Casual Friday Series

GI ResQ Protocol 101: Multiple Applications

A BIOGENETIX CLINICAL PRESENTATION

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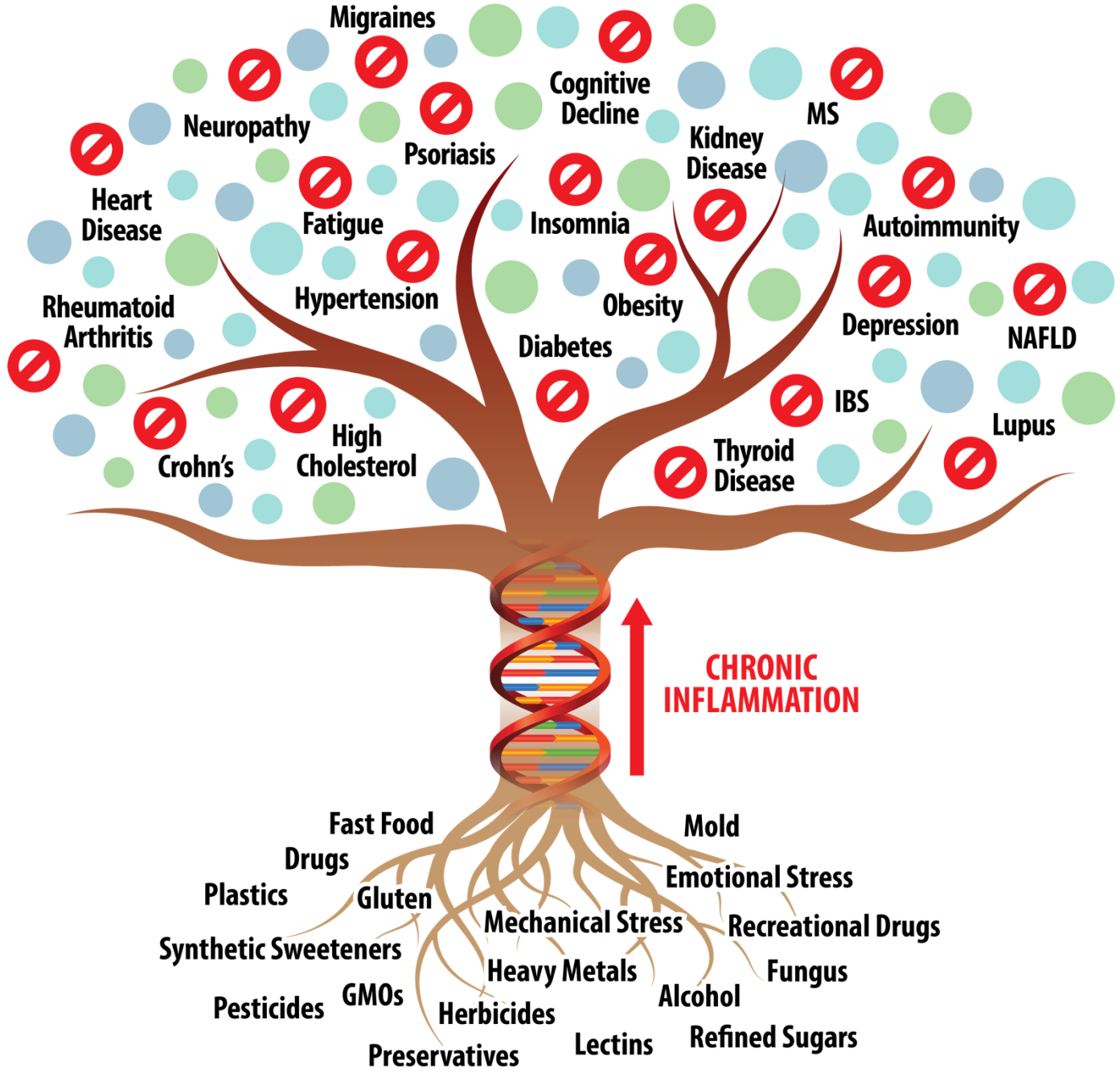
Migraines
Neuropathy
Psoriasis
Cognitive Decline
MS
Kidney Disease
Heart Disease
Fatigue
Insomnia
Autoimmunity
Rheumatoid Arthritis
Hypertension
Obesity
Depression
NAFLD
Diabetes
IBS
Lupus
Crohn's
High Cholesterol
Thyroid Disease
CHRONIC INFLAMMATION
Fast Food
Drugs
Plastics
Gluten
Mold
Emotional Stress
Mechanical Stress
Recreational Drugs
Synthetic Sweeteners
Heavy Metals
Fungus
Pesticides
GMOs
Herbicides
Alcohol
Preservatives
Lectins
Refined Sugars

