Mold, Mycotoxins, and More

Clarifying Some Common Confusions…
And Expanding Our Understanding

12/7/21
Webinar

Agenda

The common clinical dilemma, "How do I make sense of these labs?“

1) Clarifying Confusions

2) Contemporary Considerations ("More")

3) Big picture perspectives

1. Clarifying Confusions

1a. Revisiting Definitions (& correlations w/labs)

1. CIRS:
   - inflammatory condition in a patient
   - reflects symptoms (denies measurement of mechanism)

2. Mycotoxin illness:
   - mechanism of illness
   - specific lab result abnormality

3. "Mold": very loose term
   - patient = symptom? (CIRS); mycotoxin-specific?
   - environment = specific? (mold environment?); general (WDB?)

A New Definition?

CIMI: Chronic Inflammatory Mold & Mycotoxin Illness

CIRS (inflammatory): √
Mycotoxins: √
“Mold”: √

Learning Point #1a:

Esp. when communicating to/with others, be clear with your terminology.

Being mindful of terms will clarify some confusion, both within yourself and between you and others.

1b. Potential Pitfalls: Mycotoxin Lab Differences

1. GPL
   - 11 markers – direct mycotoxin testing
   - Mass Spec + Liquid Chromatography

2. Real Time
   - 15 markers – direct mycotoxin testing
   - ELISA

3. Vibrant America
   - 31 markers – direct mycotoxin testing
   - Mass Spec

4. MyMyco Lab
   - indirect (antibody) testing

(Only 6 common markers between GPL and RT)

Learning Point #1b:

A rather negative set of mycotoxin results by a lab with a smaller set of markers does not necessarily mean the patient has no mycotoxins. (Or for that matter, that the patient does not have CIRS.)

1c. “Mold” (~”Lyme”): where is it coming from?

Source: internal vs. external?

Patient (internal – colonization, or more):
-based on symptoms or serum inflam markers? = CIRS (external source)
-based on mycotoxin? (external source)
-based on **OAT**? = colonization, or perhaps even more actively so (the ONLY internal source)

Environment (external):
-mold-specific WDB?
-more generalized WDB?

Learning Point(s) #1c:

Lack of mold markers on OAT does *not* mean the patient "doesn't have mold" (as defined by mycotoxins, or CIRS) – only that s/he doesn't have internal (gut) mold colonization

Mold markers on OAT does *not* mean the patient absolutely has "mold" (mycotoxins, or CIRS)

OAT mold markers may help explain why some patients continue to have symptoms or mycotoxins despite having remediated their external environment (reduces chasing mold sources?)

2. Contemporary Considerations

Introduction

Peg DiTulio, APRN

Ritchie Shoemaker certified practitioner
Past Treasurer for ISEAI
Practices in southern NH

2a. How does the GENIE fit in?

CIRS vs. Immunoreactivity

Gene Expression: Inflammation Explained

Uses differential gene activation to identify underlying complex physiology in these chronically ill patients.

“FAB”

Fungi (AKA Mold)

Actinomycetes
(A filamentous Gram-Positive Bacteria)

Bacterial Endotoxins (Lipid Molecules From The Cell Wall Of Gram-Negative Bacteria)

Fungi (AKA Mold)

Mold, Conidia, Hyphal Fragments, Spirocyclic Drimanes, Mannans

Actinobacteria

Anaerobic Gram-positive bacteria that normally colonize subdermal extracellular vesicles.

Bacterial Endotoxin

Endotoxins are complex lipopolysaccharides (LPS) which form an inherent fraction of the outer cell wall of all gram-negative bacteria and are responsible for the organization and stability of the cell wall (Kim et al., 2012).

Chronic Inflammatory Response Syndrome (CIRS) is basically an inflammatory response to the DNA of different pathogens: FAB, Tick Borne Diseases, Pfiesteria, Ciguatera, COVID and others.

Immunoreactivity

No proteomic markers yet

On GENIE testing you see regulatory changes in T cell synapses, Tubulins, T Regulatory deficiencies, MAPKS and Toll Receptors

Cytokines

The most significant for immunoreactivity are TGFBR1, 2, 3

TGFB1 and TGFBR1, 2, 3 are markers for specific exposure to Actinomycetes along with MAPK.

