Diagnostic Solutions laboratory

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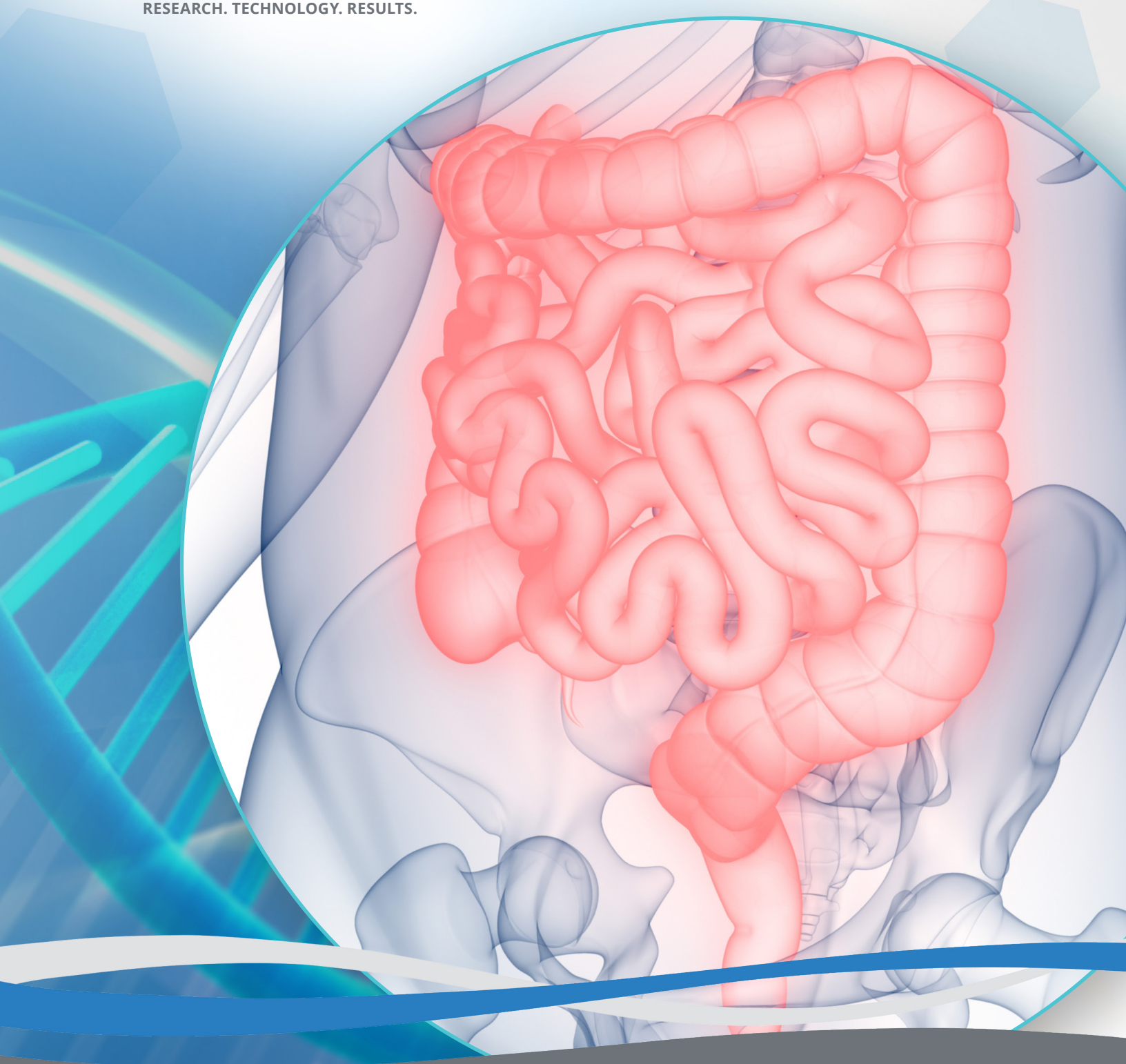


2025 EDITION

REFERENCE GUIDE

GI-MAP® Add-On Test

Our mission: to deliver innovative, accurate and clinically relevant diagnostic testing in a timely and cost-effective manner

StoolOMX™

Advanced Bile Acid Testing and Short Chain Fatty Acid Evaluation

StoolOMX™

***GI-MAP*® Add-On Test**

FIRST OF ALL
THANK YOU
FOR CONSIDERING US!

"At Diagnostic Solutions Laboratory, we're not content with the range of clinical testing currently available to practitioners. We believe that every patient should achieve optimal health, and we're driven to give clinicians the tools to do so. Our mission, therefore, is to use our resources to bring the most advanced, innovative, and clinically relevant testing to healthcare providers worldwide."

Tony Hoffman
President and CEO

CONTENTS

SUPPORT INFO

877-485-5336

StoolOMX™

StoolOMX measures gut microbial metabolites—a comprehensive set of bile acids (BA) and short chain fatty acids (SCFA) via LC-MS/MS. This data offers an in-depth understanding of the stool metabolome and its clinical applications. The StoolOMX profile can be ordered as an **add-on** to the GI-MAP.

