Fumonisin Toxicosis and its Effects on Human Health: Sources, Detection, and Risk Mitigation

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ABSTRACT

Fumonisins are mycotoxins produced primarily by *Fusarium* species that frequently contaminate maize and other cereal crops, posing significant food safety and public health concerns worldwide. Chronic exposure to fumonisins, particularly fumonisin B_1 (FB_1), has been associated with adverse human health outcomes, including disruption of sphingolipid metabolism, oxidative stress, immunotoxicity, neural tube defects, and increased risk of oesophageal cancer in high-exposure populations. This review synthesizes scientific knowledge on the sources, global occurrence, and epidemiology of fumonisin contamination, highlighting environmental and storage factors that influence toxin production. The key molecular mechanisms of fumonisin toxicity, especially inhibition of ceramide synthase and consequent alterations in membrane lipid homeostasis, are discussed alongside evidence from epidemiological and experimental studies. Advances in fumonisin detection methods and international regulatory guidelines established by WHO and EFSA are also reviewed. The paper emphasizes the need for continued surveillance, improved food safety practices, and effective mitigation strategies, particularly in regions where maize-based diets predominate.

Keywords: Fumonisin, Mycotoxin, Mycotoxicosis, Fusarium, Human health.

INTRODUCTION

Mycotoxin is a broad term for the harmful substances produced by molds. Mycotoxins are the most toxic chemical agents found in food and feed, posing the greatest threat to human and animal health (Tian *et al.*, 2022). Mycotoxicosis is the name of the illness caused by mycotoxins (Misihairabgwi *et al.*, 2016). The World Food and Agriculture Organization (FAO) estimates that, every year, over 25% of cereal products are wasted globally due to mycotoxin infection (Zhou *et al.*, 2018). Hepatotoxicity, nephrotoxicity, immunotoxicity, reproductive toxicity, and carcinogenicity of mycotoxins pose serious threats to human and animal health (Rocha *et al.*, 2017).

Fumonisins are toxic, low-molecular-weight, and water-soluble mycotoxins mainly produced by *Fusarium verticillioides* and *Fusarium proliferatum* (Zhou *et al.*, 2018). Furthermore, fumonisins are produced by *A. nigri* in crop plants such as peanut, maize, and grape (Astoreca *et al.*, 2007). Fumonisins can be found in a variety of grains and grain products, but they are most typically found in maize and maize-based products (rice, wheat, barley, maize, rye, oat, and millet) (Cendoya *et al.*, 2018).

Aims and Objectives

The aim of this review is to critically evaluate fumonisin toxicosis and its implications for human health, with emphasis on exposure pathways, mechanisms of toxicity, and associated public health risks.

Specific Objectives

1. To describe the sources and routes of human exposure to fumonisins, particularly through contaminated maize and cereal products.

2. To review epidemiological and experimental evidence linking fumonisin exposure to adverse human health outcomes such as oesophageal cancer, neural tube defects, and growth impairment.

3. To discuss regulatory guidelines and risk assessment frameworks established by international bodies such as WHO and highlight research gaps related to fumonisin exposure, especially in developing countries

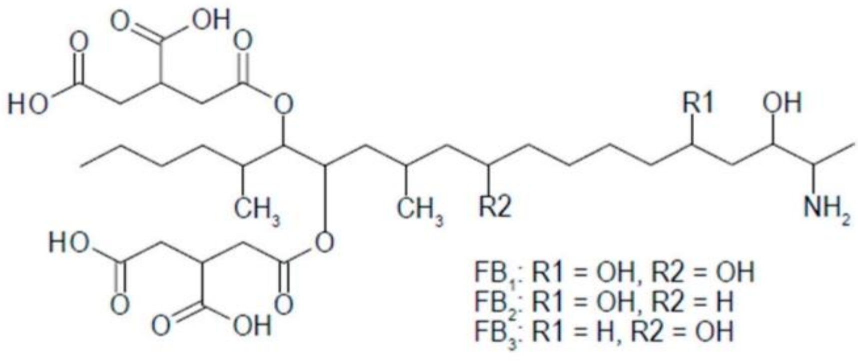
Rationale and Contribution of the Review

Despite the extensive body of literature on fumonisins, important gaps remain in the integrated understanding of their implications for human health. Many existing reviews focus either on agricultural contamination, animal toxicosis, or analytical detection methods, with comparatively limited emphasis on the convergence of exposure pathways, mechanisms of toxicity, and human health outcomes. This paper integrates fumonisin contamination with mechanistic insights and public health perspectives. In addition, the review critically examines the regulatory guidelines, detection strategies, and mitigation approaches in light of emerging scientific evidence. By bridging mechanistic toxicology, epidemiology, and food safety regulation, the review also provides a comprehensive resource for researchers, public health professionals, and policymakers, and identifies priority areas for future research and risk management.

