

Dr. Jasmyne

Mitochondria & Mycotoxins

Identifying and Understanding the Impact of Mycotoxin Exposure on Mitochondrial Function

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Dr. Jasmyne

Hi! I'm Dr. Jasmyne



Education

- Graduated from the University of Bridgeport in Naturopathic Medicine and Human Nutrition in $_{\circ}$ 2017
- Bachelor Degree in Chemistry from Alabama Agricultural and Mechanical University



Experience

- Clinical Educator at Mosaic 0 Diagnostics
- **Current Naturopathic Doctor** Consultant with Nutrafol
- at Fullscript



• Current Medical Science Liaison

Continued Growth

- Owner and operator of Natural Lyfe Health Consulting.
- I also engage in regular learning in the world of natural health



Objectives

- Importance
- and Mitochondrial Dysfunction
- Mitochondrial Dysfunction
- Health

 Define Mycotoxins and Their Sources • Explain Mitochondrial Function and Its

• Explore the Connection Between Mycotoxins

Identify Symptoms and Health Impacts of

 Discuss Diagnostic Methods for Mycotoxin Exposure and Mitochondrial Dysfunction

 Present Strategies for Mitigating Mycotoxin Exposure and Supporting Mitochondrial

What Are Mycotoxins?



- Toxic substances normally produced by mold
 - Molds are fungi similar to yeast
 - Yeast is unicellular and can be round, oval, or filamentous.
 - mold is multicellular and appears as masses of hyphae or mycelium
 - Yeasts are microscopic fungi consisting of solitary cells that reproduce by budding.
 - Molds, in contrast, occur in long filaments known as hyphae, which grow by apical extension
 - Appearance: Molds are fuzzy and spread across surfaces, while yeast forms smooth colonies.
 - Health Impact: Molds may produce harmful toxins; yeast can cause infections, especially in moist areas of the body.



Sources of Mycotoxins

We can get exposed to mycotoxins from various sources

Foods

NUTS AND GRAINS

these foods go through a drying process that increases risk of mold growth some are grown in the soil that exposes them to molds

COFFEE, WINE, CHEESE

production processes and fermentation increases risk of mold growth and mycotoxin exposure

Environment

BUILDINGS

any building with water supply has the potential for mold high humidity can increase risk of growth building material are food for molds

VEHICLES

flooded vehicles grow mold faulty A/C can grow mold then blow it throughout the cabin change air filters regularly





Mycotoxin Exposure

| INHALATION | |
|------------|--|
| | |

take

eating too many mold contaminated foods

brushing up against or using products that have mold growth on our skin- mold car seats, towels, clothes, books, etc

INGESTION

DERMAL CONTACT



Toxic Effects of Mycotoxins

mycotoxins have a myriad of toxic effects. they do nothing positive to our bodies and over exposure is detrimental

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NEUROTOXIN

disrupt nerve conduction leading to neuropathy and cognition issues

ENDOCRINE TOXIN

impairs hormonal balance by mimicking estrogen leading to premature puberty, infertility, PCOS

5



IMMUNOTOXINS

immunosuppressive effects that disrupt immune activation, shift in autoimmune Th1 and Th2 imbalance

CARDIOTOXINS

3

vascual, muscular, endothelial, protein synthesis disruptions. Increased inflammation and fibrosis

CARCINOGENS

incresaes risk of certain cancers like liver, kidney, esophageal, breast



Functions of the Mitochondria

Mitochondria are the cell's powerhouses, responsible for producing energy in the form of ATP (adenosine triphosphate) through the process of cellular respiration. They also regulate key cellular processes like apoptosis (cell death, calcium storage, and the generation of reactive oxygen species.

ENERGY PRODUCTION

ATP), the main energy currency of the cell, through a process called oxidative phosphorylation. This occurs in the inner mitochondrial membrane, where the electron transport chain creates a proton gradient that drives the synthesis of ATP.

CALCIUM HOMEOSTASIS

regulate intracellular calcium levels, which are critical for various cellular processes, including muscle contraction, neurotransmitter release, and cell signaling. They act as buffers, absorbing and releasing calcium as needed. They release cytochrome c and other proapoptotic factors in response to cellular stress, triggering the apoptotic pathway.

