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Mycotoxins and Microbial Disruption: Insights Into Fungal Pathogenesis and Gut Health: A Literature Review

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Abstract
The human body contains more microbial cells than human cells, with the gut microbiota playing a central role in maintaining homeostasis and regulating immune responses. Mycotoxins, toxic secondary metabolites produced by fungi, often enter the body through contaminated crops and poorly stored food. These compounds can disrupt the gut microbial balance, compromise intestinal barrier integrity, and suppress immune function, increasing susceptibility to infection and disease. Understanding these interactions is essential in evaluating the full impact of mycotoxins on host health. This review explores the interplay between host cells, gut microbiota, and six major food-associated mycotoxins. Findings from both human and animal studies are discussed, focusing on how these toxins disturb microbial communities, induce epithelial damage, and interfere with immune regulation. Mycotoxins contribute to dysbiosis by suppressing beneficial bacteria such as *Lactobacillus* and *Bifidobacterium* while promoting the overgrowth of inflammatory species like *Escherichia coli* and *Clostridium perfringens*. These microbial shifts are closely linked to increased intestinal permeability and pro-inflammatory signalling. A significant concern involves masked mycotoxins—plant-conjugated derivatives that microbial enzymes reactivate in the gut, leading to enhanced toxicity. These forms can escape early detoxification and damage intestinal cells by disrupting membranes and inducing oxidative stress. This review emphasizes the need for therapeutic strategies addressing fungal invasion and mycotoxin toxicity. Potential approaches include inhibiting fungal adhesion, blocking microbial activation of masked toxins, and restoring microbial balance through targeted interventions.

Keywords: mycotoxins; gut microbiota; dysbiosis; immune modulation; intestinal barrier; masked mycotoxins; fungal metabolites; microbial biotransformation; host-microbe interaction

Introduction

Mycotoxins are toxic secondary metabolites produced by various fungal species, particularly those of the Aspergillus, Penicillium, and Fusarium genera, and are commonly found in contaminated food and feed. These compounds pose a serious public health threat due to their widespread occurrence, chemical stability, and biological potency [1, 2]. Chronic exposure, even at low doses, has been associated with various adverse health outcomes, including hepatotoxicity, immunotoxicity, carcinogenesis, and disruption of gut homeostasis [3, 4]. One of the most prevalent and potent mycotoxins is Aflatoxin B1 (AFB1), which is produced by the common soil fungus Aspergillus flavus. Other classes of mycotoxins include ergot alkaloids, which are mycotoxins derived from tryptophan and can be produced by various fungi [5]. Among these, ochratoxin A (OTA), deoxynivalenol (DON or vomitoxin), and fumonisin B1 (FB1) are especially relevant due to their frequent contamination of staple foods such as cereals, roots and tubers, and their well-documented impacts on both cellular and microbial targets. OTA is primarily nephrotoxic and impairs mitochondrial function, DON disrupts protein synthesis and elicits strong inflammatory responses, while FB1 interferes with sphingolipid metabolism and epithelial integrity. Given their structural diversity and widespread occurrence, these toxins provide a valuable framework for investigating how mycotoxins disrupt intestinal function at multiple levels. The gut is essential for nutrient absorption and immune regulation, serving as a barrier between the body and the external environment. The gut microbiota supports digestion, detoxification and protection against pathogens, highlighting the importance of gut integrity when assessing the impact of mycotoxins. Given that the gastrointestinal tract is the primary entry point for these toxins and considering the gut's role as a central immune and metabolic interface, understanding how mycotoxins interact with host cells and gut microbiota is essential. Recent advances in microbiome research and toxicology have underscored a bidirectional relationship whereby

mycotoxins modulate gut microbial communities, and the microbiota, in turn, influence mycotoxin metabolism and toxicity [2, 6]. This discussion explores the mechanisms by which significant mycotoxins such as AFB1, OTA, DON, ZEN, and FB1 compromise intestinal structure, immune function, and microbial balance while also examining the microbiota's potential to bioactivate or detoxify these compounds.

