Casual Friday Series

Functional Considerations in IBS and IBD

A Biogenetix Clinical Presentation

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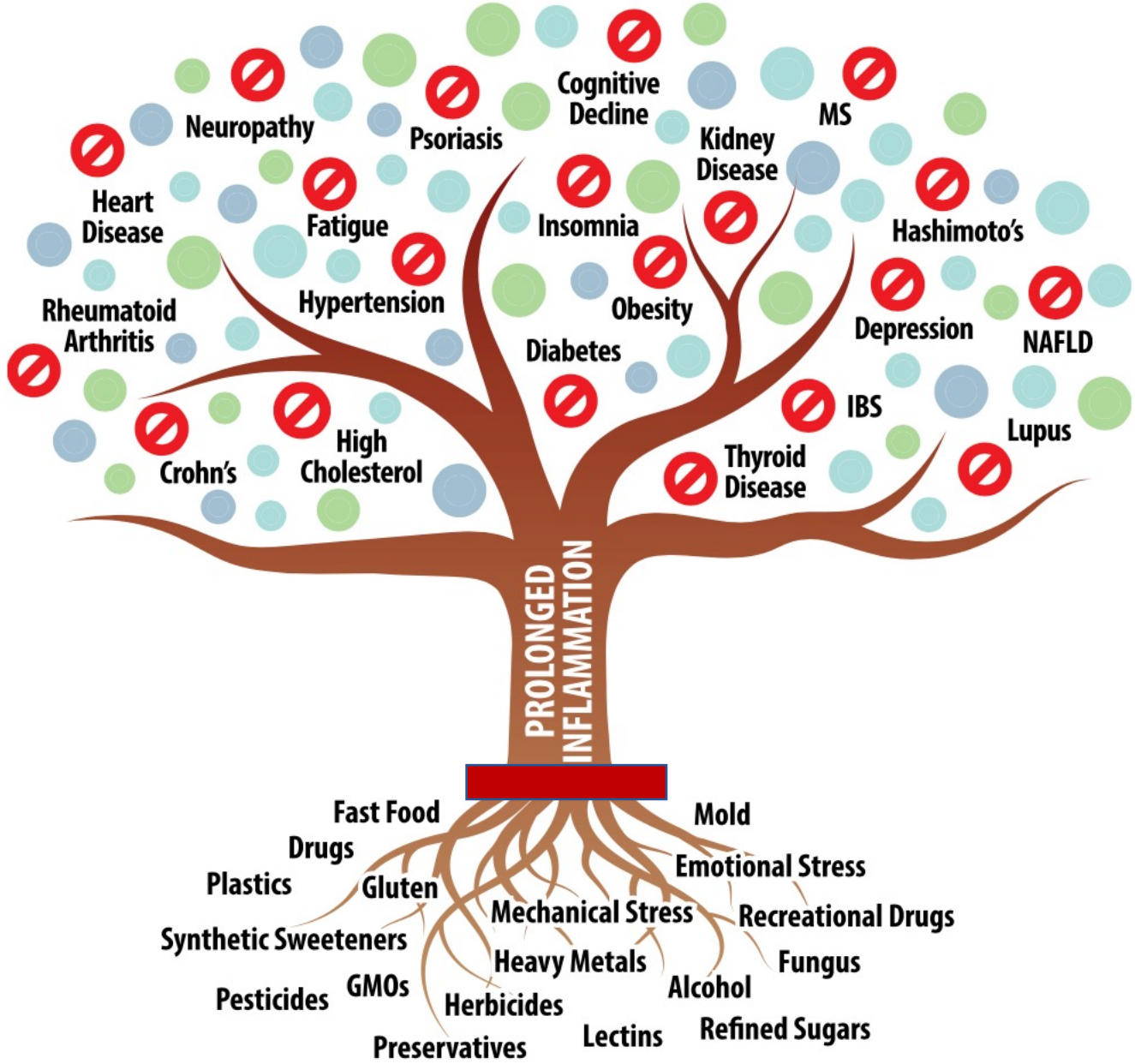
Disclaimer

