The Great Plains Laboratory, Inc.

William Shaw, Ph.D Director | 11813 W. 77th Street, Lenexa, KS 66214 | (913) 341-8949 | Fax (913) 341-6207

Requisition #:	9000000	**Physician Name:**	NO PHYSICIAN
Patient Name:	Sample	**Date of Collection:**	Jul 13, 2021
Date of Birth:	Aug 15, 2008	**Time of Collection:**	Not Given
Gender:	M	**Print Date:**	Oct 6, 2021
Specimen Id.:	9000000-2		

Mycotox Profile

Creatinine Value: **190.08 mg/dl**

Metabolite	Results (ng/g creatinine)	Normal Range *	Abnormal Range
Aspergillus			
Aflatoxin-M1	15.45	< 0.5	▲ 0.5
Ochratoxin A	38.67	< 7.5	▲ 7.5
Gliotoxin	256.78	< 200	▲ 200
Penicillium			
Sterigmatocystin	12.44	< 0.4	▲ 0.4
Mycophenolic Acid	67.80	< 37.4	▲ 37.4

**** The normal range was calculated using the median + 2 times the standard deviation***

Testing performed by The Great Plains Laboratory, Inc., Lenexa, Kansas. The Great Plains Laboratory has developed and determined the performance characteristics of this test. The test has not been evaluated by the U.S. Food and Drug Administration. The FDA does not currently regulate such testing.

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Mycotox Profile

Metabolite	Results (ng/g creatinine)	Normal Range *	Abnormal Range
Stachybotrys			
Roridin E	9.22	< 0.2	▲ 0.2
Verrucarin A	6.89	< 1.3	▲ 1.3
Fusarium			
Enniatin B	8.77	< 0.3	▲ 0.3
Zearalenone	5.34	< 3.2	▲ 3.2

** The normal range was calculated using the median + 2 times the standard deviation*

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Chaetomium globosum

Chaetoglobosin A	**21.76**	**< 10**	[redacted]

▲ 10

Multiple Mold Species

Citrinin (Dihydrocitrinone DHC)	**32.98**	**< 25**	[redacted]

▲ 25

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Aflatoxin M1: Aflatoxin M1 (AFM1) is the main metabolite of aflatoxin B1, which is a mycotoxin produced by the mold species Aspergillus. Aflatoxins are some of the most carcinogenic substances in the environment. Aflatoxin susceptibility is dependent on multiple different factors such as age, sex, and diet. Aflatoxin's may source is water damage in buildings. Aflatoxin can also be found in beans, corn, rice, tree nuts, wheat, milk, eggs, and meat. Aflatoxin can lead to liver damage, cancer, mental impairment, abdominal pain, hemorrhaging, coma, and death. Aflatoxin has been shown to inhibit leucocyte proliferation. Clinical signs of aflatoxicosis are non-pruritic macular rash, headache, gastrointestinal dysfunction (often extreme), lower extremity edema, anemia, and jaundice. Treatment should include fluid support to prevent dehydration. The toxicity of Aflatoxin is increased in the presence of Ochratoxin and Zearalenone. Aflatoxin is removed through the glutathione S-transferase system. This system can conjugate activated aflatoxin with reduced glutathione. This leads to aflatoxin becoming more water soluble, which assists in its excretion. It is theorized that variations in levels of P450s, glutathione transferase, and transporters can account for the variation in response patients have to aflatoxin exposure. Retesting is recommended after 3-6 months of treatment.

(PMID: 11724948, 12628519, 27017951, 26596546, 15027811, 15531656, 12573908, 20381597, 27470613, 18286403, 10050868, 7585637, 16762476, 16019795, 18286403)

Ochratoxin: Ochratoxin A (OTA) is a nephrotoxic, immunotoxic, and carcinogenic mycotoxin. This chemical is produced by molds in the Aspergillus and Penicillium families. Exposure is done primarily through water damaged buildings. Minimal exposure can occur through contaminated foods such as cereals, grape juices, dairy, spices, wine, dried vine fruit, and coffee. Exposure to OTA can also come from inhalation exposure in water-damaged buildings. OTA can lead to kidney disease and adverse neurological effects. Studies have shown that OTA can lead to significant oxidative damage to multiple brain regions and is highly nephrotoxic. Dopamine levels in the brain of mice have been shown to be decreased after exposure to OTA. Some studies have hypothesized that OTA may contribute to the development of neurodegenerative diseases such as Alzheimer's and Parkinson's. Treatment should be aimed at removing the source of exposure. Agents such as oral cholestyramine, charcoal, and phenylalanine can help prevent the absorption of these toxins from food. Antioxidants such as vitamins A, E, C, NAC, rosmarinic acid, and liposomal glutathione alone or in combination have been shown to mitigate the oxidative effects of the toxin. Bentonite or zeolite clay is reported to reduce the absorption of multiple mycotoxins found in food, including OTA. Studies have also shown that OTA is present in sweat, which supports the use of sauna as a treatment to increase the excretion of OTA. Retesting is recommended after 3-6 months of treatment.

