Research Article

Potential of Protective Effect of Hydrated Sodium Calcium Aluminosilicate and *Saccharomyces cerevisiae* against Dietary Aflatoxicosis in Broiler Chicks

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Abstract | This study investigated the defensive effect of dietary Hydrated sodium calcium aluminosilicate (HSCAS) (0.5%) and *Saccharomyces cerevisiae* (SC) (0.1%) on the preclusion of aflatoxin-B1 (AFB1) toxicity in broiler chicken during 1 – 42 days of age. Eight hundred broiler chicks were arbitrarily assigned for 8 treatments of 5 replicates (each contained 20 chicks). On the other hand, chickens were offered the basal dietas Negative Control (NC, group 1); the other three groups 2, 3, and 4 were fed NC supplemented with 0.5% HSCAS, SC and HSCAS plus SC respectively, group 5 chicks were fed a basal diet contaminated with 1ppm AFB1 as Positive Control (PC), the other three groups 6, 7 and 8 were fed PC supplemented with 0.5% HSCAS, SC, and HSCAS plus SC respectively. Results exhibited that broiler chicks fed with a PC diet showed significantly (P<0.05) a worse feed conversion ratio and inferior daily body weight during the experimental period. Furthermore, AFB1 toxicity at 1mg dose significantly (P<0.05) increased creatinine, uric acid concentrations, alanine aminotransferase (ALT), aspartate aminotransferase (AST) activities and significantly (P<0.05) reduced the antibody titer against sheep red blood cells (SRBC), total protein, albumin values in broilers serum compared with ngative control. Aflatoxins supplementation significantly (P<0.05) increased malondialdehyde values in liver and significantly (P<0.05) diminished the reduced glutathione (GSH), glutathione S- transferase (GST). In addition to, dissemination of aflatoxin residue in broilers liver was detected. Nevertheless, dietary addition of HSCAS and SC, in separate and combined forms, alleviated the above-mentioned alterations. The combination of HSCAS with SC was more efficient than having them in a separate form.

Keywords | Aflatoxin, Broiler, Adsorbent compounds, Detoxification

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INTRODUCTION

Aspergillus flavus and Aspergillus parasiticus produce aflatoxins (AF), which are the greatest dangerous and extensively confronted mycotoxins. Aflatoxins are known to resulted in severe growth reduction, immunosuppression, and mortality in chickens (Bilal et al., 2014; Attia et al., 2013). Aflatoxins are likewise a source of toxin residues in poultry meat, which may create a carcinogenic danger to persons (Nazir et al., 2014). Consequently, AF prevention, decontamination, and detoxification are chief global attentions. Among various detoxification methods, addition inert sorbents, for example hydrated sodium calcium aluminosilicate (HSCAS), zeolites, bentonites, and activated carbons, is one of the techniques to reduce aflatoxicosis in animals. These chemicals boundary mycotoxins' bioavaila-

bility and hinder their absorption into the gut. Nevertheless, many of them diminish amino acid and/or mineral bioavailability (Dawson, 1999).

Among the biological techniques, microbial degradation of AF is a new method and appears extra appropriate since it is extra specific, practical and environmentally helpful. Among microbes, *Saccharomyces cerevisiae*.

Because yeast cell walls include polysaccharides, proteins and lipids with absorption centers that bind mycotoxins by hydrogen and ionic bonding or hydrophobic interactions, this material can absorb mycotoxins (Huwig et al., 2001; Attia et al., 2016). Yeast culture can be used as a probiotic to enhance the immune system and encourage gut health, reducing the influences of mycotoxin. This research anticipates that chicks fed contaminated diets with AF will have less weight gain, a debilitated immune system, and organ damage, while using HSCAS and/or yeast additives will alleviate these harmful effects. This research examined the effects of aflatoxin B1 on growth performance, organ health, and immunological responses of broiler chcikens and the effective of HSCAS and Saccharomyces cerevisiae on alleviated of the harmful effects of AFB1.