Classification of Fumonisin

Since they were discovered in 1988, about 28 fumonisins have been recognized and categorized into the four most important groups: A, B, C, and P, and the 28 analogies are; FA1, FA2, FA3, PHFA3a, PHFA3b, HFA3, FAK1, FBK1, FB1, Iso-FB1, PHFB1a, PHFB1b, HFB1, FB2, FB3, FB4, FB5, FC1, N-acetyl-FC1, Iso-FC1, N-acetyl-iso-FC1, OH-FC1, N- acetyl–OH–FC1, FC3, FC4, FP1, FP2, FP3. (Rheeder *et al.*, 1992).

The most abundant fumonisin B forms are FB1, FB2, and FB3, with FB1 being the most dangerous form that may coexist with the other fumonisin forms, FB2 and FB3 (Damian *et al*., 2019). The three categories of food pollutants (FB1, FB2, and FB3) are the most common. FB1 is a diester composed of propane-1,2,3-tricarboxylic acid (TCA) and 2-amino-12,16-dimethyl-3,5,10,14,15-pentahydroxyleicosane, with C-14 and C-15 hydroxyl (OH-) forming an ester with TCA carboxyl groups (-COOH). FB2 and FB3 are the C-5 and C-10 dehydroxy counterparts of FB1, respectively (Shephard, 1998). FB1 affects the nutritional value and sensory characteristics of feed, leading to a decrease in food intake and production performance, and causing huge economic losses (Rheeder *et al*., 1992). At the same time, this mycotoxin will contaminate various food and its products, and cause 70% of global food contamination. Long-term contact with the mycotoxin will cause harm to the health of those exposed (Rocha *et al*., 2017).



(Anumudu *et al*., 2025).

Fig. 2 Chemical structure of fumonisin B1, B2, B3

The international agency for research on cancer (IARC) also identified FB1 as a class 2B carcinogen, which may be carcinogenic to humans. It has been reported FB1 can be hydrolyzed into FB1 (hFB1) by enzymatic degradation, so FB1 degradation enzyme can be used as feed additive and reduce animal fumonisin exposure (Rocha *et al*., 2017).

Common sources of fumonisins

Fumonisins are mostly generated by *F. verticillioides, F. proliferatum,* and other Fusarium spp. Fusarium, a member of the Nectriaceae family, can be found as saprophytes in soil and plants all around the world (Burgess, 1981). Fusarium spp. colonises plant rhizospheres and eventually penetrates the plant system. Furthermore, the most common maize (Zeamays) infections are *F. verticillioides* and *F. proliferatum* (Marasas, 2001). Several Fusarium species, including *F. oxysporum, F. foetens, F. hostae, and F. redolens*, regularly attack many popular decorative plants (e.g., aster begonia, carnation, chrysanthemum, gladiolus, etc.) at various stages of production (Gullino *et al.*, 2002). Fusarium, on the other hand, causes both pathogenic and non-pathogenic infections in orchids. Non-pathogenic forms are either decomposers or mutualists, helping with seed germination and seedling color development. (Vujanovic *et al*., 2000).

Epidemiology

Fumonisin contamination is widespread throughout the world in corn, wheat, rice, millet, oats, sorghum, soya beans, and relevant products (Seo *et al.*, 2013). An investigation indicated mycotoxin contamination exists in different regions of the world, including North America, Central Europe, Africa, South Asia, and Southeast Asia. Approximately 27% to 58% of crops were contaminated in these areas. The highest positive rate, 76%, was reported in South America (at a mean contamination concentration of 1.50 mg/kg). In 2012, FB1 and FB2 contamination rates in animal feeds in Korea were 50% and 40%, respectively (Kim *et al.*, 2013). From 2011 to 2013, the contamination rate of fumonisins (FB1 + FB2) in China's Hebei province increased to 46.4% (Li *et al*., 2015). In 2014, the FB1, FB2, and FB3 contamination rate in maize products from Shandong province was 98.1%, with the highest levels being 5046, 1350, and 712.1 µg/kg, respectively, and 76.7% of maize samples were contaminated with FB1, FB2, and FB3 (Li *et al*., 2015).

