

## Mycotoxigenic Fungi, Mycotoxins and Mycotoxicosis: A Review

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### ABSTRACT

Mycotoxins are toxic secondary metabolites produced by molds. These mycotoxins can enter food chain either directly from plant based food components or by indirect contamination from the growth of toxigenic fungi on food. The objective of this review is to compile different literatures which indicate mycotoxins, their mycotoxigenic fungi and the effect they can cause. Members of fungal genera such as *Aspergillus*, *Fusarium*, and *Penicillium* are the major mycotoxin Producers. Over 300 mycotoxins have been identified and the major ones are aflatoxins, trichothecenes, zearalenone, fumonisins, ochratoxins, and patulin which are regularly found in food. Mycotoxins can be found in corn, cereals, soybeans, sorghum, ground nut, pepper and other food and feed crops. They can be produced at time of pre harvesting, harvesting and post harvesting of grain. Consuming mycotoxin-contaminated food or feed can cause acute or chronic toxicity in human and animals. In addition to concerns over adverse effects from direct consumption of mycotoxin-contaminated foods and feeds, there is also public health concern like nephrotoxic, genotoxic, carcinogenic hepatotoxic effect over the potential ingestion of animal-derived food products, such as meat, milk, or eggs, containing residues or metabolites of mycotoxins. To minimize these risks, different management practices has to be applied from which good agricultural practice, good processing practices and good manufacturing practices should taken to consider.

**Keywords:** Mycotoxin, Mycotoxigenic fungi, Mycotoxicosis

### INTRODUCTION

Mycotoxins got concern since the outbreak of the mysterious turkey X disease in 1960 which killed approximately 100,000 turkeys in England due to consumption of a groundnut meal contaminated with secondary metabolite aflatoxin from *Aspergillus flavus* (Blount, 1961; Kensler et al., 2011). Secondary metabolites which have deleterious effects are called mycotoxins, referring to their fungal origin and toxic nature (Bennet and Klich, 2003). Mycotoxins can be developed during production, harvesting, or storage of grains, nuts, and other crops (Ramesh and Siruguri, 2003). Prolonged exposure for mycotoxins through diet has been linked to kidney and liver cancer as well as weakening of the immune-system (Rios et al., 2013).

Mycotoxins occur more frequently under tropical climate where high temperature and humidity are prevailing. In addition, diets in many tropical countries are more heavily concentrated on crops corn and nuts that are susceptible to mycotoxins; consequently, chronic health risks are particularly prevalent in such countries (Ramesh and Siruguri, 2003).

Some mycotoxins are produced before harvest (DON, ergot); some occur following harvest (fumonisin, ochratoxin); and a few predominantly occur during storage (aflatoxin) (Ramesh and Siruguri, 2003).

Mycotoxins are chemically stable, they are not easily degradable by normal food processing or autoclaving (Eriksen, 2003; Wannemacher et al., 2000). These toxic substances end up in the chain of food and feed.

The effects of these toxic substances are carcinogenicity, genotoxicity, nephrotoxicity, hepatotoxicity, reproductive disorders, immune-suppression and dermal effects (Bennett and Klich, 2003). The severity depends, however, on various factors including the type and concentration of mycotoxin, the route and duration of exposure, mode of action of the toxin, the animal species as well as gender, age, body weight and health status of the animal (Hussein and Brasel, 2001; Avantaggiato et al., 2005). The symptoms of mycotoxicosis are almost as diverse as the chemical structures of the compounds themselves. Some compounds may elicit few symptoms until death results, while others may produce severe effects

including skin necrosis, leucopenia and immunosuppression. Doses producing chronic disease are usually far below those responsible for acute effects, and so long-term effects such as cancer or tumor induction are undetected at the time of ingestion and, indeed, may remain so until disease is quite advanced (Pitt, 2000).

### MYCOTOXIGENIC FUNGI AND THEIR MYCOTOXINS

#### Aflatoxins

Aflatoxins are secondary metabolites of the molds *Aspergillus flavus*, and *Aspergillus parasitica*. They are highly toxic, mutagenic, teratogenic and carcinogenic compounds found to contaminate a wide variety of important agricultural products such as peanuts, maize, rice and cottonseed.

