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POSSIBILITY OF ALLEVIATING FOODBORNE AFLATOXICOSIS EFFECTS ON PERFORMANCE AND BIOCHEMISTRY OF MALE ALBINO WHITE RATS

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ABSTRACT

One hundred and twenty six albino male rats (average initial body weight of 105 g) were divided into 14 dietary groups (3 replicates X 3 animals/group). They fed for 21 weeks on a control (C) diet, C + 0.5% or 1% tafla, C + 1 or 3% ammonia, C + 3 or 6% H_2O_2, C + 1000 ppb aflatoxin (A), A + tafla or ammonia or H_2O_2 (at the same previous levels). The animals were housed in wire cages and fed *ad libitum* and water was available for 24 hours daily. The obtained results cleared that feeding rats on the aflatoxin(AF)-diet alone depressed the live body weight, daily body weight gain, feed intake, feed efficiency and increased mortality rate. The reduction in live BW was increased with the progress of feeding period, being 28.3% at 13 weeks old and 41.3% at 21 weeks old. Addition of different additives (tafla, ammonia and hydrogen peroxide) to the AF-diet decreased the deleterious effects of aflatoxin on rats performance compared with those fed the AF-diet alone. However, the effective additive to protect against the negative effect of aflatoxin was tafla. Aflatoxin excreted in faeces (as % of intake) was higher in rats fed the AF-diet with different additives than in those fed the AF-diet alone. Rats fed the AF-diet alone had higher level of serum urea-N, uric acid, creatinine, AST, ALT and alkaline phosphatase and lower levels of serum cholesterol, triglycerides, total protein and albumin than those fed the uncontaminated diet (control). Addition of different additives to the AF-diet decreased the deleterious effects of aflatoxin on blood parameters. Tafla was the best additive to protect against the negative effect of aflatoxin on blood. The relative weights of the internal organs (liver, kidneys, spleen, testes, heart and lungs) increased by feeding the contaminated diets. The different dietary additives did not improve the toxic effects of aflatoxin. Therefore it becomes clear to what extent is the seriousness of foodborne aflatoxicosis by animals (and consequently consumers). Even adsorbents (although they slightly reduce absorption) still neither obstacle nor sufficient mean for removing aflatoxins and its toxic effects. Where it is to impose upon the concern of prophylaxis against fungal invasion of feedstuffs and their ingredients to prevent consequently mycotoxin production on feeds.

Keywords: Rats, Aflatoxin, Performance, Feed efficincy, Biochemical parameters.

INTRODUCTION

Aflatoxin is the common name for a group of extremely toxic chemically related compounds produced by the mould *Aspergillus flavus* and *Aspergillus parasiticus* during growth, harvest or storage of different foods and feeds (Wood, 1989). Aflatoxins cause clinical illness and death when consumed in high quantity, at lower levels they reduce the growth rate, feed intake, feed efficiency and suppress immunity of young animals (Harvey *et al.*, 1989 and Abdelhamid *et al.*, 1992). Aflatoxins are the most important

problems and the most toxic mycotoxins to animals and man (Abdelhamid *et al.*, 1990 and 1999 and Abdelhamid and Saleh, 1996). The aflatoxins have been shown to be potent hepatotoxic, carcinogenic, teratogenic and mutagenic, which can lead to genetic damage (Wogan, 1973). Aflatoxicosis is the disease caused by ingestion of aflatoxins. Among aflatoxic compounds, aflatoxin B_1 is the most toxic followed by AFG_1, AFB_2 and AFG_2 in a decreasing order being as a ratio of 8 : 4 : 2 : 1, respectively (Abdelhamid, 1990). Abo-Hagar (2000) showed that the exposure of deliberately infected peanuts and yellow corn to microwaves at low power setting for 3, 6, 9 and 12 min. cause gradual decrease on the level of aflatoxin. The percentage of aflatoxin reduction was dependent on the exposure time. Recent information suggests that clay minerals and aluminosilicates added to mycotoxin-contaminated diets reduce the bioavailability of toxins and their hazardous effects in some animal species. The major advantages of the adsorbent include low cost, safety and easy addition to animal feed. Therefore the efficacy of different adsorbents for the binding of mycotoxins are variable (Huwig *et al.*, 2001). Nowar *et al.* (2001) added bentonite at different levels to the AF-diet and proved that bentonite diminished the toxic effects of aflatoxins on all parameters of rabbit growth performance. The relative weight of liver, carcass and dressing percentages in rabbits fed AF-diet plus bentonite were not statistically different from the control.Abd El-Baki *et al.* (2002) found that addition of tafla to AFB_1 contaminated diet improved the growth performance daily body weight gain, feed consumption and feed efficiency in comparison with the aflatoxin contaminated diet. Yet, Abdelhamid *et al.* (2002-b) showed that the adsorbents (Antitox plus and Fix-a-tox) were less effective in reducing aflatoxin productivity in YES-media for *Aspergilus flavus*, being 62.5 and 47.5%, respectively, but tafla reduced the productivity of aflatoxin by 91.5% comparing with the control.Soliman (1998) showed that the detoxification with 2% and 3% ammonia gas appears to be the most successful process for 4 weeks incubation for all tested ingredient comparing with 14 days incubation period. Soliman *et al.* (2001) studied the prevention of aflatoxicosis in growing rabbits fed aflatoxin contaminated diet using 5% hydrogen peroxide and gamma-radiation. The obtained data indicated that the use of H_2O_2 and gamma-radiation for the destruction of aflatoxin in contaminated diet induces adverse effects in the animals. The aim of the present study was to evaluate the effectiveness of some treatments in reducing the effects of dietary aflatoxins on performance, feed efficiency and biochemical parameters of rats.

MATERIALS AND METHODS

This part of study was carried out after completion of the laboratory experiment (the *in vitro* study by Abdelhamid *et al.,2002-b*). One hundred and twenty six males Albino white rats (average weight of 105 g) were bought from the local market and randomly divided into 14 similar groups (9 animals for each group, i.e. treatment, at 3 replicates, i.e. cages). The animals were housed in wire cages (three animals in each cage) provided with feed and water troughs and fed *ad libitum*. Water was available for 24 hours daily, and cages were kept in a conditioned room. The animals were marked on their

limbs and tails using colored liquid (dye). The feed ingredients and natural additives used in this work were obtained from the local sources. The diet was tested and proved that it was free from aflatoxins. The tested diets were prepared by adding the experimental materials to the basal diet. The medium contained a mixture of aflatoxins B_1, B_2, G_1 and G_2 at a total level of 18 ppm was added to the basal diet to be contained 1000 ppb aflatoxins.