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


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

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Gastrointestinal Symptom Severity in Irritable Bowel Syndrome, Inflammatory Bowel Disease and the General Population

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Irritable bowel syndrome (IBS) and inflammatory bowel disease (IBD) are gastrointestinal (GI) disorders that are associated with abdominal pain, alteration in bowel habits, relapsing-and-remitting courses, and psychological distress [1]. In comparison to IBS in which disease severity is usually based on patient reported symptoms, current research in IBD has focused on the use of serum, fecal, and colonic mucosal inflammatory biomarkers as surrogates for disease severity [2-4]. Relatively less studied are patient-reported severity of GI symptoms between these groups and the general population (GP).

IBS is a functional bowel disorder in which abdominal pain is associated with changes in bowel habits and disordered defecation. It occurs in 10-20% of the general population and is more predominant in women and those with underlying psychological comorbidities or co-existing functional disorders [5-7]. The etiology of IBS is multifactorial but the pathogenesis is thought to be due to dysregulated brain-gut interactions in which peripheral and central sensitization can occur. Central sensitization at the spinal cord and brain level is associated with increased activation in brain regions involved in emotional arousal and pain modulation [8].

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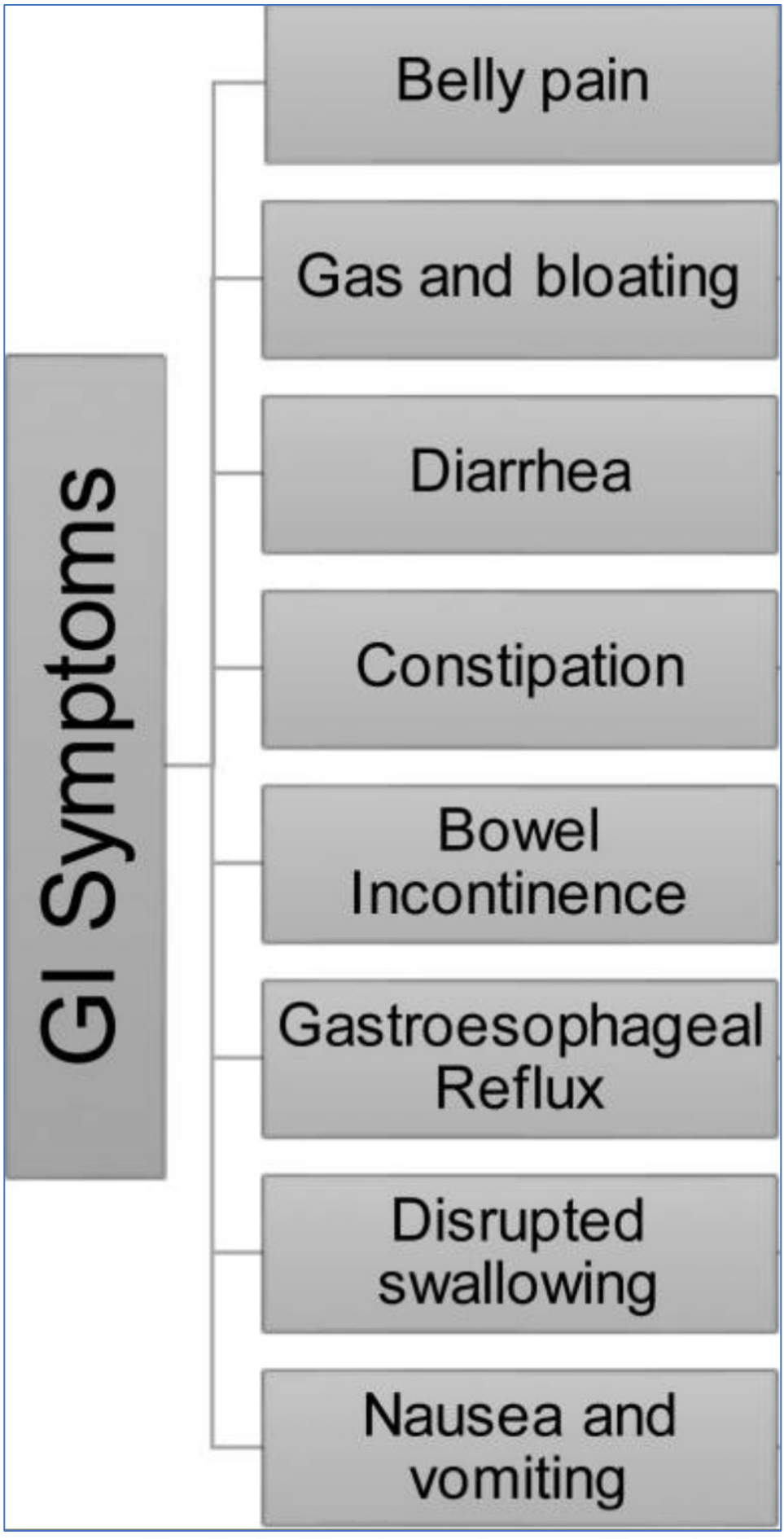
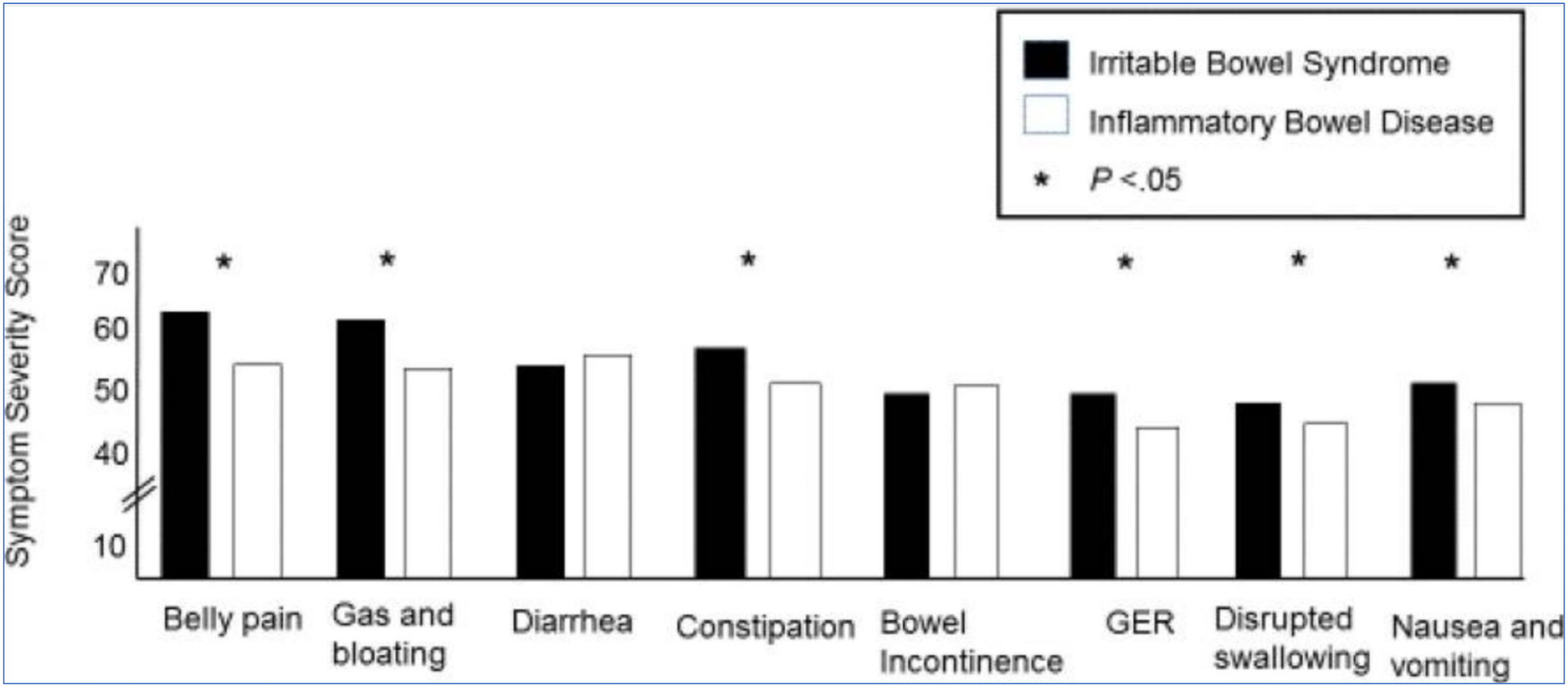
Gastrointestinal Symptom Severity in Irritable Bowel Syndrome, Inflammatory Bowel Disease and the General Population