A survey in Algeria showed that fumonisins frequently contaminate maize in the country. Fumonisin FB1 was present in29 out of 30 samples, and FB2 was detected in 27 of 30 samples. The mean concentration of fumonisins in positive samples were 14,812 µg/kg for FB1 and 8603 µg/kg for FB2. (FAO, 2009). Another study from 20 districts in Egypt revealed that the fumonisin content in maize was only 33 µg/kg, while that of rice was 1014 µg/kg (Hussein *et al*., 2017). Twenty samples each of maize, wheat and barley were collected on the markets of Rabat and Salé in Morocco and analysed for fumonisins. The average fumonisin concentration was 1930 µg/kg and the co-occurrence of several mycotoxins was determined (El-Sayed *et al*., 2003). In Tunisia, fumonisins were detected in 10.5% of 180 maize food samples with levels from 70 to 2130 µg/kg. All contaminated samples contained FB1 and 31.5% of the samples contained FB2. (Marocco *et al.,* 2008).

High fumonisin contamination of maize has been detected in Benin Republic. Local fumonisin levels were reported to range 8240–16,690 µg/kg, with variation from year to year and throughout the storage period (Jedidi *et al*., 2021). Analysis of 122 samples, mainly from maize and groundnuts collected in Burkina Faso, included analysis of fumonisin B1. The fumonisin incidence was 81% and the median fumonisin contamination was 269 µg/kg (Nikièma *et al.,* 2004). Altogether 55 samples of raw maize and 12 samples of sorghum were collected in the market in Togo and analysed for fumonisins. Of the maize samples, 88% contained fumonisins in concentrations from 101 to 1831 µg/kg, and 67% of the sorghum samples contained from 81.5 to 361 µg/kg fumonisins (Bankole and Adebanjo, 2003).

In Nigeria during during a one year survey in Ogun State, revealed that 13% of the maize samples contained fumonisins above the limit set by the Regulatory Authorities. In North Central Nigeria the fumonisin contamination was 50–8400 µg/kg in maize harvested from the fields, 50–8150 µg/kg in maize collected from stores, and 10–6150 µg/kg in maize offered on the markets (Liverpool-Tasie *et al.*, 2019).

Recent epidemiological data confirm that fumonisin contamination of cereals remains a global food safety challenge. In Europe, surveillance between 2023 and early 2025 reported fumonisins in approximately 60–61%

of maize and cereal samples, highlighting persistent exposure risks (Schieszl *et al*., 2025). Multi-year monitoring in Serbia documented fumonisin occurrence across 2021–2023 with significant levels often above safety thresholds, reflecting climatic and agricultural influences on contamination (Penagos-Tabares *et al*., 2025).

At the global scale, meta-analytic data indicate that fumonisins remain one of the most frequently detected mycotoxins in cereal-based foods (Farhadi *et al*., 2021). Longitudinal food product surveys also show high occurrence rates over 2020–2024, with some years' exposure estimates exceeding health-based guidance values (Gökışık, and Kahtalı, 2025).

Factors Affecting the Occurrence of Fumonisins

The occurrence of fumonisins in agricultural products is dependent on a range of factors, such as geographical region, season, and particular environmental conditions in which the food product is grown, harvested, and stored. The tropical and subtropical regions of the world, such as sub-Saharan Africa, are the most favorable regions for fungi development on food commodities and mycotoxin production (Zakaria, 2023). Moisture content and temperature have been demonstrated to be the critical environmental factors that affect the production of fumonisins during storage. The effect of different temperatures and water activities on fungal growth and fumonisin production by Aspergillus species was studied by Perera *et al.* (2021). The study demonstrated the effect of environmental factors on fumonisin production by Aspergillus niger and Aspergillus welwitschiae at varying temperatures and water activity.

Numerous studies have been undertaken from both natural occurrences and experimental settings on factors affecting fumonisin production. These studies have highlighted the importance of drought conditions in the occurrence of fumonisins. In the planting season of 1993, there were variations in the occurrence of measured fumonisins in corn crops in Ontario, Canada, due to changing rainfall patterns. Areas that received high rainfall (95% of normal value) had low incidences of fumonisin contamination with an average FB1 concentration of 0.4 µg/g, while areas with lower rainfall (49% of normal value) had high incidences of fumonisin contamination with an average FB1 concentration of 1.4 µg/g (Miller *et al*., 2014). This indicates that drought conditions favor the occurrence of fumonisins in crops. Another study conducted by Kos *et al*. (2016) reported by Rheeder *et al*. (2016) in Serbia related the increase in fusarium toxins contamination of maize in the farming season of 2012 to drought conditions and the associated increase in temperature. This co-relates with previous data obtained from samples collected in South Africa.