REGULATION OF CELLULAR METABOLISM

They are involved in the breakdown of carbohydrates (glycolysis), fats (betaoxidation), and proteins to produce ATP.

APOPTOSIS



Functions of the Mitochondria

Regulation of Cell Growth and Differentiation:
 Mitochondria influence cell proliferation and differentiation by regulating energy production and the balance of metabolic intermediates. They play a role in signaling pathways that control cell cycle progression and differentiation.

PRODUCTION OF REACTIVE OXYGEN SPECIES (ROS)

generate reactive oxygen species (ROS), such as superoxide. While ROS play a role in cell signaling and defense against pathogens, excessive ROS can lead to oxidative stress and damage cellular components.

SYNTHESIS OF METABOLIC PRECURSORS

involved in the synthesis of several important molecules, including heme (a component of hemoglobin) and steroid hormones. They also contribute to the urea cycle and gluconeogenesis (glucose production).

REGULATION OF CELLULAR REDOX STATE

maintain the balance between oxidation and reduction (redox state) within cells, which is crucial for cellular function and defense against oxidative damage.

In brown adipose tissue, mitochondria are involved in thermogenesis, where they produce heat instead of ATP. This process is essential for maintaining body temperature, especially in newborns and during cold exposure.



THERMOGENESIS

Common Signs and Symptoms of Mitochondrial Dysfunction

MUSCLE WEAKNESS AND FATIGUE

decrease recovery of muscle tissue from simple movement or exercise intolerance

MEMORYAND SENSORY ISSUES

reduced cognition, hearing or vision issues



NEUROLOGIC anxiety, depression, irritability, developmental, learning difficulties

nausea, vomiting, diarrhea, constipation



GI DISTRESS





- Headaches
- Seizures
- Alzheimer's disease
- Parkinson's disease
- Obesity
- Type 2 Diabetes
- Chronic Fatigue Syndrome
- Fibromyalgia
- Osteoporosis and Sarcopenia
- NAFLD
- Cancer
- Peripheral Neuropathy





Mycotoxin--> Mitochondria

OCHRATOXIN

mitochondrial function disturbance, apoptotic and autophagic cell death and also induce mitochondrial biogenesis

ALFLATOXIN

disrupts the levels <u>phospholipids</u>, mitochondrial membrane which affect the mitochondrial membrane transport. reduces mitochondrial membrane potential and promotes mitochondrial permeability, induced oxidative stress and apoptosis

ZEARALENONE

increases cell division, autophagy, LDH activity



Mycotoxin--> Mitochondria

MYCOPHENOLIC ACID

induced hyperpolarization of the mitochondrial membrane potential and the translocation of Cytochrome C and Bax proteins from the cytoplasm to the mitochondria





inhibits several enzymes, including malate dehydrogenase and glutamate dehydrogenase, as well as the ATP synthase complex involved in the How do we evaluate this?

Functional testing option support practitioners

ORGANIC ACID TEST

organic acids supply information about mitochondrial function as it relates to glucose, fat, and amino acid usage

MYCOTOXIN TESTS

urine test capture what mycotoxins are being excreted from the body. unbound mycotoxins help us understand what has not been conjugated by the liver and removed unchanged

ENVIRONMENTAL TESTS

identifies environmental source of exposure. once you stop the exposure you can get on top of the detox process

Mitochondrial Function Evaluation

| Glycolytic Cycle Metabolites | | | |
|------------------------------|-------|-----|--|
| | | | |
| 22 Lactic | ≤ 48 | 22 | |
| 23 Pyruvic | ≤ 9.1 | 2.4 | |

- Low levels show proper usage of these metabolites for energy production
- Lactic Acid:
 - elevated: increased production with decreased transformation
 - lack of oxygen delivery to cells
 - iron deficiency
 - microorganism production
 - poor oxygenation states
- Pyruvate:
 - elevated increased production with decreased usage
 - poor B vitamin availability
 - B2, Bl, B3
 - poor oxygenation