The best three detoxification methods (ammoniation, hydrogen peroxide and tafla) were selected to evaluate their effectiveness(after aerating the ammoniated diet)in reducing the negative effects of dietary aflatoxin on performance and biochemical parameters of rats. All materials and methods(including statistical analysis) used herein are the same as described in Abdelhamid *et al.*(2004-a). Where a basal diet - prepared from locally purchased ingredients according to Ahmed (1976) contained 46% crushed wheat, 40% shredded barley, 9% fish meal, 3% dried milk, 1% yeast and 1% minerals and vitamins mixture - was offered to rats for 21 weeks. The experimented diets used in this study were as follows

1 - Control diet without any addition(C).	8-C-contaminated with 1000 ppb (AF).
2 - C + 0.5% tafla.	9-AF + 0.5% tafla.
3 - C + 1% tafla.	10 – AF + 1% tafla..
4 - C + 1% ammonia solution.	11 - AF + 1% ammonia solution..
5 - C + 3% ammonia solution.	12 – AF +3% ammonia solution.
6 - C + 3% hydrogen peroxide solution.	13–AF+3% hydrogen peroxide solution.
7- C + 6% hydrogen peroxide solution.	14-AF+6% hydrogen peroxide solution.

Biweekly live body weight for the individuals and feed intake were measured. During the period of the experiment. The rats were inspected daily for any clinical symptoms and mortality was recorded. Organs weight was recorded at slaughtering or autopsy. Aflatoxin concentration was determined using immunoaffinity column coupled with solution fluorometry or liquid chromatography postcolumn derivatization according to Truckess *et al.* (1991). At the end of the experiment, blood samples were collected from three rats from each group, from the orbital plexus vein of the rat eyes using a heparinized capillary tubes. The blood samples were allowed to stand at room temperature (20 – 25°C) for 10 min for coagulation then centrifugated at 4000 rpm for 3 min and the colourless upper layer was carefully separated and transferred into sterilized epindorff tubes and kept frozen until analysis. Serum concentrations of total protein (Gornall *et al.*, 1949) and albumin(Doumas *et al.* ,1971) and transaminases (AST and ALT, Cambell and Hornby ,1975) and alkaline phosphatase (Rick, 1990) activity besides cholesterol (Allain *et al.*, 1974), creatinine (Bartels and Boehmer, 1971), urea (Fawcett and Scott, 1960), uric acid (Gupta and Bhargava, 1985), and triglycerides (Fossati and Principle, 1982) levels were determined.

RESULTS AND DISCUSSION

Growth performance:

Body weight (BW):

Data in Table (1) show a reduction in average live body weight (BW) of rats fed a diet contaminated with 1000 ppb aflatoxins mixture when compared with those fed the uncontaminated diet (control).

Table (1): Average biweekly body weights (g) of the experimental rats in different experimental treatments.

Weeks	C	C + 0.5% Tafla	C + 1% Tafla	C + 1% NH_3	C + 3% NH_3	C + 3% H_2O_2	C + 6% H_2O_2	A 1000 ppb	A + 0.5% Tafla	A + 1% Tafla	A + 1% NH_3	A + 3% NH_3	A + 3% H_2O_2	A + 6% H_2O_2
W_1	105.2 ± 1.2	104.1 ± 2.4	107.3 ± 1.9	104.8 ± 4.5	105.4 ± 3.2	104.0 ± 3.3	105.2 ± 1.4	103.1 ± 4.1	105.4 ± 4.8	106.1 ± 2.1	103.6 ± 3.1	106.8 ± 0.9	105.0 ± 7.7	106.1 ± 2.1
W_3	144.0 ± 2.5	148.9 ± 4.4	147.4 ± 8.1	144.7 ± 1.9	147.7 ± 2.8	147.1 ± 4.2	147.3 ± 4.6	143.2 ± 2.3	152.7 ± 3.2	148.1 ± 4.1	147.1 ± 4.2	151.8 ± 2.7	150.6 ± 5.3	150.3 ± 6.1
W_5	203.0 ± 1.2	204.1 ± 7.9	204.6 ± 4.1	200.2 ± 2.1	200.6 ± 3.6	198.9 ± 4.8	192.8 ± 5.3	168.7 ± 2.8	191.3 ± 6.2	186.9 ± 8.1	174.7 ± 6.1	187.9 ± 9.4	180.9 ± 11.9	178.4 ± 12.3
W_7	217.1 ± 3.4	216.0 ± 6.7	215.8 ± 9.8	214.0 ± 2.2	210.8 ± 2.2	205.8 ± 4.7	200.2 ± 8.1	181.9 ± 2.6	199.8 ± 6.7	189.9 ± 6.1	187.6 ± 4.1	196.1 ± 7.6	190.1 ± 7.7	185.0 ± 9.2
W_9	234.0 ± 1.3	229.8 ± 5.5	231.0 ± 8.9	229.6 ± 1.1	225.6 ± 3.5	220.0 ± 4.5	220.4 ± 10.1	184.4 ± 2.2	217.6 ± 7.4	201.6 ± 6.5	198.3 ± 7.2	214.2 ± 9.6	206.9 ± 8.2	198.7 ± 9.4
W_{11}	255.9 ± 4.7	249.6 ± 5.9	251.4 ± 5.6	249.2 ± 4.6	246.4 ± 3.8	244.1 ± 3.7	244.0 ± 8.4	188.0 ± 1.5	220.3 ± 6.6	214.1 ± 3.5	210.8 ± 8.6	219.7 ± 7.5	215.6 ± 12.1	207.2 ± 10.3
W_{13}	263.4 ± 4.3	254.1 ± 6.4	251.4 ± 6.8	252.6 ± 3	252.6 ± 3.7	248.3 ± 3.7	249.7 ± 8.5	188.9 ± 3.9	219.6 ± 5.1	214.0 ± 3.3	206.7 ± 7.5	216.8 ± 10.1	213.3 14.7	206.6 ± 7.5
W_{15}	267.1 ± 2.4	258.1 ± 5.4	257.8 ± 5.5	258.6 ± 1.7	257.9 ± 3.9	254.0 ± 5.9	250.1 ± 9.8	184.7 ± 3.9	210.8 ± 8.1	203.4 ± 1.8	199.4 ± 7.4	209.1 ± 11.3	206.8 ± 15.8	198.4 ± 9.8
W_{17}	275.2 ± 2.5	267.8 ± 4.3	267.1 ± 4.8	268.9 ± 2.1	267.8 ± 4.5	265.0 ± 1.9	258.3 ± 9.8	177.3 ± 1.3	200.6 ± 6.5	199.7 ± 5.8	188.0 ± 1.7	195.2 ± 6.1	195.0 ± 9.6	181.3 ± 8.7
W_{19}	280.1 ± 2.3	275.0 ± 3.4	273.3 ± 4.4	273.9 ± 1.5	272.4 ± 4.3	268.8 ± 2.1	263.3 ± 7.3	168.8 ± 3.1	195.8 ± 7.8	184.0 ± 2.6	187.6 ± 6.3	191.0 ± 8	191.6 ± 12.7	194.8 ± 2.4
W_{21}	281.1 ± 1.5	278.3 ± 8.6	279.4 ± 3.9	278.3 ± 1.9	276.7 ± 4.8	274.4 ± 4.9	270.0 ± 4.8	165.0 ± 1.4	190.6 ± 7.1	191.9 ± 2.9	184.4 ± 9.1	189.2 ± 3.6	181.3 ± 12.1	181.1 ± 2.8

