Updates in Mold-Related Illness

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Course Details

Continuing Ed Certificate ~
Link for attestation emailed upon completion

Unlimited access for 1 year

Downloadable slides & Technical Sheets

Please support my work. Resources are for course enrollees only.

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Learning Objectives

- Brief review of the basics
- Mast cells & mold
- Sensitivities (histamine/mast cell, oxalate, sulfur, salicylate)
- Diagnostic updates
- Other damp building characters
- Precision mycotoxin treatments
- Prescriptions for mold
- Points of consideration
- A note on coherence

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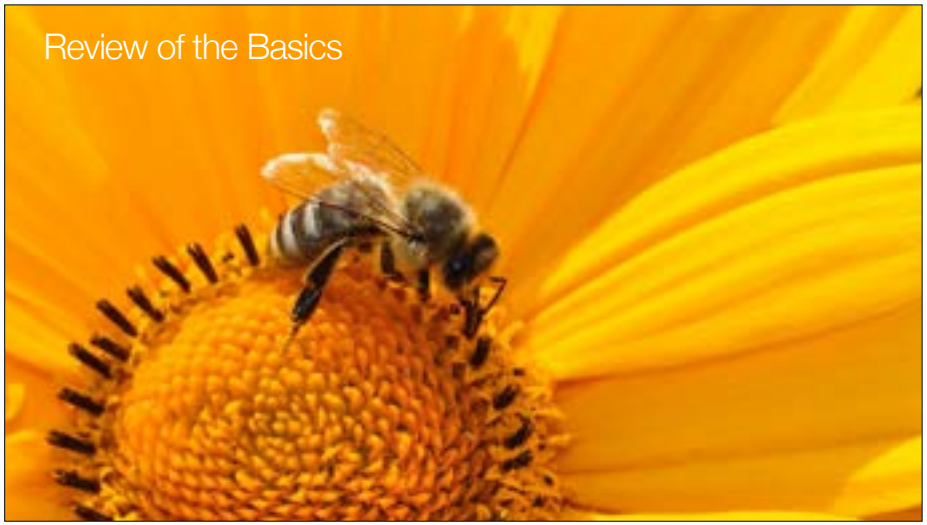
Review of the Basics



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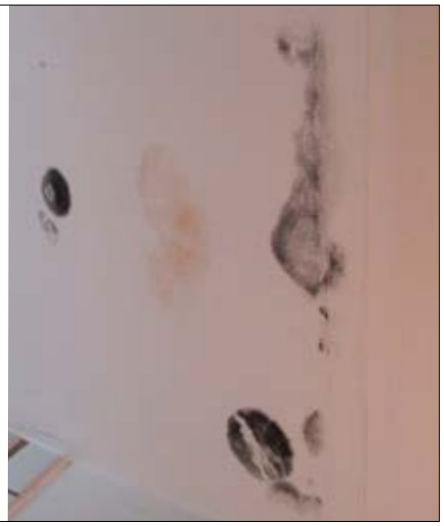
Multisystem Multisymptom

More common than not that each person in the environment has a completely different presentation.

Every living being is affected.

Depends on type of mold, presence of mycotoxins, duration and dose of exposure, and individual susceptibility.



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Damp Buildings

① Mold spores

② Mold spore fragments

③ Mold off-gassing

④ Mold mycotoxins

⑤ Bacteria

⑥ Bacterial off-gassing

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Allergic Reactions

① Mold spores

② Mold spore fragments

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Toxin Reactions

③ Mold off-gassing

④ Mold mycotoxins

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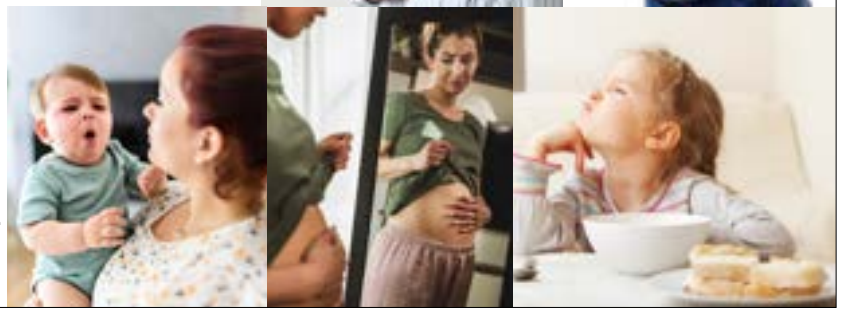
Gut & Lung

⑤ Bacteria

⑥ Bacterial off-gassing



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More Than Spore Illness

Spores

IgE | Allergic rhinitis, asthma, hypersensitivity pneumonitis (CDC)

Non-IgE | Non-IgE mediated Asthma exacerbation (CDC)

Infection | Aspergillosis (CDC)

Mast cell | Recruitment, degranulation, enhanced survival

Fragments

"Mold-othelioma"

Other Mold Dangers

Chemical off-gassing | VOCs, aldehydes, alcohols, MPA

Competition/Colonization | Mycotoxins

Biofilm

Water-damage = increased microbial diversity

Actinomycetes | gram(+) antibiotic metabolic byproducts

Endotoxins | gram(-) cell wall LPS

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Intention of the toxicant

Intention helps us determine the level of harm

Mycotoxins are formed with the intention of harming or killing another living being

Mycotoxins are made on purpose, only in certain circumstances, and even though they cost the mold a lot of energy to make

In comparison, the bacterial products found in damp buildings are byproducts of metabolism, ie: Actinomycetes metabolites have antibiotic activity

Bacterial byproducts are not selectively made in reaction to the surroundings

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Mycotoxins & Mold Sources

Aflatoxin
- Aspergillus flavus, A. parasiticus

Chaetoglobosin A,C
- Chaetomium globosum

Citrinin
- Aspergillus, Penicillium, Monascus

Enniatin B_1
- Fusarium spp

Fumonisin
- Fusarium spp

Gliotoxin
- Aspergillus fumigatus, Candida spp

Ochratoxin A
- A. ochraseus, A. niger, Penicillium verrucosum, P. nordicum, P. chrysogenum

Patulin
- Aspergillus spp, Penicillium spp, Mucor, Fusarium spp

Sterigmatocystin
- Precursor of Aflatoxin, A. versicolor

Trichothecenes (Roridin, Verrucarin, Nivalenol, Deoxynivalenol, Diacetoxyscirpenol, Satratoxin)
- Stachybotrys chartarum, Trichoderma viridae, Fusarium spp, Myrothecium

Zearalenone
- Fusarium spp

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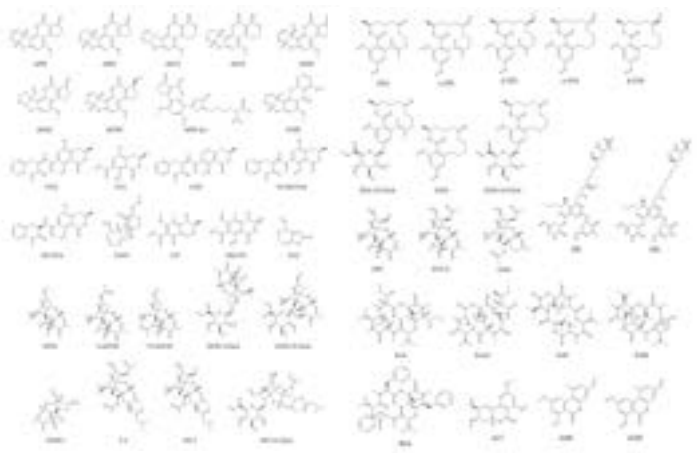

PMID 32121036

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Mycotoxins

Lipid soluble
Mitochondrial toxic
Immunotoxic
Neurotoxic
Alimentary toxic
Dermatoxic
Nephrotoxic
Nephrocarcinogenic
Hepatotoxic
Hepatocarcinogenic
Genotoxic
Teratogenic
Carcinogenic

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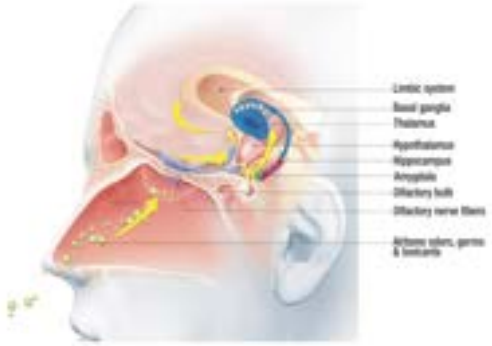
Mycotoxins on the Mind



Neuroinflammation
Microglial dysfunction
Cognitive difficulties
Neurotransmitter imbalance
Inhalational Alzheimer's
Limbic and vagal dysfunction

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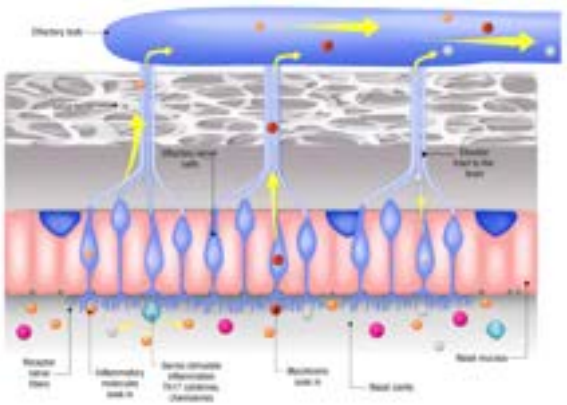
Olfactory bulb “elevator to the brain”



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Lung microbiome and the brain

Damp and WDBs modify the lung microbiome.

There’s a tight interconnection between the lung microbiota and immune reactivity in the brain.

A dysregulation in the lung microbiome significantly influenced the susceptibility of rats to developing autoimmune disease of the CNS.
*The connection between mold and PANS.

Shifting the lung microbiota towards LPS-enriched phyla induces a type-I-interferon-primed state in brain-resident microglial cells.

PMID: 35197636, 35417673, 35197592, 32140452, 19793773

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Analysis for Fusarium toxins in various samples implicated in biological warfare in Southeast Asia

Abstract

Mycotoxins have a long history of use as a biological warfare weapon.
“Yellow rain” mixed mycotoxins used against the Hmong people in Southeast Asia.
So, yes, they affect everyone.

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Sensitivities

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Figure 1

Highly simplified overview of the diverse stimuli and potential consequences of mast cell activation and secretion of cytokines, chemokines and growth factors

PMID: 29431212

Spores, Fragments & Mast Cells

"For their strategic location at vascularized mucosal surfaces, combined with a unique versatility, mast cells are well positioned to respond to fungi and/or fungal allergens."

Mast cells influence innate immune responses against fungal infections via multiple mechanisms, not all beneficial, reaction varies by genetics.

Actively living mold ~
Influence mast cell homeostasis to enhance survival
IgE allergic response influences the number and function of mature mast cells
Induce pulmonary mast cell degranulation even in the absence of antigen-specific IgE

Killed hyphae (#fragments) induced significant degranulation as compared to live (DIY remediation/remodel of dried water event)

Increased allergic responses to other respirable and ingested antigens (foods, dust, grass, pollen, pet dander, exhaust)

Empirically, increased recruitment at stage of fungal evasion → invasion

PMID: 19527167, 19201896, 29431211

Published in final edited form as:
J Immunol. 2008 February 15; 180(4): 2434–2438.

