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Detoxification of Aflatoxin B1 and Fumonsinin B1 in Broiler chickens by grape seed extract and hydrated sodium calcium aluminosilicate

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Abstract: A feeding trial was conducted on commercial broilers for a period of 35 days to determine the individual effects of aflatoxin B1 (AFB1) and Fumonsinin B1 (FB1) toxins on performance, organ weights and immune status. The efficacy of dietary grape seed extract (GSE) and hydrated sodium calcium aluminosilicate (HSCAS) in preventing the adverse effects of AFB1 and FB1 toxin was also evaluated. Seven dietary treatments were tested on 420 commercial broiler chickens divided randomly into 42 replicates of 10 chicks each. the nutritional treatments: (1) control (non-contaminated diet); (2) non contaminated diet + 1mg/kg of AFB1; (3) non-contaminated diet + 400 mg/kg FB1 ; (4) 1 mg/kg AFB1 + 500 mg/kg GSE; (5) 1 mg/kg AFB1 + 5 g/kg HSCAS; (6) 400 mg/ kg FB1 + 500 mg/kg GSE; and (7) 400 mg/kg FB1 + 5 g/kg HSCAS. Feed intake and body weight gain were recorded weekly. On the 35th day, organ morphology and antibody titers for infectious bursal disease (IBD) and Newcastle disease (ND) were assessed. Individually, AFB1 and FB1 reduced weight gain while raising the feed conversion ratio (FCR) and mortality rate ($p \leq 0.05$). AFB1 alone ($p \leq 0.05$) raised the weights of the liver, kidney, gizzard, and spleen while decreasing the weights of the thymus and bursa. FB1 increased liver and gizzard weights and decreased thymus weight. Individual doses of AFB1 and FB1 substantially impacted NDV and IBD titers. GSE supplementation increased weight gain, enhanced feed conversion efficiency, and restored organ weights ($p \leq 0.05$). With the addition of GSE, antibody titers against NDV and IBD were dramatically increased. Adding HSCAS increased body weight gain and restored organ weights in birds fed AFB1 alone but not in birds given FB1 ($p \leq 0.05$). FCR in groups fed toxins was unaffected by HSCAS addition. Adding HSCAS ($p \leq 0.05$) increase antibody titers against NDV and IBD. In conclusion, mycotoxins cause adverse effects, especially aflatoxin B1, which is more severe than Fumonsinin B1. Whereas HSCAS (5 g/kg diet) is effective solely against AFB1, GSE (500 mg /kg diet) prevents the individual toxicity of AFB1 and FB1 in commercial broilers.

Keywords: Aflatoxin B1, Fumonsinin B1, broiler chickens, grape seed, HSCAS

1. Introduction

Mycotoxins are secondary metabolites produced by fungi that can enter the human and animal diet through direct or indirect contamination of cereals and grains. Mycotoxins are challenging to identify once consumed since they typically induce modest syndromes that are easily confused with illnesses brought on by other microorganisms. Each mycotoxin can specifically damage one organ or system, resulting in specific clinical manifestations of an acute or chronic nature, depending on its physicochemical characteristics and the animal species involved. As a result, mycotoxicoses are frequently challenging to diagnose. Still, they can be based on detecting the toxin in foods, epidemiological information, histological findings, assessing metabolic residues in tissues and clinical indications (Attia et al., 2013; Yang et al., 2020).

Two major groups of mycotoxins significant in public and animal health comprise aflatoxins, formed by *Aspergillus flavus* and *Aspergillus parasiticus*, and fumonisins, which are chiefly produced by *Fusarium verticillioides* and *Fusarium proliferatum*. The aflatoxins cause various of biological effects, including liver disorders, changes in growth rates, modifications to the immunogenesis mechanism, and carcinogenic and mutagenic effects in numerous animal species, particularly domestic poultry (Pier, 1973; Attia et al., 2016). The soft tissues and

fat deposits of poultry frequently become contaminated with aflatoxins, according to Sawhney et al. (1973). However, the largest buildup is found in biotransformation-related organs, including the liver and kidney.

The most prevalent member of the recently identified class of mycotoxins known as fumonisins is Fumonisin B1 (FB1), a water-soluble Fusarium metabolite (Bezuidenhout et al., 1988). Fumonisin B1 has been linked to animal pulmonary edema, hydrothorax syndrome in humans, and equine leukoencephalomalacia (Marasas et al., 1988). According to Norred (1993), fumonisin B1 also damages the liver in rats and induces liver cancer and atherosclerosis in monkeys. In South Africa and China, fumonisins have been linked epidemiologically to a high incidence of esophageal cancer (Yoshizawa et al., 1994). According to studies conducted on chicks (Li et al., 1997), FB1 is linked to poor performance, increased free sphinganine: sphingosine ratios, increased organ weights, impaired immunological responses, and organ diseases. According to Asrani et al. (2006), FB1 in quail has been linked to ruffled feathers, decreased feed and water intake, poor body growth, greenish mucus diarrhoea, and 59% mortality.

Because there are no practical ways to ultimately prevent mycotoxin toxcity, binders like HSCAS have been proposed to sequester aflatoxins, bind with them, and form a more stable complex that controls the toxins from being absorbed in the animal's digestive tract, thereby limiting and their impact on animals and transfer to edible animal products (Pasha et al., 2007; Attia et al, 2013; 2016). However, it was not without their drawbacks. However, it was discovered that adsorbents have limited effectiveness against some mycotoxins, require high levels of incorporation, and can have adverse effects on some dietary nutrients, reducing the nutritional value of animal diets. They may also contain dioxins and heavy metals, significantly limiting their use as a feed additive (Jouany et al., 2005).

One of the most popular fruits consumed worldwide is the grape (*Vitis vinifera*), which contains certain phytogenic chemicals. Because of its cleaning qualities, the grape called the "queen of fruits". According to Bown (2001), it is used for cleansing and enhance cleanses and liver function. Furthermore, the grape significantly controls several liver illnesses, excessive blood pressure, and anemia. Additionally, grape's fibers and fruit acids are crucial parts of the kidney and digestive system's blood-cleansing processes (Celik et al., 1998). Grape Seed Extract (GSE) is a medical herb used chiefly for its great proanthocyanidin content. GSE is a naturally found plant material containing many antioxidant nutrients common as oligomeric proanthocyanidins (OPCs), which are more influential antioxidants than vitamins E, C, and β-carotene. GSE has been found to be the highest source of (OPCs).

