Chapter

Growth of Fungal Cells and the Production of Mycotoxins

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Abstract

Some filamentous fungi are able to grow in food and produce toxic metabolites. It occurs mainly in grains, cereals, oilseeds and some by-products. The growth of fungi in a particular food is governed largely by a series of physical and chemical parameters. The production of toxic metabolites is not confined to a single group of molds irrespective of whether they are grouped according to structure, ecology, or phylogenetic relationships. Mycotoxins can be carcinogenic and cause several harmful effects to both human and animal organisms, in addition to generating large economic losses. The major mycotoxins found in food are the aflatoxins, fumonisins, ochratoxins, patulin, zearalenone, and trichothecenes, generally stable at high temperatures and long storage periods. Considering the difficult prevention and control, international organizations for food safety establish safe levels of these toxins in food destined for both human and animal consumption. Good agricultural practices and control of temperature and moisture during storage are factors which contribute significantly to inhibit the production of mycotoxins. The use of some fungistatic products, such as essential oils and antioxidants, as well as physical, mechanical, chemical, or thermal processing, represents important methods to have the concentration of mycotoxins reduced in food.

Keywords: aflatoxins, ochratoxins, patulin, fumonisins, zearalenone

1. Introduction

Microorganisms constitute the main cause of deterioration and losses in food. Fungi can be mono- or multiple-cell organisms, mostly aerobic, which survive within a wide range of moisture, temperature, and pH. They inhabit nature freely and feed on the absorption of organic matter.

Their presence in food can be derived from the field, such as parasites, plant pathogen, and even coming from the soil or equipment used in the management of culture crops. In addition, they appear as storage microbiota and develop during the entire storage process, which may lead to great physical-chemical and sensory losses in food products, in addition to the production of mycotoxins.

Mycotoxins are substances secreted by the secondary metabolism of filamentous fungi, which are produced by certain fungus lineages and in particularly favorable conditions. A few hundreds different mycotoxins are known, some characterized by their antibiotic potential and others extremely toxic to men and animals. This chapter will present the fungi growth conditions to produce mycotoxins, the major mycotoxins occurring in food, levels of toxicity, favorable conditions to excretion, and control measures regarding their production.

2. Fungi

Fungi are able to grow in practically all ecological niches; however, they can be found prevailing particularly in dead organic matters present in the soil. They include eukaryotic organisms commonly known as yeasts, which normally grow in the form of single cells, and molds, which grow by forming ramified chains called hyphae. Even though most fungi are harmless to human beings, the exposition to specific lineages and their metabolites may result in some clinical manifestations in men and other animals.

a. Infections or diseases derived from the invasion of living tissue. The growth of a fungus on the top of or inside a body is named mycosis. Mycoses can have varied severity, encompassing from relatively benign and superficial infections to severe diseases that threaten life.

b. Hypersensitivity reactions. Some fungi promote an immune response which can result in allergic reactions after exposition to a specific fungus antigen. Exposition to fungi, either by developing in a host or in the environment, may cause the development of allergic symptoms in the case of a re-exposition. For example, *Aspergillus s*pp., a common saprophyte, often found in nature as a filamentous fungus from leaves, corresponds to a potent, common allergen, which often triggers asthma and other hypersensitivity reactions.

c. Mycotoxicosis. It is an intoxication resulting from ingestion of food or feed containing toxic metabolites, that is, mycotoxins [1].

The major toxigenic strains of interest for the public health belong to the genera *Aspergillus* spp., *Penicillium* spp., and *Fusarium* spp. It is important to highlight that not all fungi produce mycotoxins as well as that a single fungus species can produce many secondary toxic compounds. The presence of a mycotoxin in food is necessarily conditioned to fungus development, but it does not mean that a product without any fungi could not contain mycotoxins [2]. A cereal stored under poor conditions of temperature and moisture provides a favorable medium for fungus development

and the production of mycotoxins. Once a mycotoxin is produced, even upon the destruction of the fungus biomass, through a drying process, for example, the metabolites excreted remain in the product.

