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Mycotoxins: Health Problems and Future Strategies

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Abstract

Mycotoxins are one of the most hazards toxins for human and animal and the most widespread are aflatoxins, fumonisins, ochratoxins, trichothecenes, zearalenone and patulin. Aflatoxin is the most hazardous contaminated many foods. Numerous studies around the world indicated to the contamination of most foods with one or more types of mycotoxins induce large economic losses and health problem. The survey studies showed that they are contaminating most cereal crops, such as wheat, rice, barley, coffee beans, peanuts, medicinal herpes, etc. Mycotoxins cause many acute and chronic diseases for human and animal due to their accumulation in the body and affect different organs depending on the type of mycotoxin. Mycotoxins may convert to other derivatives, when introduce into the human body, animals and plants or during food processing and not appear in the known detection methods so the advanced detection method should be taken into consideration. Mycotoxins are hepatocarcinogenic, immunosuppressive; induced kidney tumor, carcinogenic, induced oesophageal cancer, etc. For these reasons the prevention or control is necessary to avoid myctoxin hazards and to find a suitable solution to the factors affecting on the climatic changes as well as good agricultural practices before and after harvest process, good manufacture practices and good storage conditions to ensure the food safety. Awareness of people to mycotoxin hazards will help and decrease the health problem as well as follow up these toxins in food and agriculture commodities.

Keywords:

Mycotoxins health hazards; Prevention strategies.

Introduction

Mycotoxins are secondary metabolites of fungi that contaminate many agricultural commodities and induced a wide hazard for people and animal as well [1]. There are four hundred different mycotoxins identified. The major concern medically and agricultural are aflatoxins (AFs), fumonisins (FBs), ochratoxins (OTA), trichothecene (TC), zearalenone (ZEA) and patulin (PAT) [1]. The mycotoxins molecular structure represents their toxic effects through the negative effects of oxidative stress and free radical production [2]. The imbalance between the antioxidant and

free radicals can cause damage to DNA, lipids, and proteins due to mycotoxins exposure [3].

A survey of mycotoxin in animal feeds in European and Mediterranean countries as well as Asia-Pacific area was carried out by Binder et al. [4], and the occurrence of DON, ZEA and T2 were the major occurrence in the European samples, while AFs, FBs, DON and ZEA mainly contaminated the Asia and Pacific areas. Herzallah [5] found aflatoxin M1 and M2 in milk and AFB1, B2, G1 and G2 in meat samples in Jordan. Reddy et al. [6] carried out a survey on rice samples in India and found many *Aspergillus* sp (Asp) and AFB1. Rice samples found to be contaminated with (Asp) and AFs in many countries such as Nigeria, United Arab Emirates and China [7]. Corn samples were highly contaminated with fungi, AFB1 and FB1 in Vietnam [8]. Alborch et al. [9] isolated different Asp, fusarium, penicillium, *Mucorales* sp, AFB1 and OTA from corn flour and popcorn. Mycotoxins were detected in solid processed foods in Spain [10].

Different toxigenic fungi isolated from medicinal herbs was reported from Argentina [11]. AFs, OTA and ZEA found to be contaminated Ginseng [12]. FB1 was reported in different dietary such as *Rumex lanceolatus* and *Zantedeschia aethiopica* etc., and different medicinal such as *Catha edulis, Dalbergia obovata* [13]. Different mycotoxins were found in medicinal herbs in Brazil [14]. The presence of AFB1 in *Mentha piperita*, *Piper nigrum, Pimpinella anisum* and *Origanum majorana* was reported by Bokhari [15] in Saudi Arabia. In Korea spices, 13.6% of processed spice products were contaminated with aflatoxin [16]. Multi mycotoxins contamination were found in 84 medicinal herbs surveyed in Spain [17].

In 2009, 324 grain, feed and feed commodity samples were taken directly from animal farms in the Middle East and Africa to evaluate the presence of A- and B-TC, ZEA, FBs, AFs and OTA and found the B-TC up to 87%. The prevalence of FBs in the studied countries was >50%. ZEA was found to be contaminated all tested commodities from the studied countries except three are Algeria, Sudan and Yemen. AFs levels varied from 0 to 94% and OTA was present in 67% of samples from Sudan and in 100% of Nigerian samples [18].

Major Mycotoxins with Important Impact to Human and Animals

Aflatoxins

Source: The major source of AFs are *Asp. falvus* and *Asp. parasiticus* as well as other *Asp*, sp such as *Emericella* spp. [19].

Occurrence: Aflatoxins are very important hazard contaminants due to their ability to invade human food and animal feeds like nuts, cereals, oilseeds, meat and meat products, milk and milk product and eggs [20].

