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Potential health risk and genotoxicity of mycotoxins associated with staled agricultural food items of covid-19 palliatives consumed by protesters in calabar, Nigeria

Abstract

The research was designed to identify mycotoxins, genotoxicity and the potential diseases that can infect or affect protesters in Calabar, Nigeria who consumed stale food items as garri, rice, indomie noodles, beans, semovita, corn and vegetable oil of COVID 19 palliatives. Samples of the stale food items were collected and cultured in vitro for fungal and bacterial isolation. Pure cultures of the fungus isolated were further subjected to mycotoxins analyses. Mycotoxins isolated include aflatoxin, ochratoxin, patulin, citrinin, fumonisin and penicillic acid. Stitching of the identified mycotoxins with homo sapiens organism revealed that the mycotoxins impedes and disrupts the expressions and functionality of over a hundred protein genes (genotoxicity) notably among which includes TP53, IL2, MAPK3, CASP3, CAT, ATF3, OCLN, SMPD2, SMPD1, UGCG, CERK, CYPSA4, CYP3A7, XRCC1, LACE1, Albumin, EPHX1, SPATA5, SOD1, G6PC, DECR1, PAK2, SLC15A2, SCL15A1, TLR2, ABCB11, IFNG, ABCO2 and TMPRSS11D. The study unveiled the potential health risk and diseases associated with mycotoxicity in the consumers using the Elsevier pathway studio software to includes cancer of the intestine, lung carcinoma, lung cancer, skin diseases, nephritis, kidney diseases, lung injury, dermatitis, malignant tumors, allergies, dysentery, cancer, ulcer and liver diseases. The study recommended the use organic acids, biological agents (bio degraders) and mycotoxin binders as reliable options for reducing or neutralize the toxicity of the mycotoxins in contaminated feed before consumption. Medical therapy should be administered to protesters already down with any of the associated ailments while consumers that are yet to experience any sign or symptom should endeavour to go for medical diagnosis/check up to prevent future loss of lives and reduce mortality.

Keywords: mycotoxins, spoilt food, covid -19 palliatives, kidney diseases, gene functions

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Godwin M Ubi,[1] Joyce F Akpan,[2] Etangetuk NA,[1] Edu N Eyogor,[3] Juliet N Ekpang,[4] Effiom B Ekeng,[5] Chinyere Osondu-Anyanwu,[6] Imaobong S Essien[6]

[1]Department of Genetics and Biotechnology, Faculty of Biological Sciences, University of Calabar, P.M.B 1115, Calabar, Nigeria
[2]Department of Soil Science, Faculty of Biological Sciences, University of Calabar, P.M.B 1115, Calabar, Nigeria
[3]Department of Linguistics, Faculty of Arts, University of Calabar, P.M.B 1115, Calabar, Nigeria
[4]Department of Guidance and Counselling Unit, Faculty of Education, University of Calabar, P.M.B 1115, Calabar, Nigeria
[5]Department of Science Laboratory Technology, Faculty of Biological Sciences, University of Calabar, P.M.B 1115, Calabar, Nigeria
[6]Department of Zoology and Environmental Biology, Faculty of Biological Sciences, University of Calabar, P.M.B 1115, Calabar, Nigeria

Correspondence: Godwin M Ubi, Department of Genetics and Biotechnology, Faculty of Biological Sciences, University of Calabar, Nigeria, Email ubiology.gu@gmail.com

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Introduction

The negative impact of COVID 19 diseases on susceptible population attained a worrisome dimension by frustrating families, entrepreneurships and bringing various governments down to its knees for the first time in the 21st century. The casualties and associated mortality rate plunge several boys, girls into orphanage homes, left many others jobless, down trodden, traumatized and sick. These may have given rise to the October, 2020 protest to #endsars# which culminated to riot, burning down of government infrastructures and assets by the protesters who are majority young people in Nigeria.[1]

The federal government of Nigeria through the Federal Ministry of Humanitarian and Social Services made available food crops palliatives including, rice, corn, beans, garri, semovita, wheat flour, corn, indomie noodles, juice, power oil and liquid magi to states to help cushion the traumatic effect of lock down occasioned by COVID 19 pandemic. Unfortunately most of the State Governors hoarded the food crop palliatives preserving them for future personal use and ceremonies. The government officials kept and hoarded large quantities food crop palliatives meant for the citizens under lock down in poor storage condition for a long time leading to abrupt deterioration of all the food items. The #endsars# protesters in Calabar and other states in Nigeria identified these store ware houses of food treasure, looted and consumed all the stale or spoiled food items not minding the consequence due to hunger, widespread poverty occasioned by the lock down.

Stale foods are spoilt foods due to metabolic processes that makes foods not to be appealing and unacceptable by desired consumers as a result of the bad sensory characteristics.[2] Foods may be considered safe to eat, indicating that such food may not induce illness because of the absence of pathogens or a toxin. However, the resulting variations in odour, texture, smell, taste, or appearance of stale food can make them unacceptable. It has been posited that this obnoxious smells constitute products of microbial entities to wade off enemies in other to store up food nutrients for self-gain.[3]

The United States Department for Agriculture (USDA) have posited that closed to a hundred billion pounds of food were lost by producers and distributors of which fresh produce such as fluid milk accounted for nearly 20% of this loss while lower percentages were accounted for by grain products (15.2%), caloric sweeteners (12.4%), processed fruits and vegetables (8.6%), meat, poultry and fish (8.5%), and fat and oils (7.1%).[4] Several of these food items were discarded because of their high perishable nature.[5]

The durability of a food item is the period in which the food item is in good condition and maintains its initial characteristics for human consumption.[6] Spoil food habour microbes which produces

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mycotoxins which have been reported to be carcinogenic, mutagenic and genotoxic.

Mycotoxins are toxic metabolites of fungal origin. Mycotoxins are hazardous to human consumers and animals and remained a source of public concern for over 30 years. Mycotoxins are toxins manufactured by specific species of fungal moulds which can be naturally obtained throughout the environment. They infect food crops and induce a seemingly greater risk to humans and livestock health. They are carcinogenic, genotoxic, teratogenic, mutagenic and neurotoxic in nature with grave effect of life in all forms. Mycotoxins presence in stale food poses a significant economic burden, causing an estimated 25% or more of the world's food crop to be destroyed annually. Mycotoxins produce toxic secondary metabolite which grows on staled unprocessed and processed food products, such as sorghum, wheat, corn, garri, semovita, beans, corn, indomie noodles, peanuts, and wheat, among others. Aflatoxin effect occurs predominantly on exposure and ingestion of spoilt food items. It also occurs if Aflatoxin infected food items such as corn, wheat, peanuts and sorghum, as well as other processed food items, are utilized as recipe in the synthesis of food. Notably of these mycotoxins is Aflatoxin, described as a group of virulent mycotoxins mostly generated by the species Aspergillus parasiticus Aspergillus flavus, and most often by other species of Aspergillus.[7]

Aflatoxin poses a grave danger and risk to the animals and humans health of humans as a result of their immunosuppressive, teratogenic, carcinogenic and mutagenic effects.[8] However, looking at the livestock industry, aflatoxin presents a huge financial and economic burden to humans by retarding the growth of experimental models and test organisms. Increasing cost of production and reduced weight gain.[9] Notably among the different kinds of mycotoxins, aflatoxin B1 (AFB1) is adjudged the most biologically virulent toxic producing species. Other mycotoxins isolated from stale food also include patulin, Ochratin A, funomisin, citrillin and Penicillic acid.