As heterotrophs, fungi require organic compounds for both synthesis of biomass (anabolic metabolism) and production of energy to drive these reactions (catabolic metabolism). These aspects are referred to as primary metabolism. The secondary metabolisms are distinct from primary metabolism in so far as they occur optimally after a phase of a balanced growth and are often, but not always, associated with morphogenetic changes such as sporulation; the production of particular secondary metabolites is usually restricted to a small number of species and may be species, or even strain, specific; it has not generally been possible to rationalize the biological function of secondary metabolites, although some are very active against microorganisms (antibiotics), plants (phytotoxins), or animals (mycotoxins). Although secondary metabolites in general, and mycotoxins specifically, do not form a neat and a recognizable group of organic structures, they can be classified in terms of the biosynthetic pathways leading to their production. This is so because the processes of primary and secondary metabolism are linked by a relatively small number of simple intermediates such as acetyl coenzyme A, mevalonic acid, and amino acids [3–5].

3. Fungal growth in food

The growth of fungi in a particular food is governed largely by a series of physical and chemical parameters, and definition of these can assist greatly in assessing the food's stability. The factors which govern spoilage are physical and chemical, and there are eight principal factors: water activity; hydrogen ion concentration; temperature, of both processing and storage; gas tension, specifically of oxygen and carbon dioxide; consistency; nutrient status; specific solute effects; and preservatives [6].

In general, fungal deterioration stands out under conditions in which bacterial deterioration is controlled, either by low water activity (aw), pH, temperature, and/or the presence of inhibitory agents. Raw material quality and contamination of the production environment will directly interfere with the initial contamination of the products. The processing and storage parameters will influence on time to the appearance of visible fungal colonies and, therefore, the shelf life of a food product [6, 7].

The deterioration of food by filamentous fungi starts with contamination of the product by fungal spores originating from the environment. When intrinsic parameters, such as water activity (aw) and pH, as well as temperature, are favorable, the spores will germinate and form a visible mycelium, deteriorating the product [8, 9].

Temperature and aw are recognized as the most important parameters for determining fungi cell growth, but pH also influences that development. The external pH value influences not only fungal growth rate but also metabolism. *Aspergillus flavus* isolates produce more aflatoxins when the external pH becomes increasingly acidic. In the case of the cereal pathogen *Fusarium graminearum*, trichothecene production is induced only under acidic pH conditions [10].

The moisture content of grains and other dried foods is such that there is seldom any problem with the growth of bacteria and yeasts, but there are frequent problems with the growth of molds (fungi). Unless the aw is reduced to below approximately 0.7, molds will grow on any food, and as the relative humidity in the humid tropics is generally more than 70%, almost all dry foods will become moldy when stored in the humid tropics unless the moisture content is reduced to an aw of less than 0.70, followed by storage that will protect that food from absorbing moisture from the high-humidity environment [11–14].

4. Mycotoxins

Mycotoxins have been known for a very long time, but they only became more intensively studied after an incident occurred in 1960 in England, involving the death of 100,000 birds fed on feed contaminated with fungus *Aspergillus flavus*.

Mycotoxins are produced mainly by mycelial structures present in filamentous fungi. Even though their function for produced lineages is yet to be clarified, mycotoxins are secondary metabolites that apparently do not present a biochemical meaning to fungus growth and development [7, 15].

According to the Food and Agriculture Organization (FAO), it is estimated that the contamination of food products by fungi and their toxic metabolites generates qualitative and quantitative losses for around 25% of the agricultural food production worldwide, occurring majorly in regions of tropical and subtropical climate, where higher temperature and moisture favor microbial proliferation [16, 17].

Primary metabolites of fungi, such as of other organisms, are essential to growth, while secondary ones are formed during the final exponential growth phase and have not a clear significance to the growth or metabolism of the organism [1–3].