Table 1 and Figure 1 showed the aflatoxins contamination levels in 324 grain, feed and other feed commodity samples that directly sourced from animal farms or animal feed production from the Middle East and some African countries during February and October 2009, and from the results, it is cleared that the most incidence aflatoxin contamination is Nigeria followed by Kenya, Ghana, Jordan, Egypt, Israel, Lebanon and Algeria [18].

Table 1. Aflatoxins contamination levels in samples from Middle East and some African countries [18].

Country	Aflatoxins
Algeria	
% of positive	0
Mean (ng g)	–
Egypt	
% of positive	19
Mean (ng g)	1
Ghana	
% of positive	72
Mean (ng g)	26
Israel	
% of positive	7
Mean (ng g)	1
Jordan	
% of positive	45
Mean (ng g)	4
Kenya	
% of positive	78
Mean (ng g)	52

Lebanon	
% of positive	0
Mean (ng g)	–
Nigeria	
% of positive	94
Mean (ng g)	115

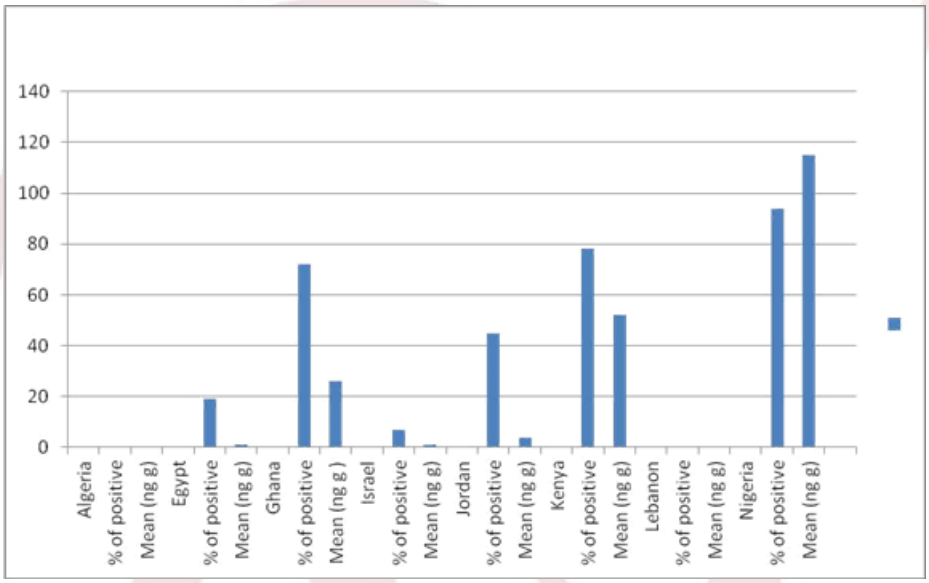

Figure 1. Aflatoxins contamination levels in samples from Middle East and some African countries [18].

Health hazards: *Human:* Aflatoxins specially B1 type is the most carcinogenic mycotoxins in human leading to aflatoxicoses [21]. Aflatoxin bind to the DNA at the N7 guanine base in hepatic cells causing several problems. AFB1 and B2 metabolism in the body are M1 and M2 and then excreted out in the urine [22].

AFs is a potent toxic carcinogen for liver and completely linked to the etiology causing hepatocellular carcinoma (HCC) which has increased up to 3.3 folds in the human with detectable AFM1 in the urine [23]. AFB1 is a potent hepato-carcinogen, generate reactive oxygen species and causes oxidative DNA damage, which may play a role in its carcinogenicity [24]. In 1993, IARC have been classified AFB1 as a class 1 carcinogen based on their chemical structures.

AFs caused acute liver and kidney lesions in children in Thailand [25] and was detected in pregnant women. Moreover, AFs at high levels cause hepatic carcinoma [26], and it was responsible for 4.6% to 28.2% of hepatocellular carcinoma worldwide [27]. Studies of AFs exposure on mice noted that mice after birth are prone towards HCC [28].

There is genetic alteration occurring in the body due to aflatoxin, since each person has a unique genetic code. These genetic codes are produced by a different level of enzymes that transform aflatoxin into epoxied. These enzymes, which carry out this process, include cytochrome P450 enzymes. When AFs epoxied produced in the body it is linked to albumin in the blood serum and adducts are formed [29].

Aflatoxins caused many genetic abnormalities including gene mutations, exchange chromatids, micronucleus formation [30]. From the biological markers during the past ten to fifteen years there are lots people exposed to high levels of AFs [31].

Animals: The toxic effects of aflatoxin are similar in all animals and are summarized as appetite loss, weight loss, disease in general, bleeding of the digestive system, lung problems, liver damage, liver clotting and hemorrhage. Aflatoxins also cause death of animals within hours, days or long periods depending on the degree of contamination and exposure to aflatoxins even from animal milk that effect on the growth small animals [32].