This research is designed to unmasked the mycotoxins in garri, rice and indomie noodles stale food item palliatives, the mycotoxins genotoxic effect as well as the associated health hazards (diseases) accompanying the consumption of the stale food items and the possible therapeutic remedy for victims.

Material and methods

Experimental site

The study was carried out in the Laboratory of the Department of Microbiology, University of Calabar, Calabar and Big made Int'l Academy of Sciences Ltd. Calabar, from March 2021 to October, 2023.

Sample collection

Six (6) stale food crops were selected for the study. The selected stale foods included samples of garri, rice, corn, semovita, flour and indomie noodles selected based on their mycotoxic growth potentials and on their availability. Sample collection was done in April 2021 in Calabar south from the remnants of originally collected stale food items of October 2020 #endsars# protest.

A total of 20 samples (10g) of ready to consume of the stale foods were collected from the food items looted during the #endsars# protest in Calabar. These were already collected and kept for this study purpose. Samples of the selected stale food were collected using sterilized flasks and brought to laboratory for microbiological analysis. Samples were kept in a refrigerator (around 4°C) till the analysis was completed.

Identification of associated microbial loads

The Sexton and Atlas method[10] was adopted in the identification of mycotoxins in the selected stale food items. The total no. of fungi present in the sample was determine using surface spreading techniques. Agar plates in triplicates were prepared and at 37 degree C were incubated for 72 hrs before counting colonies. This units per gram of the sample was measured in colony forming units per gram (CFUg-. Fungus such as Aspergillus, Pennicillium, Curvularia, Neurospora, Mucor, Fusarium and Geotrichum were implicated in the spoilage of the food products. Aspergillus, Penicillium sp., Citrinins, Patulins, Funomisins and Ochratin have been reported to be the most common mycotoxins obtained from most food items.[11]

Isolation of mycotoxins from stale food crops palliatives

For isolation of lactic acid bacteria, samples of the rice, corn and indomie noodles, (25g of samples) were homogenized with 225ml sterilized buffered peptone water for about 1-3 minutes aseptically. From the homogenate mixture 1ml of suspension was transferred in to a test tube containing 9ml of buffered peptone water and test tubes were mixed using a vortex and dilution was made up to 10-6. A volume of 0.1 ml of appropriate dilutions of food samples were plated on MRS (OXOID) agar plates. Then the plates were incubated for 48hours in anaerobic jar at 32oC.

Characterization and identification of microbes in stale food palliatives

Twenty four individual isolates/colonies from MRS agar plates were randomly-picked, representatives from all morphologically distinct colonies and were sub-cultured and purified 3 times on the appropriate MRS agar medium. Identification of spoilage microbes was done based on morphology, physiology and biochemical characteristics. Twelve[12] pure microbial isolates were further tested for cell morphology, motility, gram reaction, catalase production, acid production from glucose and growth at 100C, 15 0C, 45 0C and 370C as a control according to the methods. Cell morphology of Cocci or rod shaped, non-motile, gram-positive, catalase-negative, isolates with characteristic cell arrangements were considered as bacteria isolates.

Identification of associated mycotoxins contamination in COVID 19 palliatives

Isolation of mycotoxin

Potato dextrose agar plate was prepared and collected. Each of the stale food sample including garri, corn, semovita, indomie, wheat flour and rice was serially diluted in saline solution. The samples were then inoculated into the agar plates. The plates (plate 1) were further inoculated at 28¬+20C for 3-5days. After incubation, the total numbers of fungal population per gram of the stale food were estimated and fungal species identified (Plate 1).

Aflatoxin and other toxins isolation

The Mycotoxin from Aspergillus flavus called Aflatoxin was isolated from collected mould rice, garri, indomie, semovita, and corn and wheat flour samples. Equipment and reagents microbes extraction includes whtmann No.1 filter paper potato dextrose agar, saline solution, chloroform, sodium bicarbonate solution, ethyl acetate, formic acid, toluene, 1% p-dimethyl aminobenaldehyde, petri dishes,

incubator, microscope, pH scale, conical flask, Whatmann No. 1 filter paper, water bath, evaporator and silica gel.

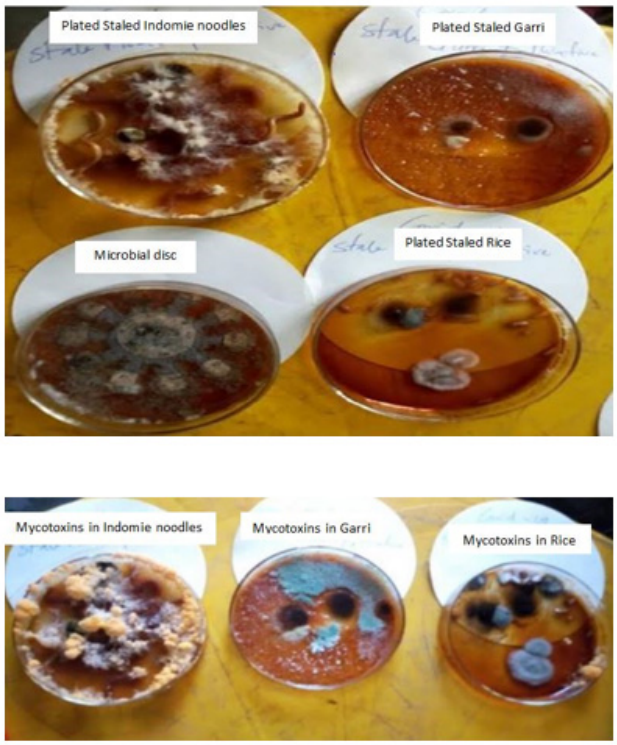

Plate 1 Agar plating of stale indomie noodles, garri, semovita, corn and rice for mycotoxins isolation

A culture filtrate that was highly concentrated was vortex vigorously with 100ml of chloroform and extract repeatedly for 2 or 3 times. The extract was combined with chloroform and filtered using Whatmann No.1 filter paper. 05M solution of Sodium bicarbonate solution was used to obtained toxins from the filtered chloroform extraction through vigorously centrifuging many times. After soaking in sodium bicarbonate for 24 hours, the oily materials were separated were separated using a separation funnel for filtration. To round it up, the hydrogen and hydroxyl content of the solution were reduced to 2.0. Aliquots of chloroform was replicative used to extract toxins with chloroform. The extracts were cooled and concentrated and the crude toxins isolated.

