Mechanisms of Mycotoxin Impact on Poultry Production Performance and Contamination Control Postprint

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Abstract

Poultry exhibit high sensitivity to mycotoxins in feed, and various mycotoxins exert toxic effects on poultry. This review summarizes the effects of common mycotoxins on poultry growth performance, immune function, and reproductive performance, along with their underlying mechanisms. Additionally, it elaborates on the detoxification efficacy of several common adsorbents against mycotoxins and provides an outlook on the application prospects of mycotoxin adsorbents.

Full Text

Influence Mechanism of Mycotoxins on Poultry Production Performance and Pollution Control

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Abstract: Poultry are highly sensitive to mycotoxins in feed, and numerous mycotoxins exert toxic effects on poultry. This article reviews the effects of common mycotoxins on the growth performance, immune function, and reproductive performance of poultry and their underlying mechanisms, while also elaborating on the detoxification efficacy of several common adsorbents against mycotoxins and prospects for their application.

Keywords: mycotoxins; toxicity; mechanism; adsorbents; poultry

Mycotoxins are toxic secondary metabolites produced by molds during their growth and reproduction on substrates. Their hazards to human health were reported as early as World War II, while the scientific community' s recognition of mycotoxin toxicity originated from a highly lethal liver disease in turkeys in the United Kingdom. The Food and Agriculture Organization (FAO) estimates that 25% of global grains are contaminated with mycotoxins to varying degrees each year, causing enormous economic losses to grain producers, livestock industries, and feed and food processing enterprises [1]. BIOMIN' s investigation of mycotoxin contamination in feed ingredients and complete feeds worldwide in 2012 revealed that mycotoxins were present in Asia, Europe, North America, and Africa, with Asia being the most severely affected. Mycotoxin concentrations in Asian samples were several to dozens of times higher than those in North America and other regions. In East Asia (China, South Korea, and Japan), the contamination rates of deoxynivalenol (DON) and zearalenone (ZEN) reached 58% and 82%, respectively, with maximum concentrations of 9,854 g/kg and 28,005 g/kg [2]. In southern China, where spring brings plum rains and summer is hot and humid, feed ingredients and complete feeds are highly susceptible to mycotoxin contamination. Ji et al. [3] collected 458 feed ingredient and complete feed samples from across China between January and June 2015. The overall detection results showed that ZEN had a 100% detection rate, DON 99.78%, while aflatoxin B1 (AFB1) had a relatively lower positive detection rate. The maximum concentrations of DON and ZEN reached 4,402.69 g/kg and 1,518.18 g/kg, respectively, with DON exceeding standards by 51.09%, representing the most severe contamination. Additionally, duck complete feeds were found to have average DON and ZEN concentrations of 1,742.91 g/kg and 371.55 g/kg, with exceedance rates of 66.67% and 16.67%, respectively. Gong et al. [4] detected mycotoxin levels in feeds and raw materials from Guangxi, Fujian, Hunan, Shenyang, and other regions between January and July 2015, finding that poultry feed had 100% detection rates for both AFB1 and DON, with maximum concentrations of 43.6 g/kg and 4,828.6 g/kg, respectively. The exceedance rates for AFB1 and ZEN were 23.1% and 7.7%, indicating serious contamination.

Extensive mold reproduction causes mold deterioration of feed ingredients and complete feeds, greatly reducing their utilization value, palatability, and nutritional value. Simultaneously, it causes acute or chronic poisoning in livestock and poultry. Moreover, certain mycotoxins exhibit carcinogenic, teratogenic, and mutagenic toxicity. Therefore, controlling mold and mycotoxin contamination of feed ingredients and feeds and their hazards to animal production represents a major global challenge. Poultry production performance and product quality are profoundly affected by mycotoxins. Currently, systematic reports elucidating the toxic effects of mycotoxins on poultry are scarce. This review summarizes the effects of mycotoxins on poultry production performance from three aspects—growth, immunity, and reproduction—and their mechanisms. It also summarizes approaches for controlling mycotoxin contamination and the

detoxification efficacy of several common adsorbents, while discussing application prospects for adsorbents and microbial degradation of mycotoxins, aiming to help feed enterprises and livestock producers actively address mycotoxin contamination issues.