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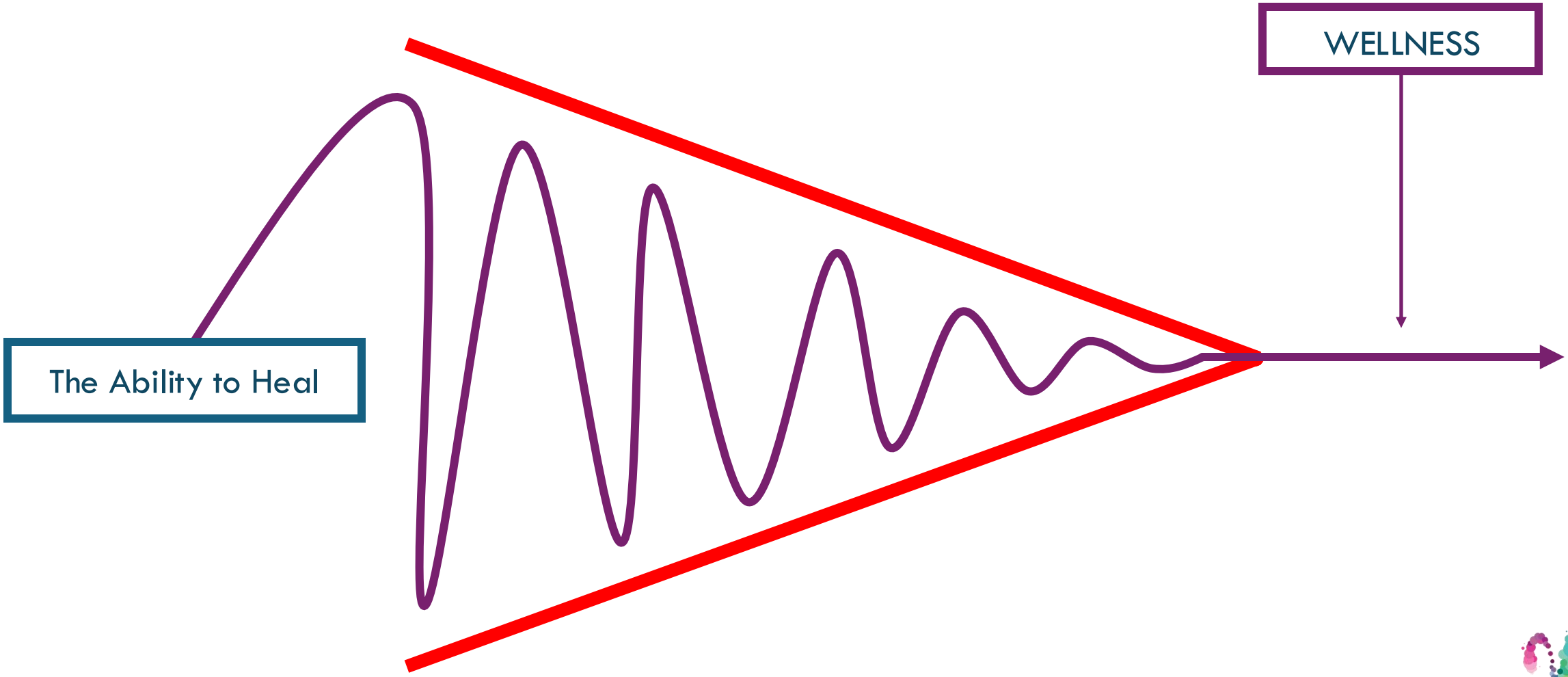
The Wedge Protocol



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Front Immunol. 2022 Oct 20;13:906258. doi: 10.3389/fimmu.2022.906258

Microbial dysbiosis in the gut drives systemic autoimmune diseases

Walaa K Mousa [1,2,3,*,†], Fadia Chehadeh [1], Shannon Husband [1]

Trillions of microbes survive and thrive inside the human body. These tiny creatures are crucial to the development and maturation of our immune system and to maintain gut immune homeostasis. Microbial dysbiosis is the main driver of local inflammatory and autoimmune diseases such as colitis and inflammatory bowel diseases. Dysbiosis in the gut can also drive systemic autoimmune diseases such as type 1 diabetes, rheumatic arthritis, and multiple sclerosis. Gut microbes directly interact with the immune system by multiple mechanisms including modulation of the host microRNAs affecting gene expression at the post-transcriptional level or production of microbial metabolites that interact with cellular receptors such as TLRs and GPCRs. This interaction modulates crucial immune functions such as differentiation of lymphocytes, production of interleukins, or controlling the leakage of inflammatory molecules from the gut to the systemic circulation. In this review, we compile and analyze data to gain insights into the underpinning mechanisms mediating systemic autoimmune diseases. Understanding how gut microbes can trigger or protect from systemic autoimmune diseases is crucial to (1) tackle these diseases through diet or lifestyle modification, (2) develop new microbiome-based therapeutics such as prebiotics or probiotics, (3) identify diagnostic biomarkers to predict disease risk, and (4) observe and intervene with microbial population change with the flare-up of autoimmune responses. Considering the microbiome signature as a crucial player in systemic autoimmune diseases might hold a promise to turn these untreatable diseases into manageable or preventable ones.