Aflatoxins are found in many countries, especially in tropical and subtropical regions where conditions of temperature and humidity are optimum for growth of the molds and for production of the toxin. Removal or inactivation of aflatoxin in food and feedstuffs is a major global concern (Rustom, 1997). Among these groups of aflatoxins, aflatoxin B<sub>1</sub> is the most prevalent, potent and class I carcinogenic (Feddern, et al., 2013). These are both acutely and chronically toxic to animals, including man, causing acute liver damage, liver cirrhosis, induction of tumours and teratogenic effects (Pitt, 2000).

#### Ochratoxins

Ochratoxins are mycotoxins produced by certain fungi (*Aspergillus ochraceus* and *Penicillium verrucosum*). Structurally, they have a particularity of containing a chlorine atom. Naturally, they are found in many plant products, such as cereals, coffee beans, cocoa, and nuts. They have been also detected in products made from cereals, wine, beer, and grape juice, as well as in animal products, such as pig kidneys (Cerain, 2007). Ochratoxin a (OTA) shows carcinogenic, nephrotoxic, teratogenic, immunotoxic, and neurotoxic properties.

It has been also associated with nephropathy in humans. OTA is a small molecule soluble in water and it is chemically constituted by a combination of an amino acid (phenylalanine) and a polyketide to carbon 10, contains one chlorine atom necessary for its biological activity, and it is stable when exposed to heat (Bueno et al., 2013).

#### Fumonisin

Fumonisin are mycotoxins which produced by *Fusarium* species which are responsible for promoting cancer (Gelderblom et al., 1991). It is also associated with human and animal diseases (Harrison et al., 1990; Marasas, 2001). The fumonisin B (FB) molecule is composed of a long hydroxylated hydrocarbon chain with added tricarballic acid and methyl and amino groups (Gelderblom et al., 1991). FB<sub>1</sub>, FB<sub>2</sub>, and FB<sub>3</sub> are the major naturally occurring FBs. FB<sub>1</sub> is carcinogen that categorized as Group 2B by the International Agency for Research on Cancer (IARC 2002). A number of industrially important strains of *Aspergillus niger* also produce FB<sub>2</sub> and FB<sub>4</sub>, additionally pointing out the potential mycotoxicological risk of some foods colonized by this species (Frisvad et al., 2007).

#### Zearalenon

This mycotoxin was mentioned earlier in that it may co-exist with deoxynivalenol (DON) as the same organisms, *Fusarium graminearum* or *Fusarium culmorum* (CAST, 2003), may produce both compounds. Grains infected with the above organism may exhibit the pink color associated with the production of a pink pigment simultaneously produced with the zearalenone. These mycotoxins frequently occur in maize. However, it is found also in other important crops such as wheat, barley, sorghum and rye throughout various countries of the world (CAST, 2003). In wheat the conditions for the occurrence of zearalenone would be essentially the same as for the occurrence of deoxynivalenol (DON) since the organism gains entry into the host plant in the same manner. Generally, the *Fusarium* species grow in moist cool conditions and similarly invade crops under these more favorable conditions. In wheat, sorghum and corn, zearaleone occurs in pre-harvest grain (WHO Food Additives Series: 44, 2000).

#### Trichothecenes

Trichothecenes are powerful inhibitors of eukaryotic protein synthesis, phytotoxic, insecticidal and toxic to animals, and some are among the most toxic non-nitrogenous compounds known to man. Several are commonly found in cereal grains, and the potential health risk from contaminated animal feed and human food is a major factor in stimulating research into this group of compounds (Grovey, 2007).

These mycotoxins produced mostly by members of the *Fusarium* genus, although other genera

are known to produce these compounds. The most frequent contaminants are deoxynivalenol (DON), also known as vomitoxin, nivalenol (NIV), diacetoxyscirpenol (DAS), while T-2 toxin is rarer (WHO, 1990). Common manifestations of trichothecene toxicity are depression of immune responses and nausea, sometimes vomiting. The first recognized trichothecene mycotoxicosis was alimentary toxic aleukia in the Union of Soviet Socialist Republics in 1932; the mortality rate was 60% (Gajdusek, 1953).

In regions where the disease occurred, 5±40% of grain samples cultured showed the presence of *Fusarium sporotrichoides*, while in those regions where the disease was absent this fungus was found in only 2±8% of samples. The severity of mycotoxicosis was related to the duration of consumption of toxic grain. In several cases, trichothecene mycotoxicosis was caused by a single ingestion of bread containing toxic flour (Bhat et al., 1989). In experimental animals, trichothecenes are 40 times more toxic when inhaled than when given orally (Smoragiewicz et al., 1993).

