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NUTRITIONAL PATHOLOGY INSTITUTE

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Navigating the Doctor's Data GI Assessment Menu

A Clinician's Guide to Stool Test Interpretation & Clinical Application

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Nutritional Pathology Institute | Harmony Healing Center

Learning Objectives

1 Navigate the DD GI test menu

Understand when to use GI 360, CSA+P, CSA, and Microbiology Only. Which are appropriate for initial vs follow-up. Understand the differences in methodology used in each test

2 Interpret microbiome and microbiology data

Apply the Dysbiosis Index and Diversity Score within clinical limitations. Interpret the dysbiosis index, commensal microbiome analysis, and antimicrobial susceptibility results to guide treatment

3 Apply Clinical Relevance Principles

Understand when a finding is clinically actionable to avoid unnecessary treatment

4 Evaluate stool chemistries

Integrate inflammatory, digestive, and immune markers for clinical decisions

5 Detect and manage C. difficile

Apply current research to carrier detection and treatment strategies

6 Apply to clinical practice

Walk through a comprehensive case study using the full DD assessment

Why Comprehensive GI Assessment Matters

The Gut as a Clinical Hub

- 70–80% of immune tissue resides in the GALT
- 95% of serotonin produced in the GI tract
- Gut dysbiosis linked to autoimmune, neurological, and metabolic disease
- Intestinal permeability drives systemic inflammation cascades
- Microbiome directly influences epigenetic expression
- SCFAs regulate HPA axis, barrier integrity, and neuroinflammation
- GI pathology often precedes and drives systemic presentation

45+

Targeted analytes

in the GI 360 microbiome profile

14

PCR pathogen targets

bacteria, viruses, parasites, worms etc.

1,600+

Species identifiable

via culture + MALDI-TOF

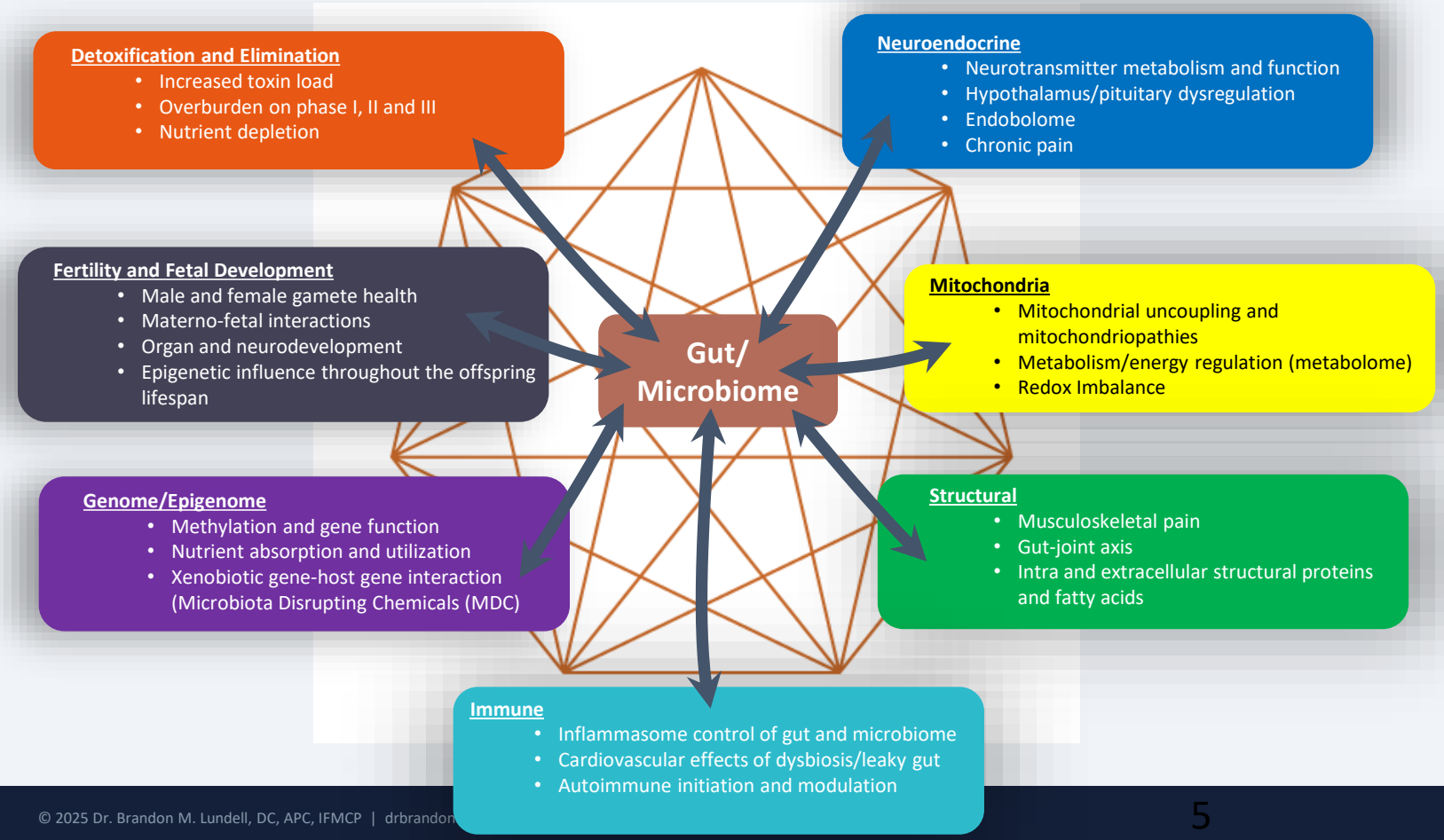
31+

Parasites screened

via 3-day microscopy

The Gut/Microbiome-Human Health Matrix

Gut-Human Health Matrix



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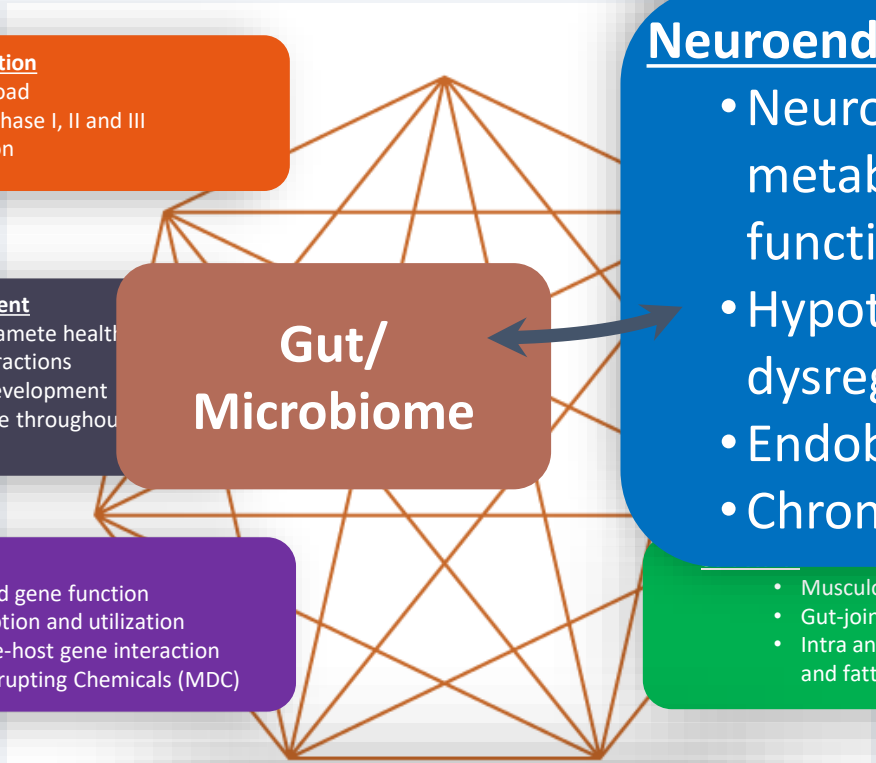
Gut-Human Health Matrix



Detoxification and Elimination
- Increased toxin load
- Overburden on phase I, II and III
- Nutrient depletion

Fertility and Fetal Development
- Male and female gamete healt
- Materno-fetal interactions
- Organ and neurodevelopment
- Epigenetic influence throughou lifespan

Genome/Epigenome
- Methylation and gene function
- Nutrient absorption and utilization
- Xenobiotic gene-host gene interaction (Microbiota Disrupting Chemicals (MDC)

Gut/ Microbiome

Neuroendocrine
- Neurotransmitter metabolism and function
- Hypothalamus/pituitary dysregulation
- Endobolome
- Chronic pain

- Musculoskeletal pain
- Gut-joint axis
- Intra and extracellular structural proteins and fatty acids

Immune
- Inflammasome control of gut and microbiome
- Cardiovascular effects of dysbiosis/leaky gut
- Autoimmune initiation and modulation

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Gut-Neuroendocrine Connection

Neurotransmitter Metabolism

- The microbiome directly influences serotonin, dopamine, and GABA production and metabolism

HPA Axis Dysregulation

- Gut dysbiosis drives hypothalamic-pituitary-adrenal axis dysfunction and chronic stress response

Chronic Pain & Gliopathy

- Activation of glial cells and neuro-glial interactions are key mechanisms underlying chronic pain — chronic pain could be a result of "gliopathy"

Glutamate Signaling

- Glutamate accumulation is a key factor in brain fatigue, poor decision making and "brain fog" — microbiome manipulation shows therapeutic promise

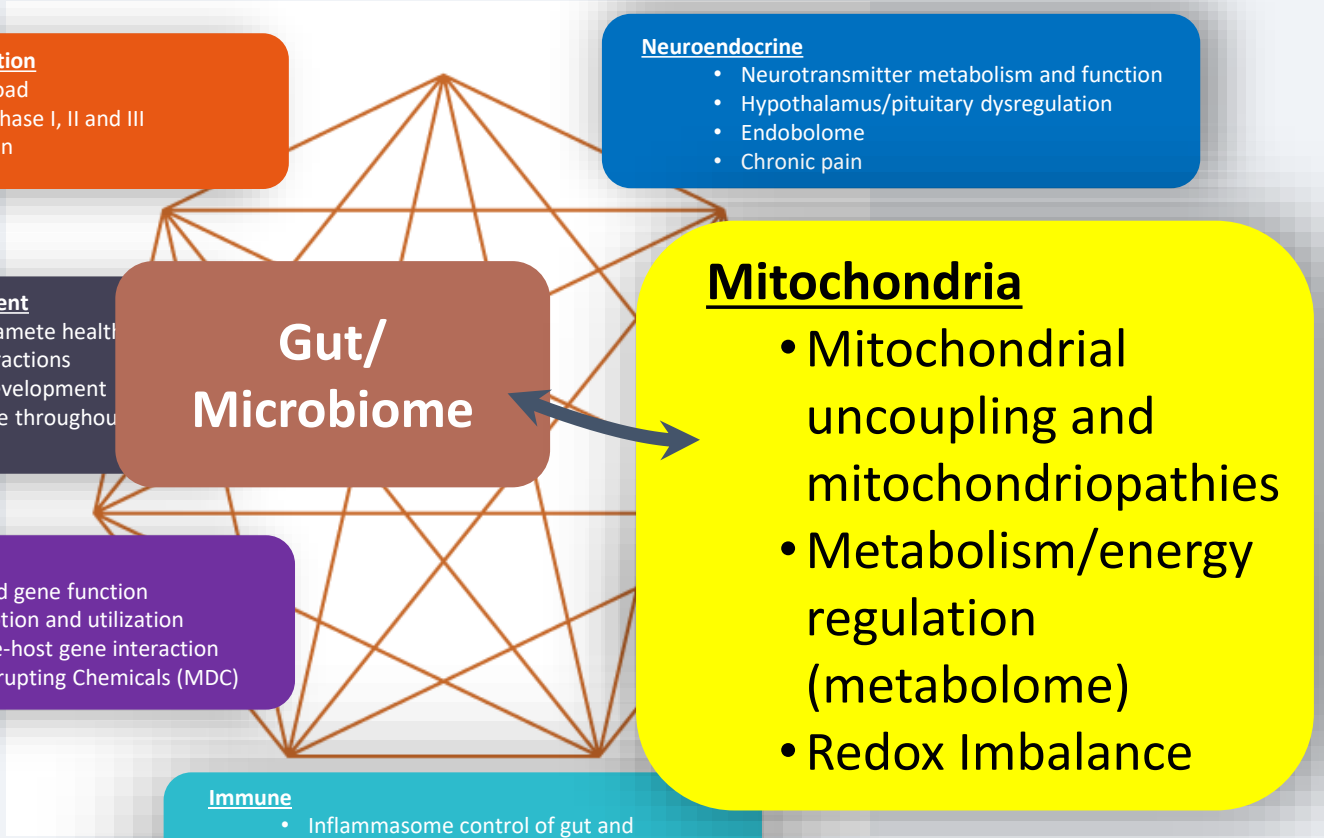
Endobolome

The collective set of gut microbial genes and pathways capable of metabolizing hormones — a critical but often overlooked axis in clinical practice

Clinical Pearl: NAC

- NAC attenuates neuropathic pain by suppressing MMP-9/2
- Induces neuroplasticity via glutamate modulation
- Normalizes spinal cord oxidative status

Gut-Human Health Matrix



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Gut-Mitochondria Connection

"Without mitochondria there is no LIFE. Without fully functioning mitochondria there is no VITALITY."

Mitochondrial Uncoupling

- Gut microbes affect ROS levels, mitochondrial homeostasis, and host health through bidirectional interactions

Metabolome & Energy Regulation

- Mitochondria perform multiple essential functions beyond energy production, impacting most areas of cell biology and medicine

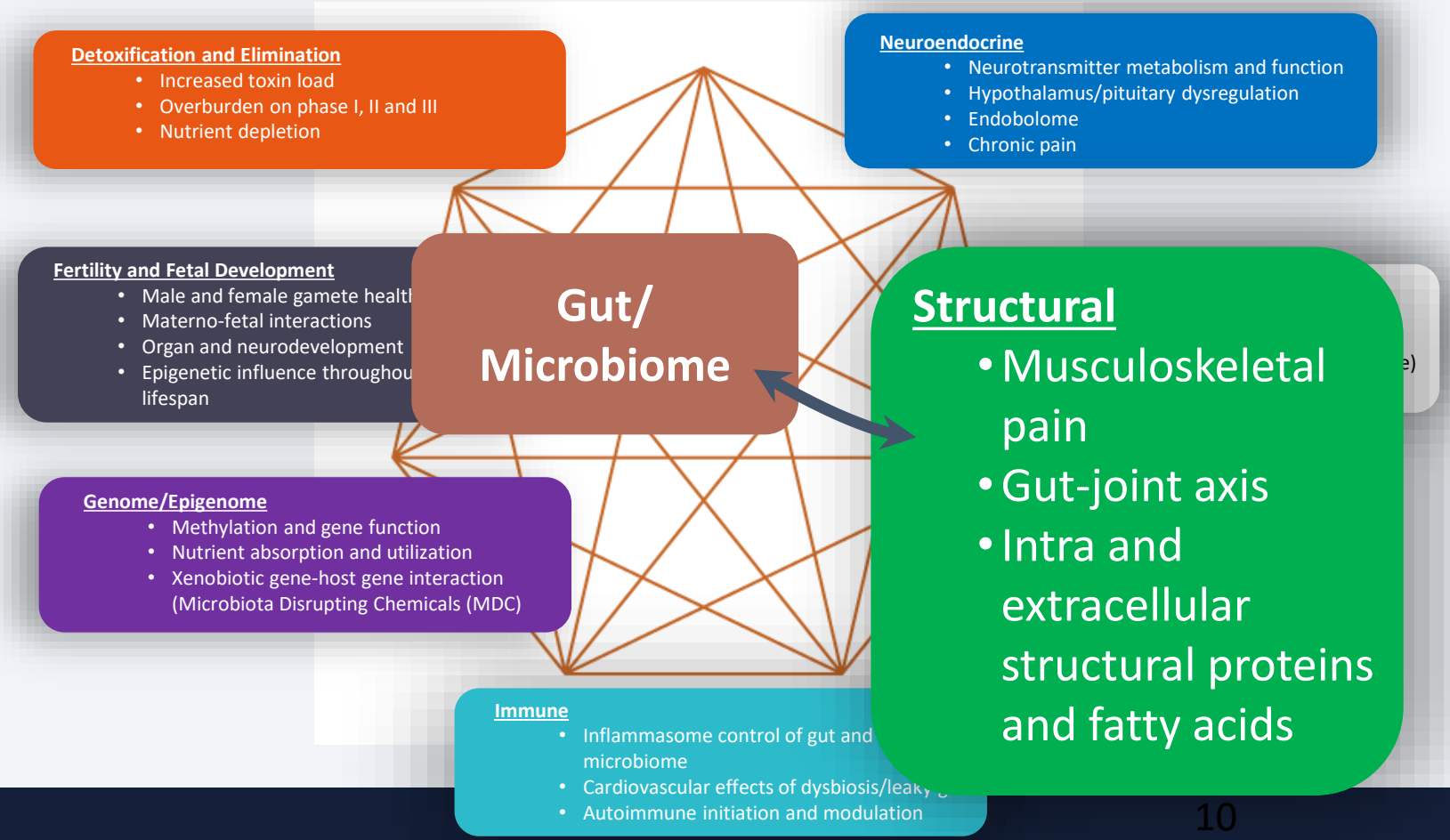
Redox Imbalance

- Gut dysbiosis directly contributes to mitochondrial oxidative stress and dysfunction across multiple organ systems

Essential Mitochondrial Support Agents

- CoQ10
- Quercetin
- Lipoic Acid
- Vitamins C, E, A, D
- NAC / Glutathione / Redox
- D-Ribose
- Adaptogens (Eleuthero, Rhodiola,
- Schisandra, Ashwagandha)
- Melatonin
- B-Vitamins (NAD)

Gut-Human Health Matrix



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Gut-Structural Connection

Gut-Joint Axis

- Articular involvement occurs in 16–33% of inflammatory bowel disease patients, increasing morbidity and reducing quality of life

Musculoskeletal Pain

- Inflammation driven by dysbiosis affects joints, connective tissue, and structural proteins through systemic inflammatory pathways

Mitochondria in Chondrocytes

- Mitochondrial integrity is a prerequisite for normal chondrocyte survival — dysfunction is found in both OA and RA

Structural Proteins & Fatty Acids

- Gut health influences intra and extracellular structural protein integrity and essential fatty acid metabolism

The Joint-Gut Axis in IBD

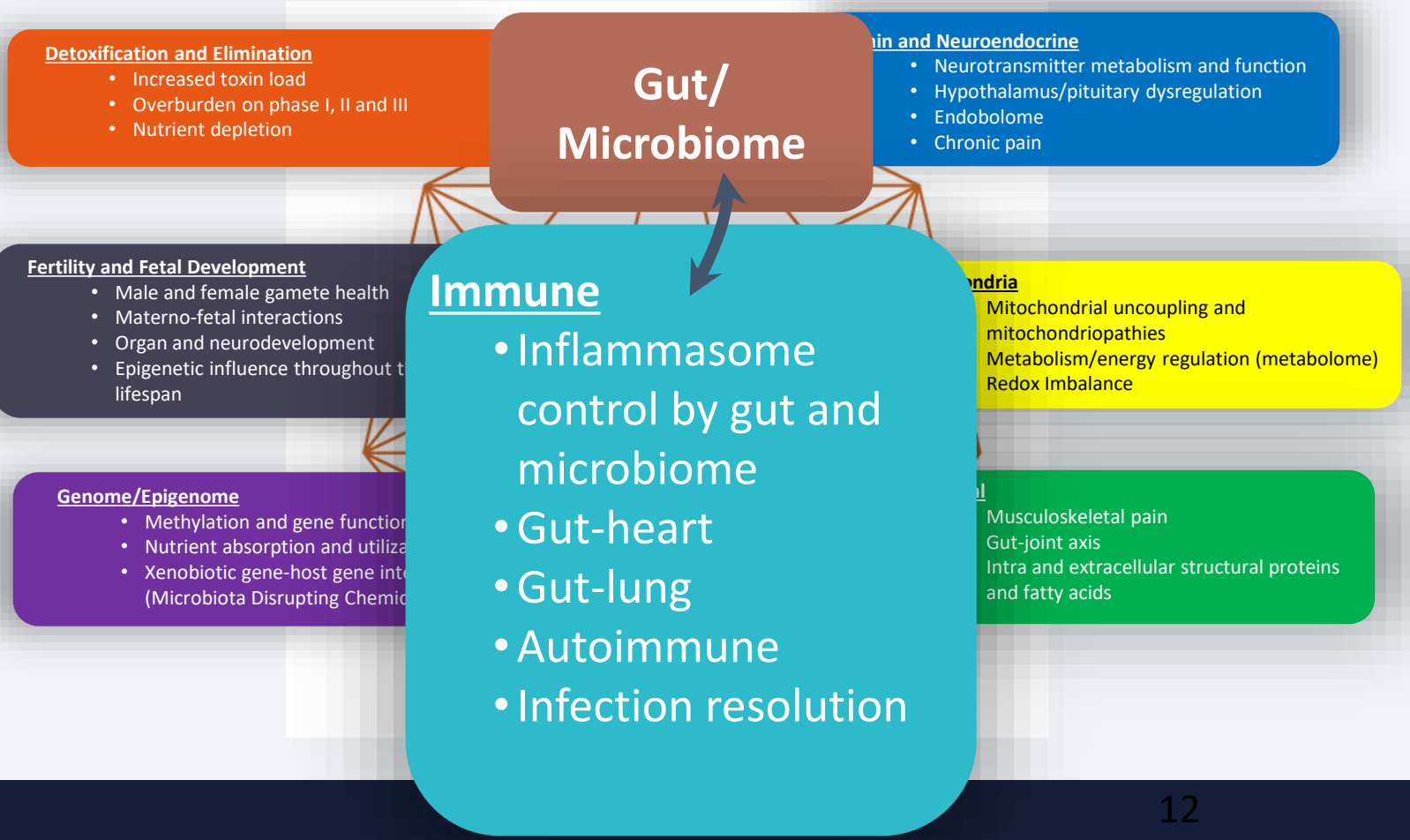
Journal of Crohn's and Colitis, Vol. 4, Issue 3

- Peripheral arthritis flares with bowel disease relapses
- Axial arthritis course is independent of IBD activity
- Linked to HLA-B27
- Treatment approach differs for peripheral vs axial

Key Support Agents

- Sulforaphane — inhibits inflammasomes
- EGCG — protects intervertebral disc cells
- Lipoic Acid + Omega-3 — neuroinflammation
- CoQ10 — reduces mitochondrial oxidative stress

Gut-Human Health Matrix



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Gut-Immune Connection

Inflammasome Control

- The gut microbiome directly controls inflammasome activation, modulating systemic inflammation

Cardiovascular Effects

- Dysbiosis and leaky gut drive cardiovascular inflammation through bacterial translocation and LPS

Autoimmune Triad

- Autoimmune initiation and modulation are directly linked to gut barrier integrity and microbial composition