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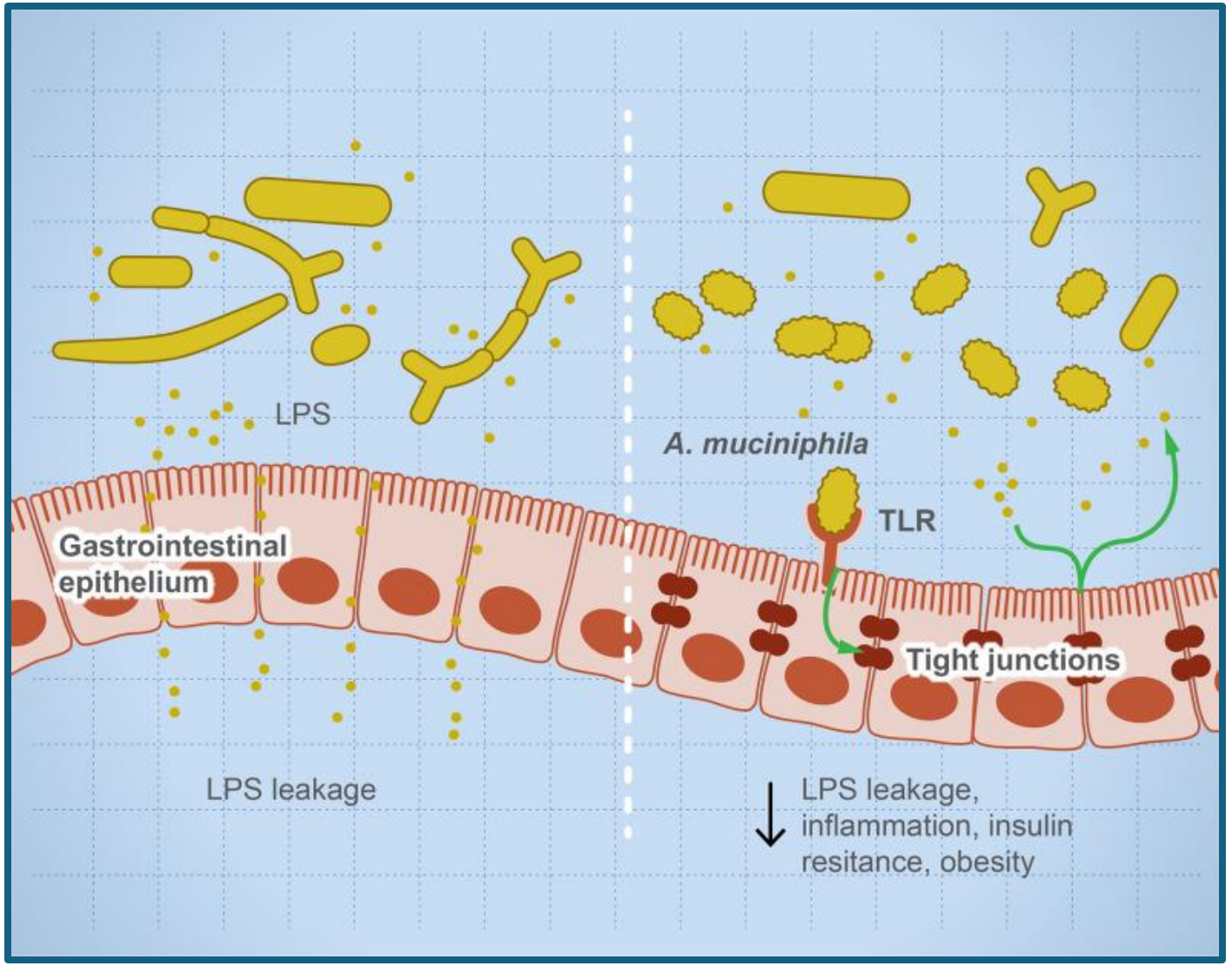
Microbial dysbiosis disturbs the immune function leading to inflammation and sensitization of the immune system and causing autoimmune diseases (35, 36). Many factors influence microbial dysbiosis such as diet, stress, drugs, diseases, age, and lifestyle. The imbalance in helper T cells/regulatory T cells drives autoimmune diseases such as colitis and multiple sclerosis (MS) (18, 33). Leakage of metabolites such as lipopolysaccharides sensitizes the immune system, leading to a higher production of pro-inflammatory interleukins, and degradation of mucin resulting in irritation of the gut lining and microbial invasion (37). **Figure 1** illustrates the role of some microbial taxa in maintaining gut barrier function and how microbial dysbiosis results in a leaky gut. Each microbe prevents or drives inflammation by a unique mechanism. For example, *Faecalibacterium prausnitzii* prevents inflammation by inducing Treg differentiation, leading to the subsequent higher production of IL-10 (an anti-inflammatory interleukin) (38, 39). In contrast, *Fusobacterium nucleatum* drives inflammation by inhibiting cytotoxic T cells and modulation of miRNAs, leading to suppression of autophagy (28). Several examples of individual microbes that modulate host immune response to prevent or drive inflammation and autoimmune reaction are noted (40–72) and summarized (**Table 2**).



