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Mycotoxins in Monogastric Animal Nutrition: A Review of the Threats and Mitigation Strategies

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ABSTRACT

Mycotoxins are low-molecular-weight secondary metabolites produced by various fungal species that threaten the health and productivity of monogastric and companion animals. Major mycotoxins, including aflatoxins, deoxynivalenol, fumonisins, ochratoxin A, trichothecenes, and zearalenone, contaminate raw ingredients and finished feeds globally. Even at low concentrations, mycotoxins can cause mycotoxicosis, while higher concentrations induce acute toxicity, organ damage, immunosuppression, reduced performance, and mortality, resulting in significant economic losses. More crucially, climate change is shifting the impact on the occurrence and geographical distribution of mycotoxins, as rising temperatures and humidity keep promoting fungal growth and toxin production in feed and food systems. This rising contamination contributes significantly to global economic losses by reducing animal performance, compromising feed quality, and increasing mitigation costs. Therefore, preventing mycotoxin contamination requires comprehensive strategies, including good agricultural practices, timely and controlled harvesting, and proper storage conditions to minimize fungal growth. Additionally, advancements in biotechnological applications, such as the development of mycotoxin-degrading enzymes, genetically engineered microorganisms capable of detoxifying mycotoxins, and the use of toxin binders, provide innovative tools to mitigate mycotoxin contamination. Such proactive measures are essential for safeguarding animal health, ensuring productivity, and reducing economic losses associated with mycotoxin exposure. This current review examines the effects of mycotoxins on monogastric and companion animals and highlights emerging threats alongside modern preventive strategies.

Keywords: *Biotechnological detoxification, feed safety, food-borne contaminants, gut microbiota, mitigation strategies, mycotoxicosis, mycotoxins, and toxin binders.*

INTRODUCTION

The term "Mycotoxin" was derived from the Greek word "mycos," meaning fungi, and the Latin word "toxicum," meaning poison. Therefore, mycotoxins are defined as the secondary metabolites produced by various fungal species, particularly moulds (Hussein and Brasel, 2001). Many of these fungal species are reported to produce mycotoxins in feed, arising mainly during crop growth, during and after harvesting, storage, transportation, and processing (Zain, 2011). The most toxic mycotoxins of concern are produced by fungi from the genera *Penicillium*, *Alternaria*, *Fusarium*, and *Aspergillus* (Allah Ditta et al., 2019). Among the various mycotoxins, zearalenone (ZEN), fumonisins (FUM), aflatoxins (AF), ochratoxin A (OTA), and trichothecenes are the most prevalent and

pose significant risks to the productivity and health of poultry and other livestock (Streit et al., 2013). These toxins are produced by fungi under specific conditions, with their growth and production influenced by several factors, including geographic location, seasonal variations, environmental conditions such as drought, and the timing of harvest (Awuchi et al., 2021). Although the overall prevalence of mycotoxin contamination may appear low, global analyses of feed and grain samples have highlighted alarming trends. These studies reveal that grains can exhibit extremely high concentrations of mycotoxins and, in many cases, are contaminated with multiple types of mycotoxins simultaneously (Streit et al., 2013). This multifaceted contamination underscores the complexity of managing mycotoxin risks and the need for comprehensive monitoring and mitigation strategies in agricultural and feed production systems. These microscopic fungi have negative effects on the nutritional and organoleptic attributes of feeds (Cegielska-Radziejewska et al., 2013). Moulds that grow on feed utilize the readily available nutrients and result in a 5 to 100% loss of feed nutrients (Okoli et al., 2006).

In addition to compromising the organoleptic and nutritional quality of feed, moulds are notorious for producing mycotoxins that contaminate animal feed, posing significant risks to both animal and human health (Shareef, 2010). When animals consume mycotoxin-laden feed, they may experience a range of adverse effects, from acute health disturbances to chronic toxic conditions, potentially leading to mortality (Binder et al., 2007; Venâncio and Paterson, 2007). This contamination, however, is not confined to the animal population; it extends to humans through secondary contamination via the consumption of animal products such as meat, eggs, or milk (Okioma, 2009). The toxic effects of mycotoxins are multifaceted, encompassing both immediate and long-term health implications. These compounds are known for their potent toxicological activities, which include estrogenic effects that disrupt hormonal balance, teratogenic effects that interfere with normal fetal development, and carcinogenic properties that increase the risk of cancer (Cegielska-Radziejewska et al., 2013). These findings underscore the critical importance of addressing mycotoxin contamination in feed to safeguard both animal welfare and public health.

The identification of harmful microbiota is crucial for quality control as it gives helpful data on mycotoxin production, thus serving as an indicator for the determination of feed hygiene quality (Khosravi et al., 2008). The European Union regulates mycotoxin levels, and the maximum limit for aflatoxin B1 in poultry feed has been set at 0.02–0.05 ppm (European Commission, 2006; 2011). Despite hundreds of mycotoxins, the regulatory guidelines for maximum tolerated levels are established for only a few mycotoxins (Bennett and Klich, 2003). Mycotoxins affect productivity and health in poultry and pig farming, which has led to regulatory guidelines for maximum tolerated levels of aflatoxins and a small number of fumonotoxins and ochratoxins. This limit varies depending on factors such as the type of mycotoxin, intended use, feed composition, raw materials, diet, and animal species, as well as differing regulations across countries and regulatory bodies. The European Union provides guidelines on diets and raw materials, with the differences linked to the stage of production and age of the animal (Guerre, 2016). The Food and Drug Administration (FDA) of the U.S. has also established guidelines that differ with the diet, intended use, the effect on age, and raw material (Food and Drugs Authority, 2011). In Sub-Saharan Africa, particularly Ghana, mycotoxin limits in food and feed are primarily regulated by the Ghana Standards Authority (GSA) and the Food and Drugs Authority (FDA). However, the permissible levels vary and depend on the mycotoxin type, commodity, and intended use (human consumption or animal feed). The major mycotoxins regulated in Ghana include aflatoxins (B_1, B_2, G_1, G_2, M_1), ochratoxin A (OTA), and fumonisins (FB_1, FB_2). Nevertheless, even with regulations, enforcement remains inconsistent, especially in informal markets where most of the commodities are traded. For instance, Kortei et al. (2021) found frequent aflatoxin exceedances in maize and ground nuts, highlighting gaps in monitoring. The intake of contaminants in low concentrations causes mycotoxicosis in poultry and swine, resulting in suboptimal production, poor feed efficiency, and poor growth. On the contrary, high concentrations of toxins are associated with acute clinical

symptoms of specific organs, avian physiology, the immune system, and mortality (Mabbett, 2004). Plants and animal-derived foods are greatly exposed to mycotoxins (Yang et al., 2020).

