 designs for health Australia

Connecting the Dots with the New Microbial Metabolite Add-On Test for GI-MAP[®]

Presented by Thomas Fabian, PhD, CNTP



Presenter | Thomas Fabian, PhD, CNTP



Functional Genomics and Microbiome Specialist

Expert on the role of the microbiome in health, immune function, chronic disease, and aging.

Dr Fabian is a translational scientist with a primary focus on the clinical application of microbiome research in the integrative and functional medicine space. He received his PhD in molecular biology from the University of Colorado Boulder and has worked as a biomedical researcher in the biotechnology industry, as well as more recently as a consultant in the microbiome testing field.

Currently, Dr Fabian serves as a consultant and science advisor at Diagnostic Solutions Laboratory, and he is also a member of the Science Advisory Board at Designs for Health. In addition, he is certified as a Nutrition Therapy Practitioner by the Nutrition Therapy Institute in Denver.

Host | Lea McIntyre



Naturopath

Lea McIntyre is head of Marketing at Designs for Health Australia.

She has 20 years experience as a qualified naturopath, herbalist and nutritionist. In her clinical practice, she has a special interest in paediatric health and gut health and the relationship between inflammation and neurological conditions.

Lea has developed a strong relationship with the Designs for Health practitioner community. She will moderate the Q&A discussion with Dr Tan in this webinar and engage our live Designs for Health practitioner community to bring insight and practical clinical pearls for all.

3-Page Report with Summary

StoolOMX™

Stool metabolomics add-on panel with GI-MAP

➤ 25 bile acids + 9 SCFAs

More complete picture of gut & microbiome health + wide range of clinical insights





Important Health Insights with Bile Acids & SCFAs

- Overall microbiome health
- Gut motility regulation
- Intestinal barrier health
- Immune responses
- Metabolic health
- Digestion & Diet



SHORT CHAIN FATTY ACIDS - SUMMARY

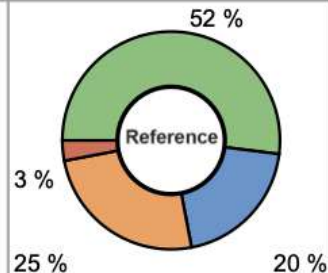
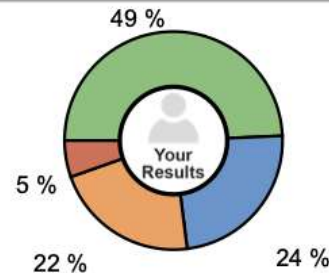
The **Postbiotic Fatty Acid Metabolite Panel** assesses fecal concentrations of straight chain and branched chain fatty acids. These metabolites provide a variety of beneficial effects for intestinal health, anti-inflammation, metabolism and immunity, and give dietary insight.

SUMMARY INFO

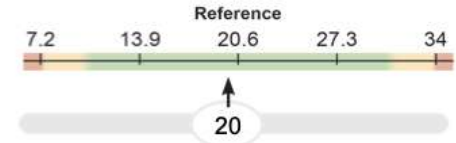
Major Straight Chain Fatty Acids - µg/g	2.26e4 H		3.63e3 - 1.95e4
Acetate - %	49.3		38.3 - 68.0
Butyrate - %	23.7		7.7 - 32.6
Propionate - %	21.6		14.1 - 33.6
Valerate - %	5.4		0.5 - 6.2

Major SCFA Percent

- Acetate
- Butyrate
- Propionate
- Valerate



Reference set at 50th percentile.



SCFA/BCFA Ratio

Ratio of total straight chain fatty acids (SCFA) to total branched chain fatty acids (BCFA).



BILE ACIDS - SUMMARY

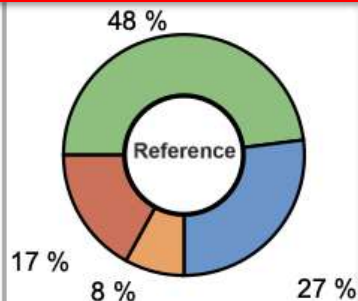
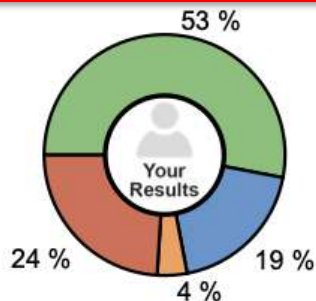
The **Bile Acids Panel** assesses fecal concentrations of primary and secondary bile acids and provides insights into microbiome diversity, digestive function, motility, and various gut-related conditions.

SUMMARY INFO

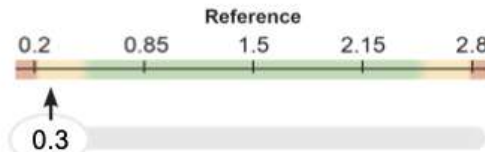
Total Bile Acids - ng/g	6.43e6 H		2.37e5 - 6.29e6
Secondary Bile Acids- %	84.6 L		> 90.5
Primary Bile Acids - %	15.4 H		< 7.8

Bile Acid Percentages

- Deoxycholic Acid-DCA
- Lithocholic Acid-LCA*
- Iso-LCA
- Other



Reference set at 50th percentile.



LCA*/DCA Ratio

Lithocholic Acid / Deoxycholic Acid Ratio based on absolute values.

*LCA value is the summation of LCA + Allo-LCA



BILE ACIDS - RESULTS

PRIMARY BILE ACIDS

	Abbreviation	Conjugation**	Result ng/g	Reference ng/g
Total Primary Bile Acids			9.92e5 H	3.50e3 - 7.90e4
Cholic Acid	CA	U	6.18e5 H	< 5.92e4
Chenodeoxycholic Acid	CDCA	U	3.67e5 H	2.16e3 - 6.87e4
Taurochenodeoxycholic Acid	TCDCA	C	5.09e2 H	< 4.14e2
Taurocholic Acid	TCA	C	5.53e2 H	< 5.19e2
Glycochenodeoxycholic Acid	GCDCA	C	1.09e3 H	1.18e1 - 8.11e2
Glycocholic Acid	GCA	C	8.90e2 H	< 7.55e2
Hyocholic Acid	HCA	U	4.71e3	< 5.50e3

SECONDARY BILE ACIDS

	Abbreviation	Conjugation**	Result ng/g	Reference ng/g
Total Secondary Bile Acids			5.44e6	1.97e5 - 6.23e6
Deoxycholic Acid	DCA	U	3.44e6 H	2.24e3 - 2.33e6
Lithocholic Acid*	LCA	U	1.20e6	6.12e3 - 1.37e6
Isolithocholic Acid	ISO-LCA	U	2.78e5	2.21e3 - 5.36e5
12-Ketolithocholic Acid	12-KLCA	U	4.42e5	1.87e3 - 5.30e5
3-oxoDeoxycholic Acid	3-oxoDCA	U	4.05e4	3.53e2 - 1.12e5
Ursodeoxycholic Acid	UDCA	U	<dl	< 5.77e4
7-Ketolithocholic Acid	7-KLCA	U	1.23e4 H	< 8.94e3
7-Ketodeoxycholic Acid	7-KDCA	U	1.89e4 H	< 1.01e4
Dehydrolithocholic Acid	DHLCA	U	<dl	< 4.52e4
Hyodeoxycholic Acid	HDCA	U	<dl	< 5.27e4
Alloisolithocholic Acid	AlloIso-LCA	U	3.51e3	< 7.53e4
3-Dehydrocholic Acid	3-DHCA	U	5.80e3 H	< 5.85e2
Glycolithocholic Acid	GLCA	C	<dl	< 2.20e2
Glycoursodeoxycholic Acid	GUDCA	C	1.71e2	< 3.08e2
Glycodeoxycholic Acid	GDCA	C	4.14e2	< 5.40e2
Taurolithocholic Acid	TLCA	C	5.87e1	< 2.68e2
Tauroursodeoxycholic Acid	TUDCA	C	<dl	< 1.28e2
Taurodeoxycholic Acid	TDCA	C	2.96e2	< 8.56e2

SHORT CHAIN FATTY ACIDS - RESULTS

Total Short Chain Fatty Acids - $\mu\text{g/g}$

2.40e4 H



4.23e3 - 2.10e4

SACCHAROLYTIC STRAIGHT CHAIN FATTY ACIDS (SCFA)

	Result $\mu\text{g/g}$	Reference $\mu\text{g/g}$
Total SCFA	2.28e4 H	3.65e3 - 1.95e4
Acetate	1.11e4 H	2.09e3 - 9.72e3
Butyrate	5.35e3	3.94e2 - 5.79e3
Propionate	4.89e3	5.91e2 - 5.45e3
Valerate	1.23e3 H	4.33e1 - 7.73e2
Caproate	2.21e2 H	7.15e-1 - 1.44e2

PROTEOLYTIC BRANCHED CHAIN FATTY ACIDS (BCFA)

	Result $\mu\text{g/g}$	Reference $\mu\text{g/g}$
Total BCFA	1.14e3	1.65e2 - 1.67e3
Iso-butyrate	4.04e2	5.65e1 - 5.64e2
Iso-valerate	4.16e2	4.45e1 - 6.58e2
2-Methylbutyrate	3.22e2	3.82e1 - 4.61e2
Iso-caproate	4.78e-1	< 9.93e0

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2025
02/17/2025

REFERENCE GUIDE
GI-MAP™ Add-On Test
An array of 25 bile acids, 10 short-chain fatty acids, and 10 inflammatory markers

StoolOMX™
Advanced Bile Acid Testing and
Short-Chain Fatty Acid Evaluation

STOOLOMX REFERENCE GUIDE

Patient Test Sample: Accession: 20240229-0002

BILE ACIDS - SUMMARY

The Bile Acid Panel assesses fecal concentrations of primary and secondary bile acids and provides insight into bile metabolism, digestive function, excretion, and various gastrointestinal conditions.