Methods

A targeted literature review was conducted using PubMed and Google Scholar, focusing on primary research articles and relevant reviews on major food-associated mycotoxins' effects on gut microbiota and immune function. Keywords included: "mycotoxin gut microbiota," "mycotoxin immune function," "aflatoxin B1 microbiome," "ochratoxin A immunity," "fumonisin B1 inflammation," and combinations of specific toxin names (OTA, DON, AFB1, FB1, ZEN, ergot alkaloids) with "gut health," "microbial metabolism," and "immune disruption." An initial pool of 126 studies was screened, of which 52 studies met inclusion criteria and were retained for full review and synthesis.

Results & Discussion

Genotoxic Effects: DNA and Protein Damage

Mycotoxins exert toxic effects through various molecular pathways, disrupting critical cellular functions and leading to severe physiological consequences. Understanding these pathways provides insights into the health risks associated with mycotoxin exposure and highlights the importance of mitigating their impact. If we look at AFB1, it has cytotoxic and carcinogenic effects and has been shown to stop RNA and DNA production in rats after 6 weeks of oral exposure to doses ranging from 0.03 to 25 mg/kg. While these doses are higher than typical human exposure, dietary intake in high-risk regions (e.g., ~48–92 µg/day) has been correlated with DNA adduct formation [7]. AFB1 has also been shown to cause severe DNA damage by forming DNA adducts [8]. DNA adducts are chemical bonds formed between a reactive chemical and DNA [9]. In the case of AFB1, bioactivation by hepatic enzymes is required for it to become toxic and reactive [10]. Our key players are a class of enzymes called cytochrome P450 (CYP) [9]. More specifically, we are looking at CYPs 1A2, 3A4, 3A5, and 3A7 in humans [10]. Those CYP enzymes oxidize AFB1 into AFB1-8,9-epoxide, which is highly electrophilic and reactive [10]. This highly reactive metabolite forms covalent adducts with N7 guanine in DNA [8]. N7 guanine refers to the nitrogen atom at the 7th position of a guanine molecule [11]. Once the adduct is formed, we call that complex the AFB1–N7-Gua, which can undergo ring-opening to create a persistent formamidopyrimidine derivative AFB1–FAPY adduct [8]. This is due to the placement of an additional positive formal charge on the guanine ring system, which enhances its overall stability [11]. The AFB1-FAPY adduct is a major contributor to human hepatocellular carcinoma (HCC) development in humans [12]. The connection can be made if we look at the p53 tumor-suppressor gene. Indeed, in over 50% of HCC cases, we can see a clear G to T mutation in the third position of codon 249 of the p53 gene [12]. The main reason why this gene is highly susceptible to this transverse mutation is because it is a G-C-rich region. This mutation makes AFB1–N7-Gua adducts valuable biomarkers of exposure as they are rapidly removed from DNA and excreted in urine [8]. Not only does AFB1 act on DNA, but it also acts at the level of proteins and cellular machinery. Actually, AFB1 can inhibit cyclic nucleotide phosphodiesterase (PDE) activity in the brain, liver, heart, and kidney tissues [13]. PDEs are enzymes that regulate cellular levels of secondary messengers, such as cAMP and cGMP, by controlling their degradation rates [13]. Naturally, alterations in PDE activity can lead to disturbances in signal transduction pathways, affecting cellular metabolism and immune responses.

While AFB1 primarily exerts its toxicity through DNA adduct formation, ochratoxins present distinct challenges to biological systems by principally acting at the mitochondrial level. Through competitive binding to active sites of mitochondrial enzymes, they impair electron transport, disturbing the overall energy production of the cell [14]. Rat experiments have shown that they act at the level of crucial enzymes involved in the citric acid cycle, specifically succinate dehydrogenase, cytochrome C oxidase, and ATP synthase [14]. OTA also competitively inhibits phenylalanyl-tRNA synthetase, a crucial enzyme needed for protein synthesis [14]. The role of this enzyme is to ensure that the correct aminoacyl-tRNAs are provided to the ribosome for protein synthesis [15]. Another experiment efficiently showcased the toxic effects of 100 nmol/l of OTA—such a low concentration induced apoptosis via extracellular protein kinase and caspase activation in kidney cells [16]. Lastly, OTA is also known to induce unscheduled DNA synthesis, as shown by an in-vitro experiment conducted by Dorrenhaus et al. [17].