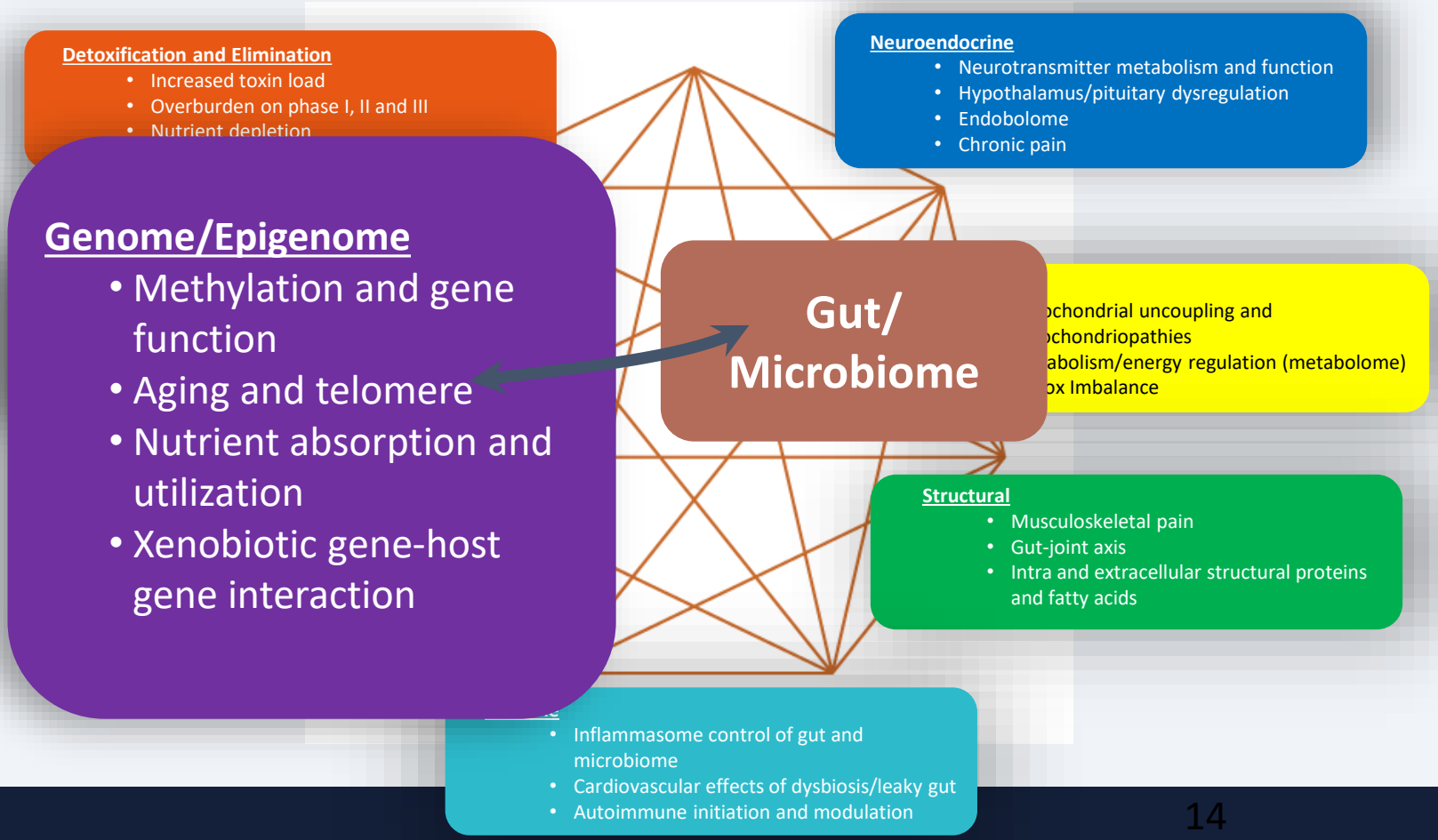
Gut-Lung Axis

- Emerging research connects gut microbiome composition to pulmonary immune responses

SCFA & Immune Function

- Short-chain fatty acids modulate immune cell function
- Butyrate regulates T-cell differentiation
- Propionate influences dendritic cell maturation
- Acetate provides energy for colonocytes and immune cells
- Melatonin from GI tract shows immunoregulatory and antioxidant functions

Gut-Human Health Matrix



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Gut-Genome/Epigenome Connection

Methylation & Gene Function

- The microbiome directly influences methylation pathways that control gene expression, impacting everything from detoxification to neurotransmitter synthesis

Nutrient Absorption & Utilization

- Gut health determines the bioavailability of key nutrients required for epigenetic regulation

Microbiota Disrupting Chemicals (MDCs)

- Xenobiotic gene-host gene interactions alter microbiome composition and epigenetic regulation simultaneously

Aging & Telomere Length

- Microbiome composition influences cellular aging processes and telomere maintenance

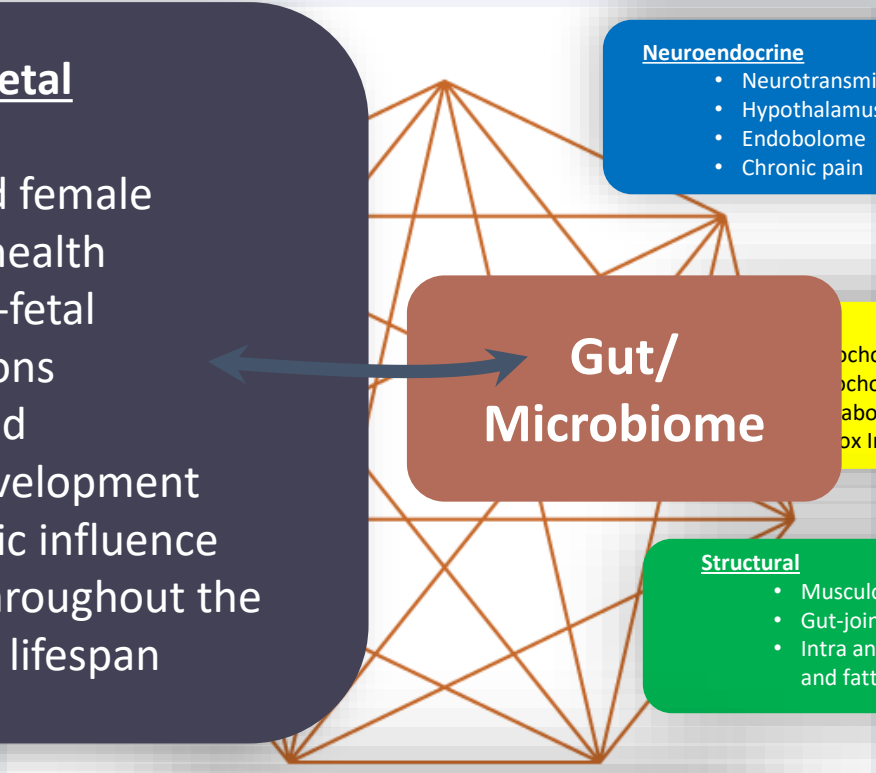
Epigenetic Effects of Probiotics

- Changes methylation patterns
- Modulates activity of cancer, oxidative, and immune genes
- Reduces SNP formation
- Can even reverse some SNPs
- Activates repair mechanisms inherent within the genome

LOE: A

Strong evidence supports the genetic and epigenetic influence of probiotics on the host genome

Gut-Human Health Matrix



Fertility and Fetal Development

- Male and female gamete health
- Materno-fetal interactions
- Organ and neurodevelopment
- Epigenetic influence lasting throughout the offspring lifespan

Gut/ Microbiome

Neuroendocrine

- Neurotransmitter metabolism and function
- Hypothalamus/pituitary dysregulation
- Endobolome
- Chronic pain

Mitochondrial

- ochondrial uncoupling and ochondriopathies
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Structural

- Musculoskeletal pain
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Immune

- Inflammasome control of gut and microbiome
- Cardiovascular effects of dysbiosis/leaky gut
- Autoimmune initiation and modulation

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Gut-Fertility & Fetal Development

Maternal Microbiome & Fetal Brain Development

- Chronic disease risk is inextricably linked to early life environment — maternal, fetal and childhood factors predict disease risk later in life
- Bacterial composition modulates weight gain and altered metabolism that drives obesity across pregnancy

Dysbiosis & Infertility

- Female microbiota dysbiosis (vaginal, endometrial, placental) and male dysbiosis (seminal fluid) can influence fertility, pre-term birth, and neonatal illness

Epigenetic Influence

- Microbiome-driven epigenetic changes can last throughout the offspring's entire lifespan

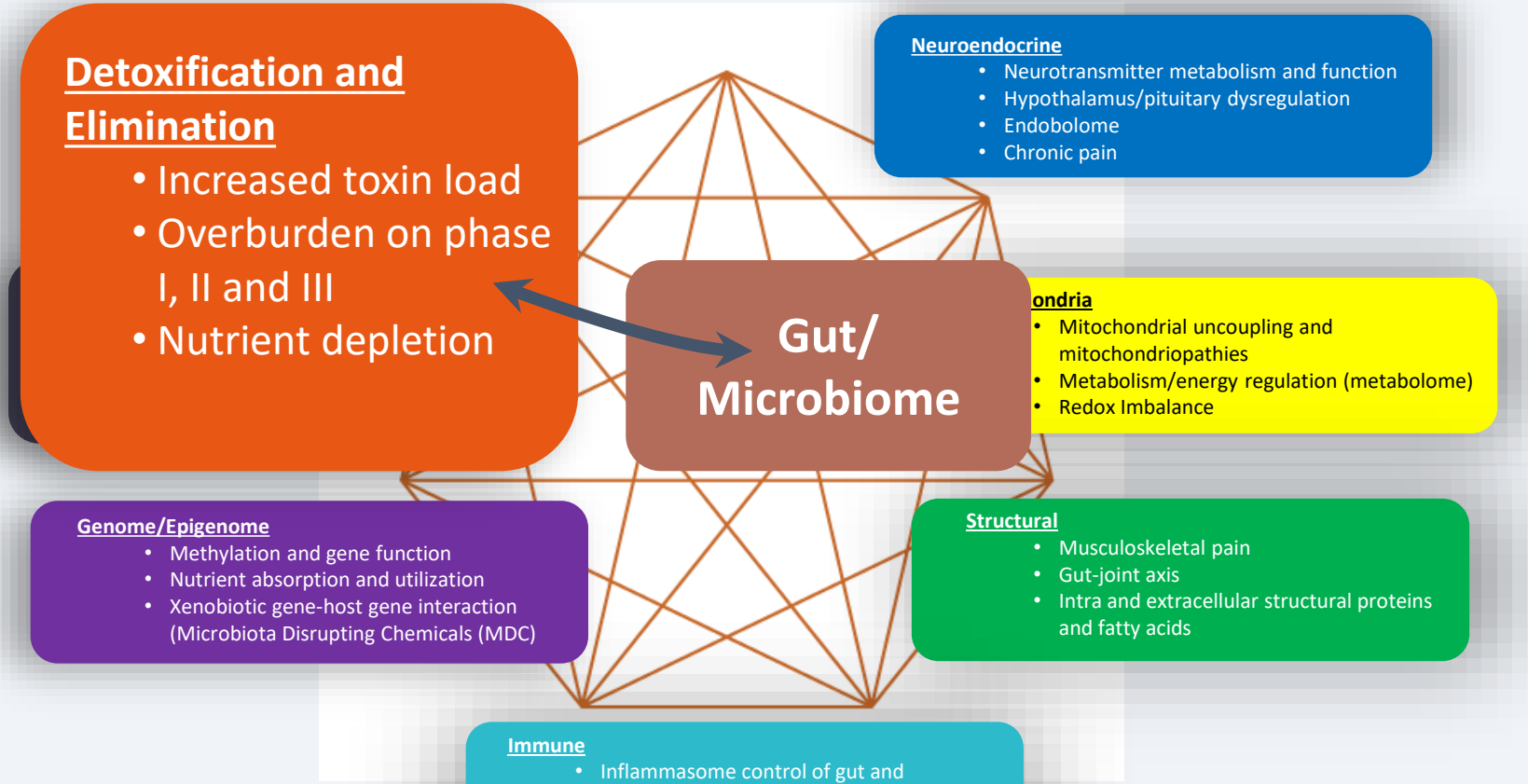
Clinical Implication

Role of stool testing in fertility and materno-fetal health: Assessing the maternal microbiome before and during pregnancy is an emerging standard in functional medicine practice.

Partner Microbiota Exchange

Unprotected sexual intercourse creates bacterial exchange between partners and can influence microbiota composition of each partner's reproductive tracts — a key consideration in fertility assessment.

Gut-Human Health Matrix



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Gut-Detoxification & Elimination

Increased Toxin Load

- Gut dysbiosis increases the body's toxic burden through impaired barrier function, allowing endotoxins and xenobiotics to enter systemic circulation

Phase I, II, and III Overburden

- Microbiome disruption overwhelms hepatic detoxification pathways, leading to accumulation of intermediate metabolites that are often more toxic than the parent compounds

Nutrient Depletion

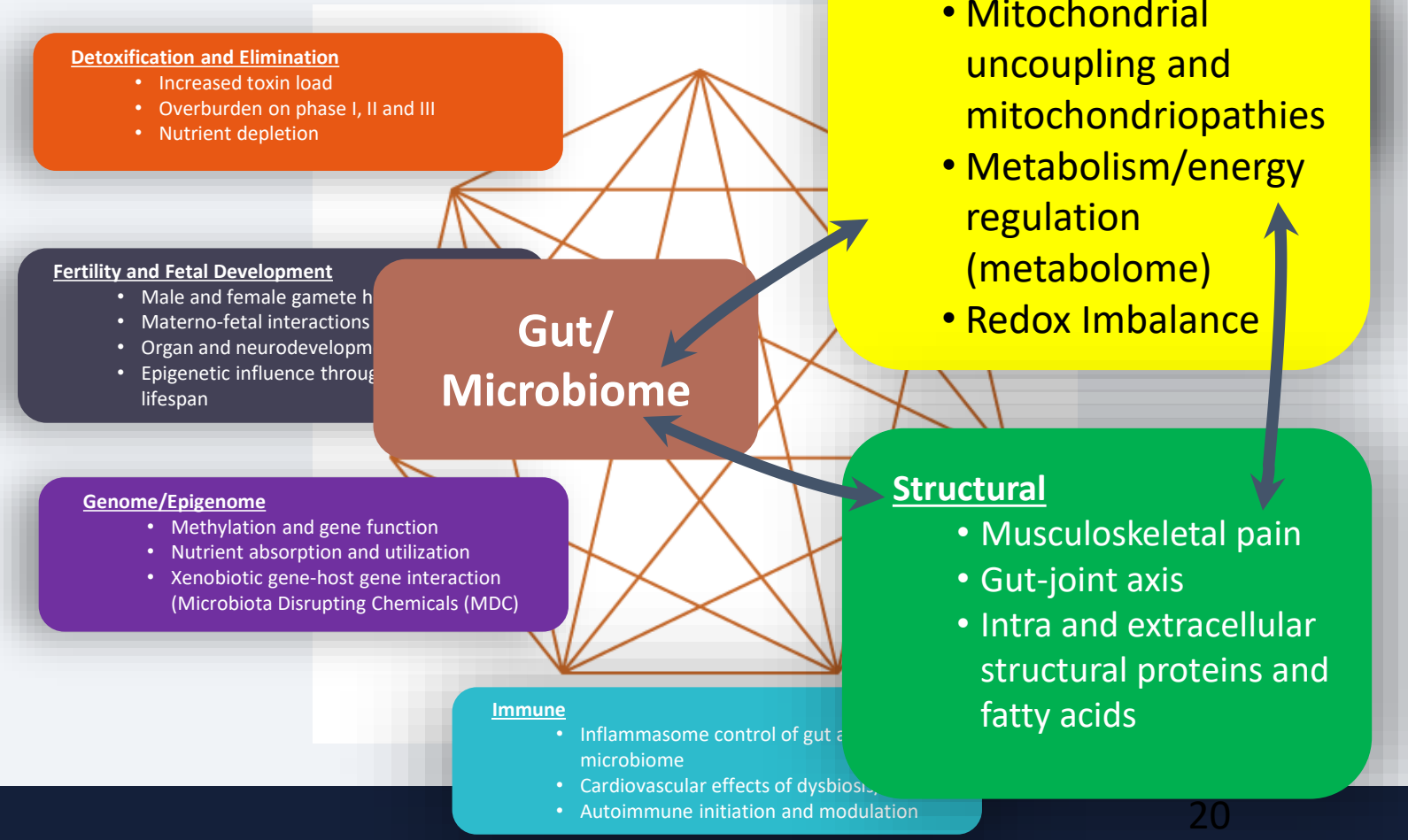
- Detoxification requires cofactors (glutathione, B vitamins, minerals) that become depleted when the gut cannot properly absorb and produce them

Raising Glutathione Naturally

The most important clinical concept to understand

- Lipoic Acid
- Grape Seed Extract
- NAC
- SAMe
- L-Glutamine
- Silymarin
- Cordyceps
- Curcumin
- Sulforaphane
- Green Tea Extract (EGCG)

Gut-Human Health Matrix



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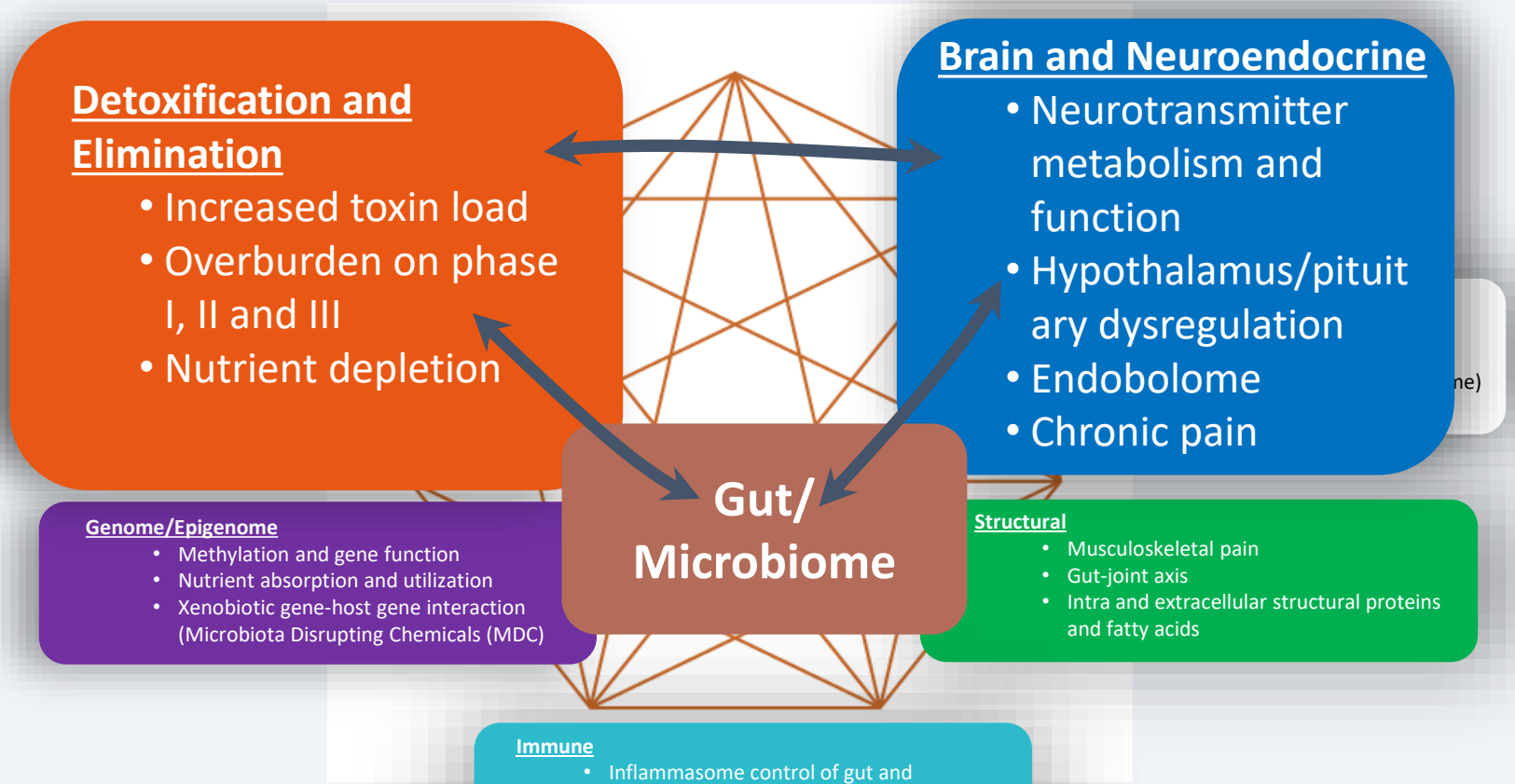

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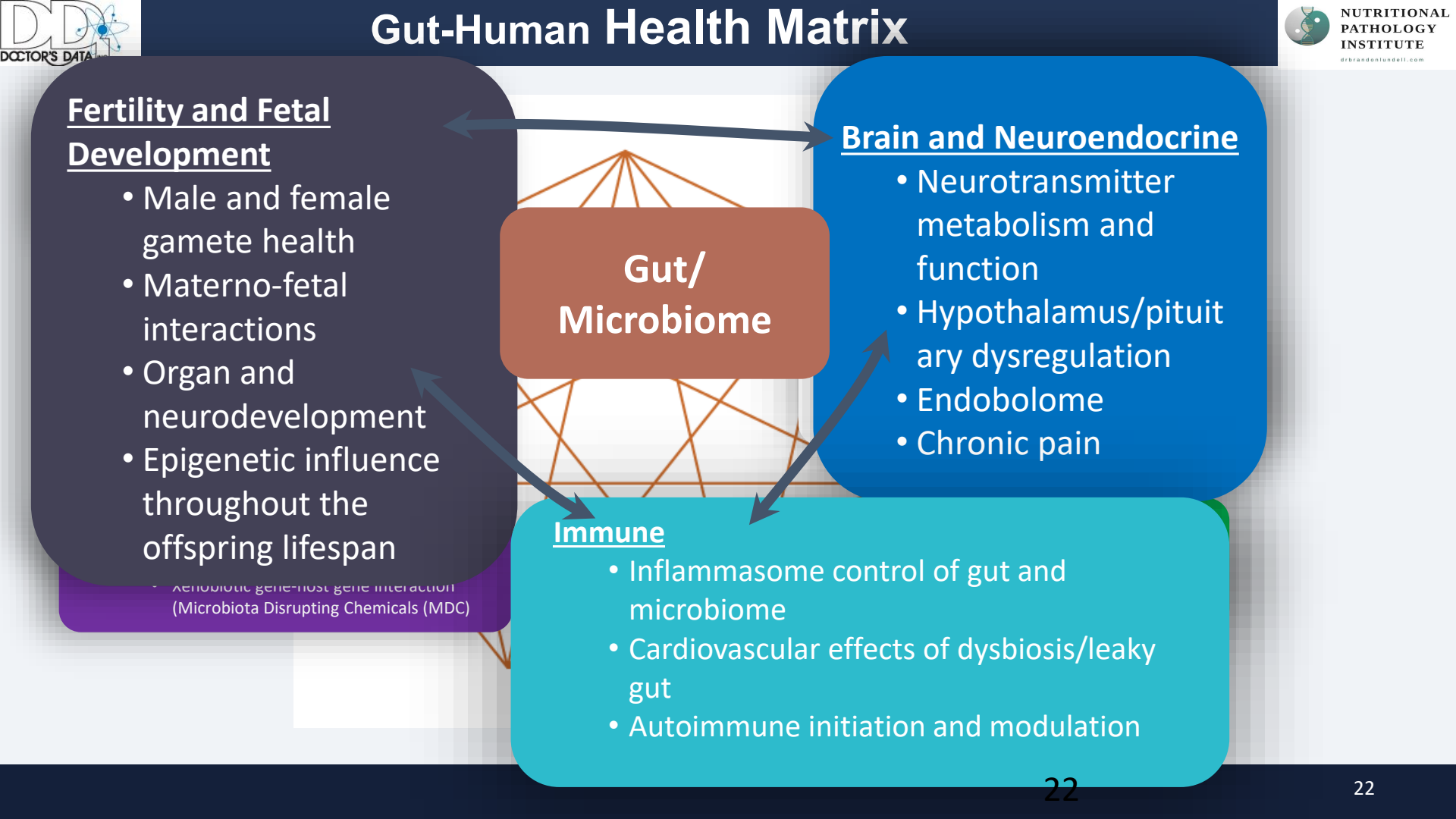

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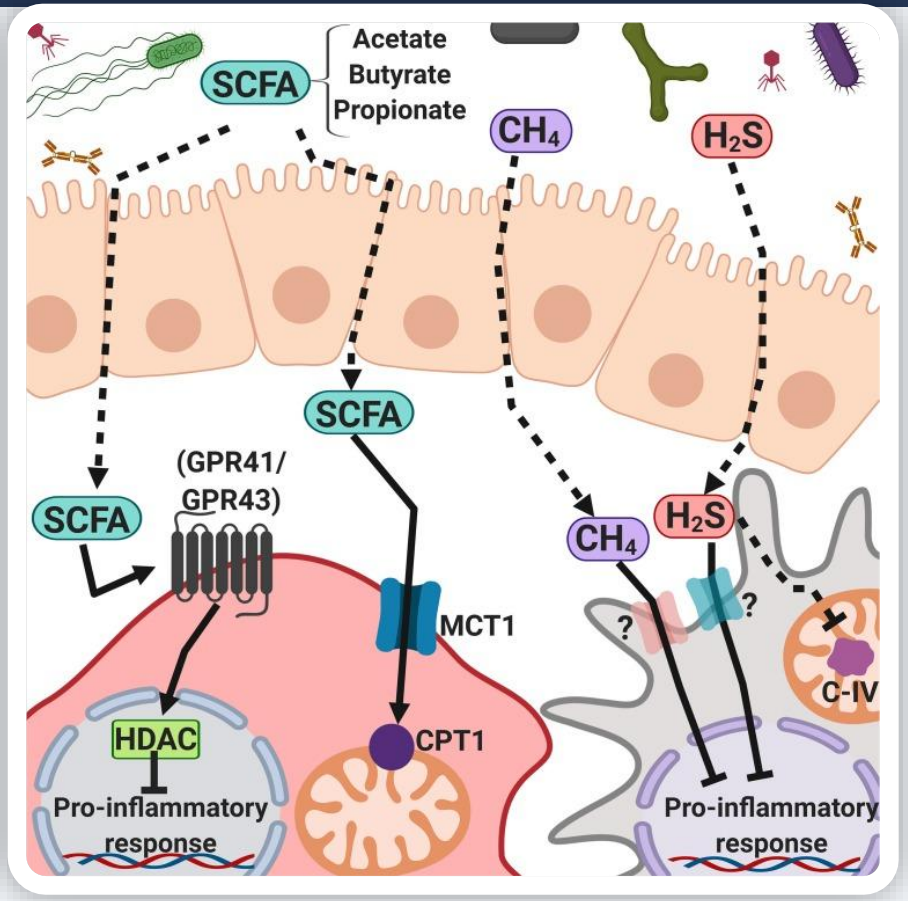
The Metabolome

- The metabolome is the repertoire of small biomolecules present in cells, tissues, and body fluids, and its composition is at the core of the health status of individuals. The development of new “metabolomic platforms” has revealed that a number of metabolites present in several biological samples, such as serum and urine, vary in concentration following a circadian rhythmicity (Martínez-Lozano et al., 2014; de Raad et al., 2016). Among them are glycolysis-related metabolites, such as glucose, glucose-6-phosphate, bisphosphoglycerate, and lactate; tricarboxylic acid (TCA) cycle-related molecules, such as acetate, acetyl CoA, citrate, isocitrate, and malonate; amino acids and their derivatives; lipid metabolites; nucleotides; antioxidants; and coenzymes such as NAD, FAD, and coenzyme A (Krishnaiah et al., 2017).
- Interestingly, the daily variation in the bacterial composition within the intestine implies a daily variation in the concentration of some bacteria-derived metabolites, and the hundreds of microbiota-derived metabolites that have been identified are regarded as components of the human metabolome (Belizário et al., 2018). Thus, linking eukaryotic- and bacterial-derived metabolites with the other three biological domains is discussed here.
- In attempting to convey the view that mitochondria support and integrate the communication between the four mentioned biological domains, the specific roles of mitochondria are discussed in the next sections.

