflatoxin unavailable for absorption from the gastrointestinal tract (Kubena et al., 1993 and Abo- Norag et al., 1995).

The role of selenium in biological system has been associated with its antioxidant activity (Schwary and Foltz, 1987). Its physiological importance was recognized when it was found to be an essential structural component of the glutathione peroxidase enzyme, the same primary site for action in the liver between selenium and aflatoxin (Dalvi and Robbins, 1978).

In an effort to develop a practical method for AF detoxification, therefore, the objective of following research using growing chicks was to examine the toxic effects of AF (1mg / kg) on lymphoid organs, immune response and residues in tissues of chicks and to evaluate the preventive efficacy of Bio – Mos, Se or HSCAS as a feed additives to suppress or counteract the severity of aflatoxicosis on local chicks.

MATERIALS AND METHODS

This experiment was carried out at Sakha Animal Research Station , Animal Production Research Institute, Ministry of Agriculture, Egypt. A total of 270 local chickens at two weeks of age were used to study the effect of basal diet contaminated with AFB₁ on (1- performance, some internal organs and blood constituents) and (immune response and AFB₁ residual in tissues) as previously described (Hassan, 2005) when given Bio-Mos and /or Se or HSCAS in growing local chicks . Aflatoxin was produced via fermentation of rice by *Asperigillns 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 spectrophotometrically for its aflatoxins content by method of Nabney and Nesbitt (1965) as modified by Wiseman *et al.* (1967). Aflatoxin in the rice powder were extracted by chloroform then incorporated into the basal diet and aflatoxin concentration were confirmed by HPLC to provide the desired level of 1 mg AFB₁ per kg diet . The birds were fed a starter mash (2950

kcal ME/kg; 19.5% crude protein) from 2 to 12 weeks of age. Aflatoxin on the basal diet showed very little amount of 5 ppb.

Experimental diets:

- 1- control diet without aflatoxin and any additives (Basal diet).
- 2- Basal diet + 1 mg AFB₁/kg diet
- 3- Basal diet + 1 mg AFB₁ + 1 g Bio-Mos⁽¹⁾ (Mos)/kg diet.
- 4- Basal diet + 1 mg AFB₁ + 0.5 mg selenomethionine⁽²⁾/kg diet.
- 5- Basal diet + 1 mg AFB₁ + 1g Bio- Mos + 0.5 Se/kg diet
- 6- Basal diet + 1 mg AFB₁ + 0.25% HSCAS⁽³⁾.

Chickens were randomly divided into six experimental groups with three replicates of 15 chicks each. The experimental groups were fed on the experimental diets from 2 weeks to 8 weeks of age (treatment period). Then they were fed on the basal diet from 8 to 12 weeks of age without any supplement (recovery period). After 8 and 12 week of age, nine birds from each treatment were slaughtered. Blood samples were collected from each bird in clean tubes with heparin and placed in a refrigerator immediately. The heparinized blood was used for determination of hemoglobin (Hb), packed cell volume (PCV), red blood cell (RBC'S) and white blood cell (WBC'S) count according to Schalm et al. (1975). Lymphoids organs (thymus and bursa of Fabricius) were removed and weighed at end of treatment and recovery periods. Serum samples were collected at 20 and 32 days of age during treatment period for HT titers against Newcastle disease vaccine (NDV) according to the method described by Anon (1980). At the end of treatment and recovery periods, samples of liver and muscles were obtained from three chicks of each group and kept in deep freezing at - 20 °C until analysis. Detection of aflatoxin residue in above mentioned organs was made by the method described in AOAC (1975). Chicks of all treatments were vaccinated a 7 days of age by Hitchner B1, lasota at 21 and 33 days of age. Data were statistically analyzed using the General Linear Model for

¹ Bio-Mos[®] was provided from Alltech, Nichola Siville, Kentucky USA, and was applied at the rate of 1g/kg of feed equivalent and mixed

²Selenomethionine as organic source of selenium provided from Calbiochem, LaJolla, California.