C = control A = aflatoxin

a – e means in the same row with different superscripts significantly ($p \leq 0.05$) differ

The reduction in live BW was increased with the progress of feeding period, being 28.3% after 13 weeks and 41.3% after 21 weeks. There were no significant differences in body weight between rats fed the control diet and the control diet with different experimental additives (tafla, ammonia and hydrogen peroxide). The decrease in BW may be attributed to a reduction in daily feed intake. In addition, aflatoxins impaire nitrogen and energy utilization of the ingested diet through the adverse affects of aflatoxin on the liver, a center of body metabolism (Reddy *et al.*, 1991). Also, aflatoxins can bind with DNA and RNA and prevent the protein synthesis in the body (Pier, 1992). Addition of different additives (tafla, ammonia and H_2O_2) to AF-diet significantly decreased the deleterious effects of aflatoxins on body weight, where BW was lower in rats fed AF-diet than those fed the AF-diet with different additives (tafla, ammonia and hydrogen peroxide). However, the effective additive to protect against the negative effects of aflatoxin on body weight were tafla. This result indicates a marked reduction in aflatoxins toxicity by adding tafla to the aflatoxins – contaminated diet. The basic mechanism of tafla and other adsorbents (binding agents) in preventing the aflatoxin toxicity, appears to involve sequestration of aflatoxin in the gastrointestinal tract and chemisorption to the adsorbent, which reduces the bioavailability of aflatoxins (Kubena *et al.*, 1990-a & b).

Body weight gain (BWG):

Table (2) shows average body weight gain (g) of the experimental rats in different experimental treatments. It could be seen that there were no significant differences between the control and the control with different additives on average daily body weight gain. A reduction in average body weight gain (BWG) of rats fed the diet contaminated with 1000 ppb aflatoxins mixture was noticed when compared with those fed the uncontaminated diet (control). Addition of different additives to AF-diet alleviated the deleterious effects of aflatoxin on daily BWG. However, tafla was the best additive to protect against the toxic effect of aflatoxin.

Feed intake:

Table (3) shows a reduction in average of daily feed intake (DFI) of rats fed the contaminated diet when compared with those fed the uncontaminated diet (control). Average DFI of rats fed the aflatoxins diet alone recorded 57.9% of the control. Feed intake may have been depressed due to decreased palatability of the AF-diet. Average DFI of the rats fed AF-diet with tafla recorded 76.1% of the control while, 67.02, 68.1, 68.1 and 67.6% were for calculated those fed the AF-diet plus 1%, 3% ammonia and 3, 6% hydrogen peroxide respectively.

Feed efficiency:

Table (4) shows that feed efficiency (g gain/g feed) had the same trend of body weight (BW), daily bodyweight gain (daily BWG) and daily feed intake (DFI). Accumulative feed efficiency for the rats group fed on the AF-diet was lower when compared with, those fed the uncontaminated diet (control).

Table (2): Average bi-weekly body gain (g) of the experimental rats in different experimental treatments.

Weeks	C	C + 0.5% Tafla	C + 1% Tafla	C + 1% NH_3	C + 3% NH_3	C + 3% H_2O_2	C + 6% H_2O_2	A 1000 ppb	A + 0.5% Tafla	A + 1% Tafla	A + 1% NH_3	A + 3% NH_3	A + 3% H_2O_2	A + 6% H_2O_2
1 – 3	38.8	44.8	40 1	39.9	42.2	43.1	42.1	40.1	47.2	42.0	43.6	45.0	45.6	44.2
3 – 5	59.0	55.2	57.1	55.6	52.9	51.8	45.5	25.5	38.7	38.8	27.6	36.1	30.3	26.1
5 – 7	14.1	11.9	11 2	13.8	10.2	6.9	7.4	13.2	8.5	3.0	12.0	8.2	9.2	8.6
7 – 9	16.9	13 8	15.2	15.6	14.8	14.2	20.2	2.6	17.8	11.7	10.8	18.1	16.8	13.7
9 – 11	21.9	19.8	20.4	19.7	20.9	24.1	23.6	3.6	2.8	12.6	12.5	5.5	8.7	8.6
11 – 13	7.6	4.6	0	3.3	6.1	4.2	5.7	0.9	-0.8	-0.1	-4.1	-2.9	-2.2	-0.7
13 – 15	3.7	4.0	6.3	6.0	5.3	5.7	0.4	-4.2	-8.8	-10.6	-7.2	-7.7	-6.8	-8.1
15 – 17	8.1	9 7	9.3	10.3	9.9	11.0	8.2	-7.4	-10.2	-3.8	-11.4	-13.9	-11.6	-17.1
17 – 19	4 9	7.2	6.2	5.0	4.7	3.8	5.0	-8.5	-4.8	-15.7	-0.4	-4.2	-3.4	+13.5
19 - 21	1.0	3.3	6.1	4.4	4.2	5.7	6.7	-3.8	-5.2	+7.9	-3.2	-1.8	-10.3	-12.8
Total	176	174.3	171.9	173.6	171.2	170.5	164.8	62.0	85.2	85.8	81.1	82.4	76.3	76.0
Daily	1.26	1.24	1.23	1.24	1.22	1.22	1.18	0.44	0.61	0.61	0.58	0.59	0.55	0.54

C = control A = aflatoxin

Table (3): Average bi-weekly feed consumption (g) of the experimental rats in different experimental treatments.

