Scholars Academic Journal of Biosciences
Abbreviated Key Title: Sch Acad J Biosci
ISSN 2347-9515 (Print) | ISSN 2321-6883 (Online)
Journal homepage: https://saspublishers.com
NLM ID:101629416
OPEN ACCESS
Biology

Mycotoxins, Food Contaminations: Effects on Human and Animals Health

Khadija Mousa Abd Al-Jabar[1*], Zainab Falih Dakhil[2], Taif Muthher Muslim[3]

[1]College of Science, Unit of Environment Researches & Prevention of Pollution
[2]Department of Biology, College of Science, Al-Qadisiyah University, Al Diwaniyah Province, Iraq.
[3]Environmental Research and Pollution Prevention Unit, Collage of Science, University of AL- Qadisiyah, Al-Diwaniyah, Iraq

DOI: https://doi.org/10.36347/sajb.2025.v13i07.006 | **Received:** 02.05.2025 | **Accepted:** 13.07.2025 | **Published:** 18.07.2025

***Corresponding author:** Khadija Mousa Abd Al-Jabar
College of Science, Unit of Environment Researches & Prevention of Pollution

Abstract | **Review Article**

Mycotoxins are fungi-produced toxins that can be found virtually wherever on Earth. Not all fungus create mycotoxins, and some species are capable of producing multiple types of toxins. Mycotoxins enter the body either directly or indirectly by the consumption of fungi-contaminated foods, such as meat, grains, fruits, and vegetables. Eating them results in a variety of harmful effects, ranging from acute toxicity to long-term illnesses or chronic diseases. Some of them trigger outbreaks of human poisoning. Aflatoxin, which causes liver cancer, and ochratoxin, which causes cancer and renal illness, are two examples. Furthermore, fumonisin toxins have been linked to poor growth, nervous system malfunction, and esophageal cancer in both humans and animals. While the toxin of deoxynivalenol is known as Vomitoxin, it is one of the most frequent trichochene compounds in this group and a major contamination of corn, wheat, and barley. From a long time ago to the present, we have noted that various significant studies on mycotoxins have been done in order to understand their mechanism of action in animals and manage them. This study looks at some of the most recent research on the biological effects of four forms of mycotoxins that are now relevant and dangerous to humans and animals: deoxynivalenol, fumonisins, aflatoxins, and ochratoxins.

Keywords: Mycotoxin, Food, Human, Animal, Toxicity.

INTRODUCTION

Climate is the primary factor that promotes the proliferation of fungus and consequently the development of mycotoxins [1]. Mycotoxins have a direct influence on food security since they significantly reduce the supply of animal feed and food for humans in underdeveloped countries [2,3]. This suggests that there is a close relationship between climate change and mycotoxins, as climate variations in temperature, humidity, and pressure have a direct impact on the type of mycotoxins produced, their concentration, and the type of effect they have on humans and animals alike [4]. A group of toxins, among the thousands of mycotoxins produced by fungi, are of great importance because of the severe toxic effect they have on living organisms in general and humans in particular, including aflatoxins (AF), ochratoxins (OT), zearalenone (ZEA), deoxynivalenol (DON), and fumonisins (FUMs), which are found in grains consumed locally and globally frequently, such as rice, wheat, and corn, as well as fruits and vegetables. As a result, they are regarded as an unavoidable issue that contributes to the development of a variety of problems, including liver cancer, malformed babies, renal toxins, and other fatal diseases [5]. Recent study has revealed that fungi in tropical and subtropical environments produce aflatoxin-type mycotoxins. As a result, aflatoxin toxins have become more prevalent in moderate-temperature regions. This toxin has gotten a lot of attention since it causes cancer in humans [4, 6, 7, 8, 9, 5]. The ability of fungus to produce fermentation was generally established by the late nineteenth and early twentieth century. Researchers discovered a huge number of toxic secondary metabolites that fungi produce during liquid and solid fermentation. Because some of these substances are ingested by humans, researchers are particularly interested in their toxicity and environmental impact. Mycotoxins, or fungi's secondary metabolites, have been linked to a number of diseases [10]. And According to a study conducted by [11, 12], these poisons are among the chemical substances found in the environment in very small amounts and constitute a significant threat to the ecosystem. However, secondary metabolites from fungus are incredibly significant. Alexander Fleming's

creation of penicillin had a major and positive impact on humanity due to the therapeutic effect that this antibiotic provides for a wide range of terrible human diseases [10,13]. Taif M. Muslim and colleagues discovered that fungi have an important part in industry, agriculture, medicine, and technology, as well as in fermentation [14].

Table 1: Mycotoxins and some of their effects on humans and animals

Mycotoxin	Toxin-producing fungus	Influence or disease	Citation
Deoxynivalenol	*F. graminearum*	It is known as emetic toxin because it causes vomiting.	(15)
Deoxynivalenol	*F. graminearu* *F. culmorum*	Inhibiting RNA and DNA synthesis, the toxin also has a noticeable effect on growth regulators, thus increasing the body's sensitivity to diseases.	(16)
FB1	*F. verticillioides*	Equine leukoencephalomalacia (ELEM)	(17)
FB2	*A.niger*	Esophageal cancer in humans	(18)
Aflatoxin B1	*A.flavus*	Turkey X disease hepatocellular carcinoma (HCC).	(19)
Aflatoxin B1	*A.flavus*	Aspergillosis in humans and animals.	(20)
Ochratoxin A	*A.niger pencillium. sp*	Liver and kidney cancer in humans and mice.	(21)
Ochratoxin A	*A.niger pencillium. sp*	Nephropathy in the Balkans (BEN).	(22)