SCFA

A higher proportion of SCFA-producing bacteria within the intestinal microbiota is associated with a reduction in the risk of developing obesity, insulin resistance, and type 2 diabetes, since these compounds, particularly butyrate, increase cellular respiration and fatty acid oxidation (Belizário et al., 2018). Acetate, butyrate, and propionate are the most abundant SCFAs and represent 90–95% of the total SCFAs present in the colon.



Front Cell Dev Biol. 2020; 8: 51.

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PERSPECTIVE
Clinical Science and Epidemiology

Do an Altered Gut Microbiota and an Associated Leaky Gut Affect COVID-19 Severity?

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ABSTRACT Coronavirus disease 2019 (COVID-19), which has been declared a pandemic, has exhibited a wide range of severity worldwide. Although this global variation is largely affected by socio-medical situations in each country, there is also high individual-level variation attributable to elderliness and certain underlying medical conditions, including high blood pressure, diabetes, and obesity. As both elderliness and the aforementioned chronic conditions are often associated with an altered gut microbiota, resulting in disrupted gut barrier integrity, and gut symptoms have consistently been associated with more severe illness in COVID-19 patients, it is possible that dysfunction of the gut as a whole influences COVID-19 severity. This article summarizes the accumulating evidence that supports the hypothesis that an altered gut microbiota and its associated leaky gut may contribute to the onset of gastrointestinal symptoms and occasionally to additional multiorgan complications that may lead to severe illness by allowing leakage of the causative coronavirus into the circulatory system.

KEYWORDS COVID-19, SARS-CoV-2, coronavirus, gut microbiota, gut barrier integrity, leaky gut

KEY MESSAGES

- While the following remains to be empirically demonstrated, accumulating evidence supports the hypothesis that an altered gut microbiota and an associated leaky gut may contribute to the onset of coronavirus disease 2019 (COVID-19)-related gastrointestinal symptoms, such as diarrhea and, in severe cases, multiorgan complications.
- Testing for a leaky gut and fecal and plasma viral loads may be useful for diagnosing the seriously ill or for preventing transmission by fecal shedding of the virus.
- Fecal microbiota transplantation (FMT), next-generation probiotics focusing on butyrate-producing gut microbes, or simply increasing the daily intake of dietary fiber may be considered in improving the gut health of COVID-19 patients.

SCFA and Immune Function

Notably, these studies demonstrating a close link between gut microbiota dysbiosis and COVID-19 severity have reported a common finding (52–54). Beneficial bacteria, whose abundance was reduced in COVID-19 patients, were reported to belong to the families *Ruminococcaceae* or *Lachnospiraceae* (52), a single species *F. prausnitzii* (53), and the class *Clostridia* (54). The class *Clostridia* includes the family *Ruminococcaceae*, which includes the species *F. prausnitzii*, which is one of the major butyric acid-producing bacteria in the gut (55). While the beneficial impact of *F. prausnitzii* on human health is well established (56), a subspecies that causes a predisposition to atopic dermatitis in infants and young children by competing with the beneficial members of the species has been identified (57). The intricate microbial interactions and the physiology involving this important butyrate-producing species warrant future investigations to understand its influence on human health and disease (57, 58).

Melatonin: its possible role in the management of viral infections--a brief review

Michela Silvestri [1], Giovanni A Rossi

Affiliations + expand

PMID: 24090288 PMCID: PMC3850896 DOI: 10.1186/1824-7288-39-61

Free PMC article

Abstract

Melatonin, a versatile molecule, is synthesized by the pineal gland but also by other organs, including gastrointestinal tract, retina, thymus, bone marrow, and by leukocytes. Besides playing an important role in various functions of the body, including sleep and circadian rhythm regulation, melatonin also shows immunoregulatory, free radical scavenger and antioxidant functions. Because of these latter characteristics melatonin has also been found to be effective in fighting viral infections in a variety of experimental animal and in vitro studies. These data suggest a possible therapeutic potential of melatonin in human virus-induced disorders.

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Ital J Pediatr. 2013 Oct 3;39:61. doi: 10.1186/1824-7288-39-61.

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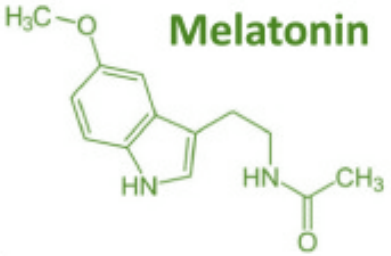
Immunomodulation

Regulation of circadian rhythms

Regulation of mitochondrial functions

Measurement of day length



Anti-inflammatory properties

Sleep initiation

Antioxidant actions

Vasomotor control

Anti-excitatory actions

Doctor's Data GI Test Menu

GI 360™ Profile

Most comprehensive assessment

- Microbiome abundance & diversity (PCR, 45+ analytes)
- GI Pathogens (14 targets, multiplex PCR)
- Parasitology (PCR + 3-day microscopy)
- Bacteriology & yeast culture (MALDI-TOF ID)
- Stool chemistries (digestive, inflammatory, immune)
- Bacterial susceptibility (Rx + natural agents)

Add-Ons: H. pylori Stool Antigen, Zonulin Family Protein, Comprehensive Clostridium Culture, Macroscopic Worm ID

CSA + Parasitology

GI 360 without microbiome PCR

- All GI 360 components EXCEPT microbiome diversity/abundance
- Includes GI Pathogen PCR (14 targets)
- Ideal for follow-up after initial GI 360
- Culture, parasitology, stool chemistries retained
- When microbiome retesting is less critical

CSA

Core stool analysis

- Bacteriology & yeast culture
- Microscopic examination
- Stool chemistries
- Does not include full parasitology (+P adds this)

Microbiology Only

Targeted follow-up

- Bacteriology culture (expected, imbalanced, dysbiotic)
- Yeast culture
- Susceptibility testing (when indicated)
- Ideal for monitoring dysbiosis resolution
- Most cost-effective follow-up option

	GI 360	CSA+P Comprehensive Stool Analysis + Parasitology	CSA Comprehensive Stool Analysis	CP+P Culture, PCR + Parasites	GI Pathogen Profile
GI Microbiome Diversity and Abundance; PCR	✓				
Viruses, Pathogens and Parasites; PCR	✓	✓	✓	✓	✓
Expected/Beneficial Bacteria Culture: Including *Bacteroides fragilis, Bifidobacteria, E. coli, Lactobacillus, Enterococcus, Clostridium* spp.		✓	✓	✓	
Dysbiotic Bacteria Culture and ID: Including *Aeromonas, Campylobacter, Plesiomonas, Salmonella, Shigella, Vibrio, Yersinia, Edwardsiella tarda*	✓	✓	✓	✓	
Commensal/Imbalanced Bacteria Culture and ID	✓	✓	✓	✓	
Yeast Culture and ID	✓	✓	✓	✓	
Pharmaceutical and Natural Agent Yeast/Bacterial Susceptibilities (performed when indicated)	✓	✓	✓	✓	
Parasitology Identification Concentrate and Trichrome Stain	✓	✓		✓	
Giardia lamblia	✓	✓		✓	
Cryptosporidium	✓	✓		✓	
Elastase	✓	✓	✓		
Fat Stain	✓	✓	✓		
Muscle and Vegetable Fibers	✓	✓	✓		
Carbohydrates	✓	✓	✓		
Lysozyme	✓	✓	✓		
Calprotectin	✓	✓	✓		
Lactoferrin	✓	✓	✓		
White Blood Cells (WBC)	✓	✓	✓		
Mucus	✓	✓	✓		
Secretory IgA	✓	✓	✓		
Short Chain Fatty Acids	✓	✓	✓		
Red Blood Cells (RBC)	✓	✓	✓		
pH	✓	✓	✓		
Occult Blood	✓	✓	✓		
Beta-Glucuronidase	✓				

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Progressive Testing Strategy

1

Initial Assessment

GI 360

Establish comprehensive baseline. Identifies dysbiosis patterns, parasites, pathogens, digestive and inflammatory status.

2

First Follow-Up

CSA+P

Confirm improvement. Contains everything except microbiome PCR. Tracks pathogen elimination and inflammatory markers.

3

Targeted Monitoring

Microbiology Only

Track bacterial/yeast balance specifically. Most cost-effective. Confirms dysbiotic organism eradication.

4

Reassessment

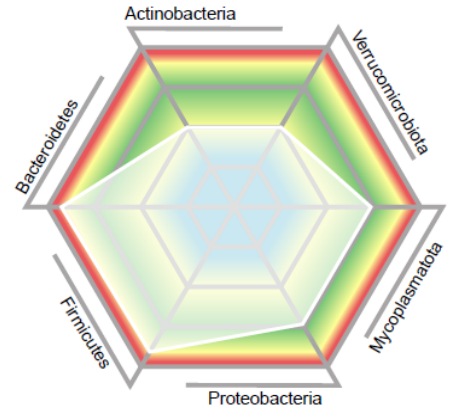
CSA+P or GI 360

Return to full panel if symptoms change, new GI symptoms develop, or for periodic comprehensive re-evaluation.

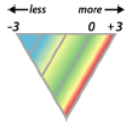
Clinical Pearl: Once expected and beneficial flora balance out on microbiology culture, the PCR microbiome diversity index typically normalizes as well.

Microbiome Abundance and Diversity Summary

The abundance and diversity of gastrointestinal bacteria provide an indication of gastrointestinal health, and gut microbial imbalances can contribute to dysbiosis and other chronic disease states. The GI360™ Microbiome Profile is a gut microbiota DNA analysis tool that identifies and characterizes more than 45 targeted analytes across six Phyla using PCR and compares the patient results to a characterized normobiotic reference population. The web chart illustrates the degree to which an individual's microbiome profile deviates from normobiosis.



LEGEND



The web image shows the relative diversity and balance among bacteria belonging to the six primary Phyla. The white shaded area represents the patient's results compared to a normobiotic reference population. The center of the web represents less abundance while the outer edges represent more than normobiotic.

Dysbiosis and Diversity Index

These indexes are calculated from the results of the Microbiome Profile, with scores ranging from 1 to 5, and do not include consideration of dysbiotic and pathogenic bacteria, yeast, parasites and viruses that may be reported in subsequent sections of the GI360™ test.

The Dysbiosis Index the (DI) is calculated strictly from the results of the Microbiome Profile, with scores from 1 to 5. A DI score above 2 indicates dysbiosis; a microbiota profile that differs from the defined normobiotic reference population. The higher the DI above 2, the more the sample deviates from the normobiotic profile. The dysbiosis test and DI does not include consideration of dysbiotic and pathogenic bacteria, yeast, parasites and viruses that may be reported in subsequent sections of the GI360™ test.

A diversity score of 3 indicates an expected amount of diversity, with 4 & 5 indicating an increased distribution of bacteria based on the number of different species and their abundance in the sample, calculated based on Shannon's diversity index. Scores of 1 or 2 indicate less diversity than the defined normobiotic reference population.

Dysbiosis Index

Diversity Score

GI Health Markers

Butyrate producing bacteria	
Gut barrier protective bacteria	
Gut intestinal health marker	
Pro-inflammatory bacteria	
Gut barrier protective bacteria vs. opportunistic bacteria	

= Expected = Imbalanced

Key Findings

Citrobacter farmeri, Cultured
Citrobacter freundii complex, Cultured
Enterobacter cloacae complex, Cultured
Klebsiella pneumoniae, Cultured

Microbiome Abundance & Diversity Analysis

Dysbiosis Index (DI)

- Scored 1–5; values above 2 indicate dysbiosis
- Calculated strictly from Microbiome Profile PCR data
- Does NOT include dysbiotic bacteria, yeast, parasites, or viruses
- Compared against normobiotic reference population (n > 1,100)
- Higher DI = greater deviation from normobiosis

Diversity Score

- Scored 1–5, based on Shannon's diversity index
- Score of 3 = expected diversity
- Scores 4–5 = increased diversity (generally favorable)
- Scores 1–2 = lower than expected
- Inversely correlated with DI (higher DI = lower diversity)

CRITICAL CLINICAL LIMITATION

⚠ The DI only evaluates eubacteria (normal bacteria) — it does NOT consider pathogenic or dysbiotic bacteria, yeast, parasites, or viruses

⚠ A patient can have a normal DI score while harboring pathogenic organisms — this represents a false negative if viewed in isolation

⚠ Only when a 3+ or 4+ growth of major dysbiotic bacteria is present will it potentially affect the microbiome score

⚠ Always integrate the DI with microbiology culture results, parasitology, and stool chemistries — never treat based on DI alone

Interpreting the Hexagonal Web Chart — Six Primary Phyla

Firmicutes

Largest phylum. Includes Clostridia, Bacilli, Lactobacillus, Faecalibacterium prausnitzii (major butyrate producer). Elevated Mediterr. gnavus = inflammation marker.

Bacteroidetes

Complex polysaccharide degradation. Bacteroides spp., Prevotella, Parabacteroides. Key for SCFA production and immune modulation.

Actinobacteria

Includes Bifidobacterium family — critical for gut barrier, SCFA production, and pathogen resistance. Low levels = significant clinical finding.

Proteobacteria

Includes Enterobacteriaceae, E. coli. Elevated levels may indicate inflammation. Contains many pathogenic genera.

Mycoplasmatota

Formerly Tenericutes. Includes Metamycoplasma hominis. Relatively minor phylum but tracked for completeness.

Verrucomicrobiota

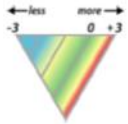
Formerly Verrucomicrobia. Primarily Akkermansia muciniphila — critical for mucin degradation and barrier function. Low = concerning.

Results shown as –3 to +3 SD vs. normobiotic reference population | Focus on ±2 and ±3 deviations for clinical decisions | White area = patient profile

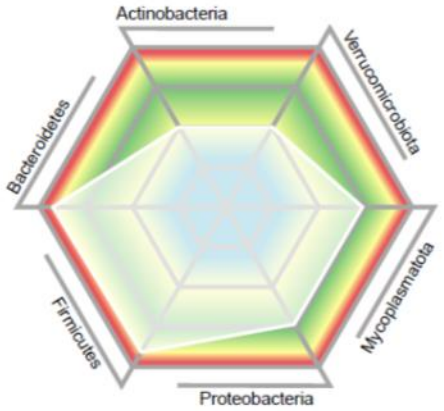
Microbiome Abundance and Diversity Summary

The abundance and diversity of gastrointestinal bacteria provide an indication of gastrointestinal health, and gut microbial imbalances can contribute to dysbiosis and other chronic disease states. The GI360™ Microbiome Profile is a gut microbiota DNA analysis tool that identifies and characterizes more than 45 targeted analytes across six Phyla using PCR and compares the patient results to a characterized normobiotic reference population. The web chart illustrates the degree to which an individual's microbiome profile deviates from normobiosis.

LEGEND



The web image shows the relative diversity and balance among bacteria belonging to the six primary Phyla. The white shaded area represents the patient's results compared to a normobiotic reference population. The center of the web represents less abundance while the outer edges represent more than normobiotic.



Dysbiosis and Diversity Index

These indexes are calculated from the results of the Microbiome Profile, with scores ranging from 1 to 5, and do not include consideration of dysbiotic and pathogenic bacteria, yeast, parasites and viruses that may be reported in subsequent sections of the GI360™ test.

The Dysbiosis Index the (DI) is calculated strictly from the results of the Microbiome Profile, with scores from 1 to 5. A DI score above 2 indicates dysbiosis; a microbiota profile that differs from the defined normobiotic reference population. The higher the DI above 2, the more the sample deviates from the normobiotic profile. The dysbiosis test and DI does not include consideration of dysbiotic and pathogenic bacteria, yeast, parasites and viruses that may be reported in subsequent sections of the GI360™ test.

A diversity score of 3 indicates an expected amount of diversity, with 4 & 5 indicating an increased distribution of bacteria based on the number of different species and their abundance in the sample, calculated based on Shannon's diversity index. Scores of 1 or 2 indicate less diversity than the defined normobiotic reference population.

GI Health Markers

Butyrate producing bacteria	
Gut barrier protective bacteria	
Gut intestinal health marker	
Pro-inflammatory bacteria	
Gut barrier protective bacteria vs. opportunistic bacteria	

= Expected = Imbalanced

Key Findings

Citrobacter farmeri, Cultured
Citrobacter freundii complex, Cultured
Enterobacter cloacae complex, Cultured
Klebsiella pneumoniae, Cultured

Parasitology Assessment — Dual Detection Methods

Multiplex PCR

3 Parasitic Targets Only:

- Cryptosporidium (C. parvum and C. hominis)
- Entamoeba histolytica
- Giardia duodenalis

Also includes 7–8 pathogenic bacteria + 3 viruses = 14 total PCR targets

Important: PCR does NOT differentiate viable from non-viable organisms. Do NOT repeat sooner than 21 days after treatment to avoid false positives from lingering DNA.

3-Day Microscopy

31+ Parasites Screened:

- Protozoa: Blastocystis, D. fragilis, Endolimax nana, E. coli, E. histolytica/dispar + more
- Nematodes: Ascaris, hookworm, Strongyloides, Trichuris, Enterobius
- Cestodes: Taenia, Hymenolepis, Diphyllobothrium
- Trematodes: Clonorchis, Fasciola, Paragonimus
- Also: Yeast, RBC, WBC, muscle/vegetable fibers, Charcot-Leyden crystals, mucus

Clinical Considerations

- 3-specimen collection increases parasitology sensitivity from ~80% to 96–98%
- Positive PCR without symptoms may indicate biofilm infestation or para-intestinal colonization
- Treating without clinical manifestations of GI disturbance is generally not indicated
- With Blastocystis or Endolimax nana: evaluate the full clinical picture before initiating treatment

Microbiology: The Four-Category Classification System

Expected Bacteria

- Bacteroides family
- Bifidobacterium family
- Escherichia coli
- Lactobacillus family
- Enterococcus family

Beneficial flora — restoration is a primary treatment goal

Imbalanced Bacteria

- Bacillus species
- Streptococcus species
- Hafnia alvei
- Low-level Klebsiella/Citrobacter
- Neither beneficial nor harmful at moderate levels

Treatment with antimicrobials is unnecessary unless elevated to dysbiotic levels or clinical presentation warrants

Dysbiotic Bacteria

- Klebsiella pneumoniae (3–4+)
- Enterobacter cloacae (3+)
- Citrobacter freundii (3+)
- Pseudomonas species
- Level-dependent classification

Thresholds per DD: dysbiotic at 3+ or greater; imbalanced at lower levels

Pathogenic Bacteria

- Aeromonas spp.
- Salmonella group
- Shigella group
- Vibrio spp.
- Yersinia spp., Edwardsiella, Plesiomonas

Always warrants treatment regardless of level — detected by culture and/or PCR

Methodology: Culture with identification by MALDI-TOF mass spectrometry and conventional biochemicals | Can identify >1,600 genera/species

Pathogenic Bacteria	Result	NG 1+ 2+ 3+ 4+	Reference Interval
Aeromonas spp.	NG		No Growth
Edwardsiella tarda	NG		No Growth
Plesiomonas shigelloides	NG		No Growth
Salmonella group	NG		No Growth
Shigella group	NG		No Growth
Vibrio cholerae	NG		No Growth
Vibrio spp.	NG		No Growth
Yersinia spp.	NG		No Growth

Imbalanced Bacteria	Result	NG 1+ 2+ 3+ 4+	Reference Interval
Bacillus licheniformis	1+		No Growth
Corynebacterium aurimucosum	2+		No Growth
Streptomyces phaeochromogenes	3+		No Growth

Dysbiotic Bacteria	Result	NG 1+ 2+ 3+ 4+	Reference Interval
Citrobacter farmeri	4+		No Growth
Citrobacter freundii complex	4+		No Growth
Enterobacter cloacae complex	4+		No Growth
Klebsiella pneumoniae	4+		No Growth

Yeast	Result	NG 1+ 2+ 3+ 4+	Reference Interval
Candida glabrata	1+		0+ – 1+

Comprehensive Stool Analysis

BACTERIOLOGY CULTURE		
Expected/Beneficial Bacteria	**Commensal (Imbalanced) flora**	**Dysbiotic flora**
4+ *Bacteroides* family	1+ *Kocuria rhizophila*	4+ *Pseudomonas aeruginosa*
3+ *Bifidobacterium* family	3+ *Streptococcus anginosus*	
4+ *Escherichia coli*		
3+ *Lactobacillus* family		
4+ *Enterococcus* family		
2+ *Clostridium* family		
NG = No Growth		