BILE ACIDS

Primary bile acids are natural products produced in the liver from cholesterol synthesis. These primary bile acids are conjugated with either taurine or glycine to increase solubility and are stored in the gallbladder as bile.

Primary, conjugated bile acids are the main component in bile. At mealtime, they aid in the emulsification and absorption of dietary fats in the small intestine. After contributing to fat absorption, the majority (~95%) of primary bile acids are reabsorbed in the distal ileum and returned to the liver via the portal vein—a process called enterohepatic circulation. A small portion (~5%) of primary bile acids will reach the colon, where they are metabolized by gut bacteria and deconjugated (glycine and taurine removed) to produce unconjugated secondary bile acids. Deconjugation is generally favorable.

Commensal gut bacteria deconjugate bile acids to facilitate their reabsorption and recycling while also preparing them for further bacterial modification.

Under normal physiological conditions, most bile acids are reabsorbed, and very few bile acids are excreted in stool. Under abnormal physiological conditions, excess bile acids enter the colon and can promote inflammation and diarrheal symptoms while exerting negative effects on commensal bacteria.

Measuring concentrations and ratios of bile acids in stool can offer root-cause insights into digestive symptoms, malabsorptive disorders, immune system regulation, and even metabolic impacts. Furthermore, altered patterns of bile acid metabolites have emerging disease state associations and can be used as part of diagnostic workup and treatment management in conditions such as bile acid diarrhea (BAD), inflammatory bowel disease (IBD), and irritable bowel syndrome (IBS) (-C, -D, -M).

StoolOMX measures 25 bile acid metabolites in total concentrations, percents, and ratios. This data offers an in-depth understanding of the stool metabolome and impactful clinical considerations.

BILE ACIDS – SUMMARY

Total Bile Acids Concentration

- **An absolute concentration of total bile acids in stool measured in ng/g**
  - » Reflects the amount of bile acids reaching the colon and not reabsorbed in the distal ileum after digestion
- **Elevated Total Bile Acids:**
  - » Excess/elevated bile acids in stool is an indication of bile acid malabsorption (BAM) and often results in symptoms such as diarrhea (bile acid diarrhea – BAD)
  - » Quantification of bile acids may be part of a comprehensive workup for irritable bowel syndrome with diarrhea (IBS-D), as it is currently estimated that up to 1/3 of IBS-D patients present with BAD
  - » Elevated stool bile acids may suggest altered bile acid production or reabsorption, which could be due to liver disease, gallbladder dysfunction, intestinal issues, or diet composition
- **Low Total Bile Acids:**
  - » Low total bile acid concentrations in stool are generally favorable but may be indicative of slow transit or constipation

Total Primary Bile Acids Percent

- **Percent concentration of primary bile acids in stool**
  - » Primary bile acids should be present in very low concentrations in stool
  - » Primary bile acids in the colon can be damaging to the gut lining and can have antimicrobial action on the microbiome
  - » Higher levels of primary bile acids in a stool sample are indicative of excess production or BAM and are associated with an array of GI symptoms and disease states, including IBD
  - » See individual primary bile acid concentrations on page 2 of the StoolOMX report for further analysis and associations

Total Secondary Bile Acids Percent

- **Inverse of total primary bile acids in stool**
  - » Listed as percent concentration of secondary bile acids in stool
  - » Secondary bile acids should be present in high concentrations in stool
  - » Secondary bile acids play diverse roles in regulating intestinal motility, gut barrier function, metabolism, and immune balance
  - » In general, a higher number of secondary bile acids relate to a healthier, more diverse microbiome and normal physiological GI function
  - » See individual secondary bile acid concentrations on page 2 of the StoolOMX report for further analysis and associations

Table 9. This section represents the most abundant bile acids.

PERCENTAGE BREAKDOWN OF MOST ABUNDANT BILE ACIDS		
SECONDARY BILE ACIDS	**FUNCTION**	**AVERAGE PERCENTAGE†**
Deoxycholic Acid (DCA)	DCA is a major secondary bile acid that is formed from the bacterial metabolism of CA. Low levels observed in ulcerative colitis (UC). Elevated levels are associated with liver and colon cancer and metabolic imbalance. Implicated in modulating the immune response by inhibiting pro-inflammatory cytokine production. Can inhibit *Lactobacillus* spp., *Bifidobacterium* spp., and *Bacteroides fragilis*.	**48%**
Lithocholic Acid (LCA)*	LCA is a major secondary bile acid that is formed from the bacterial metabolism of CDCA. Low levels observed in UC. Elevated levels are associated with liver and colon cancer and may contribute to blood sugar dysregulation. Modulates immune responses at normal levels.	**27%**
Isolithocholic Acid (Iso-LCA)	Prevalent secondary bile acid in stool that has positive associations with longevity. Influences metabolic health, interacts with cellular receptors, and modulates immune responses at normal levels. Influences the differentiation and function of T cells, particularly T helper 17 (Th17) cells, which play a critical role in the inflammatory response.	**8%**
Other	The remaining primary and secondary bile acid metabolites measured on StoolOMX.	**17%**

** On StoolOMX, the LCA value is a summation of LCA + Allo-LCA.; † Reference set at 50th percentile.*

LCA/DCA Ratio

LCA and DCA are secondary bile acids formed from CDCA and CA in the colon. CDCA is converted to LCA, and CA is converted to DCA. This ratio can be useful in determining the risk of certain disease states and conditions.

