Casual Friday Series

Unlocking the Cognitive Decline Code – Part 3

BIOGENETIX.COM

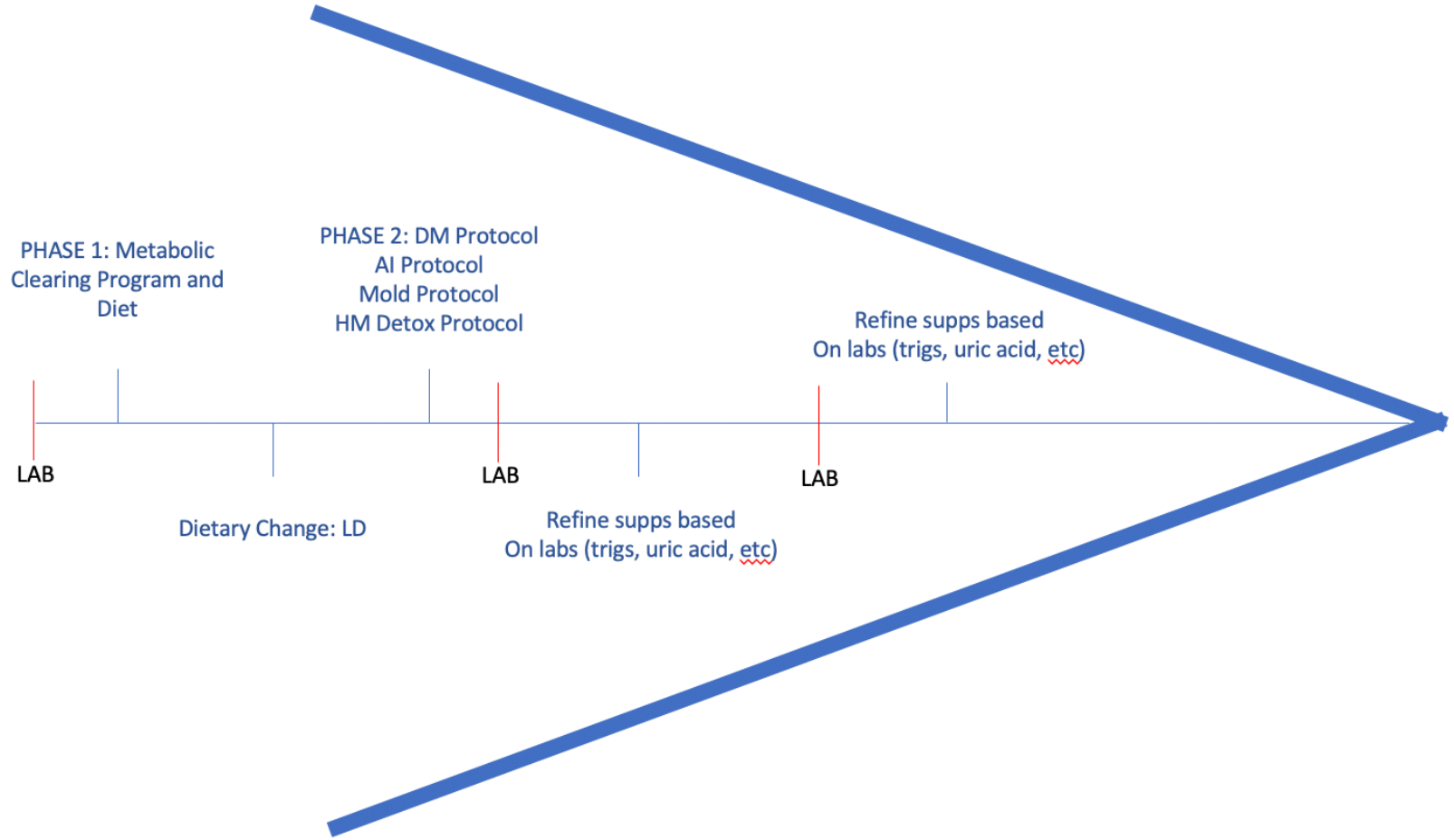
Disclaimer

- *Information in this presentation is not intended, in itself, to diagnose, treat, reverse, cure, or prevent any disease. While this presentation is based on medical literature, findings, and text, The following statements have not been evaluated by the FDA.*
- *The information provided in this presentation is for your consideration only as a practicing health care provider. Ultimately you are responsible for exercising professional judgment in the care of your own patients.*