Identification of associated health hazards (diseases) and toxicity associated with mycotoxins contaminated covid-19 food palliatives in calabar using elsevier pathway studio software

Disease and toxicity prediction was performed using Toxtree 2.6. And the Chembi KEGG online programme resources. The comparative molecular similarity index analytical model was employed in Chemprot (Chemotherapy protocol) tool for the determination of the quantitative structural activity relationship and the associated diseases of the mycotoxins on the target (absorption, metabolism, distribution, excretion and toxicity) ADMET pathways of the human consumers. The online interactive programme of expasy.org. Was exploited. The click2drug option and the pathway databases of stitch online resource programme was chosen and adopted to for the determination of the mycotoxins genomic interactions for the determination of associated biological processes, molecular partner and cellular function. The Elsevier pathway studio was used to determine the likely health consequences related to the utilization of the stale food items containing the identified mycotoxins.[12]

Identification of therapies for associated diseases and make recommendations

Based on the identified diseases and toxicities associated with consumption of stale food palliatives using CHEMPROT and QSAR software, the KYOTO Encyclopedia of genes and genomics (KEGG) of the Japanese disease therapy databases was used to identify potentials and possible therapies for the identified diseases.[13]

Results and discussion

Mycotoxins in stale food items of COVID-19 palliatives in calabar

The results of associated mycotoxins isolated from the stale food crop items of COVID 19 palliatives is presented in Table 1 as extracted from figure 1. There was significant variation in the different mycotoxins isolated from the different food items. The results (Table 1 and Fig.1) revealed that aflatoxin mycotoxin was more predominant and found in garri (37%), indomie (25%), rice and semovita (15%) and least isolated from vegetable oil (5%) and Margarine (3%) (Figure 1).

Table 1 Mycotoxins isolated from the stale food crops of COVID-19 palliatives in order of magnitude (extracted from Figure 1)

S/N	Mycotoxins	Order of major occurrence in stale food items	Order of minor occurrence in stale food items
1	Aflatoxin	Garri (37%) Indomie noodles (25%) Rice (15%) Semovita (15%)	Vegetable Oil (5%) Margarine (3%)
2	Citrinin	Rice (40%) Indomie noodles (25%) Semovita (12%) Garri (10%)	Vegetable oil (6%) Margarine (2%)
3	Fuminosin	Rice (30%) Garri (30%) Indomie noodles (15%) Semovita (15%)	Vegetable oil (5%) Margarine (5%)
4	Penicillin	Garri (30%) Indomie noodles (20%) Semovita (20%) Beans (20%)	Vegetable oil (5%) Margarine (5%)
5	Ochratoxin A	Rice (20%) Beans (20%) Semovita (20%) Garri (20%) Indomie noodles (15%)	Margarine (3%) Vegetable oil (2%)
6	Patulin	Garri (25%) Indomie noodles (20%) Rice (20%) Semovita (20%)	Beans (5%) Margarine (5%) Vegetable oil (5%)

Ochratoxin showed 20% presence in rice, beans, semovita and garri and recorded 15% in indomie noodles. It was slightly 3% in margarine and 2% in vegetable oil (Table 1, Figure 1).

Citrinin mycotoxins were 40% in rice, 25% in indomie noodles, 12% in semovita and 10% in garri. Only 6% and 2% of the mycotoxin was isolated from vegetable oil and margarine respectively (Table 1, Figure 1).

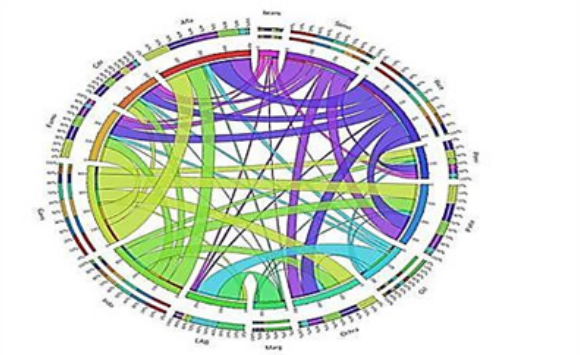

Figure 1 circos analysis and visualization of mycotoxins associated with the different food items of covid 19 palliatives consumed by protesters in calabar, Nigeria.

Key: Size of standard represents the volume of mycotoxins isolated from the different staled food items

Mycotoxins: Afla = Aflatoxin; patu= patulin; ochre= ochratin A; citri = citirin; Fumo= funomisin; peni= pencillin; lab= lactic acid bacteria Food items: Gari= Garri; indo= indomie; Rice= Rice; semo= semovita; Marg= margarine, oil= vegetable oil

Mycotoxin genome interactions (Genotoxicity) of consumed stale foods in consumer biological system

Genotoxicity of aflatoxin

Aflatoxin is a class of genetically related toxic metabolites that are described as mycotoxins. They are generated from Aspergillus flavus and A. parasiticus. The class members involved aflatoxin B1; aflatoxin B2, aflatoxin G1, aflatoxin G2; aflatoxin M1; and aflatoxin M2. When aflatoxin is ingested by humans, they impair gene function and become toxic to the following protein genes as shown in Figure 2.

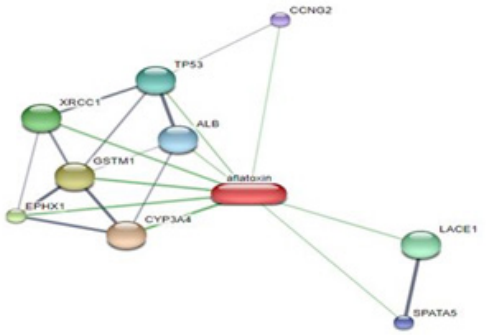

Figure 2 Genes and aflatoxin mycotoxin interactions revealing genotoxicity of aflatoxin.

CYP3A4 is a member of cytochrome P450, family 3, subfamily A, polypeptide 4 protein gene. The cytochrome P450 are a group of the heme-thiolate monooxygenases found in the liver microsomes. This protein gene (fig.3) is involved in an NADPH –dependent electron transport pathway. It performs a variety of oxidation reactions (e.g. controls caffeine 8-oxidation, omeprazole sulphoxiadtion, midazolam 1-hydroxylation and midazolam 4 hydroxylation of structurally unrelated compounds including steroids, fatty acids and xenobiotics. It acts as a 1,8-cineole 2-exomonooxygenase. The protein further hydrolates the etoposides and contains 503 amino acids. The active exon site for this protein is 20-349KDa in the genome. Aflatoxin disrupts the oxidations of toxic harmful substances which enters the liver such as steroid, unsaturated fatty acids and xenobiotics. Aflatoxin also inhibits the NADPH –dependent electron transport pathway thus disrupting biochemical processes which requires high energy for complete cellular activities.

Figure 3 Protein structure of the CYP3A4 cytochrome P450 family protein gene.

CYP3A7 is a member of the cytochrome P450, family 3, subfamily A, polypeptide 7 protein gene chain which is similar in action but at a lower dimension to the polypeptide 4 chain. This cytochrome P450 are also a group of the hemethiolate monooxygenases which occupies the liver microsomes. This protein gene (fig.4) is also involved in an NADPH –dependent electron transport pathway. The active exon site for this protein is 31-505 KDa in the genome. Aflatoxin disrupts the oxidations of toxic harmful substances which enters the liver such as steroid and fatty acids. Aflatoxin also inhibits the NADPH –dependent electron transport for this pathway, thus disenabling and uncoupling biochemical processes which requires high energy for complete coupling and complex formation (Figure 3–5).

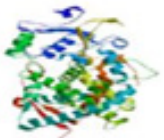

Figure 4 Protein structure of the CYP3A7 cytochrome P450 family protein gene.

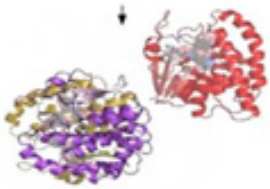

Figure 5 Protein structure of the GSTM1 protein gene.

