MycoTOX PROFILE

REQUISITION #	9900001		
PATIENT NAME	**Report Sample**	COLLECTION TIME	10:00 AM
DATE OF BIRTH	Mar 9, 1960	COLLECTION DATE	Dec 1, 2022
GENDER	F	SAMPLE TYPE	Urine
PRACTITIONER	NO PHYSICIAN	REPORT DATE	Jun 10, 2024

Summary of Elevated Results

The results below lists mycotoxin(s) with elevated results detected in this profile. You can find all tests results and a more detailed description of each mycotoxin starting on the MycoTOX Profile Results section. Please note that each value in this report needs to be considered in the context of your overall health and environment. Contact a qualified healthcare provider for further assistance in interpretation of results.

For information about mold species correlated to specific mycotoxins please refer to the detailed interpretations and/or the source chart found at the end of this test report.

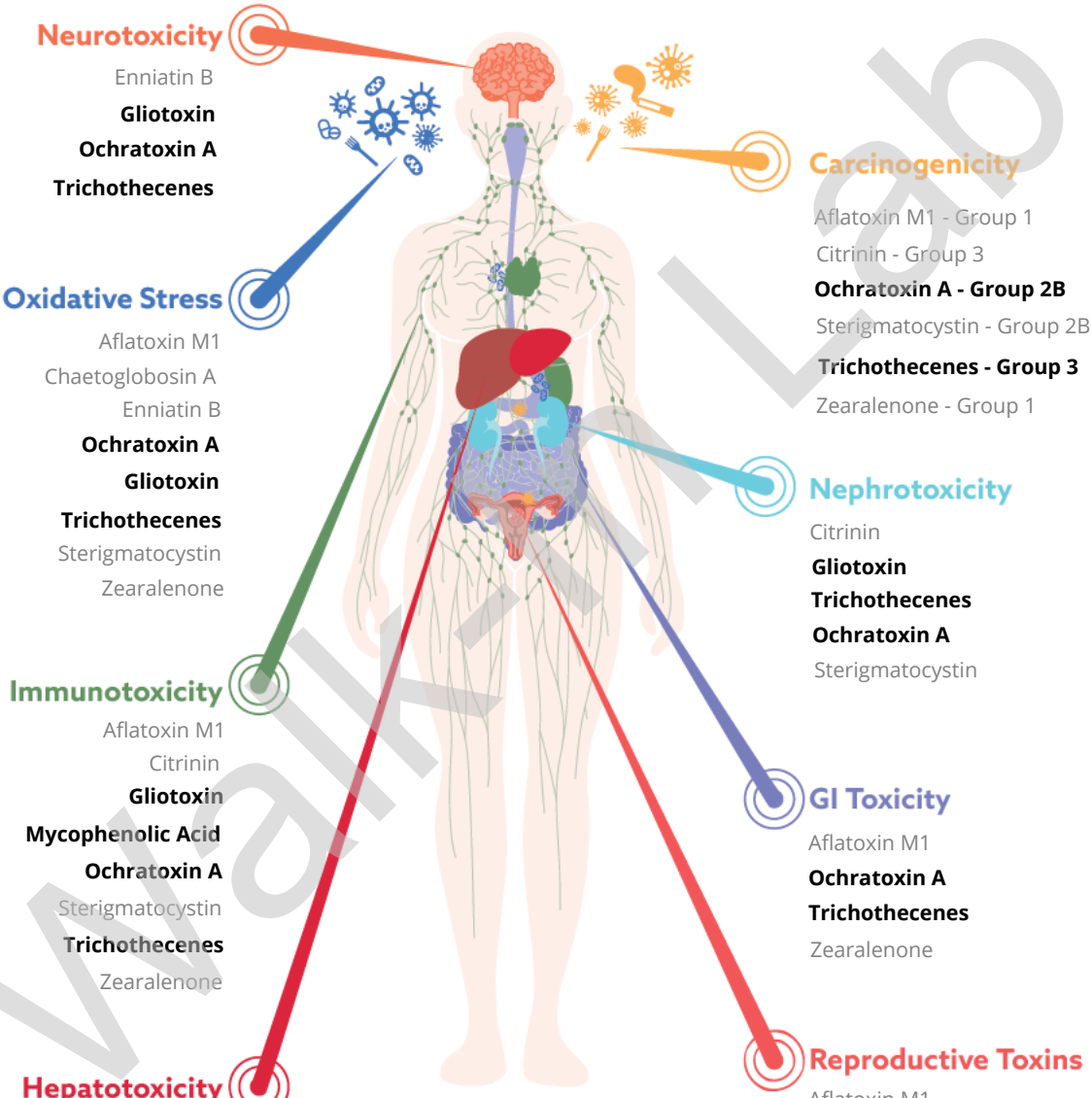
Color Key ● NORMAL ● HIGH

Creatinine Value: 100.00 mg/dl

	NORMAL RANGE (ng/g creatinine)	RESULTS (ng/g creatinine) | DL – Detectable Limit
Ochratoxin A (OTA)	**< 7.5**	**54.00**
Roridin E (ROE)	**< 0.2**	**56.00**
Verrucarin A (VRA)	**< 1.3**	**97.00**
Gliotoxin (GTX)	**< 200**	**205.00**
Mycophenolic Acid (MPA)	**< 37.4**	**40.00**

How Mycotoxins Affect Your Body

The image below visually represents the potential toxicity of specific mycotoxins and their health impact. Mycotoxin analyte(s) with elevated results are shown below in **bold, black text**.



Neurotoxicity

Enniatin B
Gliotoxin
Ochratoxin A
Trichothecenes

Oxidative Stress

Aflatoxin M1
Chaetoglobosin A
Enniatin B
Ochratoxin A
Gliotoxin
Trichothecenes
Sterigmatocystin
Zearalenone

Immunotoxicity

Aflatoxin M1
Citrinin
Gliotoxin
Mycophenolic Acid
Ochratoxin A
Sterigmatocystin
Trichothecenes
Zearalenone

Hepatotoxicity

Aflatoxin M1
Citrinin
Ochratoxin A