³ Hydrated sodium calcium aluminosilicate clay (HSCAS) is a chemical compound that contains silicon oxide (64.7%), aluminum oxide (15.5%), oxides of iron, magnesium, calcium, sodium, potassium (8.9%) and moisture (10.9%) provided from Integrated World Enterprises Co. USA.

analysis of variance (SAS, 1990). Duncan's multiple range test (Duncan 1955) was used for test the significance ($p < 0.05$) of differences among means.

RESULTS AND DISCUSSION

1- Lymphoid organs:

Relative weights of lymphoid organs (thymus and bursa of fabricius) are presented in Table (1). It is clear that aflatoxin fed group showed significantly ($p < 0.05$) decreases in relative weight of bursa (42.42%) and thymus (51.22%) by 1mg AFB₁ at the end of treatment period compared with the control group. Decreasing bursa of fabricius and thymus glands may be attributed to the depletion of follicular lymphocytes (Abd El-Hamid *et al.*, 1992). Also, the present results confirmed those obtained by Kubena *et al.*, (1990b), Edrington *et al.* (1997), Hassan (2000) and Qota *et al.* (2005). Adding the studied additives decreased the severity of aflatoxin diets effects on the bursa and thymus weights. Protections of organs weight by Bio – Mos were (42.86, 57.15 %), Se (42.86, 47.62 %), Bio- Mos plus Se (71.43, 85.71 %) and HSCAS (42.86, 52.38 %) for bursa of fabricius and thymus respectively, against 1mg AFB₁ / kg diet at the end of treatment period. These results confirmed those reported by Kubena *et al.* (1990 a&b and 1993), Abo-Norag *et al.* (1995), Hassan (2000) and Qota *et al.* (2005).

At the end of recovery period, all chicks of the different treatments were recovered except those fed AFB₁- contaminated diet without the detoxification additives (Table 2). This finding was in agreement with those of Marsh *et al.* (1986) , Genedy *et al.* (1999), Huang *et al.* (1999), Hassan (2000), Hegazy and Adachi (2000) and Qota *et al.* (2005) who found that after 4 weeks of recovery period, thymus and bursa of chicks fed AF- diet were not completely recovered .

2- Immune response:

The geometric mean of HI antibody response to NDV was presented in Table (1). No significant difference in HI antibody response was found between aflatoxicated diet and control group during the treatment period indicating that aflatoxin has no effect on HI antibody response to NDV. It appears that aflatoxin induced marked effect on cell- mediated than on humoral immunity and / or impaired phagocytosis. These results agree with Giambrone *et al.* (1978, 1985 a, b, c). Abd EL-Hamid *et al.* (1992) and Hassan (2000). They showed no effect of crude aflatoxin on humoral immunity or on the development of acquired immunity against NDV. They attributed this to the aflatoxin alteration of B and T cell functions.

On the other hand, some work with broilers indicated that AF produced by *A. parasiticus* on rice, produced immunotoxicity at 625 ppb as measured by antibody suppression and thymic and bursal atrophy (Thaxton et al., 1974) and impaired phagocytosis (Michael et al., 1973). In contrary, Campbell et al. (1983) reported that significant depression in the relative weight of bursa of fabricius was seen only in the interaction between 2.5 ug aflatoxin and 2 ug ochratoxin A but not with aflatoxin alone. This means that the effect of aflatoxin on the immune related organs (thymus and bursa) appears equivocal. Differences could result from age or genetic strain of birds, serologic techniques, nutrient content of diet, source of mixture of aflatoxin duration of toxin feeding or environment in which birds are kept (Abd El-Hamid et al., 1992).

Santin et al. (2003) found that mannan-oligosaccharide (MOS) partially and /or completely reversed the adverse effect of AF on immune response of birds.

3-Heamatological parameters:

After 6- weeks of treatment period (Table 1), analyses of blood revealed significant alterations in aflatoxicated diet groups. Significant decreases were realized in values of RBC'S, WBC'S, Hb% and PCV%. The data of the present blood profile are in accordance with those of Zilva and Pannall, (1983) who refer to presence of diseases or functional disorders in some organs may cause anemia (low Hb content and PCV%). Although, Bio-Mos and /or Se or HSCAS alleviated the toxic effect on heamatological parameters, yet the Bio-Mos or selenium had more pronounced effect especially, when Bio-Mos and Se was combined. Also, Huang and Chen,(1999) found that Se or Vit. E. improved markedly hematological parameters. Additionally, Devegowda et al. (1994) and Raju and Devegowds (2000) showed that MOS and yeast culture respectively, partially and /or completely reversed the adverse effect of AF on hematology of birds such as Hb and PCV%.