This review examines key aspects of mycotoxins in animal feed, with a particular focus on monogastric species, particularly poultry, swine, and companion animals. It explores the prevalence of mycotoxins in feed ingredients and the consequences for animal health when contaminated feed is consumed. Of particular importance in this review are recent developments and strategies to mitigate mycotoxin exposure by preventing mold growth. Finally, this review addresses mycotoxin production and its implications for monogastric animal producers, feed manufacturers, and animal productivity, providing critical insights to mitigate risks and enhance feed safety.

Literature Search and Strategy

We conducted a systematic literature search to identify relevant studies on mycotoxins, their impacts on monogastric and companion animals, and the preventive strategies. The search was made in major scientific databases, including Scopus, MDPI, Web of Science, PubMed, ScienceDirect, African Journal Online, and Google Scholar, for peer-reviewed literature. Search terms were applied using combinations of the following keywords: mycotoxins, mycotoxicosis, feed safety, food-borne contaminants, monogastric and companion animals, gut microbiota, biotechnological detoxification, toxin binders, and mitigation strategies. Where applicable, database-specific controlled vocabulary terms were also used to enhance retrieval accuracy.

The search was limited to journal articles published in the English Language, including original research papers and review articles relevant to mycotoxin occurrence, toxicological effects, performance and health impacts, and preventive or mitigation approaches in animal production systems. Studies that were not directly related to animal health, feed contamination, mycotoxicosis, or mycotoxin control were excluded. Additionally, reference lists of selected publications were manually screened to identify further relevant studies.

Mycotoxins Present in Monogastric and Companion Animal Feeds

About 300-400 groups of mycotoxins have been reported until now (Yang et al., 2020a). However, only a few classes are under consideration concerning safety and economic impacts (Santos Pereira et al., 2019). Concerns associated with feeds affecting livestock health are related to aflatoxins (AFs), fumonisins (FUMs), ochratoxins (OTAs), trichothecenes (TRCs), and zearalenone (ZEN) (Pinotti et al., 2016). Table 1 highlights major mycotoxins, including their fungal sources, commonly contaminated feed ingredients, principal toxic effects in animals, species sensitivity, and established regulatory or guideline limits. The International Agency for Research on Cancer (IARC) categorizes mycotoxins into three major groups according to their demonstrated carcinogenic potential in humans. The most hazardous group, Group 1, includes Aflatoxin B1 (AFB1) and its hydroxylated metabolite Aflatoxin M1 (AFM1), which has conclusive evidence of carcinogenicity in humans. Group 2 contains mycotoxins classified as possibly carcinogenic, namely Ochratoxin A (OTA) and Fumonisin B1 (FB1), where limited evidence exists for human carcinogenicity but sufficient evidence in experimental animals. Lastly, Group 3 comprises mycotoxins, including Deoxynivalenol (DON), Zearalenone (ZEN), Patulin (PAT), and Nivalenol (NIV), that currently cannot be classified regarding their carcinogenicity to humans due to inadequate evidence from both human and animal studies (Smith et al., 2016). This systematic classification by IARC provides a crucial framework for understanding the potential cancer risks posed by different mycotoxins in human populations. Figure 1 illustrates the major mycotoxins and the fungal species responsible for their production.

Aflatoxin

Aflatoxins (AF) are highly toxic secondary metabolites predominantly produced by *Aspergillus flavus* and *Aspergillus parasiticus*. These mycotoxins thrive in warm climates and can contaminate crops both pre-harvest (in the field) and post-harvest (during storage), and animal feed. Hot and dry conditions favour pre-harvest contamination, whereas hot and humid storage conditions increase mold growth, making post-

Table 1: Summary of major mycotoxins in animal feed and products, including fungal sources, affected feed ingredients, toxic effects, and regulatory limits (Streit et al., 2012; Peng et al., 2018; Xu et al., 2022)

Mycotoxin	Major Fungal Sources	Common Feed Ingredients Affected	Main Effects in Animals	Species Most Sensitive	Common Regulatory/ Guideline Limits*
Aflatoxins (AFB1, AFB2, AFG1, AFG2)	*Aspergillus flavus*, *Aspergillus parasiticus*	Maize, groundnut cake, cottonseed cake, stored grains	Reduced growth, liver damage, immunosuppression, poor feed efficiency, and mortality	Poultry, pigs, fish, young animals	5–20 ppb in complete feeds; dairy cattle often ≤20 ppb
Ochratoxin A (OTA)	*Aspergillus ochraceus*, *Penicillium verrucosum*	Cereals, wheat bran, barley, stored feed	Kidney damage, reduced egg production, infant deformity, and immunosuppression	Pigs and poultry	Commonly 50–250 ppb in feeds
Fumonisins (FB1, FB2)	*Fusarium verticillioides*, *Fusarium proliferatum*	Maize and maize by-products	Pulmonary oedema, liver damage, reduced performance, and neural disorders	Horses and pigs	Usually 5–50 ppm, depending on species
Zearalenone (ZEN)	*Fusarium graminearum*, *Fusarium culmorum*	Maize, wheat, barley	Oestrogenic effects, infertility, swollen vulva, reproductive failure, and weak piglets	Swine	Typically, 0.1–3 ppm
Deoxynivalenol (DON / Vomitoxin)	*Fusarium graminearum*, *Fusarium culmorum*	Wheat, maize, barley	Feed refusal, vomiting, poor growth, and immune dysfunction	Swine	Usually 0.9–10 ppm
T-2 Toxin / HT-2 Toxin	*Fusarium sporotrichioides*, *Fusarium poae*	Oats, maize, wheat	Oral lesions, hemorrhage, reduced immunity, poor growth	Poultry and pigs	Commonly 0.1–1 ppm
Ergot Alkaloids	*Claviceps purpurea*	Rye, wheat, barley, grasses	Reduced blood circulation, gangrene, and reproductive disorders	Cattle, sheep, horses	Often <1,000 ppb total alkaloids
Patulin	*Penicillium*, *Aspergillus*, *Byssochlamys* spp.	Fruits, silage, spoiled feed	Gastrointestinal irritation, immune suppression	Young animals	Usually controlled mainly in food products rather than feeds
Citrinin	*Penicillium citrinum*, *Monascus* spp.	Stored grains	Kidney toxicity, reduced growth	Poultry and pigs	No universal limits; monitored in high-risk feeds

