Advances in Research on Mycotoxins Affecting Intestinal Mucosal Barrier Function: Postprint

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Abstract

Mycotoxins are a class of hazardous agents widely present in feed ingredients and human food, posing severe threats to animal and human health. As the first line of defense against exogenous contaminants, the intestinal mucosal barrier is primarily composed of interconnected mechanical, chemical, immunological, and biological barriers. Based on existing domestic and international research, this paper provides a comprehensive review of the effects of mycotoxins on intestinal mucosal barrier function and the underlying mechanisms.

Full Text

Effects of Mycotoxins on Intestinal Mucosal Barrier Function: A Review

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Abstract: Mycotoxins are hazardous substances widely present in feed ingredients and human food that pose serious threats to animal and human health. As the first line of defense against external contaminants, the intestinal mucosal barrier consists of four interrelated functional components: the mechanical barrier, chemical barrier, immune barrier, and biological barrier. This review summarizes the effects of mycotoxins on intestinal mucosal barrier function and their underlying mechanisms based on existing research worldwide.

Keywords: mycotoxins; intestinal mucosa; intestinal barrier

Mycotoxins are toxic secondary metabolites produced by various fungi, including *Aspergillus*, *Penicillium*, and *Fusarium* species, and are widely found in feed and food [1-3], posing significant threats to both animal and human health [4]. As the primary barrier against external pollutants [5-6], the intestine is responsible for 70% of the body' s immune defense [5]. Since mycotoxins are mainly absorbed through the intestinal tract, intestinal epithelial cells are the first to encounter high concentrations of these toxins, resulting in intestinal functional damage [7]. Mycotoxins such as deoxynivalenol (DON), ochratoxin A (OTA), and T-2 toxin exhibit strong intestinal pathogenicity, frequently causing gastrointestinal dysfunction, diarrhea, vomiting, and malnutrition [8-9]. Numerous studies have demonstrated that mycotoxins can disrupt intercellular tight junctions, induce intestinal lesions, modulate intestinal immune responses, alter intestinal immune barrier function, destabilize intestinal microbial flora, and trigger intestinal inflammation. This review examines the effects of mycotoxins on intestinal mucosal barrier function and their mechanisms of action, providing a theoretical foundation for future in-depth research in this field.

1. Effects of Mycotoxins on Intestinal Mucosal Mechanical Barrier Function

The intestinal mucosal mechanical barrier, also known as the physical barrier, is primarily composed of intestinal epithelial cells and the tight junction proteins between them. This structure effectively prevents harmful substances such as bacteria, toxins, and inflammatory mediators from the intestinal lumen from crossing the mucosa into the bloodstream, thereby maintaining the integrity of the intestinal epithelial barrier [10-11].

Intestinal epithelial cells possess rapid proliferative and regenerative capacities that sustain mechanical barrier function [12]. Goossens et al. [13] found that treatment of porcine intestinal epithelial IPEC-J2 cells with DON and T-2 toxin significantly reduced cell viability in a dose-dependent manner, although low concentrations of these toxins did not cause significant changes. Ivanova et al. [14] demonstrated that high concentrations (25 mol/L) of enniatin B (ENB) arrested the cell cycle of human colon cancer Caco-2 cells at the G2/M phase, leading to necrosis. Animal studies have shown that oral administration of DON to mice or piglets significantly reduced villus height in the intestinal epithelial zone compared to control groups fed normal diets [15-16]. Kolf-Clauw et al. [17] reported that exposure to DON for 4 hours significantly decreased villus length in jejunal explants from 4-5-week-old and 9-13-week-old pigs, though low concentrations (0.3 mg/kg) had no significant effect on villus length in younger pigs. These findings indicate that under short-term feeding conditions, animal age is a primary factor influencing mycotoxin effects, and animals possess a certain tolerance to low-dose mycotoxins.

Tight junctions between intestinal epithelial cells maintain the integrity of the mechanical barrier. Diesing et al. [18] showed that high concentrations (2000 ng/mL) of DON reduced the expression of tight junction protein ZO-1 in porcine intestinal epithelial IPEC-1 and IPEC-J2 cells, compromising barrier integrity. Interestingly, low concentrations (200 ng/mL) not only showed no toxicity but actually promoted cell proliferation. These results suggest that disruption of mechanical barrier integrity may be one pathway through which mycotoxins exert their toxic effects, and that different doses may operate through distinct mechanisms.