In general, these metabolites appear to be formed whenever large amounts of primary metabolites precursors, such as amino acids, acetate, and pyruvate, among others, are accumulated. The synthesis of mycotoxins represents a way for fungi to reduce the amount of precursors, which are not required to metabolism [1–3].

They are constituted by a large variety of chemical assembles, which provides them with several biological activities, classified according to the toxicity level exerted on human and animal organisms [2] possibly with carcinogenic, mutagenic, teratogenic, cytotoxic, neurotoxic, nephrotoxic, immunosuppressant, and estrogen effects. However, its toxicity largely depends on the amount ingested, time of exposition, and possible synergy with the ingestion of many different mycotoxins simultaneously, in addition to individual physiological conditions [4].

Mycotoxin ingestion can produce both acute and chronic toxicities. Acute is characterized by a rapid onset and an obvious toxic response including rapid death. Chronic is resulting from low-dose exposure to mycotoxins over a long period of time, with toxic responses including cancers such as hepatocellular carcinoma [18, 19].

The International Agency for Research on Cancer (IARC) in Lyon (France)—through its IARC Monographs program—has performed the carcinogenic hazard assessment of some mycotoxins in humans, on the basis of epidemiological data, studies of cancer in experimental animals, and mechanistic studies. There are five groups classified according to the scientific evidence for their carcinogenicity: Group 1, carcinogenic to humans; Group 2A, probably carcinogenic to humans; Group 2B, possibly carcinogenic to humans; Group 3, not classifiable as to its carcinogenicity to humans; and Group 4, probably not carcinogenic to humans. Carcinogenic effects and related mechanisms of some mycotoxins (e.g., aflatoxins) are well-known. However, for some other important mycotoxins (e.g., OTA, FUM B1, and FUM B2), there is a need for continued research on understanding these mechanisms [20–24].

4.1 Aflatoxins

Many types of aflatoxin (14 or more) occur in nature, but only four of them are particularly dangerous to humans and animals. Aflatoxins are mainly produced by species of *Aspergillus flavus* and *Aspergillus parasiticus* and classified in aflatoxins B1 and B2 and G1 and G2. Their name derives from the fluorescence emitted after absorption of ultraviolet light at 365 nm (B, blue; and G, green). They are characteristically heat-resistant and bear the process of sterilization with a structure

remaining unaltered for long periods of storage. Their chemical structure allows good solubility in organic solvents and are insoluble in water proving sensitive in alkaline medium, which decreased their toxicity [22]. The group of aflatoxins is considered by the IARC as belonging to Group 1—cancer-causing substances to men [21].

Mycotoxin B1 is pointed out as the most toxic in the group and can generate metabolites in the organism of mammals (M1). Aflatoxin M1, generated by B1 metabolism, is transported to milk at a proportion of 1% of the total ingested and can also be found in animal tissues in which there is a high exposition to this toxin. Subsequently, humans can ingest this aflatoxin through breast milk, milk, and milk-derived products, especially in areas where grains and cereals of poor quality are used to feed animals [17–23].

Birds are the most sensitive animals to aflatoxins, and whenever ingested in contaminated feed, they present fast absorption through the gastrointestinal tract. The effects include damages to the liver, harm to the productivity and reproductive efficiency, decreased production of eggs, lower quality of egg shell, lower quality of carcass, and increased susceptibility to diseases. Pigs are, somehow, less sensitive than birds. Aflatoxin is also hepatotoxic to these animals, and its chronic effects are largely attributed to damage to the liver. In cattle, the primary symptom is reduction in weight gain. In addition, milk production is reduced [25–27].

Aflatoxins occur mainly in products such as peanut and nuts, maize, and cottonseed, among others, which is associated with pre-crop contamination. Cultures such as coffee, rice, and spices can also be contaminated by these toxins post-crop [26].

Many countries introduced legislation specific to mycotoxins. Most of these legislation rules are regarding aflatoxins, alkaloids of ergot, deoxynivalenol, and ochratoxins. Even though legal measures are yet to be uniformed at a worldwide level, the Codex Alimentarius Commission is gathering efforts to establish international guidelines regarding levels of mycotoxins. For aflatoxins, the Food and Drug Administration established the maximum limit of 20 ppb to maize, peanut, cotton bran, and other food and ingredients for animals [27–31].