Hemoglobin and haematocrite values, were increased due to addition of Bio-Mos, and /or Se or HSCAS. This indicates the positive effects of these supplementations on heamatological parameters and the positive impact on liver and spleen as well as other tissues like bone marrow where red blood cells are synthesized.

After recovery period, all groups were partially recovered for heamatological parameters except WBC'S values were completely recovered (Table 2).

4- Aflatoxin residual:

AFB1 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 AFB1 in their tissues (Table 1). 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 AF1 in their meat and liver tissues . Concentrations of AFB1 residue were higher in liver (7- 10 times) than in meat of all aflatoxin groups at the end treatment period (Table 1). These results agree with those obtained by El- samra et al. (1991b); Rizk et al. (1993); Abdelhamid et al. (1995b); Abu Sree et al. (1999) and Qota et al. (2005) who found that liver accumulate more aflatoxin than meats of aflatoxin fed chickens. Adding studied additives to aflatoxin diets decreased residues of AFB1 in the liver and meat with Bio- Mos by (59.0 , 56.5 %), Se by (53.6 , 48.4 %), Bio- Mos plus Se by (83.3 , 74.2 %) and HSCAS by (55.7 , 51.6 %), respectively, compared with aflatoxin diet without additives (Table 1). The efficiency of the combination of Bio – Mos plus Se was higher than that Bio- Mos , Se or HSCAS for diminishing aflatoxicosis and decreased AFB1 residue in liver and meat at the end of treatment and recovery periods . Concentrations of AFB1 residues in the liver were decreased by 57 -88 % after 4 weeks recovery period compared to that of the treatment period (Table 2). After 4 weeks of recovery period, there was no residual AFB1 in meat tissue when Bio- Mos and /or Se and HSCAS were supplemented to AFB1 contaminated diet during the treatment period . Aflatoxin was absent from tissues or presented with low concentrations in breast and leg and removed one to two week after withdrawing the toxic diet (EL-Shaarawi et al., 1983 and Micco et al., 1988).

Concerning, clinical symptoms. Chickens showed sever signs in aflatoxicated-diet, but little severity was observed in chicks fed aflatoxicated diet containing Se or HSCAS and very little in chicks fed aflatoxicated diet containing Bio-Mos or/and Se. These symptoms included low viability, anorexia, perosis, ataxia, scattered feathering, nasal hemorrhage, cramps and stinked (greenish – bloody – watery) excrements. Most of these sings were recovered during the recovery period especially for chicks fed diets containing Bio-Mos or/and Se. Such clinical symptoms were reported often in aflatoxicosis, whether hemorrhages (Baker and Green, 1987 and Abdelhamid et al., 1993a), morbidity (Moreau, 1979), and abnormal feathering (Abdelhamid et al., 1993a). These symptoms may be due to aflatoxin which has an antivitamin action (Moreau, 1979), thus presents vitamins deficiency and immune suppression (Bassuni et al., 1990

and Mansy et al., 1993). Therefore, detoxification additives reflected less severity of the toxicity perhaps because of the action of these supplementations as immune stimulators as well as antioxidants, coagulants or coenzymes. Addition of *Saccharomyces cerevisius* to the aflatoxin contaminated feeds resulted in less severe pathological lesions (Churchil et al., 2001). Most of these signs were recovered during the 4th week of the withdrawal period, especially, when chicks fed diet supplemented Bio-Mos plus Se, due to indirect synergetic effect between Bio-Mos and Se.

The role of MOS in AF detoxification was attributed to its selective capacity for AF binding molecules in the gastrointestinal tract. (Faulkner, 1999), to modulate the immune response (Fernandez et al., 2002; Shashidhara and Devegowda, 2003), to provide nutrients to beneficial gut flora and this improve animal performance (Fritts and Waldroup 2003, Santin et al., 2003 and Hooge 2004).