Trichothecenes
Zearalenone

Carcinogenicity

Aflatoxin M1 - Group 1
Citrinin - Group 3
Ochratoxin A - Group 2B
Sterigmatocystin - Group 2B
Trichothecenes - Group 3
Zearalenone - Group 1

Nephrotoxicity

Citrinin
Gliotoxin
Trichothecenes
Ochratoxin A
Sterigmatocystin

GI Toxicity

Aflatoxin M1
Ochratoxin A
Trichothecenes
Zearalenone

Reproductive Toxins

Aflatoxin M1
Chaetoglobosin A
Citrinin
Enniatin B
Ochratoxin A
Trichothecenes
Zearalenone

Mycotoxin impacts noted in the figure above have been compiled from a literature review of in vitro, in vivo animal and human studies.

MycoTOX PROFILE

MycoTOX Profile Results

The profile results offer a comprehensive breakdown of mycotoxin levels, grouped by class, which includes Aflatoxin, Ochratoxin, Trichothecene, Zearalenone, and Other Mycotoxins.

Color Key NORMAL HIGH

Creatinine Value: 100.00 mg/dl

	NORMAL RANGE (ng/g creatinine)	RESULTS (ng/g creatinine) | DL – Detectable Limit
AFLATOXIN		
Aflatoxin M1 (AFM1)	< 0.5	<DL
OCHRATOXIN		
Ochratoxin A (OTA)	< 7.5	54.00
TRICHOTHECENE		
Roridin E (ROE)	< 0.2	56.00
Verrucarin A (VRA)	< 1.3	97.00
ZEARALENONE		
Zearalenone (ZEA)	< 3.2	<DL

MycoTOX PROFILE

MycoTOX Profile Results – continued

Color Key NORMAL HIGH

Creatinine Value: 100.00 mg/dl

	NORMAL RANGE (ng/g creatinine)	RESULTS (ng/g creatinine) | DL – Detectable Limit
OTHER MYCOTOXINS		
Chaetoglobosin A (CHA)	< 10	3.00
Citrinin (Dihydrocitrinone DHC)	< 25	20.00
Enniatin B (ENB)	< 0.3	0.20
Gliotoxin (GTX)	< 200	205.00
Mycophenolic Acid (MPA)	< 37.4	40.00
Sterigmatocystin (STC)	< 0.4	0.10

Overview of MycoTOX Profile

WHAT IS THE MYCOTOX PROFILE?

The Mosaic Diagnostics MycoTOX Profile is a urine-based assay that assesses levels of 11 different mycotoxins, including metabolites of the most toxigenic classes: Aflatoxins, Ochratoxins, Trichothecenes, and Zearalenones.

