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Mitigating Aflatoxin Exposure Risk Using *Saccharomyces cerevisiae*: A Probiotic Eco-Friendly Approach to Enhance Biochemical Parameters and Histological Integrity in Albino Mice

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Abstract

AFLATOXINS represent a major global public health concern, contaminating 25% of food crops worldwide. Human exposure to these toxins either occurs directly through the consumption of contaminated foods or indirectly via animal products derived from livestock that have ingested aflatoxin-contaminated feed. This study aimed to evaluate the protective efficacy of probiotic baker's yeast (Saccharomyces cerevisiae) as a natural and eco-friendly agent against aflatoxicosis in female albino mice under in vivo conditions. Five experimental groups were used, each receiving either of two aflatoxin concentrations (0.81 ng/ml from Aspergillus flavus isolate Z/N/22 and 0.66 ng/ml from A. parasiticus isolate A/N/94). The results showed that Mice exposed to aflatoxins alone exhibited signs of toxicity, including reduced weight gain, increased liver and kidney weights, elevated biochemical markers (ALT, AST, alkaline phosphatase, creatinine, and urea), and histopathological alterations. In contrast, co-treatment with S. cerevisiae significantly mitigated these effects, with improved biochemical profiles and preserved tissue morphology comparable to the control group. These results concluded that Probiotic yeast demonstrates significant potential as a natural, non-toxic, and environmentally sustainable approach for mitigating the toxic effects of aflatoxins. Its application represents a promising strategy for improving food and feed safety. Regular consumption of probiotic-rich fermented foods (e.g., yogurt or cultured dairy beverages) or the use of probiotic supplements in capsule or powder form may offer effective and practical means of reducing aflatoxin exposure. Further research and integration into public health and agricultural practices is recommended to support its broader implementation.

Keywords: Mice, *Aspergillus* spp., Aflatoxins, Probiotics, *Saccharomyces cerevisiae*.

Introduction

Among the approximately 300 known mycotoxins, aflatoxin (AF) is uniquely regulated by the U.S. Food and Drug Administration due to its significant health risks. Aflatoxin contamination is one of the most pressing challenges in food safety, affecting an estimated 25% of global food crops [1]. In developing countries, nearly 5 million people are potentially exposed to aflatoxins [2]. These toxins have been associated with severe adverse effects on multiple organ systems, including the liver, kidneys, brain, lungs, gastrointestinal tract, and cardiovascular system [3].

Aflatoxins are potent secondary metabolites naturally synthesized by various *Aspergillus* species, particularly *A. flavus* and *A. parasiticus*. Multiple strategies have been developed to reduce mycotoxin contamination, including physical, chemical, and biological approaches [4]. Among these, biological detoxification using microorganisms such as yeast has gained increasing attention due to its eco-friendly nature. Yeast can produce antimicrobial compounds, grow rapidly, and are capable of degrading or

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binding toxins. Fermentation is considered a cost-effective and practical method for reducing mycotoxin levels in food, though the nature and toxicity of fermentation by-products must be carefully evaluated [5].

Probiotics represent a promising biological approach for controlling aflatoxin toxicity. These beneficial microorganisms can reduce the bioavailability of aflatoxins and limit their absorption in the gastrointestinal tract. Regular intake of probiotic-rich fermented foods (e.g., yogurt or dairy-based beverages), or probiotics in pharmaceutical forms (capsules, tablets, or powders), may help mitigate the toxic effects of aflatoxins [6]. Beyond detoxification, probiotics have demonstrated general health-promoting effects, making them valuable agents in food and feed safety [7].

This study aimed to investigate the effects of aflatoxins on the health, growth performance (e.g., body and organ weights), and biochemical and histopathological profiles of female albino mice. Additionally, it evaluated the efficacy of the probiotic yeast *Saccharomyces cerevisiae* in counteracting the adverse effects of aflatoxicosis under in vivo conditions. Given its availability, low cost, and environmental safety, *S. cerevisiae* was selected as a candidate for biological mitigation of aflatoxin toxicity.

Material and Methods

Preparation of Yeast Extract Sucrose (YES) Medium

A medium containing 2% yeast extract and 15% sucrose was prepared by dissolving the components in distilled water and dispensing the solution into 250 ml conical flasks. The medium was then sterilized by autoclaving at 126°C and 1.5 psi for 20 minutes, following the protocol described by [8].

Preparation of Saccharomyces cerevisiae Strain

Commercially available lyophilized probiotic baker's yeast (*S. cerevisiae*) was purchased from the local market and stored at 4°C. Under sterile conditions in a laminar flow biosafety cabinet, the yeast powder was reactivated in sterilized YES medium and incubated at 23 ± 2°C for 72 hours. The resulting yeast suspension was diluted with sterile distilled water to achieve a final cell concentration of 1.0×10^9 cells/mL. Each treatment was prepared in triplicate, as described in previous studies [9,10].

Preparation of Aflatoxins

Aflatoxins were produced by inoculating liquid YES medium with a 1 mL suspension containing *S. cerevisiae* (1.0×10^9 cells/mL) and/or 1 mL of fungal spore suspensions (10^5 CFU/mL) of either *Aspergillus flavus* isolate Z/N/22 or *A. parasiticus* isolate A/N/94. Each treatment was performed in triplicate and incubated at 28 ± 2°C for 15 days. After incubation, fungal mats were disrupted using a sterile glass rod, and the cultures were filtered. The filtrate, referred to as the "mother solution," contained the total aflatoxins and was stored at 4°C for further use.The concentration of aflatoxins was determined using high-performance liquid chromatography (HPLC) as described by [11]. *A. flavus* isolate Z/N/22 produced 0.81 ng/mL of total aflatoxins, whereas *A. parasiticus* isolate A/N/94 produced 0.66 ng/mL.

Experimental Design

Six-week-old female albino mice were obtained from the Animal House Laboratory, National Research Centre, Dokki, Cairo, Egypt. The animals were housed under controlled conditions (temperature: 25°C, relative humidity: 50–55%, and a 12-hour light/dark cycle) in a contamination-free environment [10].

Experimental Protocol and Grouping

The mice were divided into five groups, with each group consisting of three mice, as outlined in Table 1. Mice in the treatment groups received daily oral doses of 2 mL aflatoxin solution for three weeks. Body weights were measured weekly, and mortality was recorded. At the end of the experiment, liver and kidney weights were also recorded.