LCA is thought to be more toxic than DCA due to its inhibitory effects on antioxidant pathways. An elevated ratio can be seen in patients with gallstones and after cholecystectomy. A higher ratio is also associated with an increased risk for colon cancers, while a lower ratio may indicate a reduced cancer risk.

Bile Acids: Therapeutic Applications

Bile acids and the microbiome have a bidirectional relationship and impact. It is important to first evaluate patient symptoms, such as stool frequency and Bristol stool type.

Always analyze StoolOMX results with GI-MAP findings and support accordingly. It is important to assess liver function, as bile acids are originally produced in the liver.

If there are significant imbalances in the bile acids summary, review the list of individual bile acids on page 2 of the StoolOMX report.

- **A healthy microbiome is typically associated with:**
  - » Total bile acid concentration in normal range
  - » High secondary bile acid percent
- **An unhealthy microbiome is typically associated with:**
  - » Elevated total bile acid concentration
  - » High primary bile acid percent, as the microbiome is responsible for converting primary bile acids to secondary bile acids
- **Common GI-MAP patterns that may be associated with abnormal StoolOMX results:**
  - » Presence of pathogens
  - » Insufficiency dysbiosis
  - » Increased *Firmicutes:Bacteroidetes* ratio
  - » Overgrowth of GI-MAP (report page 3) inflammatory opportunists
  - » Elevated Steatocrit
  - » Decreased Elastase-1
  - » Elevated Calprotectin
- **Lifestyle Considerations**
  - » Evaluate macronutrient composition of diet with respect to calories from fat. Patients may need to adopt a lower fat diet if StoolOMX results are abnormal.
  - » Evaluate and ensure adequate dietary fibers
  - » Anti-inflammatory dietary practices
  - » Support liver and gallbladder
  - » Incorporate regular exercise
- **Medication/Supplement Considerations for BAM**
  - » Bile acid sequestrants
    - – ex: cholestyramine, colesevelam
  - » Tauroursodeoxycholic acid (TUDCA)
  - » Digestive support as needed
    - – Digestive enzymes, betaine HCL, digestive bitters, ox bile, etc.
  - » Activated charcoal
  - » Pectin fibers
  - » Probiotics
  - » Polyphenols such as resveratrol

Table 10. GI-MAP microbes involved in deconjugation of primary bile acids to secondary bile acids.

GI-MAP MICROBES
***Firmicutes* phyla**
***Bacteroidetes* phyla**
***Escherichia* spp.**
***Bacteroides* spp.**
***Bifidobacterium* spp.**
***Enterococcus* spp.**
***Lactobacillus* spp.**
***Methanobacteriaceae* family**