Breakdown of immune activators in water damaged buildings

(Based on over 1,000 GENIE reports)

Actinomycetes – 42%
Fungi/Mycotoxins – 7-10%
Endotoxins – 28%

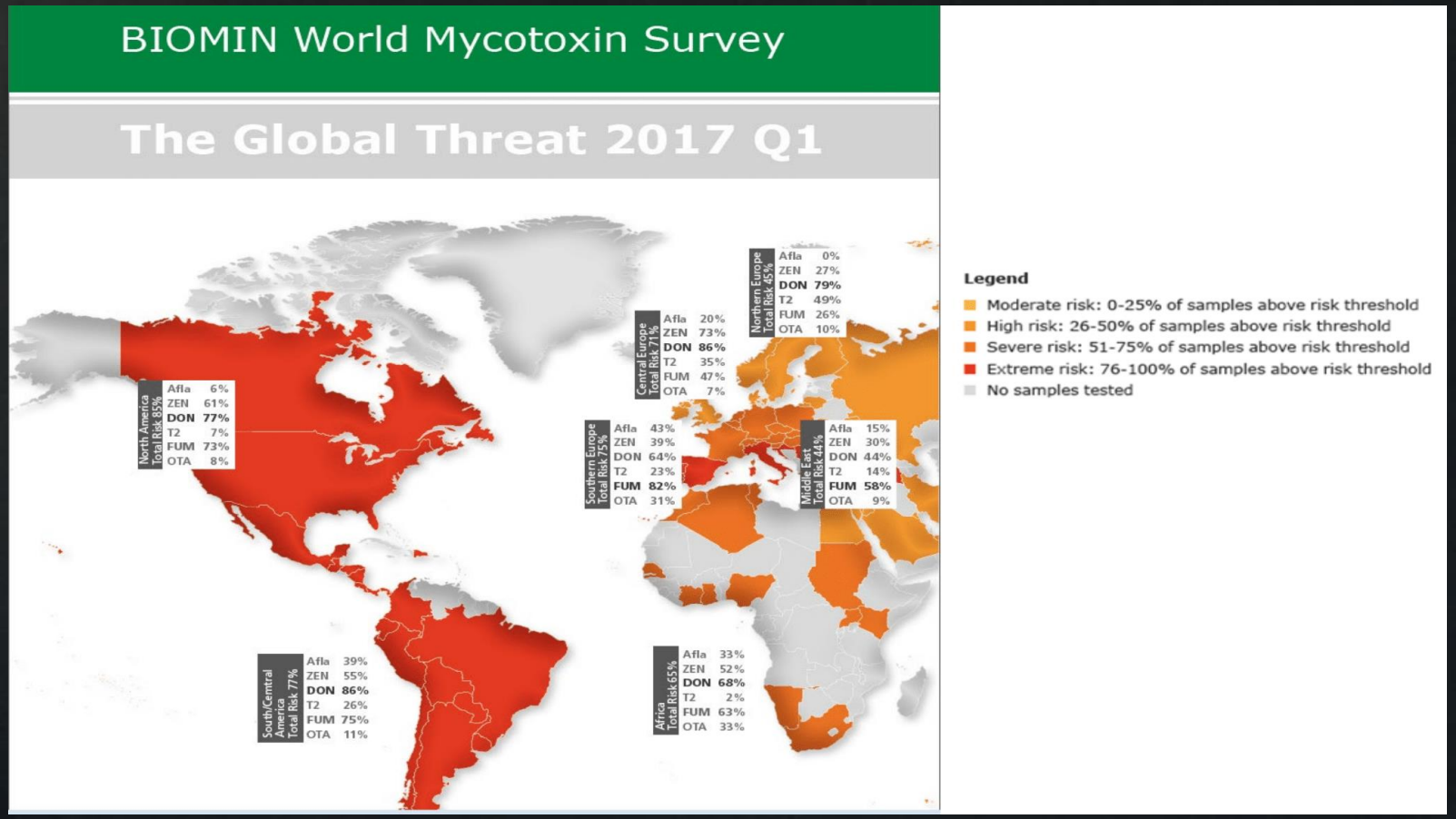
(Some GENIE report did not show immune activation)

GENIE is a gene expression assay composed of 188 genes.