**Limits vary by country, species, age, and feed type (FDA, EU, Sub-Saharan Africa, and national regulations may differ).*

harvest contamination of mycotoxins more likely. These toxic compounds are further classified into four major types: B1, B2, G1, and G2. Among these, aflatoxin B1 (AFB1) is the most notorious; not only is it the most prevalent, but also the most toxic. It contaminates staple crops such as maize, peanuts, and oilseeds, posing a significant risk to both human and animal health. For food-producing animals and humans, the danger is particularly alarming. This is because, when consumed, AFB1 works as a potent procarcinogen, directly connected to liver cancer. Animals that consume contaminated feed suffer disastrous effects, causing a ripple effect on food safety and agricultural productivity. In poultry, even low levels of aflatoxin exposure result in stunted growth, weakened immunity, and toxin residues in eggs and meat (Gao et al., 2020). Studies have shown that AFB1 suppresses egg production and compromises the immune systems of hens, with research on turkeys revealing similar damage to spleen function (Ochieng et al., 2021). Pigs and dairy animals are equally vulnerable, facing reproductive failures, immune dysfunction, and cellular damage in critical tissues, such as the mammary glands (Fouad et al., 2019; Park et al., 2019). Dairy cows may undergo one of the most insidious changes. When they consume AFB1-contaminated feed, their livers convert it to aflatoxin M1 (AFM1), which enters the milk supply. Although less effective than AFB1, AFM1 is far from harmless; sustained exposure, even at low levels, can cause chronic toxicity, raising serious concerns among dairy consumers worldwide.

Recent studies indicate that 64% to 100% of poultry feed samples tested in various regions show detectable levels of aflatoxin contamination, with contamination rates varying significantly based on geographical location, storage conditions, and feed ingredient sourcing (Gao et al., 2020). In countries such as Nigeria, Ghana, Kenya, and Uganda, contamination frequently exceeds the European Union's safety limit of 20 µg/kg (Aboagye-Nuamah et al., 2021), putting both animal and human populations at risk. The severity of aflatoxin's impact depends on exposure duration and concentration, but one thing is clear: this is not a localized issue. It is a global food safety emergency, demanding immediate action in monitoring, mitigation, and public awareness. Without intervention, aflatoxins will continue to undermine health, agriculture, and economic stability, especially in regions where food security is already fragile.

Ochratoxin

Ochratoxin A (OTA) is a mycotoxin produced primarily by the fungal species *Aspergillus carbonarius*, *Aspergillus ochraceus*, and *Penicillium verrucosum*. It is commonly produced under improper storage conditions, particularly in humid environments, and therefore more prevalent in cooler climates (Yang et al., 2020b). Research has demonstrated that OTA-producing *Penicillium* and *Aspergillus* species are strongly linked to nephropathy, renal fibrosis, and other renal and urinary tract pathologies (Kępińska-Pacelik and Biel, 2021). Analysis of feed ingredients such as rye, barley, wheat, maize, millet, sorghum, rice, and oilseeds revealed the presence of OTA (Barlow et al., 2006; Bryden, 2012). Toxin production often occurs during the storage period, but pre-harvest contamination has also been reported. Structurally, OTA consists of an isocoumarin scaffold conjugated to phenylalanine through an amide bond. This unique architecture mediates dual effects, thereby inhibiting eukaryotic protein synthesis and strongly binding to plasma proteins through hydrophobic and ionic interactions (Neme and Mohammed, 2017). Due to its high affinity for plasma proteins, particularly albumin, OTA forms stable complexes that prolong its systemic circulation. This strong binding reduces renal clearance and hepatic metabolism, resulting in extended tissue persistence and bioaccumulation in animals, especially in the kidneys, liver, and adipose tissue (Liu et al., 2022).

Pigs are particularly vulnerable to OTA, and evidence also points to a connection between OTA and swine nephropathy (Heussner and Bingle, 2015). Liver damage and necrosis of intestinal and lymphoid tissue can be the consequence of the high dietary dosage of this toxin. Furthermore, there has been public concern about swine health due to the presence of OTA in animal-derived food (Edite Bezerra da Rocha, 2014). OTA has been frequently reported in the breast muscles of chickens. Despite their low level in poultry tissues, they are significant due to the high consumption of poultry products. Regular

consumers of poultry meat and eggs can be at risk of exposure, as this toxin has also been reported in small amounts in eggs (Streit et al., 2012).

Fumonisins

Fumonisins (FUM) are mycotoxins produced by several species of the genus Fusarium, notably *Fusarium moniliforme*, *F. verticilioides*, and *F. oxysporum*. The B-series FUM: B1, B2, B3, and B4, are the most important fumonisins out of 16 fumonisins analogs known to date. These analogs include A1, A2, A3, B1 (FB1, FB2, FB3 and FB4), C1, C3, C4, AK1, PH1a, PH1b, P1, P2, and P3. Fumonisins are hydrophilic, and this hydrophilicity hinders their study as it has rendered various other mycotoxins undiscovered. The most extensively researched fumonisin is B1 (Heussner and Bingle, 2015).