Crohn's disease (CD) and ulcerative colitis (UC) are chronic immune-mediated disorders classified as inflammatory bowel diseases (IBDs) that affect less than 1% of the US population [9]. Increased prevalence is seen in genetically predisposed individuals and certain ethnic groups. These diseases are thought be caused by chronic dysregulation of mucosal immune function and therapies directed against suppression or modulation of inflammation are generally effective.

Although the extent to which these disease processes have overlapping pathologies is controversial [10], traditional thinking attributes the etiology of pain in IBD to objective inflammatory changes within the bowel as well as associated complications. It is commonly assumed that worsened symptom severity correlates with increased prevalence of inflammatory lesions and complications, however this simplistic view of pain pathogenesis does not account for the fact that patients with IBS often will have similar complaints without objective disease pathology. While IBS and IBD have both been associated with worse general health-related quality of life (HRQOL) [11], it is unclear the extent that specific GI symptoms affect patients. GI symptom questionnaires such as the Gastrointestinal Symptom Rating Scale (GSRS) and Quality of Life in Reflux and Dyspepsia (QOLRD), which measure the degree of GI symptom discomfort, have been developed but have only been evaluated in patients with reflux disease and IBS and may not be applicable to a wider range of GI disorders and the GP [12-15].


GI Symptoms
Belly pain
Gas and bloating
Diarrhea
Constipation
Bowel Incontinence
Gastroesophageal Reflux
Disrupted swallowing
Nausea and vomiting


Irritable Bowel Syndrome
Inflammatory Bowel Disease
* P <.05
Symptom Severity Score
70
60
50
40
10
Belly pain
Gas and bloating
Diarrhea
Constipation
Bowel Incontinence
GER
Disrupted swallowing
Nausea and vomiting

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The greater GI symptom severity seen in IBS patients is likely multifactorial. Patients' illness experience reflects upon how they perceive their sickness in the context of psychosocial and demographic conditions [31]. IBS is a stress-sensitive disorder in which stress is associated with enhanced colonic motility and enhanced visceral perception [32-33]. Hypervigilance, an increased attention to noxious stimuli, or an increased tendency to report sensations as bothersome has been demonstrated in IBS [34]. In fact, patients with UC in remission with IBS symptoms were found to have worse GI symptoms, psychological distress and poorer physical and mental quality of life than patients with UC in remission without IBS [35]. Although not directly examined in this study, these neurobiological and behavioral changes may explain why there is significantly greater severity of GI symptoms in IBS than IBD and the GP. Prior brain imaging studies have suggested that patients with IBS have increased activation of limbic and paralimbic circuits involved with emotional stress and pain, while patients with ulcerative colitis and healthy controls show an inhibition of these central pathways [36]. This is supported clinically by the fact that IBD patients showing mild inflammation of their disease have rectal hyposensitivity (i.e., lower sensitivity) when undergoing rectal distention studies compared to IBS patients [37].