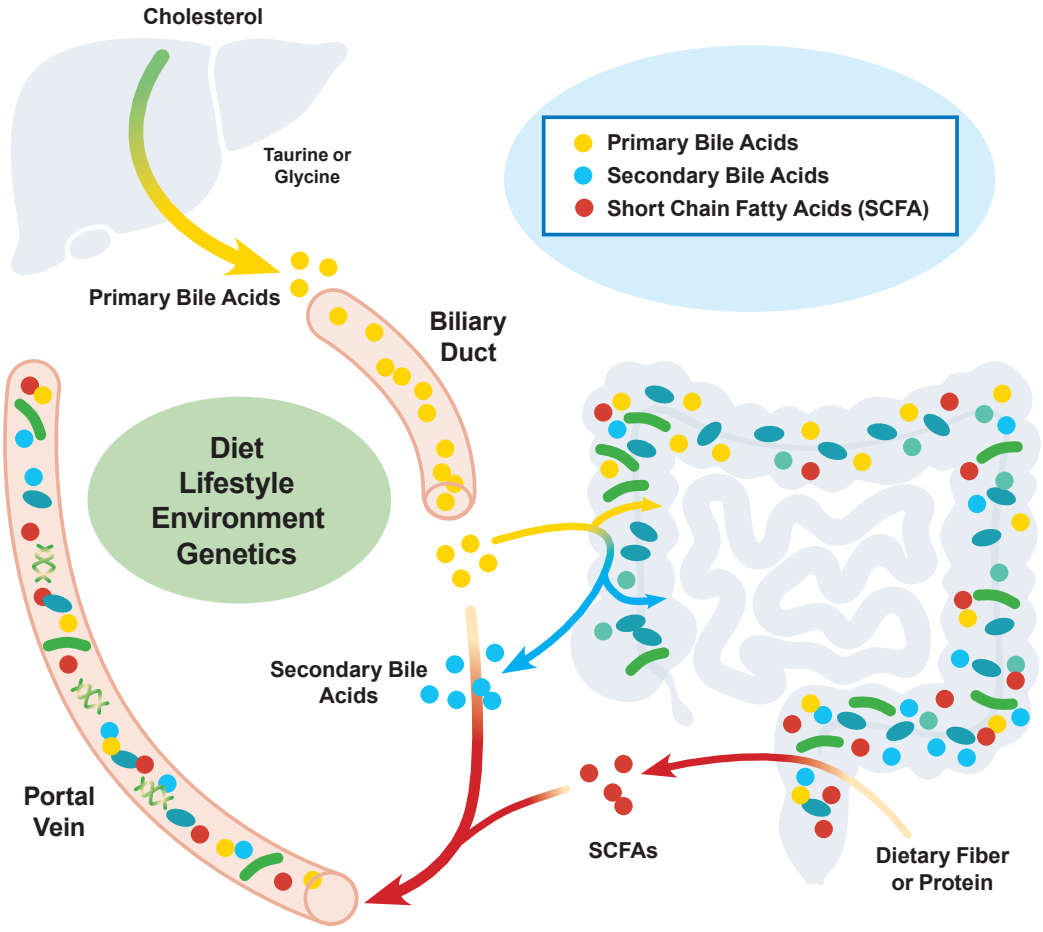


Figure 6. Bile Acids and Fatty Acids Overview.

Primary bile acids are synthesized from cholesterol in the liver and conjugated with either taurine or glycine. They are stored in the gallbladder and released during digestion to assist with the absorption of fat and fat-soluble vitamins. Normally, ~95% of primary bile acids are reabsorbed via the portal vein, while ~5% are metabolized by gut bacteria to produce secondary bile acids.

Saccharolytic short chain fatty acids (SCFAs) are primarily metabolites of dietary fiber fermentation in the gut while proteolytic branched chain fatty acids (BCFAs) are metabolites of protein fermentation. Acetate, propionate, and butyrate are three major SCFAs, which account for ~90% of the SCFAs produced by gut microbiota. SCFAs are known to have numerous health effects and can enhance fecal excretion of bile acids.

SHORT CHAIN FATTY ACIDS – SUMMARY

Short chain fatty acids (SCFAs) are small (0-6 carbon) saturated fatty acids that are produced from the microbiome (gut bacteria) by fermentation of food substrates. Fermentation of carbohydrate—called saccharolytic fermentation—produces straight chain fatty acids (SCFA). Fermentation of protein—called proteolytic fermentation—produces branched chain fatty acids (BCFA).

StoolOMX measures 9 short chain fatty acid metabolites in percentages, total concentrations, and ratios. The test also includes the ratio of straight chain fatty acids to branched chain fatty acids. Ratios can provide insight into dietary composition and digestive impacts such as saccharolytic and proteolytic (putrefactive) fermentation and digestive insufficiency. Assessing SCFA levels in stool offers a deeper look into digestive health and the overall balance of the gut microbiome.

Major SCFAs

Acetate, butyrate, propionate, and valerate are the major primary straight chain metabolites. As preferred fuel for intestinal cells, these metabolites provide a variety of beneficial effects for intestinal health.

SCFAs serve as an energy source for colon cells, strengthen the gut barrier and provide metabolic and immune system signaling. They have a profound anti-inflammatory effect by inducing and selectively expanding T-regulatory cells (T regs) in the large intestine, which, in turn, suppresses the pro-inflammatory action of Th17. As metabolites of bacterial fermentation, their levels can reflect gut microbiota composition.

Low levels of SCFAs are linked to various conditions, including irritable bowel syndrome, obesity, and inflammatory bowel disease. Low levels may also be related to slow motility.

Table 11.

PERCENT BREAKDOWN OF MAJOR SHORT CHAIN FATTY ACIDS REPRESENTED ON STOOLOMX		
MAJOR SCFA	FUNCTION	AVERAGE PERCENTAGE†
Acetate	Most abundant SCFA. Involved in lipid synthesis and appetite regulation, maintains energy balance and metabolic homeostasis. Resists oxidation and mitochondrial stress.	**52%**
Butyrate	Primary energy source for colonic cells. Supports intestinal barrier integrity, reduces intestinal inflammation, promotes motility, enhances fatty acid oxidation, inhibits tumor cell progression, and fosters a balanced microbiome.	**20%**
Propionate	Supports intestinal barrier integrity, impacts energy balance, gluconeogenesis, and lipid metabolism. Involved in appetite regulation. Proposed as a biomarker for IBS.	**25%**
Valerate	Stimulates intestinal epithelium growth, inhibits colon cancer cell production, modulates glucose and lipid metabolism. Antimicrobial effects against *C. difficile*.	**3%**

† Reference set at 50th percentile.

SCFA/BCFA Ratio

Saccharolytic straight chain fatty acids (SCFAs) are primary metabolites of dietary fiber fermentation in the gut while proteolytic branched chain fatty acids (BCFAs) are metabolites of protein fermentation.

Ideally, saccharolytic SCFAs should make up ~95% of total SCFAs, while BCFAs should make up ~5% of total SCFAs.

The production of branched chain fatty acids leads to other fermentation products that can be harmful to the colon epithelium, such as ammonia, phenol, p-cresol, or biogenic amines. BCFAs are mainly produced during fermentation of branched chain amino acids (valine, leucine, and isoleucine) by the intestinal microbiota.