Mitochondrial Markers - Krebs Cycle Metabolites

| 24 | Succinic | | ≤ | 9.3 | н | 18 | |
|----|--|------|---|------|---|------|--|
| 25 | Fumaric | | ≤ | 0.94 | | 0.82 | |
| 26 | Malic | 0.06 | - | 1.8 | | 1.7 | |
| 27 | 2-Oxoglutaric | | ≤ | 35 | | 11 | |
| 28 | Aconitic | 6.8 | - | 28 | | 14 | |
| 29 | Citric | | ≤ | 507 | н | 610 | |
| Ι | Mitochondrial Markers - Amino Acid Metabolites | | | | | | |
| | | | | | | | |
| 30 | 3-Methylglutaric | | ≤ | 0.76 | | 0.35 | |
| 31 | 3-Hydroxyglutaric | | ≤ | 6.2 | | 5.4 | |
| 32 | 3-Methylglutaconic | | ≤ | 4.5 | | 1.4 | |







Ketone and Fatty Acid Oxidation

| 43 | 3-Hydroxybutyric | | ≤ | 3.1 | | 1.7 | |
|----|------------------|------|---|------|---|------|-----|
| 44 | Acetoacetic | | ≤ | 10 | | 1.3 | 1.3 |
| 45 | Ethylmalonic | 0.44 | - | 2.8 | | 2.1 | |
| 46 | Methylsuccinic | 0.10 | - | 2.2 | Η | 4.1 | |
| 47 | Adipic | 0.04 | - | 3.8 | | 2.0 | |
| 48 | Suberic | 0.18 | - | 2.2 | Η | 3.2 | |
| 49 | Sebacic | | ≤ | 0.24 | | 0.21 | |

- 43 and 44 are ketones- by products of beta oxidation used in Krebs cycle for energy production
- 45-49 are dicarboxylic acids
 - formed outside of the mitochondria and decreases efficiency of energy production from mitochondria
 - can elevated with high intake of omega fatty acids-fatty fish, fish oil, avocado/olive oils.





Nutritional Markers

| Vita | min B12 | | | | | | |
|------|--|------|---|--------------|---|------|------|
| 50 | Methylmalonic * | | ≤ | 2.3 | Η | 2.8 | |
| Vita | min B6 | | | | | | |
| 51 | Pyridoxic (B6) | | ≤ | 34 | | 3.7 | 3.7 |
| Vita | min B5 | | | | | | |
| 52 | Pantothenic (B5) | | ≤ | 10 | н | 23 | |
| Vite | min D2 (Dibeflewin) | | | | | | |
| vita | min B2 (Ribonavin) | | | | | | |
| 53 | Glutaric * | 0.04 | - | 0.36 | н | 0.89 | |
| Vita | min C | | | | | | |
| 54 | Ascorbic | 10 | - | 200 | L | 0.56 | 0.56 |
| Vita | min 010 (Co010) | | | | | | Ť |
| 55 | 3-Hydroxy-3-methylalutaric # | 0 17 | _ | 30 | | 29 | |
| 55 | 5-nyuloxy-5-methyigiutane * | 0.17 | - | 55 | | 25 | |
| Glu | tathione Precursor and Chelating Agent | | | | | | |
| 56 | N-Acetylcysteine (NAC) | | ≤ | 0.28 | | 0.04 | 0.04 |
| Bio | tin (Vitamin H) | | | | | | |
| 57 | Methylcitric # | 0 10 | _ | 27 | | 11 | |
| 91 | | 0.15 | _ | 6 . 1 | | | |

* A high value for this marker may indicate a deficiency of this vitamin.



| Color Key ONRMAL | HIGH | | | | | |
|--------------------------------|-----------------------------------|-----------|--|--|--|--|
| Creatinine Value: 100.00 mg/dl | | | | | | |
| | NORMAL RANGE (ng/g creatinine) | (ng/g cre | | | | |
| Ochratoxin A (OTA) | < 7.5 | | | | | |
| Roridin E (ROE) | < 0.2 | | | | | |
| Verrucarin A (VRA) | < 1.3 | | | | | |
| Gliotoxin (GTX) | < 200 | 205.00 | | | | |
| Mycophenolic Acid (MPA) | < 37.4 | 40 | | | | |

RESULTS eatinine) | DL - Detectable Limit



So is it Food or a Water Damage Building (VVDB)?