### NITROPROPIONIC ACID

It is a secondary metabolite of *Arthrimum* sp., considered to cause a form of acute food-poisoning called "mouldy sugarcane poisoning" (Liu, 1988). The incubation period is generally 2±3 hours following the ingestion of mouldy sugar-cane and the main clinical symptoms are vomiting, dystonia, staring to one side, convulsions, carpopedal spasm and coma. Delayed dystonia develops in 10±50 % of patients as a consequence of bilateral symmetric necrosis of the basal ganglia.

The development of delayed symptoms can be predicted by abnormality in the basal ganglia on cranial computed tomography (CT) scans (Ming, 1995). It is a suicide inhibitor of succinate dehydrogenase a widely distributed plant and fungal neurotoxin known to induce damage to basal ganglia, hippocampus, spinal tracts and peripheral nerves in animals. Reports from Northern China indicate that 3-NPA is also likely to be responsible for the development of putaminal necrosis with delayed dystonia in children after ingestion of mildewed sugar cane. (Ludolph et al., 1991)

### Ergot Alkaloids

The ergot alkaloids are among the most fascinating of fungal metabolites. They are

classified as indole alkaloids and are derived from a tetra cyclic ergo line ring system. Lysergic acid, a structure common to all ergot alkaloids, was first isolated in 1934. The clavines have ergoline as a basic structure but lack peptide components; the lysergic acid alkaloids include ergotamine and lysergic acid amide (ergine) (Bennett& Bentley, 1999).

The human disease acquired by eating cereals infected with ergot sclerotia, usually in the form of bread made from contaminated flour, is called ergotism or St. Anthony's fire. Two forms of ergotism are usually recognized, gangrenous and convulsive. The gangrenous form affects the blood supply to the extremities, while convulsive ergotism affects the central nervous system (Bennett& Bentley, 1999). Stereoselectivities of several ergot alkaloids, added to the background electrolyte (BGE), towards some racemic hydroxy organic acids are compared. The 1-allyl derivative of (5*R*, 8*S*, 10*R*)-terguride (allyl-TER) proved to be the best chiral selector for these analytes (Ingelse et al., 1996).

### Patulin

This mycotoxin is produced by *Bysochlamys*, *Eupenicillium*, *Penicillium*, *Aspergillus* and *Peacylomyces* in a variety of food products (Kadagal and Nas, 2002). The main sources of patulin intake in human diet that was shown for EU consumers are apple juice and nectar and for this reason, apple-based food is most often monitored for this mycotoxin. However, the presence of patulin in other fruits, including stone fruits, and soft fruits has been reported as well (Sadok et al., 2019). Several studies have shown that patulin is stable in dry cereals, and in apple and grape juice, but that it is decomposed in wet cereals and during production of cider (Most and Long, 2002).

The International Agency for Research on Cancer IARC (2012) classified several mycotoxins as carcinogenic or potentially carcinogenic to humans according to the following groups:

Group 1: The agent is carcinogenic to humans.  
Group 2A: Probably carcinogenic agent in humans, there is limited evidence in humans but sufficient animal.

Group 2B: Possibly carcinogenic agent, the evidence in humans is limited and there is no sufficient evidence in experimental animals.  
Group 3: The agent is not classified as a human carcinogen, and cannot be included in another group.

Group 4: The agent is probably not carcinogenic to humans; the available evidence from both human and animal studies suggests so.

### Mycotoxicosis

Mycotoxicosis is a toxic conditions caused by the ingestion of feed and food contaminated with the toxins of different mycotoxigenic fungi. This mycotoxicosis is nephrotoxicity, hepatotoxicity, genotoxicity and immunotoxicity.

### Nephrotoxicity

It is a poisonous effect of some substances, both toxic chemicals and medications, on kidney function. A long time exposure to mycotoxins is account for nephropathies and urinary tract tumors (Radovanovic et al., 1991). Ochratoxin A binds to a serum macromolecule of low relative molecular weight accounting for the nephrotoxic effects in mammals due to its accumulation in the kidney (Stojković et al., 1984). Ali and Abdu, (2011) studied that ochratoxin A treatment caused the declines in kidney weight and its relative weight and the increases in serum urea and creatinine levels. Bayman and Baker, (2006) reported that ochratoxin A treatment increased renal disease followed by proximal tubular atrophy and also cortical interstitial fibrosis.