Lactobacilli and *Bifidobacterium* increase bile salt hydrolyses, leading to GLP-1 hormone stimulation. If this microbial role is disturbed, inflammation and autoimmune diseases arise.

https://pmc.ncbi.nlm.nih.gov/articles/PMC9632986/

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GI ResQ Protocol

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Functional Imbalance Scores

Key (< 2) : Low Need for Support (2-3) : Optional Need for Support (4-6) : Moderate Need for Support (7-10) : High Need for Support

	Need for Digestive Support	Need for Inflammation Modulation	Need for Microbiome Support	Need for Prebiotic Support	Need for Antimicrobial Support
	MALDIGESTION	**INFLAMMATION**	**DYSBIOSIS**	**METABOLIC IMBALANCE**	**INFECTION**
	0	8	10	9	0
Biomarkers	Products of Protein Breakdown ▽ Pancreatic Elastase ● Fecal Fats ●	Occult Blood ▲ Eosinophil Protein X △ Calprotectin ● Secretory IgA ●	IAD/Methane Score △ Reference Variance △ PP Bacteria/Yeast ● Total Abundance ●	Total SCFA's ▼ n-Butyrate Conc. ▼ Beta-glucuronidase △ SCFA (%) ●	Parasitic Infection ● Pathogenic Bacteria ● PP Bacteria/Yeast ● Total Abundance ●
Therapeutic Support Options	• Digestive Enzymes • Betaine HCl • Bile Salts • Apple Cider Vinegar • Mindful Eating Habits • Digestive Bitters	• Elimination Diet/ Food Sensitivity Testing • Mucosa Support: Slippery Elm, Althea, Aloe, DGL, etc. • Zinc Carnosine • L-Glutamine • Quercetin • Turmeric • Omega-3's • GI Referral (If Calpro is Elevated)	• Pre-/Probiotics • Increase Dietary Fiber Intake • Consider SIBO Testing • Increase Resistant Starches • Increase Fermented Foods • Meal Timing	• Pre-/Probiotics • Increased Dietary Fiber Intake • Increase Resistant Starches • Increase Fermented Foods • Calcium D-Glucarate (for high beta-glucuronidase)	• Antibiotics (if warranted) • Antimicrobial Herbal Therapy • Antiparasitic Herbal Therapy (if warranted) • *Saccharomyces boulardii*

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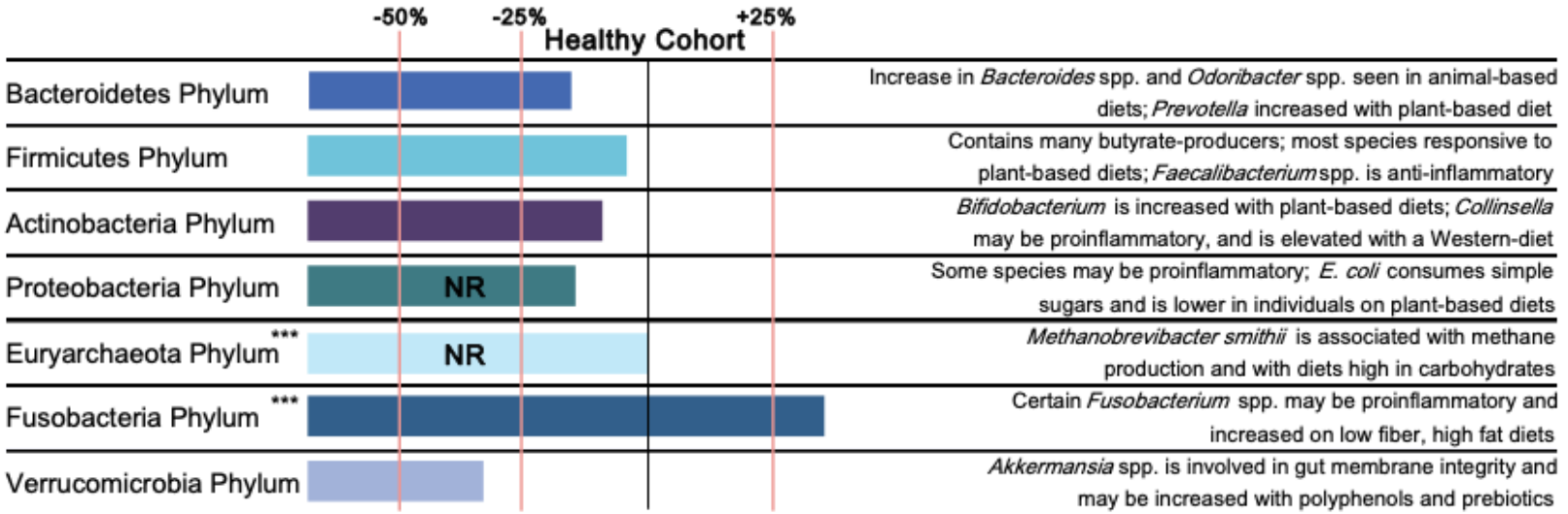
Relative Commensal Abundance



Additional Results

Methodology: Fecal Immunochemical Testing (FIT)

	Result	Expected Value
Fecal Occult Blood◆	Positive	Negative
Color††	Brown	
Consistency††	Formed/Normal	

