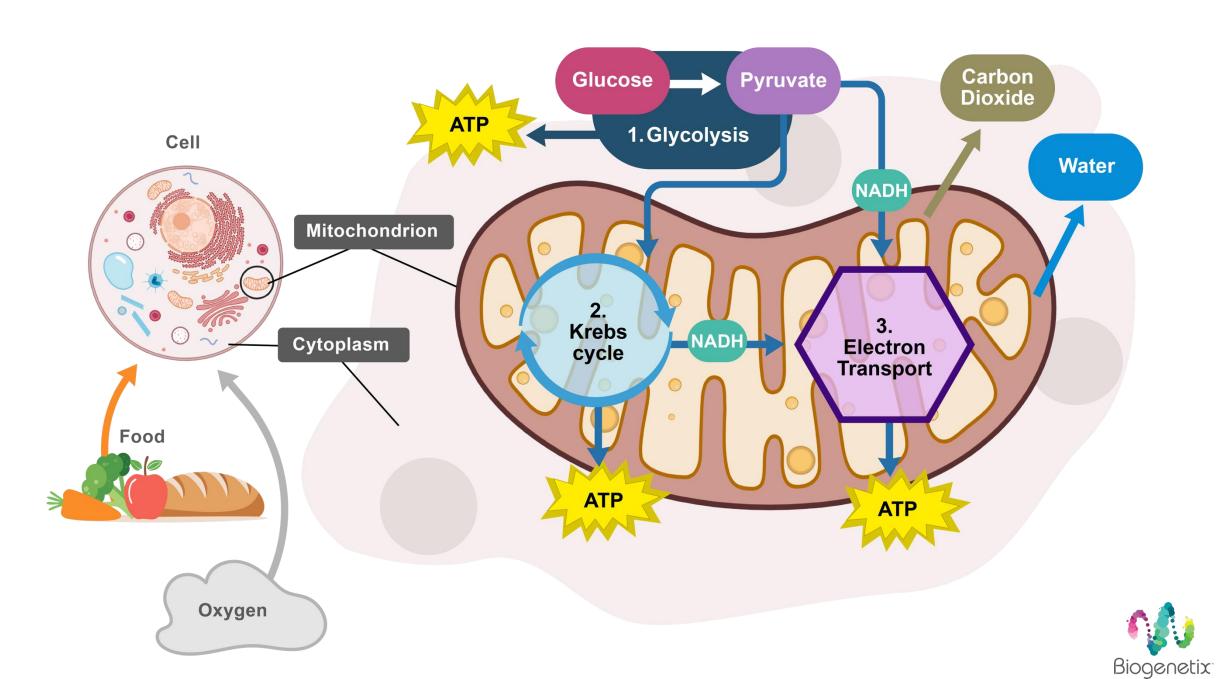
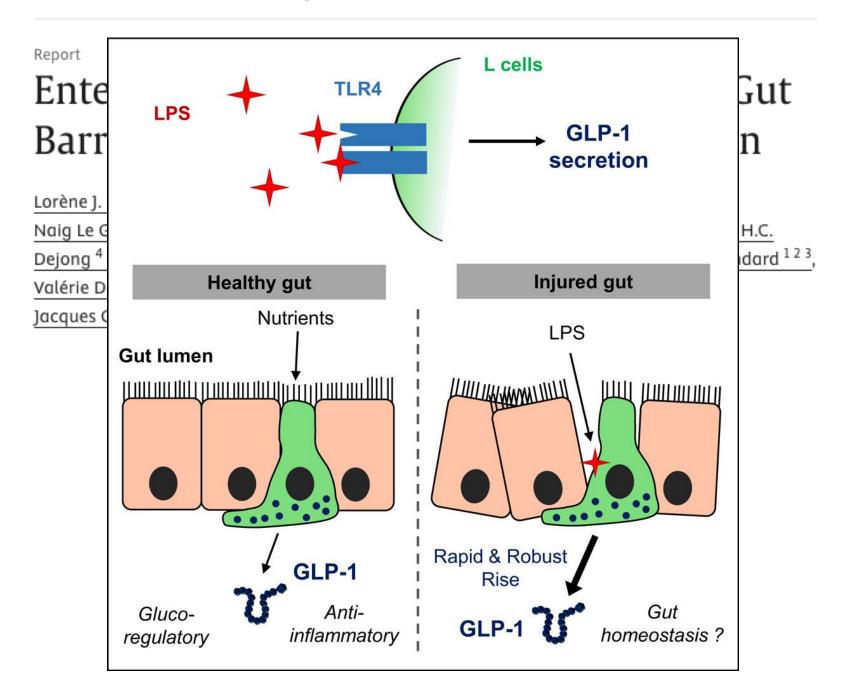
#### **Casual Friday Presents**

# Peptide Primer III (the FM plan)









#### **Concept Overview**

Lab review concept is a way to forecast metabolic and tolerance outcomes from GLP-1 receptor agonist therapy.