Furthermore, increased global temperatures also indirectly contribute to fumonisin contamination by exacerbating insect damage to crops, such as kernel damage in corn, which facilitates fungal and increased production/accumulation of mycotoxins (Stathas *et al*., 2023). This increase in kernel damage may be because insects are ectotherms and become more active as ambient temperatures rise, leading to an increase in their metabolic and developmental rates and activity patterns. Associated with increased global temperature are other changes in climatic conditions such as variations in rainfall patterns, humidity, drought, atmospheric carbon dioxide, etc. These variations in climatic conditions impact agricultural production and further predispose crops to fungal infection and mycotoxin contamination (Wu *et al*., 2011).

Tolerable daily intake

In general The World Health Organization's International Programme on Chemical Safety (IPCS) and also the Scientific Committee on Food (SCF) of the European Commission have evaluated the risks related to FB1, and they have concluded that the tolerable daily intake for FB1, FB2, and FB3, alone or in combination, is 2 µg/kg body weight per day. The daily intake of fumonisins in different food commodities among various countries has been documented (Farhadi *et al*., 2019).

The European Food Standards Agency (EFSA) set up the EFSA panel on contaminants in the food chain (CONTAM), which established health-based guidance values for fumonisins and their modified forms (Knutsen *et al*., 2018). In the European diet, for instance, the total daily intake of FB1 was estimated to be 1.4 µg/kg of body weight per week by the European Mycotoxin Awareness Network (EMAN, 2000). EMAN proposed a "provisional-maximum-tolerable-daily-intake" (PMTDI) of 2 µg/kg body weight/day for FB1. This was obtained by dividing the "no-observable–effect-level (NOEL mg/kg of body weight/day) by a safety factor of 500. This level was in line with the value set by the World Health Organization's International Programme on Chemical Safety (IPCS) and the Scientific Committee on Food (SCF) of the European Commission of 2 µg/kg body weight/day for FB1 (WHO, 2018).

In Switzerland, the official tolerance value is 1 mg/kg FB1+FB2 in dry corn products (WHO, 2002). Another study by the EFSA Panel on Contaminants in the Food Chain (CONTAM) determined a tolerated daily intake (TDI) of 1.0 µg/kg body weight (bw) per day for FB1 based on a benchmark dose lower confidence limit (BMDL10) of 0.1 mg/kg bw per day and an uncertainty factor (UF) of 100 for intra and interspecies variability (Knutsen *et al*., 2018). However the tolerable daily intake of fumonisin in Nigeria is guided by international standards.

Effects of Fumonisins on Human health

Exposure to fumonisins poses a great risk to human health because of their interference with basic cellular processes, leading to a wide range of adverse effects;

Esophageal cancer

Exposure to aflatoxins from contaminated food, have been associated with the development of Esophageal Squamous Cell Carcinoma (ESCC) in Africa (Brown *et al.*, 2020). Epidemiologic studies across multiple regions have suggested a correlation between the occurrence of Fusarium verticillioides–produced fumonisins and the incidence of human esophageal cancer, particularly in areas where maize and other cereals form the main dietary staple. In high-risk regions such as eastern South Africa's former Transkei, China's Huaian area, and Golestan Province in Iran, staple foods often contain high levels of fumonisin B1 (FB1), and populations exposed to these diets exhibit disproportionately high rates of ESCC. Biomarker studies have shown that individuals with ESCC in these regions tend to have higher levels of fumonisin biomarkers compared to controls, and dietary fumonisin exposure is significantly elevated in high-risk areas, supporting the hypothesis that fumonisins may contribute to esophageal carcinogenesis in conjunction with other factors (Come *et al*., 2019; Xue *et al.*, 2019).

The classic observation linking F. verticillioides contamination in Transkei maize with high esophageal cancer rates originally dates from older ecological and descriptive studies, but recent work by Mulisa *et al*. (2025) emphasizes ongoing high fumonisin exposure in high-risk populations rather than direct proof of causality. Contemporary reviews note this correlation and continue to investigate it with improved biomarker methods

Fumonisins-induced modifications to the membrane lipid profile

Membrane fatty acids have been shown to provide a general image of cell metabolism and disease progression, thus establishing them as reliable biomarkers. In this respect, lipidic biomarkers vary across pathophysiological conditions, depending on the animal, investigated tissue, and severity of the condition. In the case of Fumonisins, the fatty acid composition of membrane lipids provides data that can potentially be incorporated into understanding the toxicity mechanism and cancer induction by fumonisins. The identification of membrane lipid modifications provides in-depth insight into cellular metabolic conditions (Omeralfaroug and Zabo, 2024).