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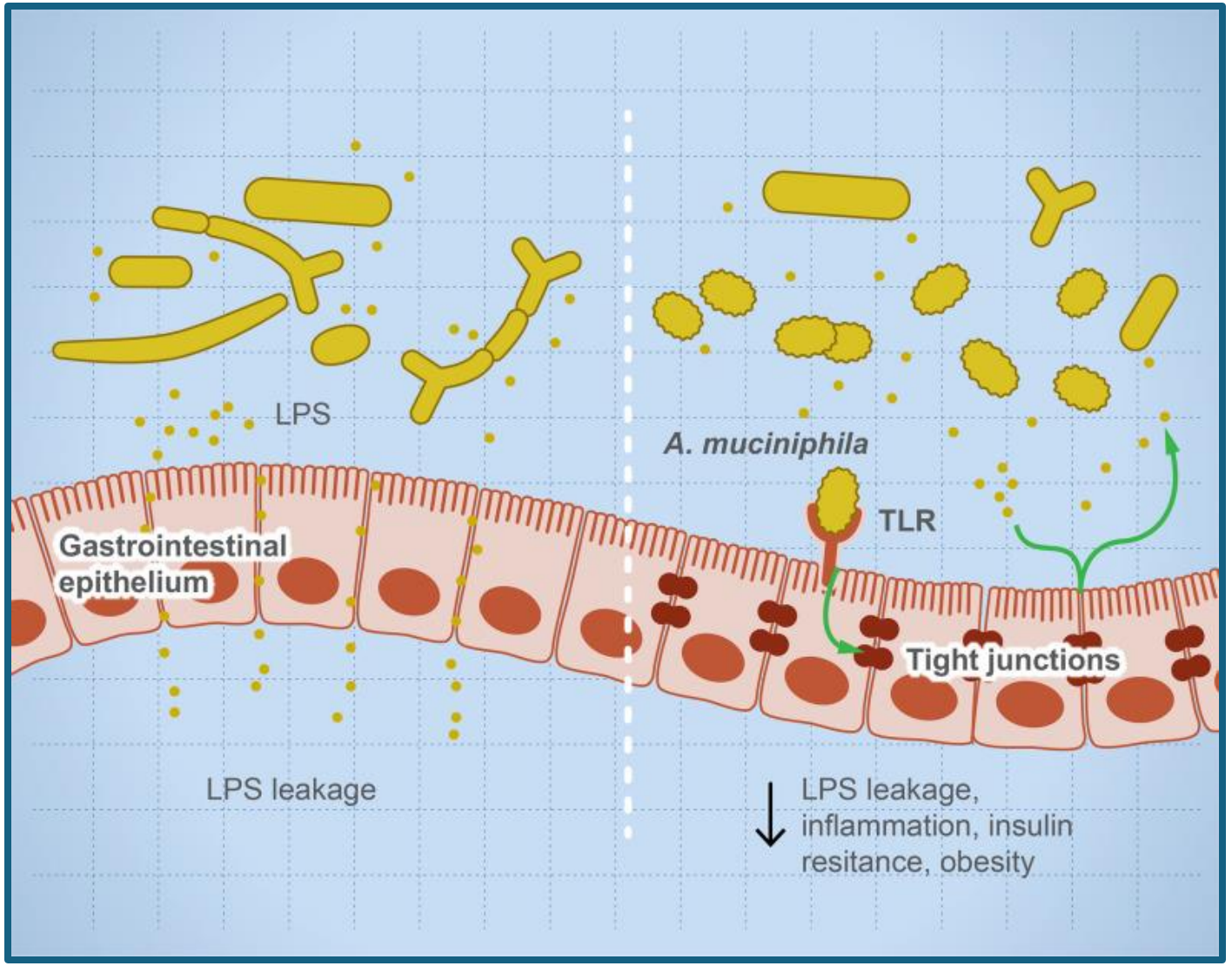