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Prior studies have demonstrated that up to 30-40% of patients with IBS will also report coexisting symptoms of GER [39-40], but the prevalence of IBD patients reporting GER symptoms has not been well studied. In the GP, the prevalence of GER ranges from 10-20% in Western populations [41]. A possible explanation for the decreased upper GI symptom severity in IBD patients is that that IBD is predominantly a disease that affects the distal bowel (ileum and colon) with rare involvement of the upper gastrointestinal tract, and in comparison, these patients may experience relatively less severe upper tract symptoms when contrasted to their severity of their lower GI symptoms. Evidence supporting this is based on prior studies demonstrating individuals distracted from pain will often report diminished pain severity [42-43]. This

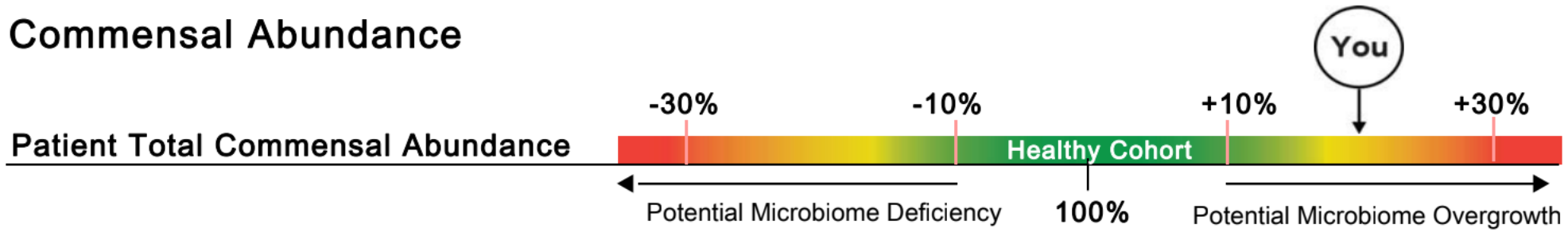
Functional Imbalance Scores

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**
	10	0	5	8	0
Biomarkers	Pancreatic Elastase ▼ Fecal Fats △ Products of Protein Breakdown ▽	Calprotectin ● Eosinophil Protein X ● Secretory IgA ● Occult Blood ●	IAD/Methane Score △ Total Abundance △ PP Bacteria/Yeast ● Reference Variance ●	Total SCFA's ▼ n-Butyrate Conc. ▼ SCFA (%) ● Beta-glucuronidase ●	Total Abundance △ Parasitic Infection ● Pathogenic Bacteria ● PP Bacteria/Yeast ●
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*

Commensal Microbiome Analysis

Commensal Abundance



2200 GI Effects™ Comprehensive Profile - Stool

Interpretation At-a-Glance									
Commensal Bacteria	Patient Results Out of Reference Range	Genova Diagnostics Commensal Bacteria Clinical Associations*							
		IBS	IBD	Metabolic Syndrome	Chronic Fatigue	Auto-immune	Type 2 Diabetes	High Blood Pressure	Mood Disorders
Bacteroidetes Phylum									
Bacteroides-Prevotella group		↑	↑	↑	↑	↑	↑	↑	↑
Bacteroides vulgatus	H	↑			↑	↑		↑	↑
Barnesiella spp.									
Odoribacter spp.									
Prevotella spp.		↑		↑	↑	↑		↑	↑
Firmicutes Phylum									
Anaerotruncus colihominis		↑	↑	↑	↑	↑	↑	↑	↑
Butyrivibrio crossotus									
Clostridium spp.									
Coprococcus eutactus		↑			↑	↑		↑	↑
Faecalibacterium prausnitzii		↑				↑			↑
Lactobacillus spp.									
Pseudoflavonifractor spp.	H	↑	↑	↑	↑	↑	↑	↑	↑
Roseburia spp.			↓						
Ruminococcus spp.		↓↑	↓	↓	↓	↓↑	↓↑	↓↑	↓↑
Veillonella spp.		↑	↑	↑	↑	↑	↑		↑

Interpretation At-a-Glance

Biomarker	Patient Results Out of Reference Range	Genova Diagnostics Biomarker Clinical Associations*							
		IBS	IBD	Metabolic Syndrome	Chronic Fatigue	Auto-immune	Type 2 Diabetes	High Blood Pressure	Mood Disorders
Pancreatic Elastase	L	↓	↓	↓	↓	↓	↓	↓	↓
Products of Protein Breakdown (Total)							↑↓		
Fecal Fat (Total*)		↑		↑	↑	↑	↓↑	↑	↑
Triglycerides		↑			↑	↑	↑	↑	↑
Long-Chain Fatty Acids	H	↑			↑	↑	↓↑	↑	↑
Cholesterol							↓↑	↑	
Phospholipids		↑	↑	↑	↑	↑	↑	↑	↑
Calprotectin			↑					↑	