Fumonisin B1 (FB1) has been identified as a possible human carcinogen and is considered the most toxic member of the FUM family (Fink-Gremmels and Merwe, 2019). Even though fumonisin B2 (FB2) has toxicological significance, it is generally regarded as less potent than FB1. In humans, FB2 specifically disrupts sphingolipid biosynthesis as it has some structural similarities, leading to severe cellular dysfunction as sphingolipids play a critical role in cell-to-cell interactions, membrane integrity, signal transduction, growth factor regulation, and cell-matrix and intracellular communication (Anumudu et al., 2025; Riley & Merrill, 2019). Importantly, FBs are not considered directly genotoxic, as they are not associated with DNA damage (Marroquín-Cardona et al., 2014). Horses, swine, and rabbits are predominantly more sensitive to FBs and are more associated with significant diseases than cattle. In horses, ingestion of FBs has been linked to leukoencephalomalacia, a fatal neurological disorder that causes lethargy, poor food intake, blindness, and ultimately death. In pigs, FB1 consumption has been associated with weakness, dyspnea, and death (Heussner and Bingle, 2015). In addition, these mycotoxins also show serious adverse effects on liver functionality (Gao et al., 2020).

Trichothecenes

More than 150 structurally related compounds are found in this class of fungal metabolites. Genus *Phomopsis*, *Trichotecium*, *Fusarium*, *Trichoderma*, *Myrothecium*, *Stachybotrys*, *Verticimonosporium*, and others produce trichothecene (TRC). The word trichothecene is derived from trichothecin, meaning the first identified member within this family of mycotoxins. All TRCs possess a tetracyclic 12,13-epoxy trichothene skeleton, which underpins their toxicological properties. While many TRC compounds have been identified, only a few of them exist naturally. The most epidemiologically and toxicologically significant TRCs include deoxynivalenol (DON), toxin T2, nivalenol (NIV), toxin HT2, and diacetoxyscirpenol (DAS). The trichothecenes are recognized for their strong characteristic to halt eukaryotic protein synthesis. They disrupt the initiation, elongation, and termination steps vital to protein synthesis. Moreover, trichothecenes were the first compounds reported to inhibit peptidyl transferase activity. Chemically, TRCs are divided into four types (A to D), McCormick et al. (2011), of which types A and B are the most toxicologically relevant. Type B TRCs are mainly represented by DON and its derivatives (Marin et al., 2013).

DON is considered the least toxic type of TRCs, but it is the most prevalent, particularly in swine and poultry production systems. They are known to halt protein synthesis and reduce the immune response in animals. They mostly contaminate cereal grains such as rye, barley, and wheat. At high dietary levels, DON ingestion can induce vomiting, nausea, and diarrhoea. Equally, symptoms of DON contamination include oral lesions, low egg and milk production, abortion, and sometimes death. In monogastric animals, especially swine, DON contamination can lead to anorexia, vomiting, and overall poor performance (Malczak et al., 2025; Crome et al., 2026). Notably, DON has not been classified by IARC as a carcinogen in humans (Rodríguez-Carrasco et al., 2013).

Zearalenones

Zearalenones (ZEN) are also produced by *Fusarium*, and their derivatives are α-Zearalenol (α-ZEL) and β-Zearalenol (β-ZEL) (Liu et al., 2022). They are mostly found in barley, maize, wheat, and some other cereals. Contamination of ZEN mostly happens concurrently with contamination of DON and only rarely with AF. ZEN

has been reported to be stable at usual cooking temperatures and is partly removed at higher temperatures. The metabolites of ZEN may show a 10-times higher estrogenic activity than ZEN itself. The major metabolites of ZEN include zearalanone (ZAN), β-zearalenol (βZOL), α-zearalenol (αZOL), β-zearalanol (βZAL), and α-zearalanol (αZAL). Analogous to trichothecenes, ZEN is rapidly absorbed from the gastrointestinal tract (GIT) and is readily metabolized in animals. ZEN biotransformation in animals occurs through two metabolic pathways: hydroxylation, leading to αZOL formation and βZOL, and glucuronic acid conjugation, thereby facilitating detoxification and excretion (Yang et al., 2020b). ZEN is classified as a nonsteroidal oestrogen as it has some structural similarities with oestradiol (female sex hormone). Although ZEN is rarely associated with acute toxicity, its oestrogenic structure enables it to compete with natural oestrogens for receptor binding, thereby disrupting hormonal balance. It has been reported to cause adverse effects such as reproductive disorders and hyperestrogenism in humans and animals. For instance, high amounts of zearalenone in pig diets are associated with abortion, impaired conception, and other reproductive problems. In studies with rats, exposure to ZEN has been found to trigger effects like those caused by oestrogen. These effects include heavier uteruses and more developing follicles in the ovaries (Heussner and Bingle, 2015; Edite Bezerra da Rocha et al., 2014).

The oestrogenic potency of ZEN metabolites varies considerably. Among these, α-ZOL exhibits a higher binding affinity to oestrogen receptors than the parent compound ZEN, whereas β-ZOL displays the lowest receptor-binding capacity. Consequently, the species-specific conversion of ZEN to α-ZOL is regarded as a bioactivation process, while its conversion to β-ZOL is considered an inactivation pathway (Frizzell et al., 2011). It is worth noting that zearalenone has not been classified as carcinogenic to humans by the IARC (Rodríguez-Carrasco et al., 2013).