In addition to these disruptions, certain mycotoxins interfere with regulatory pathways in ways that extend beyond cellular damage. Ergot alkaloids, for example, are known for disturbing multiple physiological functions, especially at the nervous system level [5]. They exercise their neurotoxic effects by disrupting neurotransmission by acting on the biogenic amine receptors [7]. They bind to those receptors and can act as either agonists or antagonists [7]. Primarily, they affect the stimulation of smooth muscle. Inhibition of prolactin secretion is also common, as it acts as a dopamine agonist, which naturally suppresses prolactin release from the pituitary [18]. Indeed, their structure, very similar to the catecholamines, allows them to act on various physiological processes, such as vasoconstriction and vasodilation [19]. They impact such processes by acting on the alpha and beta adrenoreceptors, usually activated by

epinephrine and norepinephrine. Based on their structures and those of the receptors, we expect a more significant effect on the Beta receptors than alpha 1 and 2. Indeed, alpha 1 and 2 have a polar, hydrophilic binding pocket that accommodates catecholamines [20]. On the other hand, β receptors have a more flexible pocket, allowing larger ligands to bind [20]. AFB1 and OTA exert genotoxic effects primarily through DNA adduct formation, mitochondrial disruption, and inhibition of critical enzymes. These mechanisms are closely linked to liver cancer and systemic toxicity. However, most evidence comes from high-dose animal models, and the effects of chronic low-dose exposure in humans are not fully understood. There is also limited insight into how co-exposure to multiple mycotoxins may amplify genotoxic outcomes (Figure 1.1).