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**
	1	8	2	7	0
Biomarkers	Fecal Fats △ Products of Protein Breakdown ▽ Pancreatic Elastase ●	Secretory IgA ▲ Calprotectin ● Eosinophil Protein X ● Occult Blood ●	Reference Variance △ Total Abundance △ IAD/Methane Score ● PP Bacteria/Yeast ●	Beta-glucuronidase ▲ Total SCFA's ● n-Butyrate Conc. ● SCFA (%) ●	Total Abundance △ Parasitic Infection ● Pathogenic Bacteria ● PP Bacteria/Yeast ●
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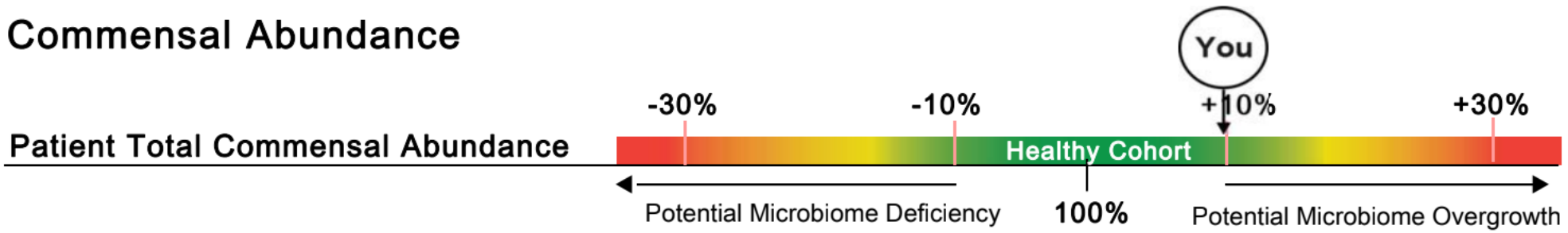

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Barnesiella spp.	H								
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Prevotella spp.		↑		↑	↑	↑		↑	↑
Firmicutes Phylum									
Anaerotruncus colihominis		↑	↑	↑	↑	↑	↑	↑	↑
Butyrivibrio crossotus									
Clostridium spp.									
Coprococcus eutactus		↑			↑	↑		↑	↑
Faecalibacterium prausnitzii		↑				↑			↑
Lactobacillus spp.									
Pseudoflavonifractor spp.	H	↑	↑	↑	↑	↑	↑	↑	↑
Roseburia spp.			↓						
Ruminococcus spp.		↓↑	↓	↓	↓	↓↑	↓↑	↓↑	↓↑
Veillonella spp.		↑	↑	↑	↑	↑	↑		↑

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Products of Protein Breakdown (Total)							↑↓		
Fecal Fat (Total*)		↑		↑	↑	↑	↓↑	↑	↑
Triglycerides		↑			↑	↑	↑	↑	↑
Long-Chain Fatty Acids		↑			↑	↑	↓↑	↑	↑
Cholesterol	H						↓↑	↑	
Phospholipids		↑	↑	↑	↑	↑	↑	↑	↑
Calprotectin			↑					↑	
Eosinophil Protein X (EPX)			↑						
Fecal secretory IgA	H	↑	↑	↑	↑	↑	↑	↑	↑
Short-Chain Fatty Acids (SCFA) (Total)					↓	↓			
n-Butyrate Concentration				↓					
n-Butyrate %									
Acetate %					↑↓		↓↑		
Propionate %				↑			↑	↑	
Beta-glucuronidase	H					↑↓			↑↓

SUGGESTED STRATEGY

GI ResQ: 1 scoop AM & PM
Multi+ Powder: 2 scoops AM & PM
UltraBiotix: 1 sachet/capsule AM