Effects of Climate Change on Mycotoxin Growth

Climate change continues to pose a significant threat to global food supply by increasing the prevalence of mycotoxins due to changes in weather patterns, rising temperatures, and increasing crop stress. Evidence shows that climate change–related factors, including elevated atmospheric CO_2 concentrations, rising temperatures, and alternating occurrence of severe droughts and heavy rainfalls, significantly influence mould proliferation and the occurrence of mycotoxins (Magan et al., 2003; Zingales et al., 2022). Notably, rising temperatures and increased humidity associated with climate change are promoting the geographic expansion and increased prevalence of mycotoxin-producing fungi, particularly species of *Aspergillus* and *Fusarium*, into previously unsuitable environments, thereby increasing contamination risks in staple crops such as maize, wheat, and groundnuts (Kos et al., 2023). It is important to note that different mycotoxigenic fungal species exhibit distinct ecological preferences, with some capable of thriving even under relatively low humidity conditions. Consequently, extreme and shifting climatic conditions can substantially influence the prevalence, geographic distribution, and diversity of mycotoxins. A recent and well-documented example of climate-induced mycotoxin risk is the increasing incidence of *Aspergillus flavus* infection and aflatoxin contamination during hot and dry summer periods in Southern Europe, areas where such contamination was previously rare under cooler climatic conditions. This change has been strongly linked with increased temperatures and prolonged drought periods that favour thermotolerant fungi and aflatoxin biosynthesis in crops such as maize and groundnuts (Casu et al., 2024; Nji et al., 2022). Similarly, in sub-Saharan Africa, climate variability characterized primarily by rising temperatures and erratic rainfall has worsened aflatoxin contamination in staple crops. For instance, a recent study in southern Africa has shown that fungal growth and host plant stress greatly increase the effects of reduced rainfall, drought stress, and aflatoxin levels in maize (Nji et al., 2024; Blackadar, 2016). Climate change may also indirectly enhance mycotoxin accumulation by altering local biological interactions within agro-ecological systems. For example, drought stress has been shown to increase insect pressure on maize, leading to kernel damage that facilitates fungal colonization and subsequent mycotoxin accumulation (Miller, 2008; Jouany, 2007),

a phenomenon now projected to intensify under future climate scenarios (Kos et al., 2023). These climatic pressures are expected to markedly influence mould proliferation and mycotoxin contamination under current conditions; therefore, concerted efforts are required to mitigate mycotoxin-related risks.

Impact of Mycotoxins on the Intestinal Microbiota in Monogastric and Companion Animals

Impact of Mycotoxins on the Intestinal Microbiota in Monogastric Animals

The gastrointestinal tract (GIT) contains a diverse and dynamic community of microorganisms that plays a vital role in the host's health. The gut microbiota contributes to protection against pathogenic bacteria through competitive exclusion and the release of antimicrobial compounds. Particularly, GIT is the primary site of mycotoxin-induced toxicity, and the resident microbiota plays a crucial role in modulating this toxicity (Guerre, 2020). Following dietary exposure, the gut microbiome modulates the absorption, metabolism, and overall toxicokinetics of mycotoxins. Accordingly, gut microbiota in monogastric and companion animals enzymatically modify mycotoxins in the GIT (Liew and Mohd-Redzwan, 2018). Depending on the type of mycotoxin, the toxic effects on monogastric animal performance can vary. The biotransformation of mycotoxins by gut microbiota may generate fewer toxic metabolites; however, microbial deconjugation can also activate masked mycotoxins, enhancing their toxic effects (Guerre, 2020). In addition, gut microbiota can reduce mycotoxin absorption across the intestine by binding to them (Guerre, 2020). Importantly, mycotoxins can modify the composition of gut microbiota, highlighting the need to study their impact on the gastrointestinal ecosystem. Coupled with the above, mycotoxins can exert antimicrobial-like properties and alter the existence of microbes in the gut. Furthermore, they can impair gut epithelial cells and stimulate intestinal secretions, enhanced mucin production, and immune cell diapedesis, leading to changes in chyme composition and the luminal environment that shapes gut microbiota (Ochieng et al., 2021). Through disruption of the gut microbial ecosystem, mycotoxin exposure can induce dysbiosis, an imbalance in gut microbiota composition that has been strongly associated with the pathogenesis of chronic diseases such as colorectal cancer, diabetes, and neurodegenerative disorders (Ochieng et al., 2021).

Several studies have demonstrated that DON and TRC facilitate the colonization of pathogenic bacteria in the gut of pigs, especially coliform bacteria (Liew and Mohd-Redzwan, 2018). Moreover, DON and OTA have been linked with the invasion of *Salmonella* into the bloodstream and other vital organs in both birds and pigs (Waché et al., 2009). FB1 can lead to an imbalance of sphingolipids in cells, especially gastrointestinal cells, and consequently facilitates the adhesion of pathogenic bacteria, leading to increased populations and prolonged infections (Grenier and Applegate, 2013). Regarding human health, colonization of *E. coli* and *Salmonella* in food-producing animals is of significant concern. These microbes cause serious health concerns. Mycotoxin exposure may increase the transmission of these pathogens, posing a risk to human health (Liew and Mohd-Redzwan, 2018). Dietary supplementation of broiler chickens with AFs has been shown to significantly increase ileal counts of *E. coli*, *Klebsiella*, *Salmonella*, and total gram-negative bacteria (Antonissen et al., 2015). The GIT of pigs is affected by the absorption of DON, which reduces nutrient absorption by the tract. This leads to epithelial injuries of the stomach and the intestine. The direct link between DON and the gut microbiota of pigs has been poorly investigated (Waché et al., 2009). Although AFB1 and DON have been reported to exert no measurable effect on the microbiota of poultry (Jahanian et al., 2017), available data remain limited, necessitating further research to evaluate the impact of mycotoxins on gut microbial dynamics.

Impacts of Mycotoxins on Companion Animals

Mycotoxins have attracted global attention due to substantial economic losses related to their effects on human health, animal production systems, household food security, and international trade (Liu et al., 2018). In recent years, the presence of mycotoxins in pet food has become a possible threat to the health of companion animals (Yang et al., 2023). In the U.S. and Canada,

mycotoxin-related losses to the livestock industry are estimated at approximately USD 5 billion annually (Liu et al., 2018). According to FAO (2001) and Leung et al. (2006), cats and dogs are particularly susceptible to the adverse effects of contaminated feed. This is due to their physiological sensitivity, relatively smaller body weight, and reliance on commercially produced feed, which increases the risk of chronic exposure to food-borne diseases. This vulnerability is further compounded by the inclusion of cereal-based products, which may be diverted into pet food formulations and can contain relatively higher levels of mycotoxins compared with raw grains. An estimated 20% of agricultural commodities within the European Union and up to 25% of global plant production are contaminated with mycotoxins, posing a significant risk to companion animal nutrition, particularly through cereal-derived ingredients commonly incorporated into pet foods. Given the high dependence of cats and dogs on commercially formulated diets, exposure to mycotoxins in contaminated feed may result in a broad spectrum of toxic effects. These compounds are classified into six major groups based on their target organs, including dermatotoxins affecting the skin and mucous membranes, cardiotoxins associated with cardiac dysfunction, nephrotoxins that impair renal function, hepatotoxins causing liver damage, neurotoxins impacting the central nervous system, and pulmonotoxins that induce inflammatory responses in lung tissue (Yang et al., 2020). Similarly, mycotoxins may be classified based on their specific effects on companion animals. In this regard, certain mycotoxins act as immunotoxins, compromising immune function and increasing susceptibility to infectious diseases.