IgE INFLUENCES THE NUMBER AND FUNCTION OF MATURE MAST CELLS BUT NOT PROGENITOR RECRUITMENT IN ALLERGIC PULMONARY INFLAMMATION[1]

Clinton B. Mathias[1,2], Eva-Jasmin Freyschmidt[1,2], Benjamin Caplan[1], Tatiana Jones[1], Dimitri Poddighe[1,2], Wei Xing[1], Krista L. Harrison[1], Michael F. Gurish[1,2], and Hans C. Oettgen[1,2]
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[3]Harvard Medical School, Boston, MA 02115

Inhalation of A. fumigatus extract in mice induced a dramatic rise in IgE *accompanied by an increase in airway mast cells*, and signs indicating an elevated systemic mast cell load.

Analyses of potential cellular targets of IgE revealed that *IgE antibodies are not required for the induction of mast cell progenitors in response to allergen* but rather act by sustaining the survival of mature mast cells.

Mast Cells & Mold

Mast cells are differently differentiated by tissue and reason for recruitment.

Dr. Theoharides - "the gateway to inflammation in the body"

MUCH more than, and not always, histamine ~ may release 1000+ cytokines and other inflammatory mediators without ever releasing histamine.

Low histamine diet may help, but may be mediators other than histamine at play.

Symptoms related to eating ~
Post-prandial flushing
Post-prandial fatigue
Post-prandial brain fog
Post-prandial drop in bp
Gastroparesis
GI: heartburn, N/V, constipation, diarrhea
Food avoidances related to histamine concentration, esp left-overs

PMID: 19527167, 19201896, 29431211

TABLE 1. Histamine Receptors

	H_1 Receptor	H_2 Receptor	H_3 Receptor	H_4 Receptor
Receptor expression	Nerve cells, airway and vascular smooth muscle, endothelial cells, epithelial cells, neutrophils, eosinophils, monocytes/ macrophages, DC, T and B cells, hepatocytes, chondrocytes	Nerve cells, airway and vascular smooth muscle, endothelial cells, epithelial cells, neutrophils, eosinophils, monocytes, DC, T and B cells, hepatocytes, chondrocytes	High expression in histaminergic neurons, eosinophils, DC, monocytes; low expression in peripheral tissues	High expression on bone marrow and peripheral hematopoietic cells, eosinophils, neutrophils, DC, T cells, basophils, mast cells
Histamine function, general	↑ Pruritus, pain, vasodilation, vascular permeability, hypotension; flushing, headache, tachycardia, bronchoconstriction, stimulation of airway vagal afferent nerves and cough receptors; ↓ atrioventricular node conduction time	↑ Gastric acid secretion, vascular permeability, hypotension, flushing, headache, tachycardia, chronotropic and inotropic activity, bronchodilation, mucus production (airway)	↑ Pruritus (no mast cell involvement), ↑ nasal congestion; prevent excessive bronchoconstriction	↑ Pruritus (no mast cell involvement), ↑ nasal congestion; differentiation of myeloblasts and promyelocytes
Histamine function in allergic inflammation and immune modulation	↑ Release of histamine and other mediators; ↑ cellular adhesion molecule expression and chemotaxis of eosinophils and neutrophils; ↑ antigen-presenting cell capacity, costimulatory activity on B cells; ↑ cellular immunity (Th1), ↑ autoimmunity; ↓ humoral immunity and IgE production	↓ Eosinophil and neutrophil chemotaxis; ↓ IL-12 by dendritic cells; ↑ IL-10 and development of Th2 or tolerance-inducing dendritic cells; ↑ humoral immunity; ↓ cellular immunity; suppresses Th2 cells and cytokines; indirect role in allergy, autoimmunity, malignancy, graft rejection	Probably involved in control of neurogenic inflammation through local neuron-mast cell feedback loops; ↑ proinflammatory activity and APC capacity	↑ Calcium flux in human eosinophils; ↑ eosinophil chemotaxis; ↑ IL-16 production (H_2 receptor also involved)
Histamine function in the CNS	Sleep/wakefulness, food intake, thermal regulation, emotions/ aggressive behavior, locomotion, memory, learning	Neuroendocrine	Presynaptic heteroreceptor; ↓ histamine, dopamine, serotonin, noradrenaline, and acetylcholine release	To be defined

APC indicates antigen-presenting cells; DC, dendritic cells; IgE, immunoglobulin E; IL, interleukin.
Adapted from Simons and Akdis.[3]

World Allergy Journal, Sept 2008

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Weakened Immunity

- More frequent infections
- Viral infections linger
- Viral infections become bacterial
- Delayed wound healing

Teens which were exposed to mold in their younger years have immune systems that are primed to preferentially choose mast cells.

PMID: 19527167, 19201896, 29431211

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Nettles *(Urtica dioica)*

Rich in quercetin, rutin, and ellagic acid. One of MANY antihistaminic herbs.

Statistically significant reduction in nasal eosinophils independent of IgE involvement.

Ameliorates allergy symptoms ~
- Antihistaminic; antagonist and negative agonist activity against H1 receptor
- Mast cell stabilizing; inhibition of mast cell tryptase preventing degranulation and release of a host of pro-inflammatory mediators
- Inhibits prostaglandins

Lowers skin irritability ~
- Decreases histamine and H4 agonist-induced IL-8 expression in keratinocytes.

Gastroprotective ~
- Protects stomach mucous membrane while hindering excess acid secretion.
- Protects against tissue damage caused by inflammatory processes in inflammatory bowel diseases
- Preserves epithelial integrity and enhances intestinal defense

Neuroprotective ~
- Improves memory function and cognition
- Reduces chronic stress-related dysfunctions of the CNS in animal models
- Positive effects on microvasculature

PMID: 37171512, 35399803, 29844782, 19140159, 31163183

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Nettles dosing

Dose is everything!

Eat it! (cooked) Used as a staple green in many indigenous diets. Very safe to use as food and in higher doses than many herbs. Often dosed too low!

Nettle lemonade (How-to video)

Daily:
- 600 mg twice daily

Allergic flare:
- 1000 mg up to four times daily

Caution:
- Fresh plant will sting; cook or dry, or handle with gloves
- Source of oxalates

PMID: 29844782

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Synergy

A note on dose (including Tech Sheets) ~

Many of the doses listed are intended for when each item is used as a standalone therapy.

When multiple items are combined, they work synergistically, which means that lower doses can typically achieve similar effectiveness due to their complementary effects.

Most don't have to be taken at the same time to have synergy.

Hops *(Humulus lupulus)*

Inhibits type-I allergic reactions, allergic rhinitis, pruritus.

Significantly inhibits histamine release from mast cells and basophils.

Significantly inhibits degranulation via inhibition of protein kinase C, which plays a pivotal role in the degranulation of chemical mediators.

Little effect on IgE, so exerts effects via prevention of degranulation.

Daily:
300 mg daily

Allergic flare:
600 mg up to three times daily

Caution:
Estrogenic, has some dopaminergic effects.

PMID: 17151464, 16595900, 20619323, 17587695

Aloe

Not just a demulcent immune modulator, also a "highly potent" mast cell stabilizer ~
"In contrast to six clinical drugs with mast cell stabilizing properties (amlexanox, tranilast, ketotifen, cromolyn disodium salt, dexamethasone and pimecrolimus), aloe-emodin showed an impressive and potent inhibitory action on the mast cell degranulation."

Aloe emodin suppresses IgE-mediated anaphylactic reaction and mast cell activation.

Antipruritic. Inhibit inflammatory-response-induced mast cell degranulation in skin lesions and suppress the expression of inflammatory cytokines, such as IL-4, IL-6.

Inhibits mediator release in lung tissue, and subsequent inhibition of histamine and leukotrienes.

Enhances nutrient absorption when co-administered *same time*.

Aloe glucomannans exhibit gastrointestinal cytoprotective action: regulate intestinal homeostasis, alleviate inflammation, relieve intestinal injury via intestinal stem-cell mediated epithelial regeneration, maintain intestinal barrier integrity.

Preserving the long-chain polysaccharides takes special non-heat processing.

Dose: 500mg concentrated 200:1 inner leaf extract, three times daily with food.

PMID: 34432461, 33607072, 21907188, 39181335, 10604937, 14510234

Antihistamine Rx considerations

A stop-gap, not the solution.

They have consequences (even SGAH) ~
Dementia, reduced bone mineral density, gastroparesis, peptic ulcer dz
H2: hypomagnesemia. Mg needed to make DAO which breaks down histamine (vicious cycle). Also reduced B12 and Ca.

Compounded often more tolerated b/c fewer excipients.

Combine Rx's with different MOAs has a synergistic effect. Also combine with herbs/ nutrients.

If responsive to Cromolyn, rule out concomitant parasitic infection, biofilm agent infection (ie: pseudomonas), food allergy

If mold was the cause, trial antifungals sooner rather than later.

PMID: 38935035

Oxalates

Fungal overgrowth.

Very few due to genetics in mold-affected individuals.

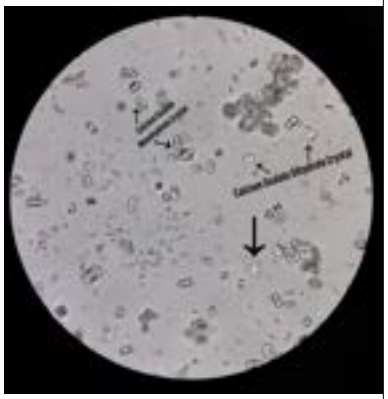
Correlated with vitamin K deficiency.

Assist with breakdown: B2, B6, magnesium, manganese, biotin.

Supplement vitamin C through whole plant/herb sources rather than ascorbic acid (rose hips, acerola, camu camu, amla).

Avoid supplemental calcium, including calcium-D-glucarate (K-H-glucarate alternative)

Antifungals as soon as possible.



Sulfur

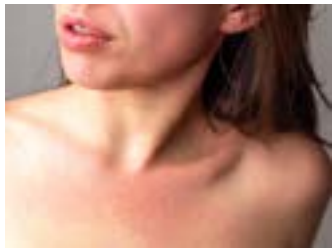
Very similar symptom picture as histamine sensitivity.

Correlated with fungal overgrowth, molybdenum deficiency.

Aspergillus spp will use sulfur to make Gliotoxin in vivo.

Temporary low sulfur diet while treating fungal overgrowth.

Optimize Mb: Molybdenum glycinate 500mcg qd
Mb-dependent enzyme sulfite oxidase (SOX) catalyzes the conversion of sulfite to sulfate, crucial for the degradation of sulfur-containing amino acids.



PMID: 39062583

Salicylate

Patulin mycotoxin (empirical) Ex. *Penicillium urticae* converts 6-methylsalicylic acid into patulin. *cert course

Sn/Sxs: pruritus, urticaria, rashes, eczema, abdominal pain, nausea, diarrhea, headache, fatigue, itchy/watery or swollen eyes, coryza, sinusitis.

Correlated with nasal polyps, atopy, asthma, anaphylaxis.

Causally correlated with low omegas, low sex hormones.

Omega-3 study: 3 patients with disabling salicylate-induced intolerance with 10 g daily of fish oils rich in omega-3 PUFAs for 6-8 weeks. All 3 had complete or virtually complete resolution of sxs allowing discontinuation of systemic corticosteroid therapy.
*Symptoms relapsed after dose reduction.

Hormone study: female patients showed an increased risk for developing acetylsalicylic acid intolerance ($p = 0.01$). Can we correlate to low T in men?

Low salicylate diet for 4-6 weeks while treat mold and optimize sex hormones.
Omega 3s: 10gm divided daily for 6-8 weeks, reduce by 1gm weekly to maintenance dose, which may be higher than typical.