AFB1 is a toxic compound, mutagenic, carcinogenic and teratogenic in experimental animals [33] and can inhibit the nucleic acid and protein synthesis in animals [34].

Unhealthy chickens and poor feed intake, low growth rate, decrease egg production, and increased mortality rate and anorexia known as aflatoxicosis were noted due to AFs [35]. The human and animal exposure to aflatoxin from contaminated grains, food and feed is presented in Figure 2.

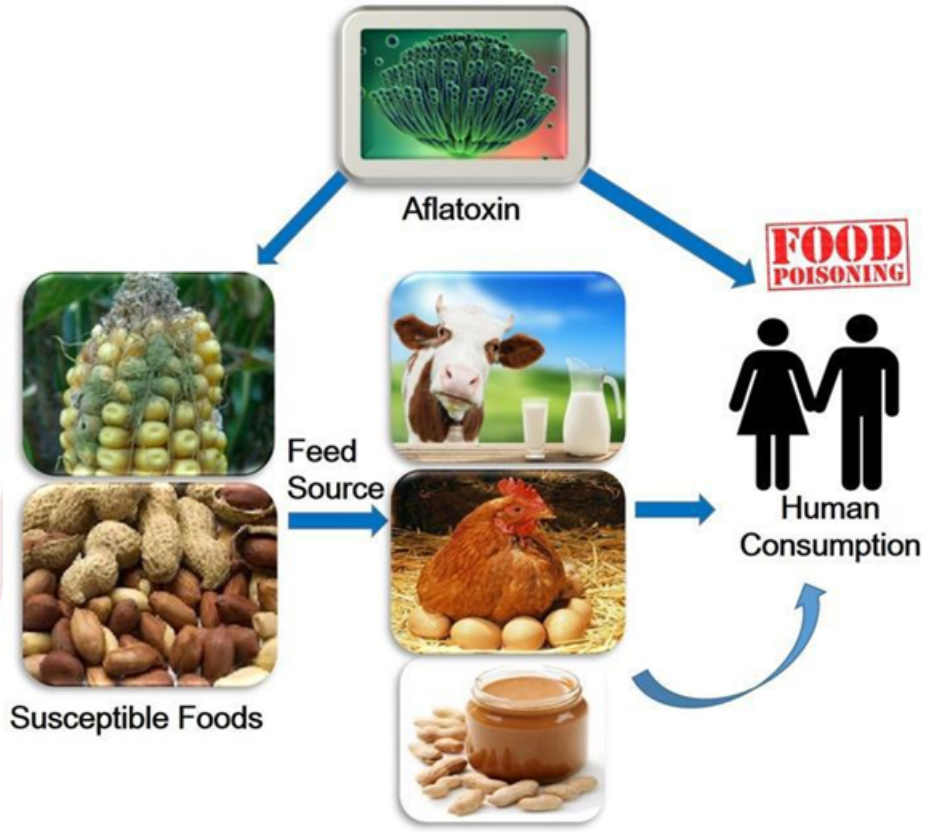

Figure 2. Showing to the source of Afs, animals and human exposure [36].

Fumonisin B1 (FB1)

Source: Fumonisin B1 elaborating by fungi are *Fusarium verticilliodes* (moniliforme) and *F. proliferatum* [37], which possess a morphology shape as shown in Figure 3.

Figure 3. The morphology of *Fusraium* sp.

Occurrence: FB1 is commonly found in corn grains and other agricultural commodities [38]. Also, FB1 was detected in rice grains, sorghum, wheat bran, poultry feed and soybean meal [39].

Table 2 and Figure 4 showing the fumonisin contamination levels in 324 grain, feed and other feed commodity samples that directly sourced from animal farms or animal feed production from the Middle East and some African countries during February and October 2009, and the results indicated that the most incidence

aflatoxin contamination is Ghana, Libanon, Joran, Egypt, Nigeria, Kenya, Algeria and Israel, respectively [18].

Table 2. Fumonisins contamination levels in samples from Middle East and some African countries [18].