High levels of BCFAs compared to saccharolytic SCFAs can indicate weak digestion, poor transit, and inflammatory dysbiosis. There may also be associations between elevated BCFA concentrations in stool and obesity, IBD, hypercholesterolemia, and metabolic-associated fatty liver disease (MAFLD).

The SCFA/BCFA ratio may decline with age, primarily due to a significant decrease in SCFA levels in stool.

Short Chain Fatty Acids: Therapeutic Applications

- **Causes for Low SCFA Levels:**
  - » Diarrhea (rapid transit leading to decreased SCFA production)
  - » Constipation (slow transit leading to increased SCFA absorption)
  - » Inflammation (high calprotectin)
  - » Chronic antibiotic use
  - » Low complex carbohydrate and fiber intake
  - » Insufficiency dysbiosis
  - » Inflammatory bowel disease
- **Cause for High SCFA Levels:**
  - » Increased transit time (diarrhea)
- **Causes for High BCFA Levels:**
  - » High protein intake and low fiber and/or polyphenol intake (Western diet)
  - » Low total SCFAs
  - » Weak digestion/hypochlorhydria
  - » Increased age
  - » Metabolic imbalance
- **Therapeutic Options and Considerations to Increase Levels of SCFAs:**
  - » Ensure diet is high and diverse in plant-based fibers, polyphenols, and fermented foods
  - » Avoid antibiotics
  - » Consider probiotics to support butyrate-producing organisms
  - » Consider support with butyrate salts
  - » Intestinal barrier support
  - » Support digestion if indicated
  - » Maintain a healthy weight

Table 12. The bacteria involved in the production of saccharolytic straight chain fatty acids and proteolytic branched chain fatty acids. Acetate, propionate, and butyrate make up ~90–95% of fecal SCFAs, with valerate and caproate comprising a smaller percentage. Isobutyrate, isovalerate, 2-methylbutyrate, and isocaproate make up ~5–10% of fecal SCFAs. Microbiota listed below each metabolite are involved in the production of that respective fatty acid.

BACTERIA INVOLVED IN SHORT CHAIN FATTY ACID PRODUCTION			
	SACCHAROLYTIC STRAIGHT CHAIN FATTY ACIDS (SCFA)	PROTEOLYTIC BRANCHED CHAIN FATTY ACIDS (BCFA)	
~90–95 Percent of Fecal SCFAs	**Acetate** • *Blautia hydrogenotrophica* • *Bifidobacterium* spp. • *Lactobacillus* spp. • *Clostridium* spp. • *Streptococcus* spp.	**Isobutyrate** • *Bacteroides* spp. • *Clostridium* spp.	~5–10 Percent of Fecal SCFAs
	Propionate • *Bacteroidetes* • *Negativicutes* • *Megasphaera elsdenii* • *Lachnospiraceae* • *Coprococcus catus* • *Akermansia municiphilia*	**Isovalerate** • *Bacteroides* spp. • *Clostridium* spp.	
	Butyrate • *Firmicutes* phyla • *Faecalibacterium prausnitzii* • *Roseburia* spp. • *Eubacterium* spp. • *Clostridium coccoides*	**2-Methylbutyrate** (from leucine) • *Bacteroides* spp. • *Clostridium* spp.	
	Valerate (potentially toxic) • *Clostridia*	**Isocaproate** (associated with disease)	
	Caproate (potentially toxic – from lactate) • *Megasphaera elsdenii* • *Clostridium* spp. BS-1		

- **Therapeutic Options and Considerations to Decrease Levels of SCFAs:**
  - » Low-FODMAP diet
  - » Maintain a healthy weight
  - » Support digestion if indicated
  - » Consider multi-strain probiotics to decrease acetate concentrations

- **Therapeutic Options and Considerations to Decrease Levels of BCFAs:**
  - » Increase fiber intake (particularly insoluble fibers) and/or decrease protein intake
  - » Consider underlying associations such as inflammatory bowel disease, chronic kidney disease, obesity, type 2 diabetes mellitus, MAFLD, or impaired digestion

INDIVIDUAL BILE ACID RESULTS

Research is more widely available on the clinical implications of certain primary and secondary bile acids compared to others.

Table 13.

PRIMARY BILE ACIDS		
MARKER	ABBR	GUIDE
Cholic Acid	**CA**	Major primary bile acid. May be increased in IBS-D patients. CA levels in stool may serve as a biomarker for IBS and UC. During a UC flare, deconjugation of CA to DCA may be impaired. Can inhibit *Roseburia* spp. and *Lactobacillus* spp.
Chenodeoxycholic Acid	**CDCA**	Major primary bile acid. May be increased in IBS-D patients and may contribute to visceral hypersensitivity. Elevated levels may be associated with metabolic imbalance.
Taurochenodeoxycholic Acid	TCDCA	Minor primary bile acid.
Taurocholic Acid	TCA	A whole grain diet may increase levels of TCA.
Glycochenodeoxycholic Acid	GCDCA	May be increased in IBS-D patients.
Glycocholic Acid	GCA	A whole grain diet may increase levels of GCA.
Hyocholic Acid	HCA	Lower levels are associated with pre-diabetes. HCA levels may serve as an indication of metabolic health.

