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Abstract

Aflatoxins, toxic compounds primarily from the fungi *Aspergillus flavus* and *Aspergillus parasiticus*, are a major global threat to poultry farming. These toxins frequently contaminate common feed ingredients like corn, cottonseed, and peanut products, causing a range of serious health issues in birds, including liver damage, weakened immunity, and poor production—all of which harm farm profitability.

This review comprehensively examines the issue, detailing how environmental factors promote fungal growth and toxin production, the biochemical mechanisms of toxicity (specifically how reactive metabolites cause cellular damage through oxidative stress), and the resulting pathology. Critically, it evaluates various control strategies designed to manage these toxins and maintain bird health. Effective interventions discussed include the use of adsorbent materials, antioxidants (like curcumin), microbial and enzymatic methods, and other phytochemicals. Given the risk of toxin residues entering the human food chain via poultry products, the text stresses the importance of global regulatory frameworks and rigorous monitoring for effective mycotoxin management, which is essential for both animal welfare and food safety.

Keywords: Mycotoxins, poultry health, toxicological effects, control strategies, *Aspergillus* contamination

Introduction

Mycotoxins produced by *Aspergillus* species are a major concern in commercial poultry farming worldwide. Among these fungal metabolites, aflatoxins stand out as particularly dangerous compounds that contaminate essential feed ingredients (Awuchi *et al.*, 2020) [6]. The dominant producers, *A. flavus* and *A. parasiticus*, colonize agricultural commodities under favorable environmental conditions, leading to widespread contamination of poultry feed sources.

The economic implications of aflatoxin poisoning in poultry production systems are significant and multifaceted. Essential ingredients such as corn, cottonseed by-products, and peanut meal commonly contain these toxins, creating ongoing challenges for feed safety management (Ochieng *et al.*, 2021) [36]. Among the various aflatoxin species, aflatoxin B1 (AFB1) exhibits the highest toxicity and prevalence in contaminated feed, posing severe risks to both animal and human health.

When birds consume feed contaminated with mycotoxins, the consequences are felt across multiple physiological systems. Acute poisoning can lead to hepatocellular necrosis, metabolic disturbance, and death, while subacute exposure results in progressive deterioration characterized by stunted growth, impaired immunity, and increased susceptibility to disease (Kibugu *et al.*, 2024) [23]. Chronic consumption of contaminated feed at low levels significantly impairs productive parameters, including feed utilization efficiency, reproductive performance, and disease resistance (Olariu *et al.*, 2025) [39].

Besides the direct effects on bird health, aflatoxins pose serious food safety concerns through bioaccumulation in edible tissues and eggs. This transfer of toxins through the food chain poses risks to human consumers, particularly with continued exposure to contaminated poultry products (Atherstone *et al.*, 2016) [5]. Regulatory authorities worldwide have set maximum residue limits for aflatoxins in animal feed and food products to mitigate these risks (Chain *et al.*, 2020) [9].

Aflatoxin contamination is a persistent issue in poultry feed, despite existing regulations and industry awareness (Shabeer *et al.*, 2022) [45]. Therefore, continuous research is vital to discover effective strategies for prevention, detection, and mitigation. To develop complete management plans that safeguard both animal welfare and food safety, it's crucial to understand the intricate connections between environmental conditions, the biology of the fungus that produces the toxin, the chemistry of the toxin itself, and how it affects avian physiology.

Etiology and Environmental Factors

1. Fungal Species and Contamination Sources

Aflatoxins, predominantly AFB1—the most toxic type and a major contaminant of poultry feed—are primarily synthesized by the fungi *Aspergillus flavus* and *Aspergillus parasiticus* (Fouad *et al.*, 2019) [17]. These adaptable filamentous fungi thrive in warm, humid conditions and can contaminate agricultural products at every stage, from field cultivation to storage and processing (Lizárraga-Paúlín *et al.*, 2011) [30].