This test was developed, and its performance characteristics determined by Mosaic Diagnostics Laboratory. It has not been cleared or approved by the US Food and Drug Administration, however, does comply with CLIA regulations for clinical use. The results should be interpreted in conjunction with the complete clinical picture, given patient history and presentation, and at the discretion of the medical provider.

WHY TEST FOR MYCOTOXINS?

Toxic secondary metabolites of many fungal (mold) species known as mycotoxins are ubiquitous in our environment. They interfere with cellular structures and important cellular processes. Most mycotoxins exert immunosuppressive effects, and many are cytotoxic (damaging to cells) and thus could potentially damage skin, lungs, and the gut microbiome. Exposure can result in mitochondrial damage as well as glutathione depletion, damage macrophage systems and raise sensitivity to bacterial endotoxins; and be associated with numerous health concerns.

- Common fungi sources of mycotoxins include species such as *Fusarium, Aspergillus, Penicillium, Alternaria,* and *Claviceps*.
- Exposure to mycotoxins may occur through a variety of routes such as inhalation, ingestion, and dermal contact from airborne mold spores, food contamination, and water-damaged building environments.
- Susceptibility to mycotoxins is influenced by an individual's age, sex, presence of other underlying diseases and/or exposures, nutritional status, and length of exposure.

Exposure to mycotoxins can have a potentially widespread toxic effect and can impact the nervous system, reproductive system, gastrointestinal system, kidney and liver; some mycotoxins are known carcinogens.

TESTING PLATFORM

Mosaic Diagnostics' MycoTOX Profile assay measures free (unconjugated) mycotoxins found in urine via an LC/MS-MS (Liquid Chromatography/Tandem Mass Spectrometry) platform. This method removes interfering and cross-reactive substances and is highly sensitive and specific for identifying and quantifying only the analytes (mycotoxins) of interest.

REFERENCE RANGE DESCRIPTION & DEPICTION

NORMAL

The result is normal relative to the reference population used to determine the reference ranges. The normal range was calculated using the mean + 2 times the standard deviation.

HIGH

The result is high relative to the reference population used to determine the reference ranges.

Interpretations

The information provided in this report, including the results and commentary, is intended solely for educational purposes and should not be construed as treatment recommendations. It is recommended that you consult with your healthcare provider for any necessary treatment. References related to this report and interpretations can be found at **MosaicDX.com/Test/MycoTOX-Profile**

AFLATOXINS

Color Key NORMAL HIGH

Aflatoxin M1 (AFM1) **<DL**
Normal Range <0.5

Aflatoxins are a group of toxic secondary metabolites of filamentous fungi, *Aspergillus flavus*, *A. nomius*, and *A. parasiticus*, and the most important mycotoxins in the world for human food and animal feed. AFM1 is a hydroxylated metabolite of AFB1 and is found in various food sources, particularly those derived from animals that have consumed aflatoxin-contaminated feed. The primary source of aflatoxin M1 contamination is milk and dairy products. AFB1 is extremely hepatotoxic and has been designated as a class 1 carcinogen by the World Health Association (WHO).

SOURCE

Aflatoxins have been found in samples taken from water-damaged buildings. Exposure to aflatoxins most often results from direct ingestion of contaminated foods such as cereals (maize, sorghum, millet, rice and wheat); oilseeds (soybean, sunflower and cotton); peanuts and tree nuts (almonds, walnuts, pistachios, coconut) and their butters; or from products created from animals that have been sustained on contaminated feed (meat, milk and milk products); and inhalation of aflatoxin dust particles – especially AFB1 – from contaminated foods in storage and processing facilities.

MECHANISM OF ACTION

Aflatoxins are metabolized via the cytochrome P450 (CYP450) pathway into reactive forms (reactive oxygen species or ROS) that preferentially bind mitochondrial DNA to form adducts and cause DNA damage with potential induction of hepatocarcinogenesis and induce apoptosis and disruption of ATP production via mutations of mitochondrial membranes. These ROS cause significant glutathione depletion with subsequent compromise of cellular antioxidant reserves. Aflatoxins also bind proteins and cause acute toxicity (aflatoxicosis); interfere with critical protein synthesis pathways; and can be transported across the placenta where they exert developmental and teratogenic effect.