The most important question that GENIE will answer is:

whether or not molecular hypometabolism or proliferative physiology are present

2b. Revisiting Mold in Foods

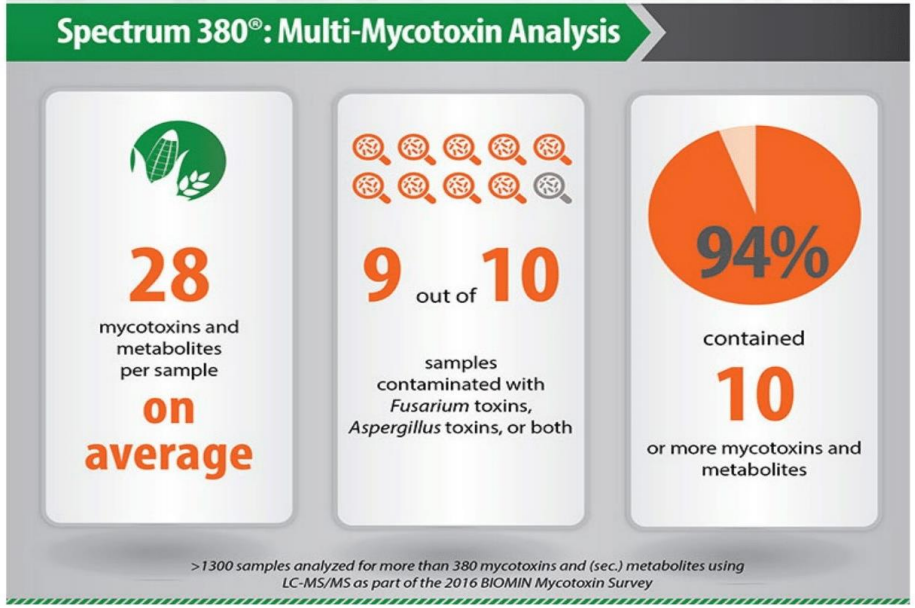


2b. Revisiting Mold in Foods

Risk Level:
The risk level expresses the percentage of samples testing positive for at least one mycotoxin above the threshold level.

Threshold levels in ppb:

Afla	ZEN	DON	T-2	FUM	OTA
2	50	150	50	500	10



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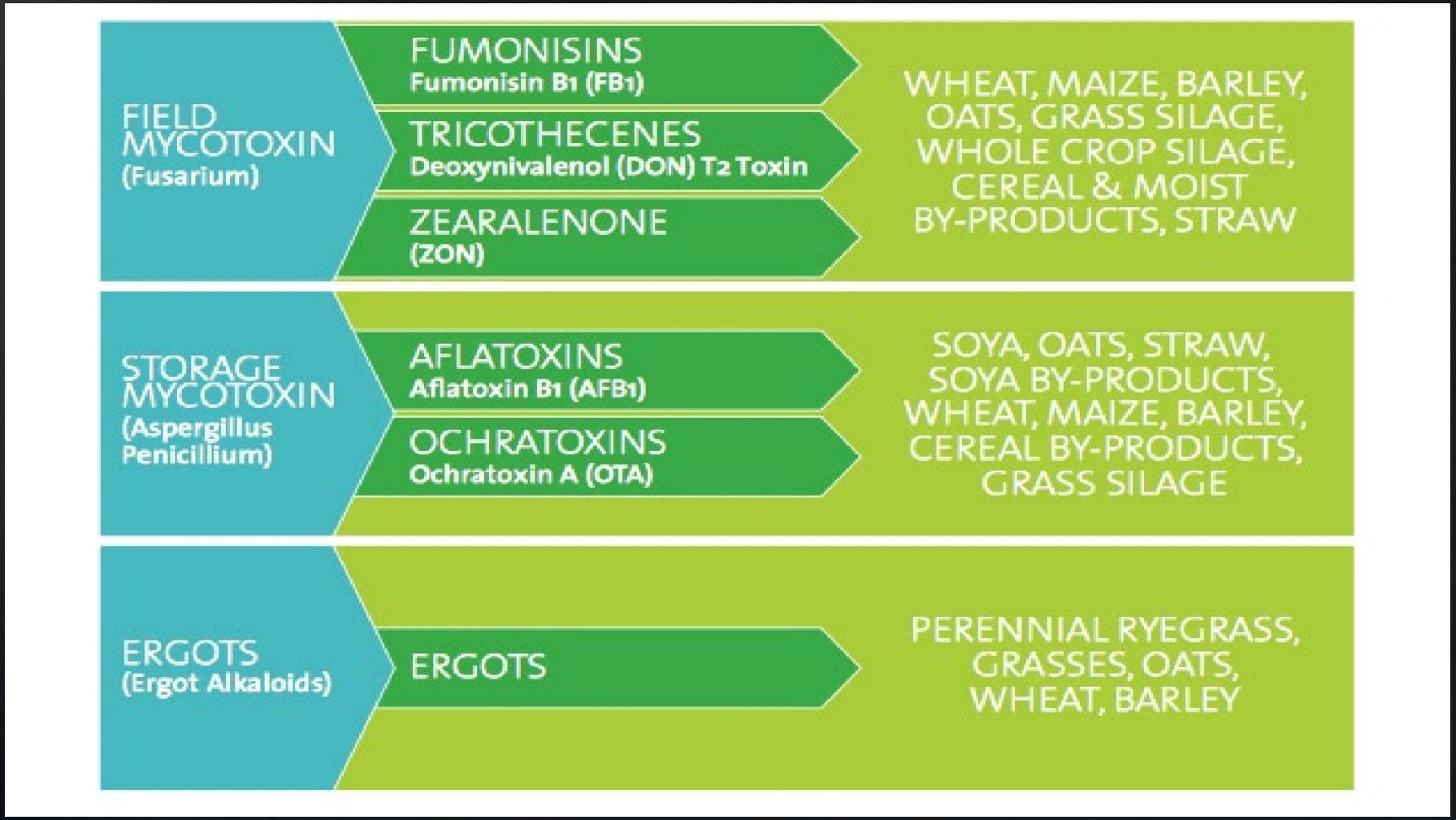