Country	**Fumonisins**
Algeria	
% of positive	64
Mean (ng g)	977
Egypt	
% of positive	81
Mean (ng g)	266
Ghana	
% of positive	89
Mean (ng g)	500
Israel	
% of positive	63
Mean (ng g)	414
Jordan	
% of positive	85
Mean (ng g)	450
Kenya	
% of positive	76
Mean (ng g)	956
Lebanon	
% of positive	86
Mean (ng g)	183
Nigeria	
% of positive	78
Mean (ng g)	919

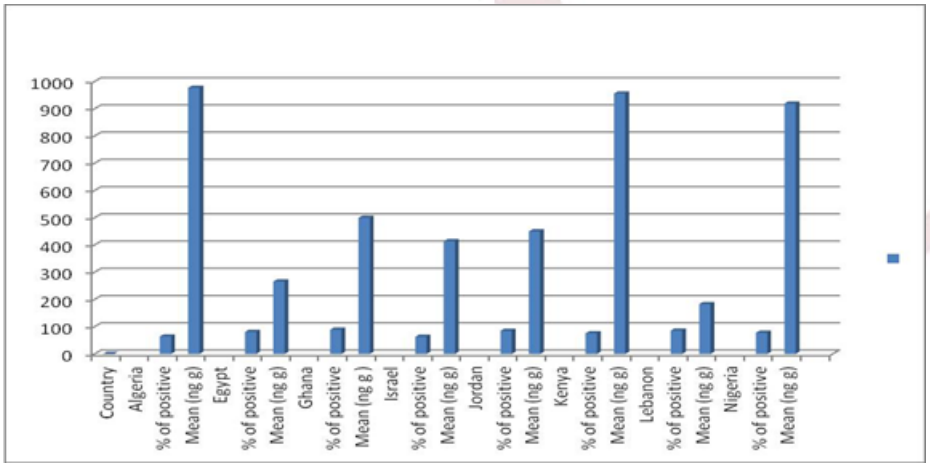

Figure 4. Fumonisins contamination levels in samples from Middle East and some African countries [18].

Health hazards: *Human:* FB1 possesses a clear structure like sphingolipids of cell tissues, leading to disturb of sphingolipids metabolism through inhibiting the enzyme ceramide synthase and therefore, accumulation of sphinganine in tissues and cells [38].

Epidemiologic studies in various countries of the world showed the correlation between human esophageal cancer incidence and the occurrence of *F. verticillioides*. Thus, esophageal cancer incidence has been associated with a poor socioeconomic status and less varied diets, consisting mainly of wheat or corn [40].

Comparative studies achieved in some areas in South Africa and China showed that higher levels of *F. verticillioides*, as well as concentrations of FB1 and FB2, occur in corn grains in the areas with high esophageal cancer incidence in comparison with corn grains in low risk areas [41]. Moreover, a high corn intake seems to be at a higher risk of developing esophageal cancer than those with a low corn intake. Similar ways were noted in the United States, Iran, Italy, Kenya, Zimbabwe, and Brazil with a high incidence of esophageal cancer [40].

In 1990-1991, outbreak of neural tube defects (NTD) in the brain and spinal cord resulting from failure of the NT occurred in the Texas Mexico border might have been due to the high levels of FB1 occurred in corn grains [42], the same NTD detected in certain areas in China and South Africa, due to high maize consumption [43].

The reactive oxygen species (ROS) caused by FB1 was studied in human and rat cell line and mouse hypothalamic cells and noted from the results an increase of ROS production due to exposure to 10-100 μM FB1 for 48-144 h [44]. The effect of FB1 on cell viability have been studied and cytotoxic effects have been observed [45].

Animals: FB is known as a toxic and has a carcinogenic effect in different animals in cells through induction of oxidative stress [45].

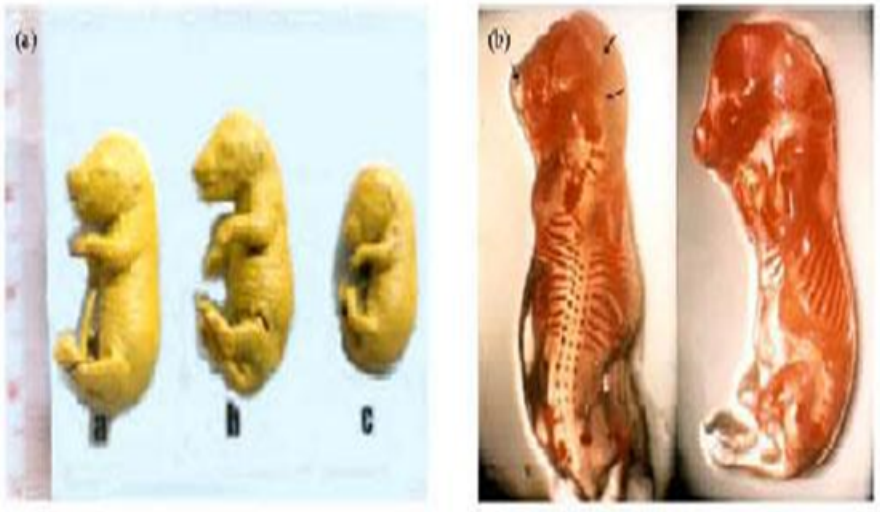

Figure 5. Rats fetuses exposed to FB [46].