HEALTH IMPACT

Carcinogenicity, GI Toxicity, Hepatotoxicity, Immunotoxicity (immunosuppressive), Oxidative Stress, Neurotoxicity, Reproductive Toxicity, Aflatoxicosis.

CLINICAL INSIGHT

AFB1, the most toxic of all aflatoxins, has a half-life of 87-91 hours in plasma, urine, and fecal excretion. P450 enzymes of Phase I and glucuronidation of Phase II liver detoxification pathways are necessary for its elimination; given that, support of these pathways is critical for therapeutically addressing aflatoxin exposure.

Interpretations – continued

OCHRATOXINS

Color Key NORMAL HIGH

Ochratoxin A (OTA) **54.00**
Normal Range <7.5

Ochratoxins A is a mycotoxin produced by several *Aspergillus* species (mainly *A. ochraceus*, *A. carbonarius*, and *A. niger*) and some *Penicillium* species (primarily *P. verrucosum*). OTA is known to induce nephrotoxicity in humans (e.g., glomerulonephritis or nephritic syndrome, Balkan endemic nephropathy and chronic interstitial nephropathy) and has been designated as a group 2B carcinogen by the International Agency for Research on Cancer (IARC).

SOURCE

Ochratoxins are highly ubiquitous in the food supply chain, and exposure is typically a result of direct consumption of contaminated foods (e.g., coffee, nuts, wine, grains, dairy) or via ingestion of contaminated animal products. Recently, the presence of OTA has been detected in bottled water, plant food supplements, and food coloring agents. According to the European Commission report, the estimated adult exposure to OTA is as follows: 44% cereals; 10% wine, 9% coffee, 7% beer, 5% cacao, 4% dried fruits, 3% meat, 3% spices, and 15% others. In addition, Ochratoxins have been found in dust samples from water-damaged buildings, offices, and ventilation systems.

MECHANISM OF ACTION

OTA appears to exert its negative health impacts via a complex series of actions including oxidative stress, mitochondrial impairment, inhibition of protein synthesis, and genotoxic effects (e.g., DNA single-strand breaks and DNA-OTA adduct formation).

HEALTH IMPACT

Carcinogenicity, GI Toxicity, Hepatotoxicity, Immunotoxicity, Nephrotoxicity, Neurotoxicity, Oxidative Stress, Reproductive toxicity, Apoptosis.

CLINICAL INSIGHT

OTA is the most toxic of all ochratoxins with the kidney being its main target organ. Given the significant public health and clinical concerns for its toxicity, it is one of only 20 mycotoxins monitored in food. Following ingestion of a single oral dosage from a contaminated food source, it has been noted to be very persistent in human beings with a half-life of 35 days. OTA is metabolized in the kidneys, liver, and intestines in humans with hydrolysis and hydroxylation being two major metabolic pathways. OTA increases oxidative stress. Preclinical studies show a positive impact when antioxidants such as NAC, CoQ10, GSH, melatonin and polyphenols are administered as well as other supportive nutrients like Vitamin C, Vitamin E, zinc, and magnesium.

Interpretations – continued

TRICHOTHECENES

Color Key NORMAL HIGH

Roridin E (ROE) 56.00
Normal Range <0.2

Verrucarin A (VRA) 97.00
Normal Range <1.3

Trichothecenes are a large group of mycotoxins that are produced by several fungal genera, which include *Cephalosporium, Fusarium, Myrothecium, Stachybotrys, Trichoderma, Tricothecium,* and *Verticimonosporium*. They are extremely potent inhibitors of protein synthesis and have been described to have wide-ranging negative systemic effects including immunotoxicity (immunosuppression), gastrointestinal toxicity, neurotoxicity, and dermatologic manifestations. They are classified as macrocyclic trichothecenes (Verrucarin A and Roridin E).

SOURCE

Trichothecenes have been found on board, wood and wallpaper in water damaged buildings. Food sources include: corn, popcorn, rice, rye, wheat, wheat flour, bread, buckwheat, barley, barley products, oats, sorghum, triticale, breakfast cereals, noodles, baby and infant foods, malt and beer.

MECHANISM OF ACTION

Much of the toxicity of tricothecenes is thought to be due to their inhibitiion of protein synthesis. Trichothecenes have been shown to increase reactive oxygen species (ROS) production in numerous tissue and organ systems with subsequent triggering of apoptosis and damage to mitochondrial function; inhibit protein transcription and translation; as well as impair immune cell proliferation.