#### It integrates:

- Organic Acids Test (OAT): mitochondrial and neurochemical readiness
- GI Effects + Zonulin: gut barrier, microbiome, and detox balance

Goal: Identify Strong, Moderate, or Poor Responders to optimize intervention sequencing.



### **Optimization Strategy**

#### Assess these 8 areas of focus:

- 1 Mitochondrial Efficiency
- <sup>2</sup> Neurotransmitter Balance
- 3 Detoxification & Phase II Capacity
- 4 Digestive Function
- 5 Barrier Integrity (Zonulin, Calprotectin)
- 6 Microbiome Composition (SCFAs,
- diversity)
- 7 Dysbiosis & Pathogenic Burden
- 8 Inflammation / Oxidative Stress



# Patients likely to struggle: stool findings

Focus	Typical Findings	Mechanism
Barrier integrity	↑ Zonulin family peptides (>75 ng/mL)	Tight-junction leak → LPS exposure → GLP-1 receptor resistance, leptin resistance
Inflammation	↑ Calprotectin, ↑ EPX	Cytokine load → central satiety blunting
Microbial balance	<ul><li>↓ Butyrate producers (Roseburia,</li><li>F. prausnitzii) / ↑ Proteobacteria</li></ul>	Low SCFA signaling → poor endogenous GLP-1 release
Digestive inefficiency	↓ Elastase, ↑ fecal fats	Slowed gastric emptying intolerance, nausea
Detox/estrogen recirculation	↑ β-glucuronidase	Hepatic congestion → drug sensitivity, plateau



# Patients likely to respond well: stool findings

Focus	Typical Findings	Mechanism
Barrier integrity	Normal zonulin, low calprotectin	Minimal endotoxemia → clean signaling
Microbiome health	High diversity, robust butyrate	Improved L-cell GLP-1 secretion
Digestive function	Elastase WNL, low fecal fat	Tolerates delayed gastric emptying
Balanced β-glucuronidase	WNL	Normal detox handling of peptide drug
SCFA ratio	Butyrate > propionate ≈ acetate	Satiety and insulin sensitivity support



FAT MALABSORPTION			
Total Fecal Fat	50.8 mg/g	2.9~37.5	High levels of fecal fat are suggestive of maldigestion or malabsorption. Consider cholagogues, betaine HCL, pancreatic enzyme supplementation to improve outcome. Phosphatidyl choline, serine and inositol can be considered when phospholipids are low.
Total Fecal Triglycerides	0.7 mg/g	0.3~2.5	
Long chain fatty acids	23.6 mg/g	0.9~28.1	
Total Cholesterol	1.9 mg/g	0.5~5.3	
Total Phospholipids	21.7 mg/g	0.3~6.4	High levels of fecal fat are suggestive of maldigestion or malabsorption. Consider cholagogues, betaine HCL, pancreatic enzyme supplementation to improve outcome. Phosphatidyl choline, serine and inositol can be considered when phospholipids are low.

SHORT CHAIN FATTY ACIDS			
Acetate	41.3 %	60.2~72.7	SCFA supplements are most commonly found as butyric acid salts.  Herbal medicines that can increase SCFA levels include berberine, resistant starch. Best sources of resistant starches include: Green bananas or banana flour to your daily routine works well. Other options include: raw plantains, raw potato starch, cooked and cooled rice, and legumes. Increasing levels of SCFA producing bacteria is important.
Butyrate	12.0 %	5.1~12.4	
Propionate	41.6 %	15.4~30.3	SCFA supplements are most commonly found as butyric acid salts.  Herbal medicines that can affect SCFA levels include berberine, resistant starch. Best sources of resistant starches include: Green bananas (adding green banana flour to your daily routine works well. Raw plantains, Raw potato starch, Cooked and cooled rice, Legumes
Valerate	2.7 %	0.8~3.5	



slgA	543.4 mcg/g	426.0~1450. 0	
ß-glucuronidase	1674 U/mL	≤2300	
Fecal Occult Blood	8.9 mcg/g	≤10.0	
рН	6.1	6.1~7.8	
Fecal Zonulin	337.6 ng/mL	25.1~160.8	Elevation indicative of intestinal permeability. Addressing gut dysbiosis and low diversity if any. Checking for food sensitivities at peptide and protein level recommended.
Fecal Anti Gliadin	107.9 U/L	≤148.0	

|--|



# Markers Suggesting Poor Response or Side Effects: OATs

- •Elevated lactate, pyruvate, or succinate: impaired mitochondrial entry of carbs → poor ATP signaling, less energy adaptation to calorie restriction.
- •High adipate/suberate:  $\beta$ -oxidation defect  $\rightarrow$  poor fat metabolism.
- •**High quinolinate / low kynurenate:** neuroinflammation → appetite dysregulation and poor GLP-1 satiety response.
- •Elevated arabinose, tartaric acid, or D-lactate: yeast/bacterial overgrowth → inflammatory burden → leptin/GLP-1 resistance.
- •Low B-vitamin intermediates (esp. methylmalonate, xanthurenate): cofactor insufficiency for mitochondrial enzymes and neurotransmitter synthesis.