GSTM1 is glutathione S-transferase mu-1 protein gene (Figure 5). The protein gene forms a conjugant with aflatoxin, thus reducing glutathione to a wide number of exogenous and endogenous hydrophobic electrophiles or substances which are useless to the human system. Glutathione is very useful in the human system as an immune booster and a correctional protein to many unstable proteins in the body. It active exon site is found within the first 1 and last 218 KDa which corresponds to the exact size of the gene (218aa). Aflatoxin reduces the metabolism and functionality of glutathione in the body thus weakening the immune system and increasing the susceptibility of the organisms to other infections. Aflatoxin mycotoxin affects the expressivity and functionality of the SPATA5 gene when ingested through food intake.

XRCC1 is an X-ray repair complementing defective repair protein gene (fig.6) found in the Chinese hamster cells 1. It functions in the correction of defective DNA strand-break repair and sister chromatid exchange following treatment with ionizing radiation and alkylating

agents. The exon active site of this protein gene is found between 1 and 183 KDa out of the 633aa protein size. The presence of aflatoxins in body impairs the repair functions of this protein gene. Alkylating agents are de-alkylated by aflatoxins and the defective repair protein is denatured in the presence of aflatoxin compound. Aflatoxin mycotoxin affects the expressivity and functionality of the XRCC1 gene when ingested through food intake (Figure 6–9).

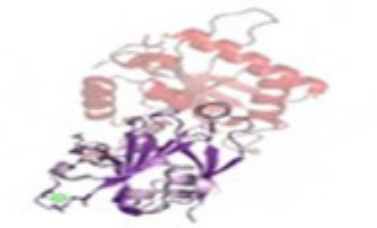

Figure 6 Protein structure of the XRCC1 protein gene.

Figure 7 Protein structure of the EPHX1 protein gene. sivate Wir Go to Setting.

Figure 8 Protein structure of the LACE protein gene.

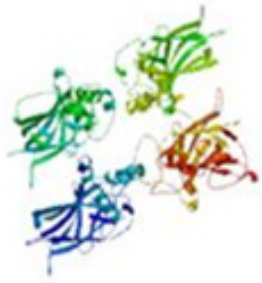

Figure 9 Protein structure of the TP53 protein gene.

EPHX1 is epoxide hydrolase 1 protein gene (Figure 7), which reacts with microsomal xenobiotic (455aa). Aflatoxin mycotoxin impedes the reactivity of EPHX1 with the microsomal xenobiotic, impairs expressivity and functionality of the EPHX1 protein gene when ingested through food intake.

LACE 1 is lactation elevation 1 protein gene (481aa) Aflatoxin mycotoxin affects the expressivity and functionality of the SPATA5 gene by otherwise reducing lactation in humans when ingested through food intake (Figure 8).

TP53 is the tumor protein 53 which acts as a tumor suppressor in many tumor types. Aflatoxin suppresses the reactivity, expressivity and functionality of the tumor suppressor gene. The TP53 gene (Figure 9) is known to induce growth arrest or apoptosis depending on the physiological circumstances and cell types with 393aa on active exon site. It is involved in cell cycle regulation as a trans-activator that acts to negatively regulate cell division by controlling a set of genes required for this process. This action is greatly impeded by aflatoxin. One of the activated genes is an inhibitor of cyclin-dependent kinases which is resisted by the presence of aflatoxin. Apoptosis induction seems to be hindered by aflatoxin.

ALB is Albumin (609aa) as shown in Figure 10. Aflatoxin mycotoxin affects the expressivity and functionality of the albumin protein gene when ingested through food intake (Figure 10,11).

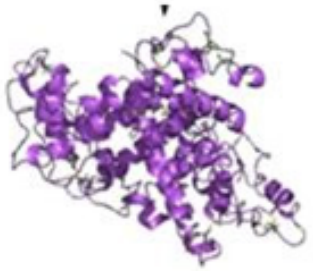

Figure 10 Protein structure of the albumin protein gene.

Figure 11 Protein structure of the SPATA5 protein gene.

SPATA5 gene is spermatogenesis associated protein 5 gene (Figure 11) which is involved in the morphological and functional mitochondrial transformations during spermatogenesis (893aa). Aflatoxin mycotoxin affects the expressivity and functionality of the SPATA5 gene when ingested through food intake.

CCNG2 is cyclin G2 protein gene (Figure 12) which plays a crucial role in the growth, regulation and in repressive regulation of cell cycle procession (344aa). Aflatoxin mycotoxin affects the expressivity and functionality of the CCNG2 gene when ingested by humans (Figure 12).

Figure 12 Protein structure of the CCNG2 protein gene.

The metabolic pathway of aflatoxins B1, B2, G1 and G2 are presented in figure below. The ingestion of aflatoxin B1 in contaminated food begins with the production of methyl-sterigmatocystin enzyme

which pave the way for the complex, Sterigmatocystin which upon demethylation produces versicolonin A (an enzyme which disrupts the colon) and versicolonin B. The two enzymes reacts to form versiconial hemiactal acetate and producing 1‘5 hydroxyaverantin which derails the optimization of the nutrient synthesis and metabolism (Graph 1).

Graph 1 Metabolic pathway for aflatoxins in stale food consumed by protesters in Calabar.

Genotoxicity of Ochratoxin A

Ochratoxin designated as SLC17A3 protein gene is also known as Mellein. It is a dihydroisocoumarin, a phenolic compound produced by Aspergillus ochraceus”. (178.2 g/mol). Ochratoxin is Solute carrier family 17 (sodium phosphate), member 3. It is an isoform 2-voltage-driven, multi-specific organic anion transporter that is able to transport para-aminohippurate (PAH), estrone sulfate, estradiol-17-beta-glucuronide, bumetanide, and ochratoxin A toxin. The isoform 2 functions as urate efflux transporter on the apical side of renal proximal tubule and is likely to act as an exit path for organic anionic drugs as well as urate in vivo. It is involved in actively transporting phosphate into cells via Na (+) cotransport (498aa). The activity of the protein gene is totally distorted in the presence of ochratoxin indicating that when Ochratoxin mycotoxin is ingested by humans, they impair and become toxic to the following protein genes as shown in (Figure 13).

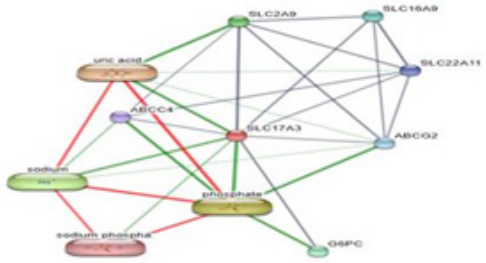

Figure 13 Genes and Ochratoxin mycotoxin interactions revealing genotoxicity of ochratoxin.

SLC2A9 is solute carrier family 2 (facilitated glucose transporter), member 9 protein gene. The gene transport urate and fructose to sites. It plays a role in the urate reabsorption by proximal tubules. Also transports glucose at low rate. It has 540aa. Ochratoxin impairs the transport of urate and fructose by this protein gene thereby affecting kidney functions.

G6PC is glucose-6-phosphatase, catalytic subunit protein gene with 357aa which hydrolyzes glucose-6-phosphate to glucose in the endoplasmic reticulum. It forms complex with the glucose-6-phosphate transporter (SLC37A4/G6PT), which is the complex responsible for glucose production through glycogenolysis and gluconeogenesis. The complex formation is hindered by ochratoxin. Hence, this key enzyme in homeostatic regulation of blood glucose levels is denatured.