Properties of mycotoxins

- Fungi produce toxic chemical compounds, primarily cyclic or open-chain hydrocarbons that dissolve effectively in organic solvents [23].
- Most mycotoxins are tasteless and odorless [23].
- Mycotoxins' low molecular weight inhibits the immune system from identifying and managing them, leading to buildup in tissues and organs like the liver, spleen, and kidney [24].
- Due to its great temperature tolerance, it can stay in the soil for a long time before spreading to crops, animals, and humans. Its durability extends to high cooking temperatures, raising concerns about mycotoxins and their health implications; it also withstands cold temperatures up to freezing [24].
- The chemical composition of mycotoxins varies, leading to different biological effects. Some of them can depress the immune system, while others can induce neurological or tissue damage [23].
- Mycotoxins enter the body through three routes: eating food infected with toxin-producing fungi, inhaling fungi and spores, or coming into close touch with them [25].
- Resistant to the breakdown processes that occur during digestion and absorption in the digestive tract [23].
- non-antigenic substances, therefore the body cannot produce antibodies against them [25].
- Not all secondary metabolites of fungi are toxic (mycotoxins). Rather, secondary metabolites of fungus are used as therapies, albeit in small quantities. Penicillin is an example of this; it is currently widely used as a treatment and is produced as a secondary metabolite by the Penicillium fungus [26].

Mycotoxins

Toxic fungi create secondary metabolites known as mycotoxins. Most fungi can create multiple forms of toxin at the same time, although not all fungus can make mycotoxins [27]. A vast group of fungi create mycotoxins, the most prominent of which are Aflatoxin, Rubratoxins, Citrinin, Fumonisins, Patulin, Ochratoxin, and Alteratoxin (AtxII & I) [28]. Mycotoxins are a chemically varied class of low-molecular-weight secondary metabolites produced by fungal molds that are generally hazardous to the environment [29,20]. Many mycotoxins enter our bodies through contaminated meals or by inhaling poisonous fungus spores. They can also enter the human body through ingesting animal products that have previously consumed mycotoxin-contaminated feed [30]. Mycotoxins may be created by fungi as a defense mechanism against competing fungal species, certain bacteria, harsh environmental conditions such as drought and extreme cold, or a lack of nutrients required for the fungus' survival [31, 32]. Because of their consequences on human health, it is increasingly critical to investigate the presence of mycotoxins in food and beverages [33]. Fumonisin, ochratoxin, zearalenone, and aflatoxin are some of the approximately 400 mycotoxins found. They are subject to international control because of the harm they do to humans and animals. [34]. Animals and humans are directly exposed to mycotoxins when they ingest food contaminated with toxin-producing fungi. These toxins target living cells during division, including bone marrow, testicles, and spleen cells. T-2 toxin, the most severe and harmful mycotoxin, produces Alimentary toxic Aleukia illness, which weakens the organism's immune system and has a more severe effect on bone marrow. Vomitoxin 'Don' toxin also suppresses the immune system, although its effects are less severe than those of T-2, particularly when it

comes to the body's resistance to infections caused by the following bacterial species: *Compylobacter spp, Listeria spp*, and *Salmonella spp* [35].

Understandably, this has been the subject of extensive inquiry. Four kinds of mycotoxins have different toxicity for humans and animals.

Deoxynivalenol (DON)

According to a 1989 study conducted in Iraq by [15], the poison is mostly produced by the *F. compactum* and *F. verticilliodes* fungi, which have been found in both imported and local wheat plants. This species of fungus attacks wheat plants, resulting in wheat blight. In 1990, [36] confirmed a direct link between wheat blight and the presence of 'DON' toxins. What adds to the fear of grains becoming infected with this type of toxin-producing fungus is that the grains appear healthy on the outside, but when examined for the presence of toxins using one of the methods used to detect the presence of mycotoxins used by [37] in 2000, it was discovered that the grains, which appear healthy on the outside, contain high levels of toxins. Toxin detection techniques include GC, TLC, ELISA, HPLC, HPTLC, UV, and Spectrophotometer. The fungi that create this sort of toxin attack grains in the field, and it is a field fungus, which means it does not survive storage or export [15]. This form of mycotoxin, also known as Vomitoxin, is one of the most frequent trichotoxin compounds in this group and the primary contaminate of corn, wheat, and barley [38, 39]. Morooka and his colleagues discovered this toxin for the first time in 1972 in Japan as a secondary metabolite of the fungus *F. graminearum* isolated from barley. Vesonder termed this toxin vomitoxin in 1973 because to its emetic characteristics [15]. When a poison-containing food is exposed to high heat, the toxic substance is reduced by 12%; when exposed to both high pressure and heat, the toxin is reduced by 26%. This shows that the toxin can withstand high heat and pressures while cooking [40]. As a result of these features, DON is extremely harmful and has a substantial impact on human health, as proved by [41]; this toxin causes serious illnesses in people such as stomach cancer, liver cancer, esophageal cancer, and arthritis. [16] in 1996 that the harmful effect of DNA is a decrease in the quantity of ribosomes, which inhibits protein synthesis of DNA and RNA, increasing the body's susceptibility to disease. Don toxic has a low molecular weight and is made up of white chemical crystals in its solid state. It is water-soluble and dissolves in polar solvents such as ethyl acetate and ethanol. It is particularly resistant to intense heat [42].

Chemically, Don toxic has its origins in the type B group of trichochenes. It consists of an epoxy group between carbon atoms 12-13 and a trichochene ring. It also contains a carbonyl group on carbon atom 8. It contains two secondary hydroxide groups and one primary hydroxide group [43, 44].