### Hepatotoxicity

Hepatotoxicity is damage of liver by toxic substances. Deoxynivalenol intoxication predisposed the animals to infectious diseases (Oswald et al., 2005). Moreover, deoxynivalenol challenge negatively affects the production of TNF- $\alpha$  from the macrophages which expose for chronic exposure to 10 mg deoxynivalenol/ kg feed suppressed the plasma TNF- $\alpha$  level responsible for inflammation (Awad et al., 2012). Alterations including bile duct proliferation, periductal fibrosis and cholestasis (Javed et al., 1993). Feeding diets containing 100 ppb aflatoxin displayed hydropic degeneration and fatty vacuoles in hepatocytes when compared to control (Ortatatli et al., 2005). Krishnamoorthy et al., (2007) exhibited pale, enlarged liver, hepatocytes necrosis and also bile duct hyperplasia in chicks exposed to T-2 toxin.

### Genotoxicity

Combinations of T-2 toxins and aflatoxin B1 are the strongest mutagens (Smerak et al., 2001). Sehata et al., (2004) studied the alterations in gene expression induced by T-2 toxin in the fetal brain of pregnant rats. The effects of the deoxynivalenol on expression of interleukin-8

gene in cloned human monocytes and peripheral blood mononuclear cells and also deoxynivalenol treatment (250-1000 ng/mL) caused an increase in interleukin 8 mRNA abundance (Islam et al., 2006).

### Immunotoxicity

The immune system is considered to be a crucial defensive mechanism against invading pathogenic bacteria and foreign cells (Sharma, 1993). The specialized cells of immune system interact with each other to produce the desired consequents (Sharma, 1993). The effect of mycotoxins on immune system is either suppressive or stimulator depending on the time, duration and dose of exposure (Pestka, 2008). Previous study has been found that there is a potent association between aflatoxins contamination and immune-suppression, reflecting that intake of aflatoxins increased the susceptibility of humans to infections (Turner et al., 2003). Many studies have indicated that mycotoxins especially aflatoxin B1 interact with biomolecules namely DNA; in turn, altering their actions (Riley, 1998). Mycotoxins always inhibit protein synthesis; consequently, they adversely influence immune cell proliferation (Gelderblom et al., 1996). Mycotoxins are Iso cytotoxic especially lymphocytes due to their impacts on membranes (Surai, 2002).

Immune cells have the high level of poly-unsaturated fatty acids on their membrane and receptors; thus, free radicals-induced by mycotoxins impose these cells to damage (Surai, 2002). It has been also demonstrated that the susceptibility of immune system to immunotoxicity resulted from mycotoxins is probably related to the sensitivity of immune cells to proliferation and differentiation that interfere with immune-mediated activities and consequently affect cellular and humoral immunity (Corrier, 1991).

### Management of Mycotoxins

Since the discovery of mycotoxin in 1960s, researchers have been thoroughly researching ways to eliminate or minimize the effects of these contaminants at different stages of agricultural products.

### Pre-Harvest

The most reliable and economic preventive mechanisms are prevention at pre-harvest stage. This implies before fungal infection and before mycotoxin production occurs on plant material. It includes biological control, development of



resistant varieties of crops through new strategies and good agricultural practices, which includes crop rotation, tillage practices, cropping pattern, reduction in plant stress through irrigation, timely planting and harvesting and protection of insect damage by the use of biopesticides (Choudhary and Koumari, 2010).

### Post-Harvest Strategies

Prevention of mycotoxin contamination in the field is the main goal of agricultural food and feed industries, but even the best management of agricultural strategies cannot totally eradicate mycotoxin contamination (Jouany, 2007). Therefore several physical, chemical and biological methods have been developed in order to remove mycotoxins from contaminated feed (Kabak and Dobson, 2009).

Physical methods like cleaning, mechanical sorting and separation, washing, density segregation, thermal inactivation, irradiation, solvent extraction, and chemical procedures like treatment with acid/base solutions or other chemicals, ammoniation and ozonation have been tested (CAST, 2003).

However, these physical and chemical detoxification methods often do not work, and often destroy or remove essential nutrients from the feedstuff, reduce palatability, and not feasible for large scale. Biological detoxification, which comprises binding of mycotoxins by adsorptive materials as well as microbial inactivation by specific micro organisms or enzymes (biotransforming agents), is the most prominent approach to reduce the risk for mycotoxicosis in farm animals (Upadhaya, 2010).