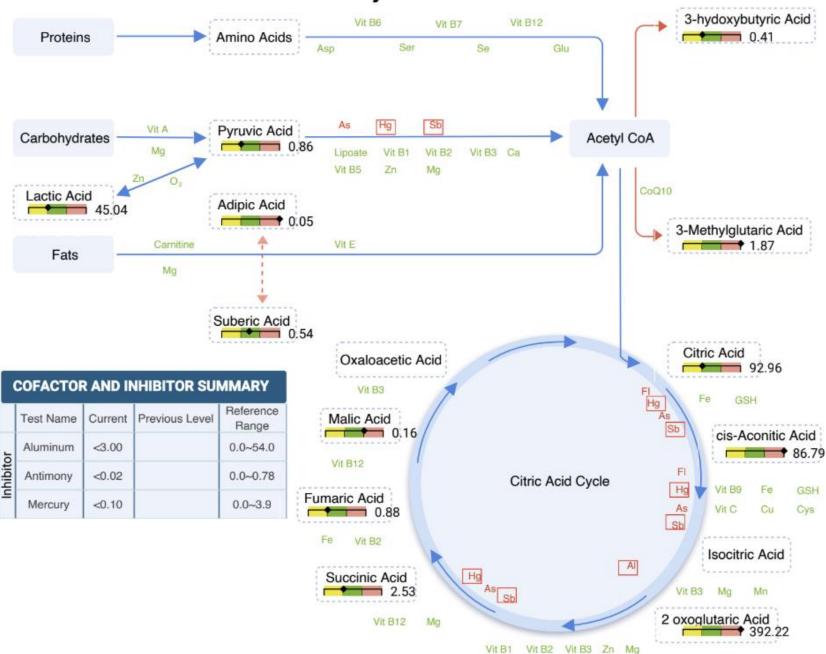


# Markers Suggesting Strong Response: OATs

- •Efficient citric acid cycle: normal/low intermediates → high mitochondrial flexibility.
- •Balanced dopamine/serotonin metabolites: normal HVA/VMA ratios → stable appetite control.
- •Low oxidative stress (8-OHdG, pyroglutamate in range): low inflammatory tone → better receptor sensitivity.

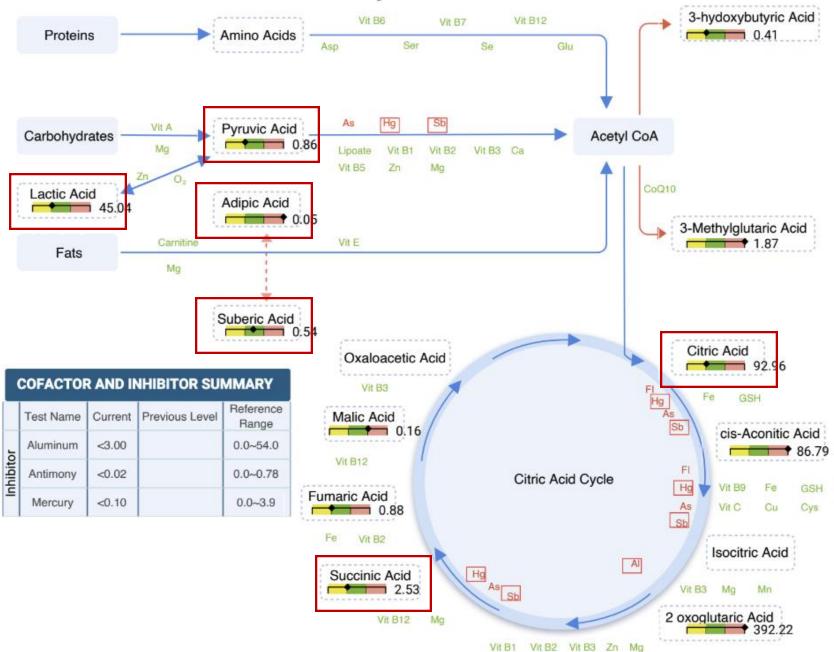


#### **Krebs Cycle At-A-Glance**





#### **Krebs Cycle At-A-Glance**





### **Blending stool with Organic Acids Testing:**

System	OAT Marker Cluster	GI Effects Marker	Predictive Outcome
Mitochondrial Efficiency	Citrate, succinate, adipate	_	Energy adaptation to calorie restriction
Gut Permeability / Inflammation	Quinolinic, pyroglutamate	Zonulin peptides, calprotectin	Appetite resistance vs. sensitivity
Microbial Metabolites	D-lactate, arabinose	Dysbiosis pattern, SCFA ratio	Drug tolerance & nausea
Detox / Phase III	Pyroglutamate, sulfate	β-glucuronidase, fecal bile acids	Plateau vs. steady weight loss



# The Antigenic Cloud