Blood Collection

At the end of the 3-week experimental period (Day 21), all mice were euthanized using ether anesthesia and then sacrificed by decapitation (guillotine method) to ensure painless termination. Blood samples were collected from the retro-orbital venous plexus into anticoagulant tubes and stored at –20°C until analysis. These samples were used for serum biochemical assessments of liver and kidney function. Additionally, liver and kidney tissues were harvested for histopathological examination following the procedures of [12].

Biochemical Analysis

All biochemical determinations were performed using commercial kits according to the manufacturer's instructions, unless stated otherwise as follows:

Liver Function Tests:

Determination of Transaminase Activity:

The activities of serum alanine aminotransferase (ALT) and aspartate aminotransferase (AST) measured calorimetrically using a spectrophotometer, following the methods outlined in [13, 14]. *Serum Albumin Level*: Serum albumin

levels (ALP) were determined calorimetrically by the PCG method using a spectrophotometer, according to the procedures described in [12, 15, and 16].

Kidney Function Analysis

Serum uric acid concentrations were quantified using an enzymatic colorimetric assay, as previously described in references [12, 17]. Serum creatinine levels were assessed using commercially available diagnostic kits (San Antonio, Texas, USA), following the procedures established by [18].

Histopathological Evaluation

For histological analysis, tissue samples were fixed in 10% neutral buffered formalin and subsequently embedded in paraffin. The samples were dehydrated through a graded ethanol series and processed for paraffin embedding, as per the method detailed in [19]. Small sections from the liver and kidneys of both control and treated animals were cut into 5 µm slices, stained with hematoxylin and eosin (H&E), and examined microscopically at the Histopathology Department, National Research Centre, Dokki, Egypt. Hematoxylin and eosin (H&E) staining was used to distinguish cytoplasmic and nuclear structures, enabling clear visualization of tissue architecture and any pathological alterations [20]. All stained tissue sections (5 µm thick) from control and treated groups were mounted on glass slides using conventional histopathological protocols. Examination was performed using a light microscope, and representative images were captured and edited using Adobe Photoshop version 8.0 [21].

Results and Discussion

Using a probiotic baker yeast (Saccharomyces cerevisiae) for enhancement mice health received aflatoxins:

No mortality was recorded in any test group. Control group (free aflatoxins) appeared normal mice and good health. Aflatoxins alone group was found to have poor health and abnormal symptoms. These mice show abnormal behavior, slow eating, slow moving and loss of functional movement when compared with control. Whereas mice received two concentrates of aflatoxins (0.81 ng/ml (AFs) produced by *A. flavus* isolate No. Z/N/ 22 and 0.66 ng/ml (AFs) produced by *A. parasiticus* isolate No. A/N/ 94) plus a probiotic yeast (*S. cerevisiae*) group were enhanced (healthy) and appear comparable to thecontrol. A probiotic yeast (*S. cerevisiae*) had to be positively enhancement of body weight gain. So, it was worthy to mention that application of bio-agent a probiotic yeast (*S. cerevisiae*) was found to effective in protected mice against aflatoxins (AFs). These results were in agreement with [22] who found that, birds received aflatoxin B_1, began to show symptoms of toxicity after 2 weeks. Aflatoxin B_1 alone was found to reduce feed efficiency and poor health can cause by an imbalance of nutrients. Abnormalities symptoms were found with all ducklings received aflatoxin B_1. These birds showing slow eating, leg paralysis, slow moving as result of loss functional movement, inflammatory edema of the eyelids (affect eyes), hair loss, and changed in the color when compared with control. [23,24] reported that, Probiotics are live microorganisms that yield health benefits to host when applied by enough amounts. [25,26] stated that positive *in vitro* results match the results of *in vivo* studies, where yeast cell walls (YCW) were able to minimize the toxicity of aflatoxins, demonstrating the beneficial effects of its addition to broiler feed. Moreover, [23] reported that the management of probiotics had positively affected the growth performance of chickens. [27] suggested that, based on the results, the investigated yeast (*S. cerevisiae*) strains show desirable probiotic characteristics and AFB adsorption capacity. Thus, the inclusion of these strains in tambaqui diets may improve health aspects and reduce the amount of AFB occasionally ingested through contaminated feed.

Improvement in Weight Gain

Table (2) shows the impact of aflatoxins (AFs) and detoxification with the bioagent probiotic yeast (S. cerevisiae) on body weight gain (g). According to the data, mice's body weight increased in every studied group with the exception of the aflatoxins (AFs) groups. Body weight gain was larger in control group 5 (Free AFs), which went from 27.0g at the beginning of the experiment to 28.5g after 7 days, 30.0g after 14 days, and 31.0g after 21 days. However, the treated mice in groups 1 and 3 weighed less than the untreated control group. Reduced food intake and/or absorption may be the cause of the total body weight deficit brought on by AF usage. Constant decline. Mice's body weight growth steadily declines as the duration of the session increases. Thus, mice in group 1 which were given 0.81 ng/ml of aflatoxins (AFs) made by A. flavus isolate No. Z/N/22 had the lowest body weights, dropping from 28.5 g at the beginning of the experiment and after one week to 28.0 g after two weeks and 27.5 g after three weeks, with reductions of 5.3 and 11.3% at the conclusion of the trial. According to the results, mice given AFs along with the bioagent S. cerevisiae (yeast) group 2 showed an improvement in all body weight increases and appeared to be on level with the control group (free AFs). As the duration of time increases, the health of the mice continues to improve.

Mice body weights group 2 were increased from 30.5g at zero time to 31.0g after 1week, 32.0 g after

2weeks and 33.0g after 3weeks equal 11.5, 8.1, 6.3 and 6.1% increasing respectively when received aflatoxins (AFs) plus a probiotic yeast (S. cerevisiae). Whereas mice group 3 received 0.66 ng/ml (AFs) produced by A. parasiticus isolate No. A/N/ 94) had less body weights gain which decreased from 27.5g at zero time and 1week to 26.5 after 2weeks and 26.0g after 3weeks equal 1.82, 3.51, 11.67 and 16.13% reduction at the end of experiment. While the obtained data indicated that mice receiving the same AFs plus bio-agent, a probiotic yeast (S. cerevisiae) group 4 were found to enhance all body weights gain and appear comparable to the control (Free AFs). Continuous enhanced mice health with increasing the period of time. Mice body weights group 4 were increased from 30.5g at zero time to 31.5g after 1week, 32.5 g after 2weeks and 33.0g after 3weeks with 11.5, 9.5, 7.69 and 6.1% increasing respectively when received aflatoxins (AFs) plus a probiotic yeast (S. cerevisiae). It was worth mentioning that mice received aflatoxins (AFs) plus a probiotic yeast (S. cerevisiae) had to be positively enhance body weight gain in the same period time.