SLC16A9 is solute carrier family 16, member 9 protein gene with 509aa (monocarboxylic acid transporter 9) which acts as a proton-linked monocarboxylate transporter. It also catalyzes the rapid transport across the plasma membrane of many monocarboxylates except intercepted by ochratoxin.

ABC02 is ATP-binding cassette, sub-family G (WHITE), member 2 protein gene with 655aa. The presence of ochratoxin impedes the release of ATP – bound energy to the cells through the ABCO2 protein gene.

SLC22A11 is solute carrier family 22 (organic anion/urate transporter), member 11 protein gene with 550aa which mediates saturable uptake of estrone sulfate, dehydroepiandrosterone sulfate and related compounds. These saturable uptake functions of the gene is greatly distorted by ochratoxin.

ABCC4 is ATP-binding cassette, sub-family C (CFTR/MRP), member 4 protein gene which is made up of 1325aa. It is an organic anion pump relevant for cellular detoxification. The presence of ochratoxin disrupts ATP –bound organic anion pump and hinders cellular detoxification functions of the liver.

Genotoxicity of citrinin

Citrinin is a mycotoxin originally isolated in 1931 by Hetherington and Raistrick from a culture of “Penicillium citrinum”. It has since been found to be produced by a variety of other fungi that are found or used in the production of human foods, such as grain, cheese, sake, and red pigments. Citrinin has also been found in commercial red yeast rice supplements (249.2 g/mol). When ingested through food intake by humans, citrinin mycotoxin reacts with the following protein genes as presented in Figure 14 causing genotoxicity (Figure 14).

Figure 14 Genes and Citrinin mycotoxin interactions revealing genotoxicity of Citrinin.

MT-CYB is mitochondrially encoded cytochrome b protein gene which is a component of the ubiquinol-cytochrome c reductase complex (complex III or cytochrome b-c1 complex). It is a respiratory chain of 380aa that generates an electrochemical potential coupled to ATP synthesis. ATP synthesis is greatly distorted by citrinin mycotoxin when ingested through the consumption of stale foods.

PTG52 is prostaglandin-endoperoxide synthase 2 protein gene with 604aa (prostaglandin G/H synthase and cyclooxygenase). Ochratoxin hinders the functions of this gene which mediates the formation of prostaglandins from arachidonate and which otherwise have a role as a major mediator of inflammation and/or a role for prostanoid signaling in activity-dependent plasticity.

CASP3 is caspase 3 protein gene, apoptosis-related cysteine peptidase protein gene with 277aa which is involved in the activation cascade of caspases responsible for apoptosis execution. At the onset of apoptosis it proteolytically cleaves poly(ADP-ribose) polymerase (PARP) at a ’216-Asp-|-Gly-217’ bond. The cleavage activates sterol regulatory element binding proteins (SREBPs) between the basic

helix-loop- helix leucine zipper domain and the membrane attachment domain. Cleavage and activation of caspase-6, -7 and -9 is impaired by citrinin mycotoxin. CASP3 is involved in the cleavage of huntingtin, a process that triggers cell adhesion in sympathetic neurons through RET cleavage.

MAPK8 is mitogen-activated protein kinase 8 gene with 427aa. This Serine/threonine-protein kinase is involved in various processes such as cell proliferation, differentiation, migration, transformation and programmed cell death. Extracellular stimuli such as proinflammatory cytokines or physical stress stimulate the stress-activated protein kinase/c-Jun N-terminal kinase (SAP/JNK) signaling pathway. In this cascade, two dual specificity kinases **MAP2K4/MKK4** and **MAP2K7/MKK7** phosphorylate and activate MAPK8/JNK1. In turn, MAPK8/JNK1 will dephosphorylates a few transcription factors, primarily components of AP-1 in the presence of citrinin mycotoxins.

MAPK9 is mitogen-activated protein kinase 9 protein gene with 424aa. Citrinin negatively influences the functionality and expressivity of the MAPK9 gene.

PAK2 is p21 protein (Cdc42/Rac)-activated kinase 2 gene with 524aa. This Serine/threonine protein kinase plays a crucial role in a variety of different signaling pathways including cytoskeleton regulation, cell motility, cell cycle progression, apoptosis or proliferation. The gene acts as downstream effector of the small GTPases CDC42 and RAC1. Activation by the binding of citrinin mycotoxin with active CDC42 and RAC1 results in a conformational change and a subsequent auto phosphorylation on several serine and/or threonine residues and acts against the PAK2 stimulation of cell survival and growth.

ABCB11 is ATP-binding cassette, sub-family B (MDR/TAP), member 11 protein gene with 1321aa that is involved in the ATP-dependent secretion of bile salts into the canaliculus of hepatocytes. This gene function is impaired by citrinin mycotoxin.

MAPK3 is mitogen-activated protein kinase 3 gene with 379aa. This Serine/threonine kinase acts as an essential component of the MAP kinase signal transduction pathway. MAPK1/ERK2 and MAPK3/ERK1 are the 2 MAPKs which play an important role in the MAPK/ERK cascade. They participate also in a signaling cascade initiated by activated KIT and KITLG/SCF. Depending on the cellular context and otherwise distorted by citrinin mycotoxin inhibition, the MAPK/ERK cascade will mediates diverse biological gene functions such as cell growth, adhesion, survival and differentiation through the regulation of transcription, translation, cytoskeletal rearrangements.

IFNG is interferon, gamma protein gene with 166aa. It is produced by lymphocytes activated by specific antigens or mitogens. IFN-gamma, in addition to having antiviral activity, has important immune-regulatory functions, which can only be inhibited by citrinin mycotoxins. It is a potent activator of macrophages and has anti-proliferative effects on transformed cells and can also potentiate the antiviral and antitumor effects of the type I interferons.

RPS6KB1 is ribosomal protein S6 kinase, 70kDa, polypeptide 1 protein gene with 525aa. This Serine/threonine-protein kinase acts as downstream of mTOR signaling in response to growth factors and nutrients to promote cell proliferation, cell growth and cell cycle progression. Regulation of protein synthesis through phosphorylation of EIF4B, RPS6 and EEF2K, and contribution to cell survival is achieved by repressing the pro-apoptotic function of BAD or mycotoxins of citrinin. Under conditions of nutrient depletion, the inactive form associates with the EIF3 translation initiation complex to achieve phosphorylation.

Genotoxicity of fumonisin

When Fumonisin mycotoxin is ingested by humans, they impair and become toxic to the following protein genes as shown in Figure 15.

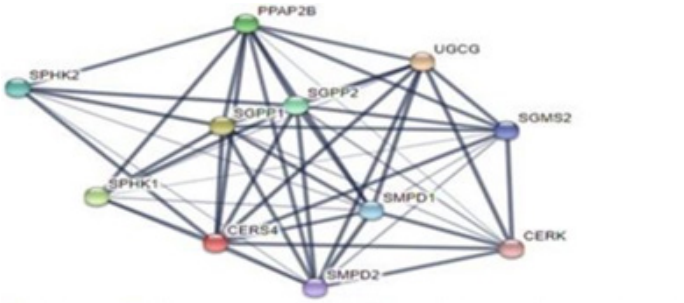

Figure 15 Genes and Fumonisin mycotoxin interactions revealing genotoxicity of Fumonisin.

