INTAS ANIMAL HEALTH

AviPod

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Multi-Mycotoxin Risk Management in Poultry

One Feed, Many Threats – Manage Mycotoxins Smartly

Dear Readers,

Our poultry industry is a major contributor to global food security; therefore, maintaining optimal bird health is critical for sustaining productivity. The term "mycotoxin" is derived from the Greek words "mykes" (fungus) and "toxicon" (poison). Mycotoxins are low-molecular-weight secondary metabolites produced by a wide range of fungi. Among the numerous mycotoxins identified, most commonly encountered include aflatoxins, zearalenone, ochratoxin A, fumonisins and trichothecenes. These toxins frequently contaminate cereal grains and oilseed meals.

Exposure to mycotoxins adversely affects poultry health and performance. Affected birds often exhibit reduced responses to routine vaccinations, while maternal exposure compromises the transfer of passive immunity to progeny. Clinically, mycotoxicosis manifests as poor growth performance, organ damage, reproductive disorders, gastro-intestinal lesions and increased mortality. Under commercial production conditions, these effects pose substantial challenges and result in measurable economic losses.

Effective mycotoxin risk management therefore requires an integrated, multi-level approach, encompassing routine feed screening, strict control of moisture and storage conditions and use of scientifically validated detoxification strategies. Such a holistic approach is essential to safeguard poultry health, optimize productivity and ensure sustainability.

We invite you to review this issue and share your valuable experiences and insights by scanning the below QR code or by contacting us *via* email at aviglo@ intaspharma.com.

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Introduction

Poultry industry is a vital, well-established and economically significant sector worldwide. Poultry meat is relatively low in fat and cost-effective, making it highly accessible to consumers. Consequently, maintaining optimal bird health is essential to meet the increasing daily demand for poultry meat and eggs.

Mycotoxins are biologically active, toxic secondary metabolites produced by toxigenic fungi, predominantly belonging to the genera Aspergillus, Fusarium and Penicillium. These fungi infect crops during pre-harvest stages and may continue to proliferate during post-harvest storage under favourable environmental conditions, particularly elevated temperature and humidity. Food and Agriculture Organization (FAO) estimates that approximately 25% of human food and animal feed worldwide is contaminated with mycotoxins. Similar to other environmental contaminants, mycotoxins adversely affect poultry health, productivity and overall economic performance.

Economic losses due to mycotoxin contamination are primarily attributed to reduced growth rates, impaired feed conversion efficiency, decreased carcass yield and quality and increased susceptibility to infectious diseases resulting from immunosuppressive effects of mycotoxins. Among the more than 350 mycotoxins identified to date, several are considered highly prevalent and economically significant in poultry production. Therefore, its prevention and effective management are of critical importance.

Prevalence and Seasonal Patterns of Mycotoxins

Indian mycotoxin survey highlights widespread contamination of feeds and feed ingredients. In 2023, 97% of samples were contaminated and 76% showed co-occurrence of multiple mycotoxins, increasing toxicological risk. Finished poultry feed exhibited 100% contamination, with high prevalence of aflatoxins (93%), fumonisins (72%), ochratoxin A (69%) and T-2 toxin (59%), often at elevated levels. Key feed ingredients, including corn (95% contaminated), soybean meal, rice bran, de-oiled rice bran and rice (100%), were severely affected, with aflatoxins and fumonisins frequently exceeding tolerable limits. Mycotoxicosis occurs most commonly during the monsoon and post-monsoon seasons, driven by warm, humid conditions that promote fungal growth and mycotoxin production (Ankit *et al.*, 2024).

Mycotoxins - Types and Sources

Mycotoxin	Fungal Source	Feed Source
Aflatoxin (B_1, B_2, G_1, G_2)	*Aspergillus flavus*, *Aspergillus parasiticus*	Maize, groundnut cake, cottonseed meal
Ochratoxin A	*Aspergillus ochraceus, A. carbonarius, A. niger, Penicillium verrucosum, P. nordicum*	Wheat, maize, barley
Deoxynivalenol (DON)	*Fusarium graminearum*	Wheat, maize, barley
T-2 toxin	*Fusarium sporotrichioides , F. poae*	Maize, wheat, oats
Zearalenone	*Fusarium graminearum, F. culmorum, F. cerealis, F. equiseti*	Corn, wheat, barley, oats, rye
Fumonisin (B_1, B_2, B_3) B_1 - most common	*Fusarium verticillioides, F. proliferatum, F. fujikuroi*	Maize and maize by-products
Citrinin	*Penicillium citrinum*	Stored grains, maize

Emerging Mycotoxins

Emerging mycotoxins represent a relatively new and chemically diverse group of fungal toxins that are not routinely monitored. These compounds are secondary metabolites produced by *Fusarium* spp. including enniatins (ENN), beauvericin (BEA), moniliformin (MON) and fusaproliferin (FUS). Large-scale global analyses of corn grain samples have demonstrated extensive co-occurrence of emerging mycotoxins, with emerging mycotoxins being the most prevalent group, detected in 31.4% of samples. Biologically, emerging mycotoxins pose a multi-faceted risk by inducing oxidative stress, cytotoxicity and disruption of intestinal integrity.

Modified Mycotoxins

Modified mycotoxins are structurally altered derivatives of fungal mycotoxins formed through chemical or biological transformations. These modifications occur in plants as part of a defense response to fungal infection and in animals through metabolic conversion of ingested mycotoxins. The most frequently detected modified mycotoxins originate from *Fusarium* spp. include derivatives of deoxynivalenol (DON), zearalenone (ZEN), HT-2 toxin, nivalenol (NIV) and fumonisins (FBs). Moreover, modified mycotoxins pose a significant analytical challenge due to their structural similarity to parent compounds and lack of validated routine detection methods.

Mycotoxins and their Effects

Effects on Intestine

Mycotoxins can exert a significant impact on intestinal epithelial cells, resulting in multiple adverse effects on poultry health and productivity. These toxic compounds adversely affect the intestinal epithelium through several mechanisms.

Cell Damage and Disruption of Tight Junctions

Mycotoxins can directly injure intestinal epithelial cells, leading to disruption of tight junctions that are essential for maintaining intestinal barrier integrity. Compromise of these junctions increases intestinal permeability, a condition commonly referred as "leaky gut". This allows bacteria, toxins and other harmful substances to translocate across intestinal wall into bloodstream, potentially triggering systemic infection and inflammatory responses.