### CONCLUSION

Mycotoxigenic fungi are filamentous fungi which are naturally associated with human foods and animal feeds. Major genera of fungi that produce mycotoxins are *Aspergillus*, *Fusarium* and *Penicillium*. Mycotoxins are toxic secondary metabolites secreted by different molds which are responsible for the cause of several adverse effects on human and animal health. These cause immunotoxicity, hepatotoxicity, nephrotoxicity and genotoxicity on animal and human health. They widespread and their controls are approximately impossible due to their nature.

These mycotoxin producing molds should be managed appropriately to minimize these adverse effects. Keeping food and feed sanitation, use of

good agricultural practices (use of resistant varieties, timely planting and harvesting) and protection of insect damage by the use of biopesticides are management options to minimize the adverse effects of mycotoxigenic fungi.

### REFERENCES

- [1] Ali A, Abdu S. (2011). Antioxidant protection against pathological mycotoxins alterations on proximal tubules in rat kidney. *Functional Foods in Health and Disease*.4:118-34.
- [2] Avantaggiato, G., Solfrizzo, M., and Visconti, A. (2005). Recent advances on the use of adsorbent materials for detoxification of *Fusarium* mycotoxins. *Food Additives and Contaminants*, **22**: 379-388.
- [3] Awad WA, Ghareeb K, Chimidtsere S, Strasser A, Hess M, Böhm J. (2012). Chronic effects of deoxynivalenol on plasma cytokines and vaccine response of broiler chickens. *Proceedings of the 34<sup>th</sup> Mykotoxin-Workshops der Ges; Für Mykotoxin Forschung e.V., Braunschweig, Germany*; 14–16 p. 29.
- [4] Bayman P, Baker J.(2006) Ochratoxins: a global perspective.*Mycopathologia*.162 :215 -23.
- [5] Bennett, J. W., & Bentley, R. (1999). Pride and prejudice: the story of ergot. *Perspectives in biology and medicine*, 42(3), 333-355.
- [6] Bennett, J. W., and Klich, M. (2003). Mycotoxins. *Clinical Microbiology Reviews*, **13**: 497 – 516.
- [7] Blount, W. P. (1961). Turkey X disease. *J. Br. Turk. Fed.*, **49**:52–4.
- [8] Bueno, D., Munoz, R.& Marty, J. L. (2013). Common Methods to Detect Mycotoxins: A Review with Particular Emphasis on Electrochemical Detection.
- [9] CAST. (2003). Mycotoxins: Risks in plant, animal, and human systems. Council of Agricultural Science and Technology (CAST), Task Force Report No. 139. P.199. Ames, Iowa.
- [10] Cerain, A. L. (2007). en: JM Soriano del Castillo (ed.), *Mycotoxinas en alimentos*, Díaz Santos.
- [11] Choudhary, A. K., & Kumari, P. (2010). Management of mycotoxin contamination in preharvest and post harvest crops: present status and future prospects. *Journal of Phytology*.
- [12] Corrier DE.(1991). Mycotoxicosis: mechanisms of immunosuppression. *Vet Immunol Immunopathol* .30:73-87.
- [13] Eriksen, G., Petterson, H., and Lindberg, J. (2003). Absorption, metabolism and excretion of 3-acetyl DON in pigs. *Archiv für Tierernährung*, **57**:335-345.
- [14] Feddern, V., Dors, C., Tavernari, F.C., Mazzuco, H., Cunha, J. R. A., Krabbe, E.L. and Scheuermann, G.N. (2013). Aflatoxins: Recent