Table 14.

SECONDARY BILE ACIDS		
MARKER	ABBR	GUIDE
Lithocholic Acid*	**LCA**	LCA is a major secondary bile acid that is formed from the bacterial metabolism of CDCA. Low levels observed in UC. Elevated levels are associated with liver and colon cancer and may contribute to blood sugar dysregulation. Modulates immune responses at normal levels.
Deoxycholic Acid	**DCA**	DCA is a major secondary bile acid that is formed from the bacterial metabolism of CA. Low levels observed in ulcerative colitis (UC). Elevated levels are associated with liver and colon cancer and metabolic imbalance. Implicated in modulating the immune response by inhibiting pro-inflammatory cytokine production. Can inhibit *Lactobacillus* spp., *Bifidobacterium* spp., *Bacteroides fragilis,* and *Clostridium difficile*.
Isolithocholic Acid	**Iso-LCA**	Prevalent secondary bile acid in stool that has positive associations with longevity. Influences metabolic health, interacts with cellular receptors, and modulates immune responses at normal levels. Influences the differentiation and function of T cells, particularly T helper 17 (Th17) cells, which play a critical role in the inflammatory response.

Table 14. *Continued.*

SECONDARY BILE ACIDS		
MARKER	ABBR	GUIDE
12-Ketolithocholic Acid	12-KLCA	Prevalent secondary bile acid in stool. May provide anti-inflammatory effects in IBD, modulate cholesterol metabolism, and improve glucose homeostasis.
3-oxoDeoxycholic Acid	3-oxoDCA	Prevalent secondary bile acid in stool that has positive associations with longevity.
Ursodeoxycholic Acid	UDCA	Enhances bile flow, hepatoprotective, and has immunomodulatory properties. Levels in stool may serve as a biomarker of IBS and UC. Elevated levels may be associated with metabolic imbalance.
Glycolithocholic Acid	GLCA	May be negatively correlated with fecal calprotectin levels in UC. Influences metabolic signaling pathways, modulates gut microbiome, involved in lipid absorption, and aids in the detoxification of potentially harmful bile acids.
Glycoursodeoxycholic Acid	GUDCA	Modulates the gut microbiome, cytoprotective, and influences lipid and glucose metabolism. May be elevated in IBS-D.
Glycodeoxycholic Acid	GDCA	Modulates the gut microbiome. Involved in lipid absorption and metabolic regulation. May be reduced in UC.
Taurolithocholic Acid	TLCA	Involved in lipid absorption and metabolic regulation. Modulates inflammation and may exert cholestatic effects when dysregulated. A whole grain diet may increase levels of TLCA.
Tauroursodeoxycholic Acid	TUDCA	Often used synthetically as a supplement. Used clinically for cholestatic liver diseases. Cytoprotective, particularly in the liver. Involved in the regulation of bile acid metabolism and lipid metabolism. Neuroprotective and modulates inflammation.
Taurodeoxycholic Acid	TDCA	Involved in bile acid metabolism and lipid metabolism. Modulates the gut microbiome and has anti-inflammatory properties.
7-Ketolithocholic Acid	7-KLCA	Involved in bile acid metabolism and cholesterol metabolism. Modulates the gut microbiome.
Dehydrolithocholic Acid	DHLCA	Involved in bile acid metabolism and modulation of the gut microbiome. Exerts anti-inflammatory properties.
Hyodeoxycholic Acid	HDCA	May be negatively correlated with fecal calprotectin levels in UC. May have anti-atherosclerotic effects. Involved in lipid absorption and cholesterol metabolism. Modulates the gut microbiome.
Alloisolithocholic Acid	AlloIso-LCA	Positive associations with longevity and exerts antibacterial effects against gram-positive pathogens.
3-Dehydrocholic Acid	3-DHCA	Involved in bile acid metabolism and exerts anti-inflammatory properties.

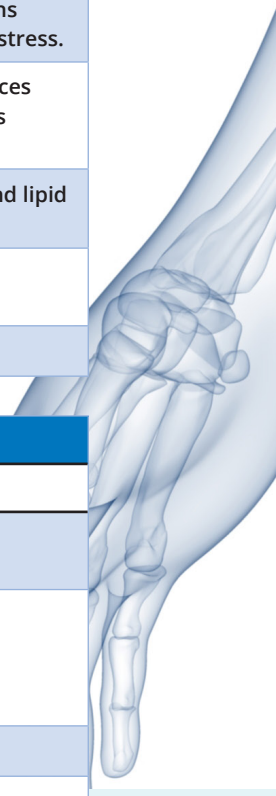
** On StoolOMX, the LCA value is a summation of LCA + Allo-LCA.*

INDIVIDUAL SHORT CHAIN FATTY ACIDS RESULTS

Table 15.