HEALTH IMPACT

Carcinogenicity, GI Toxicity, Hepatotoxicity, Immunotoxicity, Nephrotoxicity, Neurotoxicity, Oxidative Stress, Reproductive Toxicity.

CLINICAL INSIGHT

Tricothecenes can be absorbed into the body through the skin, and also have the capability of passing through the blood-brain barrier. Considering the critical role oxidative stress plays in the toxicity of trichothecenes it is important to employ antioxidant agents to prevent induced oxidative stress.

Interpretations – continued

ZEARALENONE

Color Key NORMAL HIGH

Zearalenone (ZEA) **<DL**
Normal Range <3.2

Zearalenone is a secondary metabolite produced from the fungi *Fusarium graminearum*, *F. culmorum*, and *F. equiseti* which are known as regular contaminants of cereal crops worldwide. The main toxic effect of Zearalenone relates to its endocrine disruptive capabilities and as such, resultant negative reproductive effects in humans. When zearalenone is present in large quantities, it can disrupt conception, cause abortion, and result in other reproductive problems.

SOURCE

Zearalenone has been noted to be present in dust samples from water-damaged buildings with mold contamination. Food exposure to this mycotoxin can occur via many foods, including barley, corn, rice, peanuts, wheat, and animal feeds.

MECHANISM OF ACTION

Zearalenone can competitively bind to estrogen receptors; damage cellular integrity within the digestive system leading to increased intestinal permeability; and has been shown to down-regulate tumor suppressor genes important for controlling tumor growth within the digestive system.

HEALTH IMPACT

Carcinogenicity, GI Toxicity, Hepatotoxicity, Immunotoxicity, Oxidative Stress, Reproductive Toxicity.

CLINICAL INSIGHT

Two major pathways have been described to biotransform zearalenone: (1) hydroxylation which produces alpha-zearalenol, a stereoisomer that has a high affinity for estrogen receptors and is more toxic than ZEA; and (2) conjugation with glucuronic acid which produces glucuronides that are excreted into bile and eliminated from the body in urine and feces.

OTHER MYCOTOXINS

Color Key NORMAL HIGH

Chaetoglobosin A (CHA) **3.00**
Normal Range <10

Chaetoglobosin A is one of two mycotoxins produced by *Chaetomium globosum*, a member of the family *Chaetomiaceae* which consists of globally ubiquitous fungal genera that are found in soil and degraded cellulosic materials such as timber, plywood, and even plastics.

SOURCE

Chaetomium globosum is frequently isolated from materials found in water-damaged buildings. It is often referred to as 'black mold'. Food sources include corn, cornstalks, chestnuts, grapes, ginkgo biloba, apple juice and cherry juice.

MECHANISM OF ACTION

Chaetoglobosin A is a mycotoxic cytochalasin that exerts its toxic effects by binding to actin in cells, thereby inhibiting cell division, locomotion, and formation of cell surface projections.

HEALTH IMPACT

Chaetomium may become the dominant fungal colonizer in moist indoor environments, with potential to impact the development and exacerbation of asthma in children. Metabolites of *Chaetomium* spp. have been noted to negatively affect the physical defense mechanisms of the respiratory tract (ciliostatic effect). Members of this genus have also been associated with a wide array of other health impacts from dermal presentations (e.g., onychomycoses, phaeohyphomycosis) to opportunistic infections in immunocompromised individuals. Low levels of Chaetoglobosin A have been shown to be lethal in various tissue culture cell lines and animal studies.

CLINICAL INSIGHT

Research has found that chaetoglobosins possess a broad range of biological activities, including antitumor, antifungal, phytotoxic, fibrinolytic, antibacterial, nematicidal, anti-inflammatory, and anti-HIV activities.

OTHER MYCOTOXINS

Color Key NORMAL HIGH

Citrinin (Dihydrocitrinone DHC) **20.00**
Normal Range <25

Citrinin is a secondary fungal metabolite produced by several species of the mold genera *Aspergillus*, *Penicillium* and *Monascus*. It is mainly found in stored grains and in many plant products. Citrinin can be found in rice fermented with *Monascus* spp. (red yeast rice) which is used for meat preservation and food coloring in Asia and is also widely marketed as a food supplement.