Pinton et al. [19] found that DON inhibited synthesis of tight junction protein claudin-4 in IPEC-1 cells by activating extracellular regulated protein kinases (ERK) in the mitogen-activated protein kinase (MAPK) signaling pathway. The detrimental effects of mycotoxins on tight junction proteins have also been confirmed in animal studies. Male B6C3F1 mice orally administered DON showed increased claudins mRNA expression and altered distribution in the distal small intestine [15]. Piglets continuously fed diets contaminated with low doses of DON (3 mg/kg) or fumonisin B1 (FB1) (6 mg/kg) for 5 weeks exhibited significantly reduced occludin expression in the small intestine [20].

Collectively, these studies demonstrate that the intestinal mucosal mechanical barrier can maintain its integrity through self-regulation when challenged by short-term, low-concentration mycotoxin exposure. However, when damage exceeds this self-regulatory capacity, intestinal epithelial cells undergo pathological changes, tight junction protein expression declines, and the mechanical barrier becomes compromised. Notably, long-term exposure to low-dose mycotoxins also adversely affects the intestine. Therefore, when establishing minimum detection limits for mycotoxins in animal feed, both toxin dosage and feeding duration must be considered.

2. Effects of Mycotoxins on Intestinal Mucosal Chemical Barrier Function

The chemical barrier consists of mucus secreted by intestinal epithelial cells, digestive fluids, and antimicrobial substances produced by normal intestinal flora [11]. The mucus layer, located on the luminal surface of epithelial cells and primarily composed of glycosylated mucins (MUC) produced by goblet cells, plays a crucial role in preventing external contaminants from reaching deeper tissues [6]. Bae et al. [21] reported that DON reduced MUC synthesis in human histiocytic lymphoma U937 cells and murine mononuclear macrophage RAW264.7 cells. Wan et al. [6] demonstrated that DON, zearalenone (ZEA), nivalenol (NIV), and FB1, individually or in combination, significantly altered MUC5AC and MUC5B mRNA expression in human intestinal epithelial cells. The similar transcriptional responses of MUC5AC and MUC5B genes to mycotoxins suggest a common regulatory mechanism. Pinton et al. [22] showed that reduced MUC expression depended on activation of ERK and MAPK p38.

Mammalian intestinal epithelial cells produce numerous antimicrobial peptides (AMPs), particularly defensins, to cope with the complex microbial environment [23]. Wan et al. [24] found that individual or combined *Fusarium* toxins (DON, NIV, ZEA, FB1) significantly increased mRNA expression of porcine β-defensin 1 (pBD-1) and pBD-2 in porcine intestinal epithelial IPEC-J2 cells, though secreted protein abundance remained unchanged. The discrepancy between mRNA and protein levels may be explained by: (1) post-transcriptional or post-translational regulatory mechanisms and protein degradation pathways affecting defensin molecules [25]; and (2) lower sensitivity of protein quantification techniques compared to mRNA measurement methods [26]. Animal studies have shown that supplementation with composite antimicrobial peptides (CAP) in DON-contaminated feed significantly improved peripheral blood lymphocyte proliferation, increased platelet counts, enhanced serum catalase activity, and reduced malondialdehyde content in piglets, indicating that CAP can ameliorate DON-induced intestinal injury by improving intestinal morphology, immune function, and antioxidant capacity while reducing organ damage [27].

Results from different experimental models (cell culture and in vivo studies) across various species (mice, pigs, humans) collectively indicate that mycotoxins can activate the intestinal mucosal chemical barrier as a defense mechanism. However, the precise mechanisms remain unclear. Future research should integrate transcriptomics and proteomics using molecular biology approaches to further elucidate how mycotoxins affect chemical barrier function.

3. Effects of Mycotoxins on Intestinal Mucosal Immune Barrier Function

The selective permeability of the intestinal mucosa—allowing entry of luminal contents such as food and drugs while blocking exogenous pathogens—is mediated by both mechanical and immune barriers [28]. The intestinal mucosal immune barrier has become a major research focus and consists primarily of gut-associated lymphatic tissue (GALT), mesenteric lymph nodes, and secretory immunoglobulin A (S-IgA) produced by intestinal plasma cells [29-30]. GALT comprises Peyer' s patches, mesenteric lymph nodes, and numerous lymphocytes within the intestinal epithelium [31]. S-IgA neutralizes endotoxins, forms antigen-antibody complexes with bacterial antigens, stimulates mucus secretion, accelerates mucus flow across the epithelial surface, and prevents pathogen adhesion [32-34], while also regulating immunity, immune exclusion, gut microbiota, and antimicrobial factor production [35].