(Lifestyle + Genetics) x Time = Chronic Health Condition

(Lifestyle + Genetics) x Time = Chronic Health IMPROVEMENT

Supplement and Diet Protocols



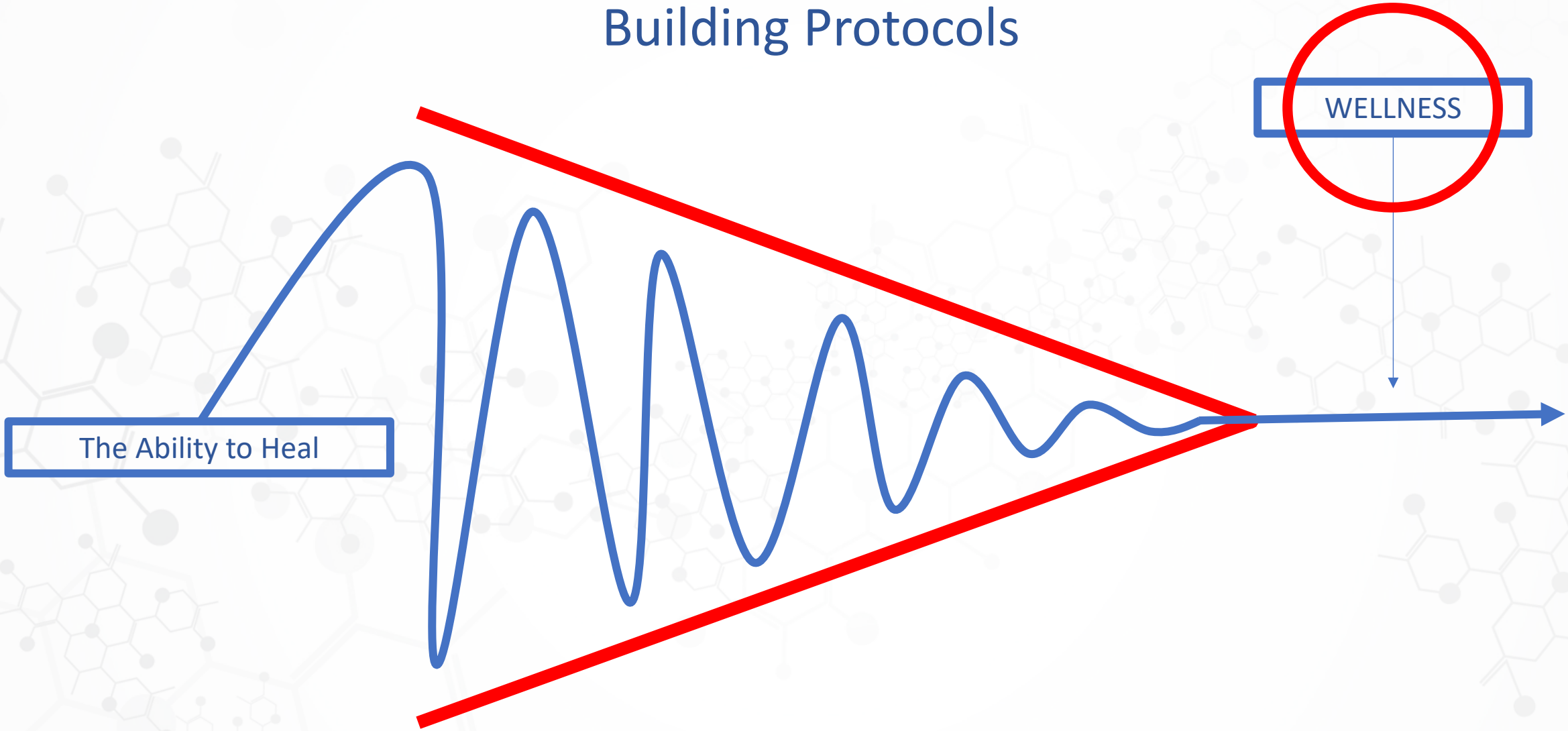
Retest a lab at least every 60 days.

85% of patients will improve with basic structures and healthy eating.

% of problem analysis: this is what the cleanse is for.