Sources of Contamination

Aflatoxin contamination is a critical concern for commercial poultry diets because the fungi primarily colonize cereals (like maize and wheat) and oilseeds (such as peanuts), which are the nutritional staples of the feed (Salisu and Almajir, 2020) [43].

Timing of Contamination

Fungal invasion and toxin production occur in two main phases:

Pre-harvest Contamination: This is a major challenge where airborne *Aspergillus* spores infect growing crops, especially under environmental stress. Conditions like drought, high temperatures, and insect damage create favorable entry points and promote fungal germination and

toxin initiation while the plants are still in the field (Dövényi-Nagy *et al.*, 2020) [12].

Post-harvest Contamination: After harvest, contamination escalates when commodities are not adequately dried or properly stored. Fungal growth and subsequent aflatoxin accumulation continue rapidly in stored grains if the moisture content exceeds critical levels (typically above 13–14%) and the facilities are warm and poorly ventilated (Lavkor and Var, 2017) [28].

Environmental Conditions Promoting Toxin Synthesis

Several environmental parameters interact to determine the extent of fungal spread and subsequent toxin production. Relative humidity exceeding 80% creates ideal conditions for *Aspergillus* growth, especially in tropical and subtropical regions where such conditions occur frequently (Molnár *et al.*, 2023) [34]. The combination of high humidity and high temperatures accelerates both fungal growth and metabolic activity.

Temperature plays a crucial role in fungal physiology and toxin biosynthesis. *Aspergillus flavus* exhibits maximum growth rates between 25°C and 30°C, temperatures commonly found in warm climates where poultry production is concentrated (Tai *et al.*, 2020) [47]. While low temperatures may temporarily inhibit fungal activity, metabolism resumes when favorable thermal conditions return, allowing for delayed contamination events.

Storage infrastructure and management practices critically influence contamination outcomes. Inadequately designed storage facilities that lack proper ventilation, temperature control, or humidity management become incubators for fungal growth (Villers, 2014) [49]. Even initially clean grain can become severely contaminated during storage if environmental conditions favor fungal activity, underscoring the importance of continuous monitoring and environmental control throughout the feed supply chain.

Toxicological Manifestations in Poultry

1. Acute and Chronic Intoxication

Toxicity in birds exposed to aflatoxin varies significantly based on the dose, duration, and individual bird susceptibility.

Acute Aflatoxicosis

Acute intoxication occurs following a single, high-level exposure, leading to the rapid appearance of symptoms like extreme lethargy, a complete refusal to eat, and sudden death (Dabuo *et al.*, 2022) [10]. Since the liver is where the toxin is primarily processed, it suffers severe oxidative injury, which causes hepatocellular necrosis and can result in deadly organ failure (Alnuimy, 2024) [3].

The mechanism for this acute toxicity is the formation of highly reactive epoxide intermediates during the liver's metabolic process. These reactive compounds covalently bond with cellular components, including DNA, proteins, and lipids, thereby interfering with normal cell function (Cao *et al.*, 2022). This resultant oxidative damage sets off a chain of pathological events, such as membrane lipid peroxidation, mitochondrial dysfunction, and ultimately, cell death (Engin and Engin, 2019) [14].

Chronic Aflatoxicosis

Chronic intoxication develops gradually from continuous, low-level exposure over long periods. Unlike the rapid onset of acute effects, chronic damage accumulates slowly, becoming noticeable only after several weeks or months (Benkerroum, 2020) [7]. Birds suffering from this exhibit a progressive decline in growth rates, significantly poorer feed conversion efficiency, and increased vulnerability to infectious diseases.

Economic Impact and Mitigation

Both acute and chronic aflatoxicosis impose a substantial economic burden on commercial poultry farming. Losses include higher mortality rates, decreased growth performance, increased feed costs due to poor conversion, and greater veterinary expenditures for managing associated infections (Yohannis *et al.*, 2025) [51].