The role of selenium in biological system has been associated with its antioxidant activity (Schwarz and Foltz, 1987). Its physiological importance was recognized when it was found to be an essential structural component of the glutathione peroxidase enzyme, the same primary site for action in the liver between selenium and aflatoxin (Dalvi and Robbins, 1978). Moreover, Se has an important potent role in preventing oxidative damage in erythrocytes or tissues (Rotruck et al., 1973). In chickens, Cantor et al. (1975) found that selenium deficiency induced oxidative diathesis or pancreatic fibrosis which has a negative effect on pancreatic enzymes secretion. Supplementation of selenium to reach the optimum and required levels in the diet was efficient in preventing such disorders.

In conclusion, the combination of Bio-Mos and Se was found to be the most effective in aflatoxin detoxification and improved lymphoid organs physiological function and hematological parameters in poultry received diets containing AFB1 and reduce AFB1 in liver and meat tissue.

Table (1): Efficacy of Bio-Mos and /or Se or HSCAS for detoxification of AFB1 contaminated diets, on relative weight of lymphoid organs (%), HI titer, hematological parameters and AFB1 residue (ppb) at the end of treatment period.

T	Lymphoid organs			Geometric mean of HI titer (Log ₂)		hematological parameters				AFB1 residue (ppb)	
	bursa%	thymus%		20 day	32 day	RBCs(x10 ⁶)	WBCs(x10 ³)	Hb(g/100ml)	PCV%	liver	meat
Control	0.33a	0.41a		3.5	4.7	2.38a	19.91a	13.16a	31.0a	0.00**	0.0**
AF-diet	0.19d	0.20d		3.2	4.3	1.86d	18.02d	9.71e	23.0e	61.00a	6.2a
AF + MoS	0.25c	0.32c		3.4	4.7	2.10c	19.22c	11.62c	27.0d	25.00c	2.7c
AF + Se	0.25c	0.30c		3.5	4.6	2.12c	19.26c	10.43d	27.0d	28.30b	3.2b
AF + Mos + Se	0.29b	0.38b		3.5	4.8	2.25b	19.62b	12.36b	29.0b	10.00d	1.6d
AF + HSCAS	0.25c	0.31c		3.3	4.5	2.06c	19.20c	10.64d	28.0c	27.00b	3.0c
SEM	0.027	0.025		0.004	0.006	0.018	0.013	0.019	0.026	0.015	0.07

** No detecting of aflatoxin B1

Means followed by different letters for each column are significantly different at (P ≤ 0.05).

Table (2): Efficacy of Bio-Mos and /or Se or HSCAS for detoxification of AFB1 contaminated diets, on relative weight of lymphoid organs (%), hematological parameters and residue (ppb) at the end of recovery period.

T	Lymphoid organs		hematological parameters				AFB1 residue (Ppb)	
	bursa%	thymus%	RBCs(x10 ⁶)	WBCs(x10 ³)	Hb(g/100ml)	PCV%	liver	meat
Control	0.29 ^a	0.42 ^a	2.50 ^a	20.21 ^a	14.38 ^a	32.00 ^a	0.0**	0.0**
AF-diet	0.20 ^b	0.26 ^b	2.10 ^c	18.92 ^b	12.62 ^d	28.00 ^c	26.0 ^a	0.0
AF + MOS	0.28 ^a	0.40 ^a	2.30 ^b	20.00 ^a	13.48 ^c	30.00 ^b	6.4 ^c	0.0
AF + Se	0.28 ^a	0.39 ^a	2.36 ^b	20.50 ^a	13.00 ^d	30.00 ^b	8.6 ^b	0.0
AF + Mos + Se	0.29 ^a	0.40 ^a	2.38 ^b	20.16 ^a	14.00 ^b	31.00 ^{ab}	3.0 ^d	0.0
AF + HSCAS	0.26 ^a	0.41 ^a	2.36 ^b	20.00 ^a	13.25 ^c	29.00 ^c	3.0 ^d	0.0
SEM	0.03	0.03	0.02	0.01	0.01	0.03	0.09	0.00