Impaired Nutrient Absorption

Intestinal epithelium plays a critical role in nutrient digestion and absorption. Mycotoxins can interfere with these processes, reducing efficiency of nutrient uptake. Consequently, poultry may experience malnutrition and impaired growth performance despite adequate feed consumption.

Inflammation and Oxidative Stress

Mycotoxins can induce inflammatory reactions and oxidative stress within intestinal epithelial cells. These responses further damage intestinal lining, exacerbate intestinal permeability and compromise bird's Immunity. Persistent inflammation and oxidative stress may also contribute to development of additional pathological conditions.

Alterations in Gut Microbiota

Intestinal health is closely linked to composition and stability of gut microbiota. Mycotoxins can disrupt microbial populations, resulting in dysbiosis. Such imbalances negatively affect digestive efficiency, immune regulation and overall physiological health.

Impaired Vaccine Efficacy

Vaccination is a cornerstone of disease prevention in poultry production; however, its effectiveness is highly dependent on integrity and functionality of host immune system. Mycotoxins, even at subclinical concentrations, can modulate immune responses and compromise the protective efficacy of commonly used vaccines. The following sections describe the mechanisms through which mycotoxins impair vaccine responses, with particular emphasis on humoral and cell-mediated immunity.

Immunosuppressive Mechanisms

Major mycotoxins, including aflatoxins, ochratoxin A, trichothecenes (such as T-2 toxin and deoxynivalenol) and fumonisins are well documented for their immunomodulatory and immunosuppressive effects (Fig. 1). Aflatoxin B_1 inhibits protein synthesis, reduces immunoglobulin production (IgA, IgY and IgM), impairs macrophage and lymphocyte function and induces apoptosis in primary and secondary lymphoid organs such as spleen, thymus and bursa of Fabricius. These alterations result in reduced lymphocyte proliferation and dysregulated cytokine production, thereby limiting bird's capacity to mount an effective immune response to antigens. Ochratoxin A exerts similar immunotoxic effects by disrupting T-lymphocyte populations and suppressing antibody production.

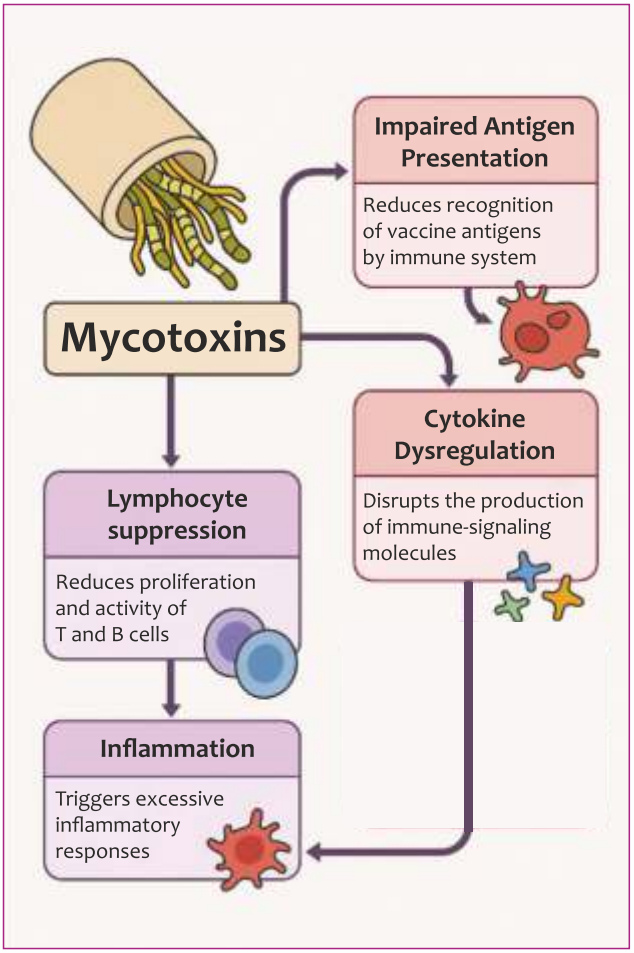


Fig. 1: Mycotoxins Immunosuppressive Pathways Impairing Vaccine Responses
(Olariu *et al.*, 2025)

Reduced Vaccine Response and Increased Disease Susceptibility

Experimental studies have demonstrated that dietary exposure to mycotoxins significantly reduces antibody titers following vaccination against infectious laryngotracheitis, newcastle disease virus and infectious bronchitis virus.

Co-Infections and Vaccine Failure

Mycotoxin induced immunosuppression predisposes poultry to secondary infections, which further compromises vaccine efficacy. Concurrent exposure to mycotoxins and enteric or respiratory pathogens such as *Clostridium perfringens, Eimeria* spp. and *Escherichia coli* has been shown to exacerbate disease severity despite vaccination. This effect is largely attributed to deficiencies in antigen processing, antibody production and cell mediated immune responses.

Maternal Immunity and Progeny Vulnerability

Maternal exposure to mycotoxins can significantly impair the transfer of passive immunity to offspring. Breeder hens consuming aflatoxin contaminated diets produce lower levels of maternal antibodies, resulting in reduced immunological protection in their progeny, even when chicks are not directly exposed to mycotoxins. This deficiency in early immune protection increases susceptibility to infectious diseases and elevates morbidity and mortality during critical early life stages, particularly around primary vaccination periods. In addition, exposure to ochratoxin A (OTA) during

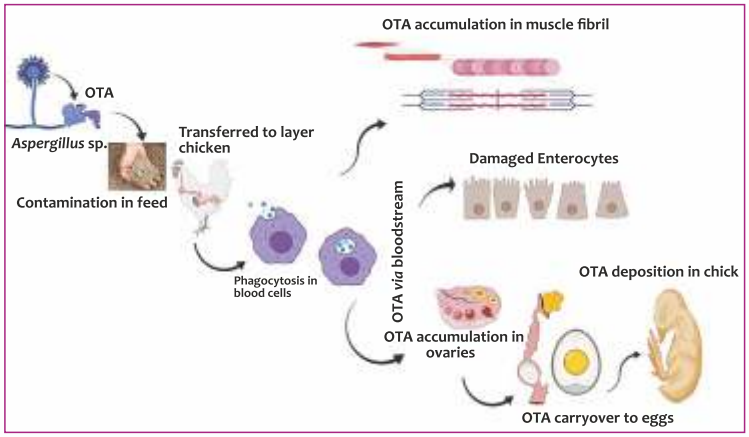


Fig. 2: OTA Carryover Pathway in Poultry
(Ganesan *et al.*, 2021)

embryonic development has been shown to induce extensive oxidative stress and organ damage, further predisposing chicks to compromised health and performance. A schematic representation of OTA carryover from contaminated feed to various tissues in poultry is highlighted in Fig. 2.