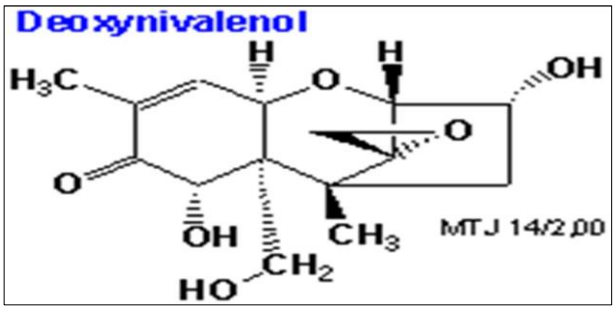


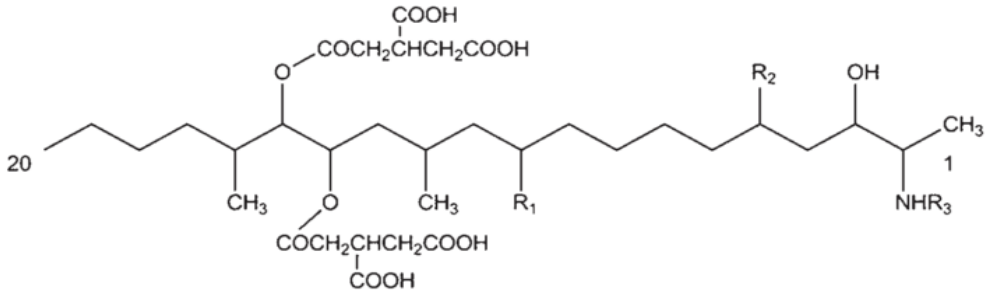
Fig. 1: Chemical structure of deoxnivalenol (44)

FUMONISINS

It is one of the most dangerous types of mycotoxins produced by the sorts of fungi that cause infections. The fungi that create these toxins are some species of Fusarium fungus, including *F.verticillioides*, *F.porliferaum*, this toxin is also produced by the fungus *Aspergillus niger*, which grows on corn and peanuts [45,46]. The Fusarium fungus, the primary cause of this toxin, was found for the first time by the scientist Link in 1909, and it causes a variety of diseases in humans, animals, and plants. Fs are classified into four kinds: A, B, C, and D, with over 15 other types [17]. Furthermore, FB1 is categorized into several types, including FB1, FB2, and FB3. The most virulent and very deadly fumonisin is FB1 [51,17]. The collection of symptoms that develop when exposed to mycotoxins is called 'Mycotoxicosis'. Mycotoxicosis symptoms in the digestive tract include vomiting, diarrhea, and abdominal discomfort, which are regarded main symptoms of fusarium toxin and aflatoxin [52,53]. Furthermore, fumonisin toxins are known to cause poor growth, nervous system dysfunction, and esophageal cancer in humans and animals [54]. FB1 is the most hazardous and most ubiquitous of the kinds of FB1, causing toxic consequences in humans and other animals alike, including neurotoxicity, malignancies, and hepatotoxicity [55]. The work conducted by [56], which discovered that FB1 produces toxicity in human esophageal epithelial cell lines, provides evidence that fumonisins are hazardous in the development of esophageal cancer. This was demonstrated in a study conducted by [57], who discovered a link between FB1

consumption and esophageal cancer. These toxins dissolve in aqueous solutions such as methanol and polar solvents such as water, and they are classified as temperature-stable chemical compounds when moisture is not present [58]. Fs are polyketides, the most abundant secondary metabolites identified in fungi and bacteria, exhibiting their varied composition [59,60].

Chemically, Fumonisin does not have a chemical ring or cyclic structure, unlike most mycotoxins but it has 19-20 carbon aminopolyhydroxyalkyl chain which decomposes with propane-1,2,3- tricarboxylic acid groups [61, 62].



	FA_1	FA_2	FB_1	FB_2	FB_3	FB_4	FC_1
R_1	OH	H	OH	H	OH	H	OH
R_2	OH	OH	OH	OH	H	H	OH
R_3	CH_2CO	CH_2CO	H	H	H	H	H

Fig. 2: Chemical structure of fumonisins [51]

Aflatoxin B1

Aflatoxins are biologically active secondary metabolites. This type of toxin is produced by Aspergillus fungus strains, mainly *Aspergillus flavus* and *Aspergillus parasiticus*. These fungi are characterized by being widely distributed in nature and capable of infecting many types of agricultural crops, which in turn become contaminated with mycotoxins under certain environmental conditions, when humans and animals consume aflatoxins-contaminated food and feed, they can develop severe liver poisoning, immune system poisoning, and birth abnormalities [63]. AFB1 is one of the chemical forms of aflatoxin. It is the most abundant and most toxic. It causes genetic mutations and is a toxic substance that is carcinogenic to humans, Hepatotoxicity in humans is associated with aflatoxin [63,64]. From an economic standpoint, approximately 25% of agricultural crops are infected with aflatoxins, causing significant economic losses [65]. When exposed to aflatoxins, two sorts of diseases develop: acute poisoning, which kills, and chronic poisoning, which causes cancer and primarily affects the liver (20). There is also some evidence that aflatoxin exposure increases the risk of developing lung and other malignancies [63]. Few fungus have had such a broad economic influence as *Aspergillus flavus*. It is a disease of plants, animals, and insects, causing storage rots in many crops and producing AFB1, a highly controlled mycotoxin. Aspergillus species have grown in importance as human diseases, owing to the fact that immunocompromised people are particularly vulnerable to these fungi. Only *A. fumigatus* is more significant than *A. flavus* among aspergilli that cause mycoses in humans [66]. AFB1 is a highly potent naturally occurring carcinogen. It is one of a few mycotoxins designed for use as a biological weapon (20). *A. flavus* is a mold that causes bronchopulmonary aspergillosis. It is an insect pathogen that affects a variety of species, such as honeybees, where it produces stonebrood [67, 68, 69]. Aflatoxin's economic costs are difficult to measure because overall estimates must encompass losses such as delayed weight increase and immunological suppression in farm animals, as well as the death of companion animals due to aflatoxicosis [70].

Chemically, the aflatoxins are difuran coumarin metabolites synthesized via a polyketide route. There are four primary aflatoxins: B1, B2, G1, and G2, where the letters represent the color of their fluorescence under UV light (blue or green) and the numbers represent their relative migration distance on a thin-layer chromatographic plate. There are also other, less prevalent aflatoxins. AFB1 is the most potent naturally occurring carcinogen [71], and when the term aflatoxin

is used in the singular, the author usually refers to aflatoxin B1.