PMID: 16247191, 18795922, 33332460

CATEGORY 1:

GENERAL:
- ☐ Fatigue that doesn't otherwise make sense
- ☐ Trouble sleeping
- ☐ Worse after eating
- ☐ Worse after exercise
- ☐ Increased thirst
- ☐ Stubborn weight gain
- ☐ Anemia

SENSITIVITY:
- ☐ Bothered by tags and seams on clothing
- ☐ Chemical sensitivities
- ☐ Sensitive to light, sound, or touch

HEAD/MIND:
- Slowed thinking or 'brain fog'
- Unwanted feeling, unwanted mood, overwhelm
- Headaches
- Dizziness, vertigo, or drunken feeling
- Unexplained mood changes, anxiety, or depression

EENT:
- ☐ Allergies/hay fever year-round
- ☐ Eye irritation
- ☐ Dark circles under eyes
- ☐ Floaters in your vista
- ☐ Vision blurry frequently changes, or difficulty reading
- ☐ Sneezing or persistent runny nose
- ☐ Acute sense of smell for mold
- ☐ Recent sinusitis
- ☐ Ears feel plugged or clogged
- ☐ Itchy or sore ear canals
- ☐ Sores in the mouth
- ☐ Post-nasal drip or frequent throat clearing
- ☐ Chronically sore throat
- ☐ Coated tongue

RESPIRATORY:
- ☐ Easily irritated lungs
- ☐ Episodic cough
- ☐ Shortness of breath, air hunger, or yawn/sigh often

CARDIOVASCULAR:
- ☐ Easy bruising
- ☐ Heart palpitations
- ☐ Lower extremity edema
- ☐ Protruding veins on limbs

DIGESTIVE:
- ☐ Nausea
- ☐ Bloated abdomen or flatulence
- ☐ Unexplained change in digestion/bowels
- ☐ Recent change in appetite
- ☐ Crave carbs, sweets, or alcohol

GENITOURINARY:
- ☐ Overactive bladder
- ☐ Bladder infections

SKIN:
- ☐ Skin rash, redness or flushing

IMMUNE:
- ☐ Frequent infections or delayed recovery from colds

MUSCULOSKELETAL:
- ☐ Increased body pain

Total CATEGORY 1 boxes checked: _____

0 - 4 symptoms = Score 0
5 - 7 symptoms = Score 1
8 - 10 symptoms = Score 2
11+ symptoms = Score 3

ENTER CATEGORY 1 SCORE: _____

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CATEGORY 2:

GENERAL:
- ☐ Voice sounds nasally
- ☐ Frequent or strong static shocks
- ☐ Histamine intolerance
- ☐ Non-obstructive sleep apnea
- ☐ React poorly to musty spaces

SENSITIVITY:
- ☐ Sensitivity to EMFs

HEAD/MIND:
- ☐ Migraines
- ☐ Difficulty thinking clearly or memory loss
- ☐ Confusion or disorientation

EENT:
- ☐ Allergies are not well-controlled by medication
- ☐ Chronic sinusitis
- ☐ Nose bleeds
- ☐ Ear ringing or ear pain that's new or worsening

RESPIRATORY:
- ☐ Asthma or wheezing
- ☐ Chronic cough
- ☐ Burning lungs

CARDIOVASCULAR:
- ☐ Episodes of fast heart beat
- ☐ Chest pain
- ☐ Low platelets

DIGESTIVE:
- ☐ Increased food sensitivities
- ☐ Frequent vomiting
- ☐ Irritable bowel or alternating constipation/diarrhea
- ☐ Digestive ulcer or blood in the stool
- ☐ Celiac or non-celiac intestinal disease
- ☐ Fatty liver
- ☐ Liver pain or swelling

GENITOURINARY:
- ☐ Unexplained menstrual changes
- ☐ Bacterial vaginosis
- ☐ Kidney pain or swelling

SKIN:
- ☐ Itchy or burning skin
- ☐ Peeling or sloughing skin
- ☐ Raynaud's syndrome
- ☐ Eczema or psoriasis

IMMUNE:
- ☐ Epstein-Barr virus activation

MUSCULOSKELETAL:
- ☐ Slow reflexes
- ☐ Balance issues or incoordination
- ☐ Joints easily injured
- ☐ New or worsening nerve pain, numbness or tingling
- ☐ Muscle weakness or spasm

Total CATEGORY 2 boxes checked: _____

0 - 2 symptoms = Score 0
3 - 5 symptoms = Score 1
6 - 8 symptoms = Score 2
9+ symptoms = Score 3

ENTER CATEGORY 2 SCORE: _____

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CATEGORY 3:

GENERAL:
- ☐ Current exposure to mold
- ☐ Previous exposure to damp, musty or water-damaged building any time in your life
- ☐ Mold allergy
- ☐ Abnormal reaction to medications or supplements
- ☐ Autism or sensory processing disorder
- ☐ Chronic fatigue syndrome
- ☐ Chronic inflammatory response syndrome (CIRS) or positive Shoemaker tests

SENSITIVITY:
- ☐ Feeling of an internal vibration

HEAD/MIND:
- ☐ Dysautonomia or Postural Tachycardia Syndrome (PoTS)
- ☐ Dementia

EENT:
- ☐ Daily use of sinus spray, sinus prescription, or Nasil gel
- ☐ Nasal polyps
- ☐ Sinus surgery at any time in your life
- ☐ Hearing loss
- ☐ MARCoNS
- ☐ Oral thrush

RESPIRATORY:
- ☐ Asthma that's difficult to control with medication
- ☐ Lung scarring or nodules
- ☐ Pulmonary Edema
- ☐ Idiopathic Pulmonary Fibrosis
- ☐ Respiratory distress or idiopathic pneumonitis
- ☐ Lung cancer

CARDIOVASCULAR:
- ☐ Arrhythmia
- ☐ Coagulation abnormalities
- ☐ Arteriovenous abnormality
- ☐ Chung Strauss Syndrome

DIGESTIVE:
- ☐ Peanut allergy
- ☐ Cyclical vomiting syndrome
- ☐ Eosinophilic esophagitis
- ☐ Non-alcoholic steatohepatitis (NASH)
- ☐ Hepatocellular carcinoma or other liver cancer

GENITOURINARY:
- ☐ Infertility
- ☐ Chronic pelvic pain
- ☐ Interstitial cystitis
- ☐ History of kidney stones
- ☐ Reduced GFR (glomerular filtration rate)
- ☐ IgA nephropathy, nephrotic syndrome, nephritis, or other kidney disease
- ☐ Kidney cancer

SKIN:
- ☐ Recurrent yeast infections or fungal skin infections, including athlete's foot, jock itch or yeast vaginitis
- ☐ Erythema nodosum
- ☐ Toenail fungus

IMMUNE:
- ☐ Autoimmunity
- ☐ Mast cell activation syndrome (MCAS)
- ☐ Aspergillosis, current or history of
- ☐ Previous or current cancer diagnosis not otherwise specified
- ☐ Aplastic anemia
- ☐ Sarcoidosis

MUSCULOSKELETAL:
- ☐ Hypermobility or Ehlers-Danlos syndrome
- ☐ Tremors or tics
- ☐ Difficulty walking

Total CATEGORY 3 boxes checked: _____

Score 1 for each box checked.
Total items checked and the Category Score will be the same for this category.

ENTER CATEGORY 3 SCORE: _____

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TOTAL MOLD RISK RESULTS

Gather your Category Scores from the 3 previous categories.

CATEGORY 1 SCORE: _____
CATEGORY 2 SCORE: _____
CATEGORY 3 SCORE: _____

Add Category Scores together to calculate your total mold risk.

TOTAL MOLD RISK _____

0 - 4 = Not Likely Mold-Related Illness
5 - 9 = Possible Mold-Related Illness
10+ = Probable Mold- or Biotoxin-Related Illness

OTHER THINGS TO CONSIDER:
Lyme Disease, MSIDS, Tick-Borne Co-Infections (Use HOROWITZ MSIDS-LYME QUESTIONNAIRE)
Other environmental toxins (IE: glyphosate, mercury load, PMS, VOCs, etc.)
Intestinal parasites
Chronic viral syndromes or other stealth infections
Food sensitivities
CVIDS or immunodeficiency syndromes

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Research Study

MoldIQ.org

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Diagnostic Updates

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The Paths of the Mycotoxin

In damp or water-damaged building exposure ~

- Inspiration
- Absorption through respiratory capillaries to the blood stream
- Carried via blood to Liver & Kidney
- Kidney - filtration to urine
- Liver - modifies to either water-soluble for excretion in urine OR modifies into bile micelle and delivers to gut as a bile conjugate

Excess absorbed into lipid-rich tissue (bioaccumulation)

Ingested mycotoxins ~
May remain unconjugated inside the intestines.

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Lab Diagnostics

All labs have strengths & weaknesses

All can be useful when appropriately applied. Both serum and urine used in research.

The choice of labs depends on what question you're trying to answer.

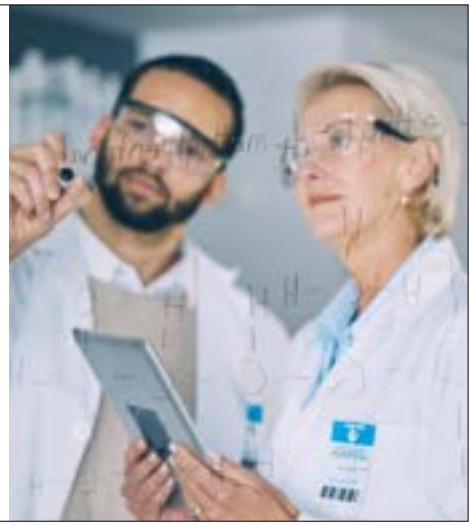
A cluster of labs raises our confidence in the result/answer.

Most common ~

Am I being exposed to mold right now?

Why have my symptoms changed?

How much longer do I have to go?



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Urine ELISA Mycotoxin Testing

Established use for ~20 years

Indirect measure

The idea - due to the body's ability to modify mycotoxins, antigen detection vs molecular matching will catch more metabolites and give a better view of body burden

Strengths ~
Detect both the mycotoxin in pure form and metabolites due to common antigens on most modified forms
Levels correlate to symptoms in majority of my patients ("bell-curve")

Challenges ~
Not controlled for creatinine
Highly dependent on the antigen selection by the lab
Non-specific reactions (aka background noise) w poss false-positives
Varying accuracy for pts with issues detoxing and excreting
Doesn't help answer the question of whether currently being exposed
Unknown degree of contamination via ingestion

6 hour urine collection directly following provocation, if used. GSH okay with this method, or pretest GSH status. Off binders.

Urine LC-MS Mycotoxin Testing

Gold standard for small molecules the size of mycotoxins

Direct measure

The idea - molecular identification as direct detection of the presence in the urine

Strengths ~
Controlled for creatinine
Specific metabolites of mycotoxins can be tested and reported as an individual finding, then grouped for a bigger picture
Levels correlate to symptoms in majority of my patients ("bell-curve")

Challenges ~
Some of the molecules are similar in structure, peak together, leading to possible cross-reporting
May miss metabolites if not specifically identified as a structure to monitor, and if partially metabolized by the body
Extraction method to prep sample varies, and is critical for QC
Varying accuracy for pts w issues detoxing and excreting
Doesn't help answer the question of whether currently being exposed
Unknown degree of contamination via ingestion

6 hour urine collection directly following provocation, if used. GSH provocation leads to false negatives. Off binders.

Serum Mycotoxin Antibody Testing

New kid on the block

Indirect measure

The idea - the mere presence of a mycotoxin not as important as knowing what the body thinks about it

Strengths ~
Not as affected by diet as urine, though can be
IgE helps to answer the question of whether currently being exposed (past 2-4 weeks to an appreciable degree)
Choice for pts with issues detoxing and excreting, because not an excretion test
Levels correlate to symptoms in majority of my patients ("bell-curve")

Challenges ~
IgG may stay elevated longer than urine reduction
Toxicant antibody patterns are different than infective (viral) patterns
Antigen selection by lab, may miss metabolites if antigens have been modified
May be elevated due to colonization, but typically due to exposure
Colonization becoming invasive may elevate IgE
May be false negatives in patients with immune compromise

Pretest total IgG and IgE for sufficiency. Different than mold spore allergy test.