Clinical Manifestations and Pathological Lesions

Mycotoxin	Target Organs	Signs and Symptoms	Pathological Lesions
Aflatoxin B1	Liver, immune organs (bursa, thymus)	Poor egg production, poor hatchability, reduced weight gain, poor feed conversion and immunosuppression	Enlarged pale fatty liver, hepatic necrosis, haemorrhages and atrophy of bursa and thymus
Ochratoxin A	Kidney, liver	Reduced feed consumption, reduced efficiency and weight gain, decreased egg production, egg quality and immunity, anaemia, feathering problems and increased mortality	Liver and kidneys show significant enlargement and histopathological changes, including vacuolar degeneration, fatty infiltration, hepatocyte necrosis, glomerular damage and bile duct hyperplasia
Zearalenone	Intestinal mucosa, immune system	Decreased egg production and egg quality	Cloacal swelling, enlargement of oviduct, reproductive tract feminization, cysts and reduced testicular weight
Fumonisin B1	Oral mucosa, intestine, liver	Diarrhoea, reduced egg production, leg weakness, lameness, mortality and poor immunity in severe cases	Hepatic necrosis, biliary hyperplasia, glomerulonephritis with degeneration and necrosis observed in both hepatic and renal tissues
T-2 toxin	Reproductive organs, liver	Reduced feed consumption, reduced efficiency and weight gain, decreased egg production and immunity, abnormal behaviour	Oral ulcers, necrosis of oral mucosa, gastrointestinal haemorrhages
Deoxynivalenol	Liver, kidney	Reduced weight gain, impaired nutrient absorption, increased feed conversion ratio. At higher doses, causes severe damage to hematopoietic tissues and disrupts immune function, making bird more vulnerable to disease	Mild intestinal inflammation, catarrhal enteritis

Managemental Approaches

Mycotoxin Screening

Routine screening of feed ingredients and finished feed for mycotoxin contamination prior to poultry ration formulation is essential. Several analytical techniques are available for mycotoxin detection, including microcolumn assays, thin-layer chromatography (TLC), enzyme-linked immunosorbent assay (ELISA), high-performance liquid chromatography (HPLC), gas chromatography and tandem gas chromatography/mass spectrometry. Among these, thin-layer chromatography and enzyme-linked immunosorbent assay are widely used due to their rapidity, simplicity and suitability for routine screening of contaminated feed and feedstuffs.

Moisture and Temperature Control

Effective control of moisture and temperature is critical for preventing fungal growth and mycotoxin production. Grain storage facilities should be well ventilated and protected from rain and extreme temperature fluctuations. Grain moisture levels should be maintained below 13%.

Amelioration/ Biological Inactivation

Mycotoxin Binding Agents

Numerous sorbent agents, including activated carbon (charcoal), clay minerals such as bentonite, zeolite and HSCAS are widely used to mitigate mycotoxicosis and to reduce the carryover of mycotoxins into meat and eggs. These sorbents are generally nutritionally inert and function by adsorbing mycotoxins onto their surface within the intestinal tract, thereby reducing toxin bioavailability.

Hydrated Sodium Calcium Aluminosilicate (HSCAS) is widely used in animal feeds for mycotoxin binding and pelletizing purposes. HSCAS is a sorbent compound composed of natural clays containing sodium, calcium, aluminum, silicon and water, present either in crystalline form or as hydroxyl groups. It functions as a "chemical sponge" by selectively adsorbing mycotoxins present in poultry feed. The molecular surface of HSCAS becomes saturated with water, which facilitates interaction with polar structures of specific mycotoxins.

Mechanism of Clay-Based Mycotoxin Binders

Addition of clay binder in feed
↓
Hydration and swelling of clays
↓
Formation of large surface area and activation of negatively charged adsorption sites
↓
Interaction between clay surface and mycotoxin molecules
(Electrostatic attraction + hydrogen bonding + Van der Waals forces)
↓
Adsorption of mycotoxins in interlayer spaces of aluminosilicate clay structure
↓
Formation of stable clay–mycotoxin complex (non-absorbable in intestinal epithelium)
↓
Reduced intestinal absorption of mycotoxins
↓
Excretion of clay–toxin complex *via* faeces

Bentonites (hydrated aluminium silicates) are sorbent materials characterised by a layered (lamellar) crystalline microstructure and variable chemical composition. Their adsorption properties primarily depend on type of exchangeable cations (Na^+, K^+, Ca^{2+} and Mg^{2+}) present within the layers. Sodium bentonite is generally more effective than calcium bentonite. Bentonite has been reported to bind aflatoxin at levels of up to 66%.

Sepiolite is a silicate clay commonly used in animal feed as a pellet binder and as an adsorbent in poultry feed to reduce the bioavailability of mycotoxins. Owing to its high surface area, fibrous structure and strong adsorption capacity, sepiolite is capable of binding polar mycotoxins such as aflatoxins, ochratoxin A and certain trichothecenes within the gastrointestinal tract, thereby preventing their absorption into bloodstream. Dietary inclusion of sepiolite at levels of 1–2% has been reported to improve feed efficiency in broiler chicks, enhance calcium (Ca^{2+}) retention and increase body weight gain. These beneficial effects are likely attributed to stabilization and/or activation of digestive enzymes through their adsorption onto the sepiolite surface, which may also protect enzymes from proteolytic degradation.