To effectively mitigate this problem, a thorough approach is necessary, encompassing rigorous feed quality control, the use of mycotoxin binders, nutritional support, and strict adherence to regulatory limits that cap AFB1 concentrations in poultry feed at 20 ppb or lower (Mengesha *et al.*, 2024; Negash, 2018) [33, 35]. Figure (1)

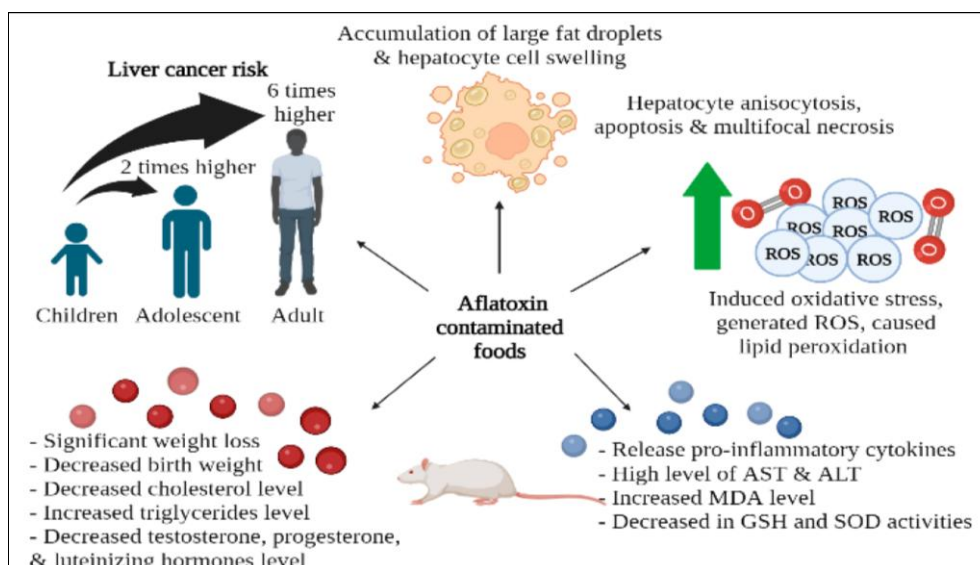


Fig 1: accumulation of aflatoxin and contaminated food chain

2. Hepatotoxicity and Pathological Changes

- **primary Target Organ:** The liver is the main target organ for aflatoxin-induced pathology due to its central role in xenobiotic metabolism (Alnuimy, 2024) [3].

Histopathological Lesions

- Hepatomegaly (enlarged liver).
- Pronounced fatty infiltration.
- Proliferative bile ductule changes.
- Hepatocellular carcinoma (in severe chronic cases) (Ali *et al.*, 2021) [2].

Progression of Damage

- Direct toxic injury and compensatory cellular responses occur.
- Continued exposure leads to progressive hepatic fibrosis, where damaged hepatocytes are replaced by fibrous connective tissue.
- This ultimately results in cirrhosis and compromised liver function (Fouad *et al.*, 2019) [17].
- The compromised liver impairs essential metabolic, synthetic, and detoxification functions, leading to overall health deterioration.

Impact on Poultry Production/Parameters

- **Laying Hens:** Marked reductions in egg production and deterioration of egg quality (shell strength, albumen quality, yolk composition) (Ojo *et al.*, 2022) [38].
- **Broiler Chickens:** Impaired growth, poor feed efficiency, and reduced carcass quality, all negatively impacting economic returns.

3. Immunosuppression and Disease Susceptibility

Aflatoxins exert profound immunosuppressive effects on both the innate and adaptive immune systems in poultry, significantly increasing susceptibility to disease (Hou *et al.*, 2022) [19]. This immune dysfunction increases morbidity and mortality rates within affected flocks, especially when birds encounter infectious challenges (Alwetaid *et al.*, 2023) [4]. The innate immune system, which provides immediate, nonspecific defense against pathogens, is significantly impaired under aflatoxin exposure. Phagocytic cells, including macrophages and dendritic cells, exhibit reduced functions in identifying, engulfing, and destroying invading microorganisms (Mehrzaad *et al.*, 2018) [32]. This compromised initial defense allows pathogens to establish infection more easily and progress more rapidly than in healthy birds, as shown in Figure (2). RetryClaude can make mistakes. Please double-check responses.