The current results align with previous studies indicating that aflatoxin exposure negatively impacts growth performance. For instance, [28] found that rats consuming a diet contaminated with aflatoxins showed notably lower feed intake and body weight compared to healthy controls. Similarly, [2] reported that ducklings receiving a diet containing 0.018 ng/mL of aflatoxin B1 had significantly reduced body weight ($P < 0.01$ and $P < 0.05$) relative to untreated counterparts. In agreement, [29] demonstrated that rats administered aflatoxins at 0.7 mg/kg of diet exhibited the lowest body weight among all groups.

In contrast, positive outcomes were observed in animals receiving probiotic supplementation. According to [30], dietary inclusion of *Saccharomyces cerevisiae* in chicks led to marked improvements in body weight gain, feed consumption, and feed conversion efficiency. These findings are consistent with [31], who noted that probiotics enhanced both body weight gain (BWG) and feed conversion ratio (FCR) in broiler diets. Supporting this, another investigation reported significant increases in BWG and FCR in broilers supplemented with probiotics. Similarly, [23] observed that probiotic-fed birds outperformed the negative control group, showing statistically higher ($P < 0.05$) BWG and more efficient FCR. As explained by [32], such improvements may be attributed to the probiotics' ability to produce antimicrobial substances and enhance nutrient utilization in the digestive system. Top of Form

According to [23], the use of probiotics has shown beneficial effects on the growth performance of poultry. Probiotics interact with the host by enhancing immune responses, improving gut structure and metabolic activity, and consequently reducing the likelihood of infections caused by opportunistic pathogens. Similarly, [10] observed that throughout the experimental period, birds fed a diet containing aflatoxin B1 (AFB1) exhibited significantly lower feed intake and daily weight gain ($P < 0.05$).

Probiotics may function as bio-detoxifying agents, exerting antioxidant properties through the stimulation of enzyme production. This mechanism can support weight gain by improving protein metabolism as well as the absorption of essential vitamins and minerals. The inclusion of such detoxifying agents in the diet positively influences the gastrointestinal tract by enhancing digestive enzyme production, ultimately leading to better digestion and increased weight gain.

Consistent with these findings, [31] reported that incorporating probiotics into broiler diets improved both body weight gain (BWG) and feed conversion ratio (FCR). Other studies also confirmed that probiotic supplementation significantly enhanced BWG and FCR in broilers. For instance, [23] demonstrated that broilers fed probiotic-enriched diets achieved significantly higher ($P < 0.05$) BWG and more efficient FCR when compared to those on non-supplemented diets.

Furthermore, yeast and yeast-derived bacterial cultures have been recognized for their role in the fermentation process, which contributes to the detoxification of mycotoxins such as aflatoxins, including ochratoxin A. Recent findings by [23] indicated that certain wine yeast strains of *Saccharomyces* can be applied effectively to reduce ochratoxin A levels in grape juice. In addition, specific components of the yeast cell wall play a crucial role in binding aflatoxins, making *S. cerevisiae* a promising candidate for aflatoxin mitigation.

Effect of Probiotic Yeast (Saccharomyces cerevisiae) on Organ Weights:

At the end of the experimental period, all liver and kidney samples were collected and weighed. The results indicated that the control group (Group 5), which was not exposed to aflatoxins (AFs), maintained normal liver and kidney weights. In contrast, groups exposed solely to aflatoxins (Groups 1 and 3) exhibited noticeable organ enlargement and increased weights compared to the control group. However, the groups treated with both aflatoxins and the probiotic yeast *S. cerevisiae* (Groups 2 and 4)

showed improvements in organ weights, with values that closely resembled those of the control group, as summarized in Table (3).

Specifically, liver weight in Group 1 (mice treated with 0.81 ng/mL of AFs produced by *Aspergillus flavus* isolate Z/N/22) increased from 1.57 g in the control group to 2.35 g—a 33.28% rise. In Group 3 (mice treated with 0.66 ng/mL AFs from *A. parasiticus* isolate A/N/94), liver weight rose to 1.65 g, a 5.14% increase over the control. Upon administering *S. cerevisiae*, liver weights decreased from 2.35 g to 1.94 g in Group 2, and from 1.65 g to 1.55 g in Group 4, showing values equivalent to the control.

Similarly, kidney weight was also elevated due to aflatoxin exposure. In Group 1, the kidney weight increased from 0.30 g (control) to 0.40 g, representing a 25.56% rise. However, probiotic treatment in Group 2 reduced kidney weight to 0.36 g—a 17.36% decrease—bringing it closer to the control value. In Group 3, kidney weight increased to 0.35 g (14.29% increase) but decreased to 0.34 g in Group 4 after *S. cerevisiae* supplementation, indicating an 11.24% reduction. These observations suggest that co-administration of *S. cerevisiae* helped mitigate aflatoxin-induced organ enlargement, restoring liver and kidney weights to levels comparable to unexposed mice.

These findings are supported by [33], who observed that feeding AFB1 alone significantly increased the relative weights of the liver, kidneys, gizzard, and spleen in broilers. Similarly, [34] reported that an average dietary AF concentration of 0.95 mg/kg induced metabolic alterations in birds, leading to the enlargement of the liver, kidneys, and spleen, along with atrophy of the bursa of Fabricius, thymus, and testes. High doses of AFs were also associated with fat accumulation in hepatic cells, making the liver appear enlarged, yellow, and fragile.