Bacterial & Yeast Susceptibility Testing

Prescriptive Agents

Results categorized as:

Susceptible:	Likely to respond to standard dosage
Intermediate:	May respond at higher dosage
Resistant:	Unlikely to respond to treatment

Tested on each patient's pure isolates — not wall charts

Natural Agents Tested by DD

Results displayed on a scale from Low to High inhibition:

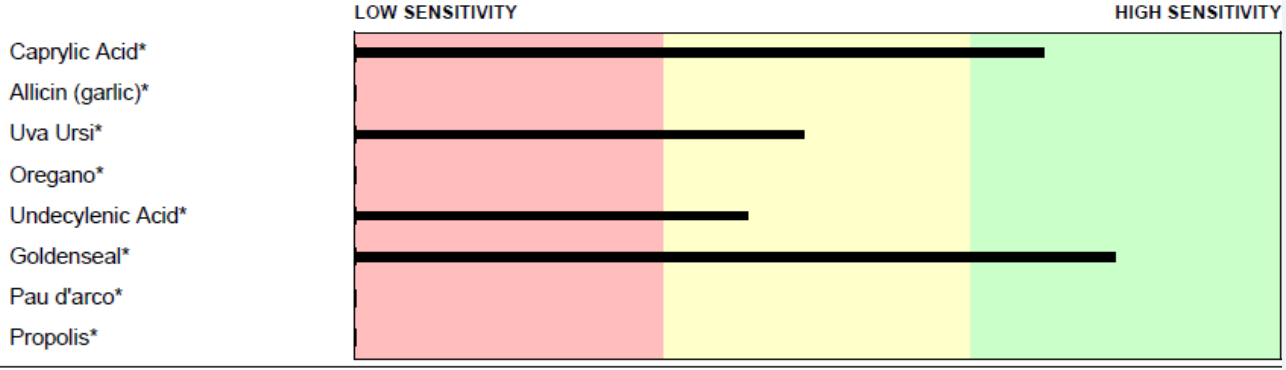
- Caprylic Acid
- Uva Ursi
- Olive Leaf Extract
- Oregano
- Goldenseal
- Ionic Silver
- Colloidal Silver

Clinical Application

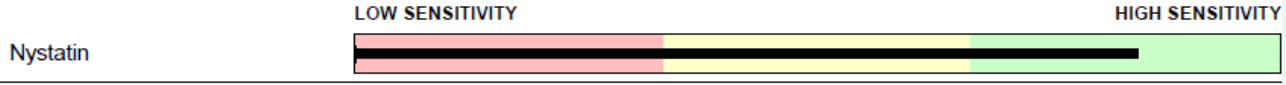
- Susceptibility testing requires cultured isolates of live bacteria and yeast — PCR alone cannot provide this
- Natural antimicrobials can be highly effective, particularly in combination therapy
- Susceptibility is performed for most dysbiotic and pathogenic species; occasionally not performed for strict anaerobes
- Additional clinically useful agents (not on DD panel): Black Walnut, Grapefruit Seed Extract, Berberine, Pau d' Arco — consider based on clinical experience
- Colloidal silver dosing: approximately 60 mL (quarter cup) twice daily for systemic infections

Candida albicans

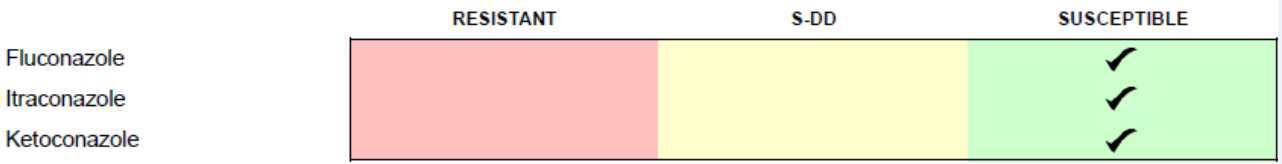
NATURAL ANTIFUNGALS



NON-ABSORBED ANTIFUNGALS



AZOLE ANTIFUNGALS



Standardized test interpretive categories established for *Candida* spp. are used for all yeast isolates.

Klebsiella pneumoniae

Natural Agents	Low Inhibition ... High Inhibition
Caprylic Acid*	
Uva Ursi*	
Olive Leaf Extract*	
Oregano*	
Goldenseal*	
Ionic Silver*	
Colloidal Silver*	

Prescriptive Agents	Resistant	Intermediate	Susceptible
Amoxicillin-Clavulanic Acid			●
Ampicillin	●		
Cefazolin			●
Ceftazidime			●
Ciprofloxacin			●
Sulfamethoxazole / Trimethoprim			●

Stool Chemistry: Digestive Function Markers

Pancreatic Elastase

- DD Reference: >200 µg/g
- <200 µg/g = pancreatic exocrine insufficiency
- Clinical interpretation: 200–400 may indicate mild insufficiency
- Correlates with need for enzyme supplementation (trypsin, chymotrypsin, lipases, amylases)
- Low elastase often correlates with low HCl
- May also indicate insufficient pancreatic bicarbonate

Fat Stain

- DD Reference: None – Moderate
- Increased = fat malabsorption or steatorrhea
- "Few" = mild fat malabsorption

Carbohydrates

- DD Reference: Negative
- Positive = impaired digestion of complex carbohydrates
- Suggests enzymatic insufficiency or rapid transit

Microscopic Markers of Digestive Function

- Muscle fibers (Ref: Not Detected – Rare): Indicator of incomplete protein digestion; associated with pancreatic insufficiency and may correlate with low elastase
- Vegetable fibers (Ref: Not Detected – Few): May indicate inadequate chewing or rapid transit
- Yeast (Ref: Not Detected – Few): Abnormal if moderate to many; may be visible on microscopy but not grow in culture

Clinical Correlation: Low elastase + undigested muscle fibers + alkaline pH = strong indication for HCl assessment and pancreatic enzyme support. Always evaluate both HCl and pancreatic function for comprehensive digestive support.

Digestion / Absorption	Result	Unit	L WRI H	Reference Interval
Elastase	343	µg/mL		> 200
Fat Stain	None			None – Few
Carbohydrates†	Negative			Negative

Inflammation	Result	Unit	L WRI H	Reference Interval
Lactoferrin	124	µg/mL		< 7.3
Lysozyme*	814	ng/mL		≤ 500
Calprotectin	12	µg/g		≤ 50

Immunology	Result	Unit	L WRI H	Reference Interval
Secretory IgA*	38.6	mg/dL		30 – 275

Short Chain Fatty Acids	Result	Unit	L WRI H	Reference Interval
% Acetate‡	60			50 – 72
% Propionate‡	17			11 – 25
% Butyrate‡	21			11 – 32
% Valerate‡	1.7			0.8 – 5.0
Butyrate‡	2.4	mg/mL		0.8 – 4.0
Total SCFA's‡	11	mg/mL		5.0 – 16.0

Intestinal Health Markers	Result	Unit	L WRI H	Reference Interval
pH	5.4			5.8 – 7.0
β-glucuronidase*	547	U/L		100 – 1200
Occult Blood	Positive			Negative

Stool Chemistry: Inflammatory Markers

Lactoferrin

Ref: < 7.3 µg/mL

- Antimicrobial protein secreted by neutrophils and epithelial cells
- Sensitive biomarker for differentiating IBD from IBS
- Increases 2–3 weeks prior to clinical relapse in IBD
- Decreases during remission and effective treatment
- Can be non-specific: infection, irritation, alcohol intake

Lysozyme

Ref: ≤ 500 ng/mL

- Enzyme secreted at sites of GI inflammation
- Moderate elevations: significant enteropathogen overgrowth
- Marked elevations: IBD (Crohn's, UC) and diarrheal diseases
- Helps differentiate pathogen-induced inflammation vs. IBD
- If markedly elevated, assess calprotectin and lactoferrin

Calprotectin

Ref: < 80 µg/g

- Calcium-binding protein released from neutrophils
- More specific for ulcerative colitis and Crohn's disease
- Stable in stool for up to one week at room temperature
- May be elevated in NSAID users and with liver disease
- Correlates well with endoscopic findings

Clinical Pearl: Lactoferrin and lysozyme can be non-specifically elevated from infection, irritation, or alcohol. Calprotectin is more specific for IBD. Use all three together for comprehensive inflammatory assessment. Elevated calprotectin >150 µg/g warrants GI referral consideration.

Stool Chemistry: Immune Function & Short Chain Fatty Acids

Secretory IgA (sIgA)

Ref: 30–275 mg/dL

- First line of mucosal immune defense
- Low (<30): Chronic stress, chronic infections, immune suppression; may correlate with increased intestinal permeability
- High (>275): Active infection or dysbiosis; appropriate immune response to antigens; may remain elevated 6+ weeks post-viral
- Support low sIgA with colostrum and IgG supplements

Short Chain Fatty Acids

Total SCFAs	**5.0–16.0 mg/mL**
Butyrate (abs)	**0.8–4.0 mg/mL**
Acetate	**50–72%**
Propionate	**11–25%**
Butyrate	**11–32%**
Valerate	**0.8–5.0%**

Butyrate: colonocyte energy, anti-inflammatory, barrier integrity. Elevated acetate/propionate percentages may reflect shifts in bacterial populations — interpret in context of overall microbiome.

Stool pH

Ref: 5.8–7.0

- Largely dependent on fermentation of fiber by beneficial flora
- Low (<5.8): Increased fermentation, rapid transit, bacterial overgrowth; may indicate insufficient pancreatic bicarbonate
- High (>7.0): Rare; may indicate insufficient fermentation, SIBO, or low HCl

Beta-Glucuronidase

*Ref: 2,800–8,000 U/h*g*

- Breaks bond between glucuronic acid and toxins
- High: Increased toxin load, dysbiosis, hormone recirculation (especially estrogen); consider calcium D-glucarate
- Low: May indicate gallbladder insufficiency

Intestinal Permeability Assessment

Zonulin Family Protein

GI 360 ADD-ON — must be specifically ordered

- Regulates tight junctions between intestinal cells
- Limited utility as both rule-in AND rule-out test
- Only positive during active tight junction breakdown
- Positive = confirms active permeability; Negative does NOT exclude it
- Most sensitive when consuming trigger foods (e.g., gluten)
- Particularly useful with HLA-DQ2/DQ8 (Celiac markers)

Indirect Permeability Indicators

- Low sIgA: Often correlates with barrier dysfunction
- Sustained inflammatory markers: Ongoing barrier damage
- Multiple food sensitivities: Frequently correlate with increased permeability
- Yeast + dysbiotic bacteria together: Strongly suggests barrier impairment
- Dysbiotic bacteria produce endotoxins that directly damage intestinal barrier

The Dysbiosis–Permeability Relationship

- The vast majority of patients with dysbiosis exhibit leaky gut concurrently
- Not all leaky gut patients have dysbiosis, but these conditions frequently co-exist
- Inflammation markers (calprotectin, lactoferrin, lysozyme) often correlate with increased permeability
- Clinical approach: Address dysbiosis and permeability simultaneously for optimal outcomes

HCl Challenge Test Protocols

Empty Stomach HCl Challenge

Gastritis Assessment

- Administer 2000 mg betaine HCl on empty stomach
- If irritation, burning, or discomfort occurs: → Positive for gastritis → Possible leaky gut / impaired mucosal barrier → Possible H. pylori infection → Immediate HCl supplementation is contraindicated
- Address underlying gastritis before implementing HCl therapy

Meal-Based HCl Challenge

Hypochlorhydria Assessment

- Administer 2000–3000 mg betaine HCl with protein meal
- Tolerates without discomfort = hypochlorhydria confirmed
- Absence of warmth/digestive sensation = insufficient acid
- Therapeutic supplementation protocol: → Start with 1 capsule (~650 mg) with protein meals → Increase by 1 cap/meal until mild warming → Reduce by 1 capsule = therapeutic dose → Patients often reduce dosage as digestion improves

Stool Test Correlations with HCl Status

- Low pancreatic elastase (<400 µg/g) often correlates with low HCl production
- Alkaline stool pH can indicate insufficient hydrochloric acid
- Undigested muscle fibers may indicate poor protein digestion from insufficient HCl
- Multiple food intolerances frequently correlate with hypochlorhydria
- Consider bile support with gallbladder insufficiency (low beta-glucuronidase may indicate this)

Follow-Up Testing Decision Framework

Scenario	Timing	Test	Rationale
Post-antimicrobial (dysbiosis)	≥21 days post-tx	**Microbiology Only**	Confirm bacterial/yeast eradication; most cost-effective
Post-pathogen treatment	≥21 days post-tx	**CSA+P**	Verify pathogen clearance + inflammation resolution
Post-parasite treatment	≥21 days post-tx	**CSA+P**	Microscopy + PCR to confirm parasite elimination
Persistent symptoms despite tx	Any time	**GI 360 (full)**	New pathology may have emerged; reassess completely
New GI symptoms after resolution	Any time	**GI 360 (full)**	Reinfection, new organisms, or relapse possible
Stable improvement, routine check	3–6 months	**Microbiology Only**	Cost-effective monitoring of flora balance

CRITICAL: NEVER retest with PCR sooner than 21 days post-treatment — lingering DNA creates false positives and unnecessary re-treatment

Let me tell you a personal story….

Clostridioides difficile: Clinical Overview

What is C. difficile?

- Gram-positive, spore-forming anaerobic bacterium
- Leading cause of healthcare-associated diarrhea
- Produces Toxin A (enterotoxin) and Toxin B (cytotoxin)
- Some hypervirulent strains produce binary toxin (CDT)
- Spores resistant to heat, acid, and standard disinfectants
- ~500,000 infections and ~29,000 deaths annually in the US

Toxin A

Enterotoxin — damages intestinal epithelium, causes fluid secretion, attracts neutrophils

Toxin B

Cytotoxin — 1,000x more potent than Toxin A, disrupts actin cytoskeleton, causes cell death

Binary Toxin

CDT — found in hypervirulent strains (e.g., ribotype 027); increases adhesion and colonization

Primary Risk Factors

Antibiotic Exposure

Fluoroquinolones, clindamycin, cephalosporins, carbapenems

Healthcare Settings

Hospital stays, long-term care, shared medical equipment

Immune Compromise

Elderly, immunosuppressed, cancer therapy, IBD

PPI Use

Reduced gastric acid barrier allows spore survival

Women at Higher Risk

Peripartum antibiotics, hormonal fluctuations, increased healthcare contact

Recurrence

20–25% recurrence rate after first episode; increases with subsequent episodes

C. difficile: Detection Methods & DD Testing Options

Molecular (NAAT/PCR)

✓ Highest sensitivity (~95%); rapid turnaround

✗ Detects DNA, not viable organisms; cannot distinguish active infection from colonization

Antigen (GDH)

✓ Fast screening; detects glutamate dehydrogenase enzyme

✗ Not toxin-specific; positive GDH requires confirmatory toxin test

Toxin EIA

✓ Detects actual toxins A and B; indicates active disease

✗ Lower sensitivity (~75%); false negatives possible

Stool Culture

✓ Gold standard for organism identification; enables susceptibility testing

✗ Slow (48–96 hrs); requires anaerobic conditions

DD Testing: GI 360 PCR detects C. difficile Toxin A/B. For positive results, follow up with Comprehensive Clostridium Culture (add-on) to confirm viable organisms and obtain susceptibility data. PCR detection alone may indicate carrier status rather than active infection.

C. difficile: Asymptomatic Carriers & Organic Acid Markers

~38%

of C. diff carriers progress to symptomatic disease

Carrier Management Considerations

- Antibiotic-based eradication: Reduces carrier rates but may increase antimicrobial resistance
- Probiotic approach: Saccharomyces boulardii, B. longum show promise in reducing colonization
- Clinical decision depends on patient risk profile, immune status, and healthcare setting exposure
- Positive PCR without symptoms: Consider culture to confirm viable organisms before treating

Organic Acid Markers Indicative of C. diff / Dysbiosis

p-Hydroxyphenylacetic Acid (p-HPA):

Elevated in C. diff carriers; produced by Clostridium species metabolism of tyrosine. Key urinary OAT marker.

Phenylacetic Acid:

Associated with anaerobic bacterial overgrowth; elevated in SCFA-producing Clostridial metabolism.

p-Cresol:

Produced by C. difficile; inhibits growth of competing bacteria, providing C. diff selective advantage.

Short Chain Fatty Acid Disruption:

C. diff disrupts normal SCFA patterns, particularly butyrate production, which impairs colonocyte energy and barrier integrity.

Normal Bile Acid Metabolism & C. diff Resistance

- Primary bile acids are made by the liver and released into the small intestine
- Gut bacteria (mainly in the colon) convert primary → secondary bile acids
- **Secondary bile acids inhibit C. diff growth and spore germination**
- A healthy microbiota with correct bile conversion = natural colonization resistance to C. diff
- This is why maintaining bile acid and gut microbiota health is critical to overall immune strength

Clinical Insight: The epithelial barrier reinforced by microbial metabolites (butyrate, indoles) works in concert with secondary bile acids to suppress C. diff — both systems must be intact.

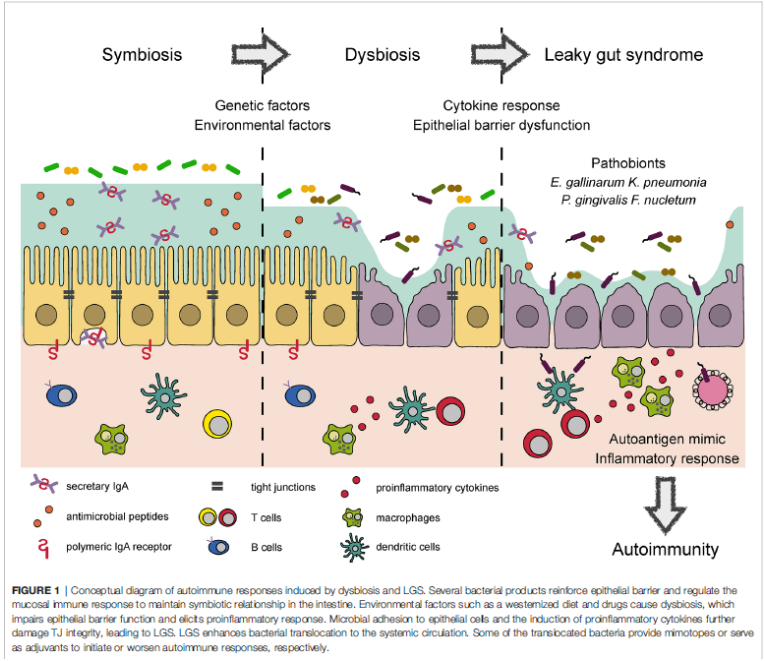


FIGURE 1 | Conceptual diagram of autoimmune responses induced by dysbiosis and LGS. Several bacterial products reinforce epithelial barrier and regulate the mucosal immune response to maintain symbiotic relationship in the intestine. Environmental factors such as a westernized diet and drugs cause dysbiosis, which impairs epithelial barrier function and elicits proinflammatory response. Microbial adhesion to epithelial cells and the induction of proinflammatory cytokines further damage TJ integrity, leading to LGS. LGS enhances bacterial translocation to the systemic circulation. Some of the translocated bacteria provide mimotopes or serve as adjuvants to initiate or worsen autoimmune responses, respectively.

Figure 1 | Kinashi & Hase, Front. Immunol., 2021

Frontiers in Immunology | Kinashi & Hase, 2021 • Cell Reports | Fachi et al., 2019

C. diff Infection Disrupts Bile Acid Balance

During C. diff Infection

- Antibiotics disrupt the microbiota, killing bacteria responsible for converting bile acids
- Primary bile acids accumulate in the colon and act as germination signals for C. diff spores
- **Secondary bile acids drop → loss of natural C. diff suppression**
- Simultaneously, butyrate-producing bacteria are decimated — HIF-1 stabilization fails and tight junctions weaken (Kinashi & Hase, 2021)
- **Result: a gut primed for C. diff colonization from both bile acid AND barrier disruption**

Even After Treatment Clears Infection

- The microbiota has NOT recovered — bile acid imbalance persists
- Surviving spores can germinate again, causing recurrent infection
- Tight junction integrity remains compromised — leaky gut syndrome amplifies the inflammatory cascade
- **This is why recurrence rates are so high after antibiotic-only treatment**
- The cycle continues until the underlying microbiota and bile acid balance is restored

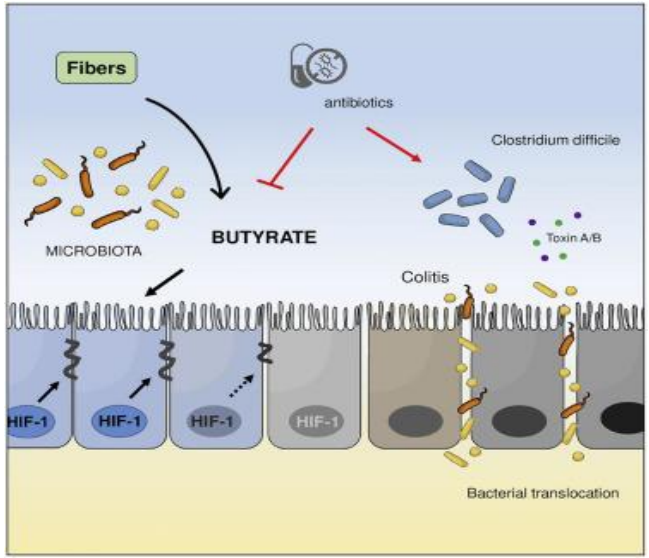
Butyrate: Bridging Barrier Function & C. diff Resistance

The Butyrate → HIF-1 → Tight Junction Pathway

- Colonocytes use butyrate as primary energy via beta-oxidation → consumes oxygen → stabilizes HIF-1
- HIF-1 upregulates Claudin-1 and Occludin expression → reinforces tight junctions
- **Fachi et al. (2019) demonstrated this specifically confers resistance to barrier disruption during C. difficile infection**
- Butyrate did NOT affect C. diff colonization or toxin production — it protected the epithelium from toxin damage

How Kinashi & Hase Connects This to C. diff

- Kinashi & Hase (2021) cited Fachi et al. directly, confirming butyrate upregulates Claudin-1 and Occludin via HIF-1, conferring resistance to C. difficile
- They further showed that loss of butyrate-producing species (e.g. F. prausnitzii) during dysbiosis is a key driver of barrier dysfunction — the same dysbiosis that enables C. diff



Graphical Abstract — Fachi et al., Cell Reports, 2019

The Dysbiosis Cycle: Why C. diff Keeps Coming Back

1. Antibiotic Trigger

- Kills bile acid-converting bacteria (e.g. C. scindens)
- Kills butyrate producers (e.g. F. prausnitzii)
- **Loss of BOTH barrier systems simultaneously**

→

2. Dual Vulnerability

- Primary bile acids accumulate → C. diff spore germination
- No butyrate → HIF-1 destabilized → tight junctions fail
- **Leaky gut syndrome develops**

→

3. C. diff Colonizes

- Toxin production → epithelial damage
- Inflammatory cascade worsens barrier
- **Bacterial translocation triggers systemic inflammation**

The Recurrence Loop — Why Antibiotics Alone Fail

- Treatment clears C. diff vegetative cells, but surviving spores remain dormant in the colon
- **Microbiota has not recovered → bile acid imbalance persists → butyrate production still suppressed → barrier still compromised**
- Spores germinate again in the same favorable environment → recurrent CDI

Restoring Balance: Breaking the Recurrence Cycle

Probiotics & Fecal Microbiota Transplant (FMT)

- Restores bacteria that convert primary → secondary bile acids (re-establishes bile acid-mediated C. diff suppression)
- Restores butyrate-producing species → HIF-1 stabilization → tight junction repair (Kinashi & Hase mechanism)
- **This dual restoration is why FMT is so effective for recurrent CDI**

Butyrate Supplementation

- Directly supports the HIF-1 pathway even before full microbiota recovery
- Fachi et al. showed butyrate reduced intestinal permeability and bacterial translocation in C. diff-infected mice
- **Can serve as a bridge therapy to protect the barrier while the microbiota rebuilds**

Key Takeaway: Treating the infection alone is not enough. Successful recovery requires restoring BOTH bile acid metabolism AND epithelial barrier function — this means rebuilding the microbiota.