SUMMARY

Total Bile Acids - µg/g	1,760	3,070 - 12,960
Secondary Bile Acids %	58.7	> 65.5
Primary Bile Acids %	41.3	< 34.5

Bile Acid Percentages

- I: Deoxycholic-Acids
- II: Lithocholic-Acids
- III: Cholic

SHORT CHAIN FATTY ACIDS - SUMMARY

The Pediatric Fatty Acid Metabolite Panel assesses fecal concentrations of straight chain and branched chain fatty acids. These metabolites provide a variety of beneficial effects for metabolic health, gut-inflammation, mucosal and immunity, and gut-biome insight.

SUMMARY

Major Straight Chain Fatty Acids - µg/g	1,556 (H)	3,020 - 13,960
Acetate %	58.0	55.3 - 65.5
Propionate %	18.0	17.3 - 24.8
Butyrate %	14.1	16.1 - 23.8
Valerate %	0.0	0.5 - 4.2

Major SCFA Percentages

- I: Acetate
- II: Butyrate
- III: Propionate
- IV: Valerate

The StoolOMX report delivers results in an intuitive, easy-to-read format, highlighting imbalances, clinical implications, and areas requiring attention. This actionable data supports tailored intervention strategies, leading to improved outcomes for patients with digestive concerns.

StoolOMX™
Empowering Practitioners to Solve Gut Dysfunction

Diagnostic Solutions
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STOOLOMX SAMPLE REPORT - SEE PAGE 6

GI-MAP Add-On

StoolOMX™

Pinpoint the Causes of IBS, IBD, and Gut Dysfunction with StoolOMX

StoolOMX™ is a powerful GI-MAP add-on test that evaluates 25 bile acids (total concentrations, percentages, and ratios) and 9 short-chain fatty acids (SCFAs), offering clinicians valuable insights into gut functionality.

Bile acids and SCFAs play critical roles in digestion, mobility, microbiome balance, and metabolic health. Imbalances in these metabolites can drive conditions such as IBS, IBD, SIBO, and acid diarrhea, and alter gut motility. By ordering StoolOMX alongside the GI-MAP, practitioners can uncover the underlying causes of these common conditions.

The StoolOMX report delivers results in an intuitive, easy-to-read format, highlighting imbalances, clinical implications, and areas requiring attention. This actionable data supports tailored intervention strategies, leading to improved outcomes for patients with digestive concerns.

Provides Insight Into The Following Areas of Gut Health

- Bile Acid Metabolism**
 - Assess for digestion, microbiome health, and motility
- SCFA Production**
 - Evaluate dietary fermentation, gut inflammation, and gut lining integrity
- GI Dysfunction**
 - Identify microbial imbalances driving symptoms like bloating, diarrhea, or constipation
- Inflammatory Pathways**
 - Explore markers of gut inflammation and barrier integrity

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PRODUCT BRIEF

Diagnostic Solutions Lab – YouTube (Webinars)



StoolOMX™



StoolOMX™

Overview of the StoolOMX™
GI-MAP® Add-on



Natalie Groenewoud, ND



Amber Sereda, ND

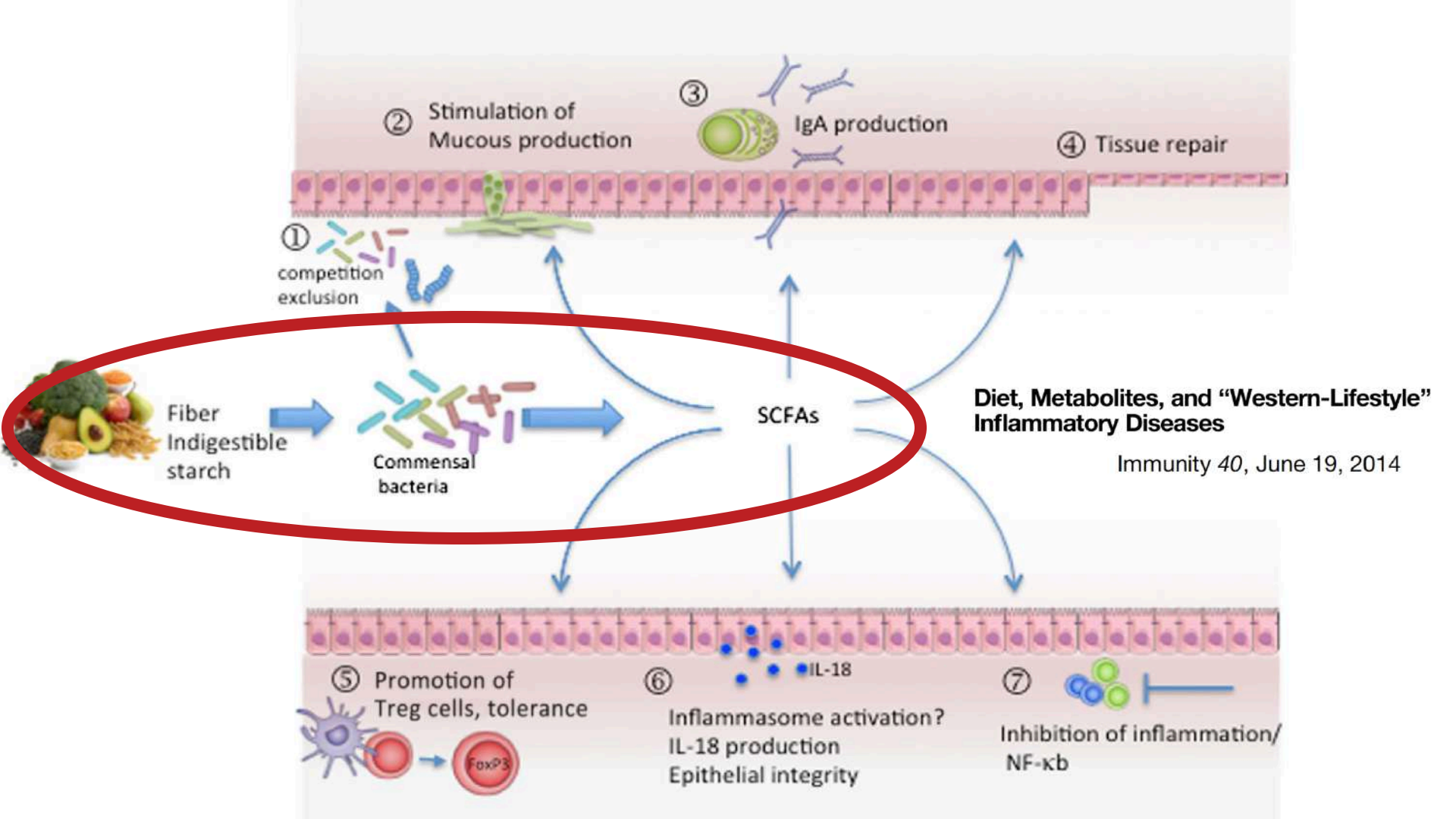
Short-Chain Fatty Acids Overview

- Wide range of established beneficial health effects
- Produced largely by commensal bacteria via fermentation of dietary carbohydrates (some from mucus breakdown, and other sources)
- Normally, at least 95% of SCFAs that are produced are absorbed by the mucosa, and ~5% are present in stool
- SCFAs levels in stool are influenced by both production **and absorption**
 - Production influenced by diet, microbiome composition, and gut function (esp. digestion)
 - Absorption influenced by transit time (stool consistency) & mucosal health (e.g., inflammation)

> Am J Physiol Gastrointest Liver Physiol. 2020 Feb 1;318(2):G361-G369.
doi: 10.1152/ajpgi.00283.2019. Epub 2019 Dec 23.

Distal colonic transit is linked to gut microbiota diversity and microbial fermentation in humans with slow colonic transit

“Despite often being used as fermentation markers, fecal SCFA concentrations reflect the net result of absorption, production, and bacterial cross-feeding.”






Supporting Microbiome & SCFA Balance (4P's)

- ❖ Prebiotics / fermentable carbs
- ❖ Polyphenols
- ❖ Probiotics
- ❖ Postbiotics
(butyrate, vitamins, indoles, urolithin A, bile acid metabolites,

COMMENSAL/KEYSTONE BACTERIA

COMMENSAL BACTERIA

	Result	Reference
<i>Bacteroides fragilis</i>	5.84e8 L 	1.6e9 - 2.5e11
<i>Bifidobacterium</i> spp.	1.95e9 	> 6.7e7
<i>Enterococcus</i> spp.	1.36e5 L 	1.9e5 - 2.0e8
<i>Escherichia</i> spp.	1.32e6 L 	3.7e6 - 3.8e9
<i>Lactobacillus</i> spp.	2.85e6 	8.6e5 - 6.2e8
<i>Enterobacter</i> spp.	5.54e6 	1.0e6 - 5.0e7
<i>Akkermansia muciniphila</i>	<dl L 	1.0e1 - 8.2e6
<i>Faecalibacterium prausnitzii</i>	<dl L 	1.0e3 - 5.0e8
<i>Roseburia</i> spp.	4.56e6 L 	5.0e7 - 2.0e10
BACTERIAL PHyla		
<i>Bacteroidetes</i>	2.17e10 L 	8.6e11 - 3.3e12
<i>Firmicutes</i>	1.38e9 L 	5.7e10 - 3.0e11
<i>Firmicutes:Bacteroidetes</i> Ratio	0.06 	< 1.0



SHORT CHAIN FATTY ACIDS - SUMMARY

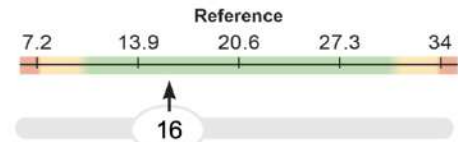
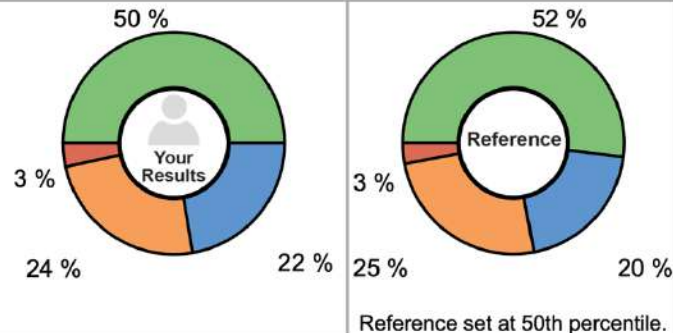
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SUMMARY INFO

Major Straight Chain Fatty Acids - $\mu\text{g/g}$	6.00e3		3.63e3 - 1.95e4
Acetate - %	50.0		38.3 - 68.0
Butyrate - %	22.3		7.7 - 32.6
Propionate - %	24.3		14.1 - 33.6
Valerate - %	3.4		0.5 - 6.2

Major SCFA Percent

- Acetate
- Butyrate
- Propionate
- Valerate



SCFA/BCFA Ratio

Ratio of total straight chain fatty acids (SCFA) to total branched chain fatty acids (BCFA).