Consistent with these results, [35] found that supplementing aflatoxin-contaminated diets with *S. cerevisiae* (2 g/kg) significantly reduced the negative effects of AFs on body weight gain, feed consumption, egg production, egg weight, and feed efficiency in quail. Additionally, [23] emphasized the role of probiotics in correcting liver enzyme disturbances, which are often elevated during aflatoxin-induced hepatotoxicity. Elevated levels of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were linked to liver damage; however, probiotic supplementation led to histological improvement and reduced fibrotic tissue formation. The reduction in serum ALT and AST levels observed in probiotic-treated animals supports the liver-protective effect of probiotics. Furthermore, [35, 36] reported that broiler chicks consuming diets containing 1.0 mg AFB1/kg showed reduced feed intake, suppressed body weight gain, and increased liver weight, confirming the hepatotoxic effects of aflatoxins and the mitigating influence of *S. cerevisiae*.

Aflatoxins (AFs) are known to exert toxic effects on several organs and systems in the body, including the liver, kidneys, brain, lungs, gastrointestinal tract, and cardiovascular system. Inhibiting fungal growth with antifungal agents may also influence the biosynthesis of AFs. In this study, the probiotic yeast *Saccharomyces cerevisiae* was tested for its ability to protect the liver from aflatoxin-induced toxicity using mice as a sensitive animal model.

As shown in Table 4, the control group exhibited normal serum biochemical values. Exposure to aflatoxins led to elevated serum levels of liver enzymes, namely alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP), indicating hepatic damage. However, mice co-treated with aflatoxins and the probiotic *S. cerevisiae* exhibited a gradual reduction in these enzyme levels over time.

By the end of the experiment (day 21), the control group (G5) maintained normal enzyme levels: 38 U/L for ALT and 89 U/L for AST. In contrast, group G1, which received 0.81 ng/ml of AFs from *Aspergillus flavus* isolate Z/N/22, showed marked increases in ALT (88.00 U/L) and AST (236.00 U/L), representing 56.82% and 62.29% increases respectively compared to the control. Similarly, group G3, which received 0.66 ng/ml of AFs from *Aspergillus parasiticus* isolate A/N/94, showed elevated levels of ALT (78.00 U/L) and AST (215.50 U/L), with increases of 51.28% and 58.71%, respectively.

The ALP level also increased significantly, from 92.00 U/L in the control group to 174.00 U/L in G1 (47.13% increase) and 165.00 U/L in G3 (44.20% increase). Conversely, serum albumin levels decreased in response to aflatoxin exposure: from 3.75 g/dL in the control group to 2.40 g/dL (a 36.00% reduction) in G1 and 2.85 g/dL (24.00% reduction) in G3.

Notably, co-administration of *S. cerevisiae* with AFs in group G2 led to improved biochemical profiles, approaching those of the control. In G2, ALT, AST, and ALP levels decreased to 48.00, 141.00, and 120.00 U/L, reflecting reductions of 20.8%, 36.88%, and 23.30%, respectively, compared to G1 and G3. Albumin levels also improved, increasing from 2.40 and 2.85 g/dL in G1 and G3 to 3.05 g/dL in both G2 and G4, showing an 18.67% improvement.

These findings indicate that *S. cerevisiae* exerts a protective effect against aflatoxin-induced hepatotoxicity, reflected in the normalization of serum biochemical markers. The results align with previous studies. For instance, [37] reported significantly increased ALT, AST, creatinine, and uric acid levels in animals fed AF-contaminated diets ($p < 0.01$). According to [28], elevated ALT, AST, and ALP are reliable indicators of liver injury, including cirrhosis and necrosis.

Similar findings were documented by [38], where aflatoxin B1 (AFB1) exposure increased hepatic lipid peroxidation, contributing to liver cell damage. Elevated levels of ALT, AST, and ALP were consistently observed in aflatoxin-exposed mice and rats, as also reported by [22] and [29], who noted significant changes in various serum biochemical parameters and albumin levels due to AF exposure.

Moreover, [39] observed enzyme activity increases of 42%, 43%, and 44% for AST, ALT, and ALP, respectively, in animals fed mold-contaminated feed. This elevation was indicative of liver mycotoxicosis. In contrast, several studies have highlighted the protective role of *S. cerevisiae*. For example, [40] found that *S. cerevisiae* could bind over 60% of AFs, while [10] demonstrated that probiotics enhanced blood parameters, liver and kidney function, and animal weight gain ($p < 0.05$).

Additionally, the inclusion of *S. cerevisiae*, bentonite, or kaolin in AF-contaminated diets mitigated toxic effects and improved liver enzyme profiles. [12] reported variable effects of *S. cerevisiae* cell wall components on ALT and AST levels across different time points. Notably, ALP levels were not significantly affected in some studies, suggesting a selective response to probiotic treatment.

Further studies confirmed that probiotics, including *S. cerevisiae*, can reduce liver enzyme levels and inflammatory markers like TNF-α and IL-6, which are commonly elevated in chronic liver diseases. Probiotic intake was also linked to improved liver histology, reduced fibrosis, and decreased aflatoxin deposits in liver tissues.

In summary, *S. cerevisiae* supplementation showed a clear hepatoprotective effect, as evidenced by improved serum enzyme levels and enhanced liver function in aflatoxin-exposed mice.

Enhancement of Kidney Functions

The impact of probiotic yeast *Saccharomyces cerevisiae* on kidney function is presented in Table 5. Combined treatment with aflatoxins and *S. cerevisiae* proved effective in mitigating the elevation of kidney function biomarkers. The control group maintained normal values within physiological limits, while exposure to aflatoxins alone significantly elevated serum creatinine and urea levels in mice.

However, the groups receiving both aflatoxins and *S. cerevisiae* showed a progressive improvement in kidney function over time. At the end of the 21-day experiment, the control group (G5), which was not exposed to aflatoxins, displayed normal creatinine and urea levels of 0.60 mg/dL and 33.00 mg/dL, respectively.

In contrast, group G1, which was administered 0.81 ng/mL of aflatoxins produced by *Aspergillus flavus* isolate Z/N/22, showed a marked increase in creatinine (2.00 mg/dL) and urea (73.00 mg/dL), representing increases of 70.00% and 54.80%, respectively, compared to the control. Similarly, group G3, exposed to 0.66 ng/mL of aflatoxins from *Aspergillus parasiticus* isolate A/N/94, recorded creatinine and urea levels of 1.75 mg/dL and 72.50 mg/dL, reflecting 65.71% and 54.48% increases, respectively.