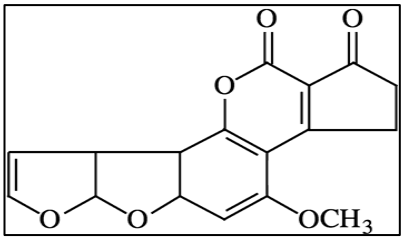


Fig. 3: Chemical structure of Aflatoxin 'B1' [72]

Ochratoxin A

Ochratoxin is a mycotoxin resulting from the secondary metabolism of a group of filamentous fungi belonging to the genera *Aspergillus* and *Penicillium* [73,74,75,76]. Several forms of ochratoxins exist naturally, including ochratoxin A, ochratoxin B 'dechlorinated OTA', and ochratoxin C 'ethylated OTA', and they are frequently co-produced. The most common toxin is ochratoxin A, which the International Agency for Research on Cancer classifies as a group 2B probable human carcinogen [77]. Ochratoxin was associated with human and animal diseases in the early seventies of the twentieth century. Most studies indicate that ochratoxin is mainly prevalent in the countries of northeastern Europe (Serbia, Bulgaria, Croatia), and Africa, such as South Africa, Tunisia, Morocco, Congo and Egypt [78,79,80,81,82,83,84,85,86]. Ochratoxin is a common contaminant found in coffee, grains, spices, grape juice, wine, beer, and cocoa products. It has a number of negative effects in humans and animals, including nephrotoxicity [87]. It has also been found to cause liver and kidney tumors in mice [21]. In addition to rats [88]. There are also other minor adverse consequences, such as liver toxicity [89]. Congenital malformations [90]. Cancerous illness [91]. It also lowers immunity (92). This suggests that Ochratoxin A is accountable for the kidney illness in the Balkans 'Balkan Endemic Nephropathy'; also, urinary tract malignancies in humans are linked to this toxin [22]. Based on a study conducted by [93] where they measured the concentration of ochratoxin A in the blood of people suffering from acute kidney disease and compared these concentrations with the concentrations of OTA in healthy individuals, they noticed that there was no significant difference between the concentration of OTA in healthy people and that found in people with kidney disease, as their serum showed an increase in the concentration of A by 1.5 nmol /L compared to what is found in healthy people ,This study reveals that when the blood concentration of OTA in patients with nephritis is 1.5 nmol/L higher than in healthy people, it may serve as a biomarker for the disease. According to a study conducted by [94] in 1999, when OTA was administered to rats during pregnancy, the toxin produced central nervous system abnormalities. Similarly, [95] demonstrated in 2001 that OTA toxin might be deemed a chemical responsible for several brain diseases. As a result, OTA might be called a highly hazardous toxin that can reach the neurological tissue (retina - brain) [96]. [97,98,99,100] mentioned in a study they conducted on laboratory mice, where a dose of poison was given to the mice during pregnancy, and then they noticed a deformity in the formation of the fetuses before birth as a result of the accumulation of poison in the tissues of the fetuses after crossing the placenta, thus causing morphological deformities in the fetuses of mice, and in the same manner in chickens [101] and hamsters. [102].

Chemically, Ochratoxin 'A' (OTA) is 7 - carboxyl -5- chloro-8-hydroxyl-3,4-dihydro-3-R-methylisocomarin-7- L-phenylalanine [103].

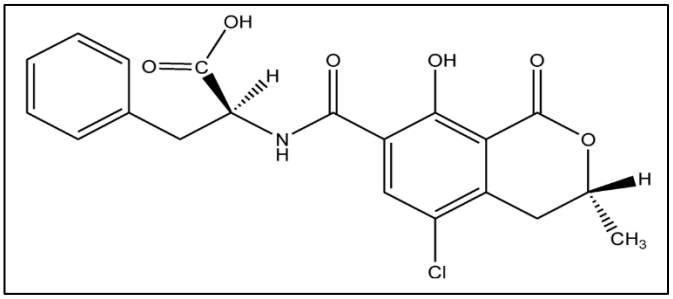


Fig.4: Chemical structure of ochratoxin' A'(103)

CONCLUSIONS

Although there is a wealth of information available that demonstrates the toxic effects of many mycotoxins produced by fungi, future research topics must continue to address this topic and its importance to human health. Continued research will contribute to the

discovery of modern, fast, simple, less expensive, and more accurate methods through which we can detect mycotoxins before contaminated foods and foodstuffs reach the market. It is necessary to identify the danger posed by mycotoxins to humans when they consume foods contaminated with toxins. Through the study conducted in this review, we noticed that the vast majority of secondary metabolites of fungi have a negative effect on humans, animals and plants. This effect can either be observed directly after eating foods contaminated with mycotoxins, or it is a cumulative effect, where symptoms appear after several years, which is the most dangerous.

There are some secondary metabolites of fungi, but on a limited scale, that have a positive effect, including penicillin, which is used in treatment. There are also some types of fungi that help repair and improve soil productivity. In addition, aflatoxin, which is considered the most powerful and severe carcinogenic substance, has been biologically developed to be used as a weapon.

RECOMMENDATIONS

Establishing inspection centers for local crop grains, fruits, and vegetables, as well as conducting periodic inspections of grains in laboratories, warehouses, and shops to ensure they are free of contaminated fungi and mycotoxins, and ensuring the cleanliness of buildings and health facilities from fungal contamination. Raising community awareness about not eating food that has been improperly stored and contaminated with fungi, while taking into account the laws governing permissible levels of contamination and toxicity, as well as using types of fungi (Mushroom) as a food substance due to their role in preventing fungi growth and their nutritional and health benefits.

Abbreviation key: DON= deoxynivalenol, FB1= fumonisinB1, F= fumonisin, AFB1= aflatoxinB1, OTA= ochratoxin A.