The current trial was conducted to estimate the effectiveness of grape seed extract and HSCAS on performance, organ morphology, and serum immunological variables in broiler chickens exposed to individual mycotoxicoses of AFB1 and FB1.

2. Materials and MethodsProduction and quantification of mycotoxins

By using solid substrate fermentation, Shotwell et al. (1966) and Burmeister (1971) developed techniques to generate aflatoxin B1 and FB1, respectively. To produce aflatoxin B1 and FB1, the fungal cultures employed were *A. parasiticus* NRRL 2999 and *Fusarium verticilloides* MRC 826 (source: Institute of Animal Health, Dokki, Egypt). Thin layer chromatography was used to determine the mycotoxin content of the culture material using the AOAC (2005) methods for AFB1 and Dilkin et al. (2002) for FB1. The corresponding contents of AFB1

and FB1 were 300 and 6000 mg/kg, respectively.

Experimental Design and Management

Four hundred and twenty 1-day-old unsexed broiler chicks (Ross 308) were purchased from a local commercial hatchery and haphazardly distributed into 7 identical groups at the first day of age. Each group was divided into six replicates (10 birds per replicate), in floor pens furnished with wood shavings. Mash feed and fresh water were given *ad-libitum* for 35 days of age. All the birds were reared under typical management circumstances from 0-5 weeks. A 23 h light:1 h dark regime was provided throughout the experimental period. Using a two-phase feeding schedule, the chicks were fed a typical commercial broiler diet of corn-and-soybean meal, from the first to the twenty-first day of the starter, and from the twenty-second to the thirty-fifth day of the grower. The National Research Council's (1994) Nutrient Requirements for Broiler Chickens NRC (1994) suggestion was used to establish the diet composition shown in Table 1.

The experimental feeding was designed as follows: The first group (T1) fed on a commercial broiler diet without supplement (control); T2 (AFB1) had aflatoxin B1 as 1mg /kg (Rajput et al., 2019), T3 (FB1) had fumonsinin B1 at 400 mg /kg (Rauber et al., 2013), T4 (AFB1+GSE) had a basal diet containing GSE 500 mg plus 1 mg AFB1/kg diet (Rajput et al., 2019), T5 (AFB1+HSCAS) fed a basal diet containing HSCAS 5 g plus 1 mg AFB1/kg diet (Neeff et al., 2013), T6 (FB1+GSE) had a basal diet containing GSE 500 mg plus 400 mg fumonsinin B1 /kg diet and T7 (FB1+HSCAS) fed a basal diet containing 5 g HSCAS plus 400 mg fumonsinin B1 /kg diet.

Grape seed extract analysis

The grape seed was collected and ground to a thickness of 2 mm. Grape seed was extracted using a mixture of 75% methanol and 25% dichloromethane. LC-MS/MS (Dionex Ultimate 3000 LC and TSQ Quantum Access Max MS, Thermo Fisher Scientific®, Massachusetts, USA) was used to analyze the phenolic components in grape seed extract. The separations were conducted at a flow rate of 700 µl/min, and the mobile phase was a gradient of the solvents A and B (methanol and 0.1% formic acid in water). The gradient of solvents utilized was as follows: 100% A for the first 0–1 min, 100–5% A for the next 1–22 min, 5% A for the next 22–25 min, and 100% B for 5 min. The injection had a 20 µL volume. On an ODS Hypersil column (250 4.6 mm 5 µm particle size, Thermo Fisher Scientific®), chromatographic separation was carried out at 30°C according to the method of Iacopini et al. (2008). Table 2 lists the quantities of the phenolic chemicals in GSE.

Table (1): Composition and calculated analysis of starter, grower and finisher diets.

Ingredients	Starter-grower (1-21d)	Finisher (22-35d)
Yellow corn	54.40	62.00
Soybean meal, 44%	27.00	24.05
Corn Gluten meal, 60%	10.00	6.19
Soy bean oil	4.55	4.00
Limestone	1.10	1.00
Di-calcium phosphate	2.20	2.05
Vit& min. premix*	0.30	0.30
DL-Methionine	0.05	0.01
L-lysine	0.15	0.15
Na Cl	0.25	0.25
Total	100	100
Calculated analysis: **		

CP, %	23.03	20.02
ME (Kcal/kg)	3004	3201
Calcium, %	1.05	0.97
Available phosphorus, %	0.45	0.42
Lysine, %	1.14	1.03
Methionine, %	0.52	0.41
TSAA, %	0.90	0.73

*Vitamin and mineral premix contains in the following per kg: vitamin A, 2,400,000 IU; vitamin D, 1,000,000 IU; vitamin E, 16,000 IU; vitamin K, 800 mg; vitamin B1, 600 mg; vitamin B2, 1600 mg; vitamin B6, 1000 mg; vitamin B12, 6 mg; niacin, 8000 mg; folic acid, 400 mg; pantothenic acid, 3000 mg; biotin 40 mg; antioxidant, 3000 mg; cobalt, 80 mg; copper, 2000 mg; iodine, 400; iron, 1200 mg; manganese, 18,000 mg; selenium, 60 mg; zinc, 14.000 mg. **According to tables of NRC (1994).

Hydrated sodium and calcium aluminum silicates (HSCAS)

A commercial mycotoxin adsorbent (Toxi-Mold Plus®) purchased from Egyco-Vet Company was blended with a basal diet through a dose of 5 kg per ton for their bird groups.

Data collection

Growth Performance

Throughout the 5-week trial, body weight and feed intake (FI) were recorded weekly on a pen basis. Final body weight and Cumulative feed consumption were determined, the obtained data were used to calculate the feed conversion ratio (FCR). Mortality was recorded daily to adjust FCR.

Relative organ weight

Six birds from each treatment were chosen randomly from the flock at 5 weeks of age, weighed, and then slaughtered. The liver, kidney, gizzard, spleen, thymus glands, and bursa of Fabricius were then removed and weighed to determine the organs' relative weights, which were then expressed as a percentage of the body weight, as described by Montgomery et al. (1986).

Blood samples collection

At the end of the experiment, six blood samples per treatment (1 bird per replicate) were harvested into non-heparinized tubes.

Determination of oxidative stress parameters in the liver

For the colorimetric evaluation of malondialdehyde (MDA) levels and total antioxidant capacity (TAC) activities, frozen samples (6 samples/treatment) of liver homogenates were used. Using commercial kits, the serum's total antioxidant capacity (TAC) was assessed (Nanjing Jianheng Bioengineering Institute, Nanjing, Jiangsu, China). Using a commercial colorimetric assay kit, the serum malondialdehyde (MDA) concentration was measured using the thiobarbituric acid reaction method (Nanjing Jiancheng Bioengineering Institute, Nanjing, Jiangsu, China).