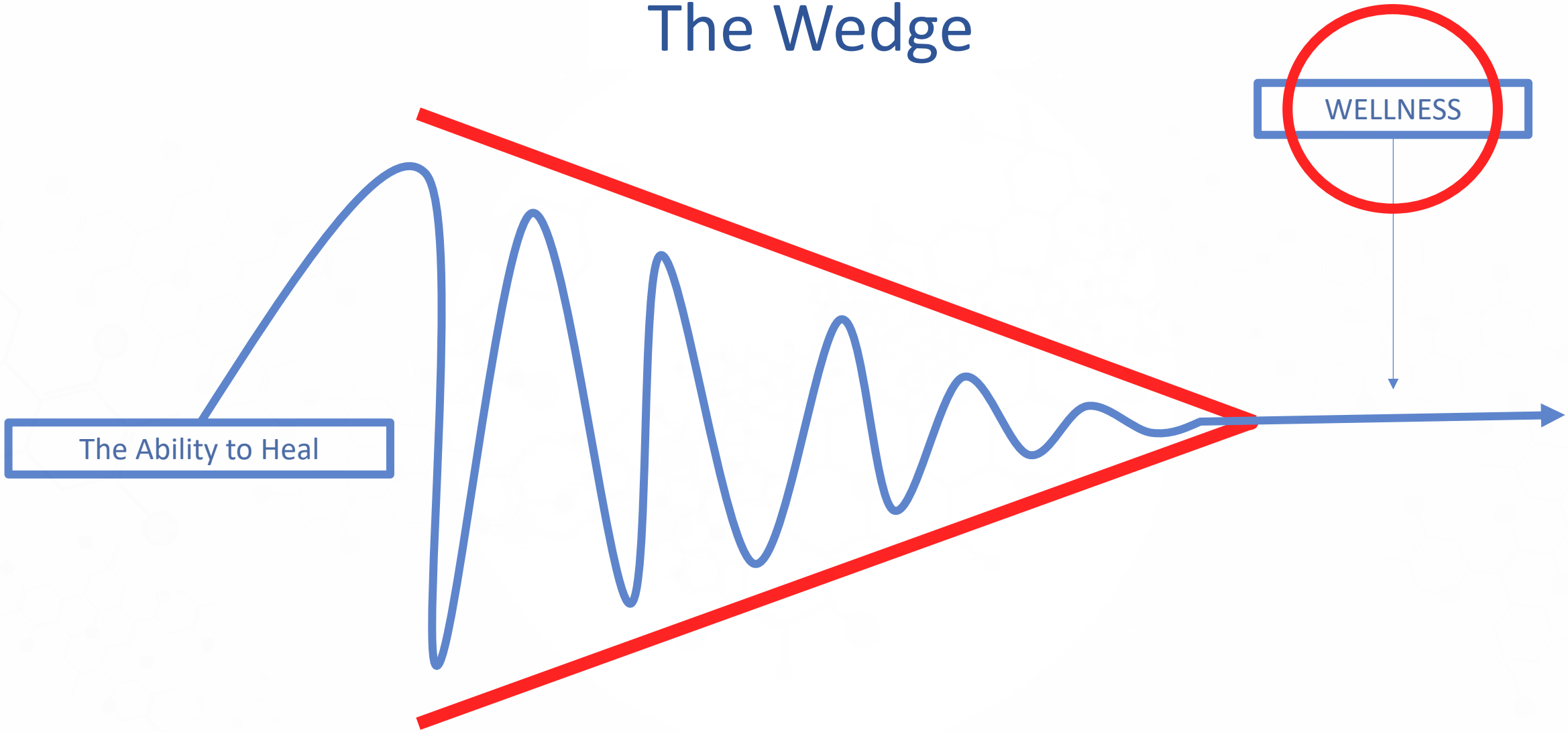
Mix into 10 ounces of cold water in the morning and the evening.

Questions? Our sales team is here for you: (833) 525-0001

30-DAY SUPPLY

1 GI ResQ
1 Multi+ Powder
1 UltraBiotix

The Wedge



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