REFERENCES

1. Magan, N.; Medina, A. Integrating gene expression, ecology and mycotoxin production by *Fusarium* and *Aspergillus* species in relation to interacting environmental factors. *World Mycotoxin J.* 2016, *9*, 673–684.
2. Miraglia, M.; Marvin, H.J.P.; Kleter, G.A.; Battilani, P.; Brera, C.; Coni, E.; Cubadda, F.; Croci, L.; De Santis, B.; Dekkers, S.; et al. Climate change and food safety: An emerging issue with special focus on Europe. *Food Chem. Toxicol.* 2009, *47*, 1009–1021.
3. Medina, A.; Rodriguez, A.; Magan, N. Effect of climate change on Aspergillus flavus and aflatoxin B1 production. *Front. Microbiol.* 2014, *5*, 348.
4. Milani, J.M. Ecological conditions affecting mycotoxin production in cereals: A review. *Vet. Med. Praha* 2013, *58*, 405–411.
5. Rafael,V.-Q., Juana,S.-A. Environment Changes, Aflatoxins, and Health Issues, a Review . Int. J. Environ. Res. Public Health 2020, 17(21), 7850.
6. Jakšić, D.; Kocsubé, S.; Bencsik, O.; Kecskeméti, A.; Szekeres, A.; Jelić, D.; Kopjar, N.; Vágvölgyi, C.; Varga, J.; Šegvić Klarić, M. Aflatoxin production and in vitro toxicity of Aspergilli section Flavi isolated from air samples collected from different environments. Mycotoxin Res. 2019, 35, 217–230.
7. Dobolyi, C.; Sebok, F.; Varga, J.; Kocsubé, S.; Szigeti, G.; Baranyi, N.; Szécsi, A.; Tóth, B.; Varga, M.; Kriszt, B.; et al. Occurrence of aflatoxin producing Aspergillus flavus isolates in maize kernel in Hungary. Acta Aliment. 2013, 42, 451–459.
8. Varga, J.; Kerepes, L.; Kocsubé, S.; Szigeti, G.; Baranyi, N.; Vágvölgyi, C. Examination of the diversity of indoor molds in a hungarian student hostel. Acta Biol. Szeged. 2013, 57, 51–54.
9. Baranyi, N.; Kocsubé, S.; Varga, J. Aflatoxins: Climate change and biodegradation. *Curr. Opin. Food Sci.* 2015, *5*, 60–66.
10. John ,L.R.,2007,Some major mycotoxins and their mycotoxicosis-An overview.International Journal of food Microbiology 199 (2007) PP. 3-10 .
11. Wanda E, Nyoni H, Mamba B, Msagati T (2017) Occurrence of emerging micropollutants in water systems in Gauteng, Mpumalanga, and North West Provinces, South Africa. Int. J. Environ.Res. public. health 14:79S .
12. Bunke D, Moritz, S, Brack W, Herráez, D.L, Posthuma, L, Nuss, M (2019) Developments insociety and implications for emerging pollutants in the aquatic environment. Environ. Sci . Europe 31:32 .
13. Skinner, C.E., Emmons, C.W., Tsuchiya, H.M., 1947. Fungus diseases of manand animals—general considerations, Henrici's Molds, Yeasts and Actinomycetes.Second ed. John Wiley and Sons Inc., New York, pp. 119–140.
14. Taif,M.M., Khetam,I.M., Mahmood, H.A.-S.(2024) Active Contribution of Soil Fungi to Sustainable Development: A review . Journal of Bioscience and Applied Research, 2024, Vol.10, No. 4, P.891-902.
15. AbdAl- Razzaq, Amani Abdel Wahab (1989). Fungal content, and the extent of the emetic toxin Deoxynivalenol In local and imported wheat. Master's thesis. Department of Life Sciences – College of Science - University of Baghdad .
16. Rotter,B.A., D.B. Prelusky, J.J. Pestka . (1996). Toxicology of Deoxynivalenol (vomitoxin). J. Toxicol Environ Health .48:1-34.
17. Marasas W. F. O. (2001). Discovery and occurrence of the fumonisins: ahistorical perspective, Environ. Health Perspect., 109 (Suppl. 2), 239 43.