Microbiological Binding Agents

Mannan oligosaccharides (MOS) derived from yeast cell wall (Fig. 3) of *Saccharomyces cerevisiae* have demonstrated broad-spectrum efficacy against multiple mycotoxins. *Saccharomyces cerevisiae* supplementation has shown beneficial effects in poultry during mycotoxicosis, with MOS identified as the primary active component. Esterified MOS-glucan complexes have exhibited strong *in vitro* binding affinity for aflatoxin B_1 (up to 81.6%), significant binding of zearalenone and T-2 toxin and moderate binding of ochratoxin A (25.5%) (Raju and Reddy, 2000). Dietary inclusion of MOS has been associated with improvements in body weight and other economically important performance parameters.

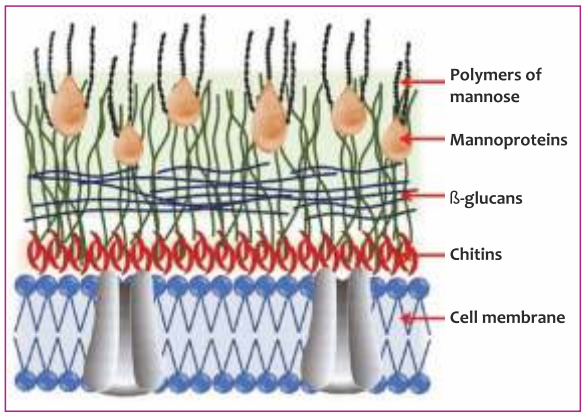
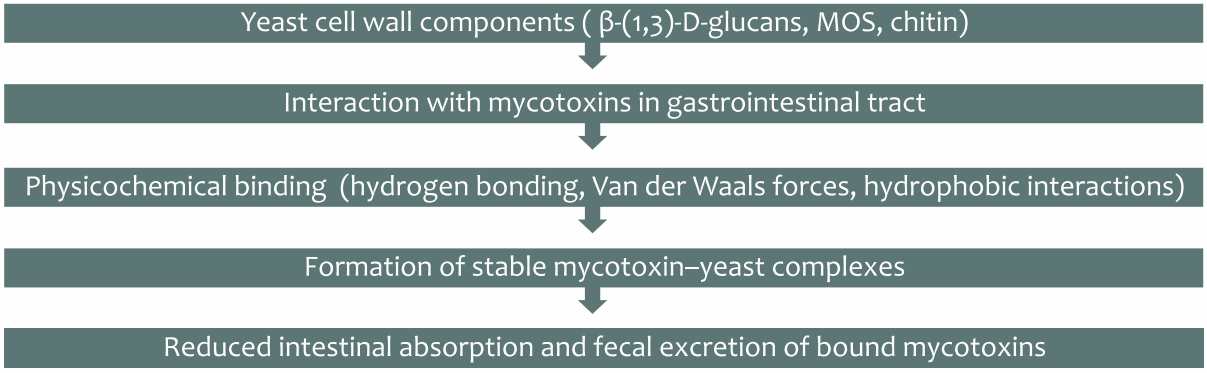


Fig. 3: Yeast Cell Wall Sheets and their Components
(Talavera *et al.*, 2013)

Certain strains of Bifidobacterium, lactic acid bacteria and Propionibacterium possess cell wall components capable of binding mycotoxins and reducing their bioavailability. Specifically, *Bifidobacterium animalis* and *Lactobacillus acidophilus* have demonstrated high binding efficiency against ochratoxin A and patulin, respectively.

Mechanism of Yeast Cell Wall-Based Mycotoxin Binders



Phytogenic Biosorbents or Natural Bio-Protectors

Currently, anti-mycotoxin phytogenic solutions are gaining prominence as promising natural alternatives, as they not only mitigate the harmful effects of mycotoxins but also provide additional health benefits. Common phytogenic biosorbents include extracts of curcumin, silymarin, grape pomace, olive pomace and orange peel.

Curcumin extract derived from *Curcuma longa*, has demonstrated significant protective effects against mycotoxicosis by reducing mycotoxin induced oxidative stress, suppressing cytochrome P450–mediated bioactivation and limiting the formation of toxin DNA adducts. Mechanistically, curcumin mitigates cellular damage during mycotoxin exposure by activating Nuclear factor erythroid 2 related factor 2 (Nrf2) mediated antioxidant pathways, inhibiting pro-inflammatory signaling, preserving intestinal barrier integrity and improving gut microbiota balance, thereby enhancing overall resilience to mycotoxin challenge.

Silymarin extract, a bioactive flavonolignan complex derived from *Silybum marianum* (milk thistle), plays a critical role in protecting against mycotoxin induced hepatic and intestinal damage. Silymarin exerts its protective effects by scavenging reactive oxygen species, enhancing endogenous antioxidant defense systems (including SOD, CAT, GST and glutathione-dependent enzymes), maintaining mitochondrial integrity and inhibiting lipid peroxidation and apoptosis. Importantly, silymarin modulates cytochrome P450 activity, thereby reducing mycotoxin bioactivation and conserving hepatic glutathione reserves, which are essential for detoxification during mycotoxicosis.

Grape pomace extract derived from winemaking by-products of *Vitis vinifera* is rich in bioactive phytochemicals that help reduce mycotoxin induced oxidative stress. It also exhibits biosorbent properties, binding major mycotoxins through hydrophobic and non-covalent interactions and supports intestinal integrity and gut microbiota balance, thereby mitigating mycotoxin related intestinal damage.

Olive pomace extract obtained from olive oil processing by-products of *Olea europaea*, contains a broad range of bioactive compounds that reduce mycotoxin induced oxidative stress through scavenging of reactive oxygen species and enhancement of cellular antioxidant defenses. It further demonstrates biosorbent activity, adsorbing key mycotoxins *via* non-covalent interactions, while also inhibiting the growth of toxigenic fungi and suppressing mycotoxin production.

Orange peel extract derived from citrus processing by-products is a rich source of bioactive compounds that reduce mycotoxin induced oxidative stress through activation of endogenous antioxidant pathways. In addition, it exhibits biosorbent and antifungal properties, enabling sequestration of major mycotoxins, while supporting gut integrity, immune modulation and microbiota balance.