Fiber
Indigestible
starch



①
competition
exclusion

Commensal
bacteria

SCFAs

Diet, Metabolites, and "Western-Lifestyle"
Inflammatory Diseases

Immunity 40, June 19, 2014

② Stimulation of
Mucous production

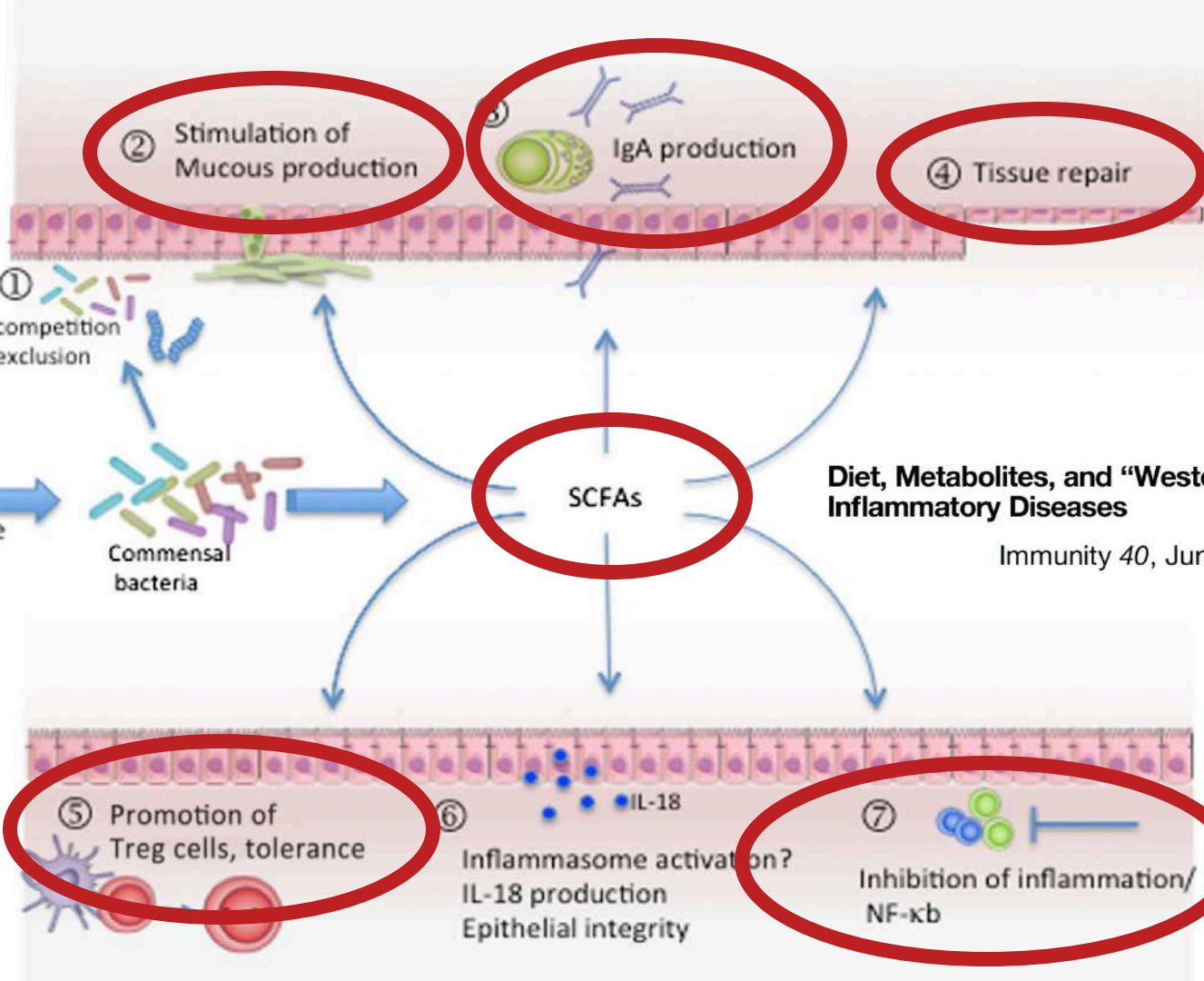
③ IgA production

④ Tissue repair

⑤ Promotion of
Treg cells, tolerance

⑥ Inflammasome activation?
IL-18 production
Epithelial integrity

⑦ Inhibition of inflammation/
NF- κ b



Gut microbiota-derived short-chain fatty acids and their role in human health and disease

Indrani Mukhopadhyay¹ & Petra Louis²✉

Abstract

Short-chain fatty acids (SCFAs) are a group of organic compounds produced by the fermentation of dietary fibre by the human gut microbiota. They play diverse roles in different physiological processes of the host with implications for human health and disease. This Review provides an overview of the complex microbial metabolism underlying SCFA

Sections

Introduction

Microbial short-chain fatty acid metabolism

Molecular host interactions of short-chain fatty acids

“There is overwhelming evidence that short-chain fatty acids (SCFAs) influence many aspects of host health, and much progress has been made in recent years in deciphering the underlying molecular mechanisms of action, which include energy provision, interaction with host receptors and epigenetic gene regulation.”

They play diverse roles in different physiological processes of the host with implications for human health and disease. This Review provides an overview of the complex microbial metabolism underlying SCFA

Microbial short-chain fatty acid metabolism

Molecular host interactions of short-chain fatty acids

“SCFAs have been implicated in many different aspects of host physiology, including gut motility, strengthening of the physical gut barrier, numerous immune interactions and the prevention of diseases including cancer, inflammatory, cardiovascular, metabolic and neurological diseases.”

They play diverse roles in different physiological processes of the host with implications for human health and disease. This Review provides an overview of the complex microbial metabolism underlying SCFA

Microbial short-chain fatty acid metabolism

Molecular host interactions of short-chain fatty acids

“SCFAs have an important role in maintaining gut health through a multitude of different actions. They contribute to colonization resistance against pathogens via several mechanistic pathways.”

They play diverse roles in different physiological processes of the host with implications for human health and disease. This Review provides an overview of the complex microbial metabolism underlying SCFA

Microbial short-chain fatty acid metabolism

Molecular host interactions of short-chain fatty acids

Short-chain fatty acids: linking diet, the microbiome and immunity

Elizabeth R. Mann¹, Ying Ka Lam² & Holm H. Uhlig^{2,3,4}✉

Abstract

The short-chain fatty acids (SCFAs) butyrate, propionate and acetate are microbial metabolites and their availability in the gut and other organs is determined by environmental factors, such as diet and use of antibiotics, that shape the diversity and metabolism of the microbiota. SCFAs regulate epithelial barrier function as well as mucosal and systemic immunity via evolutionary conserved processes that involve G protein-coupled receptor signalling or histone deacetylase activity. Indicatively, the anti-inflammatory role of butyrate is mediated

Sections

Introduction

Factors affecting SCFA levels

SCFA-induced cellular signalling

Functions of SCFAs at mucosal barriers

SCFAs in intestinal immune disorders

“Microbiota-derived metabolites such as short-chain fatty acids (SCFAs) — which include acetic acid (acetate), propionic acid (propionate) and butyric acid (butyrate) — have marked effects on mucosal and systemic immune responses, and low levels of SCFAs in the intestine have been directly associated with susceptibility to inflammatory and allergic diseases.”

SCFAs regulate epithelial barrier function as well as mucosal and systemic immunity via evolutionary conserved processes that involve G protein-coupled receptor signalling or histone deacetylase activity. Indicatively, the anti-inflammatory role of butyrate is mediated

Functions of SCFAs at mucosal barriers

SCFAs in intestinal immune disorders



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Masse KE and Lu VB (2023) Short-chain fatty acids, secondary bile acids and indoles: gut microbial metabolites with effects on enteroendocrine cell function and their potential as therapies for metabolic disease. *Front. Endocrinol.* 14:1169624. doi: 10.3389/fendo.2023.1169624

Short-chain fatty acids, secondary bile acids and indoles: gut microbial metabolites with effects on enteroendocrine cell function and their potential as therapies for metabolic disease

Karly E. Masse and Van B. Lu*

Department of Physiology and Pharmacology, University of Western Ontario, London, ON, Canada

The gastrointestinal tract hosts the largest ecosystem of microorganisms in the body. The metabolism of ingested nutrients by gut bacteria produces novel chemical mediators that can influence chemosensory cells lining the gastrointestinal tract. Specifically, hormone-releasing enteroendocrine cells which express a host of receptors activated by these bacterial metabolites

“Changes in the levels of SCFAs, indoles, and secondary bile acids are associated with metabolic disease, and restoration of levels can attenuate disease progression and severity.”