18. Barry , J . ; Robert , W. and Mechelle , M.(2007).stored Green. Toxigenic Fungi .,55:23-28.
19. Peraica, M., Radic, B., Lucić, A., & Pavlovic, M. (1999). Toxic effects of mycotoxins in humans. Bulletin of the World Health Organization, 77(9), 754.
20. Bennett, J.W. and Klich, M.A. (2003) Mycotoxins. Clin. Microbiol. Rev. 16, 497–516.
21. Kanizawa, M.; Suzuki, S. Induction of renal and hepatic tumors in mice by ochratoxin A, a mycotoxin. Gann, 1978, 69(4), 599-600.
22. Vukelic, M.; Sostaric, B.; Belicza, M. Pathomorphology of Balkan endemic nephropathy. Food Chem. Toxicol., 1992, 30, 193-200.
23. Fung , F and R . Clark . 2004 . Health effects of my cotoxius toxicological overview Jousnal of toxicology , 42:217 – 243.
24. Al-Jumaili, Sami Abdul-Ridha Ali. (2014). Mycotoxins. Dar Al-Kutub for Printing and Publishing, Iraq, p. 32.
25. Nakheelan, Abdul Aziz Majeed. (2011). Mycotoxins. Dar Dijlah, Amman, Jordan, 1st ed., p. 32.
26. Sun, L.-H., Zhang, N.-Y., Zhu, M.-K., Zhao, L., Zhou, J.-C., & Qi, D.-S. (2015). Prevention of aflatoxin B1 hepatoxicity by dietary selenium is associated with inhibition of cytochrome P450 isozymes and up-regulation of 6 selenoprotein genes in chick liver. The Journal of Nutrition, 146(4), 655–661.
27. Liew, W.-P.-P., & Mohd-Redzwan, S. (2018). Mycotoxin: its impact on gut health and microbiota. Frontiers in Cellular and Infection Microbiology, 8, 60.
28. Abd AL-Hamid, Muhammad. (2000) Fungi and mycotoxins. Universities Publishing House -Egypt.
29. Bennett, J. W. (1987). Mycotoxins, mycotoxicoses, mycotoxicology and Mycopathologia. Springer.
30. Ghasaq Al-Barqawi.(2022). Efficiency of Pleurotus ostreatus and bicarbonate Sodium inhibition in the Monascus purpureus production Tioxins isolated from some dried foods.
31. Bacon, C. W.; Glenn; A. E. and Yates, I. E. (2008). Fusarium verticillioides: Managing the endophytic association with maize for reduced fumonisins accumulation. Toxin Rev., 27: 411-446.
32. Ramesh, G. (2012). Symptoms, Diagnosis, and Pathophysiology of Mycotoxin Exposure. Murray State University, Hopkinsville, Kentucky. USA.
33. Heshmati, A., Zohrevand, T., Khaneghah, A. M., Nejad, A. S. M., & Sant'Ana, A. S. (2017). Co-occurrence of aflatoxins and ochratoxin A indried fruits in Iran: Dietary exposure risk assessment. Food and Chemical Toxicology, 106, 202–208.
34. Ediage, E. N., Van Poucke, C., & De Saeger, S. (2015). A multi-analyte LC--MS/MS method for the analysis of 23 mycotoxins in different *sorghum* varieties: The forgotten sample matrix. Food Chemistry, 177, 397–404.
35. Moss, M.O. (2002). Mycotoxin Review- 2. Fusarium. Mycologist. 16 part. P:45-70.
36. AbdAl- Razzaq, Amani Abdel Wahab (1989). Fungal content, and the extent of the emetic toxin Deoxynivalenol In local and imported wheat. Master's thesis. Department of Life Sciences – College of Science - University of Baghdad .
37. Snijders, C.H. and J. Perkowski.(1990). Effects of head blight causedby Fusariumculmorum on toxin content and weight of kernels. Phytopathology. 80: 566–570.
38. Ostry, V. and j. skarkova. (2000). Development of an HPLC method forthe determen of Deoxynivalenol in cereal products. JPC. J. of planar chromatography Modem. TLC. 13 : 443-446.
39. Deijns, A.J., H.P. Egmond, G.A.J. Speijers, andH.V.Loveren.(1997).Immunotoxicity van natuurlijketoxinen .Eenliteratuuroverzicht. RijksInstituutvoor Volksgezondheid en Milieu, Bilthoven. pp. 16-17.
40. Krska, R. and R.Josephs . (2001). The state of the art in the analysis ofestro-genic mycotoxin in cereals. Fresenius journal of Analytical chem.369:469-476.
41. Wolf-Hall, C.E., A.H. Milford, and L.B. Bullerman. (1999). Stability of Deoxynivalenol in heat-treated foods. J. Food Prot. 62: 962–96 .
42. Wild, C.P. and A.J. Hall.(1996). Epidemiology of Mycotoxin-Related disease.The Mycota .VI., Berlin: Springer. pp 213-225 .
43. Eriksend, G.S and J.Alexander. (1998). Fusarium toxins in cereals – ariassessment. Nordic Council of Ministers; pp 502.
44. Hadeel,A.A., (2015) Contamination of maize and wheat grain name Deoxynivalen (DON) in the provinces of Karbala and Babylon .
45. Rheeder, J. P.; Marasas W. F. O. and Vismer H. F. (2002). Production of fumonisin analogs by *Fusarium* species, Appl. Environ. Microbiol., 68 2101–2105.
46. Kumar, V.; Basu, M. S. and Rajendran, T. P. (2008). and mycroflora in some
47. commercially important agricultural commodities Crop Protection, 27: 891-905.
48. Mirocha, C. J.; Abbas, H.K.; Windels, C. E. and Xie, W. (1989). Variation in deoxynivalenol, 15-acetyldeoxynivalenol, 3-acetyldeoxynivalenol, and zearalenone production by *Fusarium graminearum* isolates. Appl. Environ.Microbiol. 55, 1315-1316.
49. Ritieni, A.; Fogliano, V.; Randozzo, G.; Scarallo, A.; Logrieco, A.;Moretti, A.; Mannina, L. and Bottalico, A. (1995) . Isolation and characterization of fusaproliferin, a new toxic metabolite from *Fusarium proliferatum* . Nat. Toxins 3: 17-20
50. Leslie, J. F. ; Marasas, W. F. O. ; Shephard, G. S. ; Sydenham, E. W. ;Stockenstrm, S and Thiel, P. G. (1996). Duckling toxicity and the production of fumonisin and moniliformin by isolates in the A and F mating populations of Gibberella fujikuroi (*Fusarium moniliforme*) Appliad Environmental Microbiology 62: 1182-1187.