Serum concentrations of immunoglobulins analysis

The serum was separated by centrifugation at 1500× g for 10 min at 4 ◦C and stored at −18 ◦C for later examination. Using commercial kits that were purchased from the Nanjing Jiancheng Bioengineering Institute in Nanjing, China, the concentrations of serum, IgA, IgM, and IgG were determined.

Immunological parameters

The serum antibody titers against ND and IBD were determined using commercial test kits and the ELISA approach (Source: Kirkegaard and Perry Laboratories, Gaithersburg, Maryland 20879, USA).

Statistical analysis

The results of all response variables were subjected to one-way variance analysis (SAS, 2004). Using Duncan's Multiple Range Test (Duncan, 1955), the mean of variables with a significant F-test ($P \leq 0.05$) were compared.

3. Results and discussion Grape seed composition and extract analysis

Table (2) shows, grape seed extract there are several phenolic acids such as (Gallic Acid, Ferulic Acid, Vanillic Acid, Caffeic acid, and Sinapic acid) and Flavonoids such as (Catechin, Epicatechin, Procyanidine B1, Procyanidine B2 and Procyanidine C), the main compounds were Catechin, Epicatechin and gallic acid. Numerous polyphenols, including flavonols and anthocyanins, have been discovered as antioxidants in grape residue (Caillet et al. 2006). These results agree with El-Shafeey et al. (2012), who found that HPLC-UV analysis of Grape Seed Extract showed two primary compounds: gallic acid and epicatechin.

Table 2: Composition of the grape seed extract analyzed by PLC method

phenolic acids (mg/100 g)		Flavonoids (mg/100 g)	
Gallic Acid	45.58	Catechin	1385
Ferulic Acid	10.66	Epicatechin	1108
Vanillic Acid	15.85	Procyanidine B1	842
Caffeic acid	25.36	Procyanidine B2	763
Sinapic acid	8.33	Procyanidine C	528

Growth performance

AFB1 and FB1 both considerably ($p \leq 0.05$) decreased body weight and cumulative feed consumption while significantly ($p \leq 0.05$) increasing FCR and mortality rate (Table 3). Due to the gastrointestinal tract's significant absorption of AFB1 and FB1, these reductions occurred. According to Yunus et al. (2011), these substances are metabolised in the liver and produce toxic byproducts that harm the liver and limit protein synthesis, which leads to anorexia. According to Bintvihok and Kositcharoenkul (2006), AFB1 can significantly increase mortality in chickens by decreasing growth rate, feed efficiency, and disease incidence.

Table 3. Individual effects of AFB1 and FB1 with and without GSE (500 mg /kg) and HSCAS (5 g/kg) on the performance of broiler chickens at 35 days of age.

Treatments			Growth parameters				
Mycotoxin	GSE (500mg/kg)	HSCAS (5g/kg)	IBW, (g)	FBW, (g)	TFI. (g/bird)	FCR (feed:gain)	Mortality %
0	0	0	40.2	2100^{a}	3450^{a}	1.67^{d}	0.00^{g}
AFB1	0	0	40.5	1890^{d}	3360^{f}	1.82^{a}	16.66^{a}
FB1	0	0	40.4	1960^{c}	3380^{e}	1.76^{b}	11.66^{b}
AFB1	500	0	40.3	2010^{b}	3420^{b}	1.74^{c}	3.33^{e}
AFB1	0	5	40.5	2000^{b}	3400^{c}	1.73^{c}	5.00^{d}
FB1	500	0	40.5	2020^{b}	3405^{c}	1.72^{c}	1.66^{f}
FB1	0	5	40.3	1975^{bc}	3390^{d}	1.75^{b}	8.33^{c}
SEM			1.22	5.32	6.26	0.022	2.005
p-value			0.852	0.0001	0.0001	0.004	0.0001

$^{a-e}$ Means within each column bearing common superscript do not differ significantly ($p \leq 0.05$); SEM: Standard error of the mean; AFB1: aflatoxin B1; FB1: fumonsinin B1; GSE: grape seed extract; HSCAS: Hydrated sodium calcium aluminum silicates; IBW: initial body weight; FBW: Final body weight; BWG: body weight gain; TFI: total feed intake; FCR: feed conversion ratio.

These findings are consistent with earlier research, which demonstrated that broilers fed diets contaminated with 1 mg/kg AFB1 experienced significant reductions in ADG and ADFI as well as a negative impact on the FCR when compared to the control group (Gowda et al., 2009). AFB1 has been linked to reluctance, anorexia, and protein synthesis and lipogenesis reduction, which explains these negative consequences (Bagherzade et al., 2012). Additionally, according to earlier studies, AFB1 can change the intestinal absorbing barrier, decrease the activity of pancreatic lipase, amylase, and trypsin, and alter the cell's energy metabolism by interfering with gluconeogenesis, the tricarboxylic acid cycle, and fatty acid

synthesis, which slows down growth (Osborne et al., 1982).

BW reductions in the FB1-fed group may be attributed to a lower feed utilisation efficiency (Kubena et al., 1997; Attia, 2023), which was most likely brought about by FB1s' disruption of sphingolipid biosynthesis (Wang et al., 1991). These sphingolipids control ion pumps, cell surface receptors, and other critical mechanisms for cell survival and function (Merrill et al., 1996). In FB1 group, the drop in BW might have also been influenced by diarrhea and intestinal diseases (goblet cell hyperplasia).

The body weight gain of the animals receiving the highest level of FB 1 was reported to be reduced by up to 18% in comparison to the treatment without mycotoxin by Rauber et al. (2013), who evaluated contamination levels of 100 and 200 mg FB1/kg feed in chicks with ages ranging from 1 to 28 days. The authors noted a decrease in feed intake of more than 30%. Nevertheless, according to Rauber et al. (2013), fumonisin contamination of the diet at levels up to 200 mg/kg did not affect mortality. When quail were fed a meal containing 150 ppm of FB1 starting at age 5 day (Deshmukh et al. 2005a), a mortality rate of 2.25% was seen. However, when FB1 (300 ppm) was added to the diet starting at age 1 day (Asrani et al. 2006), a death rate of 59% was seen.