4.2 Ochratoxin A

The major toxins produced by *Penicillium verrucosum*, *Aspergillus ochraceus*, and *Aspergillus carbonarius* are ochratoxin A, ochratoxin B, and ochratoxin C. Among these toxins, ochratoxin A is considered the most toxic and, according to the IARC, a possible cancer-causing substance to humans (category 2B). Recent researches conducted over the past 6 years related to ochratoxin toxicity encompassed the identification of factors involved in carcinogenesis and provided strong evidence to a reclassification of the Group 2B into the Group 2A (probably carcinogenic to human beings) [1, 4, 20].

Among the species of *Penicillium*, *P. verrucosum* is the major source of ochratoxin A and the most common species in countries of temperate, cold climate, while *A. ochraceus*, *A. carbonarius*, and other species from the Group A niger are the most common in tropical, hot climates. Another species of *Penicillium* produced from ochratoxin A is *P. nordicum*. *P. verrucosum* is especially associated with stored cereals, that is, post-crop fungi. This mycotoxin is often found in animal feed and food as wheat, rye, coffee, nuts, and, at a lower degree, grapes, raisins, wine, or products derived from pork. There have been reports of this mycotoxin detected in blood and milk breast of individuals exposed to its ingestion [1–4]. The levels can accumulate in the tissues of the body and fluids of human beings or animals who consume contaminated food. Evidence shows that ochratoxin A is slowly eliminated from the body [17, 25].

The structure of these toxins is derived from L-phenylalanine, which makes it a potent inhibitor of the enzyme phenylalanine-RNAt synthase, responsible for the synthesis of proteins of high turnover rich in phenylalanine—a functional role for kidney homeostasis. In addition, it interferes in the lipid peroxidation causing damages to the DNA and oxidative stress. Therefore, it is suspected that ochratoxin A is one of the cancer-causing agents in the urinary tract as well as related to the damages to kidneys occurred in Eastern Europe. Researches indicate that practically all Europeans have some ochratoxin concentration in their blood. Human exposition to ochratoxin occurs primarily from brown bread. In some parts of Europe, the most significant exposition derives from the consumption of animal products, especially those formulated based on pig blood [32, 33].

Considering the toxic effects of ochratoxins, a tolerable weekly intake (TWI) of 120 ng/kg of body weight (pv) was established by the European Food Safety Authority (EFSA). The meeting of the Committee of Specialists on Agricultural Contaminants in food (European Commission, DG Health, and food safety) has been considering establishing limits to herbal teas, infusions, and baking [32]. Even though ochratoxins B and C are hepatotoxic, immunotoxic, teratogenic, and genotoxic, maximum tolerable limits are yet to be established regarding these toxins [34, 35].

4.3 Patulin

Patulin (polyketide lactone 4-hydroxy-4H-furo (3.2c) pyran-2 (6H)-one) is a secondary metabolite produced by several species of *Penicillium*, *Aspergillus*, and *Byssochlamys* in conditions of high activity of water (0.95–0.99) and temperature of 0–31°C. Food which are more susceptible to contamination by patulin in human diet are apples and by-products (puree and juices). Even though contamination with patulin is mainly associated with areas of contaminated tissue, it can penetrate around 1 cm in healthy regions of the fruit [1].

Patulin has been reported as mutagenic, neurotoxic, immunotoxic, and genotoxic and to cause gastrointestinal damages in rodents. There is also some concern that similar effects may occur in humans through a long-term consumption of food and beverage contaminated with this mycotoxin. The IARC classified patulin as Category 3, non-classifiable regarding its carcinogenicity to human beings. Because of its toxicity, the Joint Food and Agriculture Organization/World Health Organization Expert Committee on Food Additives (JECFA) established a maximum tolerable limit for daily intake (PMTDI) for patulin of 0.4 μg/kg of body weight [34]. The Codex Alimentarius established a maximum level for patulin of 50 μg/kg in apple juice, and the European Union (EU) adopted a maximum level of 50 μg/kg 1 for juices, beverages, and fermented milk products containing apple juice, 25 μg/kg in solid products containing apple, and 10 μg/kg in apple-based products as well as baby food. Although some limits have been established, some countries, such as Pakistan, do not have any specific legislation for this toxin [36].