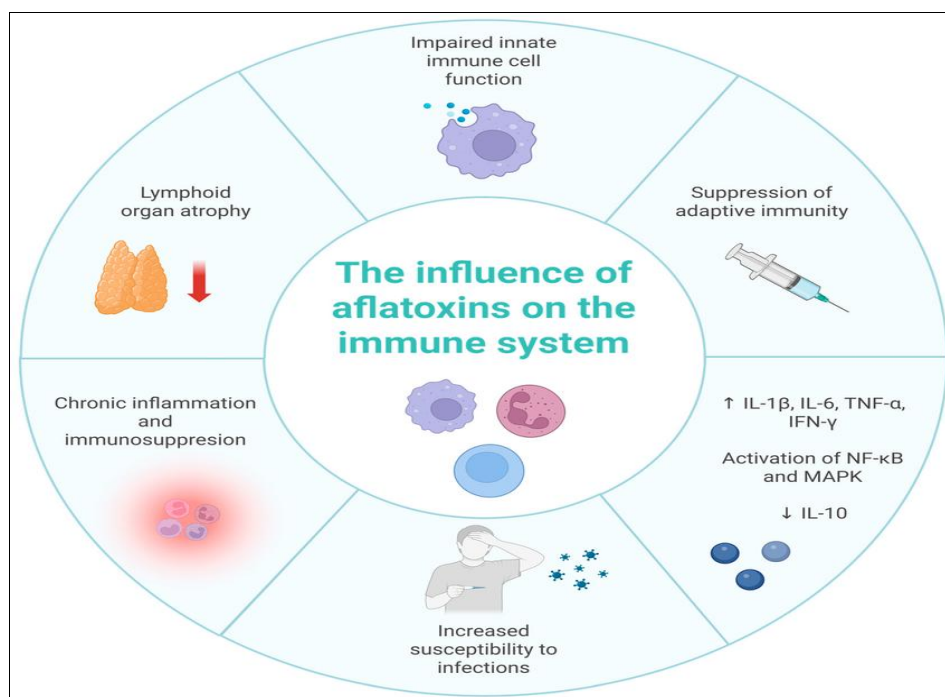


Fig 2: Exposure to aflatoxins can lead to immune dysregulation, impaired response to vaccination, upregulation of pro-inflammatory cytokines and genes, inflammation, and overall immunosuppression.

Adaptive immunity, responsible for developing long-term, specific protection against specific pathogens, is similarly compromised. Aflatoxin exposure inhibits the proliferation and differentiation of lymphocytes, impairing the development of both cellular and humoral immune responses (Kraft *et al.*, 2021) [25]. Antibody production after vaccination or natural infection is significantly reduced, leaving vaccinated birds inadequately protected against target diseases (Li *et al.*, 2019; Olariu *et al.*, 2025) [39]. The practical consequences of immunosuppression are particularly evident in increased susceptibility to common poultry diseases.

Viral infections, including Newcastle disease and avian influenza, bacterial pathogens such as *Escherichia coli*, and parasitic challenges cause more severe outbreaks in flocks exposed to aflatoxin (El Miniawy *et al.*, 2014; Sattar *et al.*, 2016) [44]. Secondary infections become more common and difficult to control, necessitating increased use of antimicrobials and raising concerns about the development of drug resistance (Valencia-Quintana *et al.*, 2020) [48]. Recovery from disease is prolonged in immunosuppressed birds, and previously controlled diseases may reappear. These immune dysfunctions directly translate into reduced

productivity, increased drug costs, and significant economic losses across the poultry industry (Chain *et al.*, 2020) ^[9].