1 Common Mycotoxins

Over recent decades, researchers have successively discovered many different types of mycotoxins. Currently, more than 300 mycotoxins are known, with approximately 150 mold species capable of producing toxins. Mycotoxins of primary concern in feed hygiene mainly originate from *Fusarium* spp., *Penicillium* spp., and *Aspergillus* spp. Presently, aflatoxins (AF), ZEN, DON, T-2 toxin, and ochratoxins (OT) are research hotspots.

AF represents the most severely contaminating and toxic class of mycotoxins, classified by the World Health Organization (WHO) as a Group IA hazard with strong carcinogenic, highly toxic, and strongly mutagenic properties. AF comprises secondary metabolites produced by fungi such as *Aspergillus flavus*, *Aspergillus parasiticus*, and *Aspergillus pseudotamarii* [5], with 18 structural derivatives including B1, B2, G1, G2, and M1, M2, among which AFB1 exhibits the strongest toxicity and carcinogenicity. The strong carcinogenicity of AFB1 results from its activation by monooxygenases to form the highly carcinogenic AFB1-8,9-epoxide, which further generates carcinogenic substances that hinder protein synthesis in the liver [6]. Many countries have established maximum limits for AFB1 in poultry feed. Some countries set the maximum allowable AFB1 content in poultry feed at 20 g/kg. Japan stipulates that AFB1 should not exceed 10 g/kg in chick feed and 20 g/kg in adult chicken feed. China limits AFB1 content to 10 g/kg in broiler starter and chick feed, 15 g/kg in broiler finisher, growing duck, and laying duck feed, and 20 g/kg in broiler finisher, growing chicken, and laying chicken feed. The European Union sets maximum allowable levels for total AF and AFB1 in agricultural products at 4 g/kg and 2 g/kg, respectively. These standards demonstrate the high priority placed on AF restrictions worldwide, with China' s limits being essentially consistent with those of developed countries.

1.2 ZEN

ZEN, also known as F-2 toxin, is a non-steroidal mycotoxin isolated from corn with gibberella ear rot. It is a secondary metabolite produced by various *Fusarium* species including *Fusarium graminearum*, *Fusarium roseum*, and *Fusarium nivale* [7], exhibiting strong toxicity with multiple derivatives such as 7-dehydrozearalenone, zearalenic acid, and 8-hydroxyzearalenone. ZEN is the most widely contaminating fusarium toxin globally, commonly found in moldy corn, sorghum, barley, and other cereal crops as well as in complete feeds. Elisabeth et al. [8] collected 13,578 feed ingredient and complete feed samples worldwide from 2004–2011, with detection results showing a 36% ZEN detection rate and a maximum concentration of 26,728 g/kg, far exceeding maximum limits.

ZEN exhibits estrogen-like effects, binding irreversibly to estrogen receptors in the uterus and triggering a series of estrogen-mimicking effects that disrupt reproductive hormones in livestock and poultry, thereby affecting animal reproductive physiology [9]. China' s *Feed Hygiene Standard* limits the maximum ZEN content in cereals such as corn and wheat and in livestock and poultry complete feeds to 500 g/kg.

1.3 Trichothecenes (TS)

TS include approximately 150 compounds, broadly divided into four subclasses including A and B. The most hazardous Type A trichothecene is T-2 toxin. In 1973, the FAO and WHO classified T-2 toxin, like AF, among the most dangerous naturally occurring food contaminants, primarily affecting the normal physiological functions of blood, liver, kidney, and lymphocytes [10]. The most common Type B trichothecene is deoxynivalenol, also known as DON, named for its ability to cause vomiting in ducklings, pigs, dogs, pigeons, and other animals. The International Agency for Research on Cancer has classified DON as a Group 3 carcinogen. Its main toxic effects include vomiting, anorexia, gastroenteritis, diarrhea, immunosuppression, and hematological disorders. The toxic group of DON is the C12,13-epoxy group, which strongly damages gastrointestinal mucosal cells, lymphocytes, and thymocytes, while also inhibiting protein synthesis and inducing mitochondrial apoptosis [11]. TS are the second most immunosuppressive mycotoxins after AF, directly acting on immune organs and tissues such as bone marrow, spleen, and lymphoid tissue in livestock and poultry, hindering division of immunocompetent cells and affecting cellular immune responses. When DON and T-2 toxin levels in feed and grains exceed 1 mg/kg, they pose health hazards to humans and animals. Therefore, the U.S. Food and Drug Administration sets the DON limit in food at 1 mg/kg. China' s *Feed Hygiene Standard* limits DON in cereals such as corn, barley, and wheat and in poultry complete feeds to 1 mg/kg, and sets the allowable T-2 toxin level in pig and poultry complete feeds at 1 mg/kg. Currently, most countries set limits of 1 mg/kg for DON and T-2 toxin in food and feed.