** No detecting of aflatoxin B1

Means followed by different letters for each column are significantly different at (P ≤ 0.05)

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الملخص العربي

قدرة البيوموس، السلينيوم العضوي وسلكات الصوديوم - الكالسيوم - الألومنيوم على إزالة سمية الأفلاتوكسين في الكتاكيت المحلية النامية.
٢-الأعضاء الليمفاوية - الاستجابة المناعية - المتبقى في الأنسجة

رضا على حسن

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تم استخدام عدد ٢٧٠ ككتوت عمر أسبوعين من سلالة السلام وقسمت إلى ٦ معاملات متساوية (٤٥ ككتوت لكل معاملة) تم تغذية كتاكيت المعاملة الأولى على عليقة مقارنة (عليقة الكنترول). غذيت كتاكيت المعاملة الثانية على عليقة الكنترول مضاف إليها ١ مجم من الأفلاتوكسين ب ١ لكل كيلو جرام علف. الكتاكيت في المعاملات ٣ - ٤ - ٥ - ٦ كانت تتناول العلف الملوث بالأفلاتوكسين مضاف إليها ١ جم بيوموس - ٥ ملليجرام سيلنيوم + ٠,٥ ملليجرام سيلنيوم - ٠,٢٥% سليكات الصوديوم والكالسيوم والألومنيوم على التوالي. غذيت كل الكتاكيت على العلائق التجريبية لفترة ٦ أسابيع من عمر ٢ - ٨ أسابيع (فترة المعاملة) ثم بعد

- ذلك غذيت جميعها على العليقة الأساسية (الكنترول) لمدة ٤ أسابيع أخرى من عمر ٨-١٢ أسبوع (فترة إستشفاء) وتتلخص النتائج المتحصل عليها في الآتي :-
- ١- قلت الأوزان النسبية لكل من البرسا والثيموس و قيم تركيز الهوجلوبيين و عدد كرات الدم الحمراء و عدد كرات الدم البيضاء بنسبة معنوية في المجاميع التي تناولت علائق ملوثة بالأفلاتوكسين.
 - ٢- متوسط الاستجابة المناعية لعدوى مرض النيوكاسل لم يتأثر معنوياً بالأفلاتوكسين خلال فترة المعاملة .
 - ٣- كانت قيم المتبقي من الأفلاتوكسين ب ١ في أنسجة الكبد أكبر مما في أنسجة اللحم في كل المجاميع التي تناولت أفلاتوكسين ٠. أدت إضافة الإضافات إلى تقليل المتبقي من الأفلاتوكسين ب ١ في الكبد واللحم بالمقارنة بمعاملة الأفلاتوكسين بدون إضافات وبعد ٤ أسابيع من فترة الإستشفاء قل تركيز الأفلاتوكسين في الكبد واللحم بالمقارنة بفترة المعاملة و كان ذلك أكثر وضوحاً في مجاميع الإضافات وخاصة عند إضافة البيموس و السلينيوم مجتمعة أو معاً .
 - ٤ - ظهرت بشدة الأعراض المرضية (قلة الحيوية - فقدان الشهية للأكل - انكماش الطائر - غشاوة العين - التواء الرجل - عدم اتزان الحركة - تساقط الريش) لمجموعة الأفلاتوكسين وقلت شدة الإصابة في المعاملات التي تناولت بيوموس أو سلنيوم أو سليكات الصوديوم و الكالسيوم والألومنيوم وتضاءلت جداً في المعاملة التي تناولت الأفلاتوكسين مع البيموس مع السلينيوم وقد تلاشت معظم هذه المظاهر خلال الأسبوع الرابع من إزالة العلائق الملوثة وخاصة في المعاملة التي تناولت البيموس مع السلينيوم.
- ومن هذه الدراسة وجد ان إضافة البيموس مع السلينيوم قد قللت التأثير الضار للأفلاتوكسين على الأعضاء اليمفاوية ومكونات الدم و متبقيات الأفلاتوكسين في الكبد و اللحم خلال فترة المعاملة وتساعد على سرعة الإستشفاء خلال فترة الإستشفاء.