Quimitōx Range - Science-Backed Mycotoxin Management

Quimitōx is a unique mixture of optimized, refined bentonite and sepiolite forming an effective adsorbent matrix. Quimitōx Plus is an advanced formulation comprising selected bentonite and sepiolite, which together form an efficient adsorbent matrix, combined with silymarin derived from milk thistle (*Silybum marianum*) extract and curcumin derived from turmeric (*Curcuma longa*) extract, collectively constituting the bio-protection complex. The formulation is further supplemented with yeast-based components, including yeast cell wall and hydrolyzed (inactivated) yeast products, classified as post-biotic fractions, along with a defined blend of flavouring compounds.

Bentonite provides high mycotoxin binding capacity through its layered aluminosilicate structure and strong cation-exchange properties, enabling efficient adsorption of polar mycotoxins. Sepiolite contributes a large specific surface area, supporting the binding of a broad spectrum of mycotoxins. Curcumin (turmeric extract) supports protection against mycotoxin induced oxidative stress and helps maintain intestinal barrier integrity, while silymarin (milk thistle extract) assists in mitigating mycotoxin related liver and intestinal damage. Yeast derived components further contribute to mycotoxin binding, support gut integrity, modulate intestinal microbiota and strengthen immune function.

The efficacy of Quimitōx Plus is supported by *in vivo* studies conducted in broiler chickens, which demonstrate protective effects against commonly occurring dietary mycotoxins under commercial broiler production conditions.

Clinical Studies

Study 1

Objective

To evaluate the efficacy of Quimitōx Plus in broilers challenged with fumonisin B1.

Introduction

Fumonisins, particularly FB1 are common mycotoxins in poultry feed that adversely affect growth performance, liver function and intestinal integrity. Exposure is characterised by reduced weight gain and feed efficiency, hepatic enlargement and dysfunction, intestinal mucosal damage (Fig. 1 and 2), bone lesions (Fig. 3) and disruption of sphingolipid metabolism, reflected by an elevated sphinganine/sphingosine (Sa/So) ratio, a recognised biomarker of fumonisin exposure.

Fig. 1: Necrotic enteritis in broiler chickens induced by the presence of Fumonisin toxins in feed (Antonissen, 2015)

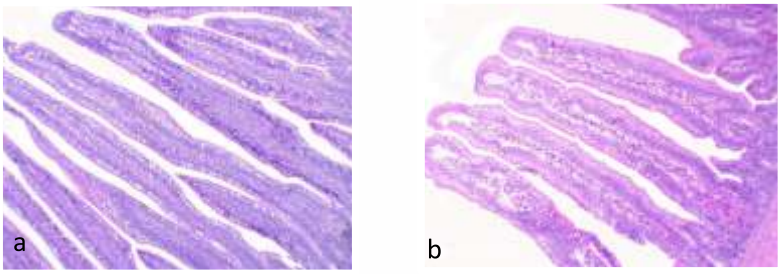


Fig. 2: Histomorphometry of jejunum in chickens: (a) control (b) Fumonisin challenge (Samitec, 2023)

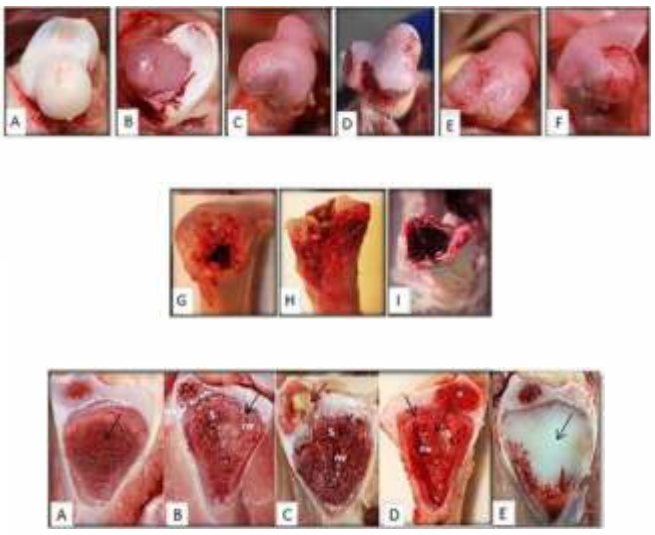


Fig. 3: Bone lesions caused by Fumonisin and DON. Bacteria chondronecrosis with osteomyelitis lameness (BCO) syndrome (Shanmugasundaram *et. al.* 2022; Alharbi *et. al.*, 2024)

Materials and Methods

The study was conducted in Santa Maria, Brazil using 600 (12 replicates/treatment, 10 broilers/replicates) one-day-old male (Cobb 500) broiler chicks housed in battery cages under standard management conditions without antibiotic use, with feed and water provided *ad libitum*. The experiment lasted 21 days, during which body weight and average daily gain were recorded at 7 and 14 days. At 21 days, evaluations included blood sphinganine/sphingosine ratio, relative liver weight and intestinal histopathology to assess the effects of fumonisin (B1+B2) exposure, with Sa/So ratio used as a biomarker of exposure.

Experimental Design

Groups			
Mycotoxin	Control	Contaminated diet	Contaminated diet + Quimitōx Plus @ 2.5 kg/ton feed
Fumonisin B1 (ppb)	–	>1000	>1000

Note: >1000 ppb represents critical level contamination. Fumonisin risk levels are classified into low at <100 ppb, medium at 100-250 ppb, high at 250-1000 ppb and critical at levels exceeding >1000 ppb.

Results

Supplementation with Quimitōx Plus effectively mitigated the adverse effects of fumonisin B1 in broilers by normalizing blood sphinganine/sphingosine ratios (Fig. 4), confirming reduced mycotoxin exposure. Birds receiving product showed improved productive performance, body weight gain and average daily gain (Fig. 5) despite fumonisin challenge, while intestinal histopathology demonstrated a significant increase in absorptive surface area (Fig. 6) and improved gut integrity (Fig. 7). Overall, Quimitōx Plus enhanced intestinal absorptive capacity and supported performance in broilers challenged with fumonisin B1 (Table 1).