Others behave as myco-hormones, disrupting normal hormonal regulation and endocrine balance, while some possess carcinogenic properties, thus promoting the development of neoplasms following prolonged dietary exposure (Bennett and Klich, 2003). Table 2 presents a comprehensive synthesis of major mycotoxins, detailing their principal contamination matrices, species-specific susceptibility, and associated target organ toxicities in animals.

Figure 1: Mycotoxin-producing fungal species (Yang et al., 2020).

Studies have revealed that a high percentage of pet food samples are contaminated with multiple mycotoxins simultaneously (Brera et al., 2006). Typically, mycotoxins commonly detected in pet foods include aflatoxins, ochratoxins, and *Fusarium*-derived mycotoxins, all of which are harmful to the health of companion animals. However, these toxins differ in their toxicological profiles and adverse health effects and may spread across various regions via contaminated raw materials. Consequently, acute mycotoxicosis in pets is frequently associated with using extremely contaminated ingredients in pet food manufacturing. It is noteworthy that, among the various mycotoxins of concern in pet food, aflatoxins are particularly hazardous to dogs. Consequently, clinical manifestations of canine aflatoxicosis commonly result in anorexia, depression, lethargy, and, in severe cases, sudden death. The heightened susceptibility of dogs to aflatoxin exposure is largely attributed to the accidental consumption of pet foods contaminated with maize or maize-derived ingredients during processing and manufacture (Brera et al., 2004; Agriopoulou et al., 2020). In some cases, an aflatoxin outbreak may continue for a few months before diagnosis, impacting a larger population of animals (Antonissen et al., 2015; Jahanian et al., 2017). Testing of pet food at the point

of manufacture and the timely identification and diagnosis by veterinarians are vital in controlling these outbreaks. Aflatoxin contamination has been reported in pet food globally, with higher prevalence observed in North and South America. Many surveys have identified contaminated wild bird feed as a potential source of aflatoxins, probably due to the extensive use of maize, nuts, and seeds as main ingredients (U.S. Government Accountability Office, 2005; Stenske et al., 2006). In two separate surveys, up to one in four feed samples of wild birds were found to contain AFB1 levels exceeding 100 µg/kg, representing a significant potential hazard (Garland and Reagor, 2007). These findings underscore the broader risk of aflatoxin entry into animal feeding systems through contaminated plant-derived ingredients. Consequently, mitigating the negative impacts of mycotoxins on animal health necessitates the use of correctly graded and quality-controlled feed ingredients, as well as regular diagnostic and monitoring procedures to ensure early detection and effective treatment of mycotoxin exposure.

Prevention and Mitigation Strategies

Several human disease outbreaks and animal poisonings attributable to mycotoxin contaminations have been documented worldwide (Henke et al., 2001). Consequently, effective prevention of mycotoxin contamination is essential for ensuring food and feed safety across the agro-food industry. According to the data provided by the Food and Agriculture Organization (FAO), 77 countries across the world have established regulatory limits or guidelines for mycotoxins in food and feed, while 13 countries, including African countries, lack mycotoxin-specific food safety regulations (Brera et al., 2004). As contamination of food and fodder by mycotoxins has emerged as a global concern, it is estimated that approximately 25% of the world's crop production is lost through mycotoxin infestations. Annually, mycotoxins are reported to be linked with huge agricultural and industrial losses in billions of dollars. The United States reports a loss of about 0.5 to 1.5 billion dollars every year (Henke et al., 2001; Penido Maia and Pereira Bastos de Siqueira, 2002).

Effective prevention of mycotoxin contamination requires the integration of good agricultural practices, controlled harvesting and storage conditions, and the application of appropriate biotechnological interventions. It is worth noting that mycotoxin mitigation strategies in animal feed include a variety of physical, chemical, and biological processes to remove, adsorb, or transform toxic compounds into less harmful forms. While individual methods may exhibit variable efficacy depending on the type of mycotoxin, integrated mitigation approaches are increasingly emphasized to address the complexity of multi-mycotoxin contamination in feed systems and to enhance overall detoxification efficiency (Zhu et al., 2016). Table 3 summarizes the effectiveness of these measures based on mycotoxin type, with integrated methods showing greater poten-

Table 2: Major mycotoxins with their target matrices, animal species sensitivity, and associated organ toxicity (Alshannaq & Yu, 2017; Khan et al., 2024)

Mycotoxin	Target materials	Target animal species	Affected organs
Aflatoxins	Milk, eggs, rice, cheese, wheat, nuts, peanuts, corn, figs, cottonseeds	Rat, fish, sheep, cattle, chicken, duckling	Bile duct, intestinal tract, kidney damage, liver tumors
Ochratoxin	Wine, coffee, cheese, cereal grains, grapes, and dry fruits	Chicken, rat, duckling	Liver damage, urinary tract tumors, and kidney tumors
Fumonisins	Polenta, corn	Rat, mouse, horse	Lungs, liver, and kidney damage
Zearalenone	Moldy hay, corn, commercial feed.	Rat, mouse, lamb, cattle, chicken	Ovaries, mammary glands, testicles, uterus, and vulva
Trichothecenes	Rice, barley, oats, maize, wheat	Pigs, sheep, poultry, cattle	Skin, intestine, testes

tial for managing co-contamination in feed systems. However, not all of these approaches are appropriate for practical applications; the acceptable ones are those that preserve the nutritional content and palatability of the products; so, inactivation of mycotoxins without toxin generation is preferable (Awuchi et al., 2022). Because mycotoxin contamination can occur at multiple points along the feed supply chain, effective prevention requires an integrated strategy involving both pre-harvest and post-harvest interventions. Pre-harvest measures focus on reducing fungal infection and toxin production in the field, whereas post-harvest strategies include proper drying, handling, storage, transportation, and processing of feed ingredients, as well as physical, chemical, and biological detoxification approaches. Pre-harvest interventions are widely acknowledged as the most critical preventive barrier against mould infection and the initial formation of mycotoxins; however, well-designed post-harvest interventions are equally indispensable for limiting further toxin development and accumulation during harvesting, storage, and feed processing. Although appropriate plant selection based on soil characteristics and the use of fungus-resistant crop varieties can significantly reduce fungal colonization and subsequent mycotoxin development, these measures must be integrated with broader pre- and post-harvest control strategies to ensure effective mitigation (Ferrão et al., 2017).