2c. A new (pathogenic) kid on the block?

Parasites as a reservoir?
(an internal source of mold and/or mycotoxins?)

Learning Point(s) #2a-c:

GENIE testing has much to offer, in certain patients; be clear on your goal in testing

Consider that foods may lead to mycotoxin levels in testing, but likely at meaningfully low levels (tbd?)

Consider parasites as a possible internal source of mold, mycotoxins (tbd?)

3. Big Picture Perspectives

3a. Passe: Silo Medicine

Conventional medicine silos:

-formally specialist/specialty-based

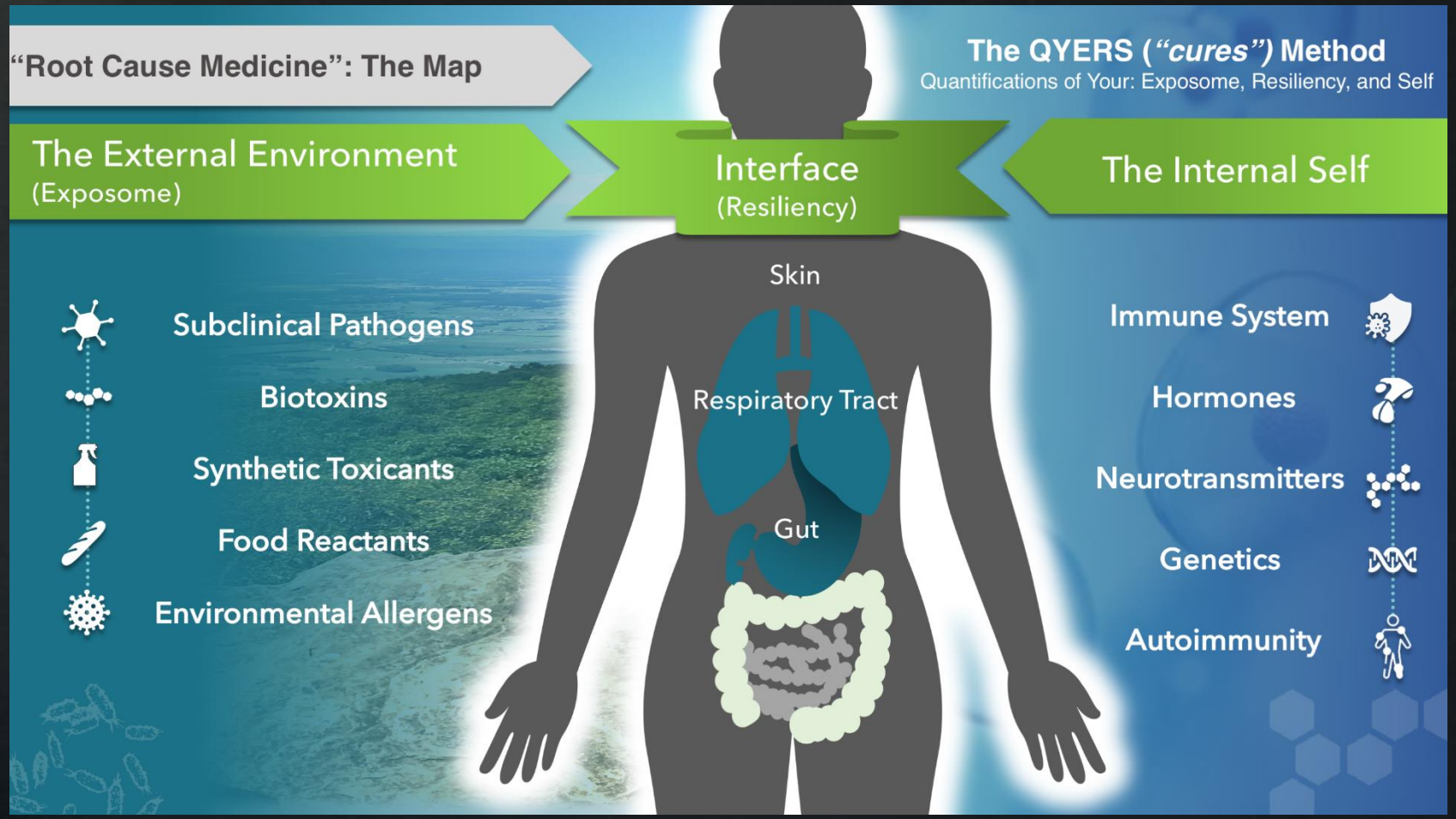
-eg, cardiology, endocrinology, gastroenterology, etc.