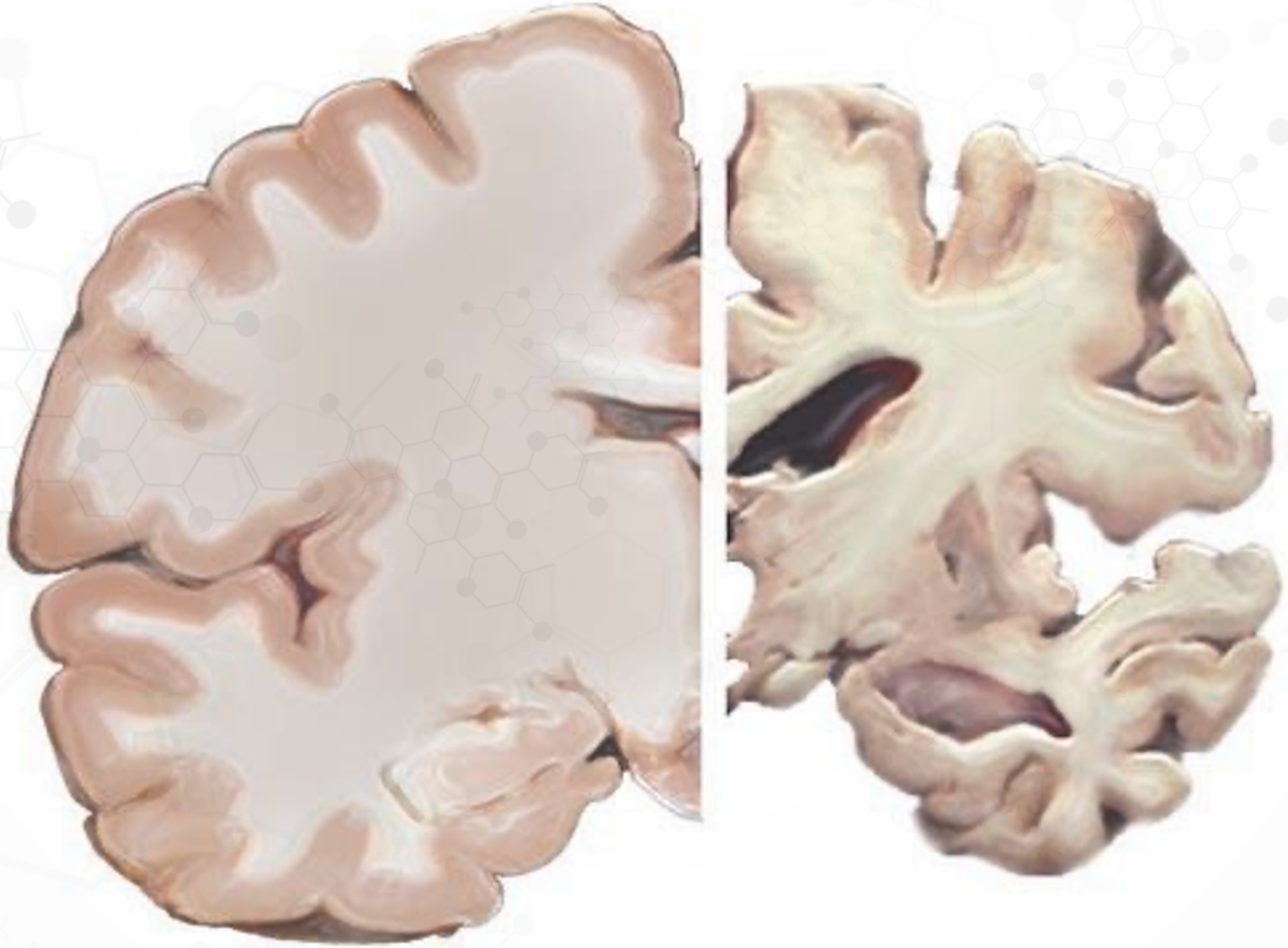
General → Fine Tune

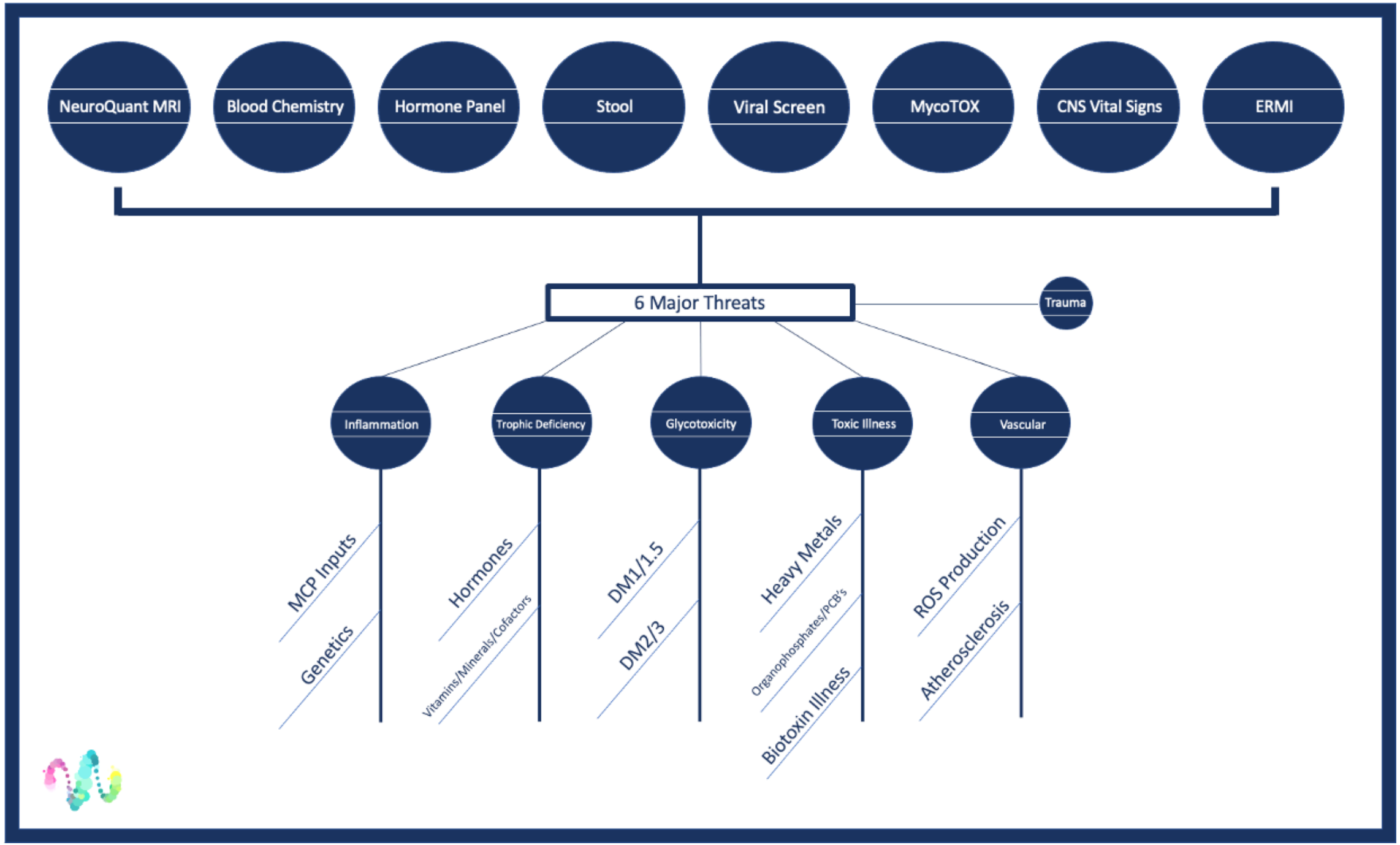
Building Protocols



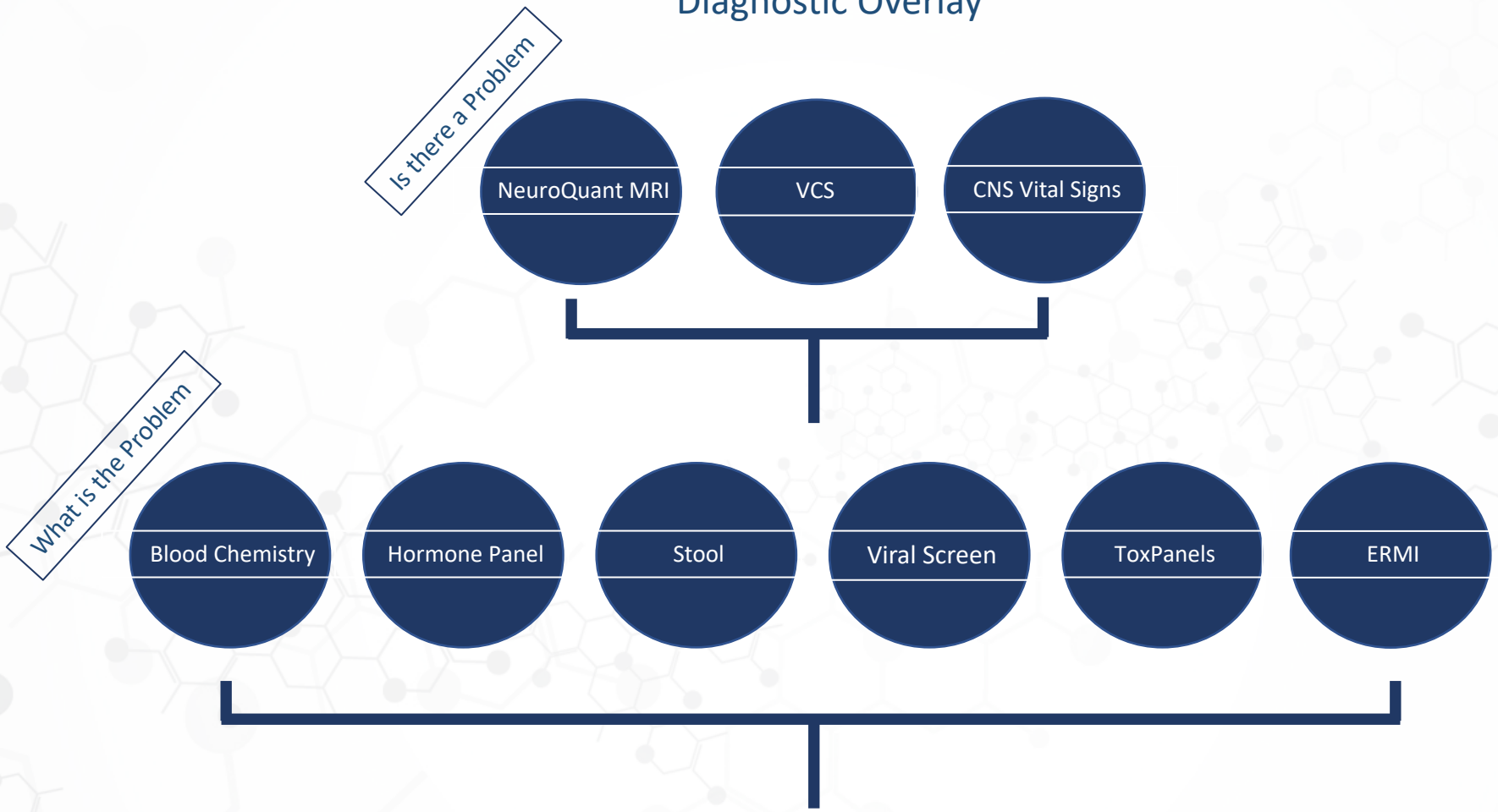
Healthy Brain

Severe AD

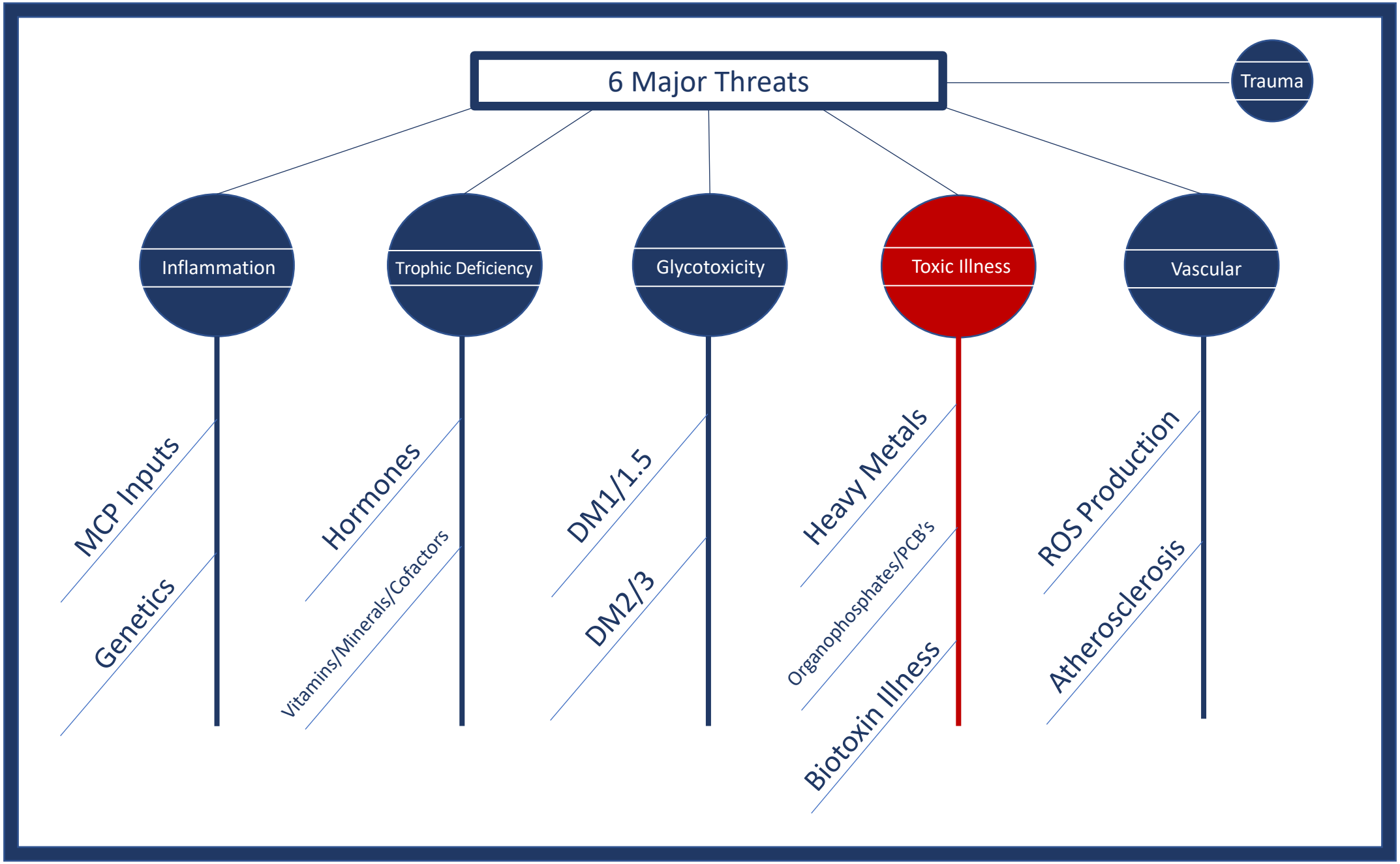



NeuroQuant MRI
Blood Chemistry
Hormone Panel
Stool
Viral Screen
MycoTOX
CNS Vital Signs
ERMI
6 Major Threats
Trauma
Inflammation
Trophic Deficiency
Glycotoxicity
Toxic Illness
Vascular
MCP Inputs
Genetics
Hormones
Vitamins/Minerals/Cofactors
DM1/1.5
DM2/3
Heavy Metals
Organophosphates/PCB's
Biotoxin Illness
ROS Production
Atherosclerosis

Diagnostic Overlay



What combination of the 6 threats (and their subcategories) are we dealing with?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PMID: 26870879

Inhalational Alzheimer's disease: an unrecognized—and treatable—epidemic

Dale E. Bredesen[1,2]

Alzheimer's disease is one of the most significant healthcare problems today, with a dire need for effective treatment. Identifying subtypes of Alzheimer's disease may aid in the development of therapeutics, and recently three different subtypes have been described: type 1 (inflammatory), type 2 (non-inflammatory or atrophic), and type 3 (cortical). Here I report that type 3 Alzheimer's disease is the result of exposure to specific toxins, and is most commonly inhalational (IAD), a phenotypic manifestation of chronic inflammatory response syndrome (CIRS), due to biotoxins such as mycotoxins. The appropriate recognition of IAD as a potentially important pathogenetic condition in patients with cognitive decline offers the opportunity for successful treatment of a large number of patients whose current prognoses, in the absence of accurate diagnosis, are grave.