International Journal of Environmental Research and Public Health

MDPI

Review

Occurrence, Toxicity, and Analysis of Major Mycotoxins in Food

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"Members of three fungal genera, Aspergillus, Fusarium, and Penicillium, are the major mycotoxin producers [in food].

While over 300 mycotoxins have been identified, six (aflatoxins, trichothecenes, zearalenone, fumonisins, ochratoxins, and patulin) are regularly found in food, posing unpredictable and ongoing food safety problems worldwide.

In addition to concerns over adverse effects from direct consumption of mycotoxin-contaminated foods and feeds, there is also public health concern over the potential ingestion of animal-derived food products, such as meat, milk, or eggs, containing residues or metabolites of mycotoxins."

Table 1. Major mycotoxins and US and EU limits on food and animal feed levels.

Mycotoxin	Fungal Species	Food Commodity	US FDA (ug/kg)	EU (EC 2006) (ug/kg)
Aflatoxins B1, B2, G1, G2	*Aspergillus flavus* *Aspergillus parasiticus*	Maize, wheat, rice, peanut, sorghum, pistachio, almond, ground nuts, tree nuts, figs, cottonseed, spices	20 for total	2–12 for B1 4–15 for total
Aflatoxin M1	Metabolite of aflatoxin B1	Milk, milk Products	0.5	0.05 in milk 0.025 in infant formulae and infant milk
Ochratoxin A	*Aspergillus ochraceus* *Penicillium verrucosum* *Aspergillus carbonarius*	Cereals, dried vine fruit, wine, grapes, coffee, cocoa, cheese	Not set	3–10
Fumonisins B1, B2, B3	*Fusarium verticillioides* *Fusarium proliferatum*	Maize, maize, products, sorghum, asparagus	2000–4000	200–3000
Zearalenone	*Fusarium graminearum* *Fusarium culmorum*	Cereals, cereal products, maize, wheat, barley	Not set	20–100
Deoxynivalenol	*Fusarium graminearum* *Fusarium culmorum*	Cereals, cereal products	1000	200–50
Patulin	*Penicillium expansum*	Apples, apple juice, and concentrate	50	10–50

Effects of Cooking

Netherlands study 2016

Pasta infected with ~
Enniatin
Deoxynivalenol (DON)
(tricothecene aka vomitoxin)

Cooked in duplicate on different days, under standardized conditions, simulating household preparation

Tested post-cooking

83-100% enniatin retained

60% tricothecenes retained

PMID: 27451245

Urine Test Prep

Diet can influence results

Low mycotoxin diet x 3 days
(48-hr washout for most mycotoxins)

D/c binders

D/c GSH with LC/MS method

Continue antifungals

Different mycotoxins on different tests

Different methods

Split sample testing to different labs; to be expected

QC should always be emphasized; concern if split sample to same lab, same collection

Margin of error leans toward false negatives

Any positive is significant

Test Cluster: Am I being exposed right now?

Visual: visual contrast sensitivity ~
High possibility this will be abnormal during active exposure.
VCStest.com

Blood: mycotoxin antibody IgE ~
High possibility this will be elevated during active exposure.

Urine: mycophenolic acid ~
This mold metabolite is excreted from living mold. Possible elevation during active exposure.

Blood: mold spore allergy/antibody IgE/IgG ~
Possible elevation during active exposure to spores.

Test Cluster: Why have my symptoms changed?

Visual: visual contrast sensitivity ~
Rule out new exposure. High possibility this will be abnormal during active exposure.
VCStest.com

Urine: mycotoxins ~
Common for underlying/buried mycotoxins to begin to excrete as you heal.
The first 3 months of treatment often increases urinary mycotoxin levels.
Original mycotoxins may have cleared but different ones come to the surface.
Follow low mycotoxin diet x 3 days prior to urine collection.

Urine: organic acids test ~
May identify colonization (note: colonized people can have a normal OAT.)
If colonized, the fungi will fight back and shift symptoms.

Test Cluster: How much longer do I have to treat?

Urine: mycotoxins ~
Possible elevation with continued body burden.
Sauna provoke followed by 6 hour urine collection.
Follow low mycotoxin diet x 3 days prior to urine collection.

Blood: mycotoxin antibody IgG ~
Possible elevation with continued body burden.

Blood: mold allergy IgG ~
Possible elevation if colonized with spores.

On the horizon

The use of capillary blood from finger-pricks versus venous blood was evaluated.
The analyte levels correlate indicating that the less invasive finger-prick sampling gives also reliable results.
No significant hematocrit effect was observed.

Journal of Fungi

MDPI

Article

In Search of Clinical Markers: Indicators of Exposure in Dampness and Mold Hypersensitivity Syndrome (DMHS)

Kirsi Vaali [1,*], Kingsley Mokolo Ekami [1], Maria A. Andersson [1,2], Marika Mannerström [3] and Taula Heinonen [4]

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Indoor air condensed water and human macrophage THP-1 test were applied to evaluate the buildings.

Basophil activation tests (BAT) - the only non-IgE-mediated test method that can be used without the requirement of exposing the patient to an allergen.

Basophils from exposed subjects died/lost activity at 225 times lower concentrations of toxic extracts from the target building than recommended in the common BAT protocol.

PMID: 36983500

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Other Damp Building Characters (and what to do about them)

58

We can't purposefully poison people in order to determine treatments

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Don't Avoid Avoidance

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Peel The Orange

1 AVOIDANCE

2 FUNDAMENTALS

3 PROTECT

4 REPAIR

5 FIGHT

Don't skip the basics!

Can you guess the 5 basic things that have reduced side-effects to allow for more "aggressive" treatments to be tolerated?

Vitamin D (+K)

DHA

Phospholipids

Vitamin E

B-vitamins (cofactors and coenzymes for detox)

Bioflavonoids as Antimycotoxin Agents

FIGURE 2 Chemical structures of plant phenolic compounds reported in literature between 2010 and 2021 as having potential antimycotoxin activity (data listed in Table 1). Glu, glucose; Neo, neohesperidoside; Rut, rutinose PMID: 35092346

Bioflavonoids Before Binders

Promotes detox via urine over stool

Protects the organs of detoxification -(don't forget the kidneys!)

Downregulating effect on the expression of mycotoxin-forming genes.

IE: Ochratoxin ~ the "persister mycotoxin"
high affinity binding to albumin
∴ kidneys (first line of detox) can't clear it

Leads to tissue accumulation in kidney and gut interstitium

Bioflavonoids (esp Astaxanthin for OTA) denatures it from albumin to allow for clearance, sparing the kidneys from damage.

Different color band for different mycotoxins.

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MPA (Mycophenolic acid)

Not a mycotoxin! It's a mold metabolite. Why important? MPA+ if active mold.

Where there is living mold, there is MPA, but not necessarily mycotoxins.

Potent immunosuppresant (CellCept).

Significant intestinal lining damage.

Quickly absorbed & excreted in bile within the first few hours of exposure ∴ beneficial to give small, frequent doses of treatments.

Phenolic substances detoxed/excreted in Phase II as glucuronides.

Tx ~
Get them out of mold!
Focus on glucuronidation (green tea, potassium-H-glucarate, I3C, DIM)
Gut protection/immunomodulation (aloe polysaccharides)
Give small frequent doses

PMID: 21049395

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Bacteria

IME occur moreso in grey-water spills

Doesn't change building remediation, but may require specific treatments

Both Gram positive and negative are found in WDBs

Think about your patient with persistent microbiome imbalance despite adequate treatment

Gram negative (ie: E. Coli) ~
Bacterial cell wall fragments
Endotoxins - lipopolysaccharides (LPS)
Not selectively made in response to environment, just dead bacteria "body parts"
Damaging to lungs and gut, increased risk of SIBO
Inhibit bile formation

https://academic.oup.com/femsre/article/41/3/392/3830259
https://microbiologysociety.org/publication/past-issues/natural-products-and-drug-discovery/article/actinomycetes-as-natures-pharmacists.html

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Endotoxins

Intranasal for LPS brain impacts ~
Ginsenosides (Synapsin)

Endotoxin binders ~
Sarsaparilla (Smilax glabra) - glycerite 1/2 tsp tid
Lactoferrin - 100mg bid

Block absorption ~
Bioflavonoids like quercetin were shown in a psoriasis study to block the absorption of endotoxins, thereby reducing symptoms.

Digest them ~
In the same study, taking Ox Bile as a supplement broke up Endotoxin inside the intestinal lumen.

(and of course treat SIBO if +)

https://www.sciencedirect.com/topics/medicine-and-dentistry/endotoxin
PMID: 29908580, 10024582

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Actinomycetes phylum

Kingdom: Bacteria
Phyla: Actinobacteria
Class: Actinobacteria
Order: Bifidobacteriales
Family: Bifidobateriaceae
Genus: Bifidobaterium
Species: B. Longum (32)

The most prevalent soil bacteria; benefic role in soil fertility.

1 of 4 major phyla of the gut microbiota ~
Make up a small % in total, but play a pivotal role in microbial balance
Ie: Bifidobacteria family is in the class Actinobacteria.

Genus Streptomyces is the source of most of antimicrobial and antiprotozoal drugs, including
Tetracyclines, Macrolides, Aminoglycosides, Rifamycins, Ivermectin

Similar to mold, may be found in all buildings to some extent due to cross-contamination from outdoors. Found in WDBs in higher number than "normal", and some of the more pathogenic species may thrive in that environment.

Secrete antibiotic metabolic byproducts (Gram positive).

Not selectively made in response to environment, just an off-gassed metabolite

Metabolites are associated with granulomatous disease-like symptoms ~
Shortness of breath on exertion
Flu-like achiness
Fever and chills

Increased risk of yeast overgrowth (think Abx)

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Actinos treatment

Intranasal probiotics ~
Lactobacillus sakei, casei

Intranasal terrain building ~
Humic acid

Aloe juice sipped throughout the day (GI immunomodulation)

Spore-based probiotics ~
Rebalance the flora rather than replace it, therefore need less as you use them.
Start extremely low in the dose, can cause significant die-off.
For sensitive patients, few flecks of powder from an opened capsule daily, using 1 capsule over 2 weeks, then 1 capsule over 1 week, then 1 capsule over 3 days, etc.

Depending on severity of exposure, may require additional probiotics and/or post-biotics.

BPC-157. 500mcg daily. Formed in the stomach to maintain the gut barrier.

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Treatment Updates

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MYCOTOXIN DETOXIFICATION

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Peel The Orange

1 AVOIDANCE

2 FUNDAMENTALS

3 PROTECT

4 REPAIR

5 FIGHT

Frontiers in Cellular and Infection Microbiology

Mycotoxin: Its Impact on Gut Health and Microbiota

"This review comprehensively discussed the role of mycotoxins (trichothecenes, zearalenone, fumonisins, ochratoxins, and aflatoxins) toward gut health and gut microbiota.

Findings revealed that the gut microbiota is capable of eliminating mycotoxin from the host naturally, *provided that the host is healthy with a balance gut microbiota.*

Mycotoxins have been demonstrated [to modulate] gut microbiota composition.

Most, if not all, of the reported effects of mycotoxins, are negative in terms of intestinal health, where beneficial bacteria are eliminated accompanied by an increase of the gut pathogen.

The interactions between gut microbiota and mycotoxins have a significant role in the development of mycotoxicosis, particularly hepatocellular carcinoma. "

Probiotics

Not "a given" with mold-related illness. SIBO common sequelae.