Meanwhile, co-treatment with *S. cerevisiae* led to substantial improvements. In group G2 (aflatoxins + *S. cerevisiae*), creatinine and urea levels were reduced to 1.65 mg/dL and 63.00 mg/dL, showing decreases of 63.64% and 47.60%, respectively, when compared to G1. Similarly, group G4 recorded values of 1.55 mg/dL for creatinine and 52.50 mg/dL for urea, corresponding to reductions of 61.29% and 37.14% compared to G3.

These results confirm the efficacy of *S. cerevisiae* in protecting kidney function from aflatoxin-induced toxicity. Mice treated with both aflatoxins and *S. cerevisiae* exhibited biochemical profiles similar to those of the control group. Additionally, mice fed aflatoxin-contaminated corn and supplemented with *S. cerevisiae* showed not only improved kidney function but also better body weight gain and normalized organ weights.

These findings are consistent with previous studies [41, 42], which identified *S. cerevisiae* as one of the most efficient microorganisms for detoxifying aflatoxin B1. Yeasts have shown a strong ability to adsorb mycotoxins in aqueous solutions, with *S. cerevisiae* showing effectiveness in binding AFB1. According to [37], *S. cerevisiae* supplementation also contributed to improved weight gain in animals. Furthermore, exposure to aflatoxins has been shown to significantly increase serum ALT, AST, uric acid, and creatinine levels, and cause severe histopathological damage in liver and kidney tissues.

However, when *S. cerevisiae* was administered prior to aflatoxin exposure, a marked improvement was observed in both serum biochemical markers and the structural integrity of liver and kidney

tissues. This supports the role of *S. cerevisiae* as a safe and effective biological agent in counteracting the toxic effects of aflatoxins.

In contrast, other studies [37, 43] found no significant changes in creatinine and urea levels when *S. cerevisiae* was added to the diets of rabbits or hens. For instance, dietary supplementation with *S. cerevisiae* extract (1 g/kg for 30 days) in hens did not significantly alter these kidney function markers. Nevertheless, overall evaluations suggest that dietary yeast does not pose any nephrotoxic risks and may, in fact, offer protective effects against renal damage.

In summary, *S. cerevisiae* demonstrates a clear protective effect on kidney function in mice exposed to aflatoxins, as evidenced by improved biochemical markers and preservation of kidney health.

Histopathological Examination of the Liver

The biochemical findings in this study were supported by histological analysis of liver tissues from experimental mice. No pathological alterations were observed in the liver tissues of the control group (G5), where microscopic examination revealed normal hepatic architecture. Hepatocytes were organized in cords radiating from the central vein, with clearly visible hepatic sinusoids and vesicular nuclei (Fig. 1).

In contrast, liver sections from the aflatoxin-treated groups (G1 and G3) showed marked histopathological changes. Group G1, which received 0.81 ng/mL of aflatoxins produced by *Aspergillus flavus*, exhibited significant structural damage, including disorganized hepatic architecture, degeneration of hepatocytes, central vein dilation and congestion, infiltration of mononuclear cells, and numerous pyknotic nuclei (Fig. 2).

Similarly, group G3, which was treated with 0.66 ng/mL of aflatoxins produced by *Aspergillus parasiticus*, showed severe liver damage. Histological changes included vacuolar degeneration of hepatocytes, congestion and dilation of the central vein, mononuclear cell infiltration both around and within focal areas, and widespread pyknotic nuclei (Fig. 3).

Conversely, groups treated with both aflatoxins and *Saccharomyces cerevisiae* (G2 and G4) demonstrated substantial histological improvement. In group G2 (0.81 ng/mL aflatoxins + yeast), liver sections showed a restored hepatic structure, with hepatocytes returning to their typical arrangement. Only mild central vein congestion, minimal localized inflammation, and activated Kupffer cells were observed (Fig. 4).

Group G4 (0.66 ng/mL aflatoxins + yeast) displayed nearly normal liver architecture, characterized by slight central vein congestion and mild inflammatory cell infiltration around the vein (Fig. 5).

These findings align with previous research by [44], which indicated that probiotics could slow the progression of fibrosis in a rat model of steatohepatitis. The variation in outcomes across different studies may be due to differences in probiotic formulations, including strains used and the presence of other additives.

Further supporting this, [23] reported that liver tissue damage due to hepatotoxicity was accompanied by elevated levels of liver enzymes such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST). Probiotic treatment not only improved liver histology but also led to a modest reduction in fibrous tissue formation.

The observed improvements may depend on the dosage and strain combination of probiotics, as well as the inclusion of prebiotics or other synergistic components. The histological improvements corresponded with significant decreases in serum ALT and AST levels, suggesting a hepatoprotective effect of probiotic administration.

This protective role may be attributed to the ability of probiotics to reduce the production or absorption of gut-derived lipopolysaccharides, thereby lowering systemic inflammation. Moreover, probiotics may enhance liver regeneration through increased expression of proliferation markers such as Ki-67, which is widely used to assess tissue regeneration.

In conclusion, the restoration of liver function in probiotic-treated mice is likely associated with hepatocyte regeneration, further confirming the protective and restorative role of *S. cerevisiae* against aflatoxin-induced liver damage.

Improvement of Kidney Tissues

The biochemical findings related to kidney function were further supported by histopathological examination of renal tissues. No structural abnormalities were observed in the kidneys of the control group (G5). Histological analysis revealed intact renal architecture, including well-formed Malpighian corpuscles containing glomeruli composed of capillary loops, clearly separated from the Bowman's capsule by a defined Bowman's space, and surrounded by normal urinary space. Renal tubules were lined with intact epithelial cells featuring normal vascular nuclei (Fig. 6).

In contrast, the groups exposed to aflatoxins (G1 and G3) displayed notable histopathological alterations in kidney tissue. In group G1, which received 0.81 ng/mL of aflatoxins derived from

Aspergillus flavus, microscopic analysis revealed glomerular atrophy, widened urinary spaces, inflammatory infiltration, degeneration of portions of the tubular epithelial cells, and signs of interstitial hemorrhage (Fig. 7).

Similarly, in group G3, exposed to 0.66 ng/mL of aflatoxins from *Aspergillus parasiticus*, kidney tissue exhibited glomerular atrophy, expansion of the urinary space, inflammatory cell infiltration, degenerative changes in the epithelial lining of renal tubules, and the presence of pyknotic nuclei, along with interstitial hemorrhage (Fig. 8).