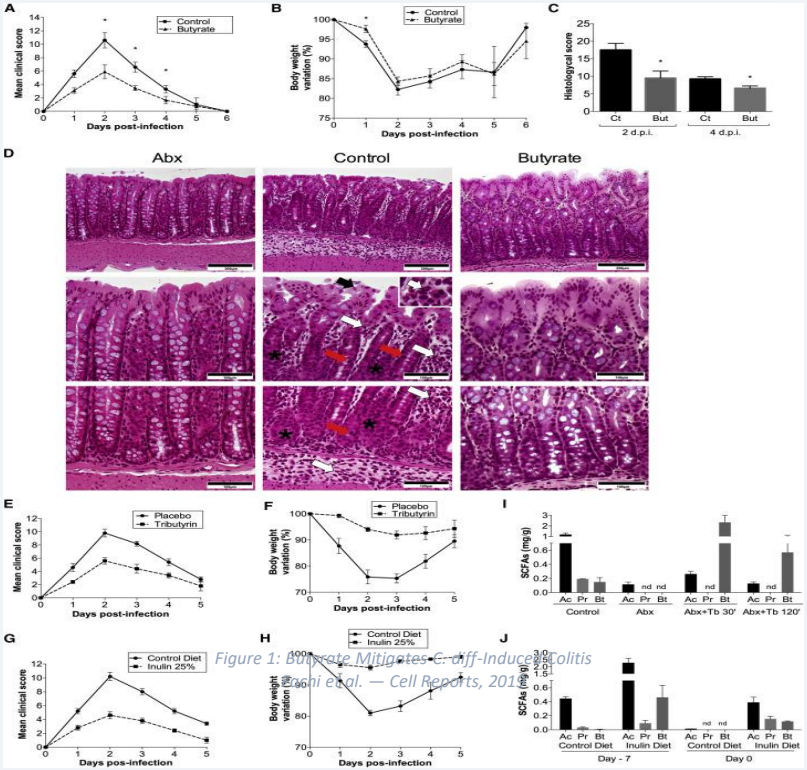


Figure 1: Butyrate Mitigates C. diff-Induced Colitis — Fachi et al. — Cell Reports, 2019

C. difficile: Functional Medicine Treatment Approaches

Probiotic Strategies

- Saccharomyces boulardii: Direct C. diff inhibition, protease that degrades toxin A receptor
- Bifidobacterium longum: Demonstrated C. diff growth inhibition (Frontiers Microbiology 2023)
- Lactobacillus reuteri: Bile acid metabolism support, colonization resistance
- Multi-strain combinations post-treatment for recolonization

Supplemental Support

- Butyrate supplementation: Protects colonocytes, enhances bile acid metabolism
- Cholestyramine: Binds C. diff toxins A and B in the gut lumen
- Black seed oil + bentonite clay: Case report showed resolution (PMC9168092)
- Colostrum / IgG: Immune barrier restoration

Bile Acid & Barrier

- C. diff disrupts primary → secondary bile acid conversion
- Primary bile acids (taurocholate) promote C. diff spore germination
- Secondary bile acids (deoxycholate) inhibit C. diff growth
- Restore bile acid homeostasis through microbiome rebalancing

Fecal Microbiota Transplantation (FMT): Emerging therapy for recurrent C. diff with >90% efficacy in clinical trials. Restores microbial diversity and colonization resistance. FDA-approved product (Vowst/SER-109) now available as oral capsules for recurrent CDI.

Comprehensive Dysbiosis Protocol

1 Biofilm Disruption

2–4 weeks

- NAC (N-acetyl cysteine)
- Enzymes: nattokinase, serrapeptase
- EDTA chelation
- Implement BEFORE antimicrobials

2 Antimicrobial Phase

4–8 weeks

- Select agents based on susceptibility results
- Rotate protocols for resistant organisms
- Natural + Rx combination when indicated
- Monitor clinical response

3 Gut Healing

Ongoing

- L-glutamine: primary enterocyte fuel
- Zinc carnosine: tight junction integrity
- Aloe vera: anti-inflammatory
- Colostrum: IgG and growth factors

4 Recolonization

Ongoing

- S. boulardii during antimicrobial phase
- Multi-strain Lactobacillus + Bifidobacterium post-protocol
- Soil-based organisms for dysbiosis
- Reduced fermentable carbs during active phase

Inflammation Support: Elevated calprotectin → curcumin, boswellia | Elevated lactoferrin → quercetin, omega-3s | Mucus present → address underlying irritants

Key Takeaways

1. Never interpret the Dysbiosis Index in isolation — always integrate with culture, parasitology, and stool chemistries
2. DD uses four microbiology categories: Expected, Imbalanced, Dysbiotic, and Pathogenic — treatment thresholds differ by category
3. PCR detects DNA, not viable organisms: wait ≥21 days post-treatment before retesting to avoid false positives
4. Blastocystis and D. fragilis are detected by microscopy, not PCR — three-day collection is essential for sensitivity
5. The GI 360 offers targeted add-ons: H. pylori Stool Antigen, Zonulin, Comprehensive Clostridium Culture
6. Progressive testing (GI 360 → CSA+P → Micro Only) is the most cost-effective monitoring strategy
7. The gut–brain axis via SCFAs directly impacts neurotransmitter function, neuroinflammation, and epigenetics
8. Always address the gut first — systemic and neurological conditions often do not resolve until GI pathology is treated

CASE STUDY

23 yr old Female with Systemic and Neurological Inflammation

Case Study: Mae — 23-Year-Old Female with Systemic & Neurological Inflammation

Presenting Complaints

- Severe depression, anxiety, ADHD
- Weight gain, fatigue, excessive sweating
- Nausea, bloating, intermittent vomiting
- Irregular periods, severe PMDD
- Chronic pain: low back, neck, shoulders
- Unable to hold a job or attend school

Current Medications

- Prozac, Wellbutrin, Remeron (psychiatric)
- Vyvanse (ADHD)
- Diclofenac, Gabapentin (pain — very little relief)
- Hormonal IUD (some PMDD relief, still variable)
- Tried: Ketamine (short-lived relief)
- Experimented: marijuana, psilocybin, MDMA (temporary only)

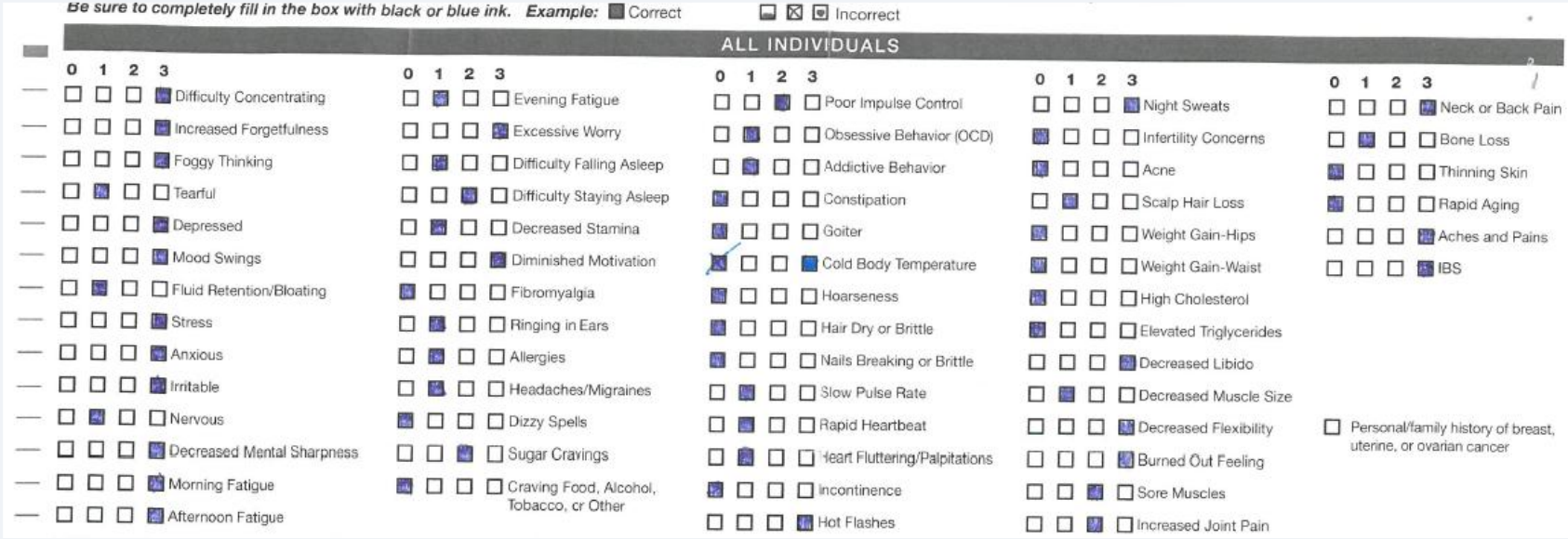
Relevant History

- Split family; not extensive trauma
- Parents not very healthy at conception
- Mother prone to anxiety; stressful pregnancy
- Consider epigenetic inheritance factors
- Poly-pharmacy creating additional burden

FM Diagnostic Framework

- Nutritional deficiencies
- Leaky gut / dysbiosis → systemic inflammation
- High viral load assessment
- Mitochondrial dysfunction
- ANS imbalance — sympathetic overload
- MAO/COMT activation; methylation abnormalities
- SNPs likely playing a role

Case Study - Mae

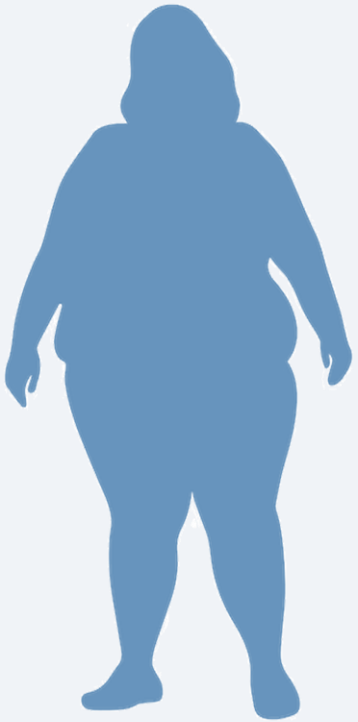


Be sure to completely fill in the box with black or blue ink. ***Example:*** ■ Correct ☒ Incorrect

ALL INDIVIDUALS

0 1 2 3		0 1 2 3		0 1 2 3		0 1 2 3		0 1 2 3	
☐ ☐ ☐ ■	Difficulty Concentrating	☐ ■ ☐ ☐	Evening Fatigue	☐ ☐ ■ ☐	Poor Impulse Control	☐ ☐ ☐ ■	Night Sweats	☐ ☐ ☐ ■	Neck or Back Pain
☐ ☐ ☐ ■	Increased Forgetfulness	☐ ☐ ☐ ■	Excessive Worry	☐ ■ ☐ ☐	Obsessive Behavior (OCD)	■ ☐ ☐ ☐	Infertility Concerns	☐ ■ ☐ ☐	Bone Loss
☐ ☐ ☐ ■	Foggy Thinking	☐ ■ ☐ ☐	Difficulty Falling Asleep	☐ ■ ☐ ☐	Addictive Behavior	■ ☐ ☐ ☐	Acne	■ ☐ ☐ ☐	Thinning Skin
☐ ■ ☐ ☐	Tearful	☐ ☐ ■ ☐	Difficulty Staying Asleep	■ ☐ ☐ ☐	Constipation	☐ ■ ☐ ☐	Scalp Hair Loss	■ ☐ ☐ ☐	Rapid Aging
☐ ☐ ☐ ■	Depressed	☐ ■ ☐ ☐	Decreased Stamina	■ ☐ ☐ ☐	Goiter	■ ☐ ☐ ☐	Weight Gain-Hips	☐ ☐ ☐ ■	Aches and Pains
☐ ☐ ☐ ■	Mood Swings	☐ ☐ ☐ ■	Diminished Motivation	☒ ☐ ☐ ■	Cold Body Temperature	■ ☐ ☐ ☐	Weight Gain-Waist	☐ ☐ ☐ ■	IBS
☐ ■ ☐ ☐	Fluid Retention/Bloating	■ ☐ ☐ ☐	Fibromyalgia	■ ☐ ☐ ☐	Hoarseness	■ ☐ ☐ ☐	High Cholesterol		
☐ ☐ ☐ ■	Stress	☐ ■ ☐ ☐	Ringing in Ears	■ ☐ ☐ ☐	Hair Dry or Brittle	■ ☐ ☐ ☐	Elevated Triglycerides		
☐ ☐ ☐ ■	Anxious	☐ ■ ☐ ☐	Allergies	■ ☐ ☐ ☐	Nails Breaking or Brittle	☐ ☐ ☐ ■	Decreased Libido		
☐ ☐ ☐ ■	Irritable	☐ ■ ☐ ☐	Headaches/Migraines	☐ ■ ☐ ☐	Slow Pulse Rate	☐ ■ ☐ ☐	Decreased Muscle Size		
☐ ■ ☐ ☐	Nervous	■ ☐ ☐ ☐	Dizzy Spells	☐ ■ ☐ ☐	Rapid Heartbeat	☐ ☐ ☐ ■	Decreased Flexibility	☐	Personal/family history of breast, uterine, or ovarian cancer
☐ ☐ ☐ ■	Decreased Mental Sharpness	☐ ☐ ☐ ■	Sugar Cravings	☐ ■ ☐ ☐	Heart Fluttering/Palpitations	☐ ☐ ☐ ■	Burned Out Feeling		
☐ ☐ ☐ ■	Morning Fatigue	■ ☐ ☐ ☐	Craving Food, Alcohol, Tobacco, or Other	■ ☐ ☐ ☐	Incontinence	☐ ☐ ■ ☐	Sore Muscles		
☐ ☐ ☐ ■	Afternoon Fatigue			☐ ☐ ☐ ■	Hot Flashes	☐ ☐ ■ ☐	Increased Joint Pain		

Case study - Mae



Some things to consider when treating from a Functional Medicine perspective:

- What is your FM approach?
- What do you know so far about what is really happening inside her mind and body?
- What labs do you want to run?
- What treatments are you thinking about?
- What are the challenges to a pt with motivation, but no ability, or plenty of ability, but no motivation?
- How do you manage the poly-pharmacy?

TESTS	RESULT	FLAG	UNITS	REFERENCE INTERVAL
CMP14+LP+TP+TSH+7AC+CBC/D/P...				
Chemistries				
Glucose	93		mg/dL	65 - 99
Hemoglobin A1c	5.1		%	4.8 - 5.6
Please Note: Prediabetes: 5.7 - 6.4 Diabetes: >6.4 Glycemic control for adults with diabetes: <7.0				
Uric Acid	5.3		mg/dL	2.4 - 6.3
Please Note: Therapeutic target for gout patients: <6.0				
BUN	9		mg/dL	5 - 18
Creatinine	0.83		mg/dL	0.57 - 1.00
eGFR If NonAfricn Am				>59
Unable to calculate GFR. Age and/or sex not provided or age <18 years old.				
eGFR If Africn Am				>59
Unable to calculate GFR. Age and/or sex not provided or age <18 years old.				
BUN/Creatinine Ratio	11			10 - 22
Sodium	141		mmol/L	134 - 144
Potassium	4.0		mmol/L	3.5 - 5.2
Chloride	102		mmol/L	96 - 106
Carbon Dioxide, Total	22		mmol/L	20 - 29
Calcium	9.6		mg/dL	8.9 - 10.4
Phosphorus	4.3		mg/dL	2.5 - 5.3
Magnesium, RBC [A]	6.0		mg/dL	4.2 - 6.8
Protein, Total	7.3		g/dL	6.0 - 8.5
Albumin	4.7		g/dL	3.5 - 5.5
Globulin, Total	2.6		g/dL	1.5 - 4.5
A/G Ratio	1.8			1.2 - 2.2

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Bilirubin, Total	0.4		mg/dL	0.0 - 1.2
Bilirubin, Direct	0.10		mg/dL	0.00 - 0.40
Alkaline Phosphatase	99		IU/L	45 - 101
Creatine Kinase,Total	70		U/L	24 - 173
LDH	185		IU/L	114 - 209
AST (SGOT)	17		IU/L	0 - 40
ALT (SGPT)	21		IU/L	0 - 24
GGT	14		IU/L	0 - 60
Iron Bind.Cap.(TIBC)	312		ug/dL	250 - 450
UIBC	243		ug/dL	131 - 425
Iron	69		ug/dL	26 - 169
Iron Saturation	22		%	15 - 55
Ferritin, Serum	49		ng/mL	15 - 77
Vitamin D, 25-Hydroxy	**28.3**	**Low**	ng/mL	30.0 - 100.0

Vitamin D deficiency has been defined by the Institute of Medicine and an Endocrine Society practice guideline as a level of serum 25-OH vitamin D less than 20 ng/mL (1,2). The Endocrine Society went on to further define vitamin D insufficiency as a level between 21 and 29 ng/mL (2).

1. IOM (Institute of Medicine). 2010. Dietary reference intakes for calcium and D. Washington DC: The National Academies Press.

2. Holick MF, Binkley NC, Bischoff-Ferrari HA, et al. Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. JCEM. 2011 Jul; 96(7):1911-30.

Cholesterol, Total	**201**	**High**	mg/dL	100 - 169
Triglycerides	**128**	**High**	mg/dL	0 - 89
HDL Cholesterol	40		mg/dL	>39
VLDL Cholesterol Cal	26		mg/dL	5 - 40
LDL Cholesterol Calc	**135**	**High**	mg/dL	0 - 109
LDL/HDL Ratio	**3.4**	**High**	ratio	0.0 - 3.2

Please Note:

LDL/HDL Ratio	Men	Women
1/2 Avg.Risk	1.0	1.5
Avg.Risk	3.6	3.2
2X Avg.Risk	6.2	5.0
3X Avg.Risk	8.0	6.1

C-Reactive Protein, Cardiac	**3.71**	**High**	mg/L	0.00 - 3.00

Relative Risk for Future Cardiovascular Event	
Low	**<1.00**
Average	**1.00 - 3.00**
High	**>3.00**

Homocyst(e)ine	11.2		umol/L	0.0 - 15.0

Thyroid				
TSH	0.889		uIU/mL	0.450 - 4.50
Thyroxine (T4)	8.5		ug/dL	4.5 - 12.0
T3 Uptake	28		%	23 - 35
Free Thyroxine Index	2.4			1.2 - 4.9
Triiodothyronine (T3)	115		ng/dL	71 - 180
Triiodothyronine (T3), Free	3.2		pg/mL	2.3 - 5.0
Reverse T3, Serum [A]	15.9		ng/dL	9.2 - 24.1
T4,Free(Direct)	1.40		ng/dL	0.93 - 1.60
Thyroid Peroxidase (TPO) Ab	9		IU/mL	0 - 26
Thyroglobulin Antibody	**5.4**	**High**	IU/mL	0.0 - 0.9

Thyroglobulin Antibody measured by Beckman Coulter Methodology

Immunoassay				
Sex Horm Binding Glob, Serum	43.2		nmol/L	24.6 - 122.
Serology/Immunology				
Antinuclear Antibodies, IFA	Negative			
				Negative <1:80
				Borderline 1:80
				Positive >1:80
RA Latex Turbid.	<10.0		IU/mL	0.0 - 13.9
Fibrinogen Activity	296		mg/dL	193 - 507
CBC, Platelet Ct, and Diff				
WBC	6.4		x10E3/uL	3.4 - 10.8
RBC	5.01		x10E6/uL	3.77 - 5.28
Hemoglobin	15.3		g/dL	11.1 - 15.9
Hematocrit	45.6		%	34.0 - 46.6
MCV	91		fL	79 - 97
MCH	30.5		pg	26.6 - 33.0
MCHC	33.6		g/dL	31.5 - 35.7
RDW	13.4		%	12.3 - 15.4
Platelets	222		x10E3/uL	150 - 450
Neutrophils	59		%	Not Estab.
Lymphs	32		%	Not Estab.
Monocytes	7		%	Not Estab.
Eos	2		%	Not Estab.
Basos	0		%	Not Estab.
Neutrophils (Absolute)	3.8		x10E3/uL	1.4 - 7.0
Lymphs (Absolute)	2.0		x10E3/uL	0.7 - 3.1

Case Study - Mae

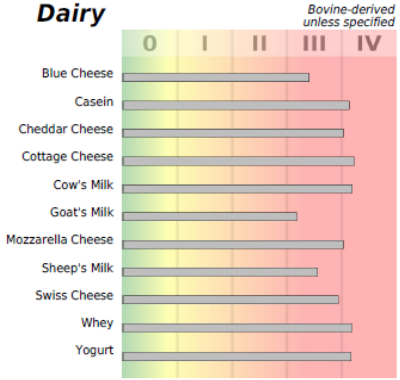
Monocytes(Absolute)	0.5		x10E3/uL	0.1 - 0.9
Eos (Absolute)	0.1		x10E3/uL	0.0 - 0.4
Baso (Absolute)	0.0		x10E3/uL	0.0 - 0.3
Immature Granulocytes	0		%	Not Estab.
Immature Grans (Abs)	0.0		x10E3/uL	0.0 - 0.1
Sedimentation Rate-Westergren	4		mm/hr	0 - 32
Urinalysis, Complete				
Urinalysis Gross Exam				
Specific Gravity	1.005			1.005 - 1.030
pH	6.5			5.0 - 7.5
Urine-Color	Yellow			Yellow
Appearance	Clear			Clear
WBC Esterase	**2+**	**Abnormal**		Negative
Protein	Negative			Negative/Trace
Glucose	Negative			Negative
Ketones	Negative			Negative
Occult Blood	Negative			Negative
Bilirubin	Negative			Negative
Urobilinogen,Semi-Qn	0.2		mg/dL	0.2 - 1.0
Nitrite, Urine	Negative			Negative
Microscopic Examination	See below:			
Microscopic was indicated and was performed.				
WBC	**6-10**	**Abnormal**	/hpf	0 - 5
RBC	0-2		/hpf	0 - 2
Epithelial Cells (non renal)	0-10		/hpf	0 - 10
Bacteria	Few			None seen/Few