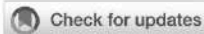
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Role of gut microbiota-derived signals in the regulation of gastrointestinal motility

Zhipeng Zheng, Jingyi Tang, Yingnan Hu and Wei Zhang*

Department of General Surgery, The Second Affiliated Hospital of Zhejiang Chinese Medical University, Hangzhou, China

The gastrointestinal (GI) tract harbors trillions of commensal microbes, called the gut microbiota, which plays a significant role in the regulation of GI physiology, particularly GI motility. The GI tract expresses an array of receptors, such as toll-like receptors (TLRs), G-protein coupled receptors, aryl hydrocarbon receptor (AhR), and ligand-gated ion channels, that sense different gut microbiota-derived bioactive substances. Specifically, microbial cell wall components and metabolites, including lipopeptides, peptidoglycan,



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SPECIALTY SECTION
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Gut bacteria regulate gastrointestinal motility through their cell wall components and metabolic products

Gut microbiota can directly influence the GI motility through bacterial cell wall components [lipopeptides, peptidoglycan, and lipopolysaccharides (LPS)] binding to TLRs expressed in the GI tract. Indirectly, gut microbiota can also modulate the GI motility *via* the release of metabolites or end products of bacterial biotransformation and fermentation. Three main groups of bacterial metabolites, including BAs, SCFAs, and tryptophan metabolites, have been well studied in the regulation of GI motility. In addition, other microbial metabolites belonging to a wide range of chemical groups have also been shown to modulate GI motility, and there are many more gut microbiota-derived

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es, called
ulation of
n array of
receptors,
that sense
microbial

SCFAs, BCFAs, Motility & Protein Fermentation

- SCFAs are produced primarily via carbohydrate fermentation
- BCFAs are produced via protein fermentation
- The balance of SCFAs and BCFAs depends upon diet, digestion, and transit time
- With constipation, SCFAs tend to be decreased and BCFAs tend to be increased
- With loose stools or diarrhea, SCFAs tend to increase and BCFAs are often decreased

Patient with Loose Stools



SHORT CHAIN FATTY ACIDS - SUMMARY

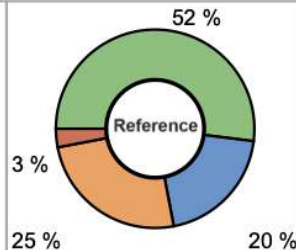
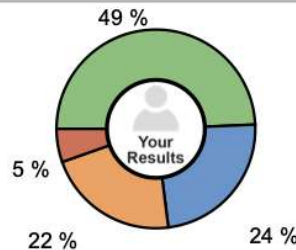
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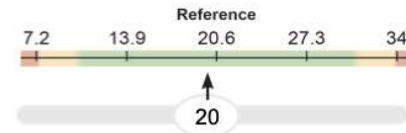
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Butyrate - %	23.7		7.7 - 32.6
Propionate - %	21.6		14.1 - 33.6
Valerate - %	5.4		0.5 - 6.2

Major SCFA Percent

- Acetate
- Butyrate
- Propionate
- Valerate



Reference set at 50th percentile.



SCFA/BCFA Ratio

Ratio of total straight chain fatty acids (SCFA) to total branched chain fatty acids (BCFA).

Patient with Constipation



SHORT CHAIN FATTY ACIDS - SUMMARY

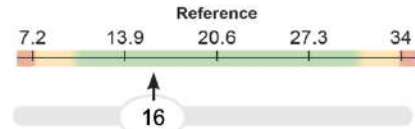
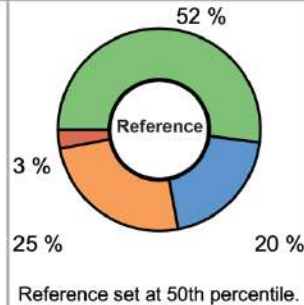
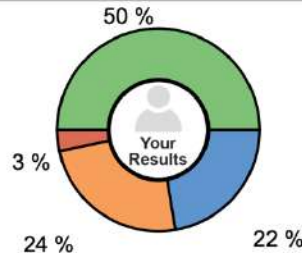
The **Postbiotic Fatty Acid Metabolite Panel** assesses fecal concentrations of straight chain and branched chain fatty acids. These metabolites provide a variety of beneficial effects for intestinal health, anti-inflammation, metabolism and immunity, and give dietary insight.

SUMMARY INFO

Major Straight Chain Fatty Acids - $\mu\text{g/g}$	6.00e3		3.63e3 - 1.95e4
Acetate - %	50.0		38.3 - 68.0
Butyrate - %	22.3		7.7 - 32.6
Propionate - %	24.3		14.1 - 33.6
Valerate - %	3.4		0.5 - 6.2

Major SCFA Percent

- Acetate
- Butyrate
- Propionate
- Valerate



SCFA/BCFA Ratio

Ratio of total straight chain fatty acids (SCFA) to total branched chain fatty acids (BCFA).

“Reduction in SCFA producers is consistent with the switch away from saccharolytic fermentation toward proteolytic fermentation in the case of constipation. Reduced SCFA production is known to weaken smooth muscle contractions that drive peristalsis, acting as a positive feedback on constipation.”

FASTER

SLOWER

Colonic transit time

Microbial metabolism

Complex polysaccharides

Primary bile acids and host-derived glucuronides

Dietary fat

Mucins/dietary proteins

Saccharolysis

↓ pH

SCFA: Acetate, Propionate, Butyrate
Lactate, Succinate, Formate

Secondary bile acids
Hydrolysed glucuronide conjugates

TMA

Proteolysis

↑ pH

BCFA: Valerate, Caproate, Isobutyrate, Isovalerate
Phenols, Indoles, NH₃, H₂S

Methanogenesis

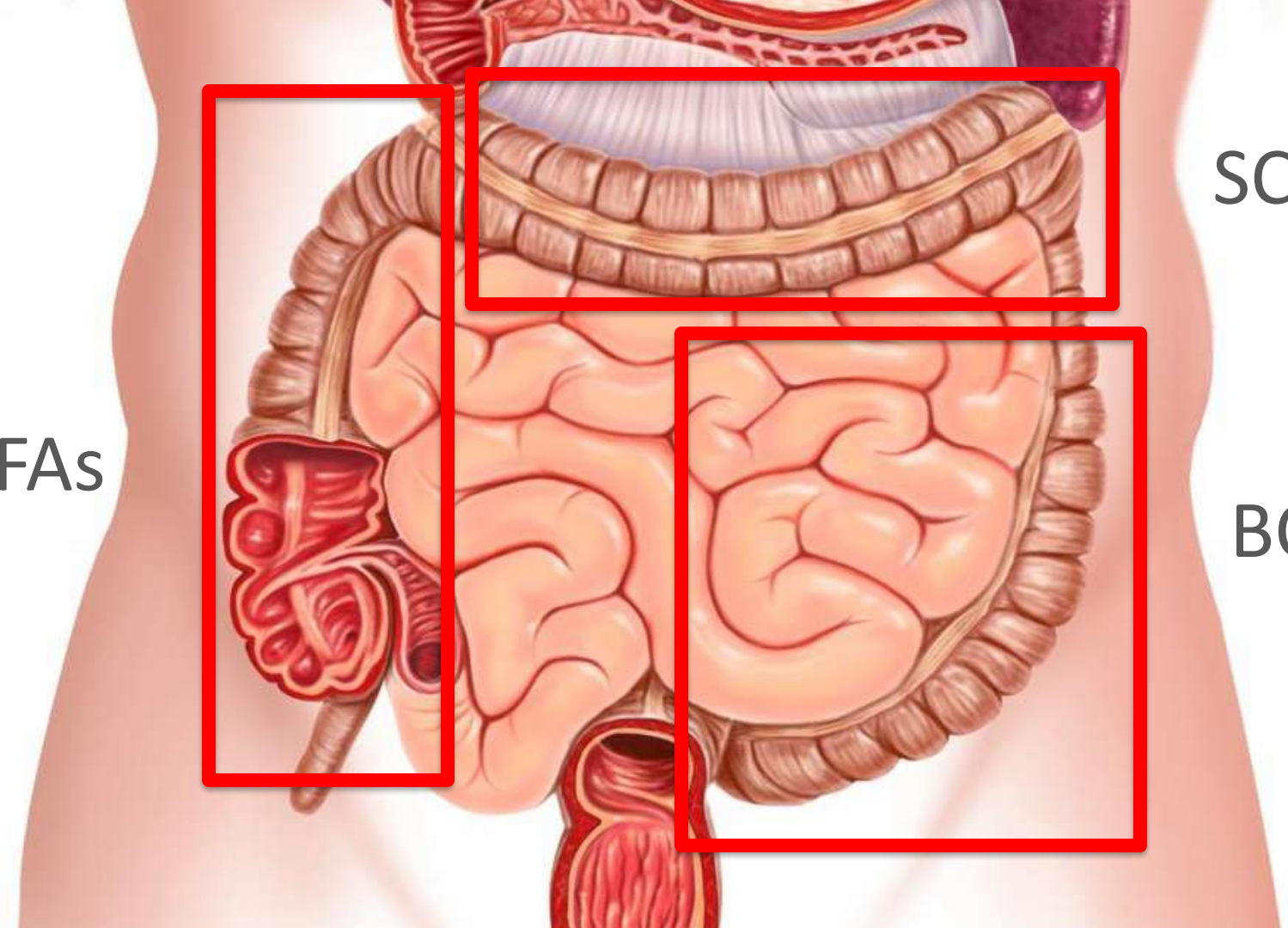
H₂+CO₂

CH₄

SCFAs

SCFAs

BCFAs





SHORT CHAIN FATTY ACIDS - SUMMARY

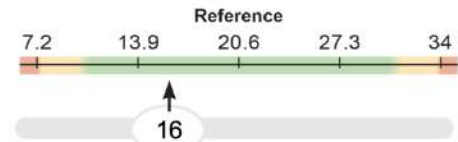
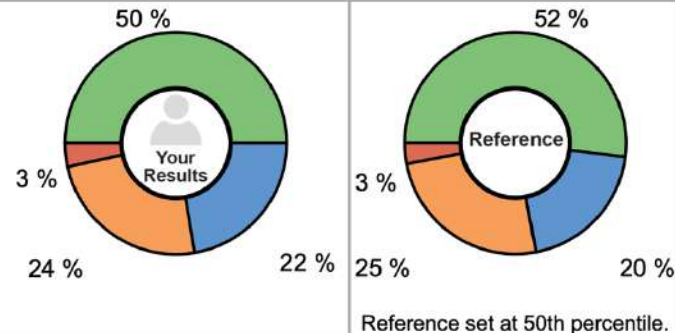
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Major SCFA Percent

- Acetate
- Butyrate
- Propionate
- Valerate



SCFA/BCFA Ratio

Ratio of total straight chain fatty acids (SCFA) to total branched chain fatty acids (BCFA).