Function as adsorbents primarily, some degrade the mycotoxin to lower-toxic derivative.
Empirically: dose with greens and bitters, increased efficacy and adherence.

L. plantarum C88/MON03 ~
Adhesion of Aflatoxin in lumen, organoprotective against Zearalenone toxicity.
Upregulates antioxidant enzymes,↑GST expression via Nrf2 pathway.

L. rhamnosus GAF01 and GG~
Binds Aflatoxin, counteracts RBC, WBC, lymph immunotoxic effects.

L. casei strain Shirota ~
Strain is impt! (strains impact histamine), hepatoprotective, chlorophyllin ↑ efficacy.

Bacillus licheniformis strain CK1 ~
More effective than clay-based binder for Zearalenone. Acidic tolerance to stomach acid.

Bacillus pumilus and licheniformis (multiple strains) ~
Degrade Enniatin B to levels below the detection limit, reduce deoxynivalenol.

PMID: 28129335, 24738739, 23030351, 21816119, 29641608, 31330922, 39338565, 36356030

A note on binder research

Many binder studies are motivated by companies trying to find a profitable use for their waste materials, so not only do they *not* have to pay to dispose of their waste material, they actually make money on it.

NOT because a binder is the best binder for the job.

Follow the $. Animal feed binders are chosen due to cost, not by what would be optimal.

Additionally, some binding is best done in the feed, not once exposed to digestive juices.
Read the whole paper to understand methods rather than take an abstract glance.

*****REMINDER: binders are not required to recover from mold-related illness*****

Concern over Rx bile sequestrants

Prescription bile sequestrates are used for far too long IMHO.

My general cut-off is 1 month. May pulse 2 days/wk for longer but not much longer.

They DO cause nutritional depletions of fat-soluble nutrients and microbiome alterations.

Cholesterol → hormone imbalance.

Case ~
18 months.
I d/c'd Rx, repleted phospholipids, EFAs, ADEK, CoQ and gave pregnenolone.
Nutritional depletion and subsequent hormone imbalance ended up being the only cause of residual CIRS sxs.
She didn't have CIRS anymore, she now had nutrient deficiencies caused by the Rx causing the same sxs.

Alternate binder refresher

Benefits ~
Can take WITH food! And usually less constipating.

Insoluble fiber ~ 2-4 Tbsp divided daily (caution SIBO)
25% as effective as CSM, and also feeds microbiome and increases serum bile acids (via epigenetic effect).
"(0.10 ± 0.007) is designated as 1.0; excretion rates are expressed as multiples of this control rate: CSM (6.8 ± 0.6), P < 0.001; Metamucil (1.7 ± 0.1), P < 0.001."
"Psyllium induced expression of genes mediating bile acids (BA) secretion. Resulted in elevated level of fecal BA, reflecting their removal from enterohepatic circulation but, in stark contrast to the BA sequestrant cholestyramine, increased serum BA levels."

Steamed kale, collards, mustard greens ~ 1 packed cup steamed, divided daily. (caution oxalates)
13% as effective as CSM, and also provides bioflavonoids for kidney detox support. Chlorophyll source.

Dried, pulverized aloe (glucomannans), okra, lemongrass, chlorella, konjac root.

SIBO friendly - sunflower, sesame, pumpkin (finely grind and use up to ¼ cup over meals).

Bile reabsorption blocker - Taurine. Empirically, minimum effective dose for this effect: 500mg qd.

PMID: 4584910, 36828279, 30187492, 19083431, 26359588, 19952359, 33529081, 32371067, 30332612

Other adsorbents

Bind unconjugated toxicants (ingested mycotoxins and some trichothecenes). Most benefit with endotoxins.

Cons ~ Not specific to bile conjugates. Impurities. Nutrient depletions. GI inflammation. Negatively modify microbiome. Thyroid hormone disruption.

Charcoal ~
Useful around the onset of antifungals; benefit with toxic/acidic die-off.
Adsorption of iodine.
I don't use more than 1 month in most cases.
(exception - concomitant parasite treatment using pulsed LT when taking antiparasitic regimen "on" days)

Clay ~ additional benefit - possible trace mineral source
I rarely use for mold; with concomitant diarrhea (rice water diarrhea). Used in animal feed b/c lowest cost.
Risk of toxic metal contamination; lead, cadmium, thallium. Decreased Vitamin A, T3, T4 hormones.
Adsorption of zinc, manganese, selenium, cobalt, plus large organic molecules, albumin, complex ions, enzymes. Nutritional safety studies in humans limited to 3 months, tested serum minerals, not RBC minerals.
Avoid in kids. Caution with LT use longer than 2 weeks. Consider pulsed dosing, if at all.
Animal research: dentition and bone, milk production, detoxification.
"…clays may directly interact with GI cells and can elicit an inflammatory response"

PMID: 37903944, 35619608, 37903944, 31515765, 18569006, 29210610

Why I don't use Sacch B

Yes, there are animal studies that show that Saccharomyces boulardii is an effective binder when animals are fed moldy feed. Fair enough.

Except, these are studies where the animals are fed controlled diets.

My patients became carb cravers when I added Sacch B, making their yeast burdens worse.

Oddly, they also had increases in neurological, cardiac, and hormonal imbalance sxs.

A review paper on Zearalenone reported that Sacch. B. can keep Zearalenone in its reduced state — meaning, it keeps it around longer and in its more damaging form.

Zearalenone is a cardiotoxic and neurotoxic endocrine disrupter.

Same symptoms aggravated in the patients I put on Sacch B.

Zearalenone is also a persister-type mycotoxin.

We have so many other options for binding. Why use one that increases the risks?

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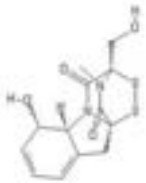
Gliotoxin



Opens the doorway to fungal infection.

Treat this first, because will complicate treatment of all other mycotoxins.

A sign that "mold is on the move" ~
- Most abundant metabolite produced during hyphal growth.
- Uses sulfur for its production.
- May be produced in vivo and contribute to the etiology of fungal infections.

Many who test positive for this have a significant yeast burden.

Correlated with sulfur intolerance and histamine sensitivity.

Primary aim of treatment is to stop its production.

Gliotoxin presence = "race to antifungals"

Mold Source: Aspergillus spp., Trichoderma spp.

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Gliotoxin | Health Impacts

One of the most diverse toxins in how it affects the body.

Cutaneous/mucocutaneous
(sinus, lung, mouth, throat, GI, bladder, vagina)
Immunosuppressive
Immunotoxic
Neuroinflammatory
Neurotoxic
Hepatotoxic
Highly oxidative
Genotoxic
Cytotoxic-potent inducer of apoptotic cell death in a number of cells
(immune, hepatic, neuro)

1° excretion - hepatic

Gliotoxin | Sn/Sxs

Pruritis
Mast cell reactions
Post-prandial bloating
Sweet cravings
Nausea, Constipation
Intolerance to sulfur-containing foods
Chemical sensitivities
Fatigue
Cognitive difficulties
Headaches
Anxiousness
Frequent mood changes
Despair/suicidality
Incoordination/MS
Insomnia
Frequent infections
Delayed wound healing

Possible signs ~
Fungal dermatological infections
Onychomycosis

Gliotoxin | Antifungal

Temporary avoidance of sulfur-containing foods ~
ie: garlic, onions, eggs, fish, and the Brassicacea family (broccoli, kale, cauliflower, cabbage, Brussels sprouts)

Binder. Aloe (antihistamine immunomodulatory adsorbent)

Antifungals ~ herbals, Rx-herbal combo
Systemic
Nasal
Topical: fungal rashes, toenails

Combine with bitters. 5-10 drops on the tongue 10 minutes before meals.
Extra-oral bitter taste receptors are involved in regulating some aspects of innate immunity.
In pts with CRS, local innate immunity deficiencies predispose to sinus mucosal bacterial colonization/infection, including *deficient functioning of the extra-oral bitter taste receptor.*

Gliotoxin | Thiol Support

Redox without harm. Excreted via GSH, but sulfur could help fungi first.

Antioxidants! (work against fungal ROS, caspases)

Mixed bioflavonoids ideal. Every color band but especially green (green tea polyphenols)

Molybdenum. 250mcg qd-bid.

Glutathione precursors. Vit C, Vit E (tocotrienols), B-complex, Mag, Se

Zinc. Use cautiously, even though a glutathione precursor.
Assists Aspergillus in biosynthesis of gliotoxin.

Thiols. Use cautiously, and not until on antifungals.
Ex 1: NAC (cell study: “completely abolished the gliotoxin-induced caspase-3-like activity, cytotoxicity, and reactive oxygen species”)
Ex 2: GSH study (cell study: “things that reduce the internal sulfide bond interfere with its effect on cell viability and apoptosis.”)
****But in vivo, may add antioxidant protection* ***to fungi.***

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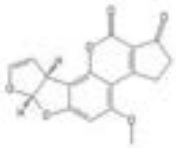
Aflatoxin



Not a liver lover.

Plus pathogens, cancer, gut, neuro, birth defects. (liver, lung, leaky, leuko, littles)

The most potent hepatocarcinogen recognized in mammals. Listed as a Group I carcinogen by the WHO International Agency for Research on Cancer.

Recognized teratogen. Pregnant patients MUST be removed from exposure.

Promotes viral replication due to innate immune effects.

Significant down-modulation of Vitamin D receptor in intestine & kidneys.

Increased gut pathogens due to microbiome impacts - even if inhaled not ingested.

BBB breakdown. Stops cell proliferation of brain astrocytes.

Secondary metabolites are more toxic than the original.

Primary aim of treatment is phase II liver support, and nutritional/immune support.

Mold Sources: Aspergillus flavus, A. parasiticus

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Aflatoxin | Health Impacts

Hepatotoxic
Hepatocarcinogenic
Nephrotoxic
Carcinogenic
Mutagenic
Genotoxic
Teratogenic
Immunotoxic
Neurotoxic
Cardiotoxic
Polymorphisms in CYP3A5 affect susceptibility.

1° excretion - hepatic

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Aflatoxin | Sn/Sxs

Possible signs ~
Dark urine

Fatigue
Insomnia
Anemia
Progressive inflammation
Metabolic acidosis
Accelerated aging
Reduced immunity
Cognitive dysfunction or decline
Incoordination
Headache
Visual fatigue
Chronic sinusitis

Hepatic pain or fullness
Loss of appetite
Nausea, vomiting
Dysbiosis
Jaundice
Hepatocellular carcinoma
Chemical sensitivity
Toxic metal accumulation
Steroidal hormone imbalance
Miscarriage or preterm birth
Stunted growth in children

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Alfatoxin | Liver

Therapeutic Diet ~
Antioxidant-rich diet, including colorful vegetables rich in bioflavonoids.
Protein-rich diet high in essential fatty acids, such as wild-caught fish, wild game, pasture-raised chicken eggs.

DHA (docosahexaenoic acid). Hepatoprotective, chemopreventive against Aflatoxin.

Green tea. 2-4 cups daily. Protective against Aflatoxin-induced cell injury.

Binder. Bile sequestrants; ideally natural in lieu of Rx.
2 Tbsp rice bran (ideally purple rice bran) daily as an insoluble fiber binder, as well as a possible antimutagen against Aflatoxin.

Liver ~
Turmeric (Curcuma longa). Hepatoprotective. Ameliorates Aflatoxin-induced lipid peroxidation.
Glutathione. Start very low if still exposed, yeasty, or Gliotoxin.
Or use glutathione inducers if not tolerated - ALA, NAC, Selenium, Milk thistle.
Red sage (Salvia miltiorrhiza/Danshen). Hepatoprotective from Aflatoxin.