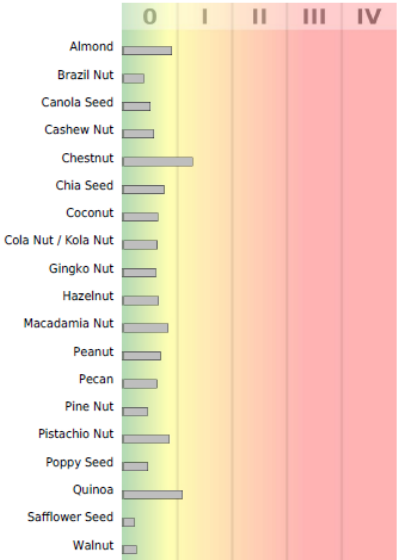
Case Study - Mae

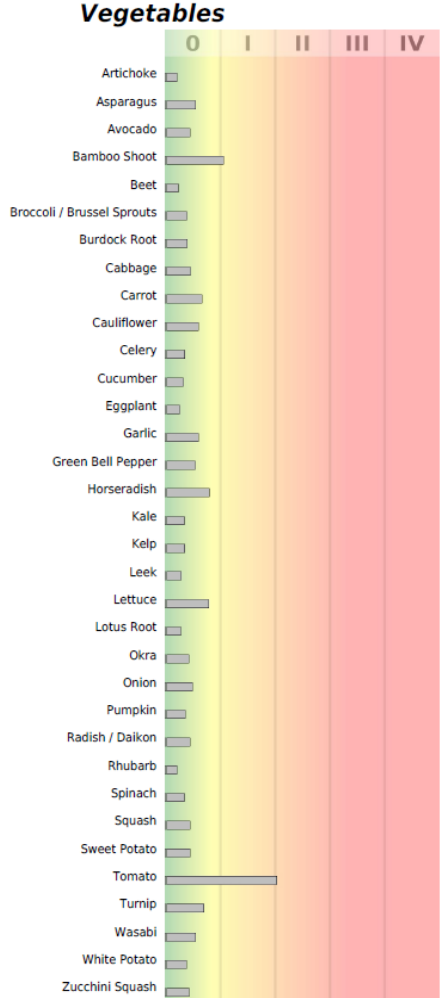
EBV Antibody Profile

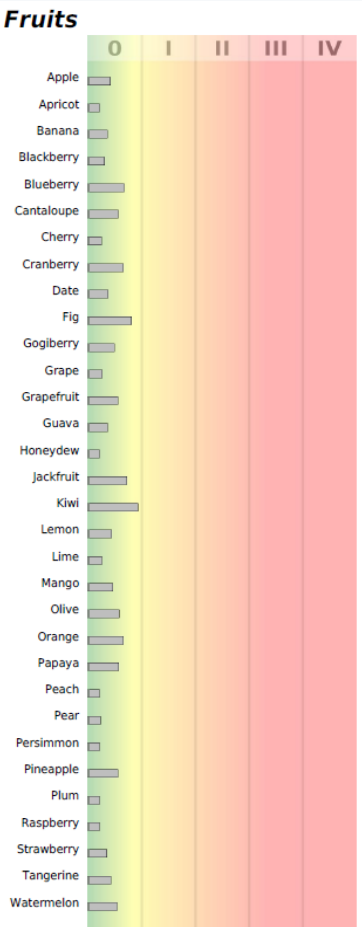
	Test	Current Result and Flag		Previous Result and Date	Units	Reference Interval
	EBV Ab VCA, IgM [01]	<36.0			U/mL	0.0-35.9
				Negative	<36.0	
				Equivocal	36.0 - 43.9	
				Positive	>43.9	
▲	**EBV Ab VCA, IgG** [01]	**387.0**	**High**		U/mL	0.0-17.9
				Negative	<18.0	
				Equivocal	18.0 - 21.9	
				Positive	>21.9	
▲	**EBV Nuclear Antigen Ab, IgG** [01]	**>600.0**	**High**		U/mL	0.0-17.9
				Negative	<18.0	
				Equivocal	18.0 - 21.9	
				Positive	>21.9	

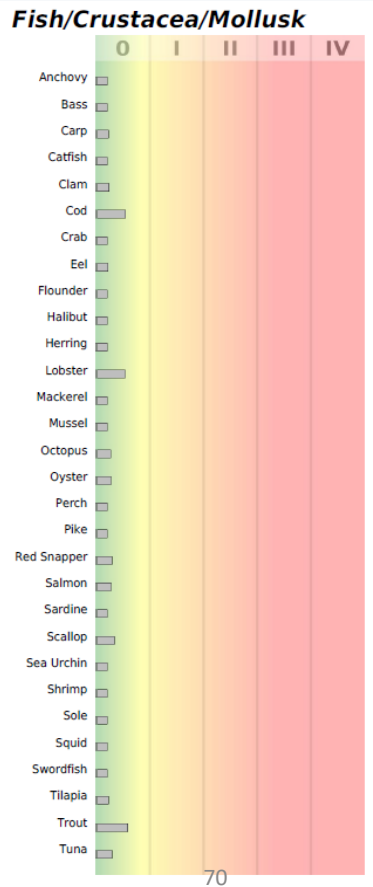
Case Study - Mae











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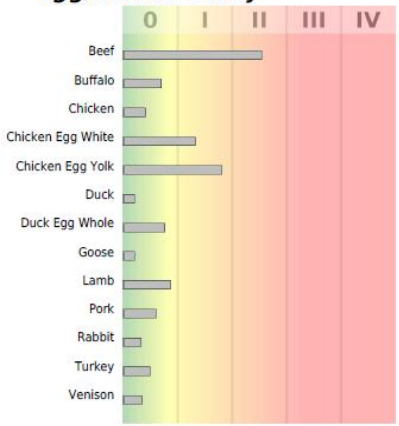
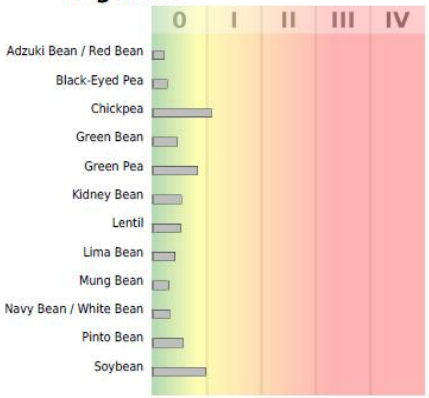
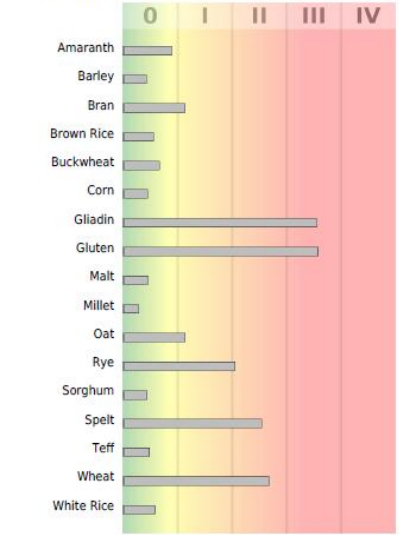
Egg/Meat/Poultry



Legumes

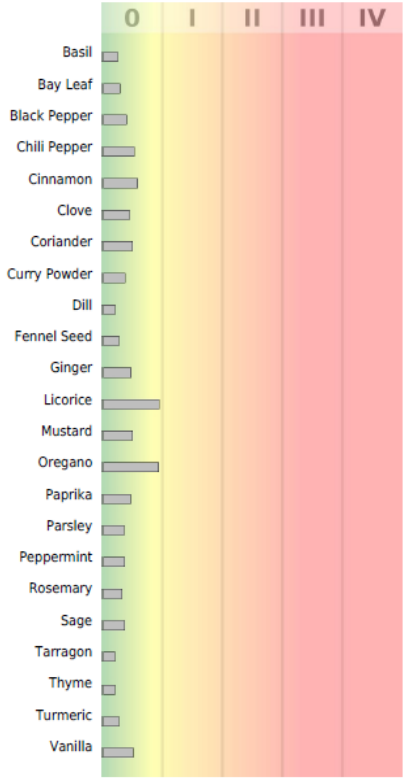


Grains

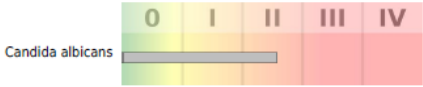


Miscellaneous

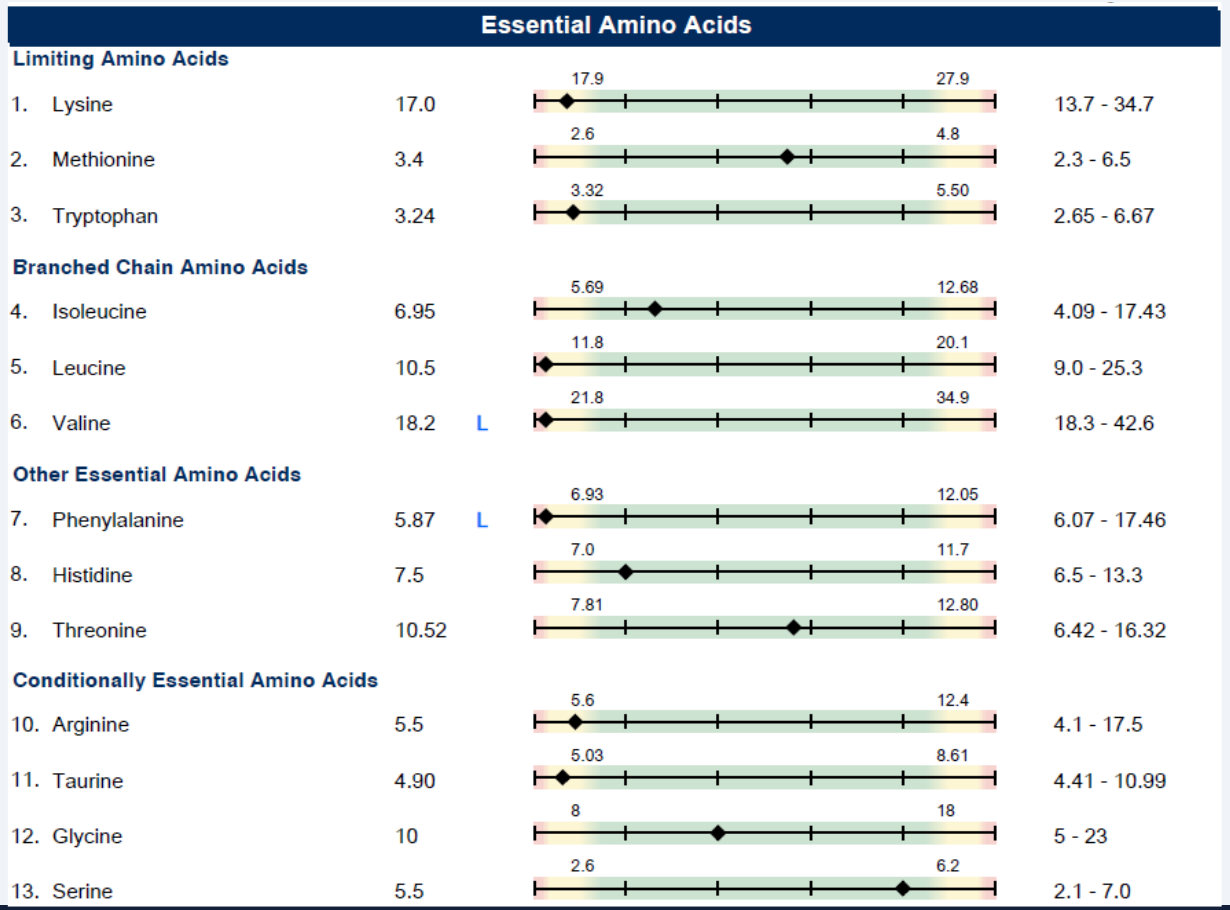
Herbs/Spices



Candida Screen



Case Study - Mae



Case Study - Mae

Vitamin B6 Status Markers			
14. α-aminoadipic acid	0.06		0.16
15. α-Amino-n-butyric acid (α-ANB)	4.73		5.65
16. γ-aminobutyric acid (GABA)	<DL		0.04
17. Cystathionine	<DL		0.06
Vascular Function			
18. Arginine	5.5		5.6 – 12.4
19. Taurine	4.90		5.03 – 8.61
20. α-aminoadipic acid	0.06		0.16
Neurotransmitters and Precursors			
21. Phenylalanine	5.87	L	6.93 – 12.05
22. Tyrosine	6.1		6.8 – 13.1
23. Tryptophan	3.24		3.32 – 5.50
24. Glutamic Acid	10.1		2.6 – 10.3
25. Taurine	4.90		5.03 – 8.61
Sulfur Amino Acids (Glutathione - related)			
26. Methionine	3.4		2.6 – 4.8
27. Cystathionine	<DL		0.06
28. Cyst(e)ine	7.7		6.9 – 15.0
29. Taurine	4.90		5.03 – 8.61

Case Study - Mae

Urea Cycle and Ammonia Detoxification

30. Urea	194	L	359 – 756	216 - 1,156
31. Arginine	5.5		5.6 – 12.4	4.1 - 17.5
32. Citrulline	1.7		2.0 – 4.0	1.6 - 5.7
33. Ornithine	7.77		4.86 – 10.61	4.38 - 15.42
34. Glutamine	53		50 – 87	41 - 111
35. Asparagine	5.6		3.9 – 8.3	3.5 - 11.6
36. Aspartic Acid	<DL		0.60	<= 0.67

Glycine, Serine and Related Amino Acids

37. Alanine	25		26 – 49	19 - 62
38. Glycine	10		8 – 18	5 - 23
39. Sarcosine	0.04		0.09	<= 0.15
40. Serine	5.5		2.6 – 6.2	2.1 - 7.0
41. Phosphoserine	<DL		0.39	<= 0.39
42. Ethanolamine	0.40		0.55	0.19 - 0.78
43. Phosphoethanolamine	0.11		0.37	0.09 - 0.57

Collagen - Related Amino Acids

44. Proline	17		17 – 40	11 - 57
45. Lysine	17.0		17.9 – 27.9	13.7 - 34.7

Case Study - Mae

Homocysteine Assay - Plasma

Methodology: Enzymatic Assay

1.	Homocysteine	11.3	H	3.7 - 10.4 umol/L

Nutrient & Toxic Elements Profile - Blood

Methodology: Inductively Coupled Plasma/Mass Spectrometry

Nutrient Elements

Erythrocytes (packed cells)

1.	Potassium	3,736	H	2,725 – 3,626 — 2,220 - 3,626 mcg/g
2.	Magnesium	55.7		38.5 – 56.5 — 30.1 - 56.5 mcg/g

Plasma

3.	Zinc	114.0		81.5 – 159.4 — 64.3 - 159.4 mcg/dL
4.	Copper	138.7		92.9 – 192.0 — 75.3 - 192.0 mcg/dL

Whole Blood

5.	Selenium	202		134 – 330 — 109 - 330 mcg/L
6.	Manganese	8.2		5.5 – 16.5 — 3.0 - 16.5 mcg/L

Toxic Elements

Whole Blood

7.	Arsenic	<DL		<= 13.7 mcg/L
8.	Cadmium	<DL		<= 1.22 mcg/L
9.	Lead	<DL		<= 2.81 mcg/dL
10.	Mercury	1.09		<= 4.35 mcg/L

Case Study - Mae

Coenzyme Q10 Plus Vitamins Profile - Serum

Methodology: High-pressure liquid chromatography (HPLC), LC/MS/MS

		Results		Reference Range
1.	Coenzyme Q10	1.06		0.43 - 1.49 mcg/mL
2.	alpha-Tocopherol	10.3		5.9 - 19.4 mg/L
3.	gamma-Tocopherol	1.0		0.7 - 4.9 mg/L
4.	Vitamin A (Retinol)	35.5		18.9 - 57.3 mcg/dL
5.	β-Carotene	11		3 - 91 mcg/dL

Glutathione Assay - Whole Blood

Methodology: Colorimetric

		Results umol/L		Reference Range
6.	Glutathione	1,045		>= 669 umol/L

DNA/Oxidative Stress Marker (8-OHdG) Assay - Urine

Methodology: LC/MS/MS, TBARS (thiobarbituric acid reactive substances), Hexokinase/G-6-PDH

		Results		Reference Range
7.	Lipid Peroxides	5.7		<= 10.0 umol/g creatinine
8.	8-Hydroxy-2-deoxyguanosine	8		<= 15 mcg/g creatinine

Vitamin D Profile - Serum

Methodology: Chemiluminescent

		Results ng/mL		Reference Range
9.	25 - Hydroxyvitamin D	20	L	30-100 ng/mL

Case Study - Mae

Polyunsaturated Omega-3

1.	Alpha Linolenic (18:3n3)	<DL	L	0.11	>= 0.09
2.	Eicosapentaenoic (20:5n3)	0.32		0.22	>= 0.16
3.	Docosapentaenoic (22:5n3)	1.44		1.40	>= 1.14
4.	Docosahexaenoic (22:6n3)	3.0		2.7	>= 2.1
5.	Total Omega-3 %	4.8		4.4	>= 3.8

Polyunsaturated Omega-6

6.	Linoleic (18:2n6)	11.3		11.5 – 16.2	10.5 - 16.9
7.	Gamma Linolenic (18:3n6)	0.09		0.04 – 0.10	0.03 - 0.13
8.	Eicosadienoic (20:2n6)	0.27	H	0.24	<= 0.26
9.	Dihomogamma Linolenic (20:3n6)	1.10	L	1.22	>= 1.19
10.	Arachidonic (20:4n6)	17		16 – 20	15 - 21
11.	Docosatetraenoic (22:4n6)	2.61		2.00 – 3.82	1.50 - 4.20
12.	Total Omega-6 %	32.9		33.5 – 38.6	30.5 - 39.7

Case Study - Mae

Nutrient Markers

Fatty Acid Metabolism
(Carnitine & B2)

1.	Adipate	1.5	2.3	<= 2.8
2.	Suberate	0.6	1.7	<= 2.1

Carbohydrate Metabolism
(B1, B3, Cr, Lipoic Acid, CoQ10)

3.	Pyruvate	14	20	7 - 32
4.	Lactate	10.3	16.7	1.9 - 19.8
5.	β-Hydroxybutyrate	2.9 H	2.2	<= 2.8

Energy Production (Citric Acid Cycle)
(B Comp., CoQ10, Amino Acids, Mg)

6.	Citrate	457	370	40 - 520
7.	Cis-Aconitate	18	22	10 - 36
8.	Isocitrate	59	65	22 - 65
9.	α-Ketoglutarate	45	27	4 - 52
10.	Succinate	3.2	3.4	0.4 - 4.6
11.	Malate	4.2 H	3.0	<= 3.0
12.	Hydroxymethylglutarate	5	14	<= 15

Case Study - Mae

Cell Regulation Markers

Neurotransmitter Metabolism Markers
(Tyrosine, Tryptophan, B6, Antioxidants)

23. Vanilmandelate	1.8		1.0 – 2.5	0.4 - 3.6
24. Homovanillate	1.9		1.7 – 3.3	1.2 - 5.3
25. 3-Methyl-4-Hyroxyphenylglycol	0.05		0.05 – 0.17	0.02 - 0.22
26. 5-Hydroxyindoleacetate	6.5		4.9 – 12.1	3.8 - 12.1
27. Kynurenate	1.6		6.0	<= 7.1
28. Quinolinate	8.6		9.1	<= 9.1
29. Kynurenate/Quinolinate	0.19	L	0.72	>= 0.44
30. Xanthurenate	0.12		0.77	<= 0.96

Oxidative Damage and Antioxidant Markers
(Vitamin C and Other Antioxidants)

31. 8-Hydroxy-2-deoxyguanosine	8		9	<= 15

(Units for 8-hydroxy-2-dexoyguanosine are mcg/g creatinine)

Toxicants and Detoxification

Detoxification Indicators
(Arg, NAC, Met, Mg, Antioxidants)

32. Orotate	0.58		0.64	0.33 - 1.01
33. α-Hydroxybutyrate	0.53		0.60	<= 0.83
34. Pyroglutamate	25		30	16 - 34
35. α-Hydroxyisobutyrate	4.1		5.4	<= 6.7
36. α-Ketophenylacetate	0.12		0.30	<= 0.46

Case Study - Mae

Compounds of Bacterial or Yeast/Fungal Origin

Bacterial - General

37. Benzoate	>0.56	H	0.05	<= 0.05
38. Hippurate	61		329	<= 603
39. Phenylacetate	0.06		0.08	<= 0.12
40. Indoleacetate	0.9		2.4	<= 4.2
41. p-Hydroxyphenylacetate	5		25	<= 29
42. m-Hydroxyphenylacetate	6.0		4.6	<= 8.1

Clostridial Species

43. 3,4-Dihydroxyphenylpropionate	1.3		2.9	<= 5.3

Yeast / Fungal

44. D-Arabinitol	16		32	<= 36
45. Citramalate	2.9		3.3	<= 5.8
46. Tartarate	7		7	<= 15

Oxalate Markers

Oxalates

47. Glycerate	12.4		13.6	3.5 - 16.4
48. Glycolate	53		43	<= 67
49. Oxalate	20		40	<= 78

Case Study - Mae

Doctor: Brandon Lundell, DC
Harmony Healing Center
714 Kimbark St
Longmont, CO 80501 USA

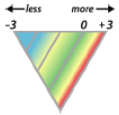
Sex: Female

Date Reported	11/17/2022
Specimens Collected	3

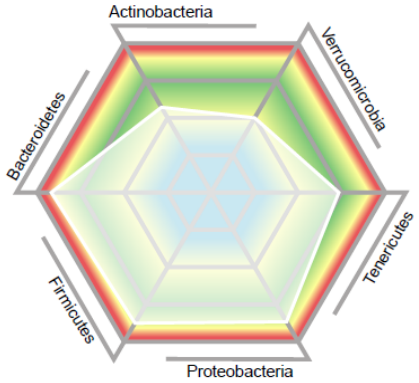
Microbiome Abundance and Diversity Summary

The abundance and diversity of gastrointestinal bacteria provide an indication of gastrointestinal health, and gut microbial imbalances can contribute to dysbiosis and other chronic disease states. The GI360™ Microbiome Profile is a gut microbiota DNA analysis tool that identifies and characterizes more than 45 targeted analytes across six Phyla using PCR and compares the patient results to a characterized normobiotic reference population. The web chart illustrates the degree to which an individual's microbiome profile deviates from normobiosis.

LEGEND



The web image shows the relative diversity and balance among bacteria belonging to the six primary Phyla. The white shaded area represents the patient's results compared to a normobiotic reference population. The center of the web represents less abundance while the outer edges represent more than normobiotic.



Dysbiosis and Diversity Index

These indexes are calculated from the results of the Microbiome Profile, with scores ranging from 1 to 5, and do not include consideration of dysbiotic and pathogenic bacteria, yeast, parasites and viruses that may be reported in subsequent sections of the GI360™ test.

The Dysbiosis Index the (DI) is calculated strictly from the results of the Microbiome Profile, with scores from 1 to 5. A DI score above 2 indicates dysbiosis; a microbiota profile that differs from the defined normobiotic reference population. The higher the DI above 2, the more the sample deviates from the normobiotic profile. The dysbiosis test and DI does not include consideration of dysbiotic and pathogenic bacteria, yeast, parasites and viruses that may be reported in subsequent sections of the GI360™ test.

A diversity score of 3 indicates an expected amount of diversity, with 4 & 5 indicating an increased distribution of bacteria based on the number of different species and their abundance in the sample, calculated based on Shannon's diversity index. Scores of 1 or 2 indicate less diversity than the defined normobiotic reference population.