CERS4 is ceramide synthase 4 protein gene with 394aa. It is a bona fide (dihydric) ceramide synthase or a modulator of its activity. When overexpressed in cells CERS4 is involved in the production of sphingolipids containing different fatty acid donors (N-linked stearoyl- (C18) or arachidoyl- (C20) ceramides) in a fumonisin B1-independent manner.

UGCG is UDP-glucose ceramide glucosyltransferase protein gene with 394aa. It catalyzes the first glycosylation step in glycosphingolipid biosynthesis and enhances the transfer of glucose to ceramide. The gene also serves as a "flippase". It functions and expression is hindered by the presence of fumonisin mycotoxins.

SGPP1 is sphingosine-1-phosphate phosphatase 1 protein gene; has enzymatic activity against both sphingosine 1- phosphate (S1P) and dihydro-S1P. Regulates intracellular and extracellular S1P levels (441aa)

SPHK1 is sphingosine kinase 1 protein gene; catalyzes the phosphorylation of sphingosine to form sphingosine 1-phosphate (SPP), a lipid mediator with both intra- and extracellular functions. Also acts on D-erythro-sphingosine and to a lesser extent sphinganine, but not other lipids, such as D,L-threo-dihydrosphingosine, N,N-dimethylsphingosine, diacylglycerol, ceramide, or phosphatidylinositol (470 aa)

PPAP2B is phosphatidic acid phosphatase type 2B protein gene with 311aa. It catalyzes the conversion of phosphatidic acid (PA) to diacylglycerol (DG). In addition it hydrolyzes lysophosphatidic acid (LPA), ceramide-1-phosphate (C-1-P) and sphingosine-1- phosphate (S-1-P). The relative catalytic efficiency is LPA = PA > C-1-P > S-1-P. It is involved in distorted cell adhesion and in cell-cell interactions in the presence of fumonisin mycotoxins.

SGPP2 is sphingosine-1-phosphate phosphatase 2 protein gene with 399aa. It shows specific phosphohydrolase activity towards sphingoid base 1-phosphates. It has high phosphohydrolase activity against dihydrosphingosine-1-phosphate and sphingosine-1-phosphate (S1P) in vitro. Fumonisin mycotoxins disrupts SGPP2 role in attenuating intracellular sphingosine 1-phosphate (S1P) and in pro-inflammatory signaling.

SPHK2 is sphingosine kinase 2 protein gene with 654aa. It catalyzes the phosphorylation of sphingosine to form sphingosine 1-phosphate (SPP), a lipid mediator with both intra- and extracellular functions. It also acts on D-erythro- dihydrosphingosine, D-erythro-sphingosine

and L-threo- dihydrosphingosine that binds Phosphoinositides. It activity is hindered by mycotoxins of fumonisin.

SMPD1 is sphingomyelin phosphodiesterase 1, acid lysosomal protein gene with 631aa. It help to converts sphingomyelin to ceramide. It also has phospholipase C activities toward 1,2-diacylglycerolphosphocholine and 1,2-diacylglycerolphosphoglycerol. The Isoform 2 and isoform 3 can easily lost catalytic activity in the presence of fumonisin mycotoxins.

SGMS2 is sphingomyelin synthase 2 protein gene with 365aa. Sphingomyelin synthases synthesize the sphingolipid, sphingomyelin, through transfer of the phosphatidyl head group, phosphatidylcholine, on to the primary hydroxyl of ceramide. The reaction is bidirectional depending on the respective levels of the sphingolipid and ceramide. Plasma membrane SGMS2 can also convert phosphatidylethanolamine (PE) to ceramide phosphatidylethanolamine (CPE). The gene is required for cell growth in certain cell types. Regulation of cell surface levels of ceramide, an important mediator of signal transduction and apoptosis by SGMS2 gene is lost when in contact with fumonisin mycotoxins.

SMPD2 is sphingomyelin phosphodiesterase 2, neutral membrane (neutral sphingomyelinase) proyein gene with 423aa which Converts sphingomyelin to ceramide. It hydrolyze 1-acyl-2- lyso-sn-glycero-3-phosphocholine (lyso-PC) and 1-O-alkyl-2-lyso- sn-glycero-3-phosphocholine (lyso-platelet-activating factor). The physiological substrate seems to be Lyso-PAF anti fumonisin.

Genotoxicity of penicillin

Penicillin Benzyl penicillin (Penicillin G) is narrow spectrum antibiotic used to treat infections caused by susceptible bacteria. It is a natural penicillin antibiotic that is administered intravenously or intramuscularly due to poor oral absorption. Penicillin G may also be used in some cases as prophylaxis against susceptible organisms. (334.4g/mol). When Penicillin mycotoxin is ingested by humans, they impair and become toxic to the following protein genes as shown in Figure 16.

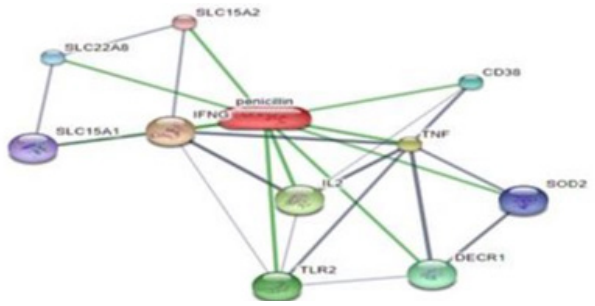

Figure 16 Genes and Penicillium mycotoxin interactions revealing genotoxicity of Penicillin.

IFNG is interferon, gamma protein gene with 166aa. It is produced by lymphocytes activated by specific antigens or mitogens. IFN-gamma, in addition to having antiviral activity, has important immune-regulatory functions. It is a potent activator of macrophages, it has anti-proliferative effects on transformed cells and it can potentiate the antiviral and antitumor effect of the type I interferons. These functions of IFNG are seemingly disrupted in the presence of penicillium mycotoxins.

TNF is tumor necrosis factor protein gene with 233aa. These functions of TNF are seemingly disrupted in the presence of penicillium mycotoxins.

IL2 is interleukin 2 immuno-protein gene with 153aa. It is produced by T-cells in response to antigenic or mitogenic stimulation, this protein is required for T-cell proliferation and other activities crucial to regulation of the immune response. It can stimulate B-cells, monocytes, lymphokine- activated killer cells, natural killer cells, and glioma cells. These functions of IL2 are seemingly disrupted in the presence of penicillium mycotoxins.

TLR2 is toll-like receptor 2 protein gene with 784aa. It aligns with LY96 to mediate the innate immune response to bacterial lipoproteins and other microbial cell wall components. It further cooperates with TLR1 or TLR6 to mediate the innate immune response to bacterial lipoproteins or lipopeptides. It acts via MYD88 and TRAF6, leading to NF-kappa-B activation, cytokine secretion and the inflammatory response. TLR2 also promote apoptosis in response to lipoproteins and recognizes mycoplasmal macrophage-activating lipopeptide-2kD (MALP-2), soluble tuberculosis factor (STF), phenol-soluble modulin (PSM) and B.burgdorferi outer surface. These functions of TLR2 are seemingly disrupted in the presence of penicillium mycotoxins.