51. Logrieco, A. ; Moretti, A. ; Castella, G. ; Kostecki, M. ; Golinski, P.Ritieni, and Chelkowski, J. (1998). Beauvericin production by *Fusarium* species. Appl. Environ Microbiol. 64, 3084-3088.
52. Domijan, A. M. (2012). Neurotoxicity of fumonisin B1. Arh. Hig. Rada.Toksikol., 63:531-544.د
53. Liu, C., Yu, H., Zhang, Y., Li, D., Xing, X., Chen, L., Zeng, X., Xu, D., Fan, Q., Xiao, Y., & others. (2015). Upregulation of miR-34a-5p antagonizes AFB1-induced genotoxicity in F344 rat liver. Toxicon, 106,46-56.
54. Sava, V., Reunova, O., Velasquez, A., Harbison, R., & Sanchez-Ramos, J. (2006). Acute neurotoxic effects of the fungal metabolite ochratoxin-A. Neurotoxicology, 27(1), 82–92.
55. Chen, C., Riley, R. T., & Wu, F. (2018). Dietary fumonisin and growth impairment in children and animals: A review. Comprehensive Reviews in Food Science and Food Safety, 17(6), 1448–1464.
56. Regnier, M., Polizzi, A., Lukowicz, C., Smati, S., Lasserre, F., Lippi, Y., Naylies, C., Laffitte, J., Bétoulières, C., Montagner, A., & others. (2019). The protective role of liver X receptor (LXR) during fumonisin B1-induced hepatotoxicity. Archives of Toxicology, 93(2), 505–517.
57. Myburg R. B.; Dutton M. F. and Chuturgoon A. A. (2002). Cytotoxicity of fumonisin B1, diethylnitrosamine, and catechol on the SNO esophageal cancer cell line, Environ. Health Perspect., 110, 813–815.
58. Zain, M. E. (2011). Impact of mycotoxins on humans and animals. J. Saudi Chem. Soc., 15: 129-144.
59. Dnpuy, J. P.; Bars.;L. Boudra.; H.; and Bars.; J.L. (1993). Thermostability of fumonisin B(in f1),amycotoxin form Fusarium moniliforme In corn. Appl.Enviro.Microbi. 59(9):2864-2867.
60. Scott, P M. (2012). Recent research on fumonisins: a review. Food Additives \& Contaminants: Part A, 29(2), 242–248.
61. Cox, R. J. (2007). Polyketides, proteins and genes in fungi: programmed nano-machines begin to reveal their secrets. Organic \& Biomolecular Chemistry, 5(13), 2010–2026.
62. Bezuidenhout S. C. ; Gelderblom W. C. A. ; Gorst-Allman C. P. ; Horak R. M. ; Marasas W. F. O. ; Spiteller G. and Vleggaar R. (1988). Structure elucidation of the fumonisins, mycotoxins from *Fusarium moniliforme*, J. Chem. Soc., Chem. Commun., 743-5.
63. Seo J-A and Lee Y-W. (1999). Natural occurrence of the C series of fumonisins in moldy corn, Appl. Environ. Microbiol., 65, 1331–1334.
64. Dvorackova, 1. (1990). Aflatoxins and Human Health. Boca Raton, FL: CRC Press.
65. Eaton, D. L. & Gallagher, E. P. (1994). Mechanisms of aflatoxin carcinogenesis. Annu Rev Pbarmacol Toxicol34, 135-1 72.
66. CAST (1 989). Council for Agricultural Science and Technology. Mycotoxins **:**Economic and Health Risks. Report 11 6.
67. Stevens, D.A., Kan, V.L., Judson, M.A., Morrison, V.A., Dummer, S., Denning, D.W., Bennett, J.E., Walsh, T.J., Patterson, T.F. and Pankey, G.A. (2000) Practice guidelines for diseases caused by Aspergillus. Clin. Infect. Dis. 30, 696–709.
68. Gilliam, M. and Vandenberg, J.D. (1990) Fungi. In: Honey Bee Pests, Predators and Diseases, 2nd edn. (R.A. Morse and R. Nowogrodzki, eds), pp. 64–90. Ithaca NY, Cornell University Press.
69. Scully, L.R. and Bidochka, M.J. (2005) Serial passage of the opportunistic pathogen Aspergillus flavus through an insect host yields decreased saprobic capacity. Can. J. Microbiol. 51, 185–189.
70. Wicklow, D.T. (1990) Adaptation in Aspergillus flavus. Trans. Mycol. Soc. Japan, 31, 511–523.
71. Robens, J. and Cardwell, K.F. (2005) The costs of mycotoxin management in the United States. In: Aflatoxin and Food Safety (H.K. Abbas, ed.), pp. 1–12. Boca Raton, FL: Taylor & Francis.
72. Squire, R.A. (1981) Rating animal carcinogens: a proposed regulatory approach.
73. KLICH, M. A. (2007). Aspergillus flavus: the major producer of aflatoxin. Molecular plant pathology, 713-722.
74. Keeper-Goodman, T.; Scott, P.M. Risk assessment of the mycotoxin ochratoxin. Biomed. Environ. Sci. 1989, 2, 179–248. [Google Scholar] [PubMed]
75. Miller, J.D.; Trenholm, H.L. Mycotoxins in Grain: Compounds Other than Aflatoxin; Eagan Press: St. Paul, MN, USA, 1994. [Google Scholar]
76. Bredenkamp, M.W.; Dillen, J.L.M.; Van Rooyen, P.H.; Steyn, P.S. Crystal structures and conformational analysis of ochratoxin A and B: Probing the chemical structure causing toxicity. J. Chem. Soc. 1989, 2, 1835–1839. [Google Scholar]
77. Budavari, S. The Merck Index, 11th; Rahway, N.J., Ed.; Merck & Co.: Rahway, NJ, USA, 1989; pp. 30–31. [Google Scholar].
78. International Agency for Research on Cancer. Some Naturally Occurring Substances: Some Food Items and Constituents, Heterocyclic Aromatic Amines and Mycotoxins. In IARC Monographs on the Evaluation of Carcinogenic Risks to Humans; IARC: Lyon, France, 1993; Volume 56. [Google Scholar].
79. Radic, B.; Fuchs, R.; Peraica, M.; Lucic, A. Ochratoxin A in human sera in the area with endemic nephropathy in Croatia. Toxicol. Lett. 1997, 91, 105–109. [Google Scholar]
80. Vrabcheva, T.; Usleber, E.; Dietrich, R.; Märtlbauer, E. Co-occurrence of ochratoxin A and citrinin in cereals from Bulgarian villages with a history of Balkan endemic nephropathy. J. Agric. Food Chem. 2000, 48 (6), 2483–2488. [Google Scholar] [PubMed].
81. Shephard, S.; Fabiani, A.; Stockenström, S.; Mshicileli, N.; Sewram, V. Quantitation of