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Strategy Adaptations

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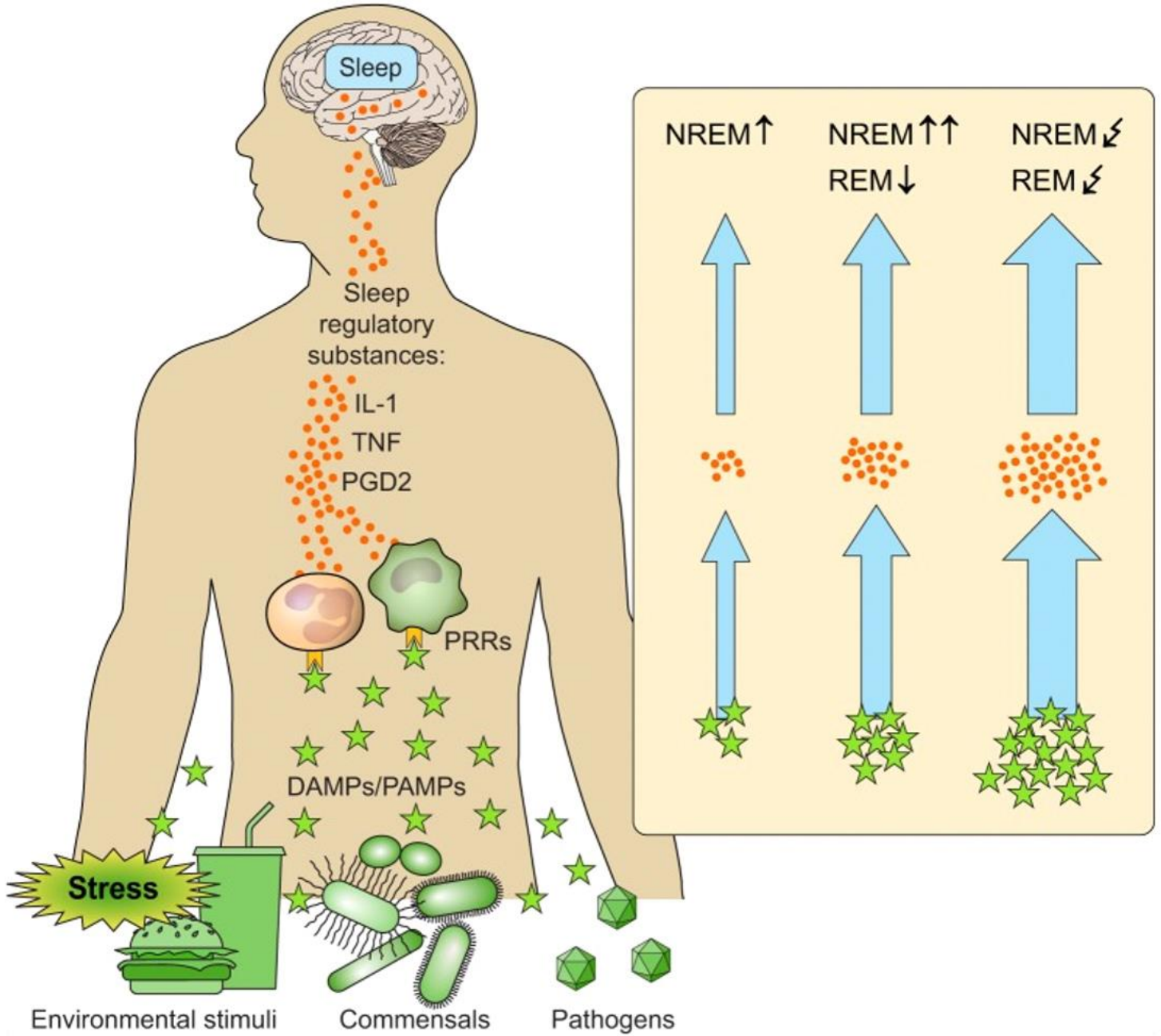