Fumonisins mycotoxins are well known to disrupt membrane lipid structures and integrity, implying that numerous cellular metabolic pathways are likely to be modified, altering signal transduction, ion transport, and cell adhesion, as well as interactions between cells and their environment, impacting tissue and organ function; however, the magnitude of the association between altered lipids and remodulated signals is still uncertain and warrants further investigation. Clearly recognizing the lipids targeted by Fumonisins would provide further biomarkers for exposure assessment and potential therapeutic approaches to mitigate or diminish the adverse effects and consequences of fumonisins, especially in potential cancer areas (Omeralfaroug and Zabo, 2024).

Disruption of sphingolipid metabolism

Fumonisins such as FB1 are structural analogues of sphingoid bases including sphinganine and sphingosine, enabling them to competitively inhibit ceramide synthase (CerS), a key enzyme in sphingolipid biosynthesis.

This inhibition disrupts the normal synthesis of complex sphingolipids, leading to elevated cellular levels of sphinganine, sphingosine, and their phosphorylated metabolites and depletion of ceramides and other complex sphingolipid species. Such alterations are reflected in changes to biomarker ratios such as sphinganine/sphingosine (Sa/So) and other sphingolipid ratios in tissues exposed to FB1 (Obafemi *et al*., 2025).

Recent mechanistic studies provide structural insights into the inhibitory interaction between FB1 and ceramide synthase, showing that FB1 and its derivatives can interfere with the catalytic activity of CerS, thereby disrupting de novo sphingolipid biosynthesis (Zhang *et al.*, 2024). Experimental animal studies have also demonstrated that exposure to fumonisins at regulatory levels leads to significant sphingolipid perturbations in liver, kidney, and lung tissues, including altered Sa:So and related sphingolipid ratios, underscoring the utility of these metabolites as biomarkers of exposure and effect (Lassallette *et al*., 2025).

The disruption of sphingolipid metabolism has downstream consequences for cell signaling, apoptosis, membrane integrity, and cellular homeostasis, which likely contribute to the diverse toxicological effects of fumonisins observed in both animal and cellular models (Abia *et al*., 2025).

Fumonisins and Neural Tube Defect (NTD)

NTD are common congenital malformations that occur when the embryonic neural tube, which ultimately forms the brain and spinal cord, fails to properly close during the first few weeks of development (CDC, 2025). NTD are among the most common of all human birth defects, yet their etiologic basis and embryology remain poorly understood. Empirical risk figures, along with numerous clinical studies, indicate that NTD are of a multifactorial origin, having both genetic and environmental components (Rheeder *et al.*, 1992).

In the early 1990s, following investigation of an NTD cluster that occurred among Mexican-American women in Cameron County, Texas, Kate Hendricks from the Texas Department of Health began questioning researchers about the possibility that exposure to fumonisin-contaminated food (tortillas) during gestation may have contributed to these birth defects (Rheeder *et al.*, 1992).

Fumonisin B1 has been implicated in neural tube defects in babies. Fumonisins reduce uptake of folates by disrupting sphingolipid metabolism and consequently folate transport across cell membranes. The correlation between fumonisin uptake in diets and incidences of neural tube defects in some populations has been demonstrated in various epidemiological survey on incidences of neural tube defects for instance along the Texas-Mexico border in the United States, correlating this to fumonisin exposure in the mothers using maternal serum measurements of the sphinganine-sphingosine (Sa/So) ratio. The study found a dose-response relationship between maternal fumonisin exposure and increased risk of NTDs in babies, highlighting that fumonisins are potential risk factors for NTD in populations in which maize is a staple diet (Anumudu *et al.*, 2025).

Acute mycotoxicosis

Fumonisins have been implicated in incidences of acute human mycotoxicosis characterized by abdominal pain, diarrhea, and borborygmi (Anumudu *et al.*, 2025). Human consumption of rain-damaged moldy sorghum and corn in 27 villages on the Deccan plateau in India in 1995 resulted in a food-borne disease outbreak characterized by abdominal pain, and borborygmi. In an epidemiological survey, the mycotoxicosis was connected to consumption of unleavened bread. The corn and sorghum samples from affected households were observed to be contaminated by Fusarium and Aspergillus fungi and contained high amounts of FB1 compared with samples collected from unaffected households (Bhat *et al.*, 1997).