MATERIALS AND METHODS

Aflatoxin B1 production

Aflatoxin B1 (AFB1) was produced on potato dextrose agar using a pure culture of *Aspergillus parasiticus* NRRL 2999 (Source: National Institute of Animal Health, Dokki, Cairo, Egypt). AFB1 was yielded on rice, and the toxin was isolated according to Rukmini and Bhat (1978). then quantified through Thin Layer Chromatography (TLC) as detailed by AOAC (2000).

Experimental Chickens

A total of 800 unsexed day-old broiler chicks from the Ross 308 strain were weighed, and distributed in a completely randomized experiment with 8 treatments and 5 replications of 20 chicks. Throughout the experimental period, the chicks were succumbed to conventional broiler chicken management and housed in floor pens in an environmentally controlled broiler house with litter floors. Chicks were offered ad libitum feed and water during the study. The temperature was kep at 30±1°C in the first week and reduced by 2.5°C each week to reach 21°C. From day one until day 4, the light cycle was 24 hours until 14 days of age, and thereafter the light cycle was 20 hours/day. Diets were formulated to offere the nutrient demands of commercial broilers during the starter - grower (0-21days) and finisher (22-42 days) periods. The composition of diets was according to NRC (1994) and is displayed in Table 1. The basal diet was formulated using feed ingredients, which were inspected for AF before diet formulation. The diets were prepared by supplememntingthe required quantity of aflatoxin to arrive at the levels of 0 and 1mg of AFB1. Diets were formulated without addition of aflatoxin and SC or HSCAS as Negative Control (group 1); NC plus 0.1% of SC (group 2); NC plus 0.5% of HSCAS (group 3); NC plus 0.1% of SC plus 0.5% HSCAS (group 4); 1mg Aflatoxin B1 as Positive Control (PC) (group 5); PC plus 0.1% of SC (group 6); PC plus 0.5% of HSCAS (group 7); and PC plus 0.1% of SC plus 0.5% HSCAS (group 8). The trail was handled according to the National regulations on animal welfare and Institutional Animal Ethical Committee (IAEC).

Hydrated Sodium Calcium Aluminosilicate (HSCAS)

Hydrated Sodium Calcium Aluminosilicate (HSCAS) is a feed additive, adsorbent, anti-caking, and toxin binder. It is mineral silicate and organic acids obtained from Trouw Nutrition International and mixed with the diet at 5 g/kg (0.5%).

Saccharomyces cerevisiae

The yeast was marked as KVASEC with about 11.6 x 10^9/gm viable cells. Active dried yeast was added to the diet at a concentration of 1 g per kg diet.

Measurements

Growth performance: Birds were individually weighed to estimate the BWG, while feed intake (FI) was valued based on replicate measurements. FCR was calculated as kg feed consumed/kg BWG. Data for productive performance were assessed at weekly periods.

Blood parameters: At the end of the experimental period, 2.5 mL of blood was taken from one chick, each replicate, and centrifuged at 3000 g for 15 minutes, then serum were separated from samples. Serum total protein, albumin, aspartate aminotransferase (AST), alanine aminotransferase (ALT) activities, cholesterol, and creatinine, were measured using commercial kits purched from Spinreact company. At day 42 of age, one bird from each replicate was select randomly, famished for 12 hours, and then slaughtered. The dressing (%) was assessed. The liver's weight was determined and represented as a percentage of the pre-slaughter BW.

Evaluation of immune system

At 42 days of age, 5.0 ml of 5 percent sheep red blood cell (SRBC) suspension was washed in sterile phosphate-buffered saline and injected into the pectoral muscle to assess humoral immunity. After 7 days, 3 ml of blood from the same birds were extracted through the wing vein. After separating serum, determine the overall response titer

Table 1: Composition and calculated analysis of starter and finisher diets.