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Strategy Adaptations

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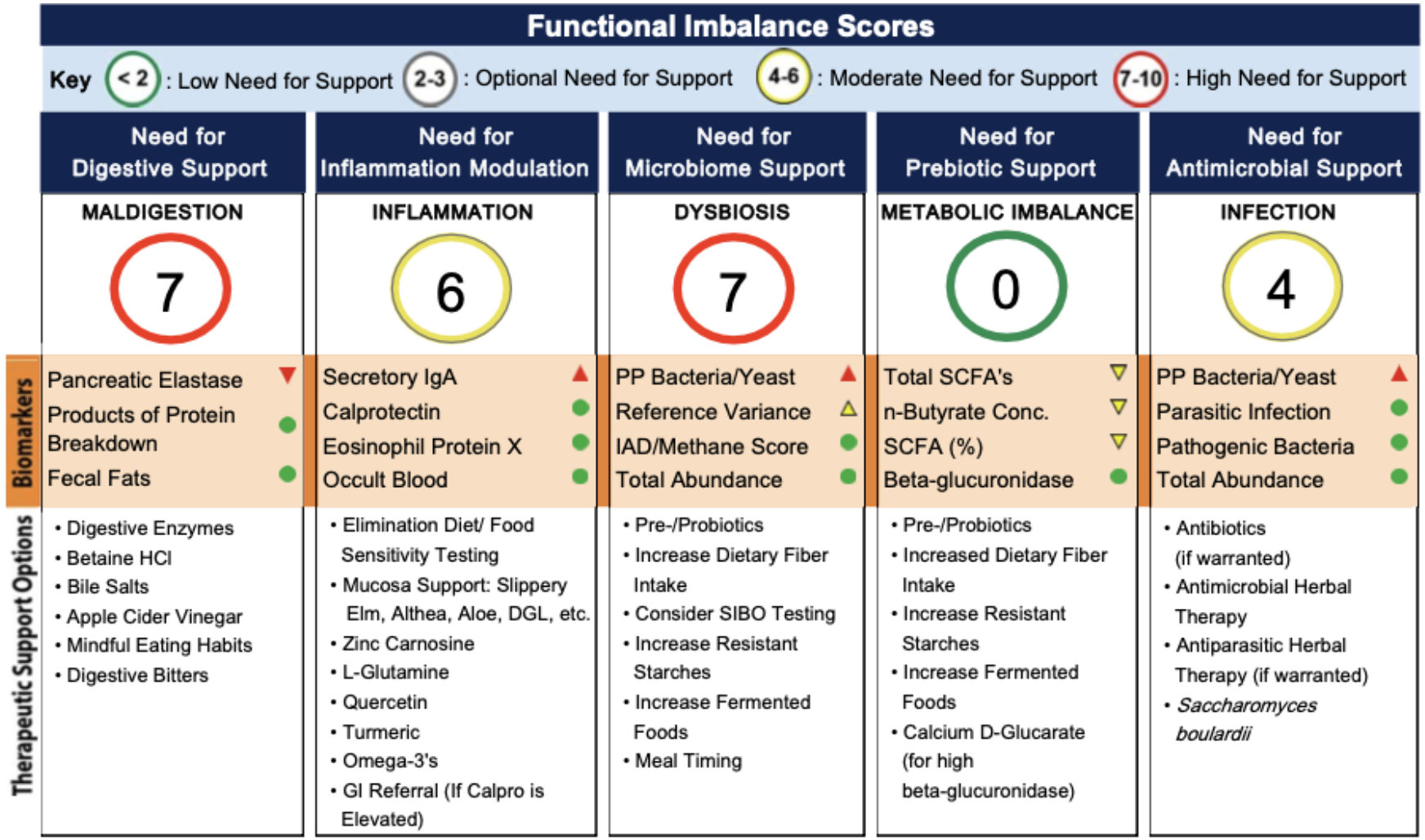
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7
Need for Inflammation Modulation
INFLAMMATION
6
Need for Microbiome Support
DYSBIOSIS
7
Need for Prebiotic Support
METABOLIC IMBALANCE
0
Need for Antimicrobial Support
INFECTION
4
Biomarkers
Pancreatic Elastase
Products of Protein Breakdown
Fecal Fats
Secretory IgA
Calprotectin
Eosinophil Protein X
Occult Blood
PP Bacteria/Yeast
Reference Variance
IAD/Methane Score
Total Abundance
Total SCFA's
n-Butyrate Conc.
SCFA (%)
Beta-glucuronidase
PP Bacteria/Yeast
Parasitic Infection
Pathogenic Bacteria
Total Abundance
Therapeutic Support Options
• Digestive Enzymes
• Betaine HCl
• Bile Salts
• Apple Cider Vinegar
• Mindful Eating Habits
• Digestive Bitters
• Elimination Diet/ Food Sensitivity Testing
• Mucosa Support: Slippery Elm, Althea, Aloe, DGL, etc.
• Zinc Carnosine
• L-Glutamine
• Quercetin
• Turmeric
• Omega-3's
• GI Referral (If Calpro is Elevated)
• Pre-/Probiotics
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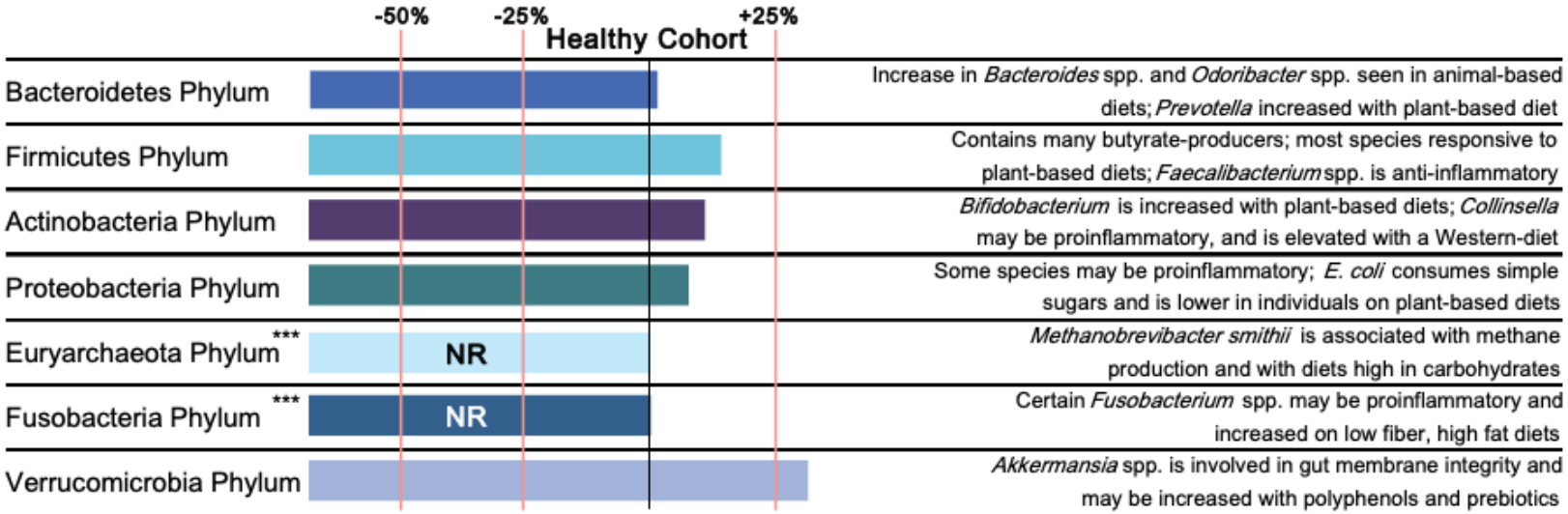