The effects of growth depression were significantly reduced when GSE was added to the mycotoxin-contaminated diets (Table 3). In the AFB1 and FB1 fed groups, adding GSE dramatically enhanced 5^{th} week body weight. In all the mycotoxin-fed groups, the addition of GSE dramatically increased cumulative feed consumption and lowered FCR. Adding GSE to the diet can prevent AFB1's harmful effect on growth performance. According to Long et al. (2016), GSE might considerably increase the body weight of mice whose AFB1 levels were lowered. This is consistent with our research, which revealed that adding GSE (500 mg/kg) to meals contaminated with AFB1 significantly enhanced ADFI, ADG, and FCR compared to the AFB1 group.

The addition of HSCAS enhanced ($p \leq 0.05$) body weight, feed intake, and FCR in the groups fed AFB1 alone. HSCAS addition showed an insignificant impact on group-fed FB1. HSCAS addition enhanced ($p \leq 0.05$) the performance of broilers in the AFB1-fed groups. These results are in harmony with prior results of the protective impacts of HSCAS compound (Ledoux et al., 1999). A projected mechanism of AF chemisorptions by HSCAS is the configuration of a complex by the B carbonyl system of the AF with uncoordinated edge site aluminum ions in HSCAS, allowing the mycotoxin to pass safely through the animal (Phillips et al., 1990a; Attia et al., 2013). On the other hand, Kubena et al. (1998) found that adding HSCAS at the amount 10 kg/ton of diet did not safeguard against toxicity of T-2 on any of the performance parameters; these results supported the present results.

In studies on chickens (Kubena et al., 1993; Harvey et al., 1994) and swine (Harvey et al., 1994), several HSCAS compounds demonstrated protection against the toxicity of AF. However, the HSCAS provided no appreciable defense against fumonisin's toxicity. The absence of protection against fumonsin toxicity is consistent with earlier research, in which no in vivo protection against T-2 toxin, fumonisin, diacetoxyscirpenol, or ochratoxin A was seen with a variety of adsorbents (Huff et al., 1992 ; Kubena et al., 1993).

Organ weights

The proportional weights of the liver, kidney, thymus, and Fabricius bursa varied significantly among the treatments (Table 4). Liver, kidney, and spleen sizes all increased substantially due to AF. Only the liver's

weights statistically increased as a result of FB1. When compared to the unsupplemented control groups, GSE supplementation significantly decreased the increase in the relative weights of the liver, kidney, and spleen in the groups fed both individual toxins (Table 4). In contrast, supplementation of HSCAS significantly reduced the toxicity in the group provided AFB1 alone.

Similar to our findings, Ortatatli et al. (2005) research on birds fed a diet containing aflatoxin discovered a rise in the absolute and relative weights of the liver, kidney, and gizzard, demonstrating the hepato- and nephrotoxicity of aflatoxins. Because most aflatoxins bioactivate in the liver to the reactive 8, 9-epoxide form, which is known to bind DNA and proteins and cause structural damage to the liver as well as an increase in liver weight, the liver is thought to be the target organ for aflatoxin B1 (Bailey et al., 2006; Pasha et al., 2007). Increased lipid deposits in the liver brought on by decreased fat metabolism may be to blame for the rise in liver weight (Hsieh, 1979). Cholesterol and phospholipid production inhibition are the primary mediators of hepatic lipidosis. This impacts lipid transport from the liver (Manegar et al., 2010). Gizzard relative weight may have increased due to mucosal layer thickening and severe inflammation (Hoerr et al., 1982).

Additionally, Gelderblom et al. (1988) noted that a diet contaminated with FB1 led to necrosis in liver cells, including Kupffer cells (hepatic macrophages), indicating a change in cell function. The findings of Raju and Devegowda (2000) are consistent with the lack of any substantial effect of FB1 on the weight of the kidney and spleen seen in our study. According to Gabal and Azzam (1998), aflatoxin is a potent nephrotoxic substance that might harm kidneys and modify its function. According to Ledoux et al. (1992), fumonisins are also hepatotoxic to broilers and are linked to biliary hyperplasia and multifocal necrosis.

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BW reductions in the FB1-fed group may be attributed to a lower feed utilisation efficiency (Kubena et al., 1997; Attia, 2023), which was most likely brought about by FB1s' disruption of sphingolipid biosynthesis (Wang et al., 1991). These sphingolipids control ion pumps, cell surface receptors, and other critical mechanisms for cell survival and function (Merrill et al., 1996). In FB1 group, the drop in BW might have also been influenced by diarrhea and intestinal diseases (goblet cell hyperplasia).

The body weight gain of the animals receiving the highest level of FB 1 was reported to be reduced by up to 18% in comparison to the treatment without mycotoxin by Rauber et al. (2013), who evaluated contamination levels of 100 and 200 mg FB1/kg feed in chicks with ages ranging from 1 to 28 days. The authors noted a decrease in feed intake of more than 30%. Nevertheless, according to Rauber et al. (2013), fumonisin contamination of the diet at levels up to 200 mg/kg did not affect mortality. When quail were fed a meal containing 150 ppm of FB1 starting at age 5 day (Deshmukh et al. 2005a), a mortality rate of 2.25% was seen. However, when FB1 (300 ppm) was added to the diet starting at age 1 day (Asrani et al. 2006), a death rate of 59% was seen.

The effects of growth depression were significantly reduced when GSE was added to the mycotoxin-contaminated diets (Table 3). In the AFB1 and FB1 fed groups, adding GSE dramatically enhanced 5th week body weight. In all the mycotoxin-fed groups, the addition of GSE dramatically increased cumulative feed consumption and lowered FCR. Adding GSE to the diet can prevent AFB1's harmful effect on growth performance. According to Long et al. (2016), GSE might considerably increase the body weight of mice whose

AFB1 levels were lowered. This is consistent with our research, which revealed that adding GSE (500 mg/kg) to meals contaminated with AFB1 significantly enhanced ADFI, ADG, and FCR compared to the AFB1 group.

The addition of HSCAS enhanced ($p \leq 0.05$) body weight, feed intake, and FCR in the groups fed AFB1 alone. HSCAS addition showed an insignificant impact on group-fed FB1. HSCAS addition enhanced ($p \leq 0.05$) the performance of broilers in the AFB1-fed groups. These results are in harmony with prior results of the protective impacts of HSCAS compound (Ledoux et al., 1999). A projected mechanism of AF chemisorptions by HSCAS is the configuration of a complex by the B carbonyl system of the AF with uncoordinated edge site aluminum ions in HSCAS, allowing the mycotoxin to pass safely through the animal (Phillips et al., 1990a; Attia et al., 2013). On the other hand, Kubena et al. (1998) found that adding HSCAS at the amount 10 kg/ton of diet did not safeguard against toxicity of T-2 on any of the performance parameters; these results supported the present results.