4.4 Trichothecenes

Trichothecenes are a group of secondary metabolites produced by fungi belonging to the genus *Stachybotrys* and mainly *Fusarium*, in which *F. graminearum* and *F. culmorum* are the most important. *Fusarium graminearum* grows greatly at a temperature of 25°C and activity of water above 0.88, while *F. culmorum* grows well at 21°C and activity of water above 0.87 [1].

The group of trichothecenes is composed of over 200 mycotoxins and carries this name because of their chemical structure constituted of a ring with tetracyclic skeleton 12,13-epoxitrichothecenes. They also present varied ligand assembles, which

provides toxicity. These toxins are classified in type A, in which toxins T-2, HT-2, 15-monoacetoxyscirpenol (15-MAS), and diacetoxyscirpenol (DAS) are found, and type B, in which deoxynivalenol (DON) occurs. Deoxynivalenol is an epoxy-sesquiterpenoid which occurs naturally combined with 3-acetyl-deoxynivalenol (3AcDON) and 15-acetyl-deoxynivalenol (15AcDON), which increases DON toxicity. According to the IARC, DON is classified in level 3, that is, it does not represent a risk of cancer induction, although co-occurrence with aflatoxin may increase aflatoxin carcinogenicity. DON can also coexist combined with zearalenone [1, 2, 37].

Despite the existence of a relatively large amount of different trichothecenes, their natural occurrence in food and feed is mainly related to nivalenol, deoxynivalenol, toxin T-2, diacetoxyscirpenol, and less often furarenone-X, toxin HT-2, and neosolaniol.

DON is probably the most largely distributed mycotoxin in food and feed often detected in wheat, barley, rye, malt, oat, maize, and consequently products derived from these cereals, such as flours and beers, and less often rice, sorghum, and triticale. It is a heat-resistant toxin which is not altered during food processing, in addition to being stable over long storage periods [37, 38].

Discovered in the early 1970s, DON is also popularly known as vomitoxin for its acute effect on the ingestion of high doses causing symptoms similar to enterotoxins of *Staphylococcus aureus* and *Bacillus cereus*, such as nausea, vomit, abdominal pains, diarrhea, leukopenia, bleeding, and even death in humans and animals [37, 38].

Pig is the most sensitive animal to DON, and the chronic effects of ingestion of contaminated feed result in reduced weight gain and growth, infertility, including the birth of animals with malformation, and miscarriage. Birds are more tolerant than pig, and the effects of intoxication are lower quality and weight of the eggs produced. Cattle are more tolerant, possibly due to the toxin degradation in secondary metabolites in the rumen. The effects in cattle include lower feed consumption and conception rate in addition to reduced milk production [2, 37].

The Codex Alimentarius Commission (CAC) establishes the maximum level of 2 μg/kg for DON for wheat, maize, and barley. The European Union, in turn, proposes 0.75 μg/kg for cereals and flour and 0.2 μg/kg for wheat germ [38].

4.5 Zearalenone

Also known as toxin F-2, it is an estrogen produced mainly by *F. graminearum*, *F. culmorum*, and *F. sporotrichioides*. Toxigenic strains of *Fusarium* can develop in soft climate and the optimum temperature to produce zearalenone is until 28°C. It is commonly found in several cereals, such as wheat, barley, sorghum, and mainly maize [6].