Biochemical Mechanisms of Toxicity

1. Absorption and Metabolic Transformation

Following ingestion, aflatoxins undergo rapid absorption through the gastrointestinal mucosa and enter hepatic circulation for metabolic processing. The liver's cytochrome P450 enzyme system, particularly CYP1A and CYP3A isoforms, catalyzes the oxidation of AFB1 to its highly reactive 8,9-epoxide derivative (Dohnal *et al.*, 2014) ^[11]. This epoxide metabolite possesses extreme electrophilic reactivity, enabling covalent binding to nucleophilic sites on cellular macromolecules.

The aflatoxin-epoxide forms adduct with DNA, RNA, and proteins, disrupting normal cellular processes and triggering carcinogenic pathways (Cao *et al.*, 2022). DNA adduct formation is particularly significant, as these lesions interfere with replication and transcription, potentially leading to mutations and neoplastic transformation. The persistence of these adducts and inadequate DNA repair contribute to the carcinogenic potential of aflatoxins.

2. Oxidative Stress and Inflammatory Cascades

The metabolic processing of aflatoxins generates substantial quantities of reactive oxygen species (ROS), overwhelming cellular antioxidant defense systems and inducing severe oxidative stress (Finotti *et al.*, 2021) ^[16]. These free radicals attack multiple cellular components including membrane phospholipids, structural and enzymatic proteins, and nucleic acids. Lipid peroxidation disrupts membrane integrity and function, while protein oxidation impairs enzymatic activities and structural integrity.

The oxidative damage initiates inflammatory signaling cascades that amplify tissue injury. Pro-inflammatory cytokines are upregulated, and inflammatory cell infiltration occurs, further contributing to tissue damage through additional ROS production and protease release (Roze *et al.*, 2015) ^[41]. This inflammatory response, while initially protective, becomes destructive when sustained, leading to chronic tissue injury and fibrosis.

Impact on Production Parameters

1. Growth Performance and Feed Utilization

Aflatoxin exposure consistently produces negative effects on growth rates and feed conversion efficiency in all classes of poultry. Broiler chickens exhibit reduced weight gain, prolonged time to market weight, and increased feed consumption per unit of gain (Fouad *et al.*, 2019) ^[17]. Layer hens demonstrate decreased body weight maintenance and impaired development during the growing phase. These growth impairments result directly from metabolic disruption, reduced feed intake, impaired nutrient absorption, and increased maintenance requirements due to toxin detoxification demands.

2. Reproductive Function

Reproductive performance suffers across multiple parameters in aflatoxin-exposed birds. Layer hens show reduced egg production, decreased egg size, and compromised shell quality (Eze *et al.*, 2018) ^[14]. Fertility rates decline in breeding stock, and hatchability of fertile eggs is reduced.

Embryonic development may be impaired, leading to increased early and late embryonic mortality. These reproductive deficits stem from both direct toxic effects on reproductive tissues and indirect effects through metabolic and nutritional imbalances.

3. Residue Transfer to Products

A critical food safety concern involves the transfer of aflatoxins from contaminated feed into poultry products consumed by humans. Aflatoxin residues, particularly aflatoxin M1 (a hydroxylated metabolite of AFB1), accumulate in eggs and can be detected in muscle tissue (Suleman *et al.*, 2022) ^[48]. While bioaccumulation factors are relatively low, sustained consumption of contaminated feed leads to detectable residues in products. This transfer pathway represents a direct human health risk and underscores the necessity of stringent feed quality control.

Detection and Analytical Approaches

1. Conventional Analytical Techniques

Multiple validated analytical methods exist for quantifying aflatoxins in feed matrices and biological samples. High-Performance Liquid Chromatography (HPLC) with fluorescence detection provides sensitive, accurate quantification and remains the gold standard method (Mahfuz *et al.*, 2020) ^[31]. Thin-Layer Chromatography (TLC) offers a more economical though less sensitive alternative for screening purposes. Enzyme-Linked Immunosorbent Assay (ELISA) provides rapid, relatively simple analysis suitable for field or feed mill applications, though sensitivity and specificity vary among commercial kits.