Ingredients	Starter-grower (1-21d)	Finisher (22-42d)
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 (HCl)	0.15	0.15
NaCl	0.25	0.25
Total	100	100
Calculated analysis: **		
CP, %	23.03	20.02
ME (Kcal/kg)	3204	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

Abbreviations: CP: crude protein; ME: metabolizable energy; TSAA: total sulfur amino acid. *Each 3kg contain: Vit A 12000000IU, Vit D3 2000 000 IU, Vit E 10g, Vit K3 2g,Vit B1 1g, Vit B2 5g, Vit B6 1.5g, Vit B12 10mg, Nicotinic acid 30g, Pantothenic acid 10g, Folic acid 1g, Biotin 50mg, Choline chloride 250g, Iron 30g, Copper 10g, Zinc 50g, Manganese 60g, Iodine 1g, Selenium 0.1g, Cobalt 0.1g and carrier (CaCo3) to 3 kg. **According to tables of NRC (1994).

Table 2: Effect of SC and/or HSCAS on growth performance with dressing % in broiler chickens fed with aflatoxin contaminated feed.

Treatments	Growth performance				Dressing %
	Daily weight gain, g	Daily feed intake (g/bird/day)	FCR (g feed/g gain)	Mortality %	
NC	54.35^{ab}	103.80^{a}	1.91^{bc}	2.00^{b}	69.80^{b}
NC +SC	55.11^{a}	103.57^{a}	1.87^{c}	1.80^{b}	70.90^{a}
NC +HSCAS	54.47^{ab}	103.69^{a}	1.90^{bc}	1.90^{b}	69.50^{bc}
NC +SC+HSCAS	54.59^{ab}	103.57^{a}	1.89^{bc}	1.50^{b}	70.00^{b}
PC	46.85^{d}	99.52^{c}	2.12^{a}	20.0^{a}	65.80^{e}
PC +SC	52.96^{c}	101.42^{b}	1.91^{b}	4.50^{b}	68.00^{d}
PC +HSCAS	53.76^{bc}	101.66^{b}	1.89^{bc}	3.00^{b}	68.05^{d}
PC +SC+HSCAS	53.78^{bc}	101.50^{b}	1.88^{bc}	3.00^{b}	69.00^{c}
SEM	0.526	0.311	0.016	1.257	0.317
p-value	0.0001	0.0001	0.0001	0.0001	0.0001

$^{a-e}$Means with different superscripts in a column differ significantly (P < 0.05). *SEM =Standard Error of the mean; NC = Negative control; SC = *Saccharomyces cerevisiae*; HSCAS = hydrated sodium calcium aluminosilicate; PC = Positive Control; FCR= Feed conversion ratio.

(SRBC) using the microtitrehemagglutination method (Grasman 2010).

Antioxidant status and AFB1 residues of liver chicks

Malondialdehyde (MDA), glutathione (GSH), and glutathione S-transferase (GST) activity were determined in liver tissues (Habig et al., 1974). For AFB1 residue analy

Table 3: Effect of SC and/or HSCAS on some blood constituents in broiler chickens fed with aflatoxin contaminated feed.

Treatments	Serum constituents						Anti-SRBC titre (Log2)
	Total protein, g/dl	Albumin, g/dl	Chol. g/dl	AST, IU/L	ALT IU/L	Creat. mg/dl	
NC	7.58^{a}	4.99^{abc}	137.5^{a}	186.5^{d}	65.3^{cd}	0.45^{b}	5.22^{ab}
NC +SC	7.62^{a}	5.15^{ab}	139.0^{a}	185.0^{d}	63.4^{cd}	0.44^{b}	6.00^{a}
NC +HSCAS	7.52^{a}	5.32^{a}	137.0^{a}	188.0^{d}	65.0^{cd}	0.46^{b}	5.30^{ab}
NC +SC+HSCAS	7.66^{a}	5.20^{ab}	136.8^{a}	185.5^{d}	62.5^{d}	0.45^{b}	5.51^{ab}
PC	5.25^{c}	3.66^{d}	119.8^{b}	362.5^{a}	115.6^{a}	1.00^{a}	3.65^{c}
PC +SC	6.39^{b}	4.75^{bc}	133.5^{a}	228.6^{b}	78.0^{b}	0.58^{b}	4.86^{a}
PC +HSCAS	6.45^{b}	4.80^{bc}	131.5^{a}	230.8^{b}	75.6^{b}	0.50^{b}	4.50^{bc}
PC +SC+HSCAS	6.66^{b}	4.55^{c}	130.8^{a}	200.0^{c}	66.8^{c}	0.48^{b}	4.90^{ab}
SEM	0.173	0.138	6.056	11.758	3.476	0.039	0.188
p-value	0.0001	0.0001	0.0001	0.0001	0.0001	0.0001	0.011