Zearalenone is a lactone of beta-resorcylic macrocyclic acid with a structure similar to 7ß-estradiol, main hormone produced in female human ovary. Zearalenones are considered micro-estrogenic due to their capacity to hamper the effect of steroid hormones interfering in human and animal reproductive capacities. It also influences the production of testosterone, progesterone, and estradiol. Zearalenone is able to imitate the activity of estrogen in the reproductive tract, including accessory glands, such as the prostate [40–42].

It causes hyperestrogenism in pig, whose symptoms are swelling and redness of the vulva and hyperdevelopment of the uterus and mammary glands. In addition to present significant effects on the increase of endometrial secretions and synthesis of uterine proteins and higher weight of reproductive organs [31].

Birds are more resistant to intoxication by zearalenone, but the many associations of fusariotoxin with other mycotoxins can result in severe losses. The production of zearalenone may occur either in the field or post-crop in inadequate storage

conditions (high moisture). The detection of this mycotoxin in bird feed has been considered a biomarker for other toxins belonging to the genus *Fusarium* [31].

Despite some evidence, the IARC assessed the carcinogenicity of zearalenone and found it to be a possible cancer-causing substance to humans. Zearalenone residues do not seem to be an issue after consumed.

4.6 Fumonisins

They were discovered in 1988 and described as fumonisins B1, B2, and B3, in which B1 occurs more frequently. However, fumonisins constitute a group encompassing over 16 substances already identified, called B1 (FB1, FB2, FB3, and FB4), A1, A2, A3, AK1, C1, C3, C4, P1, P2, P3, PH1a, and PH1b. They are highly water-soluble unlike other mycotoxins and do not have an aromatic structure or a single chromophore to analytically facilitate its identification, therefore being difficult to identify through ultraviolet spectrum [25].

These substances are produced by several species of the genus *Fusarium*, especially by *Fusarium verticillioides* (previously classified as *F. moniliforme*), *F. proliferatum*, and *F. nygamai*, in addition to *Alternaria alternata*. Other species, such as *F. anthophilum*, *F. dlamini*, *F. napiforme*, *F. subglutinans*, *F. polyphialidicum*, and *F. oxysporum*, also have been included in the group of these mycotoxin products [25, 38].

Fumonisins have been found as a common contaminant in maize-based food and feed. When ingested, fumonisins present low bioavailability and are rapidly metabolized and excreted. The carcinogenic nature of fumonisins does not seem to involve an interaction with DNA. Their mode of action is related to their toxicity in the interference of the biosynthesis of sphingolipid, which are very important to maintain the integrity of the cell membrane, regulation of receptors of cell surface, ion pump, regulation of growth factors, and other vital systems for the functioning and survival of the cell. In addition, fumonisins are potent immunosuppressant agents and can enhance the susceptibility to diseases [38–44].

These toxins cause many diseases in animals, such as leukoencephalomalacia (LEME) in horses and pulmonary edema in pigs. LEME is a noninfectious, highly fatal disease which affects the central nervous system of horses and other equines with a large distribution worldwide and considered a disease derived from regions of temperate, tropical climate. LEME involves metabolic alterations that produce the softening of the white substance of the encephalon as well as its liquefaction, which occurs due to a mycotoxin present in the feed. The disease occurs because of the need to supplement horse diet with grains of maize or feed containing them in their formulation due to the lack of fodder in pastures [43].

Even though their effects on human beings are difficult to determine, fumonisins have been statistically associated with high occurrence of esophageal cancer in South Africa and liver cancer in certain endemic areas in China. Based on toxicological evidence, the IARC declared the toxins of *F. moniliforme* as potentially carcinogenic to humans (Class 2B) [44].

4.7 Modified mycotoxins

The major mycotoxins in food (aflatoxins, ochratoxins, patulin, deoxynivalenol, zearalenones, and fumonisins) occur freely and coexist with modified mycotoxins. The term "mask mycotoxin" was used for the first time for mycotoxin M1, derived from the hydroxylation of aflatoxin B1, excreted in the milk of animals which consume contaminated feed with aflatoxin B1. In the mid 1980s, a new compound derived from zearalenone was found to be involved in cases of smycotoxicosi and no correlated to the mycotoxins found in the food matrix in question [45].