2. Advanced Detection Technologies

Recent technological developments have expanded analytical capabilities for rapid aflatoxin detection. Fluorescence spectroscopy coupled with multivariate statistical analysis and machine learning algorithms enables rapid, non-destructive screening of feed and grain samples (Wang *et al.*, 2025) ^[50]. These emerging technologies promise improved efficiency in surveillance programs, allowing more frequent testing and better contamination control throughout the feed production chain.

Mitigation and Management Strategies

More than 500 mycotoxins have been identified in animal feed, with AFB1, ZEN, DON, FB1, OTA, and T-2 being the most prevalent in maize, wheat, barley, peanuts, and their by-products. These mycotoxins exhibit cytotoxicity, hepatotoxicity, genotoxicity, immunotoxicity, and reproductive toxicity, seriously threatening animal health, productivity, and product safety. Recent surveys indicate that 60–80% of feed samples show detectable mycotoxin contamination, with prevalence rates exceeding 79% for certain toxins in Chinese feed in 2021. Therefore, developing effective mycotoxin control strategies has become a priority for researchers and the feed industry. Current mitigation approaches focus on biodegradation using novel microorganisms, modified sorbents, nutritional interventions, and elucidating toxicity mechanisms to develop targeted antidotes, as illustrated in Figure (3).

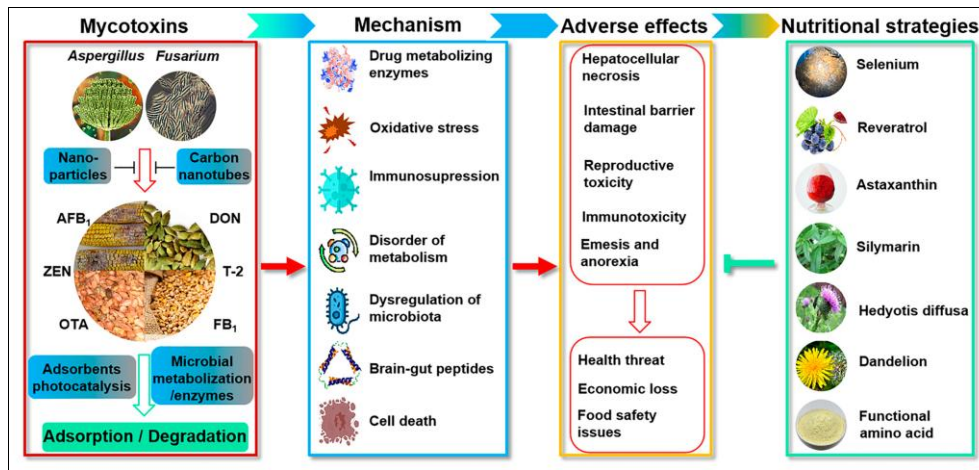


Fig 3: Occurrence, toxicity and control of mycotoxins in animal feeds.

1. Mycotoxin Binding Agents

The incorporation of adsorbent materials into contaminated feed represents a practical approach to reducing aflatoxin bioavailability. Aluminosilicate clays, particularly bentonite and montmorillonite, along with activated carbon products, effectively bind aflatoxins in the gastrointestinal tract, preventing absorption (Ahmadou *et al.*, 2021) ^[1]. These binders work through physical adsorption mechanisms, forming stable complexes that pass through the digestive system without releasing bound toxins. Efficacy varies among products depending on clay composition, particle size, and inclusion rate (Kolawole *et al.*, 2022) ^[24].

2. Antioxidant Supplementation

Nutritional strategies employing antioxidant compounds help counteract the oxidative stress induced by aflatoxin metabolism. Vitamin E and selenium supplementation enhances endogenous antioxidant defense systems, supporting glutathione peroxidase activity and scavenging reactive oxygen species (Panda and Cherian, 2014) ^[40]. These nutrients work synergistically to protect cell membranes from lipid peroxidation and maintain cellular redox balance. Vitamin E stabilizes membrane structures through its lipid-soluble antioxidant properties, while selenium serves as a cofactor for glutathione peroxidase enzymes that neutralize peroxides (Finotti *et al.*, 2021) ^[16].