Other effects of fumonisin B1(FB1) Toxicity

Effect of FB1 on Liver

FB1 also inhibits the value addition and prolonged cell cycle of the human normal hepatocyte line HL-7702, which may be related to FB1-induced changes in the expression levels of cyclins E and P21. In a human hepatocellular carcinoma cell line (HepG2), the expression levels of miR-27b and CYP1B1 protein were

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significantly negatively correlated, which may be a mode of liver tumor transformation. However, after studying FB1 levels in 271 liver cancer patients and 280 normal subjects, some scholars concluded that there is no direct association between FB1 intake and liver cancer (Chen *et al.,* 2021)

FB1 mediated oxidative stress

Under normal physiological conditions, oxidants and antioxidants are maintained in dynamic equilibrium. However, exposure to fumonisin B1 (FB1) disrupts this balance, leading to excessive production of reactive oxygen species (ROS) that overwhelm cellular antioxidant defenses. FB1 has been shown to increase lipid peroxidation, deplete glutathione (GSH), and reduce activities of key antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione-peroxidase (GSH-Px). These oxidative changes occur in various cell types and tissues and help explain part of the cytotoxicity and tissue damage associated with FB1 exposure (Qu *et al.*, 2022).

In several in vitro models, fumonisin B1 (FB1) has been shown to induce oxidative stress in a cell-type- and dose-dependent manner. Recent studies demonstrate that exposure of neural-derived cells, including glioblastoma, neuroblastoma, and hypothalamic cell lines, to micromolar concentrations of FB1 results in a significant increase in intracellular reactive oxygen species (ROS) following prolonged exposure. In contrast, some non-neural cell types, such as human fibroblasts and primary astrocyte cultures, appear to be more resistant to FB1-induced ROS generation, highlighting differential cellular susceptibility to oxidative damage (Riley *et al*., 2020).

Evidence of FB1-induced lipid peroxidation has been reported across multiple experimental systems, including human intestinal epithelial cells, neural cell lines, embryonic fibroblasts, and endothelial cells. These effects are characterized by elevated malondialdehyde (MDA) levels, increased membrane lipid oxidation, and disruption of mitochondrial integrity. The concentrations and exposure durations required to induce lipid peroxidation vary considerably, ranging from low micromolar doses in sensitive cell lines to higher concentrations following prolonged exposure in neural cells (Chen *et al.*, 2021; Anumudu *et al.*, 2024). In vivo studies further support these findings, showing that chronic dietary exposure to FB1 leads to increased lipid peroxidation and oxidative damage in hepatic and renal tissues. FB1 exposure has also been associated with depletion of intracellular antioxidant defenses, particularly reduced glutathione (GSH), in the liver, spleen, and neural tissues. Collectively, these data indicate that oxidative stress and lipid peroxidation are central mechanisms contributing to FB1-induced cytotoxicity and tissue injury (Anumudu *et al.*, 2024).

Effect of FB1 on Human Heart

FB1 also reduces cardiac contractility in humans, triggering idiopathic congestive cardiopathy (ICC). This is caused by massive blood flow into the heart and weakness of the heart (Chen *et al.,* 2021).

FB1-induced toxicity to the immune system

According to what is known, FB1 can have significant immunological effects. Numerous papers have detailed the impact of FB1 on the cytokine profile of many organs and cell types. Following exposure to FB1, elevated expression of TNF-α and interleukin-1β (IL-1β) has been observed in mouse liver and kidney as well as in primary cultures of hepatic cells. It has been demonstrated that FB1 increases the production of IFN-γ and the associated chemokine CXCL in human dendritic cells (Stockmann *et al.*, 2008).

However, FB1 may decrease the lipopolysaccharide (LPS)-induced brain and liver expression of IFN-γ and IL-1β, as well as the LPS-induced expression of IL-6, IL-1β, and the chemokines CCL3 and CCL5 in human dendritic cells. Elevated TNF-α expression, after activation of the cells with LPS in vitro, has also been observed in peritoneal macrophages of mice and in mouse macrophage cell line cells treated with FB1. (Osuchowski and Sharma, 2008).