- Advances and Future Prospects, Aflatoxins Importance on Animal Nutrition.
- [15] Frisvad JC, Smedsgaard J, Samson RA, Larsen TO, Thrane U (2007) Fumonisin B2 production by *Aspergillus niger*. *J Agric Food Chem* 55(23):9727–9732.
- [16] Gajdusek, D. C. (1953). Acute Infectious Hemorrhagic Fevers and Mycotoxicoses in the Union of Soviet Socialist Republics. Acute Infectious Hemorrhagic Fevers and Mycotoxicoses in the Union of Soviet Socialist Republics.
- [17] Gelderblom WC, Snyman SD, Abel S.(1996). Hepatotoxicity and carcinogenicity of the fumonisins in rats a review regarding mechanistic implications for establishing risk in humans. In: Jackson LS, DeVnes JW, Bullerman LB, eds. *Fumonisin in Food*. New York: Plenum: 251-64.
- [18] Gelderblom WCA, Kriek NPJ, Marasas WFO, Thiel PG (1991). Toxicity and carcinogenicity of the *Fusarium moniliforme* metabolite, fumonisin B1, in rats. *Carcinogenesis* 12:1247–1251.
- [19] Grovey, J.F., (2007). The trichothecenes and their biosynthesis. In *Progress in the Chemistry of Organic Natural Products* pp. 63-130. Springer, Vienna.
- [20] Harrison LR, Colvin BM, Greene JT, Newman LE, Cole JR (1990). Pulmonary edema and hydrothorax in swine produced by fumonisin B1 a toxic metabolite of *Fusarium moniliforme*. *J Vet Diagn Invest* 2:217–221.
- [21] Hussein, H., and Brasel, J. (2001). Toxicity, metabolism, and impact of mycotoxins on humans and animals. *Toxicology*, **167**:101-34.
- [22] Ingelse, B.A., Flieger, M., Claessens, H.A. and Everaerts, F.M., 1996. Ergot alkaloids as chiral selectors in capillary electrophoresis determination of the separation mechanism. *Journal of Chromatography A*, 755(2), pp.251-259.
- [23] International Agency for Research on Cancer (IARC) (2002) IARC monographs on the evaluation of carcinogenic risks to humans. Some traditional herbal medicines, some mycotoxins, naphthalene and styrene. IARC, Lyon, pp 301–366.
- [24] International Agency for Research on Cancer, 2012. A review of human carcinogens: personal habits and indoor combustions (Vol. 100). World Health Organization.
- [25] Islam Z, Gray JS, Pestka JJ.(2006). p38 Mitogen activated protein kinase mediates IL-8 induction by the ribotoxide oxynivalenol in human monocytes. *Toxicol Appl Pharmacol*. 213: 235 -44.
- [26] Javed T, Bennett GA, Richard JL, Dombrink-Kurtzman MA, Côté LM, Buck WB.(1993). Mortality in broiler chicks on feed amended with *Fusarium proliferatum* culture material or with purified fumonisin B1 and moniliformin. *Mycopathologia* .123:171-184.
- [27] Jouany, J. P. (2007). Methods for preventing, decontaminating and minimizing the toxicity of mycotoxins in feeds. *Animal Feed Science and Technology*, 137(3-4), 342-362.
- [28] Kabak, B., & Dobson, A. D. (2009). Biological strategies to counteract the effects of mycotoxins. *Journal of food protection*, 72(9), 2006-2016.
- [29] Kadakal, C., & Nas, S. (2002). Effect of activated charcoal on patulin, fumaric acid and some other properties of apple juice. *Food/Nahrung*, 46(1), 31-33.
- [30] Kensler, W., Roebuck, D., Wogan, N., and Groopman, D. (2011). Aflatoxin: A 50-Year Odyssey of Mechanistic and Translational Toxicology. *Toxicological Sciences*, **120**: 28 – 48.
- [31] Krishnamoorthy P, Vairamuthu S, Balachandran C, Muralimanohar B.( 2007). Pathology of chlorpyrifos and T-2 toxin on broiler chicken. *Veterinarski Arhiv*.77:47-57.
- [32] Liu, X., Luo, X., & Hu, W. (1988). Arthrinium sp. and the deteriorated sugarcane poisoning. In *Mycotoxins and phycotoxins. Abstracts of the Seventh International IUPAC Symposium*, Tokyo pp. 16-19.
- [33] Ludolph, A.C., He, F., Spencer, P.S., Hammerstad, J. and Sabri, M.(1991). 3-Nitropropionic acid-exogenous animal neurotoxin and possible human striatal toxin. *Canadian journal of neurological sciences*, 18(4), pp.492-498.
- [34] Marasas WFO (2001) Discovery and occurrence of the fumonisins. A historical perspective. *Environ Health Perspect* 109[Suppl 2]:239–243.
- [35] Ming, L. (1995). Moldy sugarcane poisoning a case report with a brief review. *Journal of Toxicology: Clinical Toxicology*, 33(4), 363-367.
- [36] Moss, M. O., & Long, M. T. (2002). Fate of patulin in the presence of the yeast *Saccharomyces cerevisiae*. *Food Additives & Contaminants*, 19(4), 387-399.
- [37] Ortatatlı M, Oguz H, Hatipoglu F, Karaman M.(2005). Evaluation of pathological changes in broilers during chronic aflatoxin (50 and 100 ppb) and clinoptilolite exposure. *Res Vet Sci*.78:61-8.
- [38] Oswald IP, Marin DE, Bouhet S, Pinton P, Taranu I, Accensi F.(2005). Immunotoxicological risk of mycotoxins for domestic animals. *Food Add Contam*.22:354-360.
- [39] Pestka JJ. (2008). Mechanisms of deoxynivalenol-induced gene expression and apoptosis. *Food Addit Contam Part A Chem Anal Control Expo Risk Assess*.25:1128-40.
- [40] Pitt, J.I., 2000. Toxigenic fungi and mycotoxins. *British medical bulletin*, 56(1), pp.184-192.
- [41] Radovanovic Z, Jankovic S, Jevremovic I. (1991). Incidence of tumors of urinary organs