SHORT CHAIN FATTY ACIDS - RESULTS

Total Short Chain Fatty Acids - $\mu\text{g/g}$

2.40e4 H



4.23e3 - 2.10e4

SACCHAROLYTIC STRAIGHT CHAIN FATTY ACIDS (SCFA)

	Result $\mu\text{g/g}$	Reference $\mu\text{g/g}$
Total SCFA	2.28e4 H	3.65e3 - 1.95e4
Acetate	1.11e4 H	2.09e3 - 9.72e3
Butyrate	5.35e3	3.94e2 - 5.79e3
Propionate	4.89e3	5.91e2 - 5.45e3
Valerate	1.23e3 H	4.33e1 - 7.73e2
Caproate	2.21e2 H	7.15e-1 - 1.44e2

PROTEOLYTIC BRANCHED CHAIN FATTY ACIDS (BCFA)

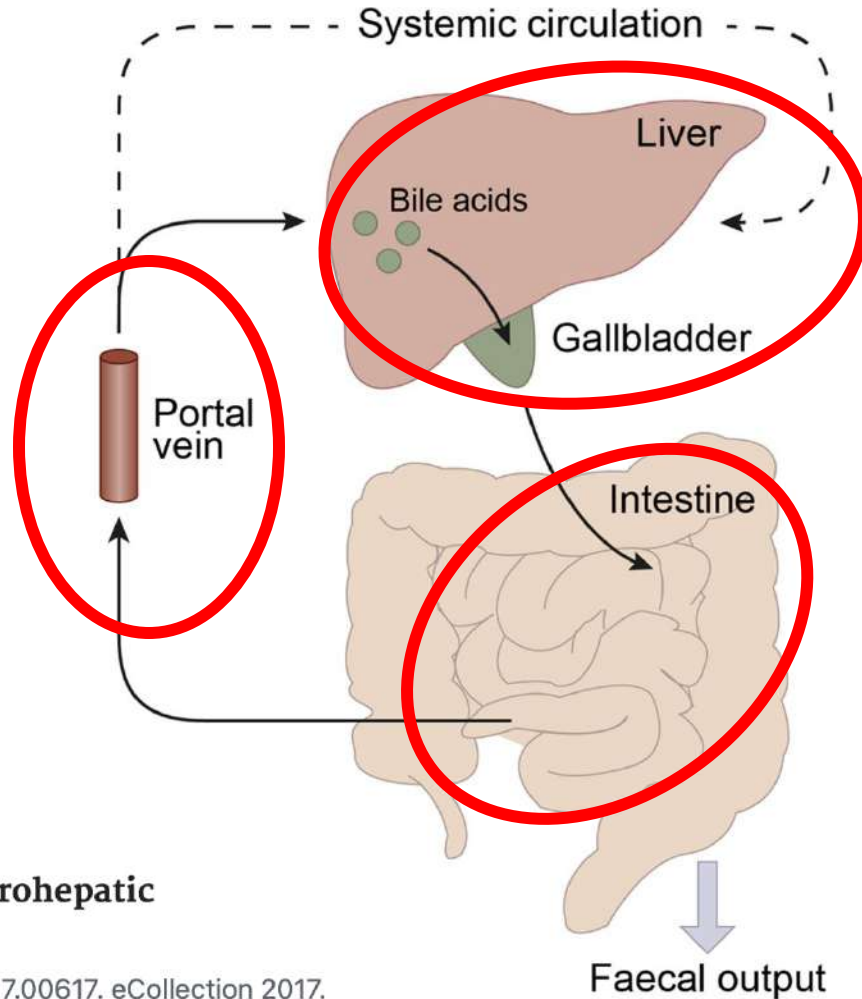
	Result $\mu\text{g/g}$	Reference $\mu\text{g/g}$
Total BCFA	1.14e3	1.65e2 - 1.67e3
Iso-butyrate	4.04e2	5.65e1 - 5.64e2
Iso-valerate	4.16e2	4.45e1 - 6.58e2
2-Methylbutyrate	3.22e2	3.82e1 - 4.61e2
Iso-caproate	4.78e-1	< 9.93e0

Bile Acids Along the GI Tract: Summary

- Produced and conjugated (taurine, glycine) in the liver, and released into the duodenum during meals
- ~95% are reabsorbed in the latter part of the ileum (facilitated by specific bile acid transporters) and returned to the liver via portal circulation
- ~5% enter the colon and are deconjugated (primarily by Bacteroidetes)
- Deconjugated bile acids are converted to secondary bile acids (primarily by certain Firmicutes; typically > 98% conversion to secondary bile acids)

Enterohepatic Circulation


- ~95% of bile acids absorbed in the terminal ileum & returned to the liver
- ~5% pass on to the colon





Bile Acid Signaling Pathways from the Enterohepatic Circulation to the Central Nervous System

INTESTINAL HEALTH MARKERS

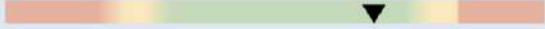


DIGESTION

	Result		Reference
Steatocrit	33 H		< 15 %
Elastase-1	>750		> 200 ug/g

GI MARKERS

β -Glucuronidase	411		< 2486 U/mL
Occult Blood - FIT	<dl		< 10 ug/g

IMMUNE RESPONSE

Secretory IgA	1450		510 - 2010 ug/g
Anti-gliadin IgA	85		< 175 U/L
Eosinophil Activation Protein (EDN, EPX)	1.35		< 2.34 ug/g

INFLAMMATION

Calprotectin	96		< 173 ug/g
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Fat Malabsorption (Steatorrhea): Possible Contributors

- High fat consumption
- Insufficient fat digestion & absorption
 - ***Low bile acid production or impaired release***
 - Pancreatic insufficiency
 - Malabsorption syndromes:
 - Small intestinal disease (e.g., Celiac)
 - Small intestinal infections (e.g., Giardia)
 - Small intestinal dysfunction (impaired mucosal barrier, inflammation, dysbiosis, food reactions, reduced absorptive capacity)



BILE ACIDS - SUMMARY

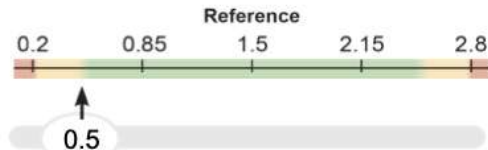
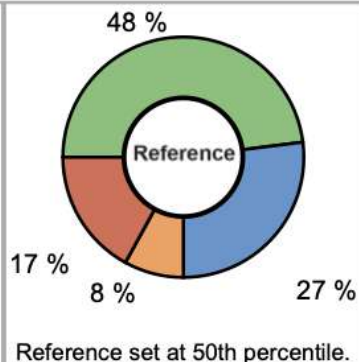
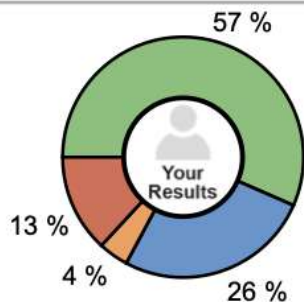
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SUMMARY INFO

Total Bile Acids - ng/g	4.29e5		2.37e5 - 6.29e6
Secondary Bile Acids- %	99.0		> 90.5
Primary Bile Acids - %	1.0		< 7.8

Bile Acid Percentages

- Deoxycholic Acid-DCA
- Lithocholic Acid-LCA*
- Iso-LCA
- Other



LCA*/DCA Ratio

Lithocholic Acid / Deoxycholic Acid Ratio based on absolute values.

*LCA value is the summation of LCA + Allo-LCA

Obesity, Motility, Diet, and Intestinal Microbiota— Connecting the Dots

“Lower levels of bile acids also cause symptoms of constipation, longer transit time, and decreased colonic muscle contraction. Furthermore, when butyrate and bile acids are supplemented, many of the constipation symptoms reverse. Studies suggest the effect of metabolites of gut microbiota on dysmotility may be mediated by changing serotonin levels.”



HHS Public Access

Author manuscript

Curr Opin Gastroenterol. Author manuscript; available in PMC 2024 May 01.

Published in final edited form as:

Curr Opin Gastroenterol. 2023 May 01; 39(3): 184–191. doi:10.1097/MOG.0000000000000916.

Bile Acid Diarrhea- As bad as it gets?

Joelle BouSaba, MD,

Michael Camilleri, MD, DSc

Clinical Enteric Neuroscience Translational and Epidemiological Research (CENTER), Division of Gastroenterology and Hepatology, Mayo Clinic, Rochester, Minnesota, USA

Abstract

Purpose of review: Bile acid diarrhea (BAD) is a common but under-recognized gastrointestinal condition that manifests with increased stool frequency and urgency, and a looser stool consistency. The aim of this review is to present recent advances in the pathophysiology, mechanisms, manifestations, diagnosis, and treatment of BAD.