Integrative medicine silos:

-informally condition/system-based

-eg, focuses/interests on gut health, hormone health, “Lyme disease”, “mold”, “food allergies”, etc

3b. Multi-Dimensional Medicine



The QYERS Map

3b. Multi-Dimensional Medicine

The QYERS (*"cures"*) Method
Quantifications of Your: Exposome, Resiliency, and Self

The External Environment (Exposome)

Subclinical Pathogens
Tickborne pathogens
Viruses
Yeast
Mold
Parasites
Other bacteria
(gut, sinuses, oral)

Synthetic Toxicants
Heavy metals
EMFs
POPs
Pesticides

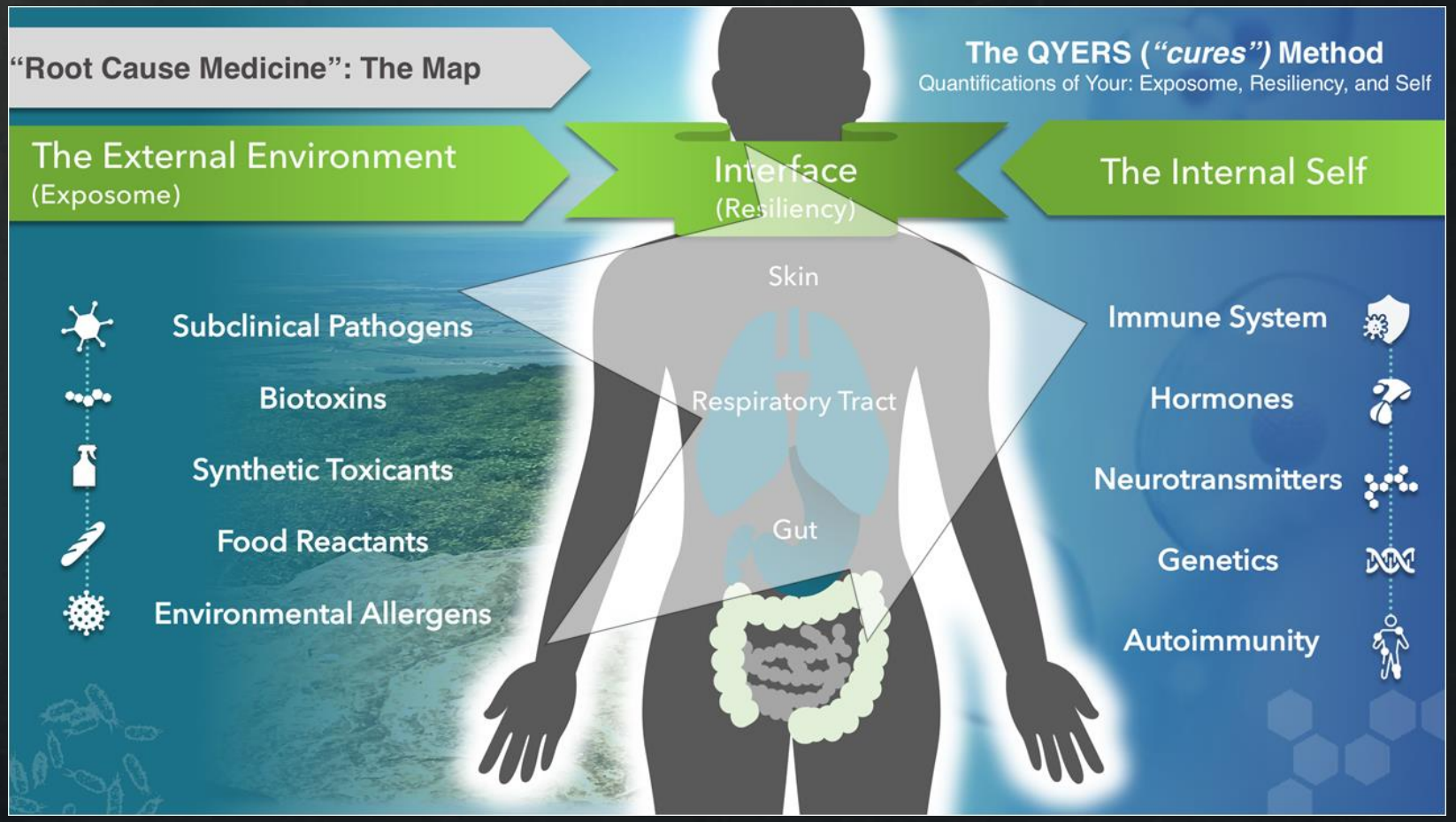
Biotoxins
Mold toxins
Cyanotoxins
Neurotoxins