FB1-induced apoptosis

Cells undergoing programmed cell death (apoptosis) exhibit some obvious morphological characteristics. Apoptosis induced by FB1 (shown mainly as caspase-3 activation or DNA fragmentation) has been observed

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in a number of cell types and tissues, including human keratinocytes, liver and kidney of rats, human colonic cell line cells, human fibro blasts, human proximal tubule-derived cells, human neuroblastoma and glioblastoma cells and mouse hypothalamic cells, and rat glioblastoma cells. However, neoplastic monkey kidney cells or murine and human leukemia cells do not undergo apoptosis when treated with FB1. It has also been shown that the induction of apoptosis by FB1 can be mediated by the accumulation of free sphingoid bases, but inhibition of ceramide synthase alone is, however, not responsible for the induction of apoptosis (Seefelder *et al.*, 2003).

Some studies have focused on the mechanisms whereby FB1 may induce apoptosis. Studies with human neonatal kidney cells, human lung fibroblasts, green monkey kidney fibroblasts, and human and murine glial and neural cell lines have indicated that FB1-induced apoptosis does not involve p53 or Bcl-2 family (Stockmann, 2004). It has also shows that abaculovirus gene, inhibitor of apoptosis (CpIAP), protects cells from apoptosis following FB1 treatment. This gene is known to block apoptosis induced via the tumor necrosis factor (TNF) pathway. In addition, it was shown that caspase-8 was leaved, and the TNF receptor-associated protein-2 was induced, which further indicates that FB1 activates apoptosis via binding to TNF receptors. Moreover, increased expression of caspase-8 and the production of proinflammatory cytokines like TNF-α seem to be important mediators of FB1-induced apoptosis in murine liver or primary hepatocytes. (He *et al.*, 2002).

Effect of FB1 on human intestine

FB1 has some toxic effects on human intestinal cell lines, but the toxicity is low. In experiments using a human gastric epithelial cell line (GES-1) as an in vitro model, FB1 was found to significantly reduce cell viability, increase membrane leakage, cell death, and induce endoplasmic reticulum stress, resulting in gastrointestinal injury. Recent studies have shown that the endoplasmic reticulum stress-associated PERK-CHOP signaling pathway plays a key role in FB1 damage to GES-1 (Chen *et al.*, 2021).

FB1-induced autophagy

Autophagy involves a process of degradation of cytoplasmic content which helps regulate cell death (or survival) and plays a role in many physiological processes. In addition, excessive autophagy can induce cytotoxicity. It has been reported that FB1 can induce cytotoxicity via autophagy. (Tian *et al.*, 2022).

Methods of Detection

Effective detection methods for fumonisins are very important in light of the health risks posed by these toxins. Current detection methods are diverse and include various technologies and approaches that enable the identification and quantification of levels of fumonisin in different samples. (Gazzotti *et al.*, 2011)

mmunological Methods

For the quick detection of fumonisins in foods and feed materials, a number of immunological techniques have been developed, including flow-through membrane-based immunoassays and Enzyme Linked Immuno-Sorbent Assay (ELISA). They rely on a particular monoclonal or polyclonal antibody's ability to recognize the fumonisins' three-dimensional structure (Berthiller *et al.*, 2018).

ELISAs are simple, inexpensive, and rapid-to-use methodologies, easily adapted for screening purposes, and can be qualitative or quantitative. Direct competitive ELISAs are commonly employed in the analysis of mycotoxins, including fumonisins (Ling *et al.*, 2014). Various studies have utilized the principle of ELISA for the determination of fumonisins and other mycotoxins in different food matrices, and a wide range of ELISA kits are commercially available (Anumudu *et al.*, 2025).

Lateral flow immunoassays (LFID) are utilized for the rapid onsite determination of fumonisins. They are based on the migration of the sample along a membrane strip as a result of capillary action and subsequent reaction between immobilized immunoreagents and the mycotoxins. They can be qualitative or used semi-quantitatively and require minimal sample extraction steps. There are widely available commercial products based on LFIDs, and numerous studies have been conducted on the use of such devices in Fumonisin analysis to improve the reliable detection of the mycotoxins in different food and feed matrixes (Anumudu *et al.*, 2025).

Chromatographic Methods and Mass Spectrometry

Thin-layer chromatography (TLC), gas chromatography coupled with mass spectrometry (GC/MS), liquid chromatography-high resolution mass spectrometry (LC-HRMS), and liquid chromatography with mass spectrometric or fluorescence detection (LC/MS) (LC/FLD) are among the chromatographic techniques for fumonisin detection that are typically coupled to a detector, primarily mass spectrometry (MS) (Medina *et al*., 2021). When compared to the modern LC, other chromatographic methods like TLC and GC still have several drawbacks despite their ongoing use. While TLC is a quick, simple, and affordable method for semi-quantitative mycotoxin testing and screening, its resolution is low, and its detection capabilities are limited to nonspecific methods (Medina *et al.*, 2021).