Exposure of rat fetuses to FB caused several fetal growth defects as in the Figure 5a and skeletal malformation as shown Figure 5b.

Studies indicate its toxicity in animals as it is a carcinogen in different animals and causes liver poisoning, liver cancer and kidney poisoning, problems in the immune system, causes pulmonary complications in pigs, leukemia, and causes fatty acid abnormalities. It has also been found to have negative effects on mice, rabbits and chickens [47].

Ochratoxin A: *Source:* Ochratoxin A (OTA) is a mycotoxin produced by several fungal species, including *Aspergillus ochraceus* (Figure 6), *Penicillium verrucosum*, *A. carbonarius* and *A. niger*.

Occurrence: Exposure to ochratoxin A (OTA) occurs principally in Europe and Canada, where people consumed processed food from barley, wheat and coffee bean. Minor sources include meat, especially pork, from animals fed contaminated grain. OTA has been the subject of an environmental health criteria document [49] and JECFA evaluations [50, 51].

Health hazards: *Human:* The target organ for OTA is kidney since it causes nephrotoxicity, renal tumors and adverse health effects [52]. OTA is a widely-spread all over the world, causing major health risks. The mode of action of OTA is not clearly understood yet and seems to be very complex. Inhibition of protein synthesis and energy production, induction of oxidative stress, DNA adduct formation, as well as apoptosis/necrosis and cell cycle arrest are possibly involved in its toxic action. Since OTA binds very strongly to human and animal albumin, a major emphasis is done regarding OTA-albumin interaction [53].

Animals: The toxic effects include cardiac and hepatic lesions in rats, lesions of the GIT and lymphoid tissues in hamsters and kidney lesions in chickens. Pigs appeared to be the most sensitive species to the nephrotoxic effects. Degeneration alteration in the proximal kidney tubules is the most common effects shown in animal species [50].

Trichothecene (TC)

Source: The main fungal genera that produce TC are *Fusarium, Myrothecium, Spicellum, Stachybotrys, Cephalosporium, Trichoderm* and *Trichothecium* [54].

Occurrence: Tricothecene toxicosis on human was reported due to ingestion of moldy rice contaminated with it in China [55], in processed foods such as wafers, biscuits, and rusks in Italy [56] and at high level in Poland [57] as well as in maize grains in Pakistan [58].

Health hazards: *Human:* All tricothecenes contain epoxide at the C12, C13, which is responsible for their toxicological effects [59]. Wang. et al. [55] reported that 65% of patients developed food poisoning symptoms such as dizziness, vomiting, abdominal pain, nausea, abdominal distension, and diarrhea.

TC targets the ribosomal subunit, suggesting that the major mechanism of toxicity is translational inhibition. TC has multiple effects on eukaryotic cells, the most effect is inhibition of protein, RNA and DNA synthesis [60]. Moreover, alteration of membrane structure and mitochondrial function, stimulation of lipid peroxidation, induction of apoptosis, activation of cytokines and chemokines, activation of mitogen activated protein kinases-alteration at neurotransmitter levels [61]. TC is known to cause immune suppression, neurotoxicity and renal toxicity. Several studies have shown that TC can cause adverse effects in humans consuming grain-based foods and in animals ingesting contaminated grain, including in chronic low-level exposure like neuroendocrine changes, immune suppression, gastroenteritis emesis, nausea, anorexia, growth retardation, and gastrointestinal toxicity [62].

Animals: The toxic effects of TC in animals like swines, dairy cattle and rats including decrease of plasma glucose, reduce of blood cell, loss of weight and alteration in histology picture for both liver and stomach [2].

Using an animal experiment, TC did induce necrotic lesions in the GIT [63]. Also, a shortening of villi height was detected due to TC treated animals. The changes on villi were due to activation of the apoptotic pathway by TC, which in turn leads to nutritional malabsorption [64].

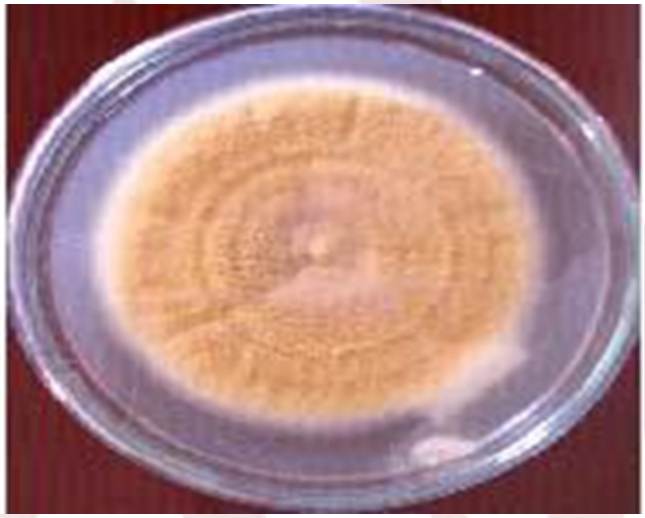

Figure 6. Colony surface of *Asp. ochraceus* [48].