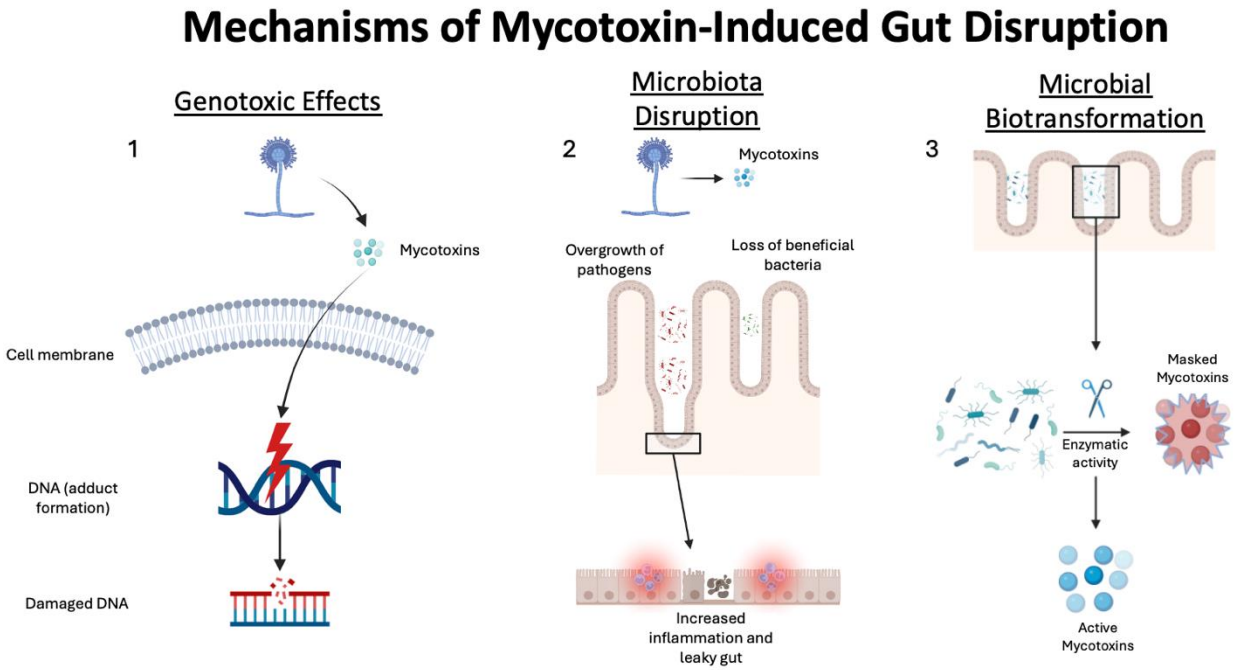

Figure 1. Three Major Pathways of Mycotoxin-Induced Gut Disruption. This figure illustrates how mycotoxins compromise gut health through three main mechanisms: (1) genotoxic effects via DNA adduct formation, (2) disruption of gut microbiota leading to inflammation and barrier dysfunction, and (3) microbial biotransformation of masked mycotoxins into active, more toxic forms. This figure was created with BioRender.

Impact of Mycotoxins on Gut Microbiota

Beyond their direct effects on DNA integrity, protein synthesis, and cellular metabolism, mycotoxins also profoundly influence the gut microbiota. Given that the gastrointestinal tract is one of the first exposure sites, mycotoxins profoundly influence gut microbiota composition, leading to dysbiosis, reduced microbial diversity, and a shift in microbial populations favouring pathogenic species. Exposure to OTA, Vomitoxin (DON), and Fumonisin B1 (FB1) has been shown to selectively inhibit beneficial gut bacteria, particularly Lactobacillus and Bifidobacterium, both of which play a critical role in gut homeostasis, immune modulation, and short-chain fatty acid (SCFA) production [2]. The depletion of these protective bacteria compromises intestinal barrier integrity, increases inflammation, and reduces microbial resilience. For instance, OTA exposure leads to a decline in *Lactobacillus reuteri*, a key bacterial species involved in gut protection, while simultaneously promoting the growth of inflammatory bacteria such as Bacteroides [2]. Additionally, AFB1 exposure has been linked to a reduction in Faecalibacterium prausnitzii, a bacterium with anti-inflammatory properties, further exacerbating gut inflammation and weakening the intestinal barrier [3]. As beneficial microbes decline, opportunistic and pathogenic bacteria such as Escherichia coli, Clostridium perfringens, and Enterobacteriaceae proliferate, intensifying gut inflammation and disrupting metabolic balance [1]. This shift is particularly evident with DON exposure, which significantly increases the abundance of Proteobacteria, a microbial marker of gut dysbiosis commonly associated with inflammatory bowel disease (IBD) and metabolic disorders [1]. Similarly, OTA has been shown to favour an increase in Bacteroidetes over Firmicutes, a microbial imbalance frequently linked to gut inflammation and metabolic dysfunction [4]. ZEN and DON further contribute to dysbiosis by increasing pathogenic Firmicutes, impairing gut homeostasis and reducing essential nutrient absorption [21]. Beyond these microbial shifts, mycotoxin exposure also significantly declines

overall gut microbiota diversity, further weakening microbial resilience and increasing susceptibility to gut-related diseases. Chronic exposure to AFB1 and OTA has been linked to a substantial reduction in alpha diversity, diminishing the microbiota's ability to adapt to external stressors [3]. Lower microbial diversity also impairs metabolic flexibility, reducing the microbiome's capacity to produce protective metabolites like SCFAs, which are essential for maintaining gut barrier integrity and immune function [2]. Additionally, DON exposure negatively impacts the functional potential of gut microbiota, leading to a decline in microbial metabolic activity and protective biochemical pathways [4]. Taken together, these microbial alterations demonstrate the significant role of mycotoxins in compromising gut health by reducing beneficial bacteria, promoting the overgrowth of inflammatory pathogens, and diminishing microbial diversity. These disruptions weaken intestinal barrier function, increase inflammation, and contribute to long-term metabolic and immune dysregulation, making the gut more susceptible to chronic diseases.