(PMID 17195275, 16293235, 27521635, 22069626, 24792326, 22253638, 16140385, 2467220, 16844142, 19148691, 22069658, 16019795, 18286403, 15781206, 11439224, 17092826, 32710148)

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Sterigmatocystin: Sterigmatocystin (STC) is a mycotoxin that is closely related to aflatoxin. STC is produced from several species of mold such as Aspergillus, Penicillium, and Bipolaris. STC is considered to be carcinogenic, particularly in the cells of the GI tract and liver. STC has been found in the dust from damp carpets. It is also a contaminant of many foods including grains, corn, bread, cheese, spices, coffee beans, soybeans, pistachio nuts, and animal feed. In cases of lung aspergilloma, STC has been found in human tissue specimens. The toxicity of STC affects the liver, kidneys, and immune system. Tumors have been found in the lungs of rodents that were exposed to STC. Oxidative stress becomes measurably elevated during STC exposure which causes a depletion of antioxidants such as glutathione, particularly in the liver. Because STC is structurally similarity to Aflatoxin, many of the same therapies will be effective. A diet of carrots, parsnips, celery, and parsley may reduce the carcinogenic effects of STC. Bentonite or zeolite clay is reported to reduce the absorption of multiple mycotoxins found in food, including STC. Supplementation with chlorophyllin, zinc, and vitamins A, E, and C has been used to treat exposure to STC. Retesting is recommended after 3-6 months of treatment.

(PMID: 10855723, 19998385, 21287681, 23705030, 24514428, 12147486, 15027811, 12244755, 11727790, 12725069, 18286403, 10050868, 7585637, 16762476, 16019795, 18286403, 15781206, 11439224, 17092826, 11724948, 12628519, 27017951, 25176419, 11727790)

Zearalenone: Zearalenone (ZEA) is mycotoxin that is produced by the mold species Fusarium, and has been shown to be hepatotoxic, haematotoxic, immunotoxic, and genotoxic. ZEA exposure is mostly through water damaged buildings, although ZEA is commonly found on several foods in the US, Europe, Asia, and Africa. The foods known to be contaminated with ZEA include wheat, barley, rice, and maize. ZEA has estrogenic activity and exposure to ZEA can lead to reproductive changes. ZEA estrogenic activity is higher than that of other non-steroidal isoflavones (compounds that have estrogen-like effects) such as soy and clover. ZEA exposure can result in thymus atrophy and alter spleen lymphocyte production, as well as impaired lymphocyte immune response, which leads to patients being susceptible to disease. ZEA is deactivated primarily through glucuronidation; individuals with impairments to this pathway will be much more susceptible to this compound even at very low levels. Treatment with the antioxidants lycopene and resveratrol has been beneficial in negating the harmful effects of ZEA in several studies. Retesting is recommended after 3-6 months of treatment.

(PMID: 17045381, 19330061, 11384734, 1387742, 698923, 1599403, 2276698, 22645433, 24632555, 6239410, 6235161, 24503513, 25682699, 27489133, 15781206, 11439224, 17092826, 16095665, 16782537, 17561436, 11245394)

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Citrinin (Dihydrocitrinone DHC): Citrinin (CTN) is a mycotoxin that is produced by the mold genera Aspergillus, Penicillium, and Monascus. CTN exposure can lead to nephropathy, because of its ability to increase permeability of mitochondrial membranes in the kidneys. The three most common exposure routes are through ingestion, inhalation, and skin contact. CTN has been shown to be carcinogenic in rat studies. Multiple studies have linked CTN exposure to a suppression of the immune response. Retesting is recommended after 3-6 months of treatment.

(PMID: 11567776, 24048364, 10788357)

Roridin E: Roridin E (ROE) is a macrocyclic trichothecene produced by the mold species Fusarium, Myrothecium, and Stachybotrys (i.e. black mold). Trichothecenes are frequently found in buildings with water damage but can also be found in contaminated grain. This is a very toxic compound, which inhibits protein biosynthesis by preventing peptidyl transferase activity. Trichothecenes are considered extremely toxic and have been used as biological warfare agents. Even low levels of exposure to macrocyclic trichothecenes can cause severe neurological damage, immunosuppression, endocrine disruption, cardiovascular problems, and gastrointestinal distress. Treatment measures are often aimed at the prevention of their absorption. Nebulized and intranasal glutathione is beneficial for those exposed to inhaled toxin. Transdermal and liposomal glutathione may also be helpful, especially in combination with sequestrants. Sequestrants bind to toxins in the GI tract making them unavailable for reabsorption. Retesting is recommended after 3-6 months of treatment.

