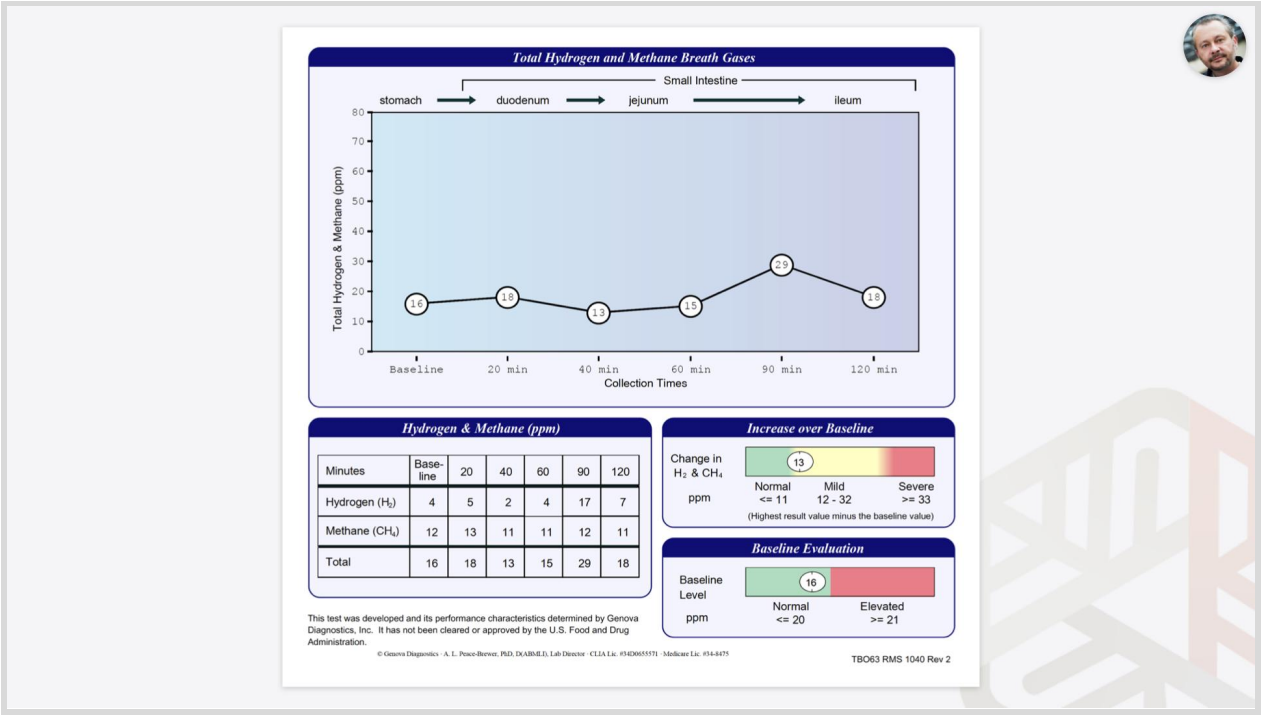
# Gut Case Studies, Part 2

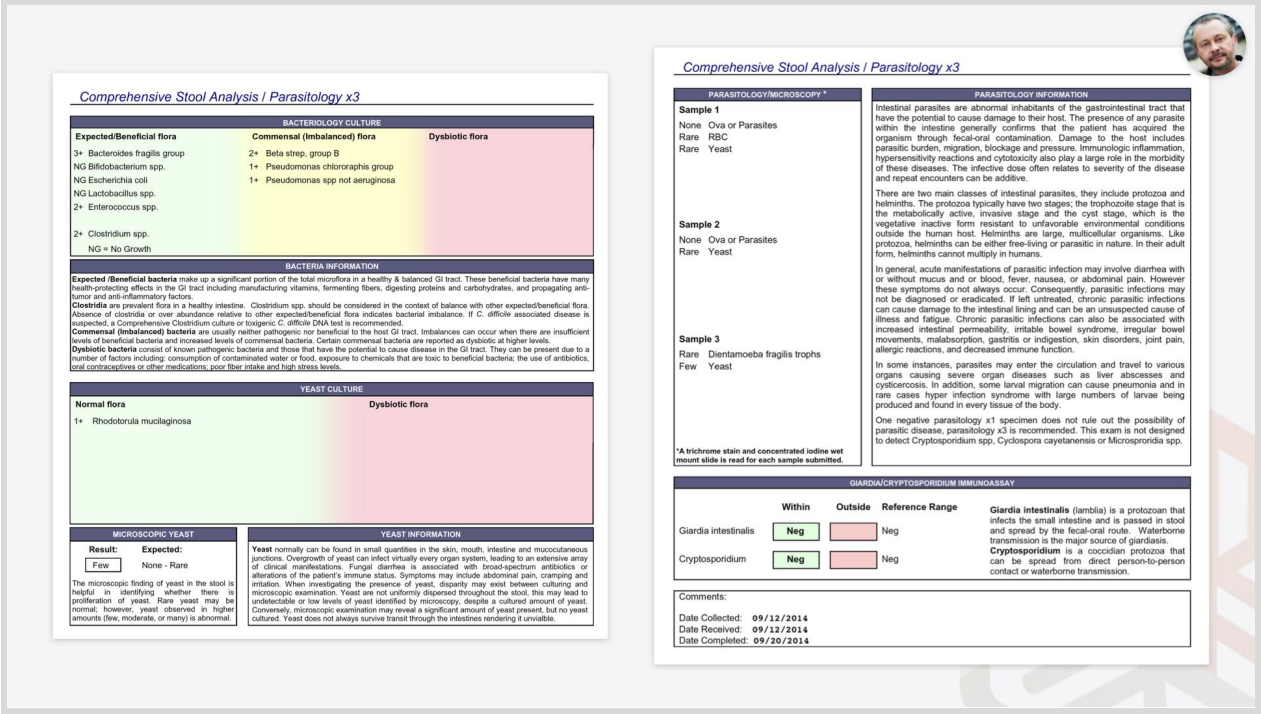
## CASE #3: 41-YEAR-OLD MALE

The next patient is a 41-year-old male. His chief complaint was very high cholesterol. And he didn't necessarily feel unwell, but he just wanted to optimize mental and physical performance and maybe even lean out a little bit. He did have some occasional postnasal drip, occasional insomnia that seems mostly lifestyle- and stress-related, and some occasional fatigue that was also related to insomnia. This is clearly connected to what was going on in his life. He is a high-powered CEO [of] a very well-known tech corporation and was burning the candle at both ends [as] is often the case of people in that position. This is another patient that Chris and I cared for a couple of years ago, so some of the labs are a little bit older. But we'll still walk through the results as it's helpful for interpretation and treatment plan design.




Here you have [a] 120-minute Genova [small intestinal bacterial overgrowth] (SIBO) breath test with just barely positive methane and with the [maximum] value being 13. With the new guidelines of intestinal methanogen overgrowth being any value over 10, this would be considered a positive result. He didn't have any known digestive symptoms that he was

complaining of, so it's just something to consider. But I would consider this a positive test, just barely positive, but still a positive. And with the lack of gut symptoms, it's not entirely clear if this could be contributing to his other issues, although I think it's important to note that I have seen cholesterol improve with treating gut infections. So it is worth considering. You'd really have to look at the rest of the gut testing, which we're about to do.



*Comprehensive Stool Analysis / Parasitology x3*

**BACTERIOLOGY CULTURE**

| Expected/Beneficial flora | Commensal (Imbalanced) flora | Dysbiotic flora |
|---|---|---|
| 3+ Bacteroides fragilis group | 2+ Beta strep, group B | |
| NG Bifidobacterium spp. | 1+ Pseudomonas chlororaphis group | |
| NG Escherichia coli | 1+ Pseudomonas spp not aeruginosa | |
| NG Lactobacillus spp. | | |
| 2+ Enterococcus spp. | | |
| 2+ Clostridium spp. | | |
| NG = No Growth | | |

**BACTERIA INFORMATION**

**Expected /Beneficial bacteria** make up a significant portion of the total microflora in a healthy & balanced GI tract. These beneficial bacteria have many health-protecting effects in the GI tract including manufacturing vitamins, fermenting fibers, digesting proteins and carbohydrates, and propagating anti-tumor and anti-inflammatory factors.
**Clostridia** are prevalent flora in a healthy intestine. Clostridium spp. should be considered in the context of balance with other expected/beneficial flora. Absence of clostridia or over abundance relative to other expected/beneficial flora indicates bacterial imbalance. If *C. difficile* associated disease is suspected, a Comprehensive Clostridium culture or toxigenic *C. difficile* DNA test is recommended.