$^{a-c}$Means with different superscripts in a column differ significantly (P < 0.05). *SEM = Standard Error of the mean; NC = Negative control; SC = *Saccharomyces cerevisiae*; HSCAS = hydrated sodium calcium aluminosilicate; PC = Positive Control; Chol= Cholesterol; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; Creat= Creatinine; SRBC=Sheep Red Blood Cells.

Table 4: Effect of SC and/or HSCAS on liver weight, hepatic MDA, glutathione levels and glutathione S- transferase activity and AFB1 residue in liver in broiler chickens fed with aflatoxin contaminated feed.

Treatments	Liver weight %	MDA (µM / g wet liver)	GSH (µM / g wet liver)	GST (mmol/min/g wet liver)	AFB1 residue in liver (µg/kg)
NC	2.09^{bc}	154.5^{bcd}	5.33^{b}	14.39^{ab}	ND
NC +SC	2.10^{bc}	145.3^{cd}	6.36^{a}	16.52^{a}	ND
NC +HSCAS	2.12^{bc}	155.5^{bcd}	5.25^{b}	14.44^{a}	ND
NC +SC+HSCAS	2.00^{c}	138.8^{d}	6.00^{a}	16.25^{a}	ND
PC	3.16^{a}	288.8^{a}	2.02^{d}	5.33^{d}	2.55
PC +SC	2.50^{b}	178.2^{bc}	5.00^{b}	13.25^{b}	0.140
PC +HSCAS	2.38^{bc}	182.5^{b}	4.28^{c}	10.68^{c}	ND
PC +SC+HSCAS	2.30^{bc}	168.8^{bcd}	5.11^{b}	16.55^{a}	ND
SEM	0.082	9.817	0.263	0.797	0.177
p-value	0.0001	0.0001	0.0001	0.0001	0.0001

$^{a-c}$Means with different superscripts in a column differ significantly (P < 0.05). *SEM = Standard Error of the mean; NC = Negative control; SC = *Saccharomyces cerevisiae*; HSCAS = hydrated sodium calcium aluminosilicate; PC = Positive Control; MDA= malondialdehyde; GSH= glutathione; GST= glutathione S- transferase; ND: not detected (determination limit of the analytical method: 0.05 µg/kg for aflatoxin B1).

sis, five liver samples from each group were maintained at -20˚C. Analysis of AFB1 residues was performed according to Tavcar-Kalcher et al. (2007).

Statistical analysis

Results from all response variables have been subjected to one-way variance analysis (SAS, 2004). Using Duncan's Multiple Range Test (Duncan, 1955), mean of variables with a significant F-test (P≤0.05) were compared.

RESULTS

Growth performance

The effects of AFB1 and dietary inclusion of SC and HSCAS on growth performance are presented in Table 2. The PC group showed significantly (P <0.05) higher FCR and lower BW and FI than the NC group at 42 day-old. Dietary inclusion of SC and HSCAS could significantly (P<0.05) alleviate the adverse effects of AFB1 on performance during the whole experimental period. AFB1 caused significantly (P<0.05) decreased dressing % while the addition of either HSCAS or SC improved dressing percentage compared to AFB1-diet.

Blood parameters

Data in Table 3 shows significant (P<0.05) difference in serum values between treatments. The PC group showed lower serum concentrations of total protein, albumin, cholesterol, and antibody titer against SRBC while there were higher serum AST, ALT activities and creatinine than the NC group. The addition of SC and/or HSCAS could alleviate the adverse effects of aflatoxin on these blood parameters. However, unlike the NC group, non-significant differences were found among groups containing additives in the absence AFB1.