However, co-treatment with *Saccharomyces cerevisiae* led to marked improvement in renal histology. In group G2 (0.81 ng/mL AFs + yeast), kidney sections displayed largely normal glomerular structure and urinary space, with renal tubules maintaining an overall healthy architecture. Only mild degenerative changes were observed in some tubules, accompanied by minimal inflammatory infiltration (Fig. 9).

Group G4 (0.66 ng/mL AFs + yeast) also exhibited a substantial recovery. The glomeruli and urinary space appeared nearly normal, and the tubular epithelium was mostly intact, with only minor interstitial hemorrhage and minimal infiltration of inflammatory cells (Fig. 10).

These histological improvements correspond with findings from [37], who demonstrated that animals receiving *S. cerevisiae* for seven days following aflatoxin exposure showed enhanced body weight gain and reduced tissue damage. Specifically, *S. cerevisiae* was reported as a safe and effective agent in mitigating aflatoxin toxicity and offering hepatoprotective benefits.

Furthermore, studies involving *S. cerevisiae* and *Lactobacillus rhamnosus* combinations showed a significant decrease in serum markers such as ALT, AST, gamma-glutamyl transferase, creatinine, and blood urea nitrogen, when compared to untreated control groups. Additionally, glutathione levels were elevated beyond those of control animals, and aflatoxin residues were significantly diminished in both *in vitro* and *in vivo* settings.

Fermented yeast derivatives have also been proposed to provide protective effects against tissue damage and elevations in liver and kidney function markers. These results emphasize the potential of *S. cerevisiae* as a promising agent in alleviating the harmful effects of aflatoxins on renal tissues.

Conclusion

The obtained data indicated that mice exposed to aflatoxins exhibited poor health conditions, including abnormal symptoms, reduced body weight gain, increased liver and kidney weights, elevated enzyme levels, and notable histopathological alterations. However, co-administration of aflatoxins (AFs) with the probiotic baker's yeast *Saccharomyces cerevisiae* significantly improved the health status of the mice. Improvements were observed in body weight gain, normalization of organ weights, and restoration of liver and kidney biochemical markers to levels comparable to the control group. These findings suggest that the application of the probiotic yeast *S. cerevisiae* was effective in protecting mice against the toxic effects of aflatoxins. Moreover, the consumption of fermented foods such as yogurt or dairy-based beverages, or the intake of probiotics in the form of capsules, tablets, powders, or sachets, may offer a practical strategy for reducing the harmful impacts of dietary toxins.

Recommendation

Utilizing yeast-based therapy presents a promising approach for combating mycotoxin contamination through the application of biological control agents (BCAs). Incorporating fermented products such as yogurt or dairy-based drinks into the diet or using probiotic supplements available in various forms—capsules, tablets, powders, or sachets—may be beneficial. The probiotic yeast *Saccharomyces cerevisiae* is widely recognized for its safety and is linked to a range of health-promoting effects. Due to its affordability, accessibility, and proven efficacy, *S. cerevisiae* is considered an effective probiotic and a cost-efficient growth enhancer.

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Declaration of Conflict of Interest

The authors declare that there is no conflict of interest.

TABLE 1. Description of Experimental Groups

Group	Treatment Description
1	Received aflatoxins (0.81 ng/mL) from *A. flavus* isolate Z/N/22 (corn flour origin)
2	Received same aflatoxins (0.81 ng/mL) + probiotic yeast
3	Received aflatoxins (0.66 ng/mL) from *A. parasiticus* isolate A/N/94 (wheat flour origin)
4	Received same aflatoxins (0.66 ng/mL) + probiotic yeast
5	Control group (fed on basal diet without aflatoxins)

TABLE 2. Enhancement of body weights by using a probiotic yeast (*S. cerevisiae*)

Groups	Av./%R	Mice body weight (g)			
		Zero	1week	2weeks	3weeks
G1	Av.	28.5	28.5	28.0	27.5
	%R	5.3	0	6.7	11.3
G2	Av.	30.5	31.0	32.0	33.0
	%R	11.5	8.1	6.3	6.1
G3	Av.	27.5	27.5	26.5	26.0
	%R	1.82	3.51	11.67	16.13
G4	Av.	30.5	31.5	32.5	33.0
	%R	11.5	9.5	7.69	6.1
Control G5	Av.	27.0	28.5	30.0	31.0

G 1 = Received 0.81 ng/ml (AFs) produced by *A. flavus* G 2 = aflatoxins + yeast G 3 = Received 0.66 ng/ml (AFs) produced by A. *parasiticus* G 4 = aflatoxins 4+ yeast. G 5 = Control free aflatoxins

TABLE 3. Enhancement of organs weights by using a probiotic yeast (*S. cerevisiae*)

Groups	Mice organs weight (g)			
	Liver		Kidneys	
	Av.	%R	Av.	%R
G1	2.35	33.28	0.40	25.56
G2	1.94	18.97	0.36	17.36
G3	1.653	5.14	0.35	14.29
G4	1.55	1.47	0.34	11.24
G5	1.57	-	0.30	-

G 1 = Received 0.81 ng/ml (AFs) produced by *A. flavus* G 2 = aflatoxins +yeast, G 3 = Received 0.66 ng/ml (AFs) produced by A. *parasiticus*, G 4 = aflatoxins 4+ yeast. G 5 = Control free aflatoxins

TABLE 4. Enhancement of liver functions by using a probiotic yeast (*S. cerevisiae*)

	Av.	Albumin	GPT(ALT) (U/I)	GOT(AST) (U/I)	Alk.
G1	Av.	2.40	88.00	236.00	174.00
	%R	36.00	56.82	62.29	47.13
G2	Av.	3.05	48.00	141.00	120.00
	%R	18.67	20.8	36.88	23.30
G3	Av.	2.85	78.00	215.50	165.00
	%R	24.00	51.28	58.71	44.20
G4	Av.	3.05	47.00	146.50	145.00
	%R	18.67	19.15	39.25	36.55
G5 Control	Av.	3.75	38.00	89.00	92.00

ALB = Albumin ALT = Alanine aminotransferase AST = Aspartate aminotransferase Alk. phosphatase
G 1 = Received 3T0.81 ng/ml3T (AFs) produced by *A. flavus* G 2 = aflatoxins plus yeast G 3 = Received 3T0.66 ng/ml3T (AFs) produced by *A. parasiticus* G 4 = aflatoxins plus yeast. G 5 = Control free aflatoxins