Weeks	C	C + 0.5% Tafla	C + 1% Tafla	C + 1% NH_3	C + 3% NH_3	C + 3% H_2O_2	C + 6% H_2O_2	A 1000 ppb	A + 0.5% Tafla	A + 1% Tafla	A + 1% NH_3	A + 3% NH_3	A + 3% H_2O_2	A + 6% H_2O_2
1 – 3	152.5	154.5	154.6	149.3	148.6	153.3	153.5	136.0	147.7	144.1	140.3	139.2	143.3	144.6
3 – 5	179.3	179.7	179.6	171.0	170.4	175.3	176.1	156.0	172.4	167.6	154.6	152.3	152.6	155.3
5 – 7	213.1	212.3	213.3	206.6	206.4	211.4	205.3	175.0	193.3	185.7	165.7	163.5	165.4	167.2
7 – 9	243.1	240.3	240.7	232.7	227.3	230.6	230.3	187.0	203.3	207.6	183.5	180.4	181.2	183.8
9 – 11	271 1	271.0	270.7	258.3	255.3	245.6	240.7	182.0	215.8	210.9	195.6	192.7	194.7	193.3
11 – 13	283 3	297.3	295.1	275.6	274.2	275.2	265.3	194.0	230.3	221.6	198.3	202.3	207.6	203.3
13 – 15	291.2	290.6	308.2	282.3	287.3	277.3	275.4	192.0	210.6	215.5	185.6	185.4	188.3	190.6
15 – 17	320 0	315.3	304.3	283.6	292.5	280.3	285.7	147.5	210.3	215.2	175.3	195.3	175.2	165.3
17 – 19	325.4	351.0	326.4	305.2	305.5	315.4	312.2	92.6	210.6	220.6	175.6	185.7	195.3	185.2
19 - 21	351.1	345.0	345.1	325.3	323.6	335.1	340.1	62.8	205.4	210.6	195.1	195.3	190.6	185.3
Total	2630.1	2641.1	2638.0	2489.9	2491.1	2499.5	2484.6	1524.9	1999.7	1999.4	1769.6	1792.1	1794.2	1773.9
Daily	18.79	18.87	18.84	17.79	17.79	17.85	17.75	10.89	14.28	14.28	12.64	12.80	12.82	12.67

C = control A = aflatoxin

Addition of different additives slightly alleviated the negative effect of aflatoxin on feed efficiency. There were no significant differences between the control and the control with different additives on feed efficiency. Each g consumed from the aflatoxin – contaminated diet alone reduced the body weight gain (BWG) by 0.02 g at the 15th week and by 0.06 g at the 21st week.

Collective performance parameters:

Data in Table (5) show a reduction in average live body weight (BW), body weight gain (BWG), daily feed intake (DFI) and feed efficiency of rats fed the diet contaminated with the mixture of aflatoxins when compared with those fed the uncontaminated diet (control). The obtained results are in agreement with those found by Schell *et al.* (1993) who recorded a significant ($P < 0.01$) decrease in daily feed intake, daily body weight gain and feed conversion efficiency in weanling pigs fed for 6 weeks a diet contaminated with 922 ppb aflatoxin B_1. Also, Nowar *et al.* (1996 and 2001) found that feeding rabbits an AF-diet depressed the live BW, daily BWG and feed intake and increased mortality rate. Moreover, Abdelhamid *et al.* (2002-c) found that adding aflatoxin at a level of 250 ppb AFB_1 led to slight decreases in BW and BWG but feed intake and feed efficiency did not affect, this may be attributed to the used low dose. Recently, Ragab (2003) showed that the aflatoxic diet led to decrease average live body weight and body weight gain of the rabbits fed a diet contaminated with aflatoxin. Abdelhamid *et al.* (2004-b) found, also, that the aflatoxic diet led to reduction in final body weight, body gain, daily gain, specific growth rate, survival rate and feed and nutrients utilization, but increase mortality rate of Nile tilapia fish. Addition of different additives significantly decreased the deleterious effects of aflatoxins on rats performance, where averages of BW, BWG, DFI and feed efficiency were lower in rats fed the AF-diet alone than those fed the AF-diet plus different additives. The best feed additives led to significant overcoming these aflatoxic symptoms was tafla. In this respect, Nowar *et al.* (1996 & 2001) and Abd El-Baki *et al.* (2002) showed that addition of tafla and bentonite at different levels to AF-diet diminished the toxic effects of aflatoxin on all parameters of rabbits growth performance. Moreover, the best feed additives let to significant overcoming aflatoxic symptoms (reduction in BW, AWG, ADG and SGR) in Nile tilapia fish were eggshell and clay (Abdelhamid *et al.*, 2004-b). Yet, Abdelhamid *et al.* (2002-c) found that dietary inclusion of tafla and aluminosilicate were not sufficient means for removing AFB_1 and its toxic effects on rabbits. Also, Abdelhamid *et al.* (2004-a) showed that tafla, ammonia and hydrogen peroxide additions to the aflatoxic diet did not overcome or ameliorate the toxic effect of aflatoxin on the histological finding of rat's liver and Kidney. Moreover, Soliman *et al.* (2001) reported that the use of H_2O_2 for the destruction of aflatoxins in contaminated diet induces adverse effects in the animals. Yet, Frayssinet and Frayssinet (1990) mentioned that ammonia treatment is a practical solution to the problem of the carcinogenic potency of contaminated oil cakes. Also, Soliman (1998) concluded that detoxification of feedstuffs and improving its feeding quality and consequently improving small ruminants performance could be occurred by using 2 – 3% ammonia gas or 6% hydrogen peroxide.

Table (4): Average bi-weekly feed efficiency (g.gain/g.feed) of the experimental rats in different experimental treatments.