1.4 Ochratoxins (OT)

In 1965, South African researcher Scott first discovered OT in moldy sorghum. OT is primarily produced by fungi such as *Aspergillus ochraceus*, *Penicillium viridicatum*, and *Aspergillus carbonarius*, designated as ochratoxin A (OTA), ochratoxin B (OTB), and ochratoxin C (OTC) based on their discovery order, with OTA being the most common [12]. OTA can poison animal liver and kidney, exhibiting teratogenic, carcinogenic, mutagenic, and immunosuppressive effects. OTA can damage the bursa of Fabricius and intestinal lymphoid tissue in poultry, reduce serum immunoglobulin (Ig) A, IgG, and IgM levels, affect humoral immunity, and decrease the phagocytic capacity of granulocytes, thereby affecting cellular immunity [6]. In 2001, WHO set the maximum OTA limit in cereals and their products at 5 g/kg, a standard subsequently adopted by many

countries. China' s GB 2761-2011 stipulates that the allowable OTA level in cereals, beans, and their products must not exceed 5 g/kg.

2 Contamination Characteristics of Mycotoxins

Feed ingredients and complete feeds are often simultaneously contaminated with multiple mycotoxins, representing the most significant characteristic of mycotoxin contamination. Numerous studies have shown that mycotoxins can interact through synergistic, antagonistic, and additive effects [13]. Swamy et al. [10] demonstrated that mycotoxin synergism poses greater hazards to livestock production and health than single mycotoxins. Wang et al. [14] reported that the detection rates of AF, DON, ZEN, OT, and T-2 toxin in complete feeds were substantially higher than in single protein and energy ingredients. Thus, formulating complete feeds with multiple feed ingredients substantially increases the risk of contamination with multiple mycotoxins.

Since certain molds in feed ingredients can produce multiple mycotoxins, such as *Fusarium*, and because complete feeds formulated with ingredients contaminated with different mycotoxins are prone to cross-contamination, Smith et al. [15] found that when grains are mixed with soybean meal, the probability of co-occurrence of AF and fusarium toxins increases significantly. This demonstrates that feed ingredients and complete feeds are vulnerable to contamination with multiple mycotoxins. Due to combined mycotoxin effects, nutrient imbalance in diets, and environmental stress factors, low concentrations of mycotoxins in feed can affect animal production and health. Furthermore, feed contaminated with mycotoxins causes enormous economic losses to feed processing enterprises and livestock farms, while mycotoxins and their metabolites residues in livestock and poultry tissues and products (meat, eggs, milk) can indirectly harm human health through the food chain.

3 Effects of Mycotoxins on Poultry Production Performance and Mechanisms

The most fundamental effects of feed mycotoxins on livestock and poultry include reduced feed intake, decreased feed conversion efficiency, and substantially lowered production performance. Additionally, certain mycotoxins exhibit oxidative damage toxicity, injuring target tissues and organs and disrupting the body' s antioxidant enzyme system, producing a series of effects on animal organisms. Moreover, mycotoxins can inhibit DNA, RNA, and protein synthesis, reducing immune function in livestock and poultry, as seen with AF, ZEN, OT, and T-2 toxin, thereby decreasing disease resistance and growth performance and causing economic losses to farms.

3.1 Effects on Growth Performance

The effects of mycotoxins on poultry first manifest as reduced growth performance. Numerous studies have shown that various mycotoxins can decrease

poultry feed intake, average daily gain (ADG), and feed conversion efficiency. Hamilton et al. [16] investigated the toxic effects of OTA on poultry, finding that 2–16 mg/kg OTA reduced feed intake in turkeys, increased mortality, decreased egg production in laying hens, caused varying degrees of kidney damage, slowed broiler growth, and increased feed-to-gain ratio. Aravind et al. [17] showed that OTA significantly decreased feed intake in poultry and slowed growth and feather development. The effects of TS on poultry growth performance primarily include reduced feed intake, decreased body weight gain and feed conversion efficiency, and poor flock uniformity. Kubena et al. [18] found that T-2 toxin reduced feed intake and ADG in chicks, while dietary supplementation with 5 mg/kg DON decreased average daily feed intake and ADG in chicks [19].