SOURCE
Citrinin has been found on damp building materials, such as wood, insulation, and drywall. Citrinin occurs mainly in stored grains, cereals, and derivatives. It has also been found in olives, apples, spices, fruit and vegetable juices, beer, cheese, infant formulas, dry meat products, and red yeast rice.

MECHANISM OF ACTION
Citrinin can inhibit protein synthesis, preventing the formation of initiation complex necessary for protein synthesis. Damages DNA by forming adducts, these can lead to mutations, increasing the risk of cancer. Citrinin can induce oxidative stress by generating reactive oxygen species (ROS). Induces inflammation by activating macrophages. In humans, citrinin has been linked to infertility, reduced sperm count, and increased rates of miscarriage.

HEALTH IMPACT
Carcinogenicity, Hepatotoxicity, Immunotoxicity, Nephrotoxicity, Reproductive Toxicity.

CLINICAL INSIGHT
Citrinin is quickly metabolized to dihydrocitrinone, and excretion from the body is through the kidney and liver. Ochratoxin A and citrinin are often found in the same foods as they are produced by many of the same mold species. Citrinin and Ochratoxin A both cause nephropathy in animals and they have also been implicated as the cause of Balkan endemic nephropathy in humans.

Interpretations – continued

OTHER MYCOTOXINS

Color Key NORMAL HIGH

Enniatin B (ENB) **0.20**
Normal Range <0.3

Enniatin B is the most studied secondary metabolite of *Fusarium* fungi, including *F. avenaceum*. Due to potent cytotoxic effects, it has been observed to exhibit antibacterial, antihelminthic, antifungal, herbicidal, and insecticidal properties.

SOURCE

F. Avenaceum has been found worldwide on a variety of crops including cereals, peaches, apples, pears, potatoes, peanuts, peas, asparagus, and tomatoes. Enniatins have also been found in fish, dried fruits, cocoa, and coffee products. ENB has also been found in water-damaged buildings.

MECHANISM OF ACTION

Enniatin B is thought to exert toxicity via its ability to act as an ionophore, changing ion transport across cellular membranes and disrupting the ionic selectivity of cell walls. ENB exerts its cytotoxic activities via oxidative stress, mitochondrial modification and cell cycle disruption, and inducement of apoptotic cell death.

HEALTH IMPACT

Neurotoxicity, Oxidative Stress, Reproductive Toxicity.
Despite its demonstrated mammalian cell line cytotoxic activity, European Food Safety Authority stated that acute exposure to ENNs, such as ENB, does not indicate concern for human health, but a concern might be the chronic exposure – especially given emerging data that suggest their toxicity can be enhanced by the concomitant presence of other ENNs or mycotoxins.

CLINICAL INSIGHT

ENB has been shown to have endocrine-disrupting properties as well as the ability to cross the blood-brain barrier in in vitro assays. As is the case with many of the mycotoxins, ENB is being explored for potential pharmacologic antimicrobial applications against pathogens of the intestinal tract.

Interpretations – continued

OTHER MYCOTOXINS

Color Key NORMAL HIGH

Gliotoxin (GTX) **205.00**
Normal Range <200

Gliotoxin is a sulfur-containing mycotoxin that belongs to a class of naturally occurring compounds produced by several species of fungi, (e.g., ***Aspergillus fumigatus***, ***Tricoderma*** and ***Penicillium*** species) especially those of marine origin. It is suspected to be an important virulence factor in ***A. fumigatus***.

SOURCE

Airborne ***Aspergillus*** fungal spores are ubiquitous in many environments making potential exposure to gliotoxin common. Gliotoxins have been found on linoleum flooring and wallpaper in water damaged buildings, as well as silage and other animal food stocks.

MECHANISM OF ACTION

Gliotoxin may exert a toxic effect via cellular organic anion and cation transporters suggesting that these transporters were the possible entrance pathway for mycotoxins in the kidney and liver, leading to the induction of adverse effects in humans. Its primary mechanisms of cellular toxicity may be related to the production of reactive oxygen species (ROS) via redox cycling and the characteristic presence of an internal disulfide bridge within the toxin that allows for the binding and inactivation of proteins. Gliotoxin has been shown to inhibit phagocytosis by neutrophils and transcription factor NF-kB, causing immunosuppression, which can potentially further influence cytokine production and mast cell degranulation.