Table 1. Performance Parameters in Fumonisin B1–Challenged Broilers Fed Quimitōx Plus

Treatment	Productive Parameters		Serum Biomarkers	Liver Integrity	Intestinal Histopathology			
	Body weight	Average daily gain	Sphinganine / sphingosine ratio	Relative liver weight	Villus height (VH)	Crypt depth (CD)	VH/CD ratio	Absorption surface
Contaminated diet (>1000 ppb FB1) + Quimitōx Plus vs Contaminated diet (>1000 ppb FB1)	+0.7%	+2.9%	−3.8%	−6.1%	+16.4%	−7.7%	+17.4%	+14.6%

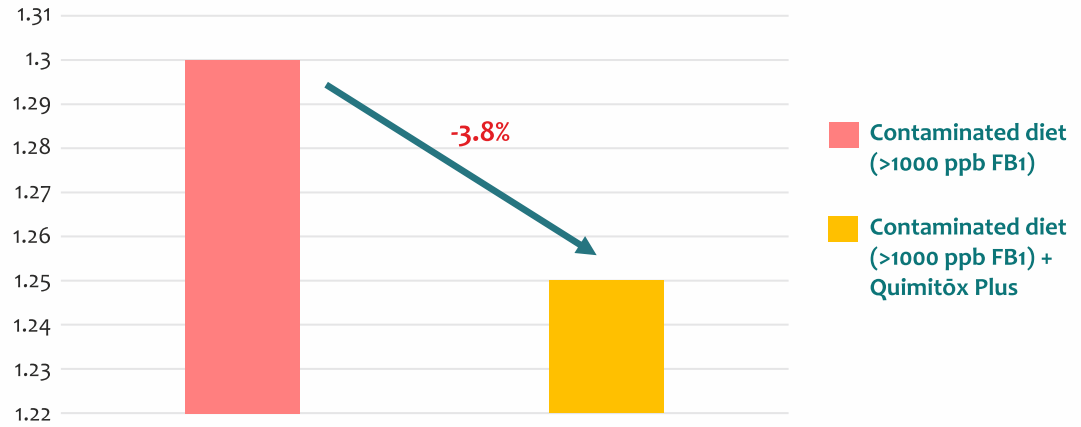


Fig. 4: Blood Sphinganine/Sphingosine Ratio

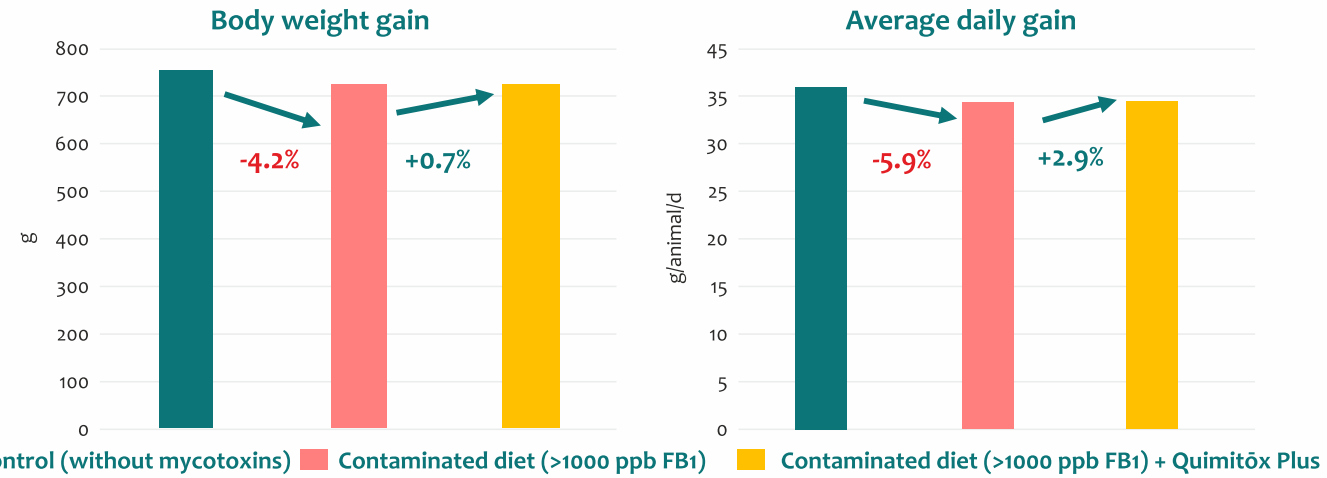


Fig. 5: Growth Performance in Fumonisin B1 Challenged Broilers

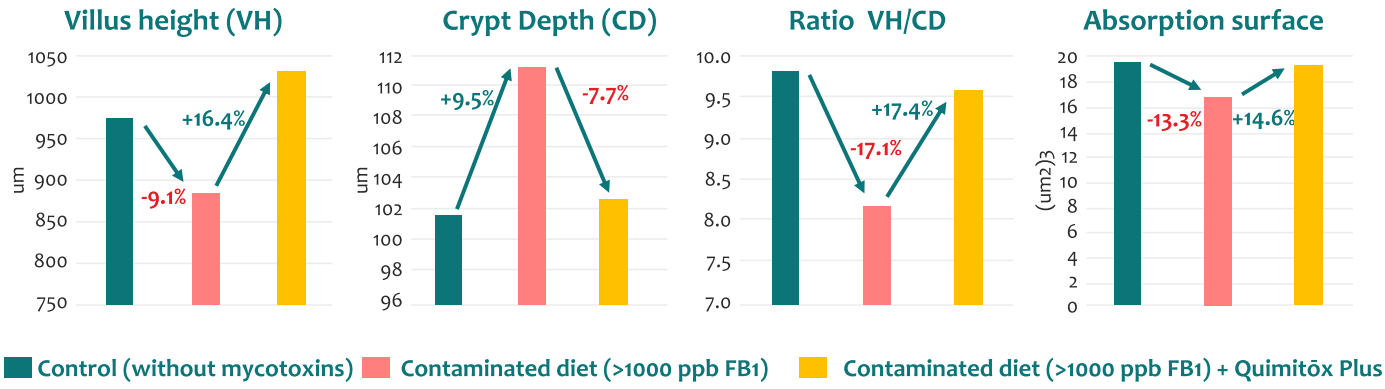


Fig. 6: Effect on Intestinal Absorptive Surface

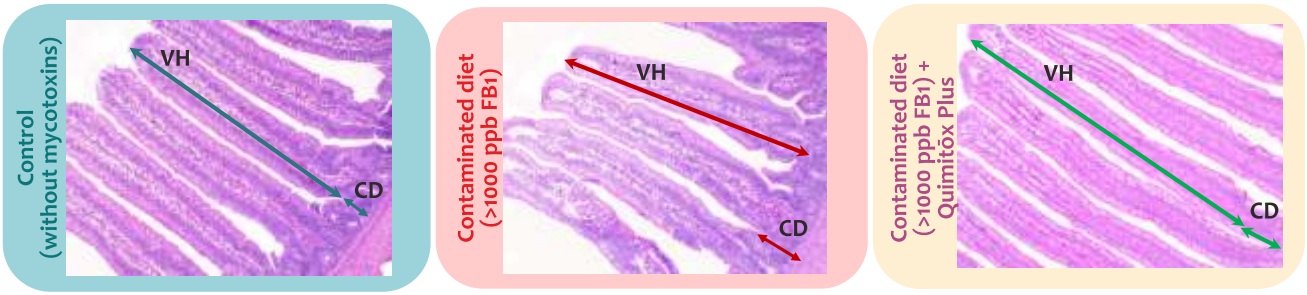


Fig. 7: Gut integrity in Fumonisin B1 Challenged Broilers

Conclusion

Quimitōx Plus effectively mitigated fumonisin B1 induced effects by improving performance, maintaining liver integrity and reducing histopathological alterations in broilers.

Study 2

Objective

Evaluation of Quimitōx Plus in broilers challenged with T-2 toxin.

Introduction

T-2 toxin is a highly toxic trichothecene mycotoxin commonly found in contaminated cereal grains used in poultry feed. In broilers, T-2 toxin primarily affects rapidly dividing tissues, leading to impaired growth, immune suppression and severe oral and gastrointestinal lesions (Fig. 1, 2, 3, 4). Exposure results in reduced feed intake, poor feathering and necrosis of beak, oral cavity and gizzard.

Materials and Methods

The study was conducted in Santa Maria, Brazil using 600 (12 replicates/treatment 10 chicks/replicates) one-day-old male (Cobb 500) broiler chickens housed in battery cages under standard management and biosecurity conditions, with no antibiotics administered. Birds were randomly assigned to treatments with 12 replicates of 10 chicks each and evaluated over a 21-day period. Productive performance, including feed conversion ratio, was recorded, while liver integrity was assessed by relative liver weight and intestinal health by histopathological analysis of villus height, crypt depth and absorptive surface.