Aflatoxin | Protective

Vitamin D. Dose to lab values of 60-90 ng/ml (150-225 nmol/L) for a minimum of 3 months in order to up-regulate receptors.

Vitamin E as tocotrienols. Immunoprotective.* (Aflatoxin-specific effect.)

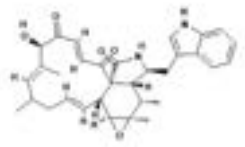
Melatonin. Hepatoprotective, cardioprotective. Can be used as a pre-treatment for known exposure events.
Dose at dinnertime to mitigate morning grogginess.

Quercetin. Hepatoprotective, nephroprotective by reducing albumin binding, and genoprotective against Aflatoxin.

Resveratrol. Chemopreventive, genoprotective.* (Aflatoxin-specific effect.)

Grape seed extract. Alleviates Aflatoxin-induced immunotoxicity and oxidative stress.

Chaetoglobosin



Resistance factor

Poor resistance to infection and leads to resistance to treatment.

Similar to Gliotoxin, it helps Chaetomium break through our immune resistance against its invasion, and subsequently reduces resistance to all pathogens.

A cytochalasin alkaloid. Chaetaglobosin-affected patients may become sensitive to other alkaloids.

Blocks actin ~
Cytotoxic effect by degrading cell infrastructure.

Blocks actin (implications-inhibit cell division, neurite pathfinding, cell motility)

Impairs cilia to reduce Chaetomium clearance, especially of nasopharyngeal cavity. Efficiently absorbs via respiratory tract, much less so in the gut, ∴ toxicity not from ingestion.

Complicates CIRS diagnosis. False negative TGF-beta.

Primary aim of treatment is nonpolar biliary conjugation and restoration of cytostructures.

Mold Sources: Chaetomium spp, Cylindrocladium floridanum, Stenocarpella spp, and some Aspergillus and Penicillium spp.

Chaetoglobosin | Health Impacts

Cytotoxic activity across a number of cell types
Myopenic
Immunosuppressive
Myelosuppressive
Neurotoxic
Toxic to the respiratory tract
Genotoxic
Reproductive toxin
Metabolic disrupting
Acute exposure may lead to liver, kidney, and pancreas injury.

1° excretion - hepatic

Chaetoglobosin | Sn/Sxs

Rapid aging
Eye, skin, and respiratory irritation
Skin rashes and thinning
Chronic sinusitis
Chronic productive cough
Fatigue, cognitive fatigue
Sarcopenia, poor muscle tone
Muscle weakness, exercise intolerance
Hypothyroid
Recurrent infections, especially of the skin, lungs, and mucosal surfaces
Neutropenia
Thrombocytopenia
Splenomegaly
Chemical sensitivity
Unstable blood glucose
Reduced sperm motility

Possible signs ~
Reduced TGF-beta

Chaetoglobosin | Basics

Therapeutic Diet ~
Proline- and tyrosine-rich animal and fish protein (muscle, glands, and skin)
Cruciferous vegetables (sulforaphane as Nrf2 activator) - enhance cellular resilience

Temporary avoidance of alkaloids, coffee, cacao, tea (caffeine, theobromine), tomatoes (tomatine) and potatoes (solanine).

Therapeutic Movement ~ resistance training for enhanced actin-myosin cross-bridging.

Binder. Bile sequestrants. One of the few times I begin with colesevelam (Welchol), dose bid for 1 month, away from meals. Then transition to insoluble fiber.

Optimize fat-soluble nutrients — phospholipids, vitamins A, D, E, K (due to its nonpolar nature, will disrupt absorption)
Take with ox bile or bile salts/TUDCA to enhance absorption.

Chaetoglobosin | Structure & Detox

Cytoskeleton ~
Bioplasma cell salts. 10 pellets under tongue throughout the day.
Amino acids - proline (1g), glycine (1g), tyrosine (500mg), taurine (500mg) as tolerated. [Actin: proline & glycine. Repletion: tyrosine (Chaetomium uses it to make chaetoglobosin). Bile: taurine and glycine]

Green tea catechins: Helps maintain cytoskeleton integrity. Actin filament stabilization, cilia movement, anti-proliferative.

Nicotinamide riboside. Plays a role in regulating the cytoskeleton by acting as a source of electrons for NADPH oxidases, directly modifying the cytoskeleton (actin, tubulin, and intermediate filaments) and cytoskeleton-associated proteins.

Protective/Detox ~
Resveratrol. Chemopreventive.
Milk thistle. Hepatoprotective.
Turmeric. Promote proliferation of certain stem cells and normal cells at low doses. May help mitigate the immunosuppressive effects of chaetoglobosin by activating and supporting adaptive immune responses.
Grape seed extract.

Chaetoglobosin | Immune

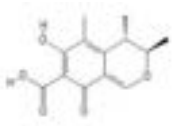
Immune ~
Thymus and spleen glandular.
Larch (Larix occidentalis). Enhances natural killer (NK) cell cytotoxicity while being anti-inflammatory. Enhances beneficial gut microflora and increase the production of short-chain fatty acids.

Melatonin. Anti-inflammatory and immunomodulatory. Could potentially help protect lymphoid organs from necrosis.

Antimicrobial ~
Thyme. Good antifungal choice for Chaetomium, as not only is it a potent broad-spectrum antimicrobial, but it also increases mucociliary-beating frequency, overcoming Chaetoglobosin's action to paralyze cilia.
Garlic. Combats both of Chaetomium's resistances; first by being an immune modulator which boosts resistance to infection, and second by combining well with antifungal agents to reduce drug resistance.

Nasal rehabilitation. Humic acid, Lactobacillus sakeii and caseii.

Citrinin



Wastes water and energy

Often found in an indoor environment along with Ochratoxin, compounding the nephrotoxic impact.

Mitochondrial dysfunction, most affecting organ systems under high demand, such as the heart, digestion, and reproduction.

Inhibits the renal vasopressin receptor.

Can impede fertility and prevent successful pregnancy.

Inhibits cholesterol and ergosterol synthesis.

Secondary metabolite is mutagenic.

Primary aim of treatment is ADH restoration to assist delivery to renal system for clearance.

Mold Sources: Aspergillus niger, A. awentil, A. ostianus, A. fumigatus, A. niveus, A.awamori, A. parasiticus, Penicillium citrinum, P. expansum, Monascus

Citrinin | Health Impacts

Alimentary toxic
Cardiotoxic
Cytotoxic
Genotoxic
Hepatotoxic
Nephrotoxic
Teratogenic
Embryotoxic
Carcinogenic

Permeates into the mitochondria, alters Ca2+ homeostasis, and interferes with the electron transport system.

1° excretion - renal

Citrinin | Sn/Sxs

Fatigue, commonly with muscle pain
Reactive blood sugar
Polyuria, Dysuria
Edema
Nephrogenic diabetes insipidus
GERD
Ulcers, hematochezia
Nausea, vomiting, diarrhea
Food sensitivities
Chemical sensitivities
Heart palpitations
Dyspnea
Angina
Menstrual changes
Miscarriage
Infertility

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Citrinin | Trophorestoration

Therapeutic Diet ~
Temporary grain-free diet.
Radishes plus vegetables/fruits in the deep red color band for pelargonidin* (*Citrinin-specific Nrf2 restorative hepatoprotection)
Butyrate-rich foods - butter, cabbage, radicchio, white part of spring onion, broccoli, Brussels sprouts

Green tea - particularly if skeletal muscle fatigue* (*Citrinin-specific effect)

Kidney ~
Homeopathic Vasopressin. Follow dosing document.
Kidney glandular.

Binder. Aloe glucomannan (promotes intestinal stem cell-mediated epithelial regeneration.)

CoQ10. Mitochondrial support.

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Citrinin | Protect

Vitamin E as tocotrienols. Genoprotective against Citrinin in hepatocytes.

Melatonin. Nephroprotective.

Resveratrol. Hepatoprotection seen when coadministered with ROS scavengers NAC & tocotrienols* (*Citrinin-specific effect)

Grape seed extract. Nephroprotective.

Glutathione. Glutathione inducers if not tolerated - ALA, NAC, Selenium.

Alpha-lipoic acid as the R-Lipoic acid isomer. Attenuates kidney injury, especially in the presence of lipopolysaccharides.

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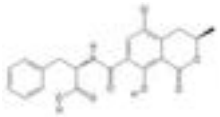
Ochratoxin



Persister mycotoxin

High affinity binding to albumin + long half life → negligible glomerular filtration, creating a mobile reserve.

Class 2B possible human carcinogen by the WHO International Agency for Research on Cancer.

Reduces immune function.

Mitochondrial dysfunction. Strong negative effect on cellular glutathione and ATP production.

Myocardial injury.

More neurotoxic and carcinogenic to males. Reduction of intracellular zinc. Correlated to the male prevalence of autism.

Crosses placenta where it concentrates → higher levels in the placenta and 2-fold higher levels in the fetus's blood than that of the mother.

Mold Sources: Aspergillus ochraseus, A. niger, Penicillium verrucosum, P. nordicum, P. chrysogenum

Ochratoxin | Health Impacts

Nephrotoxic
Nephrocarcinogenic
Hepatotoxic
Neurotoxic
Cardiotoxic
Immunotoxic
Genotoxic
Carcinogenic
Embryotoxic
Teratogenic

1° excretion - renal

Ochratoxin | Sn/Sxs

Fatigue
Brain fog
Headache, migraine
Hyporexia
Nausea
Chemical sensitivity
Pruritus
Hyperuria, or may progress to oliguria
Edema
Hypertension
Angina
Muscle weakness and/or cramps
Exercise intolerance
Frequent infections

Possible signs ~
Low albumin
Reduced/ing GFR

Ochratoxin | Bioflavonoids before binders

Flavonoids displace Ochratoxin from albumin, and prevent or attenuate its toxic consequences on albumin binding.
First-pass metabolism of flavonoids is high, so frequent, repeated dosing is recommended.
A mixed bioflavonoid supplement is ideal as it covers the wide range of the flavonoids studied.

Focus on *astaxanthin, luteolin, quercetin, lycopene* for Ochratoxin-specific albumin displacement activity.

Astaxanthin. Red fat-soluble pigment protects against Ochratoxin-induced myocardial and lung injury via Nrf2 pathway.

Luteolin. Attenuates viability loss in kidney cells and lymphocytes, while decreasing DNA damage of blood cells.

Quercetin. Suppresses cytotoxicity, oxidative stress, and alteration of antioxidant defenses via activation of Nrf2 pathway and down regulation of NF-κB and COX-2. Zinc ionophore.

Lycopene. Alleviates Ochratoxin-induced DNA damage, and renal oxidative stress and apoptosis.

Ochratoxin | Nephroprotective

Therapeutic Diet ~
Eat the rainbow of colorful vegetables, 5-7 servings daily.
Plenty of good fats every day.
Flavor dishes with rosemary, mint, sage, and thyme. (Ochratoxin-specific mycotoxin neutralizing activity)

Rosemary (Rosmarinus officinalis)
Rosmarinic acid has a significant cytoprotective effect against Ochratoxin via decreased ROS production and improvement in viability with less inhibition of protein and DNA synthesis. Also antifungal.

Binder. Bile sequestrants; ideally natural using insoluble fiber.