The most significant effect of AF on egg-laying poultry production performance is reduced egg production and egg weight. Mukhopadhyay et al. [20] added 500 g/kg AF to commercial laying hen diets and fed them for 90 days, resulting in significantly decreased egg production. Ogido et al. [21] fed Japanese quail diets containing 50 g/kg and 10 mg/kg AFB1 for 140 days, finding increased feed consumption but decreased egg weight. Growth performance in meat poultry is also affected by AF-contaminated diets. Dietary supplementation with 0.1 mg/kg AFB1 significantly reduced ADG and increased feed-to-gain ratio in yellow-feathered broilers [22]. Shi et al. [23] reported that oral administration of AFB1 at 0.05, 0.10, and 0.20 mg/kg body weight to meat ducklings significantly inhibited their growth. The mechanism by which AF reduces weight gain in livestock and poultry may involve increased urinary nitrogen excretion and decreased serum total protein, globulin, and triglyceride levels [24]. DON and T-2 toxin can alter levels of tryptophan, dopamine, serotonin, and their metabolites in the body, regulating receptors for serotonin and catecholamines in the small intestine through the blood-brain barrier, inhibiting normal small intestine peristalsis, and causing feed refusal [25]. Additionally, mycotoxins may adversely affect livestock and poultry growth performance by inhibiting enzyme activity. Beri et al. [26] found that after mycotoxin poisoning in chicks, activities of alkaline phosphatase, lactate dehydrogenase, and succinate dehydrogenase in heart, spleen, liver, and other organs were significantly reduced. Decreased activity of these enzymes affects organism metabolism and cellular respiration, thereby causing slowed weight gain and growth retardation.

Currently, most reports on mycotoxin effects on poultry growth performance have used relatively high mycotoxin concentrations (some far exceeding national standards), proving adverse effects on poultry growth performance. However, mycotoxin levels in complete feeds often do not reach those used in experiments. Additionally, some reports indicate that certain ZEN concentrations have no significant effect on piglet ADG and feed-to-gain ratio, and structural analysis of ZEN has revealed potential growth-promoting effects. Therefore, it is necessary to investigate the effects of different mycotoxin levels (especially lower levels) on poultry growth. Furthermore, researchers have mostly used purified mycotoxins in experiments, whereas naturally contaminated feeds generally contain multiple mycotoxins. Due to interactions among mycotoxins, co-occurrence may produce

greater toxicity. Future research should deeply explore the effects of combined mycotoxin actions on poultry growth performance. Current mechanistic studies on mycotoxin effects on poultry growth performance have focused on impacts on nutrient metabolism. However, mycotoxin absorption occurs primarily in the intestine, and the digestive system, particularly the digestive tract, may be the first to be poisoned after intoxication. Therefore, it is essential to study the effects of mycotoxins on poultry intestinal morphology, microbiota, and related gene expression to explore the toxicity of mycotoxins on poultry digestive physiology and its mechanisms.

3.2 Effects on Immune Function

The immune system in poultry consists of the thymus, spleen, bursa of Fabricius, tonsils, and bone marrow. Common feed mycotoxins affect cellular and humoral immunity to varying degrees, with AF exerting the strongest immunosuppressive effects on poultry. Wang et al. [27] found that 200 g/kg AFB1 significantly inhibited Newcastle disease virus vaccine antibody production in commercial broilers. Yunus et al. [28] demonstrated a high correlation between Newcastle disease outbreaks in broilers and AF contamination in diets. Betina [29] showed that AFB1 affected macrophage and liver function, interfered with T lymphocyte production of interleukins (IL) and other lymphokines, and inhibited complement 4 (C4) generation. Current research conclusions indicate that AF primarily reduces phagocytic capacity of phagocytes, increases susceptibility to diseases caused by viruses, bacteria, and parasites, reduces vaccine efficacy, and directly affects immune function. T-2 toxin primarily reduces T lymphocytes, IL-2, and other lymphokines, inhibits white blood cell and complement 3 (C3) production, and decreases serum IgM and IgA levels, affecting immune function. Additionally, T-2 toxin suppresses poultry immunity by damaging immune organs such as the spleen, thymus, and bursa of Fabricius [30]. Boonchuvit et al. [31] fed broilers T-2 toxin-contaminated feed while simultaneously inoculating them with *Salmonella*, finding that mortality in the experimental group reached 28.5%, with spleen and bursa atrophy occurring after T-2 toxin inoculation. Comprehensive analysis shows that long-term, high-dose T-2 toxin intake in poultry causes immune organ atrophy, induces lymphocyte necrosis, and may increase susceptibility to bacterial and viral infections such as *Salmonella* and Newcastle disease virus. OT primarily reduces poultry cellular immunity by causing lymphocyte degeneration and decreasing lymphocyte numbers, also reducing immune system effector cells such as macrophages, further decreasing immunoglobulin levels in circulation and suppressing humoral immunity. Lesson et al. [32] showed that OTA affects cell-mediated immune responses, causes lymphoid tissue regression, reduces heterophil phagocytosis, and increases susceptibility to airsacculitis caused by *E. coli* in poultry. Xue et al. [33] demonstrated that OT and T-2 toxin can act synergistically to reduce plasma IL-2 and interferon-γ mRNA expression.