Weeks	C	C + 0.5% Tafla	C + 1% Tafla	C + 1% NH_3	C + 3% NH_3	C + 3% H_2O_2	C + 6% H_2O_2	A 1000 ppb	A + 0.5% Tafla	A + 1% Tafla	A + 1% NH_3	A + 3% NH_3	A + 3% H_2O_2	A + 6% H_2O_2
1 – 3	0.25	0.29	0.26	0.27	0.28	0.28	0.27	0.29	0.32	0.29	0.31	0.32	0.32	0.31
3 – 5	0.33	0.31	0.32	0.33	0.31	0.30	0.26	0.16	0.22	0.23	0.18	0.24	0.20	0.17
5 – 7	0.07	0.06	0.05	0.07	0.05	0.03	0.04	0.08	0.04	0.02	0.08	0.05	0.06	0.05
7 – 9	0.07	0.06	0.06	0.07	0.07	0.06	0.09	0.01	0.09	0.06	0.06	0.10	0.09	0.07
9 – 11	0.08	0.07	0.08	0.08	0.08	0.09	0.09	0.02	0.01	0.06	0.06	0.03	0.04	0.04
11 – 13	0.03	0.02	0.0	0.01	0.02	0.02	0.02	0.004	-0.004	-0.001	-0.02	-0.01	-0.01	-0.003
13 – 15	0.01	0.01	0.02	0.02	0.02	0.02	0.001	-0.02	-0.04	-0.05	-0.04	-0.04	-0.04	-0.04
15 – 17	0.03	0.03	0.03	0.04	0.03	0.04	0.03	-0.05	-0.05	-0.02	-0.07	-0.07	-0.07	-0.1
17 – 19	0.02	0.02	0.02	0.02	0.02	0.01	0.02	-0.09	-0.02	-0.07	-0.002	-0.02	-0.02	0.07
19 - 21	0.003	0.01	0.02	0.01	0.01	0.02	0.02	-0.06	-0.03	0.04	-0.02	-0.01	-0.05	-0.07
Accum-ulative feed effici- ency	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.04	0.043	0.043	0.045	0.046	0.043	0.043

C = control A = aflatoxin

Table (5): Effect of different experimental treatments on the total performance parameters on the experimental rats.

Items	C	C + 0.5% Tafla	C + 1% Tafla	C + 1% NH_3	C + 3% NH_3	C + 3% H_2O_2	C + 6% H_2O_2	A 1000 ppb	A + 0.5% Tafla	A + 1% Tafla	A + 1% NH_3	A + 3% NH_3	A + 3% H_2O_2	A + 6% H_2O_2
Initial body weight (g)	105.2	104.1	107.3	104.8	105.4	104.0	105.2	103.1	105.4	106.1	103.6	106.8	105.0	106.1
Finial body weight (g)	281.1	278.3	279.4	278.3	265.7	274.4	270.0	165.0	190.6	191.9	184.4	189.2	181.3	181.9
Body gain (g)														
Total	175.9	174.2	172.1	173.5	171.3	170.4	164.8	61.9	85.2	85.8	80.8	82.4	76.3	75.8
Daily	1.26	1.24	1.23	1.24	1.22	1.22	1.18	0.44	0.61	0.61	0.58	0.59	0.55	0.54
Feed intake (g/ animal)														
Total	2630.1	2641.1	2638.0	2489.9	2491.1	2499.5	2484.6	1524.9	1999.7	1999.4	1769.6	1792.1	1794.2	1773.9
Daily	18.79	18.87	18.84	17.79	17.79	17.85	17.75	10.89	14.28	14.28	12.64	12.80	12.82	12.67
Feed effic-iency	0.07	0.07	0.07	0.07	0.07	0.07	0.07	0.04	0.043	0.043	0.045	0.046	0.043	0.043

C = control A = aflatoxin

Absolute weights of internal organs:

As shown in Table (6), rats fed the aflatoxin contaminated diet had slightly higher absolute weights of liver, spleen and kidneys than those fed uncontaminated diet (control). The differences in absolute weights of rat organs fed the aflatoxin contaminated diet, the contaminated diet with additives (tafla, ammonia and hydrogen peroxide) and the control were not significant. These results disagreed with those of Nowar *et al.* (1981) who reported that the weights of internal organs and their weights as percentages of the body weight in male and female rats ingested aflatoxins mixture (B_1 + G_1) were heavier than those of untreated ones. However, Abdelhamid *et al.* (2002-a) reported that, there were no changes among treatments for absolute weights for liver and kidneys. On contrast, Abd El-Mageed (1987) show that aflatoxins, aflatoxin with Vit. C and aflatoxin with soil in male rats significantly ($P < 0.05$) decreased the absolute weight of liver, kidneys, spleen, testes and lungs. Rabbits fed aflatoxin contaminated diet had a lower ($P < 0.01$) absolute weights of lungs and kidneys than those fed uncontaminated diet (control) Shehata (2001). However, Singh *et al.* (1998) found that dietary aflatoxin led to enlargement of liver and congestion of kidneys and lungs of rabbits. Some investigators (Huff *et al.*, 1986-a & b) have attributed the increase in the weight of liver due to aflatoxins to an accumulation of lipids in the liver, which provide characteristics of hepatomegaly, friable and fatty liver associated with aflatoxicosis.

Clinical Sings and post-mortem examination:

The aflatoxicated male rats before death exhibited loss of appetite, weakness and emaciation. These rats after death showed hemorrhage in the abdominal cavity, the liver was pale in appearance, friable and gall bladder was enlarged and filled with bile. The lungs and kidneys showed congestion and urinary bladder was enlarged and distended with urine. Similar clinico-anatomically and post – mortem finding were reported by many authors (Abdelhamid *et al.* 1995-a & b; Nowar *et al.* 1996 and Ragab 2003). However, Bains (2002) cited that the mechanism of mycotoxin induced tissue and organ damage is the result of complex biochemical processes that may react with other enzyme and cofactors, such as vitamins which interrupt cell membrane transport, interrupt cellular metabolism, block metabolic pathway – lesions and interact with DNA and RNA protein synthesis. He added that mycotoxins lead to depressed appetite, poor growth rate and feed conversion, immuno – suppression, diarrhea, rickets, fatty infiltration in both of liver and kidneys, and bile duct proliferation.

Mortality rate:

Data presented in Table (7) show that mortality rate (%) reached to 56% in rats fed the AF-diet at the end of feeding period. Addition of different additives decreased the mortality (%) in rats fed the AF-diet plus additives than those fed the AF-diet alone. Mortality rate (%) of rats fed the AF-diet plus ammonia and H_2O_2 were 22, 22, 11 and 22% at levels of 1, 3, 3 and 6%, respectively. No mortality was recorded in rats fed the AF-diet plus tafla.The obtained results are in agreement with those found by Shehata (2001) on tafla.