Increase in *Bacteroides* spp. and *Odoribacter* spp. seen in animal-based diets; *Prevotella* increased with plant-based diet

Contains many butyrate-producers; most species responsive to plant-based diets; *Faecalibacterium* spp. is anti-inflammatory

Bifidobacterium is increased with plant-based diets; *Collinsella* may be proinflammatory, and is elevated with a Western-diet

Some species may be proinflammatory; *E. coli* consumes simple sugars and is lower in individuals on plant-based diets

Methanobrevibacter smithii is associated with methane production and with diets high in carbohydrates

Certain *Fusobacterium* spp. may be proinflammatory and increased on low fiber, high fat diets

Akkermansia spp. is involved in gut membrane integrity and may be increased with polyphenols and prebiotics

Digestion and Absorption

Pancreatic Elastase 1 †	88	L	100 / 200	>200 mcg/g
Products of Protein Breakdown (Total*) (Valerate, Isobutyrate, Isovalerate)	3.2			1.8-9.9 micromol/g
Fecal Fat (Total*)	14.3			3.2-38.6 mg/g
Triglycerides	0.4			0.3-2.8 mg/g
Long-Chain Fatty Acids	8.3			1.2-29.1 mg/g
Cholesterol	5.2	H		0.4-4.8 mg/g
Phospholipids	0.4			0.2-6.9 mg/g

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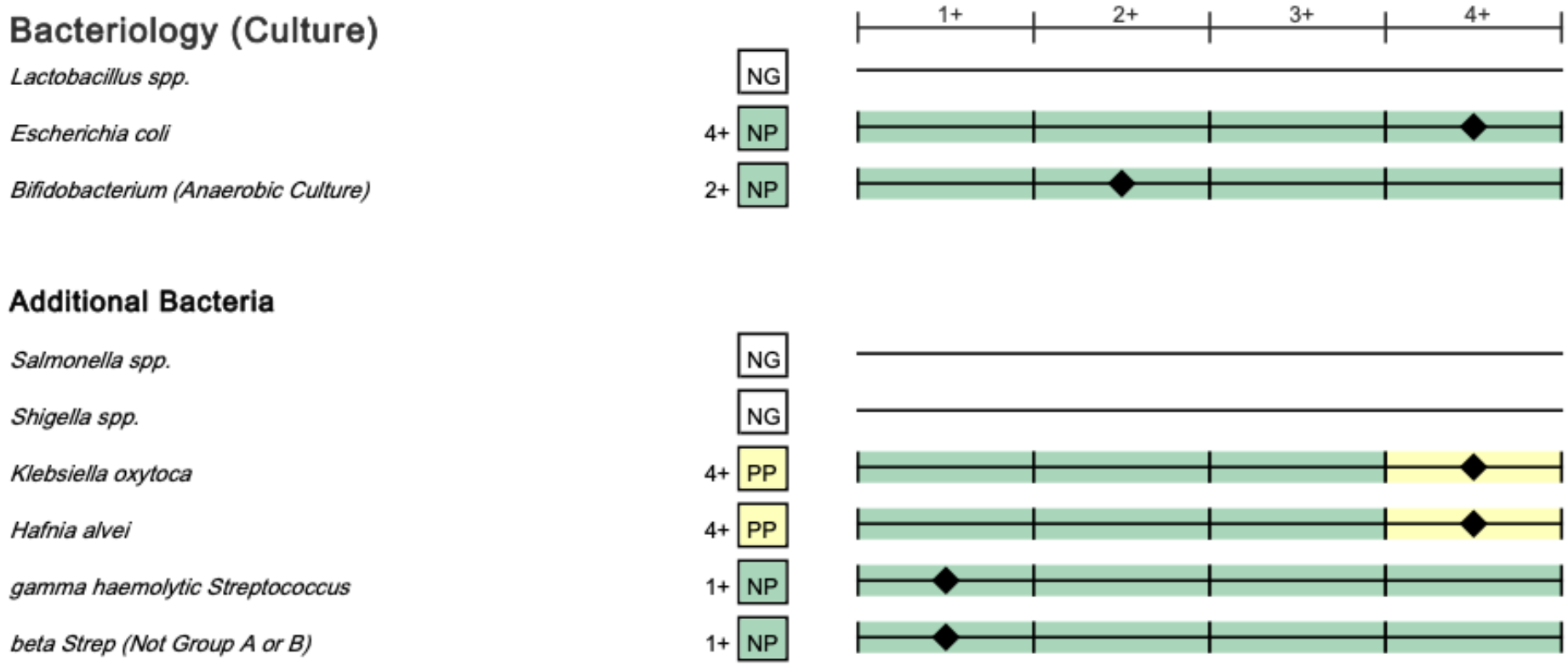
Bacteriology (Culture)



Additional Bacteria

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Strategy Adaptations

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Key Principle

Protocol specialization **requires data** for decision making.

- Blood
- Stool
- Symptoms Questionnaire

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Reach out to your Biogenetix Rep.

Submit your case to the CC team

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