- in a focus of Balkan endemic nephropathy. *Kidney Int.*34:S75-6.
- [42] Ramesh, V.B. and Siruguri, V.(2003). Food safety in food security & food trade. *Mycotoxin food safety risk in developing countries: Food Agriculture environ*, 16-20.
- [43] Riley RT. (1998). Mechanistic interactions of mycotoxins: Theoretical considerations. In: Sinha KK, Bhatnagar D, eds. *Mycotoxins in Agriculture and Food Safety*. New York: Marcel Dekker, Inc: 227-53.
- [44] Rios, L.M. and Sahinidis, N.V.(2013). Derivative-free optimization: a review of algorithms and comparison of software implementations. *Journal of Global Optimization*, 56:1247-1293.
- [45] Rustom, I.Y.(1997). Aflatoxin in food and feed: occurrence, legislation and inactivation by physical methods. *Food chemistry*, 59(1), pp. 57-67.
- [46] Sadok, I., Stachniuk, A. and Staniszewska, M. (2019). Developments in the monitoring of patulin in fruits using liquid chromatography: An overview. *Food Analytical Methods*, 12(1), pp.76-93.
- [47] Sehata S, Kiyosawa N, Sakuma K, Ito K, Yamoto T, Teranishi M, et al.(2004). Gene expression profiles in pregnant rats treated with T-2 toxin. *Exp Toxicol Pathol.*55:357-66.
- [48] Sharma RP. (1993). Immunotoxicity of mycotoxins. *Journal of Dairy Science*. 76:892-897.
- [49] Smerak P, Barta I, Polivkova Z, Bartova J, Sedmikova M.(2001). Mutagenic effects of selected trichothecene mycotoxins and their combinations with aflatoxin B1. *Czech J Food Sci.*19:90-6.
- [50] Smoragiewicz, W.Cossette, B., Boutard, A., & Krzystyniak, K. (1993). Trichothecene mycotoxins in the dust of ventilation systems in office buildings. *International archives of occupational and environmental health*, 65(2), 113-117.
- [51] Stojković R, Hult K, Gamulin S, Plestina R.(1984). High affinity binding of ochratoxin A to plasma constituents. *Biochem Int.*9:33- 8.
- [52] Surai PF.(2002). *Natural Antioxidants in Avian Nutrition and Reproduction*. Nottingham, UK: Nottingham University Press.
- [53] Turner PC, Moore SE, Hall AJ, Prentice AM, Wild CP.(2003). Modification of immune function through exposure to dietary aflatoxin in Gambian children. *Environ Health Persp.*111:217-220.
- [54] Upadhaya, B. K., Sharma, A., Khaira, A., Dinda, A. K., Agarwal, S. K., & Tiwari, S. C. (2010). Transient IgA nephropathy with acute kidney injury in a patient with dengue fever. *Saudi Journal of Kidney Diseases and Transplantation*, 21(3), 521.
- [55] Wannemacher, R., Stanley, L., and Wiener, M. (2000). Trichothecenes mycotoxins. In: *Medical aspects of chemical and biological warfare*, pp. 656-676, (Zajtchuk R, ed). Washington DC: Department of the Army.
- [56] World Health Organization. (1990). *Selected mycotoxins: ochratoxins, trichothecenes, ergot*.

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