Dysbiosis Index

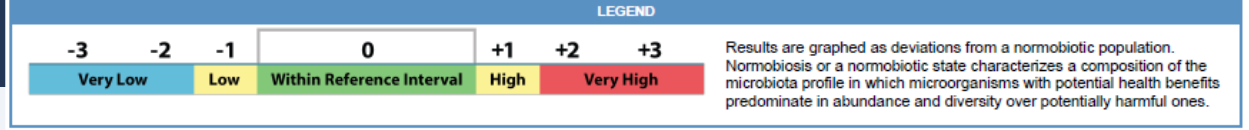
Diversity Score

Key Findings

Butyrate producing bacteria		Total SCFA's, Low
Gut barrier protective bacteria		*Candida albicans*, Cultured
Gut intestinal health marker		
Pro-inflammatory bacteria		
Gut barrier protective bacteria vs. opportunistic bacteria		

= Expected = Imbalanced

Case Study - Mae



Actinobacteria	Result	Reference Interval
Actinobacteria	0	0
Actinomycetales	-1	0
Bifidobacterium spp.	0	0

Bacteroidetes	Result	Reference Interval
Alistipes spp.	+2	0
Alistipes onderdonkii	+3	0
Bacteroides fragilis	0	0
Bacteroides spp. & *Prevotella* spp.	0	0
Bacteroides spp.	0	0
Bacteroides pectinophilus	0	0
Bacteroides stercoris	0	0
Bacteroides zoogleoformans	0	0
Parabacteroides johnsonii	+1	0
Parabacteroides spp.	+1	0

Firmicutes	Result	Reference Interval
Firmicutes	0	0
Bacilli Class	0	0
Catenibacterium mitsuokai	0	0

Case Study - Mae

Firmicutes	Result	-3 -2 -1 0 +1 +2 +3	Reference Interval
Clostridia Class	-1		0
Clostridium methylpentosum	0		0
Clostridium L2-50	0		0
Coprobacillus cateniformis	0		0
Dialister invisus	0		0
Dialister invisus & Megasphaera micronuciformis	0		0
Dorea spp.	-1		0
Eubacterium biforme	0		0
Eubacterium hallii	-1		0
Eubacterium rectale	-1		0
Eubacterium siraeum	0		0
Faecalibacterium prausnitzii	0		0
Lachnospiraceae	0		0
Lactobacillus ruminis & Pediococcus acidilactici	0		0
Lactobacillus spp.	0		0
Phascolarctobacterium spp.	+3		0
Ruminococcus albus & R. bromii	0		0
Ruminococcus gnavus	0		0
Streptococcus agalactiae & Eubacterium rectale	0		0
Streptococcus salivarius ssp. *thermophilus & S. sanguinis*	0		0

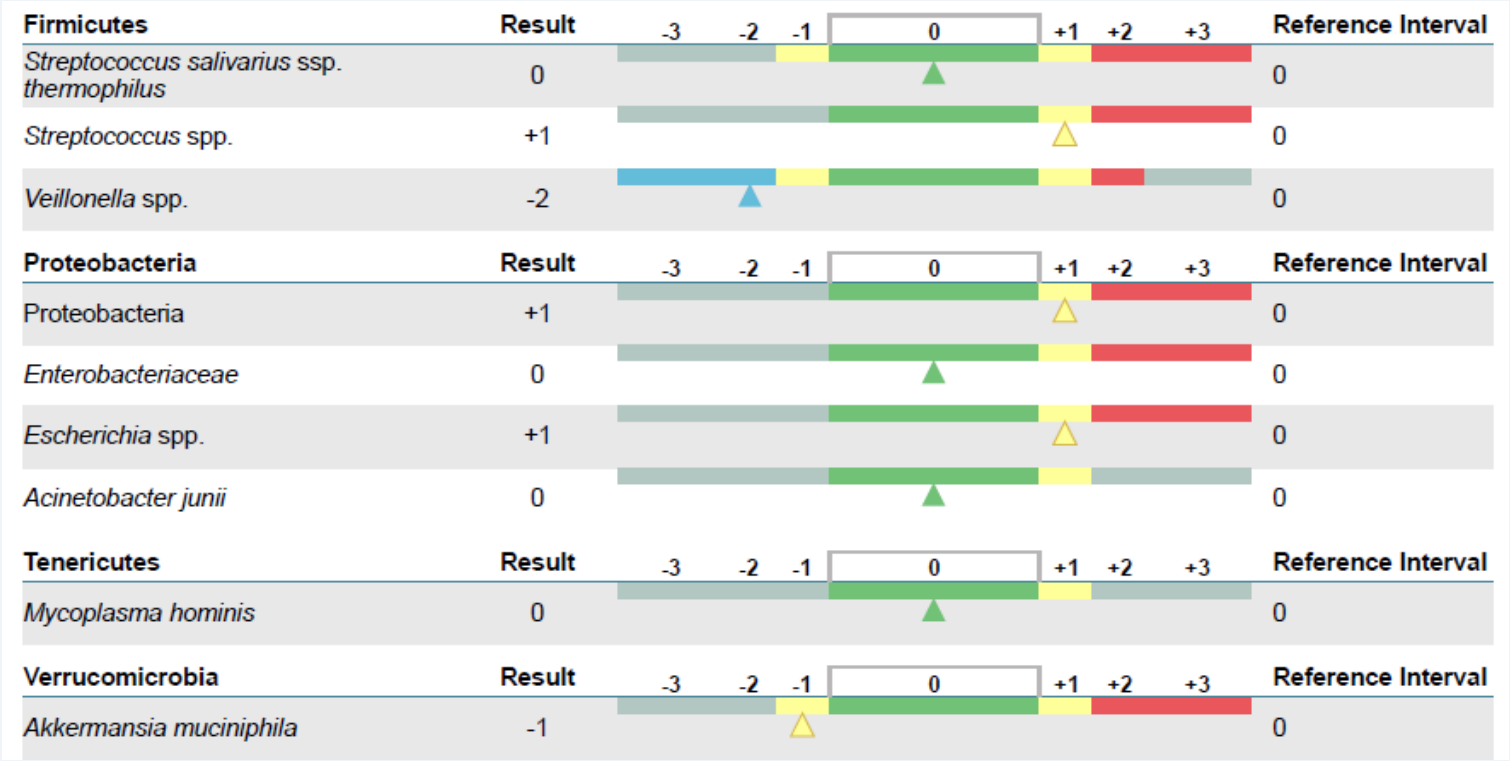
Firmicutes	Result	-3 -2 -1 0 +1 +2 +3	Reference Interval
Streptococcus salivarius ssp. *thermophilus*	0		0
Streptococcus spp.	+1		0
Veillonella spp.	-2		0

Proteobacteria	Result	-3 -2 -1 0 +1 +2 +3	Reference Interval
Proteobacteria	+1		0
Enterobacteriaceae	0		0
Escherichia spp.	+1		0
Acinetobacter junii	0		0

Tenericutes	Result	-3 -2 -1 0 +1 +2 +3	Reference Interval
Mycoplasma hominis	0		0

Verrucomicrobia	Result	-3 -2 -1 0 +1 +2 +3	Reference Interval
Akkermansia muciniphila	-1		0

Case Study - Mae



Firmicutes	Result	Reference Interval
Streptococcus salivarius ssp. *thermophilus*	0	0
Streptococcus spp.	+1	0
Veillonella spp.	-2	0

Proteobacteria	Result	Reference Interval
Proteobacteria	+1	0
Enterobacteriaceae	0	0
Escherichia spp.	+1	0
Acinetobacter junii	0	0

Tenericutes	Result	Reference Interval
Mycoplasma hominis	0	0

Verrucomicrobia	Result	Reference Interval
Akkermansia muciniphila	-1	0

Case Study - Mae

Protozoa	Result	
Balantidium coli	Not Detected	
Blastocystis spp.	Not Detected	
Chilomastix mesnili	Not Detected	
Dientamoeba fragilis	Not Detected	
Endolimax nana	Not Detected	
Entamoeba coli	Not Detected	
Entamoeba hartmanni	Not Detected	
Entamoeba histolytica/Entamoeba dispar	Not Detected	
Entamoeba polecki	Not Detected	
Enteromonas hominis	Not Detected	
Giardia duodenalis	Not Detected	
Iodamoeba bütschlii	Not Detected	
Isospora belli	Not Detected	
Pentatrichomonas hominis	Not Detected	
Retortamonas intestinalis	Not Detected	

Cestodes - Tapeworms	Result	
Diphyllobothrium latum	Not Detected	
Dipylidium caninum	Not Detected	
Hymenolepis diminuta	Not Detected	
Hymenolepis nana	Not Detected	
Taenia	Not Detected	

Trematodes - Flukes	Result	
Clonorchis sinensis	Not Detected	
Fasciola hepatica/Fasciolopsis buski	Not Detected	
Heterophyes heterophyes	Not Detected	
Paragonimus westermani	Not Detected	

Nematodes - Roundworms	Result	
Ascaris lumbricoides	Not Detected	

Case Study - Mae

Nematodes - Roundworms	Result
Capillaria hepatica	Not Detected
Capillaria philippinensis	Not Detected
Enterobius vermicularis	Not Detected
Hookworm	Not Detected
Strongyloides stercoralis	Not Detected
Trichuris trichiura	Not Detected

Other Markers	Result	Reference Interval
Yeast	Rare	Not Detected – Rare
RBC	Not Detected	Not Detected – Rare
WBC	Not Detected	Not Detected – Rare
Muscle fibers	Not Detected	Not Detected – Rare
Vegetable fibers	Rare	Not Detected – Few
Charcot-Leyden Crystals	Not Detected	Not Detected
Pollen	Not Detected	Not Detected

Macroscopic Appearance	Result	Reference Interval
Color	Brown	Brown
Consistency	Soft	Soft
Mucus	Negative	Negative

Case Study - Mae

Pathogenic Bacteria	Result	NG 1+ 2+ 3+ 4+	Reference Interval
Aeromonas spp.	NG		No Growth
Edwardsiella tarda	NG		No Growth
Plesiomonas shigelloides	NG		No Growth
Salmonella group	NG		No Growth
Shigella group	NG		No Growth
Vibrio cholerae	NG		No Growth
Vibrio spp.	NG		No Growth
Yersinia spp.	NG		No Growth

Imbalance Bacteria	Result	NG 1+ 2+ 3+ 4+	Reference Interval
Klebsiella oxytoca	1+		No Growth
Staphylococcus epidermidis	2+		No Growth
Streptococcus mutans	3+		No Growth

Yeast	Result	NG 1+ 2+ 3+ 4+	Reference Interval
Candida albicans	2+		0+ – 1+

Case Study - Mae

Candida albicans

Case Study - Mae

Digestion / Absorption	Result	Unit	L WRI H	Reference Interval
Elastase	>500	µg/g		>200
Fat Stain	None			None – Moderate
Carbohydrates†	Negative			Negative
Inflammation	**Result**	**Unit**	**L WRI H**	**Reference Interval**
Lactoferrin	1.7	µg/mL		<7.3
Lysozyme*	449	ng/mL		≤500
Calprotectin	13	µg/g		<80
Immunology	**Result**	**Unit**	**L WRI H**	**Reference Interval**
Secretory IgA*	99.6	mg/dL		30 – 275
Short Chain Fatty Acids	**Result**	**Unit**	**L WRI H**	**Reference Interval**
% Acetate‡	60	%		50 – 72
% Propionate‡	17	%		11 – 25
% Butyrate‡	20	%		11 – 32
% Valerate‡	3.1	%		0.8 – 5.0
Butyrate‡	0.83	mg/mL		0.8 – 4.0
Total SCFA's‡	4.3	mg/mL		5.0 – 16.0
Intestinal Health Markers	**Result**	**Unit**	**L WRI H**	**Reference Interval**
pH	6.7			5.8 – 7.0
β-glucuronidase*	8170	U/h*g		4000 – 9400
Occult Blood	Negative			Negative

Case Study: Mae — The Gut-Brain Connection via SCFAs

SCFA Neurological Mechanisms

"SCFA-producing bacteria are especially sensitive to antibiotics and antimicrobial therapy may directly influence epigenetics by inhibiting HDAC activity, DNA methylation status and gene transcription in the host."

- Cross blood-brain barrier via specialized transporters
- Maintain blood-brain barrier integrity
- Modulate microglial function (brain immune cells)
- Influence neurotransmitter levels and neurogenesis
- Anti-inflammatory properties in the brain
- Ameliorate stress-induced hyperthermia and corticosterone response
- Reduce anxiety- and depressive-like behavior in preclinical models
- Enhance dopamine receptor D1a expression
- Prevent stress-induced intestinal permeability increase
- Increase BDNF (brain-derived neurotrophic factor)

Why This Matters for Mae

- Dysbiosis disrupting SCFA production
- Impaired gut barrier → systemic inflammation → neuroinflammation
- Depleted butyrate → compromised BBB
- Altered neurotransmitter metabolism
- Epigenetic effects of SCFA depletion
- Poly-pharmacy further disrupting microbiome

SCFA Impact on Brain Disorders

- Mood Disorders: Antidepressant-like effects in animal studies
- ASD: Mixed — butyrate beneficial, propionate potentially harmful
- Alzheimer's: May reduce amyloid accumulation
- Parkinson's: SCFA dysregulation observed
- MS: Beneficial anti-inflammatory effects

Silva YP et al. (2020) The Role of Short-Chain Fatty Acids From Gut Microbiota in Gut-Brain Communication. Frontiers in Endocrinology, 11:25.

frontiers in Cellular Neuroscience

MINI REVIEW
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Epigenetic Regulation of Enteric Neurotransmission by Gut Bacteria

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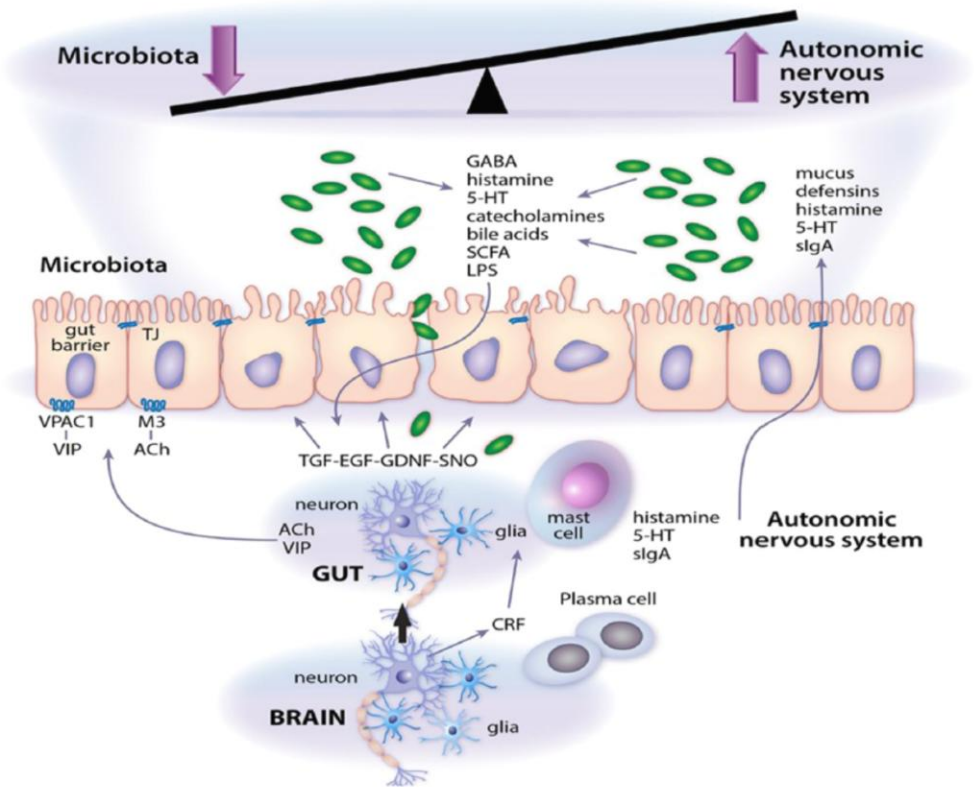
"Short chain fatty acid producing bacteria are especially sensitive to antibiotics and antimicrobial therapy may directly influence epigenetics by inhibiting histone deacetylase activity, DNA methylation status and gene transcription in the host."

community structure are increasingly found to associate with alterations in enteric neurotransmission and disease. At present, it is not known whether shifts in microbial community dynamics represent cause or consequence of disease pathogenesis. The discovery of bacterial-derived neurotransmitters suggests further studies are needed to establish their role in enteric neuropathy. This mini-review highlights recent advances in bacterial communications to the autonomic nervous system and discusses emerging epigenetic data showing that diet, probiotic and antibiotic use may regulate enteric neurotransmission through modulation of microbial communities. A particular emphasis is placed on bacterial metabolite regulation of enteric nervous system function in the intestine



IGURE 1 | Microbial neurotransmitter crosstalk with the autonomic nervous system. As outlined in the article, a system of checks and balances operate to egulate gut function. Abbreviations: 5-HT, serotonin; Ach, acetylcholine; CRF, corticotrophin releasing factor; EGF, epidermal growth factor; GDNF, glial cell ne-derived neurotrophic factor; LPS, lipopolysaccharide; M3, M3 muscarinic receptor; SCFA, short chain fatty acids; sIgA, secretory IgA; SNO, s-nitrosothiol; TGF, ansforming growth factor; VIP, vasoactive intestinal peptide; VPAC1, VIP and PACAP receptor 1.

The Role of Short-Chain Fatty Acids From Gut Microbiota in Gut-Brain Communication

*Ygor Parladore Silva[1], Andressa Bernardi[2] and Rudimar Luiz Frozza[1]**

[1] Laboratory on Thymus Research, Oswaldo Cruz Institute, Oswaldo Cruz Foundation, Rio de Janeiro, Brazil, [2] Laboratory of Inflammation, Oswaldo Cruz Institute, Oswaldo Cruz Foundation, Rio de Janeiro, Brazil

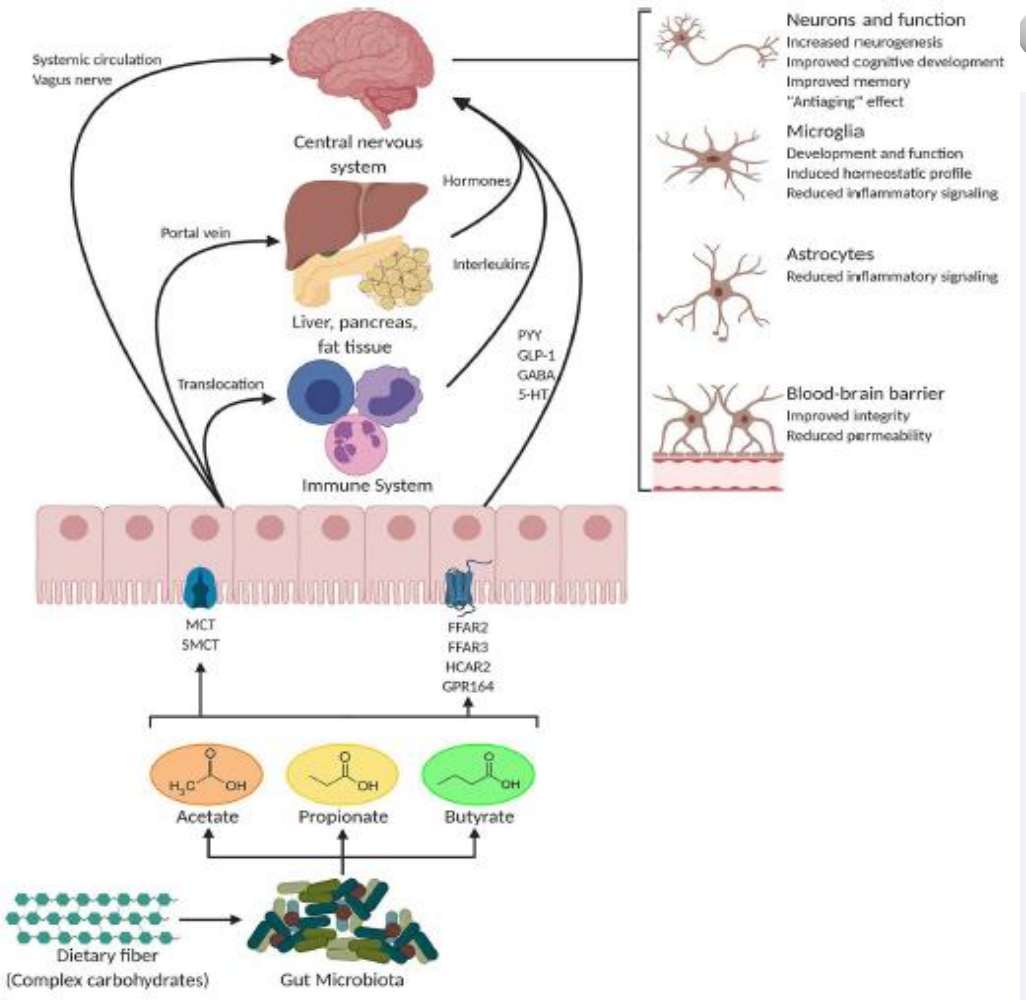
A substantial body of evidence supports that the gut microbiota plays a pivotal role in the regulation of metabolic, endocrine and immune functions. In recent years, there has been growing recognition of the involvement of the gut microbiota in the modulation of multiple neurochemical pathways through the highly interconnected gut-brain axis. Although amazing scientific breakthroughs over the last few years have expanded our knowledge on the communication between microbes and their hosts, the underpinnings of microbiota-gut-brain crosstalk remain to be determined. Short-chain fatty acids (SCFAs), the main metabolites produced in the colon by bacterial fermentation of dietary fibers and resistant starch, are speculated to play a key role in neuro-immunoendocrine regulation. However, the underlying mechanisms through which SCFAs might influence brain physiology and behavior have not been fully elucidated. In this review, we outline the current knowledge about the involvement of SCFAs in microbiota-gut-brain interactions. We also highlight how the development of future treatments for central nervous system (CNS) disorders can take advantage of the intimate and mutual interactions of the gut microbiota with the brain by exploring the role of SCFAs in the regulation of neuro-immunoendocrine function.

Basic Overview:

- SCFAs (mainly acetate, propionate, and butyrate) are metabolites produced by gut bacteria when they ferment dietary fibers and resistant starch
- They can influence brain function through multiple pathways including the vagus nerve, immune system, and direct effects on the brain

Key Mechanisms:

- SCFAs can cross the blood-brain barrier through specialized transporters
- They help maintain blood-brain barrier integrity
- They influence microglial function (brain immune cells)
- They modulate neurotransmitter levels
- They can affect neuronal function and neurogenesis
- They have anti-inflammatory properties in the brain



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Case Study - Mae

VAGINOSIS PATHOGEN PANEL PCR

Ordered by Physician Jana Wynett, MD

Collected on 2/14/23 from Vagina (Swab)

Resulted on 2/15/23

(see .jpg file in Shared Files)

RESULTS

BV Result

Value **Negative**

Standard Range Negative

Trichomonas Results

Negative

(!) Candida Species Result

Value **Positive**

Standard Range Negative

Candida glabrata Result

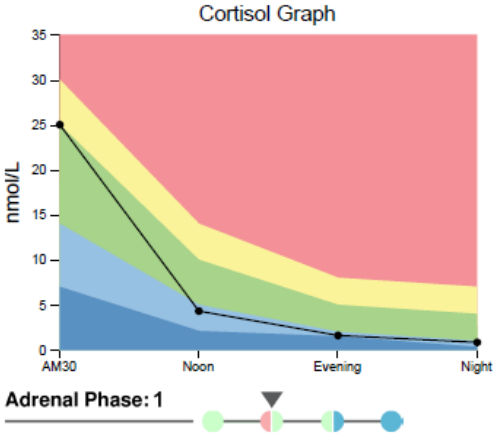
Value **Negative**

Case Study - Mae

BACTERIOLOGY CULTURE		
Expected/Beneficial flora	**Commensal (Imbalanced) flora**	**Dysbiotic flora**
2+ Lactobacillus spp.	3+ Alpha hemolytic strep	4+ Gardnerella vaginalis
	1+ Escherichia coli	
	2+ Gamma hemolytic strep	
	2+ Staphylococcus not aureus	
NG = No Growth		

Case Study - Mae

Analyte	Result	Unit	L	WRI	H	Optimal Range	Reference Interval
Cortisol AM30	25	nmol/L		◆		14.0 – 25.0	7.0 – 30.0
Cortisol Noon	4.3	nmol/L	◆			5.0 – 10.0	2.1 – 14.0
Cortisol Evening	1.6	nmol/L	◆			2.0 – 5.0	1.5 – 8.0
Cortisol Night	0.83	nmol/L	◆			1.0 – 4.0	0.33 – 7.0
DHEA*	187	pg/mL		◆			106 – 300



Hormone Comments

- AM cortisol level appears adequate, although the suboptimal diurnal cortisol pattern is suggestive of early (Phase 1) HPA axis (adrenal gland) dysfunction.