Table 2

Symptoms, signs, and laboratory values suggestive of type 3 Alzheimer's disease

Characteristic	Comment
Age at symptom onset less than 65 years.	Symptoms often begin in the 50s or late 40s.
ApoE ε4-negative genotype.	Typically ApoE3/3 unless there are other risk factors.
Negative family history or family history positive with symptom onset only in much older individuals than the patient.	
Symptom onset in association with menopause or andropause.	
Depression as a preceding or significant accompaniment of the cognitive decline.	
Headache as an early or preceding symptom.	

Atypical presentation, in which memory consolidation is not the initial and dominant characteristic.	Typical deficits include executive deficits, dyscalculia, paraphasias, or aphasia.
Precipitation or exacerbation by a period of great stress (e.g., loss of employment or marriage dissolution or family change) and sleep loss.	The degree of dysfunction is also markedly affected by stress and sleep loss.
Exposure to mycotoxins or metals (e.g., inorganic mercury via amalgams, or organic mercury via the consumption of large fish such as tuna) or both.	
Diagnosis of CIRS with cognitive decline.	Cognitive decline is common with CIRS.
Imaging suggestive of more than typical Alzheimer's involvement.	FDG-PET may show frontal as well as temporoparietal reductions in glucose utilization, even early in the course of the illness; MRI may show generalized cerebral and cerebellar atrophy, especially with mild FLAIR (fluid-attenuated inversion recovery) hyperintensity.
Low serum triglycerides or triglyceride:total	Triglycerides are often in the 50s.

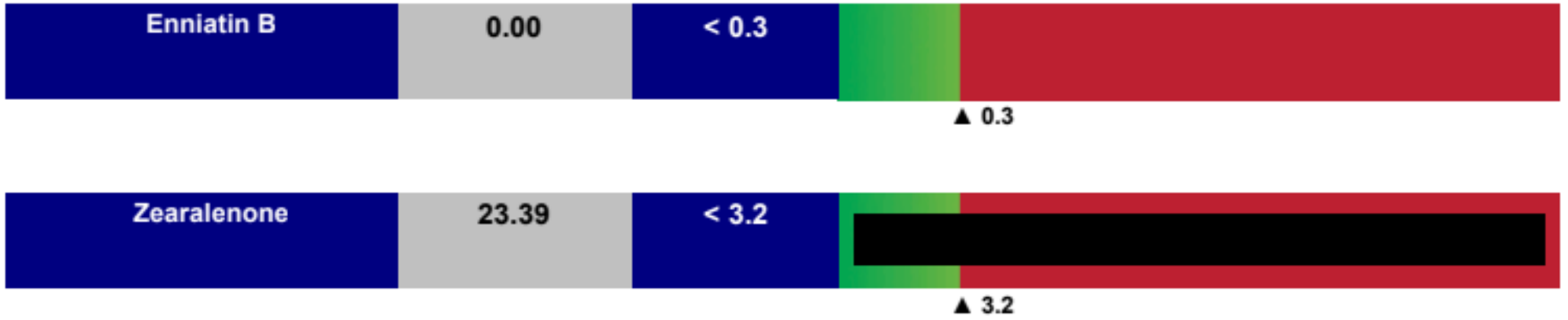
Low serum triglycerides or triglyceride:total cholesterol ratio.	Triglycerides are often in the 50s.
Low serum zinc (<75mcg/dl) or RBC zinc, or high copper:zinc ratio (>1.3).	
HPA axis dysfunction, with low pregnenolone, DHEA-S, and/or AM cortisol.	
High serum C4a, TGF-β1, or MMP9; or low serum MSH (melanocyte-stimulating hormone). Positive deep naso-pharyngeal culture for MARCoNS.	See reference 5.
HLA-DR/DQ associated with multiple biotoxin sensitivities or pathogen-specific sensitivity.	See reference 5.