3. Biological Detoxification Approaches

Probiotic microorganisms and aflatoxin-degrading enzymes offer biological approaches to reducing toxin levels and mitigating toxic effects. Certain bacterial and yeast strains bind aflatoxins or enzymatically degrade them to fewer toxic metabolites (Guan *et al.*, 2021) ^[18]. These biological agents operate in the gastrointestinal tract, reducing toxin absorption and subsequent systemic exposure. Research demonstrates particular efficacy in protecting intestinal, hepatic, and renal cells from aflatoxin-induced cytotoxicity (Kępka-Borkowska *et al.*, 2025) ^[22]. Probiotic supplementation additionally supports intestinal health and immune function, providing multiple benefits beyond toxin mitigation (Krysiak *et al.*, 2021) ^[26].

4. Phytochemical Interventions

Plant-derived bioactive compounds demonstrate protective effects against aflatoxicosis through multiple mechanisms. Curcumin, the principal bioactive component of turmeric, has received particular attention due to its multifaceted

protective properties (Jafarzadeh *et al.*, 2022) ^[20]. As a potent antioxidant, curcumin neutralizes reactive oxygen species and prevents oxidative damage to cellular components (Finotti *et al.*, 2021) ^[16]. Its anti-inflammatory properties reduce cytokine production and inflammatory enzyme activity, limiting inflammatory tissue damage (Kumar *et al.*, 2025) ^[27].

Curcumin exhibits hepatoprotective effects by supporting liver cell regeneration and enhancing detoxification pathways, thus protecting against hepatic necrosis and fibrosis. Additionally, curcumin supports immune function, helping maintain defense mechanisms against infectious challenges (Saha Turna *et al.*, 2024) ^[42]. These combined properties make curcumin and similar phytochemicals valuable components of comprehensive aflatoxicosis management programs.

Regulatory Framework and Food Safety

Governmental and international regulatory agencies establish maximum permissible concentrations of aflatoxins in animal feeds and food products to protect public health (Ogodo and Ugbogu, 2016) ^[37]. These regulatory standards vary among jurisdictions but typically set very low limits for AFB1 in feeds and even lower limits for aflatoxin M1 in milk and eggs. Rigorous monitoring programs and enforcement of these standards are essential for ensuring food safety (Negash, 2018) ^[35].

The transfer of aflatoxin residues from feed through animals to human food creates direct public health risks. Prolonged human exposure to aflatoxin-contaminated foods is associated with serious health consequences including hepatotoxicity and hepatocellular carcinoma (Awuchi *et al.*, 2020) ^[6]. By implementing comprehensive control programs including feed testing, proper storage management, regulatory compliance, and product monitoring, the risks of aflatoxin contamination in the food supply can be substantially reduced (Jallow *et al.*, 2021; Negash, 2018) ^[21, 35].

Conclusion

Aflatoxin contamination poses multifaceted challenges to poultry production systems, affecting animal health, productivity, and food safety. The complex interaction between fungal ecology, environmental conditions, toxin biochemistry, and avian physiology requires comprehensive and integrated management approaches. Effective control

requires attention to all stages of the production chain, from crop cultivation and harvest, through feed manufacturing, storage, and feeding.

Multiple mitigation strategies show promising results in reducing aflatoxin exposure and its effects, including physical adsorbents, nutritional antioxidants, biological detoxification agents, and phytochemical supplements. The implementation of sensitive detection methods and rigorous monitoring programs enables early identification of contamination events and timely intervention. Adherence to established regulatory standards protects both animal welfare and human food safety.

Continued research into novel control approaches, improved detection technologies, and better understanding of toxicity mechanisms remains critically important for addressing this persistent challenge in global poultry production. The development and adoption of comprehensive mycotoxin management programs will be essential for ensuring sustainable poultry production and maintaining food security in the face of ongoing contamination risks.

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