In studies on chickens (Kubena et al., 1993; Harvey et al., 1994) and swine (Harvey et al., 1994), several HSCAS compounds demonstrated protection against the toxicity of AF. However, the HSCAS provided no appreciable defense against fumonisin's toxicity. The absence of protection against fumonsin toxicity is consistent with earlier research, in which no in vivo protection against T-2 toxin, fumonisin, diacetoxyscirpenol, or ochratoxin A was seen with a variety of adsorbents (Huff et al., 1992 ; Kubena et al., 1993).

Organ weights

The proportional weights of the liver, kidney, thymus, and Fabricius bursa varied significantly among the treatments (Table 4). Liver, kidney, and spleen sizes all increased substantially due to AF. Only the liver's weights statistically increased as a result of FB1. When compared to the unsupplemented control groups, GSE supplementation significantly decreased the increase in the relative weights of the liver, kidney, and spleen in the groups fed both individual toxins (Table 4). In contrast, supplementation of HSCAS significantly reduced the toxicity in the group provided AFB1 alone.

Similar to our findings, Ortatatli et al. (2005) research on birds fed a diet containing aflatoxin discovered a rise in the absolute and relative weights of the liver, kidney, and gizzard, demonstrating the hepato- and nephrotoxicity of aflatoxins. Because most aflatoxins bioactivate in the liver to the reactive 8, 9-epoxide form, which is known to bind DNA and proteins and cause structural damage to the liver as well as an increase in liver weight, the liver is thought to be the target organ for aflatoxin B1 (Bailey et al., 2006; Pasha et al., 2007). Increased lipid deposits in the liver brought on by decreased fat metabolism may be to blame for the rise in liver weight (Hsieh, 1979). Cholesterol and phospholipid production inhibition are the primary mediators of hepatic lipidosis. This impacts lipid transport from the liver (Manegar et al., 2010). Gizzard relative weight may have increased due to mucosal layer thickening and severe inflammation (Hoerr et al., 1982).

Additionally, Gelderblom et al. (1988) noted that a diet contaminated with FB1 led to necrosis in liver cells, including Kupffer cells (hepatic macrophages), indicating a change in cell function. The findings of Raju and Devegowda (2000) are consistent with the lack of any substantial effect of FB1 on the weight of the kidney and spleen seen in our study. According to Gabal and Azzam (1998), aflatoxin is a potent nephrotoxic substance that might harm kidneys and modify its function. According to Ledoux et al. (1992), fumonisins are also hepatotoxic to broilers

and are linked to biliary hyperplasia and multifocal necrosis.

Table 4. Individual effects of AFB1 and FB1 with or without GSE (500 mg/kg) and HSCAS (5 g/kg) on relative organ weights at 35 days.

Treatments			Organ weights %					
Mycotoxin	GSE (500mg/kg)	HSCAS (5g/kg)	Liver	Kidney	Gizzard	Spleen	Thymus	Bursa
0	0	0	2.26^{d}	0.861^{d}	2.32^{d}	0.248^{b}	0.350^{a}	0.280^{a}
AFB1	0	0	3.86^{a}	1.095^{a}	2.78^{a}	0.301^{a}	0.300^{c}	0.248^{c}
FB1	0	0	3.48^{b}	0.870^{d}	2.58^{b}	0.250^{b}	0.305^{c}	0.262^{b}
AFB1	500	0	2.63^{cd}	0.938^{c}	2.35^{d}	0.250^{b}	0.342^{a}	0.286^{a}
AFB1	0	5	2.70^{c}	0.960^{b}	2.45^{cd}	0.250^{b}	0.326^{b}	0.290^{a}
FB1	500	0	2.56^{cd}	0.860^{d}	2.36^{d}	0.246^{b}	0.340^{a}	0.279^{a}
FB1	0	5	3.36^{b}	0.875^{d}	2.56^{c}	0.252^{b}	0.310^{bc}	0.280^{a}
SEM			0.040	0.016	0.020	0.005	0.007	0.055
p-value			0.0001	0.0001	0.0001	0.032	0.002	0.001

$^{a-e}$ Means within each column bearing common superscript do not differ significantly ($p \leq 0.05$); SEM: Standard error of the mean; AFB1: aflatoxin B1; FB1: fumonsinin B1; GSE: grape seed extract; HSCAS: Hydrated sodium calcium aluminum silicates.

In the current investigation, HSCAS was added to the aflatoxin B1 diet to considerably ($p \leq 0.05$) reduce the increase in organ weights that was shown in hens that were only fed aflatoxin B1. By including HSCAS in the diet, Ledoux et al. (1998), and Gowda et al. (2008) showed that a rise in the organ weights due to aflatoxin B1 in broiler chickens could be prevented. Additionally, Sehu et al. (2007) showed under a microscope how adding HSCAS to quail diet partially reduced the fat deposition brought on by aflatoxin in the liver and thus decreased the weight of the liver.

In the current investigation, adding GSE (500 mg/kg) to contaminated meals effectively reduced the significant increase in liver-relative weight in the AFB1 or FB1 group. These outcomes, therefore, supported the hypothesis that GSE protects against the liver damage brought on by AFB1 and FB1.

Dietary interventions had a sizable impact on the relative weights of the Fabricius bursa and thymus. The findings of the current trial are consistent with past reports of lymphoid organ atrophy during mycotoxicoses (Devegowda et al., 1995). The mycotoxins may have caused necrosis and cellular depletion, resulting in these organs' shrinkage (Hoerr et al., 1981; Attia, 2023). Raju and Devegowda (2002) reported similar outcomes.

The weights of the bursa and thymus were restored when GSE was added to diets containing toxins. These findings support the finding of Hajati et al. (2018) that GSE may benefit broilers by reducing the atrophy caused by heat stress on lymphatic organs. The weights of the thymus and bursa were restored by HSCAS supplementation in the AFB1-fed groups but not in the FB1-fed groups. Chestnut et al. (1992) and Kubena et al. (1998) noted that the toxicity of T-2 (the secondary metabolite of the Fusarium fungi) on thymus and bursal weights could not be reversed by HSCAS.

Oxidative stress

The mean serum and liver level of malondialdehyde (MDA) was found to be significantly greater in the A1FB1 and FB1 groups than in the control group. While HSCAS with AFB1 only reported a significant decrease in the mean level of serum and liver MDA, a substantial decrease in the mean level of serum MDA was seen in GSE with AFB1 and FB1 when compared to that of AFB1 or FB1 groups without

additives. On the other hand, with total antioxidant capacity (TAC) (Table 5).