Recent findings: Patients with BAD have evidence of accelerated colonic transit, increased gut mucosal permeability, altered stool microbiome composition, and decreased quality of life. Single, random stool measurements of bile acids, alone or in combination with fasting serum 7 α C4, have

2.4.a- Effect of BAs on gastrointestinal motility -- BAs exert a prokinetic effect on colonic motility, mainly through TGR5-mediated actions.(14) In a placebo-controlled, double-blind trial of 4 days' treatment, ileocolonic release of sodium chenodeoxycholate (CDC), 500 mg or 1000 mg, significantly accelerated overall and ascending colonic transit in 60 healthy volunteers. CDC was also associated with increase in stool frequency and looser stool consistency. (15) Intraluminal administration of CDC was associated with stimulation of colonic high amplitude migrating contractions. (16)

2.4.b- Effects of BAs on colonic fluid and electrolyte secretion -- BAs have been shown to increase fluid and electrolyte secretion in the colon through a variety of mechanisms (22). First, stimulation of intracellular mediators, mainly cAMP: DCA increased rabbit colon mucosal adenylate cyclase activity in a dose-dependent manner. (17, 18) Second, BAs increase water secretion by upregulating expression of colonic aquaporin channels in rat colon. (19) Other mechanisms include enteroendocrine mechanisms such as increased serotonin-induced secretion (20), neurocrine secretion through activation of TGR5 and submucosal cholinergic neurons (21), decreased water absorption in human colon (22), and increased mucosal permeability (based on biochemical tests as well as scanning electron

Comparison of biochemical, microbial and mucosal mRNA expression in bile acid diarrhoea and irritable bowel syndrome with diarrhoea

Previous studies in patients with irritable bowel syndrome (IBS) reported alterations in barrier function, immunological factors and serine protease activity in jejunal and colorectal mucosa.¹ About 30% of patients with IBS with diarrhoea (IBS-D) or functional diarrhoea have markers of abnormal bile acid (BA) metabolism (ABAM) and are diagnosed as bile acid diarrhoea (BAD).² Cholecystectomy may result in BAD; however, it is unclear whether the biochemical parameters related to BA synthesis and excretion or colonic transit differ between patients with BAD, with or without prior cholecystectomy.

Crohn's Disease Patient with Chronic Diarrhea



BILE ACIDS - SUMMARY

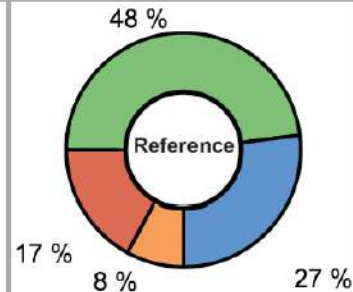
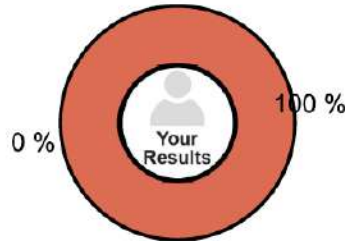
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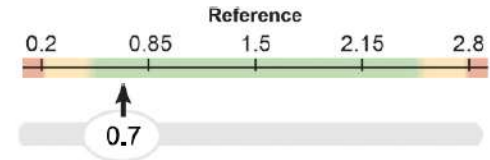
Total Bile Acids - ng/g	6.28e6		2.37e5 - 6.29e6
Secondary Bile Acids- %	50.6 L		> 90.5
Primary Bile Acids - %	49.4 H		< 7.8

Bile Acid Percentages

- Deoxycholic Acid-DCA
- Lithocholic Acid-LCA*
- Iso-LCA
- Other



Reference set at 50th percentile.



LCA*/DCA Ratio

Lithocholic Acid / Deoxycholic Acid Ratio based on absolute values.

*LCA value is the summation of LCA + Allo-LCA

Summary: Bile Acids in Gut Motility

- Elevated levels of bile acids (total & primary) may accelerate motility, while insufficient bile acids may slow motility
- Bile acid malabsorption (due to reduced absorption in the ileum) is present in at least 30% of IBS-D cases and many IBD cases
- Excess primary bile acids in the large intestine may promote inflammatory dysbiosis and exacerbate inflammation in inflammatory bowel disease

Article

Lithocholic acid phenocopies anti-ageing effects of calorie restriction

<https://doi.org/10.1038/s41586-024-08329-5>

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 Check for updates

Qi Qu^{1,10}, Yan Chen^{1,10}, Yu Wang^{1,10}, Shating Long¹, Weiche Wang¹, Heng-Ye Yang¹, Mengqi Li¹, Xiao Tian¹, Xiaoyan Wei¹, Yan-Hui Liu¹, Shengrong Xu¹, Cixiong Zhang¹, Mingxia Zhu¹, Sin Man Lam², Jianfeng Wu³, Chuyu Yun⁴, Junjie Chen⁵, Shengye Xue¹, Baoding Zhang¹, Zhong-Zheng Zheng¹, Hai-Long Piao⁶, Changtao Jiang⁷, Hao Guo^{1,8}, Guanghou Shui⁹, Xianming Deng¹, Chen-Song Zhang^{1,10} & Sheng-Cai Lin^{1,10}

Calorie restriction (CR) is a dietary intervention used to promote health and longevity^{1,2}. CR causes various metabolic changes in both the production and the circulation of metabolites³; however, it remains unclear which altered metabolites account for the physiological benefits of CR. Here we use metabolomics to analyse metabolites that exhibit changes in abundance during CR and perform subsequent functional validation. We show that lithocholic acid (LCA) is one of the metabolites that alone can recapitulate the effects of CR in mice. These effects include activation of AMP-activated protein kinase (AMPK), enhancement of muscle regeneration and rejuvenation of grip strength and running capacity. LCA also activates AMPK and induces life-extending and health-extending effects in *Caenorhabditis elegans* and *Drosophila melanogaster*. As *C. elegans* and *D. melanogaster* are not able to synthesize LCA, these results indicate that these animals are able to transmit the signalling effects of LCA once administered. Knockout of AMPK abrogates LCA-induced phenotypes in all the three animal models. Together, we identify that administration of the CR-mediated upregulated metabolite LCA alone can confer anti-ageing benefits to mice and in an AMPK-dependent manner

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SECONDARY BILE ACIDS	Abbreviation	Conjugation**	Result ng/g	Reference ng/g
Total Secondary Bile Acids			5.44e6	1.97e5 - 6.23e6
Deoxycholic Acid	DCA	U	3.44e6 H	2.24e3 - 2.33e6
Lithocholic Acid*	LCA	U	1.20e6	6.12e3 - 1.37e6
Isolithocholic Acid	ISO-LCA	U	2.78e5	2.21e3 - 5.36e5
12-Ketolithocholic Acid	12-KLCA	U	4.42e5	1.87e3 - 5.30e5
3-oxoDeoxycholic Acid	3-oxoDCA	U	4.05e4	3.53e2 - 1.12e5
Ursodeoxycholic Acid	UDCA	U	<dl	< 5.77e4
7-Ketolithocholic Acid	7-KLCA	U	1.23e4 H	< 8.94e3
7-Ketodeoxycholic Acid	7-KDCA	U	1.89e4 H	< 1.01e4
Dehydrolithocholic Acid	DHLCA	U	<dl	< 4.52e4
Hyodeoxycholic Acid	HDCA	U	<dl	< 5.27e4
Alloisolithocholic Acid	AlloIso-LCA	U	3.51e3	< 7.53e4
3-Dehydrocholic Acid	3-DHCA	U	5.80e3 H	< 5.85e2
Glycolithocholic Acid	GLCA	C	<dl	< 2.20e2
Glycoursodeoxycholic Acid	GUDCA	C	1.71e2	< 3.08e2
Glycodeoxycholic Acid	GDCA	C	4.14e2	< 5.40e2
Taurolithocholic Acid	TLCA	C	5.87e1	< 2.68e2
Tauroursodeoxycholic Acid	TUDCA	C	<dl	< 1.28e2
Taurodeoxycholic Acid	TDCA	C	2.96e2	< 8.56e2



GI-MAP™

+

StoolOMX™

Healthy Male, 60s



BILE ACIDS - SUMMARY

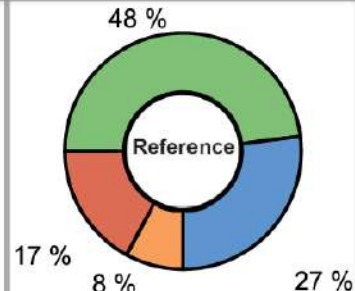
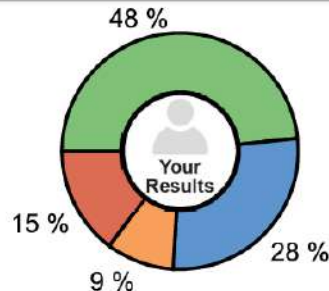
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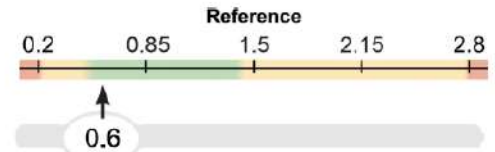
Total Bile Acids - ng/g	5.93e5		2.37e5 - 6.29e6
Secondary Bile Acids- %	99.4		> 90.5
Primary Bile Acids - %	0.6		< 7.8

Bile Acid Percentages

- Deoxycholic Acid-DCA
- Lithocholic Acid-LCA*
- Iso-LCA
- Other



Reference set at 50th percentile.