He et al. [36] reported that dietary aflatoxin B1 (AFB1) at 0.3 mg/kg reduced IgA-positive (IgA+) cell numbers and decreased S-IgA, IgA, IgG, and IgM contents in the intestine of male broilers. Reduced S-IgA increases interactions between intestinal bacteria/endotoxins and epithelial cells, promoting bacterial translocation and endotoxin absorption, which may contribute to compromised intestinal immunity [37]. Li et al. [38] found that mycotoxin-contaminated feed significantly reduced serum IgA levels in broilers. Since intestinal mucosal im-

munity is IgA-mediated and IgA neutralizes viruses and other antigens within cells while returning products to the lumen to prevent epithelial damage, decreased IgA can lead to loss of mucosal immune responses. Studies confirm that mycotoxins damage the immune barrier by reducing immunoglobulin expression. Grenier et al. [39] observed that DON and FB1 decreased serum IgG levels and lymphocyte proliferation in piglets. As IgG represents the second line of defense in inflammatory responses [40], its reduction can disrupt immune balance and compromise the barrier. However, Swamy et al. [41] reported no significant changes in serum immunoglobulin levels in male broilers fed high levels of *Fusarium* toxins (8.2 mg/kg DON, 0.56 mg/kg ZEA) for 56 days, possibly due to variations in mycotoxin type, concentration, exposure duration, and differences in animal species, age, and sex.

Lymphocytes secrete various cytokines and inflammatory mediators that regulate intestinal immune function through anti-infective humoral and cytotoxic cellular immunity, protecting against pathogenic antigens [11,29]. Mahmoodi et al. [42] demonstrated that FB1 dose-dependently promoted expression of macrophage chemotactic factors and pro-inflammatory cytokines in gastric epithelial AGS and human colon adenocarcinoma SW742 cell lines. Kadota et al. [43] showed that DON stimulated interleukin-8 (IL-8) secretion in Caco-2 cells. In IPEC-1 cells, ZEA increased synthesis of IL-8 and IL-10 [44]. Taranu et al. [45] found that ZEA alone did not significantly affect cytokine expression in IPEC-1 cells, but when combined with *E. coli*, it markedly increased secretion of interferon-γ (IFN-γ), IL-10, and tumor necrosis factor-α (TNF-α). These findings indicate that mycotoxins exert both direct pro-inflammatory effects on the intestine and indirect inflammatory effects through alterations in intestinal function [46]. Increased pro-inflammatory cytokine secretion reduces tight junction integrity and increases intestinal permeability, facilitating translocation of luminal hazards into the bloodstream [47].

4. Effects of Mycotoxins on Intestinal Mucosal Biological Barrier Function

The intestinal microbiota constitutes an important biological barrier, with obligate anaerobes as the dominant flora that prevent pathogen invasion and colonization. Reductions in anaerobic bacteria destabilize the microbial community, decrease colonization resistance, and allow exogenous pathogens to adhere to the mucosa, causing diarrhea, enteritis, and other intestinal diseases [10,32]. Niderkorn et al. [48] reported that fermentative bacteria in the gastrointestinal tract can bind ZEA and FB1, effectively reducing their toxicity. Young et al. [49] used liquid chromatography-UV mass spectrometry to demonstrate that intestinal microbes can degrade trichothecene mycotoxins through deacetylation. Wachéy et al. [50] observed dynamic changes in intestinal flora of animals exposed to DON using capillary electrophoresis single-strand conformation polymorphism.

The intestine serves as the first barrier against external pollutants, comprising

four interconnected components: mechanical, chemical, immune, and biological barriers. Damage to any component can compromise overall barrier function. Research demonstrates that mycotoxins disrupt intestinal epithelial barrier function and induce intestinal lesions in animals and humans. To protect animal and human health, mycotoxin production must be controlled in production practices through proper mold prevention and detoxification. Current research on mycotoxin effects on intestinal barrier function has primarily focused on humans and monogastric animals, with limited studies in ruminants. Furthermore, mechanisms underlying mycotoxin-mediated barrier damage, such as the regulatory mechanisms of immunoglobulin expression in the immune barrier, remain poorly understood. Future studies should employ molecular biology and toxicogenomics approaches to investigate mycotoxin-induced barrier damage at the molecular level, establishing a comprehensive theoretical framework.

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