DECR1 is 2, 4-dienoyl CoA reductase 1, mitochondrial protein gene with 335aa. It is an auxiliary enzyme responsible for beta-oxidation. It participates in the metabolism of unsaturated fatty enoyl-CoA esters having double bonds in both even- and odd-numbered positions. It catalyzes the NADP-dependent reduction of 2, 4-dienoyl-CoA to yield trans-3- enoyl-CoA. These functions of DECR1 are seemingly disrupted in the presence of penicillium mycotoxins.

CD38 is **CD38** molecule gene with 300aa. It synthesizes cyclic ADP-ribose, a second messenger for glucose-induced insulin secretion. It also has cADPr hydrolase activity and are moonlights as a receptor in cells of the immune system. These functions of CD38 are seemingly disrupted in the presence of penicillium mycotoxins.

SLC22A8 is solute carrier family 22 (organic anion transporter), member 8 protein gene with 542aa. It plays an important role in the excretion/detoxification of endogenous and exogenous organic anions, especially from the brain and kidney. It is involved in the transport basolateral of steviol, fexofenadine. It transports benzyl penicillin (PCG), estrone- 3-sulfate (E1S), cimetidine (CMD), 2,4-dichloro-phenoxyacetate (2,4-D), p-amino-hippurate (PAH), acyclovir (ACV) and ochratoxin (OTA). These functions of SLC22A8 are seemingly disrupted in the presence of penicillium mycotoxins.

SOD2 is superoxide dismutase 2, mitochondrial protein gene with 222aa. It destroys radicals which are normally produced within the cells and which are toxic to biological systems. These functions of SOD2 are seemingly disrupted in the presence of penicillium mycotoxins.

SLC15A1 is solute carrier family 15 (oligopeptide transporter), member 1 protein gene with 708aa. It is a proton-coupled intake of oligopeptides of 2 to 4 amino acids with a preference for dipeptides. It constitutes a major route for the absorption of protein digestion end-products. These functions of SLC15A1 are seemingly disrupted in the presence of penicillium mycotoxins.

SLC15A2 is solute carrier family 15 (H+/peptide transporter), member 2 protein gene with 729aa. It is also a proton-coupled intake of oligopeptides of 2 to 4 amino acids with a preference for dipeptides. These functions of SLC15A2 are seemingly disrupted in the presence of penicillium mycotoxins.

Genotoxicity of patulin

Patulin is a mycotoxin produced by a variety of molds, in particular, “Aspergillus” and “Penicillium” and “Byssochlamys”.

Most commonly found in rotting apples, in general the amount of patulin in apple products is viewed as a measure of the quality of the apples used in production. In addition, patulin has been found in other foods such as grains, fruits, and vegetables. While patulin is not considered a particularly potent toxin, a number of studies have shown patulin to be genotoxic, which has led some to theorize that it may be a carcinogen, although animal studies have remained inconclusive. Patulin has shown antimicrobial properties against some microorganisms. When Fumonisin mycotoxin is ingested by humans, they impair and become toxic to the following protein genes as shown in Figure 17.

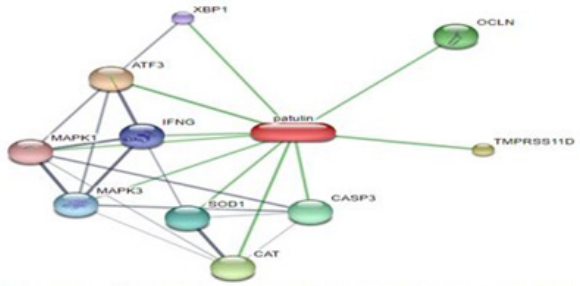

Figure 17 Genes and Patulin mycotoxin interactions revealing genotoxicity of patulin.

ATF3 is activating transcription factor 3 protein gene with 181aa. This protein binds the cAMP response element (CRE) (consensus-5'-GTGACGT [AC][AG]-3'), a sequence present in many viral and cellular promoters and represses transcription from promoters with ATF sites. It represses transcription by stabilizing the binding of inhibitory cofactors at the promoter. Patulin and Isoform 2 activates transcription presumably by sequestering inhibitory cofactors away from the promoters.

TMPRSS11D is transmembrane protease, serine 11D protein gene with 418aa. It plays some biological role in the host defence system on the mucous membrane independently of or in cooperation with patulin mycotoxin in airway mucous or bronchial secretions.

CAT is catalase protein gene with 527aa which occurs in almost all aerobically respiring organisms and serves to protect cells from the toxic effects of hydrogen peroxide. CAT promotes growth of cells including T-cells, B-cells, myeloid leukemia cells, melanoma cells, mastocytoma cells and normal and transformed fibroblast cells. These functions of CAT are seemingly disrupted in the presence of patulin mycotoxins.

OCLN is occludin protein gene with 522aa. Patulin deactivates occludin phosphorylation role in the cells when present.

CASP3 is caspase 3 protein gene with 277aa, an apoptosis-related cysteine peptidase which is involved in the activation cascade of caspases responsible for apoptosis execution. At the onset of apoptosis it proteolytically cleaves poly (ADP-ribose) polymerase (PARP) at a '216-Asp-|-Gly-217' bond. It cleaves and activates sterol regulatory element binding proteins (SREBPs) between the basic helix-loop-helix leucine zipper domain and the membrane attachment domain. It also cleaves and activates caspase-6, -7 and -9 and Involved in the cleavage of huntingtin. These functions of CASP3 are seemingly disrupted in the presence of patulin mycotoxins.

SOD1 is superoxide dismutase 1 protein gene with 154aa. SOD1 destroys radicals which are normally produced within the cells and which are toxic to biological systems. This function of SOD1 is seemingly disrupted in the presence of patulin mycotoxins.

MARK3 is mitogen-activated protein kinase 3 protein gene with 379aa. The Serine/threonine kinase acts as an essential component of the MAP kinase signal transduction pathway. MAPK1/ERK2 and MAPK3/ERK1 are the 2 MAPKs which play an important role in the MAPK/ERK cascade. They participate also in a signaling cascade initiated by activated KIT and KITLG/SCF. Depending on the cellular context, the MAPK/ERK cascade mediates diverse biological functions such as cell growth, adhesion, survival and differentiation through the regulation of transcription, translation, cytoskeletal rearrangements. These functions of MARK3 are seemingly disrupted in the presence of patulin mycotoxins.

IFNG is interferon, gamma protein gene with 166aa. It is produced by lymphocytes activated by specific antigens or mitogens. IFN-gamma, in addition to having antiviral activity, has important immune-regulatory functions. It is a potent activator of macrophages, it has anti-proliferative effects on transformed cells and it can potentiate the antiviral and antitumor effect of the type I interferons. These functions of IFNG are seemingly disrupted in the presence of patulin mycotoxins.

XBP1 is X-box binding protein 1 gene with 261aa. It transcription factor is essential for hepatocyte growth, the differentiation of plasma cells, the immunoglobulin secretion, and the unfolded protein response (UPR). It acts during endoplasmic reticulum stress (ER) by activating unfolded protein response (UPR) target genes via direct binding to the UPR element (UPRE). It binds DNA preferably to the CRE-like element 5'- GATGACGTG [TG] N(3)[AT] T-3', and also to some TPA response elements (TRE). It equally binds to the HLA DR-alpha promoter and to the Tax- responsive element (TRE) of HTLV-I. These functions of XBP1 are seemingly disrupted in the presence of patulin mycotoxins.