Food

certain toxins contaminate food more often- OTA, AF, MPA, CIT, ZEA, GLIO

amount of toxin present is usually less than double the upper limit

certain toxins are rarely contaminating foods- roridin e, verucarrin a, enniatin b, chateoglobosin a

WDB

food contaminates are usually more than double the upper limit

Questions to Consider

DIET

excessive intake of coffee, teas, nut butters, homemade breads, grains, corn, cheese, wine

> **LENGTH OF TIME** how long has someone engaged in the behavior, do symptoms follow the pattern

CHANGES

if they were to stop do their symptoms subside

is the food organic or a new brand you have switch to



ORGANIC



Always consider WDB

Even if food seems to be the reason or a part of the picture, if mycotoxins are present in a severe clinical presentation, do not rule out WDB. These off occur together and progress can be limited by only changing the diet.







Home Testing Options

- Air testing- good go to as it is inexpensive, but may miss mold contamination
- Dust Collection- mold expensive initially but usually catches mold the first time
- Visual Inspection- usually best to have positive dust/air test before hiring someone to come in unless it is visible to the naked eye.





Mitochondrial Support

Mitochondrial health relies on key nutrients which support energy production, protect against oxidative stress, and facilitate enzymatic reactions. Deficiencies in these nutrients can impair mitochondrial function, leading to reduced ATP production and increased cellular damage.

B VITAMINS

Krebs cycle, glycolytic, amino acid and fatty acid oxidation rely on these

COQ10

ETC is reliant on CoQ10. Transfers electrons between complexes I-III to create proton gradient for ATP synthesis in Complex IV



L-CARNITINE

2

Transporter of fatty acids and amino acids into mitochondria for usage

protect the mitochondria from oxidative stress caused by ROS, byproducts of energy production in the electron transport chain. function and energy production.

MAGNESIUM

Involved in Krebs cycle and electron transport chain. Regulates calcium levels preventing excessive calcium accumulation that can lead to mitochondrial dysfunction.

ANTIOXIDANTS





Routes of Excretion

Conjugated toxins are excreted via kidneys into

Toxin packaged in bile is excreted via the stool

heat and sweating increase toxin release via



Binders for Stool Excreation

Enterohepatic circulation is the process by which bile acids, along with other substances like drugs and toxins, are secreted by the liver into the bile, stored in the gallbladder, and then released into the small intestine to aid in digestion. After their role in digestion, these substances are reabsorbed into the bloodstream from the intestine and returned to the liver, where they can be reused, creating a recycling loop.

Strong Binders

PRESCRIPTIONS

Wechol and cholestyramine

NON-RX

Charcoal, zeolite, betonite clay

NATURAL PRODUCTS

humic and fulvic acid, glucomannan, modified cirus pectin, peach stone, micro chitosan

DIETARY FIBER

fiber supplements, okra, turnips, green beans, carrots, and cauliflower

***non-discriminating





Weaker Binders



Urinary Excretion

Urine excretion is a primary pathway for the body to eliminate water-soluble toxins, including metabolic waste products, environmental chemicals, and certain medications. The kidneys filter toxins from the bloodstream, which are then diluted in urine and excreted, helping to maintain the body's internal balance and detoxification processes.

- GSH (Glutathione): A powerful antioxidant that helps neutralize mycotoxins and supports liver detoxification.
- NAC (N-Acetyl Cysteine): A precursor to glutathione, NAC aids in detoxifying mycotoxins by boosting antioxidant levels and protecting cells.
- Hydration: Ensures efficient toxin elimination through urine by promoting kidney function and maintaining optimal cellular detox.
- Sulforaphane: A compound found in cruciferous vegetables that enhances detoxification enzymes and protects
 against oxidative damage from mycotoxins.
- Resveratrol: A potent antioxidant that helps reduce inflammation and supports mitochondrial health during mycotoxin detox.
- ALA (Alpha-Lipoic Acid): An antioxidant that assists in detoxifying mycotoxins by regenerating other antioxidants and promoting liver function.