ochratoxin A in South African wines. J. Agric. Food Chem. 2003, 51 (4), 1102–1106. [Google Scholar] [CrossRef] [PubMed].
82. Romani, S.; Sacchetti, G.; López, C.C.; Pinnavaia, G.G.; Dalla Rosa, M. Screening on the occurrence of ochratoxin A in green coffee beans of different origins and types. J. Agric. Food Chem. 2000, 48 (8), 3616–3619. [Google Scholar] [CrossRef] [PubMed]
83. Abouzied, M.M.; Horvath, A.D.; Podlesny, P.M.; Regina, N.P.; Metodiev, V.D.; Kamenova-Tozeva, R.M.; Niagolova, N.D.; Stein, A.D.; Petropoulos, E.A.; Ganev, V.S. Ochratoxin A concentrations in food and feed from a region with Balkan Endemic Nephropathy. Food Addit. Contam. 2002, 19 (8), 755–764. [Google Scholar] [CrossRef] [PubMed]
84. Pfohl-Leszkowicz, A.; Manderville, R. Review on Ochratoxin A: an overview on toxicity and carcinogenicity in animals and humans. Mol. Nutr. Food Res. 2007, 51, 61–99. [Google Scholar]
85. Mally, A.; Hard, G.C.; Dekant, W. Ochratoxin A as a potential etiologic factor in endemic nephropathy: lessons from toxicity studies in rats. Food Chem. Toxicol. 2007, 45 (11), 2254–2260.
86. Krogh, P. Mycotoxic porcine nephropathy–a possible model for Balkan (endemic) nephropathy. In Endemic Nephropathy, Proceedings of the Second International Symposium on Endemic Nephropathy; Bulgarian Academy of Sciences: Sofia, Bulgaria, 1972; pp. 266–277.
87. Chernozemsky, I.N.; Stoyanov, I.S.; Petkova-Bocharova, T.K.; Nikolov, I.G.; Draganov, I.V.; Stoichev, I.T.; Tanchev, Y.; Naidenov, D.; Kalcheva, N.D. Geographic correlation between the occurrence of endemic nephropathy and urinary tract tumours in Vratza district Bulgaria. Int. J. Cancer 1977, 19, 1–11. [Google Scholar]
88. Krogh, P. Role of chratoxin in disease causation. Food Chem**.** Toxicol., 1992, 30(2), 213-224.
89. Bendele, A.M.; Carlton, W.W.; Krogh, P.; Lillehoj, E.B. Ochartoxin.A carcinogenesis in the C57BL/6JxC3HF1 mouse. J. Natl**.**Cancer I., 1985, 75(4), 733-742.
90. Chopra, M.; Link, P.; Michels, C.; Schrenk, D. Characterization ofochratoxin A-induced apoptosis in primary rat hepatocytes. Cell Biol. Toxicol., 2010, 26, 239-254.
91. Mayura, K.; Reddy, R.V.; Hayes, A.W.; Berndt, W.O. Teratogenic and toxic effects of ochratoxin A in rats. Toxicol. Appl. Pharmacol**,.**1976, 37(2), 331-338.
92. Haubeck, H.D.; Lorkowski, G.; Kolsch, E.; Roschenthaler, R.Immunosuppression by ochratoxin A and its prevention by phenylalanine.Appl. Environ. Microb., 1981, 41(4), 1010-1042.
93. Schwerdt, G.; Schuster, C.; Silbernagl, S.; Gareis, M.; Gekle, M. Preliminary results on Ochratoxin A concentrations in blood of patients with various kidney diseases in Germany. Mycotoxin Res. 2001, 17 (2), 146–149. [Google Scholar] [CrossRef]
94. Pfohl-Leszkowicz, A.; et Castegnaro, M. L'Ochratoxine A dans: Mycotoxines: Evaluation et gestion du risqué, Lavoisier; Tec & Doc: Paris, France, 1999; pp. 249–278. [Google Scholar]
95. Soleas, G.J.; Yan, J.; Goldberg, D.M. Assay of ochratoxin A in wine and beer by high-pressure liquid chromatography photodiode array and gas chromatography mass selective detection. J. Agric. Food Chem. 2001, 49, 2733–2740.
96. Brown, M.H.; Szczech, G.M.; Purmalis, B.P. Teratogenic and toxic effects of ochratoxin A in rats. Toxicol. Appl. Pharmacol. 1976, 37, 331–338.
97. Mayura, K.; Reddy, R.V.; Hayes, A.W.; Berndt, W.O. Embryocidal, fetotoxic and teratogenic effects of ochratoxin A in rats. Toxicology 1982, 25, 175–185.
98. Mayura, K.; Parker, R.; Berndt, W.O.; Philips, T.D. Effect of simultaneous prenatal exposure to ochratoxin A and citrinin in the rat. Toxicol. Environ. Health 1984, 13, 553–561.
99. Mayura, K.; Stein, A.E.; Berndt, W.O.; Philips, T.D. Teratogenic effects of ochratoxin A in rats with impaired renal function. Toxicology 1984, 32, 277–285.
100. Mayura, K.; Parker, R.; Berndt, W.O.; Philips, T.D. Ochratoxin A-induced teratogenesis in rats: Partial protection by phenylalanine. Appl. Environ. Microbiol. 1984, 48, 1186–1188.
101. Gilani, S.H.; Bancroft, J.; Reily, M. Teratogenicity of ochratoxin A in chick embryos. Toxicol. Appl. Pharmacol. 1978, 46, 543–546.
102. Hood, R.D.; Naughton, M.J.; Hayes, A.W. Prenatal effects of ochratoxin A in hamsters. Teratology 1976, 13, 11–14.
103. Lalini R, K. B. (2010). Ochratoxins Food Contaminants: Impact on Human Health. Toxins, 771-779.