The use of antioxidants to counteract the damaging effects of aflatoxins has recently piqued the interest of scientists studying. This is because aflatoxins have been shown to increase the formation of reactive oxygen species (ROS), and oxidative stress has been proposed as one of the underlying mechanisms for cell injury and DNA damage caused by AFB1 (Yang et al., 2000). AFB1 causes oxidative damage because it enhances the generation of reactive oxygen spices (ROS), which in turn damages the lipids in cell membranes and changes the fluidity and permeability of the cell membrane (Choi et al., 2010).

Table 5. Individual effects of AFB1 and FB1 with or without GSE (500 mg/kg) and HSCAS (5 g/kg) on oxidative stress in serum and liver at 35 days

Treatment			Serum		Liver	
Mycotoxin	GSE (500mg/kg)	HSCAS (5g/kg)	TAC U/ml	MDA nmol/ml	TAC mmol/g tissue	MDA nmol/mg protein
0	0	0	8.22^{a}	1.86^{d}	5.61^{a}	36.30^{e}
AFB1	0	0	3.62^{e}	3.48^{a}	2.81^{d}	62.70^{a}
FB1	0	0	4.17^{d}	3.06^{b}	3.24^{c}	56.58^{b}
AFB1	500	0	7.80^{ab}	2.16^{c}	4.91^{b}	40.26^{d}
AFB1	0	5	8.00^{a}	2.08^{c}	4.73^{b}	43.18^{d}
FB1	500	0	8.08^{a}	2.00^{c}	5.00^{b}	39.50^{de}
FB1	0	5	5.00^{c}	2.84^{b}	3.8^{bc}	50.80^{c}
SEM			0.246	0.142	1.18	3.86
p-value			0.0001	0.009	0.0001	0.0001

$^{a-e}$ Means within each column bearing common superscript do not differ significantly ($p \leq 0.05$); SEM: Standard error of the mean; AFB1: aflatoxin B1; FB1: fumonsinin B1; GSE: grape seed extract; HSCAS: Hydrated sodium calcium aluminum silicates; TAC: total antioxidant capacity; MDA: malondialdehyde.

Measuring the amount of MDA, one of the primary by-products of polyunsaturated lipid peroxidation, can help determine the stage of cell damage and lipid peroxidation (Naaz et al., 2014). According to Yener et al. (2009), consuming AFB1 can lower antioxidant levels and cause oxidative stress. Our findings demonstrated that compared to the control group, the feed contaminated with AFB1 significantly raised the concentration of MDA and decreased the activity of TAC in the serum of broilers. Similar harmful effects on the oxidative state were seen in the liver and serum of broilers at various dosages of AFB1 (Liu et al., 2016).

According to El-Shafeey et al. (2012), FB1 increased the amount of lipid peroxides in rats, which led to toxicity caused by free radicals. Critical biomolecules like nucleic acids, proteins, and lipids are frequently the focus of oxidative damage (Hoehler, 1998). According to research by Stockmann-Juvala et al. (2004), FB1 induces oxidative stress, which may, at least in part, be responsible for FB1 toxicity and carcinogenicity.

Adding GSE to a diet contaminated with AFB1 and FB1 reduced lipid peroxidation and raised the serum antioxidant level. Previous research found that adding GSE to the diet reduced the oxidative stress caused by AFB1 and dramatically reduced immunological injury in mice (Long et al., 2016). Additionally, through increasing antioxidant capacity, GSE further established its protective impact against zearalenone-induced adverse effects (Long et al., 2016). Therefore, the antioxidant activity of GSE in broiler chickens contributes significantly to the prevention of the oxidative damage brought on by AFB1 and FB1.

The antioxidant is short for antioxidant free radical, a physiological phenomenon that slows down the gradual loss of function in numerous animal organs and acts as a vital safeguard for the well-being of the animal body (Jia et al. 2019). Numerous polyphenols, including flavonols and anthocyanins, have been discovered as antioxidants in grape residue (Caillet et al. 2006). Previous studies have demonstrated that grape polyphenols can scavenge free radicals, stop oxidation reactions, and increase the antioxidant capacity of grape seeds up to 20–50 times higher than vitamin E and vitamin C (Yang et al. 2016; Abu Hafsa and Ibrahim 2018). Our research showed that grape seed extract decreased the amount of MDA, showing that grape seed extract might control tissue lipid oxidation and increase antioxidant capacity.

Total antioxidant capacity (TAC) is a comprehensive index that reflects the body's potential to scavenge free radicals. The higher TAC, the stronger the antioxidant ability (Attia et al., 2016; Cong et al. 2021). It was discovered that adding grape seed could significantly increase the contents of CAT, GSH-Px, and TAC, stimulating the ability to scavenge free radicals, and improving the body's antioxidant capacity, following the findings of prior reports (Viveros et al. 2011; Iqbal et al. 2015). In light of the bird's performance results, it was hypothesized that grape seed would enhance birds performance by increasing the body's antioxidant capacity.

Serum immunoglobulins

Mycotoxins' effects on animal immunosuppression are a welfare concern as they may raise the risk of exposure to infectious diseases, which could lead to economic losses (Fink-Gremmels 2008). The most popular technique to evaluate the humoral immune response is to measure the concentration of serum immunoglobulins such IgA, IgG, and IgM (Meissonnier et al., 2008). In the present investigation, compared to the control group, broilers given a meal contaminated with AFB1 and FB1 significantly reduced serum IgA, IgG, and IgM levels (Table 6). These findings are in line with a prior study by Liu et al. (2016), which demonstrated that AFB1 is known to be immunosuppressive in birds and found that broiler's serum levels of IgA, IgG, and IgM were considerably decreased when fed an AFB1-containing diet. According to these findings, AFB1 may damage the body's humoral function. However, according to research, the serum IgM concentration of broiler chickens exposed to AFB1 did not significantly reduce (Shi et al., 2006). The different results can be explained as the effects of AFB1 on humoral immunity depending on the dosage and species of chicken (Attia et al., 2013; Attia, 2023), because AF hinders protein synthesis, which lowers the amount of immunoglobulins present. Additionally, aflatoxins impede the transport of amino acids and mRNA transcription, which inhibits DNA synthesis and reduces antibody titers (Yunus et al., 2011). Further, according to Ul-Hassan et al. (2012), the frequency of IgA, IgG, and IgM-bearing cells in the bursa of Fabricius decreased due to aflatoxins, which also drastically reduced the immunoglobulins.