Table (6): Effect of dietary treatments on absolute weights (g) of different organs of the experimental rats.

Dietary treatments	Liver	Kidneys	Spleen	Testes	Heart	Lungs
Control (basal)	6.63 ± 0.09	1.30 ± 0.06	0.61 ± 0.12	2.20 ± 0.06	0.84 ± 0.02	2.00 ± 0.06
Basal + ½% tafla	6.40 ± 0.21	1.30 ± 0.12	0.62 ± 0.02	2.13 ± 0.03	0.84 ± 0.01	1.97 ± 0.06
Basal + 1% tafla	6.47 ± 0.20	1.33 ± 0.06	0.60 ± 0.01	2.10 ± 0.05	0.83 ± 0.01	2.00 ± 0.12
Basal+1% ammonia	6.37 ± 0.09	1.30 ± 0.06	0.62 ± 0.01	2.10 ± 0.03	0.83 ± 0.01	2.00 ± 0.06
Basal+3% ammonia	6.53 ± 0.21	1.30 ± 0.06	0.62 ± 0.01	2.10 ± 0.03	0.83 ± 0.01	2.03 ± 0.12
Basal + 3% H_2O_2	6.50 ± 0.23	1.30 ± 0.07	0.62 ± 0.01	2.11 ± 0.05	0.83 ± 0.01	2.00 ± 0.17
Basal + 6% H_2O_2	6.67 ± 0.18	1.33 ± 0.03	0.62 ± 0.02	2.17 ± 0.03	0.83 ± 0.01	2.00 ± 0.06
Aflatoxin (Afla.)	7.27 ± 0.09	1.37 ± 0.03	0.63 ± 0.01	2.17 ± 0.06	0.84 ± 0.01	1.97 ± 0.06
Afla. + ½% tafla	6.43 ± 0.24	1.27 ± 0.06	0.62 ± 0.02	2.11 ± 0.05	0.83 ± 0.01	1.97 ± 0.06
Afla. + 1% tafla	6.37 ± 0.09	1.30 ± 0.06	0.60 ± 0.02	2.17 ± 0.03	0.83 ± 0.01	1.97 ± 0.06
Afla. +1% ammonia	6.27 ± 0.17	1.30 ± 0.03	0.31 ± 0.01	2.12 ± 0.04	0.83 ± 0.01	1.97 ± 0.12
Afla. +3% ammonia	6.50 ± 0.15	1.37 ± 0.03	0.60 ± 0.01	2.13 ± 0.03	0.83 ± 0.01	1.97 ± 0.06
Afla. + 3% H_2O_2	6.53 ± 0.13	1.27 ± 0.06	0.59 ± 0.01	2.13 ± 0.03	0.83 ± 0.01	1.97 ± 0.12
Afla. + 6% H_2O_2	6.60 ± 0.36	1.27 ± 0.03	0.59 ± 0.01	2.10 ± 0.05	0.83 ± 0.01	1.97 ± 0.06

They found that addition of tafla or bentonite to diets naturally contaminated with 860 ppb aflatoxins (B_1 + G_1) prolonged the survival period of rabbits when compared with those fed the AF-diet alone. Therefore, it could be concluded that tafla and other adsorbents play a definite role in binding aflatoxin and reducing its toxicity.

Aflatoxins excretion in faeces:

Data in Table (8) show that aflatoxin excreted in faces (as % of intake) was higher in rats fed the AF-diet with different additives than in those fed the AF-diet alone. Faecal aflatoxin of rats fed the AF-diet alone recorded 3.63% versus 24.7, 24.4, 6.5, 5.5, 4.2 and 3.9% for those fed the AF-diet with 0.5, 1% tafla, 1, 3% ammonia and 3, 6% hydrogen peroxide, respectively. These data indicate that tafla has a high affinity adsorbability for the aflatoxin when added to the infected diet. These results agree with those found by Nowar *et al.* (1996) who reported that faecal aflatoxin of rabbits fed AF-diet without tafla recorded 7.5% versus 72.7, 58.1 and 50% for those fed the AF-diet with 1, 2 and 3% tafla, respectively. The protective effects provided by tafla may be due to the absorption or adsorption of aflatoxin on the surface of the tafla layers, consequently prevent the bioavailability of aflatoxins. This is supported by the larger quantities of aflatoxins excreted in the faces when tafla is added to AF-diet.

Blood serum parameters:

As shown in Table (9), rats fed the AF-diet alone had higher levels of serum urea-N, uric acid, creatinine, AST, ALT and alkaline phosphatase and lower levels of serum total protein, cholesterol and triglycerides than those fed the uncontaminated diet. These findings can reflect well the signs of aflatoxicosis as described by some investigators (Lindemann *et al.*, 1993 and Nowar *et al.*, 1996 and 2001). The elevated levels of AST and ALT and decreased level of cholesterol indicate to a hepatic damage, while the increased concentrations of urea-N, uric acid and creatinine indicate to kidney injury caused by aflatoxins toxicity. The lower level of total protein and higher level of urea-N indicate a higher level of protein catabolism with a concurrent lowering of protein synthesis. Fernandez *et al.* (1994) reported that liver is the main target of aflatoxicosis, which causes important changes in hepatic metabolism, affecting protein, lipids and enzyme synthesis. This finding was supported by the obtained lower live body weight and daily body gain of rats fed the aflatoxins contaminated diet. Generally the obtain results agreed with those of Abdel-Wahhab *et al.* (1999) on rats, and Abdelhamid *et al.* (2002-c) on rabbits. However, addition of different additives to AF-diet decreased the deleterious effects of aflatoxins on blood parameters. However, the effective additive to protect against the negative effects of aflatoxin on blood parameters was tafla. The obtained results agreed with those of Abdelhamid *et al.* (2002-c) who reported that adding clay to AF-diet minimized the negative effects of aflatoxin on blood parameters. On the other hand, Soliman (1998) found that the used H_2O_2 and NH_3 for the destruction of aflatoxins in contaminated diet protected against the negative effects of aflatoxins in blood parameters.

Table (7): Number of rats/treatment throughout the experimental period and mortality rate.