TABLE 5. Enhancement of kidney functions in mice exposed to aflatoxin by using a probiotic yeast (*S. cerevisiae*3T)

Groups	Av.	Creatinine (mg/dl)	Urea (mg/dl)
G1	Av.	2.00	73.00
	%R	70.00	54.80
G2	Av.	1.65	63.00
	%R	63.64	47.60
G3	Av.	1.75	72.50
	%R	65.71	54.48
G4	Av.	1.55	52.50
	%R	61.29	37.14
G5 Control	Av.	0.60	33.00

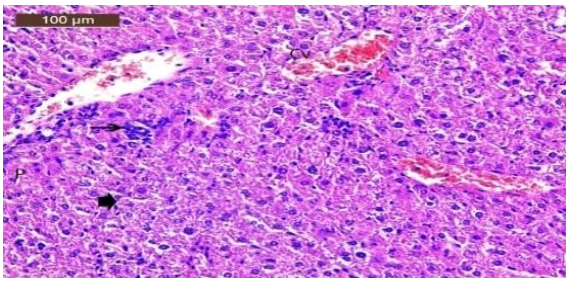

Fig. 1. Photomicrograph of liver tissue from control group 5 showing intact hepatic structure with clearly defined central vein (CV), normal blood sinusoids (S), and distinct nuclei (N).

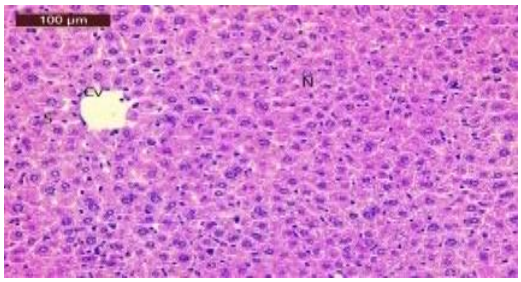

Fig. 2. Photomicrograph of liver tissue from the Aflatoxin-treated group (G1) displaying disrupted hepatic architecture, hepatocyte degeneration (arrowhead), marked central vein dilation and congestion (CV), mononuclear cell infiltration (arrow), and numerous pyknotic nuclei.

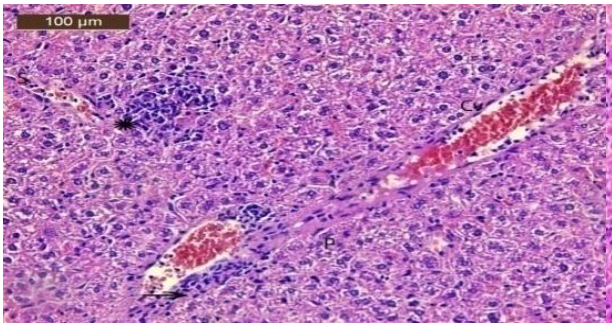

Fig. 3. Photomicrograph of liver tissue from the Aflatoxin-treated group (G3) illustrating severe hepatic architectural loss, vacuolar degeneration of hepatocytes (arrowhead), central vein dilation and congestion (CV), mononuclear cell infiltration surrounding the central vein (arrow), focal mononuclear infiltration (star), and abundant pyknotic nuclei (P).

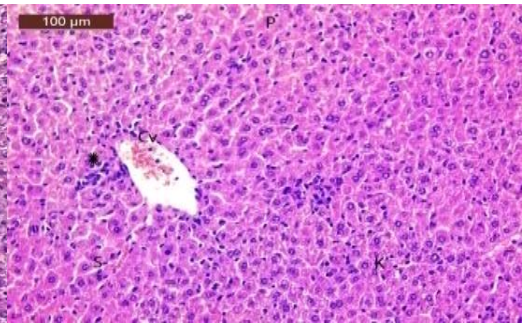

Fig. 4. Photomicrograph of a liver section of Aflatoxin + Yeast (G2) group showing restoration of normal arrangement of hepatocytes with mild congestion central vein (CV) minimal focal cellular inflammation (star) and activated Kupffer cells (K).

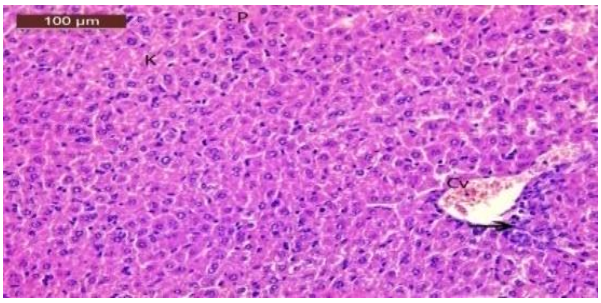

Fig. 5. Photomicrograph of a liver section of Aflatoxin + Yeast (G4) group showing nearly normal arrangement of hepatocytes with mild congestion central vein (CV) minimal cellular inflammation around central vein (arrow).

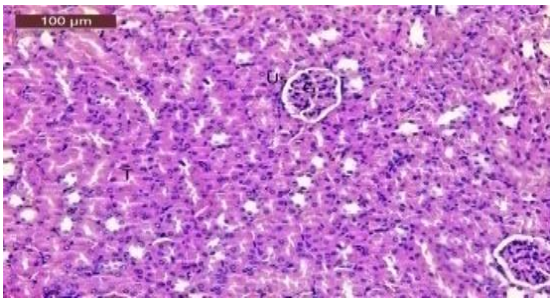

Fig. 6. A photomicrograph of rat kidney of control group (G5) showing intact glomerulus (G) normal urinary space (US) in between with normal tubular structures (*T*).

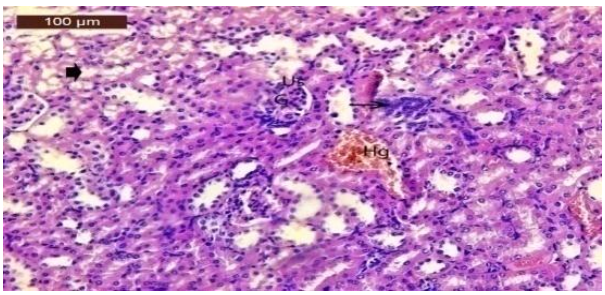

Fig. 7. A photomicrograph of rat kidney of Aflatoxin (G1) group showing atrophy of glomeruli (G) with mild wide urinary space (US) and inflammatory infiltrations (arrow), degeneration in some parts of the tubular epithelial cells (arrowhead) with interstitial hemorrhage (Hg).