Table 6. Individual effects of AFB1 and FB1 with or without GSE (500 mg/kg) and HSCAS (5 g/kg) on serum immunoglobulins at 35 days

Treatments			Serum immunoglobulins (mg/ml)		
Mycotoxin	GSE (500mg/kg)	HSCAS (5g/kg)	IgA	IgG	IgG
0	0	0	2.86^{a}	2.60^{a}	2.36^{a}
AFB1	0	0	1.92^{d}	1.87^{d}	1.90^{d}
FB1	0	0	2.46^{c}	1.90^{c}	2.00^{c}
AFB1	500	0	2.68^{b}	2.50^{b}	2.28^{ab}
AFB1	0	5	2.62^{b}	2.48^{b}	2.15^{b}
FB1	500	0	2.63^{b}	2.60^{b}	2.30^{a}
FB1	0	5	2.50^{c}	2.00^{c}	2.05^{c}
SEM			0.185	0.240	0.176
p-value			0.004	0.036	0.001

a-e Means within each column bearing common superscript do not differ significantly ($p \leq 0.05$); SEM: Standard error of the mean; AFB1: aflatoxin B1; FB1: fumonsinin B1; GSE: grape seed extract; HSCAS: Hydrated sodium and calcium aluminum silicates.

In earlier research, FB1 also noted reduced humoral immunity. White Leghorn chicks fed diets containing 61 mg of FB /kg diet, 10.5 mg of FB /kg diet, and 42.7 mg of moniliformin /kg diet for six weeks showed a significant decrease in total Ig and IgG levels in the primary and secondary response to SRBC (Qureshi et al., 1995).

Our findings demonstrated that, compared to the contaminated groups, adding GSE to diets contaminated with AFB1 and FB1 significantly enhanced the blood IgA, IgG, and IgM content. These findings demonstrated that both AFB1 or FB1 compromised the immune system. However, the adverse effects of AFB1 or FB1 on the immune system were mitigated by the addition of GSE.

Immune response

At the end of the fifth week, all mycotoxin-fed groups had significantly ($p \leq 0.05$) reduced antibody titers against ND and IBD. The antibody titers against both illnesses were considerably ($p \leq 0.05$) decreased by both mycotoxins (Table 7). In groups that were fed AFB1, the lowest ND titer was observed. While supplementation of HSCAS demonstrated benefit ($p \leq 0.05$) against antibody titers only in the AFB1-fed group, supplementation of GSE significantly improved antibody titer against both ND and IBD in all mycotoxins-fed groups. The documented decline in the current study's antibody titer values for ND and IBD is a blatant sign of the immunosuppressive effect. It is widely acknowledged that mycotoxins' direct cytotoxic impacts cause necrotic and degenerative changes in immunological organs, additionally; AFB1 prevents the synthesis of DNA and proteins, which reduces the generation of antibodies (Yang et al., 2020). Similar findings were made by Tessari et al. (2006), who reported that broilers given rations treated with aflatoxin B1 displayed decreased geometrical mean antibody titers against NDV.

However, when diets containing various levels of FB1 were administered to chicks and turkeys, the weights of the lymphoid organs (bursa, spleen, and thymus) were shown to be reduced (Li et al., 2000). The humoral immune response has been linked to decreased bursa weights, and broiler chicks and turkey poults given 100 mg of FB1/kg of feed had considerably lower secondary anti-Newcastle disease virus antibody titers than control birds (Li et al., 2000). Earlier research with chicks fed FB1-containing diets revealed that FB1 may inhibit the cell-mediated immune response by significantly lowering white blood cell counts and the number of viable lymphocytes (Javed et al., 1995).

Table 7. Individual effects of AFB1 and FB1 with or without GSE (500 mg/kg) and HSCAS (5 g/kg) on the immune response to NDV and IBD at 35 days.

Treatments			Antibody (ELISA) titer	
Mycotoxin	GSE (500mg/kg)	HSCAS (5g/kg)	NDV	IBD
0	0	0	4216^{a}	4415^{a}
AFB1	0	0	3220^{d}	3316^{e}
FB1	0	0	3580^{c}	3262^{d}
AFB1	500	0	4150^{b}	4126^{b}
AFB1	0	5	4100^{b}	3990^{c}
FB1	500	0	4200^{ab}	4000^{c}
FB1	0	5	3600^{c}	3270^{d}
SEM			62.50	84.76
p-value			0.0001	0.0001

a-e Means within each column bearing common superscript do not differ significantly ($p \leq 0.05$); SEM: Standard error of the mean; AFB1: aflatoxin B1; FB1: fumonsinin B1; GSE: grape seed extract; HSCAS: Hydrated sodium calcium aluminum silicates; NDV: Newcastle disease virus; IBD: infectious bursal disease

When present in the ration at 200 mg FB 1/kg food, FB1 is immunosuppressive in chicks (Li et al., 1999). According to research by Wang et al. (1991), FB1 disrupts complex sphingolipids and accumulates free sphinganine and sphingosine as part of its mechanism of action. Yoo et al. (1992) demonstrated a correlation between decreased cell proliferation and FB1's inhibition of sphingolipid production. Martinova (1996) recognized sphingolipid breakdown products as antiproliferative lipids and showed that they reduced humoral and cell-mediated immune responses. Additionally, Qureshi and Hagler (1992) state that the suppression of chemical synthesis, modifications to metabolic processes, or changes to membrane shape and function may all be possible mechanisms by which FB1 may affect immune function. In mice that consumed FB1, there were signs of decreased activity and immunological responses. The administration of FB1 in mice stimulates caspase-8 enzyme activities. Caspase-8 can be specified as one of the intracellular downstream signaling molecules included in the tumor necrosis factor-alpha (TNF-α) apoptotic pathway, also having a role in FB1-induced apoptosis (Bhandari and Sharma 2002).

The improvement in humoral immune response observed in this study after the addition of binders like HSCAS is consistent with findings made by Ibrahim et al. (2000), who found that the addition of sodium bentonite binder was significantly effective in reducing the adverse effects of aflatoxin on the percentage and meant of phagocytosis and HI-titer in chicks immunized against NDV. Additionally, Sehu et al. (2007) examined the impact of HSCAS on the humoral immune response of quails given a diet polluted with aflatoxin B1. They discovered that it significantly reduced the decline in antibody titer brought on by the ND vaccine that was caused by aflatoxins.