Zearalenone (ZEA)

Source: Zearalenone is a mycotoxin that mainly produced by *Fusarium culmorum* and *Fusarium graminearum* [65].

Occurrence: ZEA has been linked to scabby grain toxicosis found in the USA, China, Japan and Australia [66]. Zearalenone occurred in different commodities such as wheat grains, barley, maize, corn silage, sorghum, rice, and sometime in the feed.

Table 3 and Figure 7 showing zearalenone contamination levels in 324 grain, feed and other feed commodity samples that directly sourced from animal farms or animal feed production from the Middle East and some African countries during February and October 2009.

Health hazards: *Human:* Toxicological studies reported its effects on the reproductive system, such as alteration of reproductive tract, decrease of fertility and abnormal level of progesterone. Also, the ingestion of ZEA during pregnancy reduced fetal weight and survival rate of embryo [67].

Table 3. Zearalenone contamination levels in samples from Middle East and some African countries [18].

Country	Zearalenone
Algeria	
% of positive	0
Mean (ng g)	-
Egypt	
% of positive	19
Mean (ng g)	9
Ghana	

% of positive	11
Mean (ng g)	178
Israel	
% of positive	19
Mean (ng g)	25
Jordan	
% of positive	5
Mean (ng g)	57
Kenya	
% of positive	56
Mean (ng g)	67
Lebanon	
% of positive	29
Mean (ng g)	19
Nigeria	
% of positive	51
Mean (ng g)	46

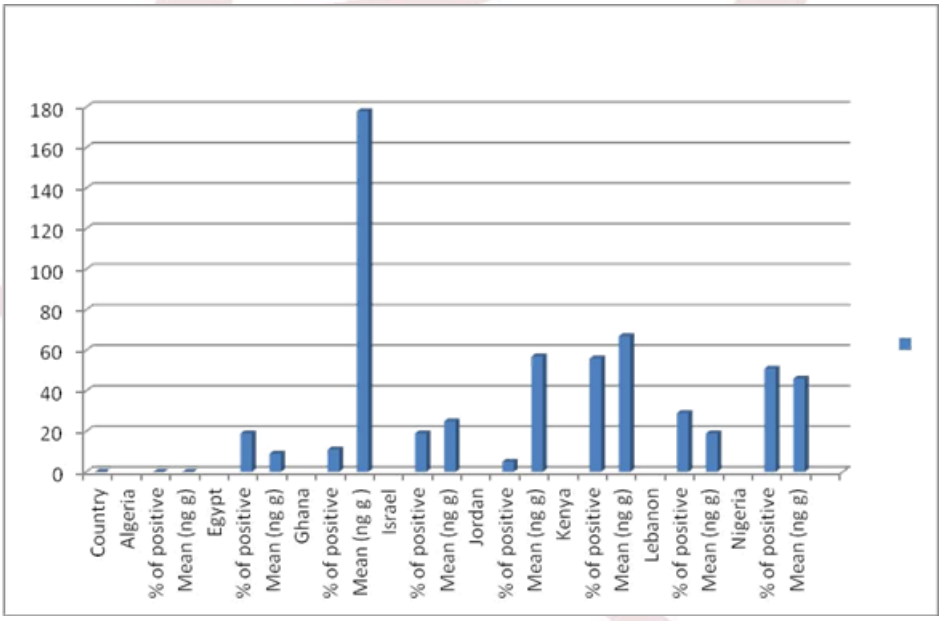

Figure 7. Zearalenone contamination levels in samples from Middle East and some African countries [18].

This phenomenon could explain through the structure of ZEA which allows it to bind to the mammalian estrogen receptor, although with lower affinity compared to the naturally-occurring estrogens [68]. Also, ZEA has been shown to be hepatotoxic, haematotoxic, immunotoxic and genotoxic [69].

The main target organ of ZEA is the reproductive organ; but the adverse effects of ZEA on GIT have been noted. Studies using intestinal epithelial cells showed that ZEA induced cell death without altering the cell integrity as indicated by transepithelial electrical resistance [70].

Animals: Several *in vitro* studies reported that exposure to ZEA leading to decrease of feed intake, refuse of feed, malnutrition in animals, diminished body weight gain and increase incidence of disease [71].