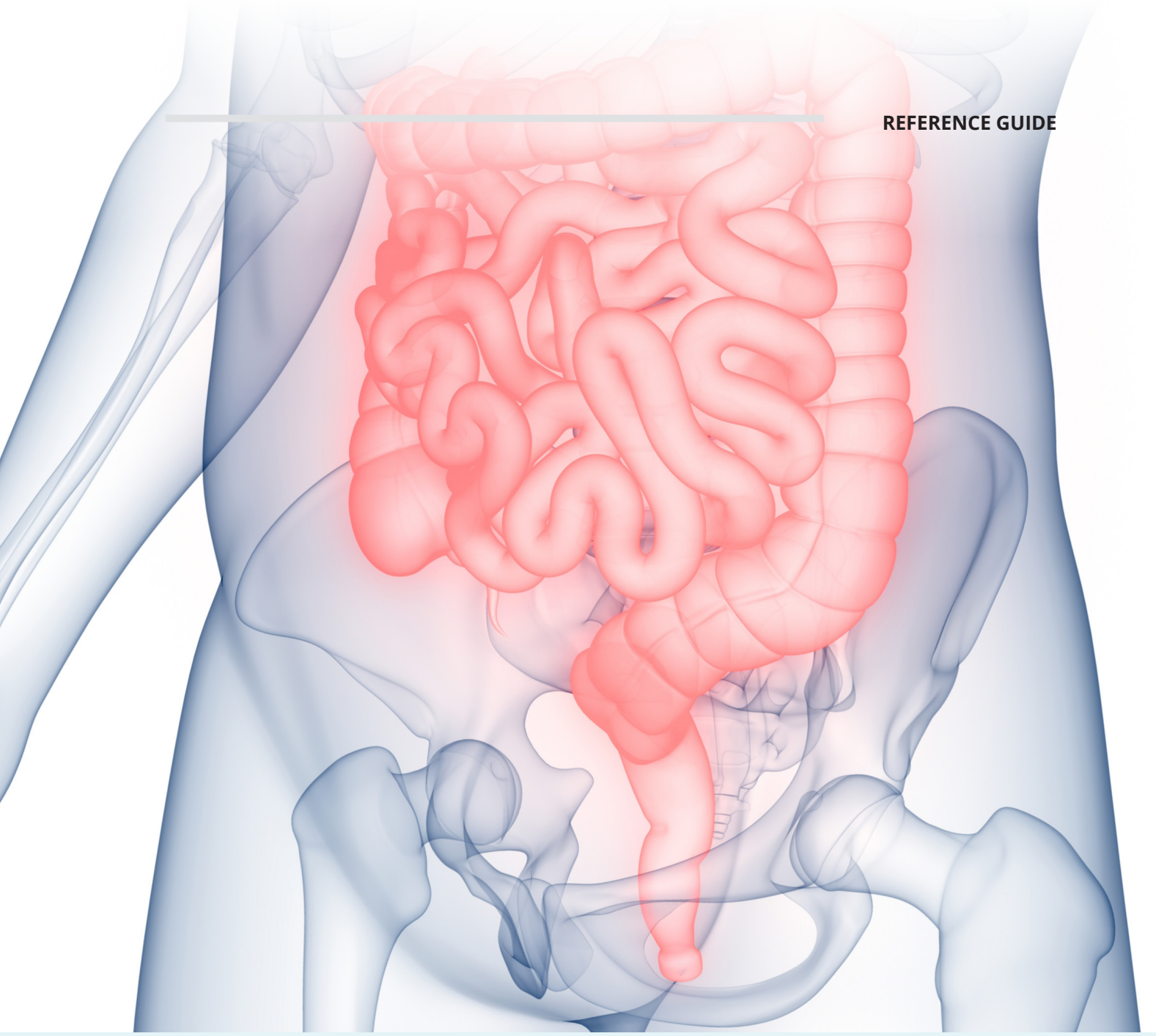
SACCHAROLYTIC STRAIGHT CHAIN FATTY ACIDS (SCFA)	
MARKER	**GUIDE**
Acetate	Most abundant SCFA. Involved in lipid synthesis and appetite regulation, maintains energy balance and metabolic homeostasis. Resists oxidation and mitochondrial stress.
Butyrate	Primary energy source for colonic cells. Supports intestinal barrier integrity, reduces intestinal inflammation, promotes motility, enhances fatty acid oxidation, inhibits tumor cell progression, and fosters a balanced microbiome.
Propionate	Supports intestinal barrier integrity, impacts energy balance, gluconeogenesis, and lipid metabolism. Involved in appetite regulation. Proposed as a biomarker for IBS.
Valerate	Stimulates intestinal epithelium growth, inhibits colon cancer cell production, modulates glucose and lipid metabolism. Antimicrobial effects against *C. difficile*.
Caproate	Antimicrobial effects against *C. difficile*.

PROTEOLYTIC BRANCHED CHAIN FATTY ACIDS (BCFA)	
MARKER	**GUIDE**
Isobutyrate	Stimulates colonic sodium absorption. Elevated levels in stool may be associated with cortisol levels and depression.
Isovalerate	Elevated levels in stool may be associated with depression by its influence on the gut flora, metabolic pathways, and inflammatory pathways. It can also interfere with neurotransmitter release. There is an association between elevated levels of isovalerate and increased cortisol levels.
2-Methylbutyrate	Branched chain fatty acid.
Isocaproate	Produced primarily through the fermentation of branched chain amino acids (BCAAs), particularly leucine. Energy source for many tissues, especially during fasting or low carbohydrate intake.