Case Study - Mae

Analyte	Result	Unit	L	WRI	H	Reference Interval	Supplementation Range**
Estrone (E1)*	14.5	pg/mL		◆		<35	
Estradiol (E2)	1.1	pg/mL		◆		0.6–4.5	1.0–6.0
Estriol (E3)*	<5.0	pg/mL	↓			7.5–66	45–680
EQ (E3 / (E1 + E2)) Ratio	0.32		↓			≥1.0	
Progesterone (Pg)	26	pg/mL	↓			127–446	400–4000
Pg/E2 Ratio†	23.6		↓			≥200	≥200
Testosterone	8	pg/mL		◆		6–49	25–60
DHEA*	187	pg/mL		◆		106–300	

Case Study - Mae

Analyte	Result	Unit per Creatinine	L	WRI	H	Reference Interval
Phenethylamine (PEA)	34	nmol/g				32 – 84
Tyrosine	48	µmol/g				32 – 80
Tyramine	1.9	µmol/g				2.0 – 4.0
Dopamine	294	µg/g				125 – 250
3,4-Dihydroxyphenylacetic acid (DOPAC)	892	µg/g				390 – 1500
3-Methoxytyramine (3-MT)	341	nmol/g				90 – 210
Norepinephrine	14.5	µg/g				22 – 50
Normetanephrine	266	µg/g				85 – 300
Epinephrine	2.8	µg/g				1.6 – 8.3
Metanephrine	97	µg/g				45 – 119
Norepinephrine / Epinephrine ratio	5.2					< 13
Tryptamine	0.19	µmol/g				0.20 – 0.90
Serotonin	80.4	µg/g				60 – 125
5-Hydroxyindoleacetic acid (5-HIAA)	8625	µg/g				2000 – 8000
Glutamate	21	µmol/g				12.0 – 45.0
Gamma-aminobutyrate (GABA)	3.0	µmol/g				2.0 – 5.6
Glycine	2549	µmol/g				450 – 2200
Histamine	9.0	µg/g				14 – 44
Taurine	286	µmol/g				320 – 1000
Creatinine	45.2	mg/dL				30 – 225

Taurine Benefits for Mental Health and Alertness

Evidence-Based Review of Neurological Effects

Taurine: Essential Neural Regulator

Key Properties:

- Sulfur-containing amino acid
- Major GABA receptor agonist
- Essential for neurotransmitter regulation
- Potent antioxidant and neuroprotectant

Brain Concentrations:

- Second most abundant amino acid in brain
- Highest in hippocampus and cerebellum
- Critical for neuronal development

Primary Citations:

Menzie J, Pan C, Prentice H, Wu JY. (2014). Taurine and central nervous system disorders. Amino Acids, 46(1):31-46.
Ripps H, Shen W. (2012). Review: taurine: a "very essential" amino acid. Molecular Vision, 18:2673-86.

Mental Health Benefits

Anxiety Reduction:

- Decreases anxiety-like behaviors
- Modulates GABA signaling
- Reduces stress hormone release
- Clinical improvement in anxiety scores

Depression Impact:

- Increases BDNF expression
- Improves neuroplasticity
- Reduces inflammatory markers
- Enhances serotonin function

Key Studies:

Wu GF, et al. (2017). Antidepressant effect of taurine in chronic unpredictable mild stress-induced depressive rats. Scientific Reports, 7(1):4989.

Toyoda A, Iio W. (2013). Antidepressant-like effect of chronic taurine administration and its hippocampal signal transduction in rats. Advances in Experimental Medicine and Biology, 775:29-43.

nutrients

MDPI

Review

Taurine Supplementation as a Neuroprotective Strategy upon Brain Dysfunction in Metabolic Syndrome and Diabetes

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Abstract: Obesity, type 2 diabetes, and their associated comorbidities impact brain metabolism and function and constitute risk factors for cognitive impairment. Alterations to taurine homeostasis can impact a number of biological processes, such as osmolarity control, calcium homeostasis, and inhibitory neurotransmission, and have been reported in both metabolic and neurodegenerative disorders. Models of neurodegenerative disorders show reduced brain taurine concentrations. On the other hand, models of insulin-dependent diabetes, insulin resistance, and diet-induced obesity display taurine accumulation in the hippocampus. Given the possible cytoprotective actions of taurine, such cerebral accumulation of taurine might constitute a compensatory mechanism that attempts to prevent neurodegeneration. The present article provides an overview of brain taurine homeostasis and reviews the mechanisms by which taurine can afford neuroprotection in individuals with obesity and diabetes. We conclude that further research is needed for understanding taurine homeostasis in metabolic disorders with an impact on brain function.

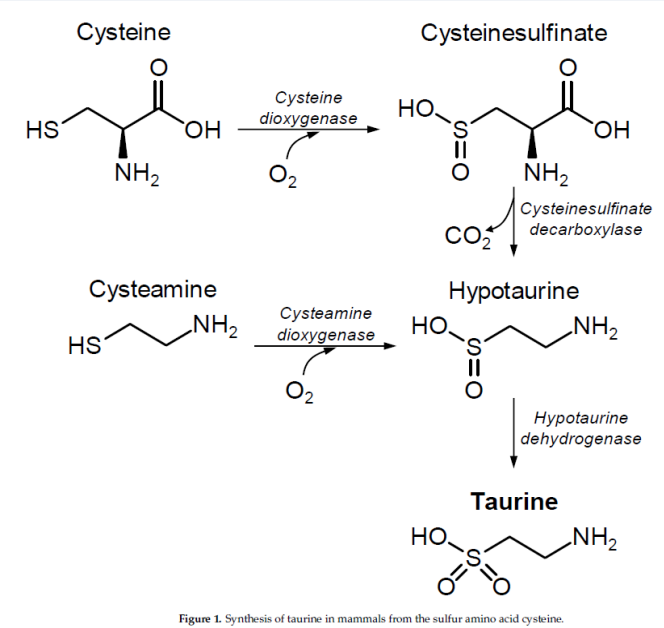
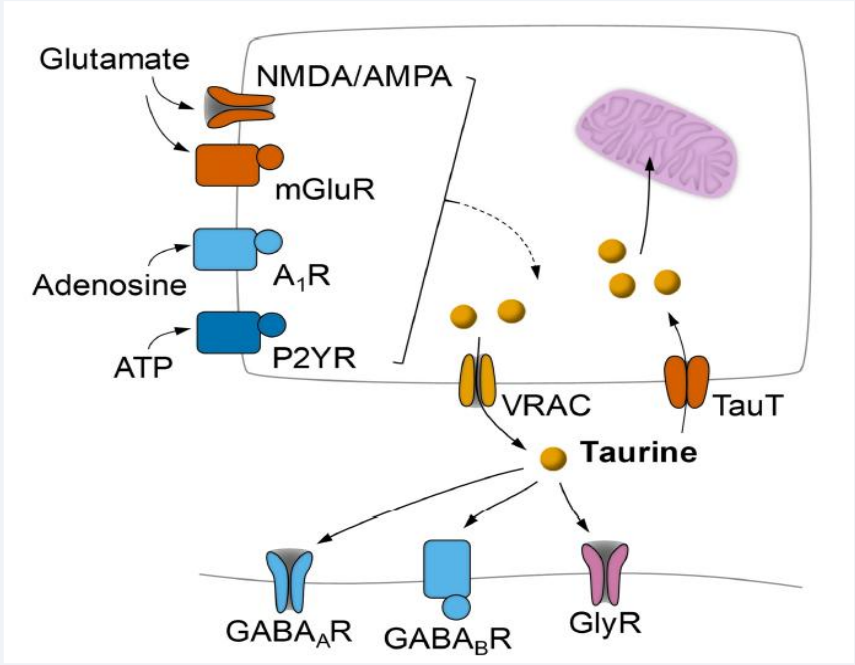


Figure 1. Synthesis of taurine in mammals from the sulfur amino acid cysteine.



In sum, taurine supplementation is proposed to improve the function of the mitochondria, contributing to the preservation of mitochondrial membrane potential, proton gradient, and matrix pH that are critical for energy metabolism and efficient oxidative phosphorylation, as well as intracellular calcium homeostasis.

Cognitive Enhancement Effects

Memory Enhancement:

- Improves spatial memory
- Enhances long-term potentiation
- Protects against memory deficits
- Supports synaptic plasticity

Learning Benefits:

- Increases focus and attention
- Enhances cognitive performance
- Improves information processing
- Supports neural development

Research Evidence:

Jakaria M, et al. (2019). Taurine: A potential molecule for Alzheimer's disease therapy. Biochemical Pharmacology, 163:12-26.
Chen C, et al. (2019). Taurine Supplementation Enhances GABA Neurotransmission and Improves Behavioral Abilities. Molecular Neurobiology, 56(3):1959-1975.

Clinical Applications & Dosing

Therapeutic Uses:

- Anxiety disorders: 500-2000mg daily
- Cognitive enhancement: 1000-3000mg daily
- Sleep support: 1000mg before bed
- Neuroprotection: 1500-3000mg daily

Safety Profile:

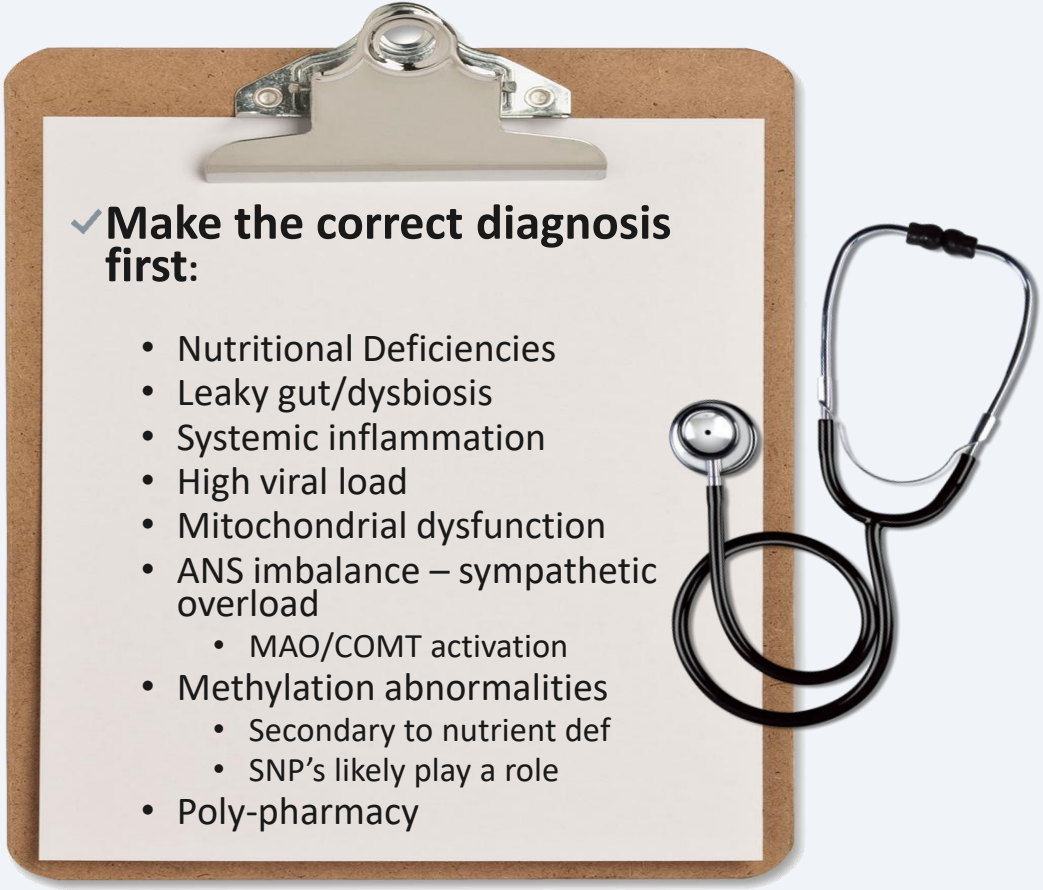
- Well-tolerated up to 3g/day
- No significant side effects reported
- Best absorbed away from meals
- Consider divided dosing

Clinical Evidence:

Schaffer S, Kim HW. (2018). Effects and Mechanisms of Taurine as a Therapeutic Agent. Biomolecules & Therapeutics, 26(3):225-241.
Oja SS, Saransaari P. (2017). Pharmacology of Taurine. Proceedings of the Western Pharmacology Society, 60:1-6.

Case Study - Mae

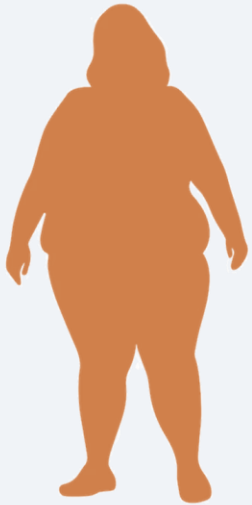
- **Treatment**



Treatment

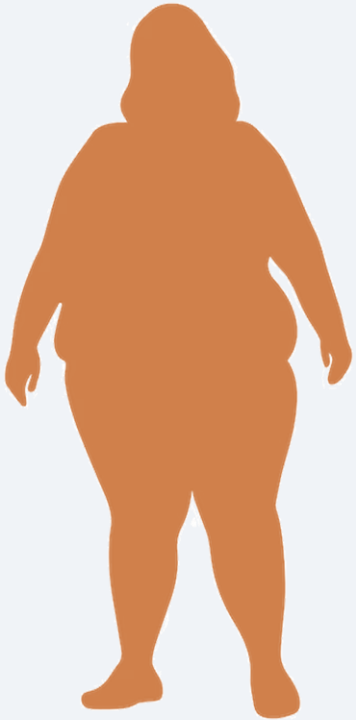
- ✓ Diet (AI and calorie restriction)
- ✓ Exercise, in sufficient amounts (HIIT and resistance, start slow).
- ✓ Mindfulness training, RR 20 min/day.
- ✓ Breathing, just 5 min twice daily - box
- ✓ Chiro Treatments, Laser, HBOT.
- ✓ Lundell Nutrient IV's – B's, NAC, ALA, C etc.
- ✓ Supplementation.

SUPPLEMENTATION



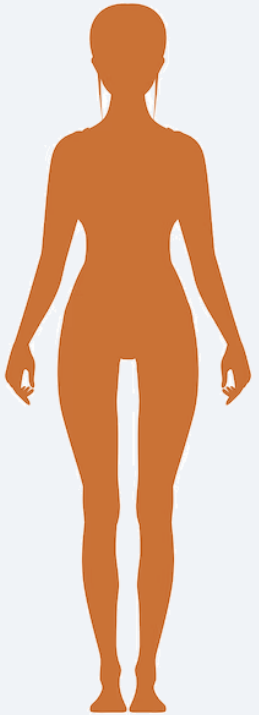
- Multi-vitamin with methyl-nutrients
- NAC
- Grape Seed and Taurine
- Liver support
- Mineral
- Vitamin C
- Vitamin D/K
- Dysbiosis Protocol – 2 months
- Gaba
- Mushroom Blend
- Hi-Potency Omega
- Butyrate
- Glutamine
- Curcumin
- Kava
- Mucuna pruriens
- Phosphatidyl Choline
- SAMe
- Adaptogens

Case Study – Mae after 4 months

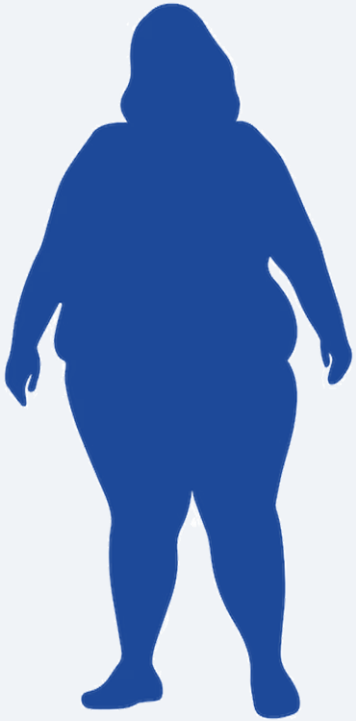


First 4 months:

- Lost 20 pounds (and kept it off)
- Wasn't sweating everyday
- Started to exercise for first time in years and actually like it
- Felt "normal" for an hour or two per day!
- No more hospital episodes
- Had trouble with all the pills – so we simplified a bit
- Was off Remeron, and Gabapentin
- Half dose of Wellbutrin and Remeron
- Started to ween off Vyvanse

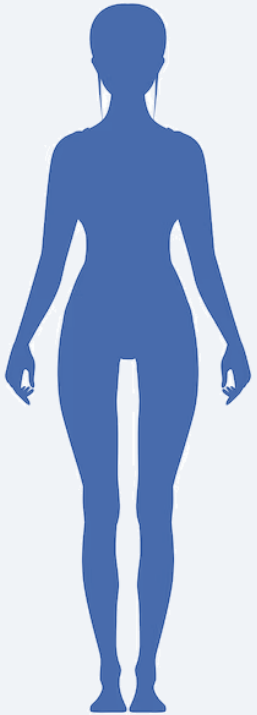


Case Study – Mae after 6 months



After 6 months:

- Lost 40 lbs.
- Got a full-time job
- Did Ketamine one more time, but this time it seemed to “stick”
- Most days feels normal, but still has episodes where she is tired and not motivated
- Still has anxiety but has more tools and she knows how and when to use them
- Does HBOT once a week to get a boost
- IV’s once per month still seem to help

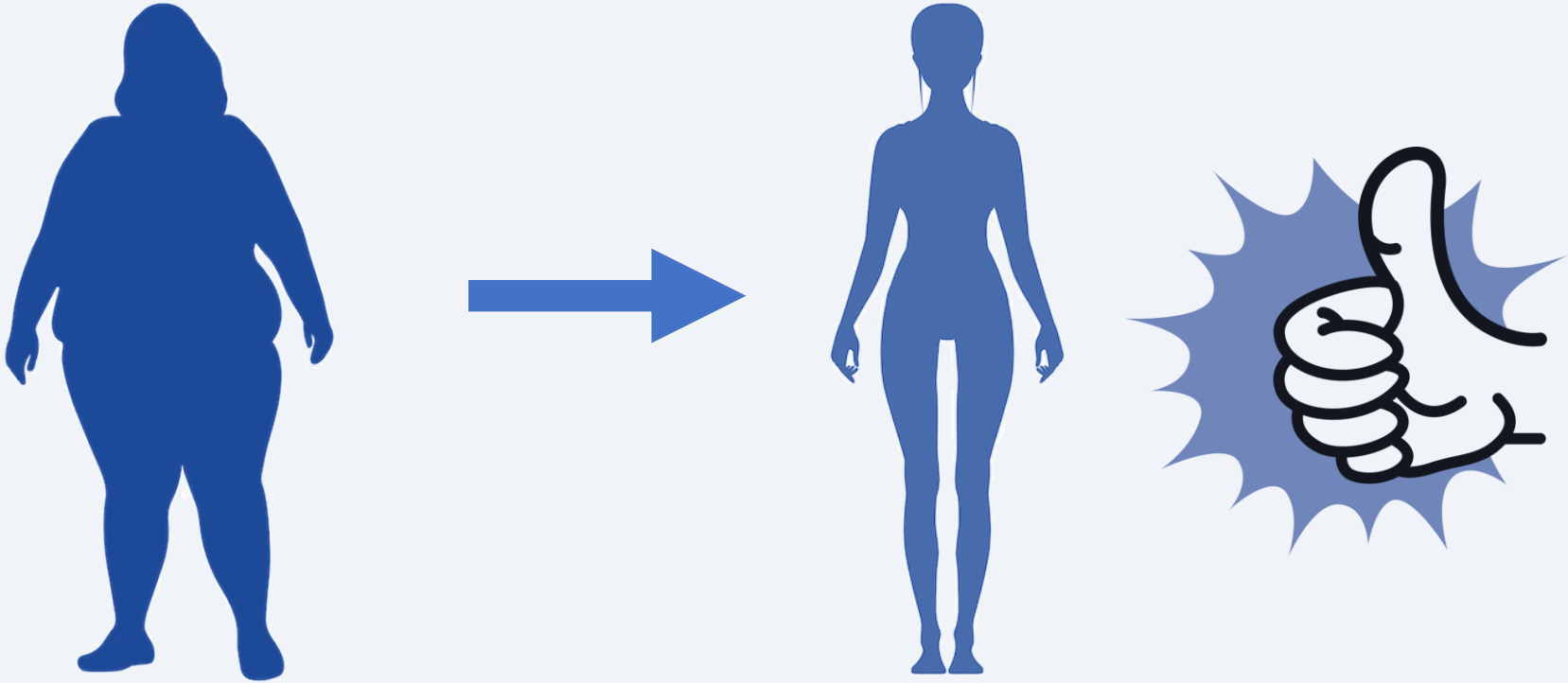


Case Study – Mae after 6 months

BACTERIOLOGY CULTURE		
Expected/Beneficial flora	**Commensal (Imbalanced) flora**	**Dysbiotic flora**
3+ *Bacteroides* family	1+ *Enterobacter cancerogenus*	
1+ *Bifidobacterium* family	2+ *Enterobacter cloacae* complex	
1+ *Escherichia coli*	2+ *Klebsiella pneumoniae*	
1+ *Lactobacillus* family	3+ *Streptococcus salivarius/vestibularis*	
2+ *Enterococcus* family		
4+ *Clostridium* family		
NG = No Growth		

YEAST CULTURE		
Normal flora		**Dysbiotic flora**
1+ *Candida albicans*	Normal flora	2+ Candida albicans

Discussion: Case Study – Mae



Resources & Next Steps

Doctor's Data: doctorsdata.com | gi360.com

GI 360 Resource Guide: gi360.com/resource-guide

Sample Report: gi360.com/sample-report

Nutritional Pathology Institute: drbrandonlundell.com

A good doctor know what to do,
A GREAT doctor knows WHY

Questions? | Contact: drbrandonlundell.com

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– Holly Green, LAc

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– Dr. Natalya Fazylova, DNP

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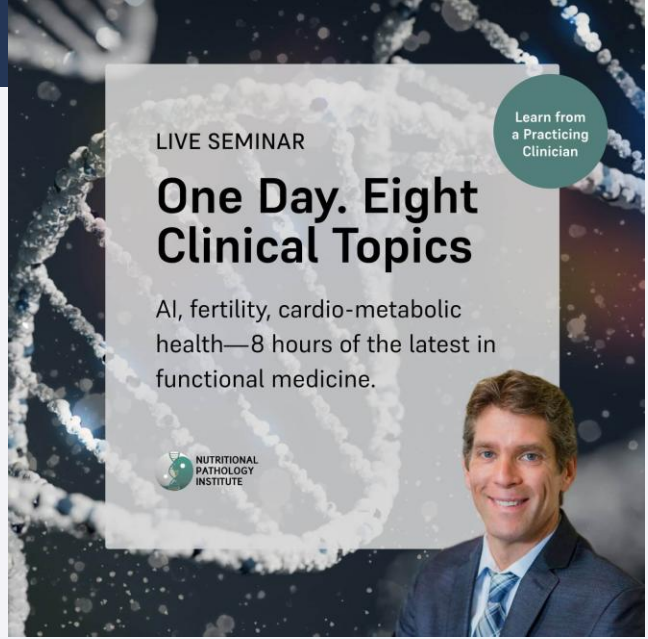

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- The gut-gonad connection matters more than most practitioners realize.
- How to assess both male and female patients for underlying causes affecting fertility
- Low sperm counts, egg quality issues, and miscarriage susceptibility
- Optimizing the fetal environment to reduce risk of mental health disorders and developmental issues
- Case studies showing successful pregnancies after multiple failed IVF attempts

Hormones and Functional Endocrinology

- Blood, urine, or saliva testing—when to use each
- Clinical decision-making for hormone assessment
- How to optimize cell sensitivity before bioidentical HRT
- Making hormone replacement safer and more effective when indicated

Cardiovascular Risk Assessment

- Pro-BNP interpretation for clinical practice
- Advanced assessment techniques beyond standard lipid panels
- Post-COVID cardiovascular health strategies
- Functional Medicine approaches to increased cardiovascular risks

Peptide Integration in Clinical Practice

- When to use peptides and which ones work
- Integration protocols backed by patient outcomes
- Practical application in various conditions

Environmental Medicine

Heavy metal testing is one of the most misused tools in functional medicine. You'll learn:

- Best practices for heavy metal testing and detoxification
- How to determine if heavy metals are actually contributing to your patient's case
- How to assess whether mold is a real clinical issue versus a distraction

Not every patient with chronic illness has mold toxicity—this section will help you make that distinction.

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- Documentation shortcuts that maintain quality
- Protocol-building tools that enhance patient outcomes
- Can save you hundreds of hours every year

Case Studies

Real cases, real outcomes. You'll see examples of autoimmune resolution, fertility success after years of failed conventional treatment, and complex cases made simple through systematic functional medicine assessment.

Resources & Next Steps

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GI 360 Resource Guide: gi360.com/resource-guide

Sample Report: gi360.com/sample-report

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A GREAT doctor knows WHY

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