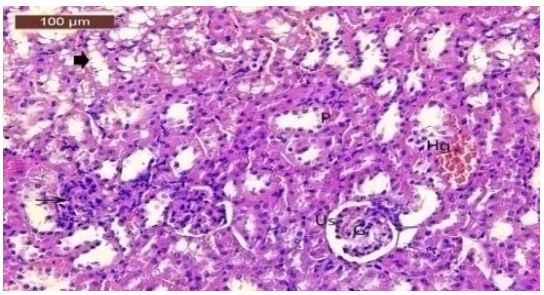

Fig. 8. A photomicrograph of rat kidney of Aflatoxin (G3) group showing atrophy glomeruli (G), wide urinary space (US), inflammatory cell infiltration (arrow) degenerative changes in the tubular lining epithelium, some cells exhibited dark pyknotic nuclei (P) and *interstitial* hemorrhage (Hg).

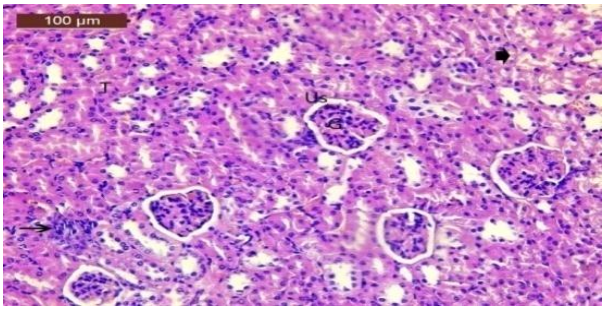

Fig. 9. Photomicrograph of a kidney section of Aflatoxin + Yeast (G2) group showing nearly normal histological structure of glomeruli (G), and urinary space (US), with normal structure of epithelium renal tubules except mild degenerative changes in other tubules (arrowhead) and minimal inflammatory cells (arrow)

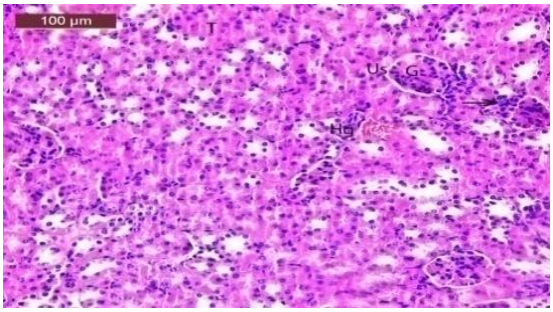

Fig. 10. Photomicrograph of a kidney section of Aflatoxin + Yeast (G4) group showing almost nearly histological structure of glomeruli (G) and urinary space (US), with normal structure of epithelium renal tubules except interstitial haemorrhage (Hg) and minimal inflammatory cells (arrow)

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التقليل من مخاطر التعرض للأفلاتوكسين باستخدام خميرة الخبز: Saccharomyces cerevisia نهج بروبيوتيك صديق للبيئة لتحسين المؤشرات البيوكيميائية وسلامة الأنسجة في الفئران البيضاء

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[4] قسم سمية الملوثات الغذائية، المركز القومي للبحوث، القاهرة، مصر.

الملخص

تمثل الأفلاتوكسينات مصدر قلق صحي عام عالمي رئيسي، حيث تُلوث حوالي 25%من المحاصيل الغذائية على مستوى العالم .ويتعرض الإنسان لهذه السموم إما بشكل مباشر من خلال استهلاك الأطعمة الملوثة، أو بشكل غير مباشر من خلال المنتجات الحيوانية الناتجة عن ماشية تغذت على أعلاف ملوثة بالأفلاتوكسين .هدفت هذه الدراسة إلى تقييم الكفاءة الوقائية لخميرة الخبز البروبيوتيكية (Saccharomyces cerevisia)كعامل طبيعي وصديق للبيئة في الوقاية من التسمم بالأفلاتوكسين في إناث الفئران البيضاء في ظروف معملية .in vivo تم استخدام خمس مجموعات تجريبية، تلقت كل منها أحد تركيزين من الأفلاتوكسين 0.81)نانوجرام/مل من عزلة *Aspergillus flavus*رقم Z/N/22، و 0.66نانوجرام/مل من عزلة A. parasiticus رقم .(A/N/94أظهرت النتائج أن الفئران التي تعرضت للأفلاتوكسين فقط أظهرت علامات تسمم شملت انخفاضاً في معدل زيادة الوزن، وزيادة في أوزان الكبد والكلى، وارتفاعاً في المؤشرات البيوكيميائية (ALT، AST، الفوسفاتاز القلوي، الكرياتينين، واليوريا(، بالإضافة إلى تغيرات مرضية نسيجية .في المقابل، أدى العلاج المشترك مع S. cerevisiaإلى تقليل ملحوظ في هذه التأثيرات، حيث تحسنت المؤشرات البيوكيميائية، واحتُفظ بسلامة الأنسجة بشكل مماثل للمجموعة الضابطة. تُظهر خميرة الخبز البروبيوتيكية إمكانات كبيرة كوسيلة طبيعية وغير سامة ومستدامة بيئيًا للتخفيف من التأثيرات السامة للأفلاتوكسينات .ويُعد استخدامها إستراتيجية واعدة لتعزيز سلامة الغذاء والعلف .إن الاستهلاك المنتظم للأطعمة المخمرة الغنية بالبروبيوتيك)مثل الزبادي أو المشروبات اللبنية المخمرة(، أو استخدام المكملات البروبيوتيكية في شكل كبسولات أو مسحوق، قد يوفر وسيلة فعالة وعملية لتقليل التعرض للأفلاتوكسين .ويوصى بإجراء مزيد من الأبحاث ودمج هذا النهج في ممارسات الصحة العامة والزراعة لدعم تطبيقه على نطاق أوسع.

الكلمات الدالة: الفئران، أنواع Aspergillus، الأفلاتوكسينات، البروبيوتيك، *Saccharomyces cerevisiae*.