HEALTH IMPACT

Gliotoxin has been isolated from the sera of patients with invasive aspergillosis, suggesting a link between gliotoxin secretion and fungal pathogenicity. Ingestion, direct contact (e.g., ocular or dermal), or inhalation may all result in acute toxicity. Gliotoxin has been shown to suppress the activity of macrophages against identification, ingestion, and destruction of pathogens (immunosuppression). It has also been demonstrated to have neurotoxic, nephrotoxic, and oxidative stress impact.

CLINICAL INSIGHT

Given the ubiquitous presence of spores from ***A. fumigatus*** (the most clinically relevant source of gliotoxin), toxin exposure is likely to occur either by accidental ingestion or by in situ generation in those with existing fungal infections.

Interpretations – continued

OTHER MYCOTOXINS

Color Key NORMAL HIGH

Mycophenolic Acid (MPA) **40.00**
Normal Range <37.4

Mycophenolic Acid is a mycotoxin produced by several species of soil-occupant fungi *Penicillium*. It is currently used as an immunosuppressive agent following organ transplantation.

SOURCE
MPA-producing strains are very common in forest soil, greenhouse, and farmland globally. It has been found in moldy foods, fruits, and dairy products – as well as in water-damaged buildings.

MECHANISM OF ACTION
MPA is thought to exert its effects by (1) depleting guanosine nucleotides preferentially in T and B lymphocytes and inhibiting their proliferation, thereby suppressing cell-mediated immune responses and antibody formation; and (2) by inhibiting the glycosylation and expression of adhesion molecules, and the recruitment of lymphocytes and monocytes into sites of inflammation.

HEALTH IMPACT
Immunotoxicity (immunosuppressant).
One study assessing indoor air water condensates found the presence of MPA in initial and follow-up urine samples of office workers with symptoms of Sick Building Syndrome. The study's authors suggest that the presence of mycotoxins in indoor air was linked to the morbidity of the office occupants.

CLINICAL INSIGHT
MPA is used as an immunosuppressive drug for the prevention of transplant rejection in the form of sodium mycophenolate (Myfortic™, Novartis) and a pro-drug, mycophenolate mofetil (CellCept™, Roche) – and as a result, its levels may be elevated on diagnostics in patients using these pharmaceuticals.

OTHER MYCOTOXINS

Color Key NORMAL HIGH

Sterigmatocystin (STC) **0.10**
Normal Range <0.4

Sterigmatocystin is a mycotoxin produced by several species of fungi including *Penicillium*, *Fusarium*, *Bipolaris* and even *Stachybotrys* and *Chaetomium* – though it is produced in particularly potent amounts by *Aspergillus* versicolor, one of the most frequent fungal indoor environment contaminants. Sterigmatocystin is a precursor of aflatoxin B1 in those cases where food sources are contaminated with fungi capable of producing aflatoxins.

SOURCE

A. versicolor is commonly observed to grow on most construction and decoration materials under appropriate environmental conditions – and Sterigmatocystin has been recovered on building materials, in dust, and in air samples. It is estimated that in northern Europe and North America, 20-40% of buildings have visible fungal growth. It has been found in a number of food products: corn, wheat, barley, peanuts, pecan nuts, soya beans, green coffee beans, ham, and cheese.

MECHANISM OF ACTION

Sterigmatocystin's carcinogenicity appears to be the result of its ability to bind to DNA and form DNA adducts. DNA adduct formation causes enhanced production of reactive oxygen species and an imbalance in antioxidant defense, leading to enhanced lipid peroxidation that causes cell damage.

HEALTH IMPACT

Carcinogenicity, GI Toxicity, Hepatotoxicity, Immunotoxicity, Nephrotoxicity, Oxidative Stress.

CLINICAL INSIGHT

Despite their similarity in chemical structure, Sterigmatocystin has been noted to be a less potent carcinogen than Aflatoxin B1 (AFB1). It is classified as a Group 2B carcinogen by the International Agency for Research on Cancer.