Weeks	C	C + 0.5% Tafla	C + 1% Tafla	C + 1% NH_3	C + 3% NH_3	C + 3% H_2O_2	C + 6% H_2O_2	A 1000 ppb	A + 0.5% Tafla	A + 1% Tafla	A + 1% NH_3	A + 3% NH_3	A + 3% H_2O_2	A + 6% H_2O_2
1	9	9	9	9	9	9	9	9	9	9	9	9	9	9
2	9	9	9	9	9	9	9	9	9	9	9	9	9	9
3	9	9	9	9	9	9	9	9	9	9	9	9	9	9
4	9	9	9	9	9	9	9	9	9	9	9	9	9	9
5	9	9	9	9	9	9	9	9	9	9	9	9	9	9
6	9	9	9	9	9	9	9	9	9	9	9	9	9	9
7	9	9	9	9	9	9	9	9	9	9	9	9	9	9
8	9	9	9	9	9	9	9	9	9	9	9	9	9	9
9	9	9	9	9	9	9	9	9	9	9	9	9	9	9
10	9	9	9	9	9	9	9	9	9	9	9	9	9	9
11	9	9	9	9	9	9	9	9	9	9	9	9	9	9
12	9	9	9	9	9	9	9	9	9	9	9	9	9	9
13	9	9	9	9	9	9	9	9	9	9	9	9	9	9
14	9	9	9	9	9	9	9	9	9	9	9	9	9	9
15	9	9	9	9	9	9	9	9	9	9	9	9	9	9
16	9	9	9	9	9	9	9	8	9	9	9	9	9	8
17	9	9	9	9	9	9	9	7	9	9	9	9	9	8
18	9	9	9	9	9	9	9	6	9	9	9	9	9	8
19	9	9	9	9	9	9	9	5	9	9	9	9	9	8
20	9	9	9	9	9	9	9	4	9	9	7	8	8	7
21	9	9	9	9	9	9	9	4	9	9	7	7	8	7
Mort-ality %	-	-	-	-	-	-	-	55.6	-	-	22.2	22.2	11.1	22.2

C = control A = aflatoxin

Table (8): Effect of different experimental treatments on aflatoxin (AF) excretion in faeces of the experimental rats.

Items	Unconta-minated diet	AF-diet	AF + 0.5% tafla	AF + 1% tafla	AF + 1% ammonia	AF + 3% ammonia	AF + 3% H_2O_2	AF + 6% H_2O_2
Aflatoxin intake								
(μg/head/day)	-	10.9	14.3	14.3	12.6	12.8	12.8	12.7
Fecal aflatoxin								
(μg/head/day)	-	0.396	3.53	3.34	0.815	0.703	0.532	0.497
As % of intake	-	3.63	24.69	23.35	6.47	5.49	4.16	3.88

Table (9): Average blood biochemical parameters of the experimental rats in different experimental treatments.

Items	C	C + 0.5% Tafla	C + 1% Tafla	C + 1% ammonia	C + 3% ammonia	C + 3% H_2O_2	C + 6% H_2O_2	A	A + 0.5% Tafla	A + 1% Tafla	A + 1% NH_3	A + 3% NH_3	A + 3% H_2O_2	A + 6% H_2O_2
Total protein (g/dl)	a 6.8 ± 0.1	ab 6.5 ± 0.03	ab 6.6 ± 0.03	a 6.8 ± 0.1	a 6 6 ± 0 1	ab 6.7 ± 0 1	ab 6.6 ± 0.03	e 4.8 ± 0.1	d 5.3 ±0.1	d 5.3 ± 0.1	dc 5.2 ± 0 1	Dc 5.2 ± 0 03	c 5.07 ± 0.1	dc 5.1 ± 0.1
Albumin (g/dl)	ab 4.5 ± 0.1	b 4 3 ± 0 1	ab 4.4 ± 0.1	a 4.5 ± 0.03	a 4.5 ± 0.1	ab 4 4 ± 0.1	ab 4.4 ± 0.1	c 3.4 ± 0.03	c 3.5 ± 0.03	c 3.5 ± 0.1	c 3.4 ± 0.1	C 3.5 ± 0.1	c 3.5 ± 0.03	c 3.5 ± 0.03
Globulin (g/dl)	a 2 3 ± 0.03	a 2 2 ± 0. 1	a 2.2 ± 0.03	a 2.3 ± 0.1	a 2.3 ± 0.1	a 2.3 ± 0.03	a 2.2 ± 0.03	c 1.4 ± 0.03	b 1.8 ± 0.03	b 1.8 ± 0.1	b 1.8 ± 0.1	b 1.7 ± 0.03	b 1.6 ± 0.1	b 1.6 ± 0.1
Got (U/L)	e 74 ± 0.9	e 74 ± 0.6	e 75 ± 0.6	e 78 ± 0.6	e 79 ± 0.6	e 76 ± 0.6	e 75 ± 0.6	a 143 ± 1.7	d 110 ± 4.4	cd 115± 2.9	b 130 ± 1.2	B 131 ± 1.9	c 120 ± 2.9	c 120 ± 5.8
GPT (U/L)	d 36 ± 0.6	d 35 ± 06	d 36 ± 1.2	d 38 ± 0.6	d 38 ± 0 6	d 35 ± 0.6	d 35 ± 0.9	c 73 ± 17	c 78 ± 0.6	c 86 ± 3.1	b 86 ± 3.1	Ab 90 ± 2.7	a 93 ± 4.4	ab 91 ± 1.9
Alkaline phospha-tes (U/L)	d 179 ± 4.9	d 178 ± 1.7	d 178 ± 4.4	d 177 ± 4.4	d 177 ± 1.7	d 181± 5.4	d 180 ± 2.9	a 315 ± 0.9	c 276 ± 1.9	c 280 ± 2.9	b 295 ± 2.9	B 298 ± 4.4	b 305 ± 1.5	b 300 ± 2.9
Total cholesterol (mg/dl)	a 80 ± 2.7	a 82 ± 2.2	a 82 ± 1.2	a 80 ± 2 3	a 81 ± 2.9	a 81± 0.6	a 81± 1.2	d 54 ± 1.9	b 64 ± 0.6	B 62 ± 0.3	b 59 ± 0.6	b 59 ± 0.9	b 58 ± 0.9	b 59 ± 0.9
Triglyce-rides (mg/dl)	a 92 ± 0.9	a 93 ± 0.6	a 92 ± 0.6	a 92 ± 0.6	a 92 ± 1.2	a 91 ± 1	a 92 ± 0.6	d 63 ± 0.3	b 71 ± 0.6	B 71 ± 0.3	c 67 ± 1 5	C 67 ± 0.9	d 64 ± 0.6	d 64 ± 0.3
Creatinine (mg/dl)	d 0 44 ± 0.01	d 0.45 ± 0 01	d 0.44 ± 0 01	d 0.45 ± 0.01	d 0.45 ± 0 01	d 0 43 ± 0 01	d 0 43 ± 0 01	a 1.22 ± 0.1	c 0.73 ± 0.01	C 0.75 ± 0.03	b 0 92 ± 0 02	B 0.90± 0.03	b 0.88± 0.1	b 0.87± 0.02
Urea-N (mg/dl)	fg 27.03± 0.9	g 26.4± 0.6	fg 27.2 ± 0.6	df 31.4 ± 0.6	d 33 6 ± 0 3	ef 29.93 ± 0 98	efg 29.1 ± 1.4	a 65.8 ± 2.2	c 49.5 ± 0.8	C 50.4± 1.6	b 56.9 ± 0.7	B 57.7± 0.97	b 55.8 ± 0.8	b 56.7 ± 1.3
Uric acid (mg/dl)	d 2 40 ± 0.1	d 2.3 ± 0.1	d 2.2 ± 0.1	d 2.3 ± 0.1	d 2.3 ± 0.1	d 2.23 ± 0 03	D 2.3 ± 0.1	a 3.5 ± 0.1	c 2.8 ± 0.1	C 2.9 ± 0.1	b 3.1 ± 0.03	B 3.2 ± 0.03	b 3.2 ± 0.1	b 3.2 ± 0.03