The immunity may be prejudiced by oxidative stress, and the enhanced antioxidant role may improve their immune function in poultry (Iqbal et al., 2015; Kamboh et al., 2015). Polyphenols might improve the immune role by reducing the inflammatory method through nuclear factor-kappaB and nuclear factor-2–dominated ways in the small intestine, which has been confirming in pigs (Gessner et al., 2013). Likewise, weaning pigs fed diets containing 0.01 to 0.015% grape seed procyanidins showed similar results (Hao et al., 2015) and sows fed diets containing 0.02 to 0.03% GSE (Wang et al., 2019). IFN-γ plays a crucial role in controlling the activation of lymphocytes and monocytes, and serum IL-2 encourages the growth of natural killer cells, B lymphocytes, T

lymphocytes, and the creation of antibodies (Ao and Kim, 2020). Complement4 is a crucial component of the body's immunological defense system and plays a significant role in the immune response. Additionally, adding GSE to the diet increased serum C4, IL-2, and IFN-γ, showing that GSE may improve immune response by controlling antibodies, complements, and cytokines (Lipinski et al., 2017).

Based on prior research reviews, certain theories on the GSE's mode of operation can be developed. The first one, which is associated with the high cellulose content (37.8%) of grape seed meal (Taranu et al., 2020), has a significant capacity to adsorb AFB1 through electrostatic attractions and hydrogen bonding, resulting in the formation of a mycotoxin monolayer on its surface (Solis-Cruz et al., 2019). The second is based on polyphenols' propensity to bind to AFB1 mycotoxins and create a complex. In a recent study, Lu et al. (2017) showed that polyphenols from fermented tea can lessen the damage that AFB1 causes to the liver binding the toxin to form a complex (C-AFB1 complex). This complex prevents AFB1 from being absorbed and increases the amount of toxin eliminated by feces. AFB1 biotransformation in the gut, which reduces AFB1 absorption, may cause the protective benefits of the bioactive component proanthocyanidin, which is found in grape seeds, according to (Rajput, et al., 2017).

4. Conclusions

In conclusion, HSCAS (5 g/kg diet) was only helpful against aflatoxin B1, whereas GSE (500 kg diet) enhanced growth performance, lymphoid organs, and immunological response in birds exposed to AFB1 or FB1. Improving the performance of broiler chickens exposed to AFB1 or FB1 could be achieved by adding 500 mg GSE/kg feed. The ability of GSE to reduce the effects of mycotoxins may be linked to immune organ development, heightened immunological response to NDV and IBD, and improved antioxidant enzyme activity. More research appears to be required before grape seed extract may be used as an effective antioxidant feed supplement for chickens suffering from mycotoxicosis.

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إزالة سميه الأفلاتوكسين ب١ و الفومونسينين ب١ في دجاج التسمين عن طريق مستخلص بذور العنب وسيليكات الصوديوم والكالسيوم والألومنيوم

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مستخلص. تم إجراء تجربة تغذية على دجاج التسمين التجاري لمدة ٣٥ يومًا وتم التقييم أيضًا فعالية مستخلص بذور العنب (GSE) وسيليكات الصوديوم و الكالسيوم و الالومنيوم (HSCAS) في منع الضرر لسموم AFB1 و FB1. تم اختبار سبع معاملات غذائية على 420 كتكوت تسمين قسمت عشوائيا إلى ٤٢ مكرره وكل مكرره تحتوى على ١٠ كتكوت. المعاملات الغذائية: (١) مجموعه التحكم (نظام غذائي غير ملوث) ؛ (٢) نظام غذائي غير ملوث + ١ مجم / كجم من الأفلاتوكسين ؛ (٣) نظام غذائي غير ملوث + ٤٠٠ مجم / كجم فيومونيسين ؛ (٤) ١ مجم / كجم افلاتوكسين + ٥٠٠ مجم / كجم من بذور العنب ؛ (٥) ١ مجم / كجم افلاتوكسين + ٥ جم / كجم سيليكات الصوديوم و الكالسيوم و الالومنيوم ؛ (٦) ٤٠٠ مجم / كجم فيومونيسين + ٥٠٠ مجم / كجم من بذور العنب ؛ و (٧) ٤٠٠ مجم / كجم فيومونيسين + ٥ جم / كجم سيليكات الصوديوم و الكالسيوم و الالومنيوم. تم تسجيل العلف المستهلك وزيادة الوزن أسبوعيا. حتى اليوم الخامس والثلاثين ، تم تقييم مورفولوجيا الأعضاء والأجسام المضادة لمرض الجمبورا (IBD) ومرض النيوكاسل (ND). بشكل فردي ، قلل الأفلاتوكسين و فيومونيسين من وزن الجسم و العلف المستهلك مع زياده معدل التحويل (FCR) ومعدل الوفيات ($p \leq 0.05$). زاد AFB1 وحده ($p \leq 0.05$) أوزان الكبد والكلى والقوانص والطحال مع خفض أوزان غدة الثيموس و البرسا. زاد فيومونيسين من أوزان الكبد والقوانص ونقص وزن غدة الثيموس. كان للأفلاتوكسين و الفيومونيسين تأثير كبير على الاجسام المضاده لمرض النيوكاسل و مرض الجمبورا. أدت اضافه بذور العنب إلى زيادة وزن الجسم والعلف المستهلك وتحسين كفاءة تحويل العلف واستعادة أوزان الأعضاء ($p <0.05$). مع إضافة بذور العنب ، زاد مستوى الأجسام المضادة لمرض النيوكاسل ومرض الجمبورا بشكل كبير. في الطيور التي تتغذى على الأفلاتوكسين ب١ وحده ، ولكن ليس في المجموعات التي تتغذى على الفيومونيسين ب١ ، ادت إضافة سيليكات الصوديوم و الكالسيوم و الالومنيوم إلى زيادة وزن الجسم واستعادة أوزان الأعضاء ($p \leq 0.05$) فقط في المجموعات التي تلقت الأفلاتوكسين ، إضافة سيليكات الصوديوم و الكالسيوم و الالومنيوم ($p \leq 0.05$) زادت الأجسام المضادة لمرض النيوكاسل و مرض الجمبورا. وخلص أخيرا إلى أن السموم الفطرية تسبب آثارا عكسية على معظم العوامل المدروسة وخاصة الأفلاتوكسين B1 وهو أكثر ضررا من الفومونسينين B1. في حين أن إضافة سيليكات الصوديوم و الكالسيوم و الالومنيوم (٥ جم / كجم علف) فعال فقط ضد الأفلاتوكسين B1 ، في حين إن إضافة مستخلص بذور العنب (٥٠٠ مجم / كجم علف) فعال في منع السمية للأفلاتوكسين B1 و الفومونسينين B1 في دجاج اللاحم.