Food Reactants

Environmental Allergens

Exposome: "Bugs and Toxins"

3b. Multi-Dimensional Medicine



QYERS Map

3c. Critical Analysis, Critical Thinking

Bias awareness
Objectivity
Hypothesis → Test → Interpret
Adjust
Repeat

Learning Point(s) #3c:

Testing, not guessing… there's a lot of data that can be gathered, even from past data

Think beyond lab and diagnostic silos (often created by unintentional biases)

Think in terms of multi-factorial ("multi-dimensional") perspectives

Case 1

71yo female, cerebellar degeneration - "idiopathic": vit E, gluten, B12, genetic evaluations normal.

Home: basement flood many years ago. No visible mold growth, dehumidifier (+). Not interested in environmental testing except agar plates, which was (-). New car; no meaningful time spent elsewhere.

Patient: no GI symptoms, no chronic/recurrent sinus symptoms.

Aug 2020 GPL MycoTox: multiple mycotoxins/mold species (+)

Treatment: 4 caps activated charcoal morning only (declined to take more often), with NAC, resveratrol, and some initial oral GSH and liver/GB support, fish oil, vits, minerals, ALA, antioxidants, and Silvercillin (broad spectrum antimicrobial).

June 2021 GPL MycoTox: meaningfully reduced Ochratoxin A, and all others completely zero

The question:

"Has anybody seen results shift like this with a similar situation? **I'm trying to make sense of this** - do we no longer suspect the home is a primary issue and these were stored from previous decades working in potentially moldy buildings? Could the results be unreliable?"

"Despite the seemingly improved toxin levels, her condition has continued to slowly decline. Funds are tight for them so I want to be very intentional on next best steps."

(→ *Diagnosis at this point, based on data?*)

My mindset:

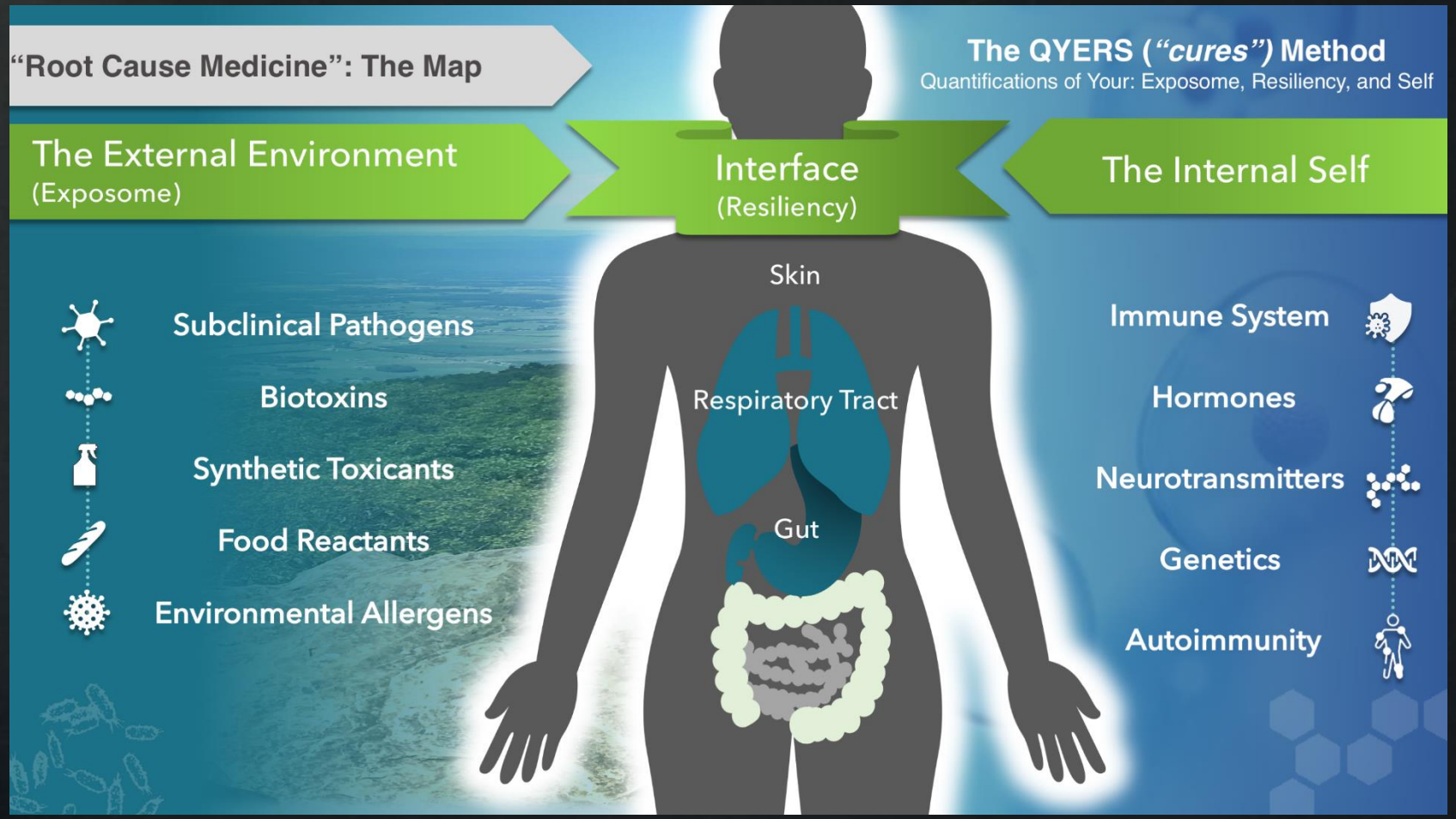
-hypothesis, run tests to support/refute, adjust hypothesis accordingly

-let the results form the clinical explanation; don't force the results to "fit" the (assumed) clinical explanation

-think broadly beyond mycotoxins (esp. if no evidence of such)

-no direct cause-effect measure, so perhaps indeed, mycotoxins helped her – just not enough to symptomatically manifest with reduced symptoms… but other matters need to be looked at as well (QYERS Map)

3b. Multi-Dimensional Medicine



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Exposome: "Bugs and Toxins"

Case 2

My (32yo) son who was dx with schizophrenia / drug induced psychosis / depression in 2006 was just dx with mold / mycotoxin illness recently.

Questions:
-what do some of these markers mean, specifically?
-do you order the same markers? and if not, what do you order
-I think many practitioners struggle to know what to order and what to do with it all once the results are received.