LCA*/DCA Ratio

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Healthy Male, 60s



SHORT CHAIN FATTY ACIDS - SUMMARY

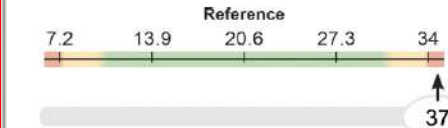
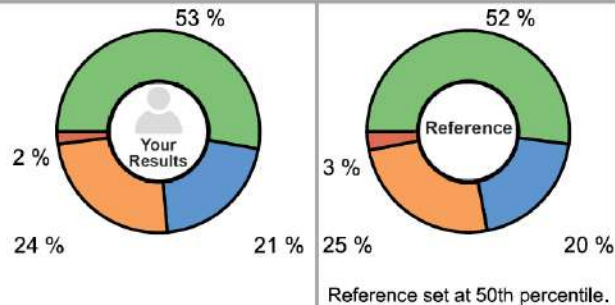
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Acetate - %	52.7		38.3 - 68.0
Butyrate - %	20.8		7.7 - 32.6
Propionate - %	24.4		14.1 - 33.6
Valerate - %	2.0		0.5 - 6.2

Major SCFA Percent

- Acetate
- Butyrate
- Propionate
- Valerate



SCFA/BCFA Ratio

Ratio of total straight chain fatty acids (SCFA) to total branched chain fatty acids (BCFA).

Healthy Male, 60s



SHORT CHAIN FATTY ACIDS - SUMMARY

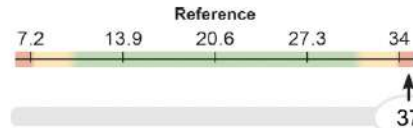
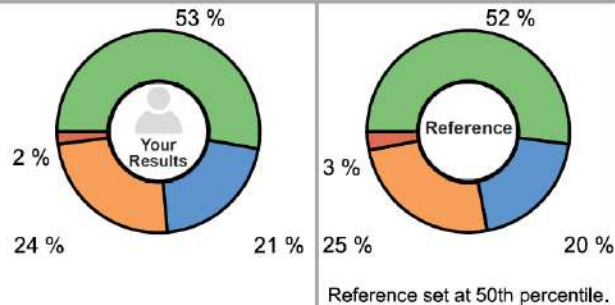
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SCFA/BCFA Ratio

Ratio of total straight chain fatty acids (SCFA) to total branched chain fatty acids (BCFA).

COMMENSAL/KEYSTONE BACTERIA



COMMENSAL BACTERIA	Result		Reference
<i>Bacteroides fragilis</i>	8.62e10		1.6e9 - 2.5e11
<i>Bifidobacterium</i> spp.	5.50e10		> 6.7e7
<i>Enterococcus</i> spp.	2.14e7		1.9e5 - 2.0e8
<i>Escherichia</i> spp.	2.96e8		3.7e6 - 3.8e9
<i>Lactobacillus</i> spp.	3.82e7		8.6e5 - 6.2e8
<i>Enterobacter</i> spp.	5.15e7 H		1.0e6 - 5.0e7
<i>Akkermansia muciniphila</i>	3.67e5		1.0e1 - 8.2e6
<i>Faecalibacterium prausnitzii</i>	1.01e6		1.0e3 - 5.0e8
<i>Roseburia</i> spp.	2.80e10 H		5.0e7 - 2.0e10
BACTERIAL PHYLA			
<i>Bacteroidetes</i>	5.63e12 H		8.6e11 - 3.3e12
<i>Firmicutes</i>	6.76e11 H		5.7e10 - 3.0e11
<i>Firmicutes:Bacteroidetes</i> Ratio	0.12		< 1.0

INTESTINAL HEALTH MARKERS

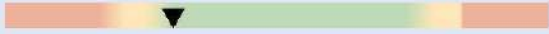

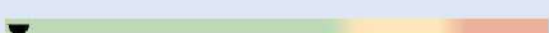
DIGESTION

	Result		Reference
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Elastase-1	240		> 200 ug/g


GI MARKERS

β -Glucuronidase	934		< 2486 U/mL
Occult Blood - FIT	0		< 10 ug/g


IMMUNE RESPONSE

Secretory IgA	666		510 - 2010 ug/g
Anti-gliadin IgA	8		< 175 U/L
Eosinophil Activation Protein (EDN, EPX)	<dl		< 2.34 ug/g

INFLAMMATION

Calprotectin	12		< 173 ug/g
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ADD-ON TESTS

Zonulin	57.4		< 175 ng/g
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Female, 60s, with Crohn's Disease, Chronic Diarrhea



BILE ACIDS - SUMMARY

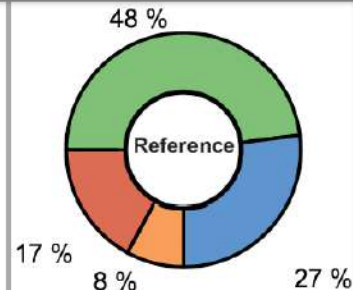
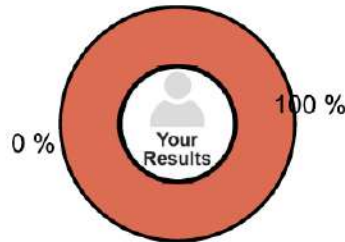
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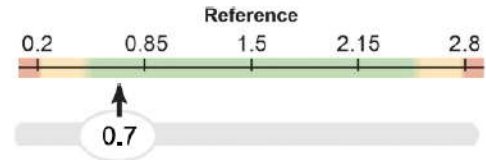
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Secondary Bile Acids- %	50.6 L		> 90.5
Primary Bile Acids - %	49.4 H		< 7.8

Bile Acid Percentages

- Deoxycholic Acid-DCA
- Lithocholic Acid-LCA*
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- Other



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Lithocholic Acid / Deoxycholic Acid Ratio based on absolute values.

*LCA value is the summation of LCA + Allo-LCA

Female, 60s, with Crohn's Disease, Chronic Diarrhea



SHORT CHAIN FATTY ACIDS - SUMMARY

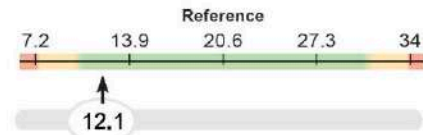
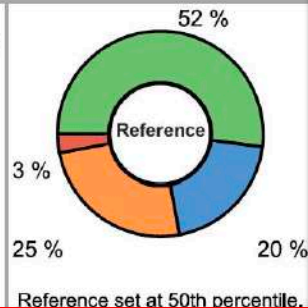
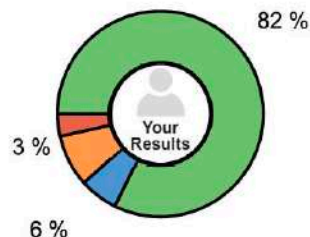
The **Postbiotic Fatty Acid Metabolite Panel** assesses fecal concentrations of straight chain and branched chain fatty acids. These metabolites provide a variety of beneficial effects for intestinal health, anti-inflammation, metabolism and immunity, and give dietary insight.

SUMMARY INFO

Major Straight Chain Fatty Acids - µg/g	8.97e3		3.63e3 - 1.95e4
Acetate - %	82.4 H		38.3 - 68.0
Butyrate - %	6.0 L		7.7 - 32.6
Propionate - %	8.2 L		14.1 - 33.6
Valerate - %	3.5		0.5 - 6.2

Major SCFA Percent

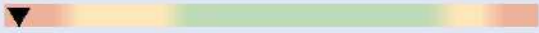

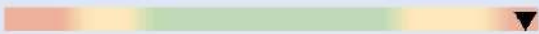
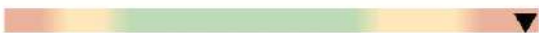
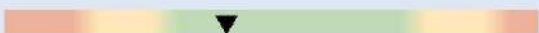

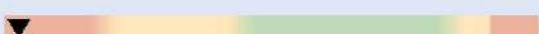



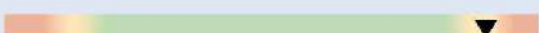

- Acetate
- Butyrate
- Propionate
- Valerate



SCFA/BCFA Ratio

Ratio of total straight chain fatty acids (SCFA) to total branched chain fatty acids (BCFA).

COMMENSAL/KEYSTONE BACTERIA

COMMENSAL BACTERIA	Result	Reference
<i>Bacteroides fragilis</i>	5.70e8 L 	1.6e9 - 2.5e11
<i>Bifidobacterium</i> spp.	6.45e9 	> 6.7e7
<i>Enterococcus</i> spp.	4.19e9 H 	1.9e5 - 2.0e8
<i>Escherichia</i> spp.	8.73e9 H 	3.7e6 - 3.8e9
<i>Lactobacillus</i> spp.	8.67e6 	8.6e5 - 6.2e8
<i>Enterobacter</i> spp.	3.71e6 	1.0e6 - 5.0e7
<i>Akkermansia muciniphila</i>	<dl L 	1.0e1 - 8.2e6
<i>Faecalibacterium prausnitzii</i>	1.12e3 	1.0e3 - 5.0e8
<i>Roseburia</i> spp.	2.34e9 	5.0e7 - 2.0e10
BACTERIAL PHYLA		
<i>Bacteroidetes</i>	3.44e9 L 	8.6e11 - 3.3e12
<i>Firmicutes</i>	2.86e11 	5.7e10 - 3.0e11
<i>Firmicutes:Bacteroidetes</i> Ratio	83.23 H 	< 1.0

OPPORTUNISTIC/OVERGROWTH MICROBES

DYSBIOTIC & OVERGROWTH BACTERIA

	Result	Reference
<i>Bacillus</i> spp.	5.80e7 High ↑	< 1.76e6
<i>Enterococcus faecalis</i>	3.09e7 High ↑	< 1.00e4
<i>Enterococcus faecium</i>	6.15e2	< 1.00e4
<i>Morganella</i> spp.	2.47e9 High ↑	< 1.00e3
<i>Pseudomonas</i> spp.	<dl	< 1.00e4
<i>Pseudomonas aeruginosa</i>	<dl	< 5.00e2
<i>Staphylococcus</i> spp.	4.47e2	< 1.00e4
<i>Staphylococcus aureus</i>	1.93e2	< 5.00e2
<i>Streptococcus</i> spp.	2.78e3 High ↑	< 1.00e3