Research has found that certain mycotoxins can indirectly harm livestock and

poultry immune systems. T-2 toxin activates the endocrine system, promoting corticosteroid release under stress conditions and indirectly suppressing immune function. ZEN exhibits estrogen-like effects, causing estrogen secretion disorders that indirectly affect immune organs as potential target organs. The neurotoxicity of DON also indirectly harms the immune system [34-35]. Abbe et al. [36] found that 40 mg/kg ZEN significantly reduced mouse spleen lymphocyte numbers, causing splenic cell, red pulp swelling, and white pulp atrophy, inducing immune system damage while significantly decreasing serum IgA and IgG levels and peripheral blood CD3+, CD4+, CD8+, and CD56+ counts, suppressing humoral immune responses. DON has a sesquiterpene structure that can inhibit transcription and translation, and in vivo, DON can suppress immune responses to pathogens, producing immunosuppression [37]. Comprehensive analysis shows that mycotoxins primarily suppress immune function by reducing antibody levels, destroying the reticuloendothelial system, decreasing cell-mediated immunity, and causing abnormal development or atrophy of immune organs such as the thymus and bursa of Fabricius. Currently, relatively few reports address the immunotoxic effects of ZEN and DON on poultry. Meanwhile, synergistic effects exist among mycotoxins, and feeds and feed ingredients are often simultaneously contaminated with multiple mycotoxins. Experiments have confirmed that mycotoxin mixtures affect immunity far more than single mycotoxins. Future research must further investigate the effects of mycotoxins and their synergistic actions on poultry immune function and the toxic mechanisms on immune organs and cells to establish a theoretical foundation for better controlling mycotoxin hazards to livestock production.

3.3 Effects on Reproductive Performance

The mycotoxins primarily harming poultry reproductive performance are AF and ZEN. AF negatively affects the reproductive performance of both meat-type and egg-type breeding poultry. Qureshi et al. [38] investigated AFB1 effects on meat-type breeding chickens and their progeny, adding 0.2 and 1.5 mg/kg AFB1 to hen diets and hatching the produced eggs. Results showed that the high-dose group had reduced hatchability, increased late embryonic mortality, and poor disease resistance in hatched chicks. Mohan et al. [39] fed immature roosters 0.2 mg AFB1 per bird daily for 35 days, finding testicular germinal epithelium lesions, testicular atrophy, reduced spermatogenesis, and substantially decreased fertilization rates. Reports indicate that AFB1 can accumulate in the reproductive organs of chickens, turkeys, and ducks and be transferred to eggs and passed to offspring [40]. Yunus et al. [41] found that AFB1 reduced egg production and hatchability in breeding hens while causing ovarian cysts and decreased estrogen secretion. Comprehensive analysis shows that AF effects on male poultry primarily include testicular atrophy, reduced sperm numbers, and decreased semen quality, while toxic effects on female poultry include ovarian cysts, inhibited estrogen secretion, and reduced egg production and hatchability. Additionally, AF is detrimental to embryonic development and reduces disease resistance in young poultry. Currently, few studies have investigated the mech-

anisms of AF effects on poultry reproduction, representing an important area for future research.