Fig. 1: Growth retardation and poor feathering

Fig. 2: Necrotic lesions in oral cavity

Fig. 3: Necrotic lesions on beak

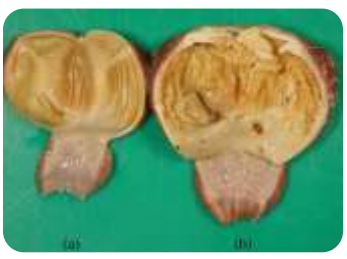


Fig. 4: Thickening and ulceration of gizzard mucosa

Experimental Design

Groups			
Mycotoxin	Control	Contaminated diet	Contaminated diet + Quimitōx Plus @ 2.5 kg/ton feed
T-2 Toxin (ppb)	–	>1000	>1000

Note: >1000 ppb represents critical level contamination. T-2 toxin risk levels are classified into low at 5 ppb, medium at 5-20 ppb, high at 20-100 ppb and critical at levels exceeding 1000 ppb.

Results

Supplementation with Quimitōx Plus markedly improved intestinal morphology in broilers challenged with T-2 toxin, as evidenced by increased villus height (VH), crypt depth (CD) (Fig. 5) an improved villus height-to-crypt depth (VH/CD) ratio and enhanced intestinal absorptive surface. These improvements indicate better gut integrity and nutrient absorption. Additionally, birds receiving Quimitōx Plus showed a significant improvement in feed conversion ratio, demonstrating enhanced productive performance under T-2 toxin challenge.

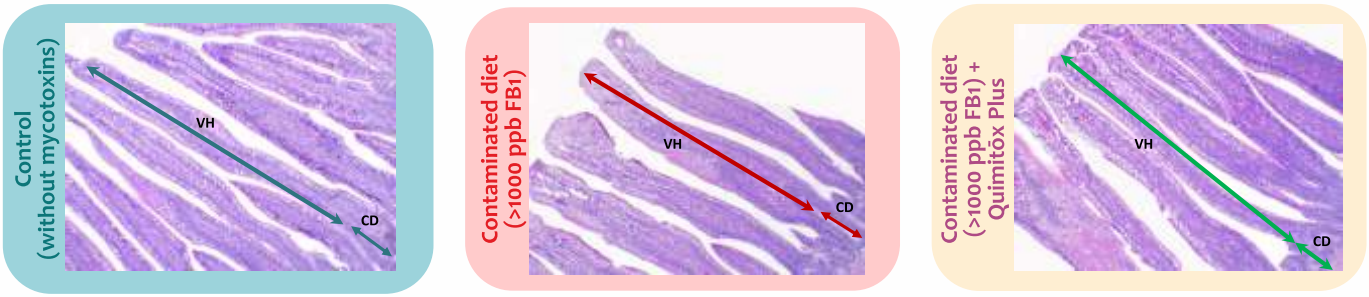


Fig. 5: Villus Height and Crypt Depth (VH, CD) in T-2 toxin Challenged Broilers

Conclusion

T-2 toxin severely impairs broiler performance through growth suppression, gastrointestinal damage and immunosuppression. Supplementation of Quimitōx Plus effectively counteracts T-2 toxin induced adverse effects by restoring intestinal integrity, thereby supporting improved productive performance in broilers.

Study 3

Objective

Evaluate the efficacy of Quimitōx Plus in broilers challenged by aflatoxins B1, B2, G1 and G2.

Introduction

Aflatoxins are highly toxic mycotoxins that frequently contaminate poultry feed and primarily target liver and rapidly developing tissues. In broilers, exposure results in growth retardation (Fig. 1), poor feed efficiency and immunosuppression, as illustrated by stunted birds. Visible lesions include hepatic enlargement and haemorrhagic liver damage (Fig. 2), along with teratogenic effects such as delayed embryonic development and skeletal abnormalities.