C = control **A = aflatoxin**

a – g means in the same column with different superscripts significantly ($p \leq 0.05$) differ

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إمكانية تخفيف آثار التسمم الغذائى الأفلاتوكسينى على أداء النمو والكيمياء الحيوية لذكور الجرذان البيضاء

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تم تغذية ١٢٦ من الجرذان البيضاء الذكور (متوسط وزنها ١٠٥ جم) لمدة ٢١ أسبوعا على ١٤ معاملة غذائية (٣ مكررات × ٣ حيوانات/معاملة) تتضمن عليقة خالية من الأفلاتوكسين بدون معاملة، أو مضافا إليها ٥ر٠ أو ١% طفله، أو معاملة بالأمونيا (١، ٣%)، أو معاملة بفوق أكسيد الهيدروجين (٣، ٦%)، أو مضافا إليها ١٠٠٠ جزء/بليون أفلاتوكسينات أو مضاف إليها التوكسين والطفلة أو الأمونيا أو فوق أكسيد الهيدروجين (بنفس التركيزات السابقة). تم إسكان الحيوانات فى بطاريات سلك (٣ حيوانات فى كل قفص) ، ويتم التغذية للشبع، والماء متاح يوميا خلال ٢٤ ساعة. ويمكن تلخيص أهم النتائج المتحصل عليها من هذه التجربة في أن تناول الغذاء الملوث بالأفلاتوكسين (١٠٠٠ جزء فى البليون) بمفرده (مقارنة بالغذاء الغير ملوث) أدى إلى انخفاض معنوى فى وزن الجسم الحى ومعدل النمو اليومى ومعدل استهلاك الغذاء اليومى وكفاءة الاستفادة من الغذاء وارتفاع معنوى فى معدل الوفيات. النقص فى وزن الجسم الحى يزيد مع زيادة فترة التغذية الملوثة حيث يكون ٣ر٢٨% عند الأسبوع الـ ١٣ و ٣ر٤١% عند الأسبوع الـ ٢١ . أدت الإضافات المختلفة (طفلة، أمونيا، وفوق أكسيد الهيدروجين) إلى للعلائق الملوثة بالأفلاتوكسين إلى تقليل التأثير السيئ للأفلاتوكسين على أداء الفئران مقارنة بتلك التى تناولت عليقة ملوثة بالأفلاتوكسين فقط، وعلى ذلك فإن الإضافة الفعالة للوقاية من التأثير السلبى للأفلاتوكسين على الأداء تكون الطفلة. الأفلاتوكسين الخارج فى الروث (كنسبة مئوية من المأكول) يكون مرتفعا فى الفئران التى تناولت عليقة ملوثة بالأفلاتوكسين مع الإضافات المختلفة مقارنة بتلك التى تتناول عليقة ملوثة بالأفلاتوكسين فقط. الأفلاتوكسين الخارج فى الروث للفئران التى تناولت عليقة ملوثة فقط سجل ٦ر٣% من المأكول، ولكن بلغ ٧ر٢٤، ٤ر٢٤، ٥ر٦، ٥ر٥، ٢ر٤ و ٩ر٣% لتلك التى تناولت عليقة ملوثة بالأفلاتوكسين مع ٥ر٠، ١% طفلة، ١، ٣% أمونيا و ٣ر٦% فوق أكسيد الهيدروجين على التوالى. أدت التغذية على الغذاء الملوث بالأفلاتوكسين إلى انخفاض محتوى بلازما الدم من البروتينات الكلية والألبيومين والكولسترول الكلى والجلسريدات الثلاثية ولكن حدث زيادة فى GPT, GOT والفوسفاتيز القاعدى واليوريا وحمض اليوريك والكرياتنين. الإضافات المختلفة تعمل على تقليل التأثير السيئ للأفلاتوكسين على مكونات الدم، وكانت الطفلة أفضل المعاملات. وجد أن الأعضاء الداخلية (كبد – كلى – طحال – خصى – قلب – رئات) قد زاد وزنها المطلق بالتغذية على العليقة الملوثة، وأن المعاملات المختلفة لم تحسن الوضع. مما سبق يتضح مدى خطورة التلوث الغذائى الأفلاتوكسينى على الحيوان والإنسان المستهلك لمنتجات لحوم هذه الحيوانات ملوثة التغذية، وأن المواد المدمصة وإن حدت لحد ما من امتصاص السموم الفطرية، أيضا وسيلة غير كافية ولا مانعة للتسمم الأفلاتوكسينى وأثاره المختلفة، مما يؤكد على أهمية الوقاية من الغزو الفطرى للعلف ومكوناته حتى يمنع بالتالى من إنتاج التوكسين على العلف، أى أن الوقاية تظل خير من العلاج.