ZEN poisoning accelerates the development of secondary sexual characteristics in poultry, causing rapid comb development in male chicks, comb enlargement in laying hens, ovarian atrophy, and substantially reduced egg production [19]. Shan et al. [42] reported that ZEN can cause reproductive tract cysts, prolapsed cloaca, and oviduct enlargement in poultry. ZEN and its metabolites can inhibit chorionic gonadotropin-induced testosterone secretion, reducing testosterone synthesis by downregulating cytochrome P450scc, 3β-hydroxysteroid dehydrogenase 1 (3β-HSD-1), and inhibiting transcription of cholesterol regulatory element-binding protein, thereby affecting spermatogenesis [43]. The toxic mechanism of ZEN on ovaries may involve inducing lipid peroxidation of polyunsaturated fatty acids in ovarian cell membranes, producing numerous lipid breakdown products that interfere with ovarian cell metabolism and function [44].

Currently, domestic and international research on mycotoxin effects on poultry reproductive performance is relatively limited, yet breeding represents the most crucial aspect of poultry production. At low doses, certain mycotoxins alone may have limited effects on poultry reproductive performance, but reproductive toxicity may manifest when they interact with other toxins. Additionally, due to mycotoxin interactions that may even enhance the reproductive toxicity of other toxins, and because complete feeds typically contain multiple mycotoxins, future research must investigate the effects of mycotoxins and their combined actions on poultry reproduction and deeply study their toxic mechanisms.

4 Control of Mycotoxin Contamination

Because mycotoxin concentrations in feed are very low, detection is difficult, clinical symptoms after livestock poisoning are not obvious or typical, and the interactive effects among mycotoxins remain unclear, controlling mycotoxin contamination is not straightforward.

4.1 Approaches for Controlling Mycotoxin Contamination

Mycotoxin contamination control can be implemented in two stages: (1) Mold prevention: controlling mold growth and reproduction during crop cultivation to reduce mycotoxin production. Primary measures include developing mold-resistant crop varieties, providing good growth conditions for crops, and avoiding mold contamination during harvest and storage. Research shows that inoculating nontoxigenic *Aspergillus flavus* strains in the field can reduce AF infection in peanuts by over 95% [45]. Currently, few reports address the application of nontoxigenic strains for other mycotoxins, representing an important area for future research. (2) Detoxification: employing appropriate methods to remove mycotoxins from feed and feed ingredients. Currently, adding mycotoxin adsorbents to feed and utilizing microbial degradation of feed mycotoxins represent two major global trends for addressing mycotoxin contamination, with

adsorbents being most widely applied in animal production.

4.2 Physical Adsorption of Mycotoxins

Mycotoxin adsorbents utilize their large specific surface area and the ionic polarity of mycotoxins to bind toxins through intermolecular forces and electrostatic adsorption, transforming them into inactive substances. Additionally, adsorbents can bind toxins within the gastrointestinal tract, substantially reducing toxin bioavailability. Currently, the most commonly used adsorbents in production include bentonite, glucomannan (GM), and esterified glucomannan (EGM).

Bentonite is a fine-grained clay whose main mineral component is montmorillonite. As early as 1983, Carson et al. [46] demonstrated that bentonite could inhibit T-2 toxin absorption in rat small intestine, alleviating its toxic effects. Subsequently, extensive research has been conducted on hydrated sodium calcium aluminosilicate (HSCAS) adsorbents such as bentonite. Devreese et al. [47] showed that HSCAS could effectively adsorb AF. In vitro studies also found that zeolite and bentonite could adsorb 99% of AFB1 [48]. Afriyie et al. [49] demonstrated that adding hectorite and montmorillonite clay to moldy feed could effectively reduce ZEN content. However, HSCAS adsorbents have limited adsorption capacity for DON, T-2 toxin, and OTA.

GM is a high-molecular-weight polysaccharide that can bind mycotoxins through ionic bonds, hydrogen bonds, and hydrophobic interactions, functioning in the gastrointestinal pH environment of most animals with minimal nutrient destruction and low addition rates. Studies have found that GM has good adsorption effects on DON and ZEN and can regulate immune function in broilers [50]. Zhang et al. [51] investigated the adsorption efficacy of brewer' s yeast β-D-glucan on ZEN, showing it could adsorb up to 2.29 g/mg of ZEN. Faixova et al. [52] used modified GM as a mycotoxin adsorbent in broilers, finding it could substantially eliminate ZEN.