COMMENSAL OVERGROWTH MICROBES

<i>Desulfovibrio</i> spp.	2.43e5	< 7.98e8
<i>Methanobacteriaceae</i> (family)	6.01e7	< 3.38e8

INFLAMMATORY & AUTOIMMUNE-RELATED BACTERIA

<i>Citrobacter</i> spp.	<dl	< 5.00e6
<i>Citrobacter freundii</i>	1.54e4	< 5.00e5
<i>Klebsiella</i> spp.	3.31e4 High ↑	< 5.00e3
<i>Klebsiella pneumoniae</i>	7.01e2	< 5.00e4
<i>M. avium</i> subsp. <i>paratuberculosis</i>	<dl	< 5.00e3
<i>Proteus</i> spp.	1.53e8 High ↑	< 5.00e4
<i>Proteus mirabilis</i>	1.50e5 High ↑	< 1.00e3

COMMENSAL INFLAMMATORY & AUTOIMMUNE-RELATED BACTERIA

<i>Enterobacter</i> spp.	3.71e6	< 5.00e7
<i>Escherichia</i> spp.	8.73e9 High ↑	< 3.80e9
<i>Fusobacterium</i> spp.	1.19e9 High ↑	< 1.00e8
<i>Prevotella</i> spp.	2.16e5	< 1.00e8

Male, 40s with Chronic Loose Stools



BILE ACIDS - SUMMARY

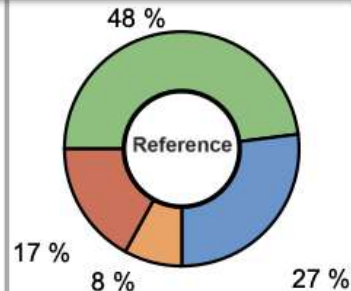
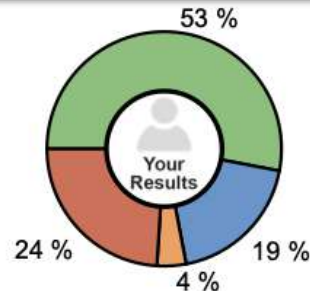
The **Bile Acids Panel** assesses fecal concentrations of primary and secondary bile acids and provides insights into microbiome diversity, digestive function, motility, and various gut-related conditions.

SUMMARY INFO

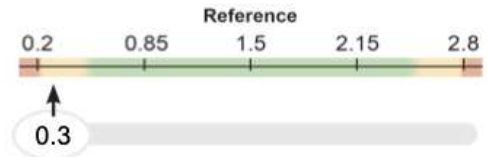
Total Bile Acids - ng/g	6.43e6 H		2.37e5 - 6.29e6
Secondary Bile Acids- %	84.6 L		> 90.5
Primary Bile Acids - %	15.4 H		< 7.8

Bile Acid Percentages

- Deoxycholic Acid-DCA
- Lithocholic Acid-LCA*
- Iso-LCA
- Other



Reference set at 50th percentile.



LCA*/DCA Ratio

Lithocholic Acid / Deoxycholic Acid Ratio based on absolute values.

*LCA value is the summation of LCA + Allo-LCA



SHORT CHAIN FATTY ACIDS - SUMMARY

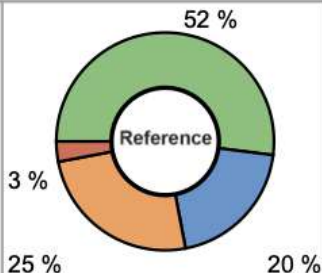
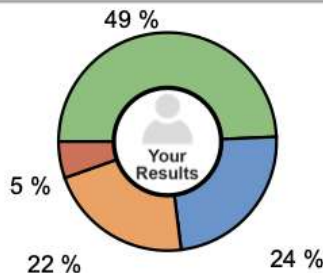
The **Postbiotic Fatty Acid Metabolite Panel** assesses fecal concentrations of straight chain and branched chain fatty acids. These metabolites provide a variety of beneficial effects for intestinal health, anti-inflammation, metabolism and immunity, and give dietary insight.

SUMMARY INFO

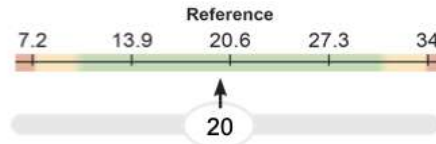
Major Straight Chain Fatty Acids - µg/g	2.26e4 H		3.63e3 - 1.95e4
Acetate - %	49.3		38.3 - 68.0
Butyrate - %	23.7		7.7 - 32.6
Propionate - %	21.6		14.1 - 33.6
Valerate - %	5.4		0.5 - 6.2

Major SCFA Percent

- Acetate
- Butyrate
- Propionate
- Valerate



Reference set at 50th percentile.



SCFA/BCFA Ratio

Ratio of total straight chain fatty acids (SCFA) to total branched chain fatty acids (BCFA).

HELICOBACTER PYLORI

H. PYLORI & VIRULENCE FACTORS

	Result	Reference
<i>Helicobacter pylori</i>	2.24e3 High ↑	< 1.00e3
Virulence Factor, babA	Negative	Negative
Virulence Factor, cagA	Negative	Negative
Virulence Factor, dupA	Negative	Negative
Virulence Factor, iceA	Negative	Negative
Virulence Factor, oipA	Negative	Negative
Virulence Factor, vacA	Negative	Negative
Virulence Factor, virB	Negative	Negative
Virulence Factor, virD	Negative	Negative

COMMENSAL/KEYSTONE BACTERIA

COMMENSAL BACTERIA	Result		Reference
<i>Bacteroides fragilis</i>	1.22e10		1.6e9 - 2.5e11
<i>Bifidobacterium</i> spp.	1.76e9		> 6.7e7
<i>Enterococcus</i> spp.	1.48e7		1.9e5 - 2.0e8
<i>Escherichia</i> spp.	7.45e7		3.7e6 - 3.8e9
<i>Lactobacillus</i> spp.	3.83e6		8.6e5 - 6.2e8
<i>Enterobacter</i> spp.	2.43e6		1.0e6 - 5.0e7
<i>Akkermansia muciniphila</i>	<dl L		1.0e1 - 8.2e6
<i>Faecalibacterium prausnitzii</i>	3.23e5		1.0e3 - 5.0e8
<i>Roseburia</i> spp.	7.06e8		5.0e7 - 2.0e10
BACTERIAL PHYLA			
<i>Bacteroidetes</i>	1.74e12		8.6e11 - 3.3e12
<i>Firmicutes</i>	7.73e10		5.7e10 - 3.0e11
<i>Firmicutes:Bacteroidetes</i> Ratio	0.04		< 1.0

OPPORTUNISTIC/OVERGROWTH MICROBES

DYSBIOTIC & OVERGROWTH BACTERIA


	Result	Reference
<i>Bacillus</i> spp.	5.83e5	< 1.76e6
<i>Enterococcus faecalis</i>	1.17e4 High ↑	< 1.00e4
<i>Enterococcus faecium</i>	9.61e2	< 1.00e4
<i>Morganella</i> spp.	2.95e4 High ↑	< 1.00e3
<i>Pseudomonas</i> spp.	<dl	< 1.00e4
<i>Pseudomonas aeruginosa</i>	<dl	< 5.00e2
<i>Staphylococcus</i> spp.	<dl	< 1.00e4
<i>Staphylococcus aureus</i>	1.83e3 High ↑	< 5.00e2
<i>Streptococcus</i> spp.	1.41e4 High ↑	< 1.00e3

COMMENSAL OVERGROWTH MICROBES

<i>Desulfovibrio</i> spp.	1.39e8	< 7.98e8
<i>Methanobacteriaceae</i> (family)	1.80e7	< 3.38e8

INTESTINAL HEALTH MARKERS

DIGESTION

	Result		Reference
Steatocrit	<dl		< 15 %
Elastase-1	313		> 200 ug/g


GI MARKERS

β -Glucuronidase	424		< 2486 U/mL
Occult Blood - FIT	2		< 10 ug/g


IMMUNE RESPONSE

Secretory IgA	5718 H		510 - 2010 ug/g
Anti-gliadin IgA	88		< 175 U/L
Eosinophil Activation Protein (EDN, EPX)	1.49		< 2.34 ug/g

INFLAMMATION

Calprotectin	27		< 173 ug/g
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ADD-ON TESTS

Zonulin	141.6		< 175 ng/g
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Case Example Summary

- M, early 40s with chronic loose stools
- High *H. pylori* (*suggests hypochlorhydria*)
- Low *Akkermansia* (*may be due to fast transit or low pH*)
- High *Staphylococcus*, *Streptococcus* & *Enterococcus* (*suggests hypochlorhydria or low pancreatic enzyme production*)
- High *Morganella* (*suggests increased LPS & histamine >> faster transit, consider food rxns*)
- Suboptimal elastase (*indicates reduced digestion*)
- High sIgA (*consider food reactions*)
- Elevated zonulin (*may be related to LPS, histamine or food rxns*)

Case Example Summary, Cont'd

- High total SCFAs (*due to fast transit and/or increased fermentable carbohydrates in large intestine*)
- High total and high % primary bile acids and low % secondary bile acids (*suggests bile acid malabsorption >> consider binders, small intestinal imbalances*)

 **GI-MAP™**
The logo features a stylized blue 'G' icon resembling a DNA double helix or a curved arrow, followed by the text 'GI-MAP™' in a bold, blue, sans-serif font.

+

StoolOMX™
The text 'StoolOMX™' is written in a bold, blue, sans-serif font, with the 'O' in 'Stool' being significantly larger than the other letters.



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