Liver status

The relative weight of the liver, liver antioxidant status, and AFB1 residues in the liver are shown in Table 4. Feeding an AFB1-contaminated diet without SC and HSCAS caused significant (P<0.05) increases in the relative weight of liver and MDA levels, whereas glutathione levels and GST activities were significantly (P<0.05) decreased. The addition of SC and/or HSCAS to the diet containing AFB1 alleviated the adverse effects of AFB1 on the liver status.

There were no detectable residues of AFB1 in the liver of chicks fed on diets consuming the uncontaminated diets. However, there was residual of AFB1 was found in the liver of the chickens fed the AFB1 alone in the diet. However, The supplementation of SC to AFB1 diets (1 mg/kg) resulted in partially protection for liver of AFB1 residue. While, the added HSCAS alone or with SC to AFB1-diets (1 mg/kg) resulted in entirely protection for liver of AFB1 residue.

DISCUSSION

AFB1 is the most predominant and essential fungal toxin, and it is associated to the initiation of possible problems in livestock feeding (Nilipour, 2002). Severe occurrences of AFB1 in poultry cause substantial economic losses in health and production (Hussein and Brasel, 2001; Attia et al., 2013). Because there are no applied techniques to eliminate mycotoxin contamination in feeds (Anderson, 1983), binders for example HSCAS have been recommended to isolate aflatoxins, bind with them, and structure a more stable complex that inhibits the toxins from being absorbed in the animal's digestive system (Attia et al., 2013; 2016). Controlling the influences of AFB1 on animals and edible animal products is likewise significant (Pasha et al., 2007). There are a few disadvantages to take into account, despite the fact that the clays showed have been confirmed to be beneficial in depressing aflatoxicosis in a diversity of animal species. They cannot bind other mycotoxins, but they can adsorb vitamins and minerals. It is likewise significant to take into account the risk of natural clays to be contaminated with dioxins should also be measured (Jouany, 2007).

In Japanese quail, Yildiz et al. (2004) presented the protective influences of yeast culture when added to contaminated diets with aflatoxin. There are numerous theories as to why yeast can diminish the influences of aflatoxicosis. Raju and Devegowda (2000) and Yildiz et al. (2004) reported on *in vivo* and *in vitro* research that demonstrated the esterified glucomannan compounds in Saccharomyces cerevisiae's cell wall have a high affinity for aflatoxins (Dalvi, and McGowan, 1984). By chelating aflatoxin that is carried and eliminated through the digestive system, Saccharomyces cerevisiae may lessen the severity of aflatoxicosis (Attia et al., 2013; 2016). Additionally, it's been demonstrated that including yeast in a diet contaminated with aflatoxin has been shown to improvement enzymes that change aflatoxin influences by rising bio-transformation, therefore diminshingthe intensity of the adverse influences (Dalvi, and McGowan, 1984).

These findings are agree with previous studies on the influences of AFB1 on the performance reducing (Denli et al., 2009). The diminution in growth after feeding aflatoxin could be attributed to diminished protein synthesis, as noted by Verma et al. (2002); elevated fat excretion in droppings, inadequate food absorption, decreased pancreatic digesting enzyme production, and decreased appetite, as reported by Osborne and Hamilton (1981).

Chicks fed an aflatoxin B1 diet died at a superior rate than the control group. Our findings suggest that aflatoxicosis-induced liver failure, anaemia, and reduced immunity may be accountable for chick mortalities. The values of mortality determined in this study is consistent with Manegar et al. (2010) and Bhaskar et al. (2003) who stated 21.66 and 23.33 % mortality with 600 and 200 g/kg AF, respectively. Nevertheless, when HSCAS or *S. cerevisiae* were introduced to an AFB1-contaminated diet, the mortality rate was inferior than when an AFB1-contaminated diet was consumed alone. This might be because antimycotoxins have the potential to bind aflatoxin in the gut, which lowers the absorption and bioavailability of the toxin (Galvano et al., 2001).