Sources of Mycotoxins

MYCOTOXIN	GENUS/SPECIES	SOURCES	POTENTIAL TOXICITY
Aflatoxins	*Aspergillus flavus* *A. nomius* *A. parasiticus* *Penicillium*	Water-damaged buildings (AFB1, AFB2). Corn, rice, pasta, Brazil nuts, peanuts, peanut butter, pistachios, cassava, tobacco, cottonseed cake, oilseeds, figs, milk, cheese, butter, yoghurt, spices, baby foods.	Carcinogenicity – Group 1 GI toxicity Hepatotoxicity Immunotoxicity Oxidative stress Reproductive toxicity
Ochratoxins	*Aspergillus* *A. ochraceus* *Penicillium* *P. nordium* *P. verrucosum*	Dust samples of water-damaged buildings, offices and ventilation systems (OTA). Corn, rice, rye, wheat, buckwheat, barley, millet, oats, cereals, raisins, currants, nuts, coffee, cocoa, spices, beer, pork, cheese, smoked and salted dried fish, dried beans, chickpeas, dried fruit, sesame seeds, grapes and grape products, wines, apples, pears, peaches, citrus, figs, strawberries.	Carcinogenicity – Group 2B GI toxicity Hepatotoxicity Immunotoxicity Nephrotoxicity Neurotoxicity Oxidative stress Reproductive toxicity
Trichothecenes	*Cephalosporium* *Fusarium* *Myrothecium* *Stachybotrys* *Trichoderma* *Trichothecium* *Verticimonosporium*	Water-damaged buildings (trichothecenes). Corn, popcorn, rice, rye, wheat, wheat flour, bread, buckwheat, barley, barley products, oats, sorghum, triticale, breakfast cereals, noodles, baby and infant foods, malt, beer.	Carcinogenicity – Group 3 GI toxicity Hepatotoxicity Immunotoxicity Nephrotoxicity Neurotoxicity Oxidative stress Reproductive toxicity
Zearalenones	*Fusarium* *F. culmorum* *F. equiseti* *F. graminearum*	Dust samples from water-damaged buildings. Corn, wheat, wheat flour, bread, breakfast cereals, noodles, rice, barley, oats, sorghum, walnuts, milk, corn beer, meat, animal-feed products, vegetable oil.	Carcinogenicity – Group 1 GI toxicity Hepatotoxicity Immunotoxicity Oxidative stress Reproductive toxicity

Carcinogenicity designations based on IARC – World Health Organization identification of carcinogenic hazards to humans.

Sources of Mycotoxins – continued

MYCOTOXIN	GENUS/SPECIES	SOURCES	POTENTIAL TOXICITY
Chaetoglobosin A	*Chaetomium globosum*	Common in water damaged buildings. Corn, cornstalks, chestnut, grapes, ginkgo biloba, apple juice, cherry juice.	Oxidative stress Reproductive toxicity (ciliostatic)
Citrinin	*Aspergillus* *A. flavus* *A. ochraceus* *Penicillum* *P. citrinin* *P. verrucosum*	Wood, insulation and drywall in water-damaged buildings. Grains, rice, cereals, cereal derivatives, olives, apples, spices, friut and vegetable juices, beer, cheese, infant formulas, dry meat products, red yeast rice.	Carcinogen – Group 3 Hepatotoxicity Immunotoxicity Nephrotoxicity Reproductive toxicity
Enniatin B	*Fusarium*	Water-damaged buildings. Cereals, peaches, apples, pears, potatoes, peanuts, peas, asparagus, tomatoes, fish, dried fruits, nuts, spices, cocoa, coffee.	Neurotoxicity Oxidative stress Reproductive toxicity
Gliotoxin	*A. fumigatus* *Penicillium* *Trichoderma*	Linoleum flooring and wallpaper in water-damaged buildings. Silage and other animal food stocks.	Immunotoxicity Nephrotoxicity Neurotoxicity Oxidative stress
Mycophenolic Acid	*Penicillium*	Water-damaged buildings. Blue cheese, gorgonzola, barley, flour, baked goods, refrigerated dough, meat, meat products	Immunotoxicity
Sterigmatocystin	*A. versicolor* *Bipolaris* *Chaetomium* *Fusarium* *Penicillium* *Stachybotrys*	Wallpaper and carpeting in water-damaged buildings. Corn, wheat, barley, peanuts, pecan nuts, soya beans, green coffee beans, ham, cheese.	Carcinogenicity – Group 2 Immunotoxicity Nephrotoxicity Oxidative stress

Carcinogenicity designations based on IARC – World Health Organization identification of carcinogenic hazards to humans.

MycoTOX PROFILE

Dr. L. G. Bates-Dubrow, PhD, CC(NRCC), Lab Director | CLIA 17D0919496
Mosaic Diagnostics | 9221 Quivira Road, Overland Park, KS 66215
MosaicDX.com

MOSAIC DIAGNOSTICS