MAPK1 is mitogen-activated protein kinase 1 gene with 360aa. The Serine/threonine kinase acts as an essential component of the MAP kinase signal transduction pathway. MAPK1/ERK2 and MAPK3/ERK1 are the 2 MAPKs which play an important role in the MAPK/ERK cascade. They participate also in a signaling cascade initiated by activated KIT and KITLG/SCF. The MAPK/ERK cascade mediates diverse biological functions such as cell growth, adhesion, survival and differentiation through the regulation of transcription, translation, cytoskeletal rearrangements. These functions of MAPK1 are seemingly disrupted in the presence of patulin mycotoxins.

Potential health risks associated with COVID-19 palliatives stale food items consumed by protesters in calabar

The analysis of potential health risk (diseases) associated with the consumption of mycotoxins infested food stuff using the Elsevier pathway studio revealed diseases such as cancer of the intestine, lung carcinoma, cancer of the lungs, skin diseases, kidney diseases, lung injury, dermatitis, malignant transformation, allergy, nephritis and ulcer (Figure 18).

Pathway analysis of mycotoxicity of the stale food items of COVID 19 palliatives showed the potentials of genomic interaction, cellular processes, and diseases induced by ingesting and exposure revealing 12 proteins, 5 cellular processes, 13 diseases, 2 small molecules and 1 functional class as shown in Figure 18.

The pathway showed MAPK, mitogen-activated protein kinase; AKT1, V-akt murine thymoma viral oncogene homolog 1; NFE2L2, nuclear factor, erythroid 2-like 2; CAT, catalase; IFNG, interferon gamma; CASP3, caspase 3, apoptosis-related cysteine peptidase; VEGFA, vascular endothelial growth factor A; TP53, tumor protein

53; BAX, B cell lymphoma 2 protein-associated X protein and ROS, reactive oxygen species as cell processes (Figure 18).

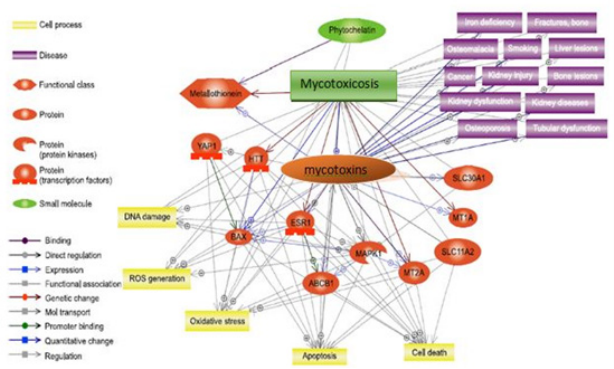

Figure 18 Associated health risk (diseases) of Mycotoxicosis in human consumers of staled food items.

Measures for reducing mycotoxicosis

A good number of methods have been investigated in connection with their effectiveness to inactivate mycotoxins in contaminated feedstuffs and the aim of this methods are either to remove or to destroy the toxins. They can be classified into physical, chemical and biological methods.[14]

Chemically, organic acid is considered one of the most widely used food additives, which is commonly used as a preservative, acid forming substances, pH control agent, flavor enhancer and antioxidant in many foods. Organic acid is one of the important factors responsible for the characteristics ordure and sour taste of food stuff with antibacterial and antifungal properties. Organic acids have been used to decrease the growth of harmful fungi and reduce secretion of aflatoxin. In addition, they have many properties on poultry health, including good growth performance, decrease feed conversion ratio, and enhanced hematological and pathological parameters through the detoxification of aflatoxin.[15]

Biologically, the use of biological methods, microorganisms and their metabolites to eliminate aflatoxin, can be a highly promising approach owing to its specific, efficient, and environmentally sound detoxification.[16] Some microbes including fungal and bacterial isolate, such as live yeast, Sacchromyces cerevisiae.[15] Flavo bacterium aurantiacum, Stenotrophomonas Maltophilia,[16] Myxococus fulvus and Aspergilus Niger[17] were reported to effectively biodegrade aflatoxin in vitro. However, little is known about their efficiency in the biodegradation of aflatoxin and effect of aflatoxicosis in vivo. As a biological product, several studies have revealed that esterified glucomannan derieved from cell wall of Sachromyces cerevisiae[18,19] have shown considerable promise in countering aflatoxins.

Physically, the use of mycotoxin binders showed that using the binder in contaminated feeds was responsible for reducing liver residual aflatoxin levels, and in ameliorating the negative effect of aflatoxin on Newcastle antibody production.[20,21] A good toxin binder should restore the nutritional values of aflatoxin contaminated feed. The quality of a good toxin binder is expressed in four different parameters, binding capacity, absorption efficacy, activation time and inclusion rate.[22,23]

Conclusion

The Mycotoxins are secondary metabolites produced by about 200 filamentous fungi, including Fusarium, Aspergilus and Penicilium species, growing under a wide range of climatic conditions on agricultural commodities (grains, spices, fruits, coffee, nuts etc.), in the field and during storage. There occurrences in food, beverage and feed has been recognized as potential threat to human health, either caused direct contamination of plant materials or products thereof or by "carryover" of Mycotoxins and their metabolites into human tissues, cells and blood after intake of contaminated foods.

Mycotoxins isolated include aflatoxin, ochratoxin, patulin, citrinin, fumonisin and penicillic acid. Stitching of the identified mycotoxins with homo sapiens organism revealed that the mycotoxins impedes and disrupts the expressions and functionality of different protein genes (genotoxicity) including TP53, IL2, MAPK3, CASP3, CAT, ATF3, OCLN, SMPD2, SMPD1, UGCG, CERK, CYPSA4, CYP3A7, XRCC1, LACE1, Albumin, EPHX1, SPATA5, SOD1, G6PC, DECR1, PAK2, SLC15A2, SCL15A1, TLR2, ABCB11, IFNG, ABCO2 and TMPRSS11D. The potential health risk and diseases associated with mycotoxicity in the consumers includes cancer of the intestine, lung carcinoma, lung cancer, skin diseases, nephritis, kidney diseases, lung injury, dermatitis, malignant tumors, allergies, dysentery, cancer, and ulcer and liver diseases.

The control of mycotoxicosis is based on preventing fungal development in the feedstuff, and on detoxifying toxin contaminated feed. At present, unfortunately, aflatoxin is considered unavoidable contaminants in most foods. The fed and agriculture organization (FAO) estimated that at least 25% of the world's cereal production is contaminated with Mycotoxins. For this reason, developments of detoxification procedures are needed. Such detoxification procedures should not only reduce the concentration of toxins to safe levels (below regulatory limits), but should also prevent the production of new products derived from aflatoxin degradation, and of course non reduction of the nutritional value of the treated commodities. Thus, it was suggested that protesters (consumers) stale food stuff oAf COVID 19 palliatives exposed to mycotoxin toxicity in the study area should seek for urgent medical attention while those yet to experience any sign or symptom should as a matter of urgency go for medical check up to prevent loss of lives and increasing mortality in the study area.

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Conflicts of interests

Authors have declared no conflict of interest in this manuscript.

Ethical approval

Ethical approval was not required as human samples were not involved.

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