Lifestyle Detox Support

| BLOOD FLOW/HEAT | sauna and exer flow and releas blood stream fo |
|------------------------|---|
| DIET | Low carb diet, ketogenic diet burn and releas cells |
| DRY BRUSHING | stimulates lymp helps remove to promoting circu detoxification p |



ercise increases blood ase of toxins into the for excretion

, fasting, and t can increase fat ase of toxin from fat

phatic drainage, which toxins from the body by culation and the natural process

- Mycotoxins and Their Sources
 - Mycotoxins are toxic compounds produced by certain molds.
 - Mycotoxins contaminate food crops like grains, nuts, fruits, and sometimes occur in water-damaged buildings.
- Mitochondrial Function and Its Importance
 - Mitochondria generate energy (ATP) through cellular respiration.
 - Essential for maintaining muscle function, brain activity, immune response, and overall metabolic health.
- The Connection Between Mycotoxins and Mitochondrial Dysfunction
 - Mycotoxins disrupt mitochondrial function by increasing oxidative stress and damaging mitochondrial DNA.
 - Mycotoxins can lead to chronic fatigue, neurodegenerative issues, and metabolic disorders due to mitochondrial damage.
- Symptoms and Health Impacts of Mitochondrial Dysfunction
 - Chronic fatigue and muscle weakness
 - Hormonal imbalances, leading to thyroid dysfunction, adrenal fatigue, and reproductive issues.
 - Long-term impacts include neurodegenerative diseases, heart problems, and metabolic conditions like diabetes.
- Diagnostic Methods for Mycotoxin Exposure and Mitochondrial Dysfunction
 - Urine mycotoxin tests to detect exposure.
 - Environmental testing of air and surfaces for mold contamination.
 - Organic Acid tests for lactate and pyruvate levels, mitochondrial function, vitamin levels
- Strategies for Mitigating Mycotoxin Exposure and Supporting Mitochondrial Health
 - Avoid mold exposure by improving air quality, using dehumidifiers, and remediating mold in homes or workplaces.
 - Follow a clean diet, avoiding moldy or contaminated foods (like spoiled grains and nuts).
 - - Support mitochondrial health with nutrients like CoQIO, magnesium, B vitamins, and antioxidants to protect against oxidative stress.
 - Use detoxification strategies, such as consuming binders (e.g., activated charcoal) and increasing liver function support to eliminate mycotoxins.

Summary





<u>1. Yeasts are microscopic fungi consisting of oval cells, which reproduce asexually by budding or fission. [National Center for Biotechnology Information (NCBI)]</u> (<u>https://www.ncbi.nlm.nih.gov/books/NBK8125/#:~:text=Yeasts%20are%20microscopic%20fungi%20consisting,a%20variable%20number%20of%20nu clei).</u>

2. Ochratoxin A causes mitochondrial dysfunction by inducing oxidative stress and apoptosis in human gastric epithelium cells. [PubMed] (https://pubmed.ncbi.nlm.nih.gov/30903243/#:~:text=Ochratoxin%20A%20causes%20mitochondrial%20dysfunction,in%20human%20gastric%20epithel jum%20cells).

<u>3. Mycotoxins like aflatoxin disrupt mitochondrial functions, leading to oxidative stress and energy metabolism interference. [PMC] (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1868791/).</u>

<u>4. The role of mitochondrial dysfunction in disease conditions linked to mycotoxin exposure. [PMC] (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9277164/).</u>

5. Mechanisms of mycotoxin-induced mitochondrial damage, emphasizing oxidative stress and apoptosis. [PMC] (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC10142723/).

6. Mitochondrial impairments from environmental mycotoxins affecting human health. [PMC](https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9277164/).

7. Ochratoxin A's impact on cellular mitochondria, causing oxidative stress and mitochondrial fragmentation. [PubMed] (https://pubmed.ncbi.nlm.nih.gov/28755482/).

8. Overview of mitochondrial dysfunction due to AFB1 toxicity, highlighting oxidative stress and apoptosis pathways. [ScienceDirect] (https://www.sciencedirect.com/science/article/abs/pii/S0308814622016703).

9. AFB1 toxicity in mitochondria, leading to oxidative stress and cell death. [ScienceDirect] (https://www.sciencedirect.com/science/article/abs/pii/B9780323884624000237#:~:text=AFB1%20toxicity%20in%20the%20mitochondria%3A%20Oxida tive%20stress%20and%20apoptosis,et%20al.%2C%202021).

<u>10. The biochemical basis of mitochondrial damage caused by mycotoxins like aflatoxin and ochratoxin. [PubMed]</u> (https://pubmed.ncbi.nlm.nih.gov/2996181/).

<u>11. Mitochondrial dysfunction linked to chronic exposure to mycotoxins, causing oxidative stress and apoptosis. [ScienceDirect]</u> (https://www.sciencedirect.com/science/article/abs/pii/S0003986183711203).



THANK YOU!

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