FB1 is characterized by the presence of a sphingoid backbone and can inhibit ceramide synthase through the modulation of two precursors in sphingolipid production: sphinganine (Sa) and sphingosine (So), resulting in an increase in the ratio of Sa/So (Westhuizen *et al*., 2001). This mechanism, which is the causal pathway of mycotoxins toxicity, is exploited as a biomarker of exposure (Turner and Synder, 2021). Thus, sphingoid bases, specifically sphinganine (Sa), sphingosine (So), and their ratio (Sa/So), have become a commonly used biomarker for fumonisin exposure in human serum. The Sa/So ratio is a preferred biomarker that may be traced in urine because of the substantial variability of both Sa and So. (Wangia-Dixon, and Nishimwe, 2020).

While newer colorimetric methods using immunologic and molecular approaches are being developed, including those using dyes, enzymes, aptamers, and even nanomaterials, chromatographic methods coupled with mass spectrometry or other detectors are considered reliable methods and most frequently used analytical method, especially in detailed quantification of fumonisins. (Gazzotti *et al.*, 2011).

Reducing risk of Fumonisin Toxicosis

It is important to note that mould that produces mycotoxins can grow on a variety of different crops and foodstuff and can penetrate deep into food and do not just grow on the surface. Mould usually does not grow in properly dried and stored foods, so efficient drying of commodities and maintenance of the dry state, or proper storage, is an effective measure against mould growth and the production of mycotoxins such as fumonisins. (WHO, 2018).

However according to WHO guidelines, to minimize the health risk from fumonisins, people are advised to:

1. Inspect whole grains (especially corn, sorghum, wheat, rice), dried figs and nuts such as peanuts, pistachio, almond, walnut, coconut, Brazil nuts and hazelnuts which are all regularly contaminated with mycotoxins for evidence of mould, and discard any that look mouldy, discoloured, or shrivelled

2. Avoid damage to grains before and during drying, and in storage, as damaged grain is more prone to invasion of moulds and therefore mycotoxin contamination

3. Buy grains and nuts as fresh as possible

4. Make sure that foods are stored properly – kept free of insects, dry, and not too warm

5. Not keep foods for extended periods of time before being used; and ensure a diverse diet – this not only helps to reduce mycotoxins exposure, but also improves nutrition.

Treatment of Foods contaminated with Fumonisin

Food processing treatments such as roasting, frying, cooking, or high-temperature extrusion of corn may result in a reduction of fumonisin concentrations in food products (Humpf and Voss, 2004). Due to their relative heat stability, fumonisins are only significantly eliminated during operations that involve temperatures above 150˚C (Milan and Maleki, 2014).

In their study, Jackson *et al.* (2011) reported by Anumudu *et al.* (2025), investigated the effect of extrusion process on fumonisins using a twin-screw press and observed a 64–72% reduction of fumonisins in extruded corn without glucose and an 89–94% reduction in corn extruded with glucose. In a feeding trial conducted by Voss *et al.* (2014l), it was found that nixtamization (alkali cooking) of corn was effective in reducing fumonisin concentrations in contaminated corn with reduced incidences of apoptotic kidney lesions in rats fed the nixtamalized corn version in comparison to those fed corn prepared by conventional means. This shows that nixtamalization is an effective method for the reduction of fumonisins and their toxicity in contaminated corn.

Similarly, the study by Xing *et al.* (2014) reported by Anumudu *et al*. (2025), found that cinnamon oil at a concentration of 280 µg/mL, a temperature of 30°C, and an incubation time of 120 h significantly led to a 94.06% reduction (from 15.03 to 0.89 µg/mL) in FB1 contamination in maize grains. A more recent study by Schambri *et al*. (2021) demonstrated that the initial fumonisin and deoxynivalenol contamination of 1351 µg/kg in maize kernels was reduced by 91% on average after undergoing three popping methods: hot air, hot oil, and microwaves. The hot oil technique appeared to be more efficient, reducing the fumonisin and deoxynivalenol levels by 98% and 58%, respectively.

CONCLUSION

Fumonisins are pervasive food contaminants with significant implications for human health. Their toxicity is mediated primarily through disruption of sphingolipid metabolism and membrane lipid homeostasis, leading to diverse pathological outcomes. Although considerable progress has been made in understanding fumonisin toxicity and improving detection methods, important gaps remain regarding long-term health effects and population-specific risks. Continued research, surveillance, and implementation of effective mitigation strategies are essential to protect public health.

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