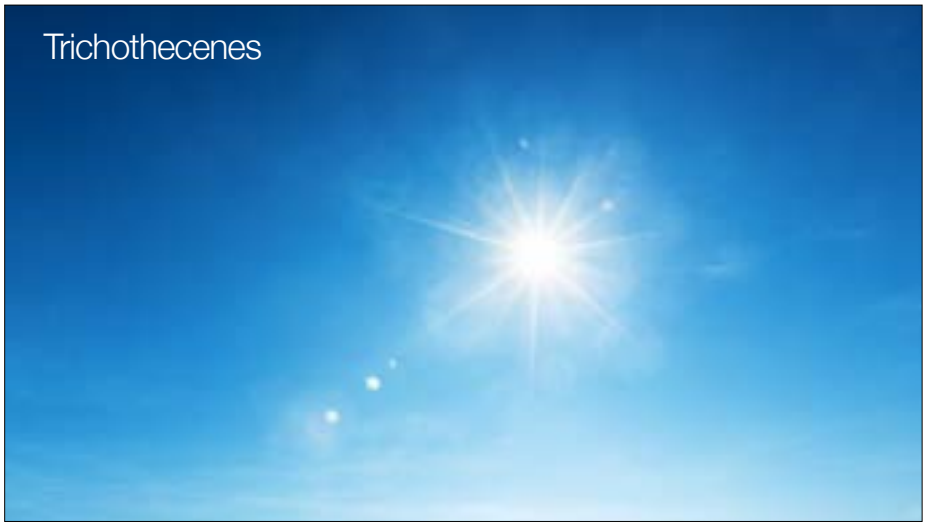
Melatonin. Nephroprotective. Zinc ionophore.

Tocotrienols. Improves blood pressure. Restores GFR, absoluted fluid reabsorption, and renal antioxidant enzyme activity.

Glutathione. Nephroprotective, hepatoprotective.

NAC+Selenomethionine - combination improved immunotoxic effects on macrophages.

Trichothecenes



Trichothecenes

Potent poisons

Large family that's chemically related. More potent that other mycotoxins due to potency, absorption, activation, and their range of systemic effects.

Require smaller parts per billion to exert their effects, with the macrocyclic trichothecenes (roridins, verrucarins, and satratoxins) generally considered to be among the most toxic.

Developed as biowarfare agents. Significantly more toxic than other well-known chemical warfare "blister agents", such as mustard gas. Yet found in WDBs.

Don't require metabolic activation. Active on contact, acting rapidly and directly on target tissues, and potentially causing blistering and bleeding.

Severe health effects: GI, skin, kidney, liver, immune and hematopoietic progenitor cellular systems, with potential for systemic toxicity to lead to weakness, shock, and even death.

Vomiting and diarrhea are warning signs, as they've been observed at ⅕-1/10 of the lethal dose.
Patients exhibiting these symptoms must be immediately removed from the environment to prevent continued exposure and risk of death.

Trichothecenes | Health Impacts

Dermatologic toxicity
Ocular toxicity
Respiratory toxicity
Gastrointestinal toxicity
Multi-organ toxicity
Reproductive toxicity
Immunotoxic
Hematologic toxicity,
Neurotoxic
Genotoxic
Protein synthesis inhibition
Mitochondria dysfunction

1° excretion - hepatic and gastrointestinal

Trichothecenes | Sn/Sxs

Skin irritation, tenderness, redness, itching, desquamation - can be severe
Weakness, muscle loss
Fatigue with lassitude
Cognitive impairment
Dizziness, loss of coordination
Blurred or changing vision
Nasal irritation, pain, itching
Throat irritation, pain, itching
Sneezing, runny nose, nosebleeds
Wheezing, cough (potentially bloody)
Difficulty breathing, chest pain

Loss of appetite
Nausea, vomiting (potentially bloody)
Abdominal pain
Diarrhea (potentially bloody)
Altered intestinal permeability
Infertility
Anemia
Hypotension
Inability to mount a fever
Increased susceptibility to infections
Bleeding disorders

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Trichothecenes | Flavonoids

Therapeutic Diet ~
Green tea
Extra-virgin olive oil
Brassicaceae family (sulforaphanes have been shown to induce glucuronidation gene expression.)

Binder. Insoluble fiber or super-fine ground dried okra: 2-4 Tbsp divided daily with food; or other bile sequestrants for bile conjugated trichothecenes.
Lemongrass powder: 1/8-1/4 tsp twice daily with food, for unconjugated trichothecenes.

Probiotic. Bacillus spp for intestinal degradation of unconjugated trichothecenes in the gut.

Bioflavonoids.
Green tea polyphenols. Cytoprotective against multiple trichothecenes.
Lycopene. Hepatoprotective. (T-2 toxin specific effect.)
Quercetin. Myoprotective.
Rutin. Hepatoprotective.
Hesperidin. Hepatoprotective.

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Trichothecenes | Protection

Milk Thistle.
Promotes regeneration of the liver via combating trichothecene protein synthesis inhibition.

Black cumin.
Hepatoprotective. (verrucarin, roridin)

Red sage, Turmeric, Schisandra.
Reduce intestinal epithelial cell injury and mitigate intestinal barrier disruption..

Melatonin. Highest tolerated dose up to 20mg hs.
Alleviates damage to spleen and thymus, and oocytes.

Combine CoQ10 and Vitamin E to support glutathione. (Trichothecene-specific effect.)

Selenium. Immunoprotective. (T-2 toxin)

Glutathione. Cytoprotective. (T-2 toxin)

Leucine. Trichothecenes strongly inhibit leucine incorporation in hypothalamus, causing brain protein synthesis inhibition and hypothalamic dysregulation, with a net antipyretic effect.

Taurine. Hepatoprotective. (T-2 toxin)

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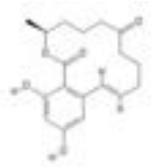
Zearalenone



Endocrine disruptor, and so much more

Non-steroidal estrogenic mycotoxin. Same binding affinity to ERβ as ERα, correlated to associated risk factors for metabolic, CV and neurological diseases, osteoporosis and some estrogenic cancers.

Binds to and lowers albumin (correlated with increased risk of C.diff).

Arrhythmic. 2nd-degree AV block, atrial bradycardia, decreased contractile power.

Immune. Impairs macrophage function, toxic to thymus, reduces B-cells.

Can be activated by Candida and Saccharomyces species.

Primary aim of treatment is enhance kidney clearance and manage endocrine effects.

Mold Source: Fusarium spp.

Zearalenone | Health Impacts

Xenoestrogenic
Xenogenic
Cardiotoxic
Immunotoxic
Hepatotoxic
Hematoxic
Embryotoxic
Genotoxic
Plausible carcinogen

1° excretion - renal

Zearalenone | Sn/Sxs

Hyperestrogenic syndromes
Acne
PMS, menstrual alterations
Precocious puberty
Osteoporosis
Hypoandrogenism in men
Central weight gain
Infertility, both genders
Fatigue
Dyspnea, with or without chest pain
Bradycardia
Palpitations
POTS
Neurocardiogenic syncope
Dizziness/vertigo

Tinnitus
Edema
Sinusitis
Frequent infections, pneumonia
EBV reactivation
C. difficele
Onychomycosis

Possible signs ~
Increased MCV and hematocrit
Reduced total protein
Reduced albumin
Increased transaminases and bilirubin
ECG: 2° AV block Mobitz type II > I

Zearalenone | Bioflavonoids

Therapeutic Diet ~
Kefir. Protection against Zearalenone-induced oxidative damage.
Brassicaceae family (broccoli, broccoli sprouts, cauliflower, kale, Brussels sprouts, cabbage, turnips)
Spices: Ginger, Cinnamon, Turmeric, Thyme, Lemongrass, and Rosemary.
Anti-mycotoxigenic and antifungal against Fusarium.
Ginger. Active constituent zerumbone prevents Zearalenone-induced liver injury.

Hawthorne (Crataegus oxycantha). Cardiotonic. Dilates arteries. Antioxidant via carotenoids which are effective in the protection against Zearalenone-induced toxicity in cardiac tissue.

Resveratrol. Protective bioflavonoid against Zearalenone-induced oxidative damage, some genoprotective activity.

Grape seed extract. Hepatoprotective. Reduces maternal developmental defects.

Binder. *Avoid the use of Saccharomyces yeasts, as they bioactivate Zearalenone.
Use bile sequestrants; ideally natural using insoluble fiber.

Zearalenone | Protect

Enhance xenoestrogen metabolism ~
Potassium-hydrogen-glucarate, DIM, indole-3-carbinol.

Melatonin. Significantly ameliorates Zearalenone-induced impairments in kidneys, reproductive cells, and embryonic development.

Glutamine. Attenuates the Zearalenone-induced increase in cytotoxicity, cell apoptosis, and intestinal permeability.
*Not appropriate for PANDAS/PANS.

Vitamin E as tocotrienols. Genoprotective against Zearalenone-induced effects on kidney and bone marrow.

N-Acetyl Cysteine (NAC). Nephroprotective. Preserves mitochondrial function. Protective to reproductive organs, testes.

Milk thistle. Alleviates Zearalenone-Induced hepatotoxicity and reproductive toxicity.

Omegas (Epanova)

Benefits. Structural component of the cell membrane and mycotoxin protective effect.

Rx: omega-3-carboxylic acids (Epanova), omega-3-acid ethyl esters (Lovaza, Omytrg), icosapent ethyl esters (Vascepa)

Dose: 4 grams daily, best divided. (For salicylate sensitivity double or more.)

Omega-3-carboxylic acids (Epanova) are the closest in molecular structure to docosahexaenoic acid (DHA) among the prescription omega-3 fatty acid products mentioned.

This is because:
Epanova contains a mixture of polyunsaturated free fatty acids, including DHA in its natural carboxylic acid form, structurally most similar to natural DHA.
Lovaza and Omtryg contain omega-3-acid ethyl esters, which are esterified forms of EPA and DHA. While they contain DHA, it is not in its natural free fatty acid form.
Vascepa (icosapent ethyl) contains only the ethyl ester of EPA and does not include DHA.

The key difference lies in the chemical structure:
DHA is a free fatty acid with a carboxylic acid group (-COOH).
Epanova contains omega-3 fatty acids in their free fatty acid form, including DHA.
Lovaza and Omtryg contain ethyl esters of omega-3 fatty acids, where the carboxylic acid group is replaced with an ethyl ester (-COOCH2CH3).
Vascepa contains only EPA in ethyl ester form and does not include DHA.

Prescription Vitamin D

50,000IU weekly of Vitamin D2 (ergocalciferol).

Weekly bolus better than daily for all vitamin D supplementation. True for mold?

D2 considered the less optimal form than D3 (cholecalciferol) but better than none.

A meta-analysis of RCTs indicated that supplementation with vitamin D3 had a significant and positive effect in the raising of serum 25(OH)D concentrations compared with the effect of vitamin D2 (P = 0.001).
*When the frequency of dosage administration was compared, there was a significant response for vitamin D3 when given as a bolus dose (P = 0.0002) compared with administration of vitamin D2, *but the effect was lost with daily supplementation.*

D2 is sourced from fungi, so some mold patients will have sensitivity. In that case, use D3 supplemental form which is sourced from lanolin from sheep's wool.

PMID: 22552031

Probiotic

Visbiome & VSL#3 ~
- 900 billion CFUs, “extra strength”
- 8 strains: Lactobacillus, Bifidobacterium, Streptococcus
- Several RCTs showing efficacy, mostly for digestive conditions.
- Dose pulsed, between antifungal pulsed days.
- Caution: contains Strep strains that may flare PANDAS/PANS

Restora Rx ~
- 12 billion. Single strain Lactobacillus casei KE-99
- Claims better attachment to intestinal wall, no human studies.
- Caution: uses folic acid (synthetic form, can lead to folinic acid deficiency)

Assess patient’s out-of-pocket amount and compare mold-specific strains available OTC.

Other cautions:
- OTC brand names: Florastor (Sacch B.)

Bile support

Prescription cholagogue “oil change” in lieu of supplemental ox bile or bile salts. Assists with mold-specific motility issues.