SCFAs are known to have numerous health effects and can enhance fecal excretion of bile acids.

Low levels of SCFAs are linked to various conditions, including irritable bowel syndrome, obesity, and inflammatory bowel disease.



StoolOMX™

Advanced Bile Acid Testing and Short-Chain Fatty Acid Evaluation in One GI-MAP Add-on Panel

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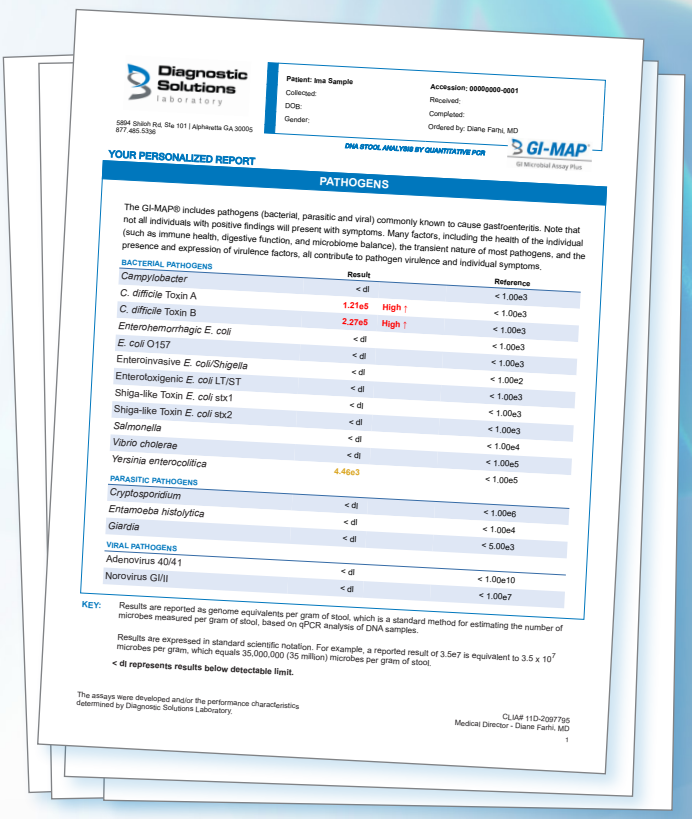

Diagnostic Solutions laboratory

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877.485.5336

Patient: Ima Sample
Collected:
DOB:
Gender:

Accession: 00000000-0001
Received:
Completed:
Ordered by: Diane Farhi, MD

DNA STOOL ANALYSIS BY QUANTITATIVE PCR — GI-MAP GI Microbial Assay Plus

YOUR PERSONALIZED REPORT

PATHOGENS

The GI-MAP® includes pathogens (bacterial, parasitic and viral) commonly known to cause gastroenteritis. Note that not all individuals with positive findings will present with symptoms. Many factors, including the health of the individual (such as immune health, digestive function, and microbiome balance), the transient nature of most pathogens, and the presence and expression of virulence factors, all contribute to pathogen virulence and individual symptoms.

BACTERIAL PATHOGENS	Result		Reference
Campylobacter	< dl		< 1.00e3
C. difficile Toxin A	1.21e5	High ↑	< 1.00e3
C. difficile Toxin B	2.27e5	High ↑	< 1.00e3
Enterohemorrhagic E. coli	< dl		< 1.00e3
E. coli O157	< dl		< 1.00e3
Enteroinvasive *E. coli*/*Shigella*	< dl		< 1.00e2
Enterotoxigenic *E. coli* LT/ST	< dl		< 1.00e3
Shiga-like Toxin *E. coli* stx1	< dl		< 1.00e3
Shiga-like Toxin *E. coli* stx2	< dl		< 1.00e3
Salmonella	< dl		< 1.00e4
Vibrio cholerae	< dl		< 1.00e5
Yersinia enterocolitica	4.46e3		< 1.00e5
PARASITIC PATHOGENS			
Cryptosporidium	< dl		< 1.00e6
Entamoeba histolytica	< dl		< 1.00e4
Giardia	< dl		< 5.00e3
VIRAL PATHOGENS			
Adenovirus 40/41	< dl		< 1.00e10
Norovirus GI/II	< dl		< 1.00e7

KEY: Results are reported as genome equivalents per gram of stool, which is a standard method for estimating the number of microbes measured per gram of stool, based on qPCR analysis of DNA samples.

Results are expressed in standard scientific notation. For example, a reported result of 3.5e7 is equivalent to 3.5×10^7 microbes per gram, which equals 35,000,000 (35 million) microbes per gram of stool.

< dl represents results below detectable limit.

The assays were developed and/or the performance characteristics determined by Diagnostic Solutions Laboratory.

CLIA# 11D-2097795
Medical Director - Diane Farhi, MD

1

REFERENCE GUIDE

GI-MAP® Add-On Test

Our mission: to deliver innovative, accurate and clinically relevant diagnostic testing in a timely and cost-effective manner

StoolOMX™

Advanced Bile Acid Testing and Short Chain Fatty Acid Evaluation