One of the most critical consequences of mycotoxin exposure is the disruption of intestinal barrier integrity, commonly referred to as "leaky gut." The intestinal epithelium forms a selective barrier regulated by tight junction proteins such as occludin, claudins, and ZO-1, which maintain cell-cell adhesion and regulate paracellular permeability. Mycotoxins such as DON, OTA, and FB1 have been shown to directly impair these tight junctions, compromising the gut barrier and facilitating the translocation of harmful luminal contents. For instance, DON significantly downregulates tight junction protein expression, resulting in greater intestinal permeability and barrier breakdown [3]. OTA exposure similarly disrupts epithelial integrity, not only by degrading tight junctions but also by inducing oxidative stress, which further destabilizes epithelial cells [4]. In addition to structural damage, OTA and FB1 have been shown to cause the shedding of epithelial villi. This process impairs nutrient absorption and leaves the mucosal surface vulnerable to pathogen invasion [3].

As the intestinal barrier weakens, bacterial endotoxins like lipopolysaccharides (LPS) can enter systemic circulation, triggering low-grade chronic inflammation and promoting the development of conditions such as IBD, metabolic syndrome, and non-alcoholic fatty liver disease (NAFLD) [4]. This translocation is particularly concerning in the context of gut dysbiosis, where pathogenic bacteria are already proliferating. Moreover, mycotoxin-induced damage to the barrier is often dose-dependent, with even low concentrations of OTA or DON being sufficient to alter gut permeability and tight junction expression. In parallel, the loss of mucin-secreting goblet cells, reported following DON and ZEN exposure, further compromises the physical mucus layer that shields epithelial cells, leaving the intestinal wall highly exposed to toxins and microbial insults (5). Beyond damaging the epithelial barrier, exposure to these toxins also interferes with the immune functions of the gut. Several studies have shown that compounds like OTA and FB1 impair mucosal immunity by reducing macrophage and dendritic cell activity, ultimately weakening the gut's ability to detect and respond to pathogens [3]. At the same time, DON has been shown to suppress the secretion of secretory IgA, a key protective immunoglobulin in the intestinal lumen [2]. These immune disruptions are compounded by the pro-inflammatory responses triggered by many mycotoxins, which can upregulate cytokines such as TNF-α, IL-6, and IL-1β, driving chronic inflammation [4]. Together, these effects contribute to a sustained inflammatory environment in the gut and may pave the way for systemic immune dysfunction and autoimmune complications. This shows that mycotoxins do not merely act as localized irritants; they systematically undermine gut integrity, increasing permeability, promoting microbial translocation, and driving inflammatory responses that can ripple beyond the gastrointestinal tract. Mycotoxins cause significant shifts in gut microbial communities, reducing beneficial bacteria, increasing pathogens, and compromising the intestinal barrier. These disruptions contribute to inflammation and metabolic dysfunction. Nonetheless, individual variability in microbiota composition and resilience remains a major gap. More research is needed to understand how factors like diet, genetics, and pre-existing gut health influence susceptibility (Figure 1.2).

Microbiota-Driven Mycotoxin Biotransformation

Another important yet often overlooked consequence of mycotoxin exposure is their biotransformation by gut microbes. While some microbial species can detoxify these compounds, others may instead convert them into more toxic or bioactive forms, enhancing their harmful potential. This phenomenon is particularly well-documented in the case of masked mycotoxins—plant- or animal-conjugated versions of mycotoxins that are not initially toxic but become harmful after microbial hydrolysis in the gut. For instance, DON-3-glucoside, a masked derivative of DON, is hydrolyzed back into its toxic parent compound by enzymes produced by intestinal bacteria [2]. Similarly, zearalenone (ZEN) can be reduced by gut microbes to α-zearalenol, a metabolite with significantly higher estrogenic activity than ZEN itself, particularly in species like pigs and ruminants [2, 4]. These microbial transformations not only increase the toxic burden on the host but may also extend the biological half-life of these compounds by modifying their absorption profile. This is especially problematic in monogastric animals and humans, where much of the microbial conversion occurs after the primary absorption site, meaning the toxic metabolite may escape hepatic detoxification altogether [1]. Adding to this complexity, certain gut bacteria possess β-glucosidases and sulfatases,