Ursodeoxycholic acid (Actigall, Ursodiol) ~
- Natural component of bile; makes up 1-3% of naturally occurring bile acids. ↓absorption of cholesterol (bile).
- Rx’d to dissolve cholesterol-based gallstones, first line alternative to surgery.
- Dose: 300mg bid with largest meals
- S/E: GI upset (esp with tid dosing; breakfast)
- Cautions: bile duct blockage

Tauroursodeoxycholic acid (TUDCA generic) ~
- Taurine conjugate of ursodeoxycholic acid

Deoxycholic Acid (Kybella) ~
- “For improvement in appearance of mod-severe fullness [from] submental fat.”
- Injectable to submentum, double chin
- Thought question: clearing fat or toxin?

Still not been approved by the regulatory authorities as a safe drug during pregnancy. Not recommended in first trimester. In 2nd and 3rd, only for dx indications.

Bile sequestrant

Colesevelam. Generic available.

Non-specific binder. Binds conjugated fat-soluble toxicants that are bound in bile, such as mycotoxins, and endotoxins.

I rarely use (Chaetoglobosin exception). My typical limit is 1 month, then I transition to a nourishing binder.

Dose: 625mg tab, 2-3 po q12 hrs
- With meals + plenty of liquid
- Oral suspension available for peds, but contains phenylalanine, caution PKU
- Chewable bar: 3.75 g (1 bar) po qd (PKU)

S/E: constipation, dyspepsia, h/a, nasopharyngitis

Interactions:
- “Monitor closely” drugs for DM, warfarin, seizure
- Impacts absorption of co-administered therapies - diuretics, CVD, Abx

Administer 4 hrs prior to colesevelam:
- Rxs w known interactions/narrow therapeutic index
- Fat-soluble vitamins
- Oral HRT & contraception

Pregnancy category: "no adequate and well-controlled studies of colesevelam HCl use in pregnant women”

Bowel motility

Prucalopride ~
- 1-2 mg po daily
- GI prokinetic for impaired motility. Selective, high affinity 5-HT 4 receptor agonist. Stimulates motility in the GI tract. Causes a release of ACh and further contraction of the muscle layer of the colon and relaxation of the circular muscle layer.
- Manages and reduces risk of SIBO.

Metamucil ~
- Psyllium husk fiber, bulk-forming fiber to manage either diarrhea or constipation. Also a bile sequestrant.
- Caution: artificial additives, aspartame (sugar-free), food colorings, fillers.
- May be contraindicated in SIBO.

Senokot ~
- Laxative made from the herb Cassia Senna, useful for constipation. Rich in anthraquinone glycosides like Sennosides which bring water to the colon.

Caution ~
- Miralax. Polyethylene glycol 3350 (PEG) contraindicated in MCAS.

Antifungal

Resets the microbiome. Fungi are a formidable force. Meet force with force.

Benefits ~
Reduces sensitivities to other treatments; histamine and mast cell, sulfur, oxalate, salicylate

Itraconazole currently most successful against resistant species.

Dose: 50-100-200mg daily x 30-90 days, as tolerated.

Pharmacokinetics for triazoles lends to qd dosing over bid dosing due to extensive first pass liver metabolism.

Can pulse long term (1+ years if needed)

Monitor liver enzymes at 45-60 days, EKG at 60-90 days if qd dosing.

Nuances of antifungals

I go into this more extensively in the mold certification course.

Herbals vs Rx? or with Rx?

Start herbals first if tolerated. Not as strong as Rx (in most cases but not all) but not as myopic either = fewer issues with resistance.

If needing Rx, when possible add rather than replace herbs. Garlic the broadest.

Intranasal a common oversight, and IME moves the needle the fastest.

Duration. Longer lower dose more effective than stronger shorter dose. Too short increases resistance.

Topical for pruritus

5% Doxepin cream. Apply a thin layer to affected area 4x/day.

Potent H1 and H2 antagonist.

In studies, significant reduction in pruritus in patients with various conditions including atopic dermatitis, urticaria, and other forms of chronic itch.

Response usually in the first 24 hours.

Cautions ~
Contact dermatitis with localized stinging or burning (improved with compounding and combining with topical LDN), drowsiness which decreases in frequency and severity over time (liposomal cream may reduce systemic effects.)

Clinical tip ~
Urge patient to start with the least amount of cream possible. Quick relief will induce them to slather it on, with higher chance for contact dermatitis reaction.

PMID: 8089287, 37869926

Consider

Route of detoxification and excretion of the toxicant

Sensitivity

Constipation

Biofilm

Level of Mold Denial

Sensitivity

Can they tolerate what you recommend?

Therapeutic diet first? (Ie: histamine, salicylate, oxalate, sulfur)

Need to go straight to antifungals?

Limbic retraining needed?
Not all limbic healing options are “mind” programs.
Aromatherapy: essential oils of lavender, blue tansy, lemon balm, bergamot, German chamomile, black spruce.
Also FSM, alpha-stim, EMDR, acupuncture, homeopathy, sound bath, laying on of hands, intercessory prayer, etc.
Get creative!

Action step: refer to a mold-literate practitioner

No Binders If Bound Up

Constipation allows for ~
Disassociation of the mycotoxin from the bile
Risk of enterocyte damage
Re-entry via enterohepatic circulation

Source of stagnation is between HCl and bile, and the neurotoxic effects on peristalsis when mold and mycotoxins are involved.

Laxatives often fail because addressing lower colon, while the stimulation needs to be earlier in the digestive cascade.

Bile, bile salt precursors, pre-binders (cholagogues, bitters), and if those don’t work, add betaine HCl. May need motility agent to overcome neurogenic paresis.

Biofilm

To what extent is biofilm playing a role and are you addressing it adequately?

Gut, nasal, lung, bladder, inner ear, endocalyx

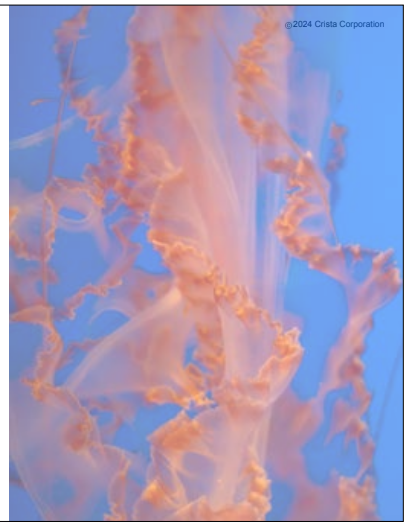
In general, wait to “poke the bear” (dissolve biofilm) until current infectious load is managed.

1st phase — enzymatic + antimicrobials

2nd phase — bisthiol + d/c mineral supps

By the 2nd phase, the infectious load is less the issue and the matrix hosting the last stem cell-based survivors is more the issue. Antimicrobials can usually be d/c’d.

3rd phase — frequency based therapies (Rife, FSM, light, sound) to dissolve the matrix/pattern.



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Mold Denial

Have they talked you into their story of denial?

*This is the most common cause for lack of progress in my peer-to-peer consults.

Use caution with these patients with mold testing that has a high potential for false negative, ie: mold plates.

Use the professionals!

Know when to “fold em”, and walk away.

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Why all the nature pics?

Coherence!

Quantum physics is verifying that everything is energy.

People whose health has been impacted by mold tend to get into coherence with mold’s vibrational energy.

Leads to being a “mold attractor”.

Talk with your patients about this, so they can take steps to move away from mold firstly energetically — to change their frequency (they’re radio dial, if you like) to a higher frequency of coherence.

*This can happen to you dear doctor if you specialize in mold, and aren’t mindfully recalibrating your and your clinical space’s energetics.

I used the nature pics to help you reset your energetics away from mold and toward nature throughout this lecture.

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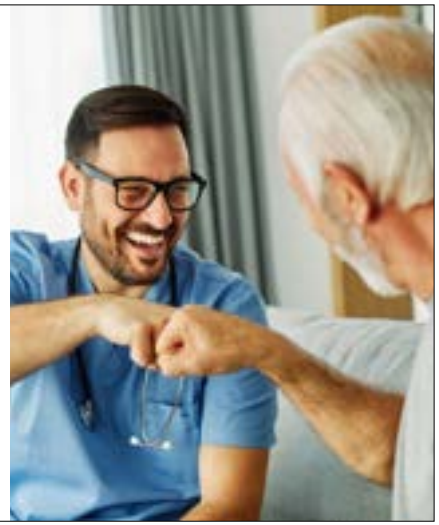
Guided Visualization: A Free Resource

#breakupwithmold

Don't forget laughter!

Comprehensive Plan

Avoidance avoidance avoidance
Diet diet diet
Bioflavonoids
Good fats
Bile movement
Binding
Detox + mitochondrial support
Immune support
Antifungals
Energetics

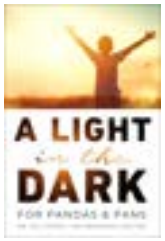


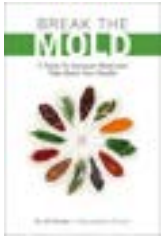
Orange Half Peeled?

Avoiding Avoidance
is the most common
barrier to recovery

must get out of mold to fully heal

Helpful Resources





Greek 

Chinese 

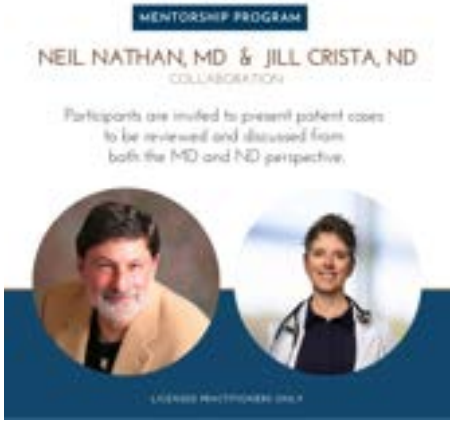
Polish 

German 

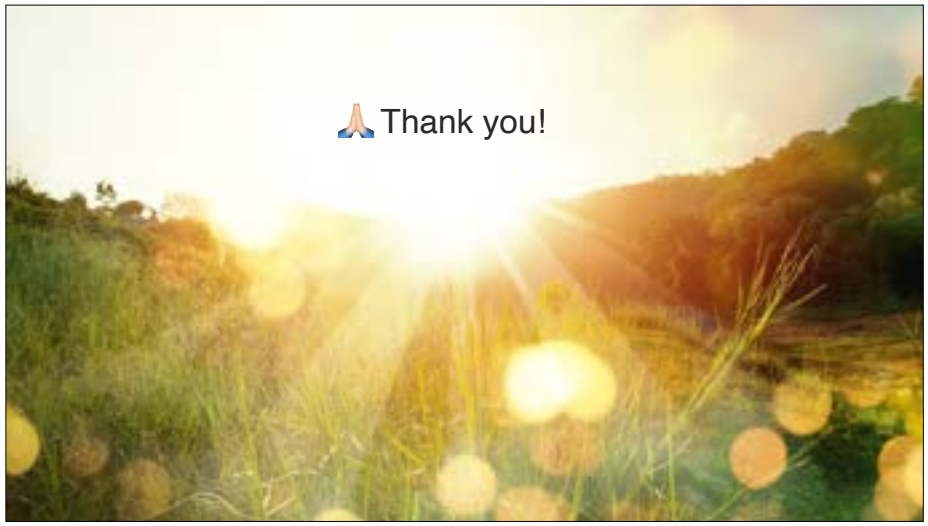
Education

For the public ~

For medical practitioners ~



Prerequisite training via mentorship or my mold certification course.
Enroll in my course or email askdrnathan@gmail.com for details.


🙏Thank you!