Mycotox Profile

Creatinine Value: 91.64 mg/dl

Metabolite	Results (ng/g creatinine)	Normal Range *	Abnormal Range
Aspergillus			
Aflatoxin-M1	0.00	< 0.5	▲ 0.5
Ochratoxin A	15.73	< 7.5	▲ 7.5
Gliotoxin	0.00	< 200	▲ 200

Fusarium



Multiple Mold Species

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

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

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

Group 1; Water Damage Molds

Species	SE/mg	
Aspergillus flavus/oryzae	65	*
Aspergillus fumigatus	14	
Aspergillus niger	170	*
Aspergillus ochraceus	1,489	* *
Aspergillus penicillioides	2,677	*
Aspergillus restrictus	678	* *
Aspergillus sclerotiorum	11	
Aspergillus sydowii	345	* *
Aspergillus unguis	1,346	* *
Aspergillus versicolor	984	* *
Aureobasidium pullulans	2,160	
Chaetomium globosum	4,980	* * *
Cladosporium sphaerospermum	421	*
Eurotium (Asp.) amstelodami	16,721	* *
Paecilomyces variotii	3	
Penicillium brevicompactum	1,494	* *
Penicillium corylophilum	435	* *
Penicillium crustosum	40	*
Penicillium purpurogenum	10	
Penicillium Spinulosum	22	*
Penicillium variabile	7	
Scopulariopsis brevicaulis/fusca	N D	
Scopulariopsis chartarum	13	
Stachybotrys chartarum	51	*
Trichoderma viride	51	*
Wallemia sebi	885	*

Sum of Logs	56.8

Group 2; Common Indoor Molds

Species	SE/mg	
Alternaria alternata	30	
Acremonium strictum	4	
Aspergillus ustus	17	
Cladosporium cladosporioides1	898	
Cladosporium cladosporioides2	227	*
Cladosporium herbarum	7	
Epicoccum nigrum	13,580	* *
Mucor amphibiorum	87	
Penicillium chrysogenum	169	*
Rhizopus stolonifer	N D	

Sum of Logs	17.7

SE = Spore Equivalents
SE/mg = SE/milligrams of sample
Logs = Logarithms
N D = None Detected

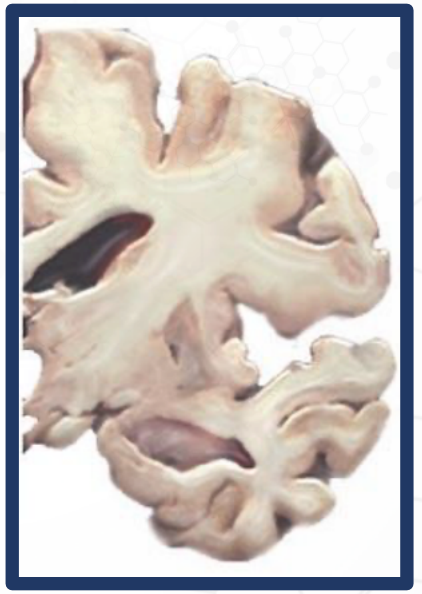
Sample Size	4.9	mg
ERMI Results= (G1-G2)	**39.1**	

(*) 10 fold higher than normal.
(**) 100 fold higher than normal.
(***) 1,000 fold higher than normal.

What's the net result? → CIRS

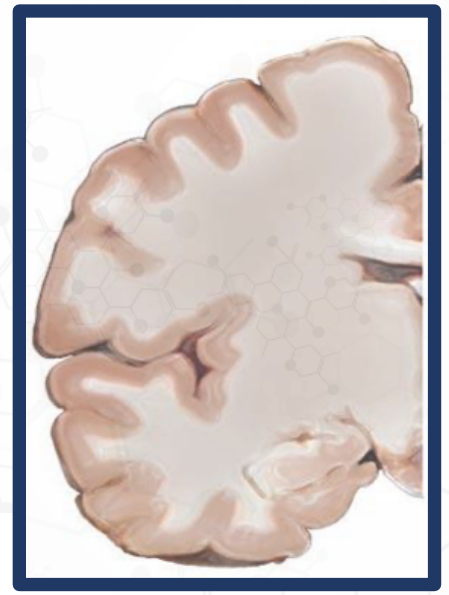
https://www.survivingmold.com/docs/April_V_CIRS_2020_216.pdf

Biotoxin Exposure → Anti-Trophic Signaling →



(→ = Promotes)

Trophic Signaling



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The Biogenetix Brain Box

Biogenetix: 833-525-0001

zeb@biogenetix.com

kim@biogenetix.com