Fig. 1: Left- Healthy Bird, Right- Poisoned Bird, Growth Retardation (Lamic, 2019)

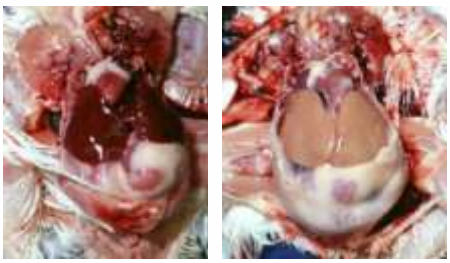


Fig. 2: Left: healthy Bird, Right: Poisoned Bird, Hepatic Damage

Materials and Methods

The study was conducted in Santa Maria, Brazil using 600 one-day-old male (Cobb 500) broiler chicks housed in battery cages under standard management conditions without antibiotic use. Birds were randomly assigned to treatments with 12 replicates of 10 chicks each and evaluated over a 21-day period. Body weight and average daily gain were recorded at 7 and 14 days, while productive parameters, relative liver weight, intestinal and bursal histopathology and microbiological analysis were assessed at 21 days.

Experimental Design

Groups			
Mycotoxin	Control	Contaminated diet	Contaminated diet + Quimitōx Plus @ 2.5 kg/ton feed
Aflatoxin (ppb)	–	>1000	>1000

Note: >1000 ppb represents critical level contamination. Aflatoxin risk levels are classified into low at 5 ppb, medium at 5-20 ppb, high at 20-100 ppb and critical at levels exceeding 1000 ppb.

Results

Broilers challenged with aflatoxins and supplemented with Quimitōx Plus showed improved body weight and growth performance, along with maintained liver integrity as indicated by normalized relative liver weight. Liver histopathology revealed reduced hepatocellular degeneration and inflammatory infiltration, demonstrating protection against aflatoxin-induced damage. Additionally, Quimitōx Plus improved intestinal microbial balance (Fig. 3) and mitigated aflatoxin-related lymphoid depletion in bursa of Fabricius (Fig. 4), supporting better immune organ structure and function.

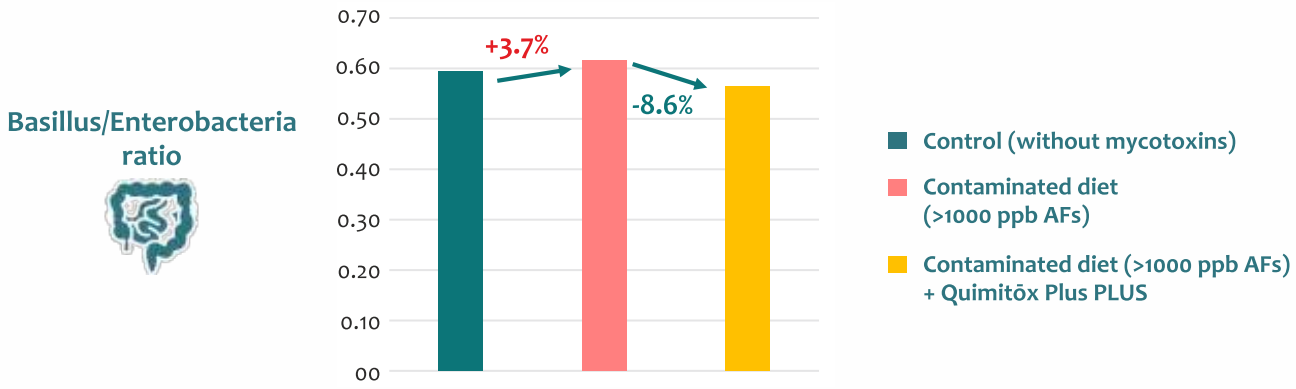


Fig. 3: Intestinal Microbial Balance in Broilers

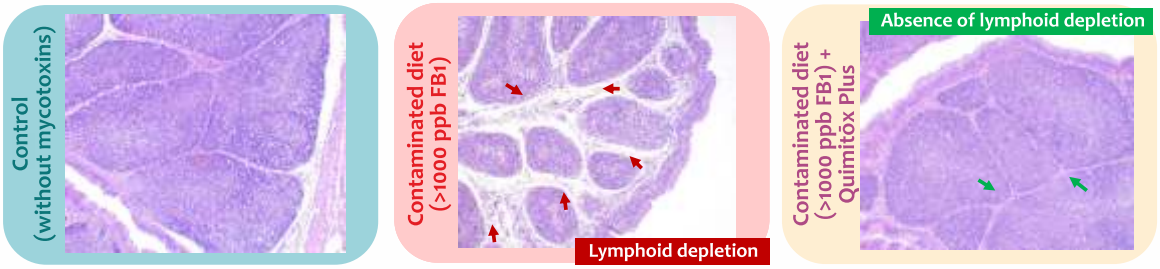


Fig. 4: Effect on Lymphoid Depletion and Function of Bursa of Fabricius in Aflatoxin Challenged Broilers

Conclusion

Aflatoxins pose a major threat to poultry health and productivity, resulting in considerable economic losses. Results from an *in vivo* broiler trial demonstrate that Quimitōx Plus effectively mitigates aflatoxin induced damage by improving productive performance and restoring hepatic, intestinal and bursa of Fabricius integrity.

Conclusion

Mycotoxins produced predominantly by Aspergillus, Fusarium and Penicillium species continue to pose a significant challenge to poultry production by causing intestinal damage, immunosuppression, disruption of gut microbiota, reduced vaccine efficacy and increased susceptibility to disease. Effective mycotoxin management therefore requires a comprehensive and preventive approach. Appropriate decontamination strategies include the use of efficient mycotoxin-binding agents, such as clay adsorbents (hydrated sodium calcium aluminosilicate, bentonite, zeolite and sepiolite), microbiological binders (mannan oligosaccharides and selected strains of Bifidobacterium, lactic acid bacteria and Propionibacterium), along with yeast-based products and phytogenic biosorbents, including extracts of curcumin, silymarin, grape pomace, olive pomace and orange peel. Specialized blends of refined clay adsorbents and yeast-derived products, such as Quimitōx and Quimitōx Plus, offer broad-spectrum protection against commonly occurring mycotoxins in poultry.

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