HLA DRB1/DQ/DRB345	7-2-53
MSH	<8
MMP9	782
TGF-beta1	6560
C4a	24892
C3a	54
VEGF	<31
VIP	<50
Leptin	58.2
ADH	<1.8
Osmolality	283
ACTH	17.6
Free Cortisol	
Testosterone Free/Total	
DHEA-S	
Estradiol, Free	
Progesterone, Free	
Aldosterone	
Pregnenolone	<10
AntiGliadin Aby	-
AntiCardiolipin Aby	+
Lyme Western Blot	-
Von Willebrands Profile	-
Nasal Culture	
CD4+CD25+	
CBC	↑Hgb, ↓Plts
CMP	↑ALT
GGT	95
Uric Acid	5.7
Hgb A1c	
Lipase	
Ferritin/Iron total	100
Vitamin D3 (25-OH)	
RBC Mg	5.5
RBC Zinc	1195
Thyroglobulin	
Free T4	1.06
Free T3	3.9
TPO	9
AntiThyroid Aby	-
B12	437
RBC Folate	871
Homocysteine	15.1
CRP	
Heavy metals	↑Cd

Surviving Mold NeuroQuant© Analysis

Date: 07/28/2021

Age of Patient: 32

Atrophic Nuclei: Pallidum

Lateral Ventricle: Normal

Mold Score	Lyme Score	Asymmetry Found
5	0	Yes

A Mold Score of 5 is consistent with CIRS-WDB. A Lyme Score of 0 is not consistent with CIRS-PLS.

Submitted Information:

Brain Structure	LH Volume (% of ICV)	RH Volume (% of ICV)	Asymmetry Index (%)
Forebrain Parenchyma	35.32	34.82	1.43
Cortical Gray Matter	17.77	17.40	2.1
Lateral Ventricle	0.68	0.64	6.06
Hippocampus	0.28	0.32	-13.33
Amygdala	0.13	0.08	47.62

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Case 3

I have an “opinionated” 3 yo (mom’s description) who has
TGF B1 in 13,000
VEGF is elevated in the 100’s
c4a is normal

He has significant eczema and food sensitivity. Would this elevate his TGFB1 ?

Mycotoxin screen is ordered.
I would love a discussion on kids/ toddlers and what products are good to use as well as dosage.

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(Additionally in this case: FAB?)

Case 4

22yo female with 2yrs of knee pain, depression, disordered eating.
6mos empirical Lyme treatment, no help.
h/o mold exposure in home (kitchen flooded) but hasn't been there for 3yrs.
→ OAT shows "no mold".
Question: isn’t it shocking to have such a clean test?

Back to definitions:
-no "mold markers" on OAT does not mean she doesn't have illness d/t mold exposure.
-also, focal symptoms less likely to be obvious for CIMI or Lyme?

Case 5

Do you have suggestions for attics that have mold?

Years ago I had it sprayed with enzymes that kill mold.

Then had another inspector say there was still mold aspergillosis and it should treated with cryotherapy.

Then another inspector say because there’s no connection to the house - just leave it because the air quality was good in the home.

Case 6

In 2007, bought a rotting, moldy, rodent-infested home. Cat urine destroyed an oak floor; bats nested behind paneling in master bedroom that only had insulation b/w room and outside siding. One side of roof looked like a barnacle-ladened ship; even more blackened similar appearance on the underside in the attic. Tearing up bathrooms, found moldy plywood under showers and window lentils in the basement from leaking tubs. And, just outside, was moist, damp, never-dried earth, plus leaves.

Over time, renovation led to house becoming more sealed.

Petri dish testing: aspergillus, penicillium, and yeast.

Individual: headaches, numbness in my left leg and arm, gunk in my throat, pain in the left lobe of "my lung", a protrusion of the right clavicle by the sternum, brain fog, emotional instability (sometimes spontaneous sobbing when going into basement), heightened sense of smell, hair and iris color losing pigment.

Case 6

Moved out in 2019. Improved tremendously but still very sensitive - easily triggered, eg church - if present for over an hour, symptoms last for 36hrs, esp. HA behind R eye and "brain fog".

MycoTox: elevated levels of Ochratoxin A, Mycophenolic acid and Citrinin.
OAT: Fusarium and Candida.

The question is, why is no one else reacting as I do? I can detect mold or toxic air immediately.
Could it be: Toxin Induced Low Tolerance (TILT)? Genetics?

Recent results: HLA-DR/DQ Risk Assessment for mold hypersensitivity (-), heterozygous for the MTHFR genes (C677T C/T and A1298C C/A), homozygous for COMT A/A.

How might the COMT mutations, specifically, and perhaps other genetic mutations, make people like me the Canary in the mine?

Closing Announcements

1) NEIA - limited # of participants
2) Case Rounds program - limited # of participants
3) FMCG - first come, first serve; options for learning/training, being part of the clinical team, and/or just being a practitioner within our referral network for patients

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