EGM is formed by esterifying GM, featuring a large surface area with numerous small pores that adsorb multiple mycotoxins. Reports indicate that EGM can adsorb 95% of AFB1, 12% of DON, 77% of ZEN, and 9% of fumonisin in feed. Raju et al. [53] showed that dietary EGM supplementation could alleviate the adverse effects of AFB1, OTA, and T-2 toxin and their synergistic actions on broiler weight gain and antibody responses, while improving serum biochemical and hematological parameters. Qi et al. [54] added 0.05%, 0.10%, and 0.15% EGM to culture media containing 5 g/mL ZEN, finding that ZEN-induced reductions in peripheral blood lymphocyte transformation rate and superoxide dismutase activity and increased malondialdehyde content were ameliorated. However, in 2002, the University of Michigan Medical School conducted scientific toxicity tests on yeast cell wall extract mannan oligosaccharide, refuting claims of its high adsorption capacity for multiple toxins [55].

4.3 Prospects for Physical Adsorption of Mycotoxins

Since feed is simultaneously contaminated with multiple mycotoxins, single adsorbents may have unsatisfactory efficacy, making the development of composite adsorbents imperative. Zhang et al. [56] investigated the application effects of a self-developed composite adsorbent (mainly composed of bentonite, yeast cell wall, and EGM), finding it could reverse oxidative damage and immunotoxicity caused by mycotoxins in broilers and improve vaccine efficacy. Meanwhile, adsorbents also adsorb other nutrients such as minerals and vitamins, reducing feed utilization efficiency. Acidifiers, enzyme preparations, and probiotics can lower gastrointestinal pH and directly participate in metabolism, promoting nutrient absorption and metabolism. Therefore, adding appropriate amounts of acidifiers, enzyme preparations, or probiotics alongside adsorbents to study their interactive effects may maximize nutrient utilization while removing mycotoxin toxicity, ensuring adsorbent efficacy. Additionally, current research on adsorbent effects on poultry intestinal structure and microecology is limited, making it highly significant to investigate adsorbent effects on poultry intestinal health and related functional gene expression to comprehensively understand adsorbent impacts on livestock production and conduct more in-depth research, promoting more scientific and widespread application of mycotoxin adsorbents in livestock production for the welfare of animals and humans.

Mycotoxins have substantial toxic effects on poultry. Researchers worldwide have revealed the effects of common mycotoxins on poultry production performance and explored their toxic mechanisms, though mechanisms for some toxins and combined toxicities remain unclear. Urgent research is needed on mycotoxin interactions and combined toxic mechanisms to establish a theoretical foundation for controlling mycotoxin contamination. Currently, adding adsorbents to feed and utilizing microbial degradation represent two major global trends for addressing mycotoxin contamination. Adsorbents are most widely applied in livestock production with good results, but certain deficiencies exist. Future efforts should focus on improving and optimizing single adsorbents, developing and promoting composite adsorbents, and investigating combined effects of adsorbents with other additives to achieve optimal production benefits.

In the early 21st century, utilizing microorganisms and their enzymes to degrade feed mycotoxins has become a research hotspot in feed hygiene. Extensive domestic and international studies have shown that microbial degradation technology offers advantages over traditional methods and adsorption, including high detoxification efficiency, strong specificity, and minimal nutrient structure destruction. In China, Professor Ji Cheng and his team at China Agricultural University and Professor Liu Daling' s research group at Jinan University have conducted extensive research in mycotoxin biodegradation, achieving breakthrough progress and obtaining numerous mycotoxin-degrading strains and enzymes while exploring their degradation mechanisms. However, overall, relatively few microbial strains have been proven to degrade mycotoxins, and even fewer probiotics can be directly applied in feed to degrade mycotoxins.

There is an urgent need to screen more probiotics with efficient degradation capacity while deeply studying their detoxification mechanisms and metabolic pathways. Furthermore, to overcome bottlenecks in large-scale application of microbial mycotoxin degradation, it is necessary to integrate knowledge and technologies from molecular biology, biochemistry, enzyme engineering, and genetic engineering to transform toxin-degrading enzyme genes into host cells for efficient expression, increasing toxin-degrading enzyme production, and ultimately developing new degrading enzyme feed additive products with high purity, strong activity, and good stability to effectively control mycotoxin contamination and reduce its hazards to animal production and human health.

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