Recently, with the modernization of the means to detect the toxins, such as high-performance liquid chromatography associated with mass spectrometry, many other compounds derived from mycotoxins were discovered, and the term "mask mycotoxin" made way to the name of modified mycotoxins. This denomination classifies all substance derived from free mycotoxin, that is, all toxic substance originated by the secondary metabolism of fungi, which through a biological process (human, animal metabolism or even through the defense mechanism of plants) or technological process (food processing), have their structure altered as well as polarity, solubility, and molecular mass, originating a new substance with characteristic toxicity or able to reinforce the damages caused by parental mycotoxin [45, 46].

Some examples of modified mycotoxins are N-(1-deoxy-D-fructos-1-yl)-fumonisin B1 (NDF-FB1) and N-(carboxymethyl)-fumonisin B1 (NCM-FB1) formed in a Maillard reaction between fumonisins and reducing sugars and reaction of reduction occurring in DON when animal feed is treated with sodium bisulfite generating sulfonated DON [46].

5. Prevention and control of mycotoxins

Cereals, grains, and oilseed are often infected by insects and toxigenic fungi when still in the field interfering directly in the quality and productivity of food. Controlling fungi infestations is not an easy task for involving climatic and environmental issues which frequently cannot be controlled by men. Therefore, it is crucial to disseminate and implement techniques for good agricultural practices, indispensable to minimize problems related to the production of mycotoxins and quality of the food in the field. Some of these techniques involve choosing the variety to be cultivated by preferring lineages that are more resistant to attacks of plagues and microorganisms, good soil preparation, and turnover of cultures. It is also important to rationally employ agricultural pesticides by replacing them with sustainable techniques for plague control whenever possible, such as biological products, oils, and natural extracts, seeking to protect the cultivation and the environment [47].

The crop at the correct maturation point and the regulation of agricultural implements to soften mechanical damages to beans and grains are factors which combined with good storage practice can reduce fungus infestations in food products. The main storage practices encompass the improvement of the products received by removing impurities derived from the field, the control of moisture through drying process at recommended levels, good ventilation, cleansing of the storage location, and control of insects and rodents, in addition to a system to relative air humidity.

In addition to good agricultural and storage practices, some strategies for the detoxification of food and feed contaminated with mycotoxins have emerged as an effort to reduce or eliminate their toxic effects through chemical, physical, and biological methods. Some of them involve the application of gamma irradiation, ozone (O_3), and some microbial strains and fungus parasites able to inhibit the production or decrease the toxicity of some secondary metabolites like *Streptomyces rimosusand* and *Gliocladium roseum*, respectively. These methods are essential to improve food safety, prevent economic losses, and retrieve contaminated products [48, 49].

6. Conclusion

Mycotoxins are a group of fungal secondary metabolites, and their production is influenced by both the genotype of the organism and the physicochemical

environment in which it is growing. Even if a strain of mold has the genetic potential to produce a particular mycotoxin, the level of production will be influenced by the nutrients available. Even when the nutritional requirements are suitable for mycotoxin biosynthesis, physical parameters, such as temperature and water activity, will influence production. In nature there are many other factors interacting with the growth and metabolism of a mold. There may be, for example, antimicrobial agents produced by other microorganisms, by the plant on which the mold is growing, or added as biocides during crop husbandry. Mycotoxins have attracted worldwide attention not only because of their perceived impact on human health but also because of the economic losses accruing from contaminated foods. Mycotoxins have been extensively studied as well as their impact on human health. It is clear that food contaminated with toxic substances are not proper for either human or animal consumption. Considering that mycotoxins are natural contaminants and practically impossible to be completely eliminated from food, international food safety organizations provide guidance on the serious risks of mycotoxins to human health by updating and establishing safe levels of ingestion for these toxins. As a short-term solution, methods of prevention and food detoxification have been offered to producers aiming at providing means to enlarge the availability of safe food to the population worldwide.

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