The use of essential oils with antioxidant properties and mould inhibitors (i.e., propionic acid) reduces the fungal growth during the storage period. Research conducted on fruits treated with essential oils revealed that they have a long shelf life in comparison to untreated fruits. Essential oils caused fruits to have high antioxidant content and physicochemical properties, protecting against the growth of yeasts and molds. Hence, essential oils are considered effective mold inhibitors (Alshannaq and Yu, 2017). Cross-contamination can also be avoided by using properly cleaned transport vehicles, monitoring humidity, temperature, and application of sodium hypochlorite to the storage area (Cinar and Onbasi 2020; Pankaj et al., 2017). Genetic engineering of plants to suppress mycotoxin production and the cultivation of fungal infestation-resistant plants are also among the best applicable strategies to stop mycotoxin production (Paster and Barkai-Golan, 2008).

To reduce the severity of mycotoxins, decontamination is also done. Decontamination is conducted via one of the three methods, i.e., biological method, chemical method, or physical method. Biological methods include using enzymes and microorganisms. The chemical method consists of treatment by ozone, ammonia, or chemical agents like chlorine, aldehyde, bases, oxidizing agents, and bisulfites. Physical method utilizes several absorbents such as aluminosilicate, activated charcoal, clay minerals, and montmorillonite (Aboagye-Nuamah et al., 2021).

Another potential strategy is to regulate mycotoxin levels by detoxification. Fermentation is the process of converting, degrading, and decontaminating food. Myco-toxin's chemical structure is altered during fermentation, and its adherence to bacterial cells lessens its toxicity (Shehata et al., 2020). However, preventive measures alone are not enough, and an integrated management system for hazard analyses and critical control point (HACCP) must be implemented. HACCP is a food management system that ensures analysis, physical control, and chemical control of the raw material before its finalization and supply. In 1992, guidelines on HACCP were published by the National Advisory Committee on microbiological criteria for foods (FAO, 2001). The application of HACCP principles, which include hazard identification and analysis, determination of critical control points (CCPs), establishment of critical limits for each class of mycotoxin, monitoring procedures, and corrective action, is critical to regulating feed safety and its associated side effects (Cinar and Onbaşi, 2020).

Economic Impact of Mycotoxins in Monogastric Animal Nutrition

Mycotoxins represent one of the most significant and persistent challenges facing modern livestock production systems. For monogastric animals, specifically poultry and swine, the economic consequences of mycotoxin contamination are particularly severe due to their simple digestive systems, which offer limited capacity for mycotoxin degradation compared to rumi-

nants (Muñoz-Solano et al., 2024). The occurrence of mycotoxins in cereals, oilseeds, stored feed commodities, and feed by-products utilized in monogastric diets has become a major global concern because of their adverse effects on productivity reduction, feed efficiency impairment, increased mortality, and animal health/veterinary costs, all of which have implications for the economics of production. The economic consequences of mycotoxin contamination in animal nutrition are extensive and multifaceted, affecting producers, feed manufacturers, processors, and consumers worldwide. Recent estimates indicate that between 60% and 80% of feed materials globally contain detectable levels of one or more mycotoxins, emphasizing the magnitude of the problem (Deng et al., 2023). The economic impact of mycotoxins can be classified as direct and indirect losses. Direct losses arise from reduced animal productivity, poor growth performance, increased mortality, reproductive inefficiency, and condemnation of contaminated feed or animal products. Indirect losses include expenditures on surveillance, feed testing, detoxification technologies, veterinary care, and regulatory compliance (Xu et al., 2022).

One of the most crucial economic impacts of mycotoxins is lowered animal performance. Monogastrics, particularly poultry and pigs, are highly susceptible to mycotoxins because they possess limited microbial detoxification capacity in the gastrointestinal tract. Exposure to aflatoxins, deoxynivalenol (DON), fumonisins, ochratoxin A, zearalenone, and T-2 toxins commonly results in depressed feed intake, reduced weight gain, poor feed conversion efficiency, immunosuppression, and impaired nutrient utilization (Lach & Kotarska, 2024), all of which have dire negative economic implications for profit. In broiler production, even low concentrations of aflatoxin B1 can significantly reduce growth rates and carcass yield, leading to lower profitability. Similarly, DON contamination in swine diets is strongly associated with feed refusal and reduced average daily gain, thereby increasing production costs per unit of meat produced (Yu et al., 2025).

Reproductive losses in livestock also contribute substantially to the economic burden of mycotoxins. Zearalenone, an estrogenic mycotoxin produced largely by *Fusarium* species, causes infertility, reduced litter size, abortion, pseudopregnancy, and other reproductive disorders in pigs and other livestock species (Xu et al., 2022). According to the authors, chronic exposure to fumonisins and ochratoxin A has additionally been linked to embryonic toxicity and impaired reproductive performance in breeding animals. Reproductive inefficiencies increase replacement costs and reduce overall herd productivity and profitability.

Mycotoxin-induced immunosuppression is also another source of economic loss. Mycotoxins impair both innate and adaptive immune responses, making animals more vulnerable to infectious diseases and reducing vaccine efficacy. Increased incidence of diseases accordingly raises veterinary expenses, mortality rates, and medication costs while decreasing production efficiency. In poultry production, immunosuppressive effects of aflatoxins and trichothecenes often predispose birds to secondary infections such as coccidiosis and necrotic enteritis, leading to economic losses (Kolawole et al., 2024).