(PMID: 18007011, 23710148, 15342078, 19333439, 20549560, 3376149)

Verrucarin A: Verrucarin A: Verrucarin A (VRA) is a macrocyclic trichothecene mycotoxin produced from Stachybotrys, Fusarium, and Myrothecium. Trichothecenes are frequently found in buildings with water damage but can also be found in contaminated grain. VRA is a small, amphipathic molecule that can move passively across cell membranes. The primary tissues affected by VRA are intestinal and gastric mucosa, bone marrow, and spleen. VRA causes damage to human cells by inhibiting protein and DNA synthesis, disrupting mitochondrial functions, and by producing oxidative stress (due to generation of free radicals). Exposure to VRA can cause immunological problems, vomiting, skin dermatitis, and hemorrhagic lesions. Nebulized and intranasal glutathione is beneficial for those exposed to inhaled toxin. Transdermal and liposomal glutathione may also be helpful, especially in combination with sequestrants. Sequestrants bind to toxins in the GI tract making them unavailable for reabsorption. These agents are not absorbed and work best for patients with GI symptoms or those whose toxin exposure is coming from food. Retesting is recommended after 3-6 months of treatment.

(PMID: 23710148, 18007011, 15342078, 19333439, 20549560, 3376149)

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Mycophenolic Acid: Mycophenolic Acid (MPA) is an antifungal, antibacterial, and antiviral mycotoxin acid. It is produced by the Penicillium fungus. MPA is an immunosuppressant which inhibits the proliferation of B and T lymphocytes. MPA exposure can increase the risk of opportunistic infections such as clostridia and Candida . MPA is associated with miscarriage and congenital malformations when the woman is exposed in pregnancy. Retesting is recommended after 3-6 months of treatment. Mycophenolic acid is used as a pharmaceutical under the brand names CellCept, Myfortic and others. Mycophenolic acid cannot be used as a marker for mold if the person is taking the pharmaceutical.

(PMID: 858824, 28646113, 27809954, 27599910)

Chaetoglobosin A: Chaetoglobosin A (CHA) is produced by the mold Chaetomium globosum (CG). CG is commonly found is homes that have experienced water damage. Up to 49% of water-damaged buildings have been found to have CG. CHA is highly toxic, even at minimal doses. CHA disrupts cellular division and movement. Most exposure to CG is through the mycotoxins because the spores tend not to aerosolize. Exposure to CHA has been linked to neuronal damage, peritonitis, and cutaneous lesions. Retesting is recommended after 3-6 months of treatment.

Enniatin B: Enniatin B (ENB) is a fungal metabolite categorized as cyclohexa depsipeptides toxin produced by the fungus Fusarium. The main cause of exposure is from water damaged buildings, although this strain of fungus is one of the most common cereal contaminants. Grains in many different countries have recently been contaminated with high levels of enniatins. The toxic effects of Enniatin are caused by the inhibition of the acyl-CoA cholesterol acyltransferase, depolarization of mitochondria, and inhibition of osteoclastic bone resorption. Enniatin has antibiotic properties and chronic exposure may lead to weight loss, fatigue, and liver disease. Sequestrants bind to mycotoxins in the GI tract making them unavailable for reabsorption. These agents are not absorbed and work best for patients with GI symptoms or those whose toxin exposure is coming from food. Retesting is recommended after 3-6 months of treatment.

(PMID: 18274964, 16730043, 21622627, 23710148)

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Gliotoxin: Gliotoxin (GTX) is produced by the mold genus Aspergillus. Aspergillus spreads in the environment by releasing conidia which are capable of infiltrating the small alveolar airways of individuals. In order to evade the body's defenses Aspergillus releases Gliotoxin to inhibit the immune system. One of the targets of Gliotoxin is PtdIns (3,4,5) P3. This result in the downregulation of phagocytic immune defense, which can lead to the exacerbation of polymicrobial infections. Gliotoxin impairs the activation of T-cells and induces apoptosis in monocytes and in monocyte-derived dendritic cells. These impairments to dendritic cells can lead to multiple neurological syndromes. Retesting is recommended after 3-6 months of treatment.

(PMID: 16712786, 27048806, 21575912, 23278106).