Zeralenone is particularly toxic to the reproductive system, induce uterine enlargement, alteration of the reproductive tract, decrease fertility, as well as a change of the progesterone levels in laboratory animals [72].

Patulin

Source: Patulin is a secondary metabolite of certain Penicillium, *Aspergillus*, and *Byssochlamys* sp., but more specifically, *P. expansum* fungus mainly responsible for production of Patulin [73].

Occurrence: Patulin can contaminate fruits and vegetables, but specifically contaminated apples and its juice are considered the main source of patulin toxin. However, patulin was firstly proposed as a drug due to its antibiotic properties [74].

Health hazards: *Human:* Patulin is classified as a group C or as not carcinogenic for human [75]. PAT has been related to DNA damage in human cell line and has apoptotic activity [76], also PAT induced DNA strand breaks, micronuclei formation in mammalian cells and chromosome aberrations [77]. It also causes carcinogenicity, mutagenicity, developmental and reproductive toxicity and immunotoxicity [74].

Animals: Most sensitive effectual signs of patulin at high doses (>16 mg/kg bw/d) in experimental animals include toxicity of GIT and lung. In the only available 2 y toxicity study, the most sensitive toxic effect of patulin was a decrease body weight in male rats at oral doses above 0.1 mg/kg bw. Patulin at oral dose of 1.5 mg/kg bw/d caused an increase the mortality in both sexes of experimental animals [78].

Modified Mycotoxins

Modified mycotoxins are secondary metabolites normally found in the substrate, but undetectable when parent mycotoxin analysis and the modified form can generate by fungi or by infected plants or during food processing and therefor the chemical structure of toxins might change and called masked mycotoxin. The new form of mycotoxins can be even more toxic than the parent mycotoxin [79]. The change in chemical structure of mycotoxins due to modification process demand to advance in the development of extraction techniques, detection and production of reference materials [80]. Mycotoxins react with food components through the covalent bond, it is conjugated mycotoxin, while, whin bind with food components through noncovalent bond it is called hidden [81].

Modification processes for mycotoxins carried out through many ways are chemical process or biological (animals, plants, microorganisms) [80].

Modified mycotoxins by plants

Plants when infected with mycotoxins producing fungi can alter the chemical structure of mycotoxin due to their defense mechanism, mycotoxin can modify through conjugate with organic molecules or by hydrolysis, (reduction and oxidation reaction). The derivatives may incorporate into the cell wall components [82].

Modified mycotoxins by microorganisms

Some strains of bacteria, yeast, and fungi can alter the mycotoxin structure as a defense mechanism through the enzymatic activity, or by the adducts which strongly reported in fermented products like beer and wine through the microorganisms used in the fermentation process [82,83].

Modified mycotoxins by yeast

Trichomonascus clade species showed to have the ability to biotransform T2 into T2 toxin-α-glucoside (by glycosylation); into 3-acetyl T-2 toxin (through acetylation action of acetyltransferases). The defense mechanism of microorganisms acts on a C-3 hydroxyl group of the parent mycotoxin, reducing its toxicity [84]. ZEN mycotoxin has been converted to α-ZEL, β-ZEL, ZEN-14G and ZEN-16G by a strain of Saccharomyces cerevisiae [85]. *Clonostachys rosea* is also able to detoxify ZEN by the action of enzyme lactonohydrolase.

Modified nycotoxins by fungi

Some fungal sp don't produce mycotoxin but can modify the parent mycotoxin leading to conjugate glucosides and sulfates like the formation of ZEN glucoside by *Rhizopus* and *Thamnidium* spp [86]. The new conjugate can be reconverted to ZEN by the enzymatic action (sulfatases) or chemical hydrolysis, and therefore the food processing, digestive process in human and animals can lead to the release of a ZEN molecule [87].

Effect of food processing on mycotoxins

Myctoxins considered heat stable when exposed to heat or during food processing, but partially may remove or alter through physical treatments like peeling and milling, or by chemical treatment such as alkali and acid, also by biological process such as fermentation [88].

Future Strategies

Control of mycotoxins

The number of strategies to control and prevention of mycotoxins has been considered in various regions in the world including African countries.

The use of innovative processing techniques to control of myctoxins (such as roasting using infrared, cold atmospheric pressure plasma, non-ionizing radiations, and neutral electrolyzed water) will greatly improve the safety of different food/feed [89].