Mycotoxin-induced mortality causes a direct and quantifiable economic loss. While acute mycotoxicosis outbreaks resulting in obvious mortality have decreased due to increased feed safety monitoring, chronic low-level exposure continues to raise baseline mortality rates in commercial operations (Kolawole et al., 2024). The replacement cost of lost animals, combined with the foregone production from animals that die before market, constitutes a substantial economic burden. Mycotoxin contamination imposes further economic burdens through feed rejection and deterioration of feed quality. Raw materials compromised by toxins are frequently deemed unsafe for animal rations, leading to significant financial setbacks for both feed producers and farmers. In critical situations, entire feed batches may be discarded or diluted to comply with regulatory standards. The increasing prevalence of co-contamination with multiple mycotoxins further complicates feed management because synergistic interactions among toxins can amplify toxicity even when individual toxin concentrations remain below regulatory thresholds (Chhaya et al., 2024).

Table 3: Innovative strategies for the mitigation of mycotoxins in animal feed systems (Zhu et al., 2016)

Category	Mitigation Strategy	Agents/Examples	Mechanism of Action	Key Outcomes/Effectiveness	Limitations
Physical	Post-harvest processing	Drying, UV irradiation, grain sorting (floating)	Reduces fungal growth or physically removes contaminated fractions	Drying reduces moisture below 20%, limiting fungal growth; UV removes ≈100% aflatoxin in short exposure; floating removes >80% contaminated kernels	Nutrient loss possible; not fully effective for internal contamination
Physical	Adsorption (inorganic binders)	Bentonite, HSCAS, zeolites, activated charcoal	Binds mycotoxins in the digestive tract to prevent absorption	Highly effective for aflatoxins due to strong binding affinity	Poor efficacy against DON, T-2 toxin, and other polar toxins
Physical/ Chemical	Modified adsorbents	Organic-modified clays, diatomite + amines	Increased hydrophobicity enhances the binding of non-polar toxins	>85% adsorption of ZEA, OTA, and fumonisins reported	Potential toxicity and unintended nutrient binding
Biological (adsorption)	Microbial cell wall binders	Yeast (β-glucans), lactic acid bacteria	Adsorption through hydrogen bonding, ionic, and hydrophobic interactions	Yeast β-glucans significantly reduce ZEA bioavailability	Binding is reversible; lower efficiency for some toxins
Biological (plant-based)	Lignocellulosic materials	Grain hulls, cocoa shells, lignin derivatives	Adsorption via increased surface area and hydrophobic interactions	Up to 59% DON and >60% OTA/T-2 adsorption under optimized conditions	Variable performance across mycotoxins
Chemical	Chemical detoxification	Bases, acids, oxidants, glycerol-based solutions	Chemical modification reduces toxicity or bioavailability	Potentiated glycerol enhances detoxification through hydroxyl ion activity	Safety concerns and regulatory limitations
Biological (microbial)	Microbial biotransformation	*Bacillus*, *Devosia*, *Sphingomonas* spp.	Converts mycotoxins into less toxic metabolites (e.g., DON → DOM)	Up to 100% detoxification of DON and ZEA in controlled conditions	Requires strict conditions (pH, temperature) for activity
Biological (enzymatic)	Enzymatic degradation	Laccase, ochratoxinase, amidases, oxidases	Enzymes catalyze toxin breakdown into less toxic compounds	Effective degradation of AFB1, OTA, ZEA, and fumonisins	Limited number of fully characterized enzymes available
Preventive (pre-harvest)	Agronomic practices	Crop rotation, resistant varieties, and fungicide use	Prevents fungal growth and toxin formation	Recommended for reducing Fusarium toxins in cereals	Sometimes ineffective; fungicides may increase toxin production
Integrated approaches	Combined strategies	Adsorbents + microbes + enzymes	Synergistic detoxification through multiple mechanisms	Enhanced mitigation across multiple mycotoxins	Cost and complexity

Furthermore, the costs associated with mycotoxin monitoring and mitigation are also considerable. Particularly, feed manufacturers and animal farmers invest heavily in routine screening, laboratory analyses, storage management, and detoxification strategies to reduce the risk of contamination. It is important to note that both commercial feed manufacturers and small-scale farmers incorporate mycotoxin binders, biological detoxifiers, adsorbents, etc., into feeds to reduce toxin bioavailability. Although these interventions help protect animal health and productivity, they increase feed formulation costs and production expenses (Deng et al., 2023).

Recently, climate change has become a critical driver of the economic burden associated with mycotoxins in monogastric animal nutrition. Rising temperatures, changing rainfall regimes, and elevated humidity foster conditions conducive to fungal growth and toxin synthesis. Emerging evidence, according to Chhaya et al. (2024), shows that climatic variability is broadening the distribution of mycotoxigenic fungi, thereby increasing both the frequency and intensity of contamination events globally. This trajectory is projected to exacerbate food safety concerns and heighten economic losses in the years ahead.

CONCLUSIONS

Mycotoxin contamination poses a significant and multifaceted challenge to global food and feed security. The productivity and financial sustainability of livestock systems are threatened by the negative impacts on animal health, which can range from acute illness and death to growth retardation and compromised immunity. Consequently, mycotoxins impose substantial economic losses through reduced growth performance, poor feed efficiency, reproductive disorders, immunosuppression, increased mortality, feed rejection, and higher veterinary and feed management costs. These risks also complicate international commodity trade and cause disruptions across the feed industry. The dynamic nature of fungal contamination cannot be adequately addressed by recent advancements in mitigation technologies, even though they offer a starting point for advancement. Therefore, future initiatives must prioritize ongoing surveillance and fortify preventative measures. Expanded toxicological research is desperately needed, especially to better understand the synergistic effects of co-exposure to multiple mycotoxins. To successfully lessen the negative effects of these harmful substances on human health and the economy, a dedication to such thorough scientific investigation and proactive risk management is necessary.

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