which are capable of cleaving conjugated mycotoxins and releasing their toxic parent structures back into circulation [3]. These processes often occur in the colon long after the small intestine has completed its role in nutrient absorption, making them harder to regulate and more dangerous in chronic exposure scenarios. While microbial metabolism is sometimes assumed to be protective, these findings emphasize that the interaction between gut microbiota and mycotoxins is not inherently beneficial. In some cases, these interactions can directly exacerbate toxicity. Understanding individual microbial communities' enzymatic profiles and metabolic potential is essential for evaluating host susceptibility to dietary mycotoxins and may offer new targets for intervention or risk stratification. Gut microbes can transform mycotoxins into either less or more toxic forms, influencing their overall impact. This is especially relevant for masked mycotoxins and conjugated metabolites. However, the enzymes and microbial species involved vary widely between individuals, and the long-term health consequences of these microbial conversions are not well defined. Further work is needed to map these interactions and assess their role in human risk (Figure 1.3).

Conclusion

The findings outlined in this discussion highlight the complex and multifaceted nature of mycotoxin toxicity, extending beyond molecular damage at the DNA and protein levels to include wide-reaching effects on the gut microbiota and host immune systems. From disrupting tight junction proteins and mucosal immune defences to driving microbial dysbiosis and systemic inflammation, mycotoxins compromise gut integrity through several converging pathways. These disruptions are not only localized but also systemic, contributing to chronic conditions such as IBD, metabolic disorders, and even hepatocellular carcinoma. Moreover, the gut microbiota plays a dual role, functioning as both a modulator and a mediator of toxicity—where, in some contexts, microbial enzymes detoxify mycotoxins. Still, in others, they convert them into more toxic metabolites, heightening their pathogenic potential. This interplay underscores the importance of microbiota-aware toxicological assessments and encourages future research into microbial-targeted interventions such as probiotics, postbiotics, and microbiome-based diagnostics. Ultimately, a comprehensive understanding of the gut–mycotoxin axis will be essential in developing strategies for risk reduction, personalized nutrition, and therapeutic prevention in populations chronically exposed to dietary mycotoxins. Despite these advancements, several limitations persist across the current literature. Many studies are based on animal models or short-term in vitro experiments, limiting their direct applicability to chronic human exposure. The combined effects of multiple mycotoxins remain poorly understood, as most studies examine single compounds in isolation. Additionally, individual differences in microbiota composition and immune responses are rarely accounted for, making it difficult to generalize findings across populations. More longitudinal, human-centered research is needed to clarify dose thresholds, synergistic toxicities, and the long-term impact of low-level exposure.

List of Abbreviations

AFB1: aflatoxin B1
CYP: cytochrome P450
DON: deoxynivalenol (vomitoxin)
FAPY: formamidopyrimidine
FB1: fumonisin B1
HCC: hepatocellular carcinoma
IBD: inflammatory bowel disease
IgA: immunoglobulin A
IL-1β: interleukin 1 beta
IL-6: interleukin 6
LPS: lipopolysaccharide
NAFLD: non-alcoholic fatty liver disease
OTA: ochratoxin A
PDE: phosphodiesterase
SCFA: short-chain fatty acid
TNF-α: tumor necrosis factor alpha
ZEN: zearalenone

Conflicts of Interest

The authors declares that they have no conflict of interest.

Ethics Approval and/or Participant Consent

This study did not require research ethics board (REB) approval or participant consent, as it is a literature-based review that did not involve the collection of new data from human participants or animals.

Authors' Contributions

AK: made substantial contributions to the conception and design of the study, conducted the literature review and analysis, drafted and revised the manuscript critically for important intellectual content, approved the final version to be published, and agrees to be accountable for all aspects of the work.

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