Pre and post-harvest process strategies are very important to manage the toxicogenic fungi in food commodities. Detoxification of mycotoxin using natural agents can suppress or reduce the absorption and promote excretion or modify the mode of action. The feed additives transform mycotoxins into less toxic metabolites either by reducing their bioavailability or by degrading them. Therefore, it can define at least two main categories; including first various mycotoxin adsorbing agents and second biotransforming agents lead to degradation of mycotoxins into non-toxic metabolites. Also, advanced detection techniques are helping in ultra-trace amount of mycotoxin in food products such as; bio-sensors (1) electrochemical biosensors, 2) optical biosensors 3) electronic biosensors, 4) piezoelectric biosensors, 5) gravimetric biosensors, 6) pyroelectric biosensors [90].

Integrated mycotoxin management programmes

Strategies to ensure the food safety and avoid the economic losses include both preharvest and post-harvest process to decrease the hazard of mycotoxin risk in food and feed.

Preharvest process includes good agriculture practices, biocontrol and advancement of resistant varieties of the crops *via* new biotechnologies. Application of the good manufacture practices in

all stages of food production. Application of hazard analysis critical control point (HACCP) system in all stages of food processing from the farm to the consumer.

Postharvest process such as good storage, detection and detoxification and continuous monitoring of potential contamination during processing and handling of food and feed. Selected feature of an integrated mycotoxin control program should involve different phases such as the ones outlined below:

Preharvest procedure: The mycotoxin producing fungi can contaminate the food crops in the field, some strategies for control in the field are

1-Reduction of plant stress through irrigation, mineral nutrition and avoid the insect damage.

2-Avoid the unsuitable infection in the field eg. delay of harvesting etc. environmental condition that encourages the drought, insect infestation, primary inoculums.

Potentially effective: 1-Application the good agricultural practices through applying the crop rotation, irrigation, timely planting and harvesting and use the biopesticides that reduce the mycotoxin contamination in the field crops.

2- Development of breeding resistant cultivars to fungal growth.

3-Use of antifungal chemical agent that protect against fungal growth and mycotoxin contamination.

4-Identification of plant constituents that suppress mycotoxin biosynthesis of fungal growth to use in new biocontrol-based breeding strategies to improve resistance in crops.

Developmental: 1-Development of resistant plant to fungal infection.

2-Development of transgenic cultivars capable of catabolism/ interference with toxin production.

3-Improve the genetically modified crops to resist insect damage.

4-Improve of crop grains containing endophytic microorganisms that exclude toxigenic fungi.

5-Supress of toxigenic fungi using pre-infection with bio competitive non toxigenic fungal strains.

6-The genome sequence of *A. flavus* has been known to help to understand the regulation of aflatoxin production by environmental factors and the obtained information can be used in improving the host resistance against aflatoxin contamination by studying the effects of various physiological parameters eg. drought stress on gene expression in toxigenic fungi.

Harvesting procedure: 1-Avoid the mechanical damage of the grains to incur during the harvesting and storage process.

2-The field crops must be harvested at the suitable time to decrease the moisture content and water activity to avoid the mycotoxin producing fungi.

Post-harvest procedure strategies: 1-Sorting the grains and remove the damaged and drying to minimal moisture content to avoid the fungal growth.

2-Monitoring of insect and rodent activity and maintenance of appropriate moisture content and temperature.

3. Select the appropriate packaging is often a successful way of excluding insects and fungi.

4. Cleaning of food/feed delivery systems and short-term storage.

5-Use of antifungal activity agents like propionic and acetic acid.

6-Use of thermal inactivation that normally used in some food processing since the FBs and OTA have been shown to be lower in thermally processed corn and wheat products [91].

Conclusion

Mycotoxins are a global problem that poses a great hazard to human and animal health. It also causes great economic losses in agricultural crops, food and feed. Therefore, the solutions should be developed to avoid these toxins. These solutions are in the pre-harvest process such as harvesting on suitable time and drying crops to be unavailable to fungi growth, the other solution is post-harvest such as good handling and good storage of crops and raise awareness of citizens the hazard of these toxins as well as applying the good manufacture practices in food processing.

Future Trends

- Adapting to the climatic changes, which is a major cause of fungal growth and the emergence of more toxic fungal strains, unlike the past, through reducing the thermal emissions and factories exhausts that effect on the climate in general.
- Application of non-conventional solutions to control these toxins through the use of good manufacturing practices, either after harvesting the crops or during food processing and trying to apply the Hazard Analysis Critical Control Point (HACCP) system in all stages of food processing.
- Continue to conduct surveys to determine the proportions and presence of these toxins in food commodities.
- Working on the development of cultivars resistant to fungi growth and thus avoid their toxicity.
- Development of detection technique to determine the derivatives of mycotoxins in food and feed.
- An attempt to analysis of toxins in human blood within the routine analyses performed in human medical examinations.
- Conduct an awareness program for citizens on the hazard of these toxins and how to avoid them in the daily food using simple ways through the media readable, audio and visual or by holding training courses for citizens in different regions.

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