Aflatoxin's influence on diminished protein synthesis (Yang et al., 2012), which results in less muscle mass formation, is related to the deteriorate in dressing percentage caused by AF. The growth performance parameters (groups 6 and 7, respectively) were improved by dietary supplementation with HSCAS or SC alone at a significant (P <0.05) level. On the other hand, the best efficient (P <0.05) performance was exhibited by the chickens in group 8 that fed on HSCAS plus SC. In this study, SC and HSCAS added to uncontaminated diets improved

growth performance metrics in compared to NC. It might be argued that HSCAS and SC assurance higher performance under abnormal conditions. According to studies, SC improves performance by diminishing the likelihood of diseases by preventing pathogenic bacteria from colonising the gut lining, slowing their growth, and lowering toxins and intestinal pathogens (Benites et al., 2007). Furthermore, SC improves performance by lengthening villi, which improves absorption and, consequently, the birds' energy-to-protein ratio (Salianeh et al., 2011).

According to previous findings of aflatoxicosis, the reduced levels of total protein and albumin show that AFB1 is toxic in hepatic and renal organs (Tejada-Castaneda et al., 2008). Diminution amino acid transport and diminished mRNA transcription caused by DNA inhibition may be accountable for the reduction in total serum protein in the aflatoxin-fed group (Kubena et al., 1993a). As per Manning and Wyatt (1984), cholesterol is mainly produced in the liver, and aflatoxin has been found to competitively inhibit mitochondria transport carrier proteins, which may reduce the energy available for cholesterol synthesis. A increase in the liver enzyme profile in aflatoxicated animals is most likely caused by liver tissue destruction, altered hepatocyte membrane integrity, and blood enzyme leakage (Duncan and Prasse, 1986). The results are in line with those of Denli et al. (2009), who discovered an increase in AST and ALT activity following feeding a ration contaminated with different concentrations of aflatoxin. Vanzytveld et al. (1970) detected toxin translocation into the liver When aflatoxin was provided directly into the crop at extremely high levels. The elevated serum level of creatinine in this investigation demonstrated aflatoxin's nephrotoxicity. An early sign of aflatoxicosis was this rise in creatinine levels (Kilany et al., 2020). HSCAS and SC substantively ($p<0.05$) improved serum biochemical items, signifying that they were successful in warding off AFB1 by inhibiting its adverse influence by raising serum conesituents (Attia et al., 2013; 2016).

Abdel-Wahhab et al. (1998) positively studied the adjustment of liver enzymes after usage of an aflatoxin-contaminated ration with HSCAS; though, SC in combination with HSCAS exhibited superior efficiency than the separate form, suggesting that both have a synergistic interaction influence on liver enzymes. Adding yeast to the diet decreased lipid concentrations and liver enzymes by lowering alanine aminotransferase and alkaline phosphatase activities, which directly or indirectly reflected a healthier liver state in the birds, according to Saadia and Nagla (2010) and Attia et al. (2013; 2016). These results suggest significant mycotoxin adsorption. In contrast to modified mannan oligosaccharides supplied from *S. cerevisiae* cells, which bind up to 95% of mycotoxins in a chicken diet, SC bound up to 77% of mycotoxins (Raju and Devegowda 2000).

AFB1 reduced immunological reaction (PC vs NC). AFB1 causes teratogenicity, hepatotoxicity, and nephrotoxicity in birds (Abidin et al., 2016). Birds fed mycotoxins have lower antibody titers (Khan et al., 2014). Due to the Fabricius bursa's possible regression, demonstrating its immunosuppressive effects (Dafalla et al., 1987). According to Ibrahim et al. (2000), the immune response in this trial improved once binders like HSCAS were added . They found that adding sodium bentonite binder lessened the negative effects of AF on phagocytosis and HI-titer in chicks that had received the NDV vaccine.

The findings demonstrated that compared to the NC (group 1) and other treated groups, the relative weight of the liver in the AFB1-fed control positive group (5) was significantly higher ($p<0.05$). These findings support those of Ortatatli et al. (2005) who found that aflatoxins are hepatotoxic and nephrotoxic based on an increase in the absolute and relative weights of the liver, kidney, and gizzard in birds fed aflatoxin-containing meals. Most aflatoxins are bioactivated in the liver to the reactive 8, 9-epoxide form known to binds DNA and proteins, damages liver structures, and increases liver weight (Pasha et al., 2007). According to Gowda et al. (2008), broiler chickens' liver weight can decrease due to the addition of HSCAS to the diet. Additionally, Sehu et al. (2007) proved microscopically that including HSCAS in the quail diet decreased the amount of fat deposited in the liver as a result of aflatoxin, hence reducing liver weight. Regarding the protection offered by SC, Khadem et al. (2012) found that included SC in a broiler chick's diet decreased the liver's relative weight. The findings of raised MDA and diminished GST and GSH values in the liver of PC birds displayed that AF (1 mg/kg) might elevate the oxidative stress in broilers fed contaminated diet with AF. These findings support those of Liu et al. (2016), who found MDA content was much higher and antioxidant enzyme activity and GSH level were significantly lower in the liver and spleen for broilers fed contaminated diets with AFB1 than in the control group. In the current study, there were reduction liver MDA values and raising of liver GSH and GST values in chicks fed contaminated diets plus HSCAS and/or SC, indicating that SC and HSCAS have clearly antioxidant activity in birds. HSCAS increases antioxidant activities by decreasing oxidative stress during aflatoxicosis, according to similar findings (Chen et al., 2014).

Due to the accretion of aflatoxin in the edible pieces of poultry, residual AFB1 in the liver not only damagingly influences the performance and health of broiler chickens but also damagingly influences the health of consumers of

broiler products. Because of this, it's significant to check the quality of chicken products and inspect afatoxin residues in numerous poultry tissues while also considering the general public welfare and safety (Salem et al., 2018). The result of the present study, which observed there was at aflatoxin residue in a group fed a contaminated diets without additives, is uniform with the findings of Hussain et al. (2016). Glucan-based binders that stick to mycotoxins during digestion and stop them from being absorbed through the digestive tract have been proven to reduce their adverse effects (Chowdhury et al., 2005). Thus, aflatoxin's interaction with the glucan in the Saccharomyces cerevisiae cell wall caused the aflatoxin residue in the group fed the contaminated diet with SC to decrease as a result (Wu et al., 2009). These findings are reinforced by those of Yiannikouris et al. (2021), who explained the adsorbent activity of the Saccharomyces crevisiae cell wall against aflatoxin in rats. Furthermore, Saccharomyces crevise's adsorbent properties were stated by Oğuz et al. (2018). According to Phillips (1999), the protective action of HSCAS is the result of the substance's quick binding to aflatoxin in chicken gastrointestinal tracts, which prevents its normal distribution to the liver and absorption. Our findings demonstrated that HSCAS, either alone or in combination with SC, was beneficial in preventing aflatoxin B1 accumulation, particularly in the liver.

CONCLUSION

This study aimed to see how effective HSCAS and SC were at promoting growth and immunity in broiler chicks fed uncontaminated and AFB1-contaminated diets. Aflatoxin-contaminated feed adversely impacted growth performance, immunity, and blood parameters. On the other hand, the inclusion of HSCAS and SC in the diet especially together may help mitigate the harmful effects of AFB1 on these parameters.

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CONFLICT OF INTEREST

There is no conflict of interest between the authors of this manuscript.

NOVELTY STATEMENT

This research work was able to determine that the adding hydrated sodium calcium aluminosilicate and Saccharomyces cerevisiae together to contaminated diets with aflatoxin B1 their a synergistic effect for alleviating the adverse effects of aflatoxin compared to hydrated sodium calcium aluminosilicate or Saccharomyces cerevisiae alone.

AUTHORS CONTRIBUTION

Reda Hassan participated in designing the experimental plan and writing the manuscript. Bahaa Abou-shehema and El-Sayed Abu El-Hassan participated in feeding the birds and collecting samples. Mahmoud El-Gbaly and Hanaa Basuony, participated in analyzing the samples, evaluating the results. Sherif Zayed and Micheal Gorgy participated in conducting the statistical analysis. Ebtehal Hassan and Shama Morsy participated in publication of this paper. All authors contributed to reviewing this manuscript.

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