**Commensal (imbalanced) bacteria** are usually neither pathogenic nor beneficial to the host GI tract. Imbalances can occur when there are insufficient levels of beneficial bacteria and increased levels of commensal bacteria. Certain commensal bacteria are reported as dysbiotic at higher levels.
**Dysbiotic bacteria** consist of known pathogenic bacteria and those that have the potential to cause disease in the GI tract. They can be present due to a number of factors including: consumption of contaminated water or food, exposure to chemicals that are toxic to beneficial bacteria; the use of antibiotics, oral contraceptives or other medications; poor fiber intake and high stress levels.

**YEAST CULTURE**

| Normal flora | Dysbiotic flora |
|---|---|
| 1+ Rhodotorula mucilaginosa | |

**MICROSCOPIC YEAST**

| Result: | Expected: |
|---|---|
| Few | None - Rare |

The microscopic finding of yeast in the stool is helpful in identifying whether there is proliferation of yeast. Rare yeast may be normal; however, yeast observed in higher amounts (few, moderate, or many) is abnormal.

**YEAST INFORMATION**

**Yeast** normally can be found in small quantities in the skin, mouth, intestine and mucocutaneous junctions. Overgrowth of yeast can infect virtually every organ system, leading to an extensive array of clinical manifestations. Fungal diarrhea is associated with broad-spectrum antibiotics or alterations of the patient's immune status. Symptoms may include abdominal pain, cramping and irritation. When investigating the presence of yeast, disparity may exist between culturing and microscopic examination. Yeast are not uniformly dispersed throughout the stool, this may lead to undetectable or low levels of yeast identified by microscopy, despite a cultured amount of yeast. Conversely, microscopic examination may reveal a significant amount of yeast present, but no yeast cultured. Yeast does not always survive transit through the intestines rendering it unviable.

*Comprehensive Stool Analysis / Parasitology x3*

**PARASITOLOGY/MICROSCOPY ***

**Sample 1**
None Ova or Parasites
Rare RBC
Rare Yeast

**Sample 2**
None Ova or Parasites
Rare Yeast

**Sample 3**
Rare Dientamoeba fragilis trophs
Few Yeast

***A trichrome stain and concentrated iodine wet mount slide is read for each sample submitted.**

**PARASITOLOGY INFORMATION**

Intestinal parasites are abnormal inhabitants of the gastrointestinal tract that have the potential to cause damage to their host. The presence of any parasite within the intestine generally confirms that the patient has acquired the organism through fecal-oral contamination. Damage to the host includes parasitic burden, migration, blockage and pressure. Immunologic inflammation, hypersensitivity reactions and cytotoxicity also play a large role in the morbidity of these diseases. The infective dose often relates to severity of the disease and repeat encounters can be additive.

There are two main classes of intestinal parasites, they include protozoa and helminths. The protozoa typically have two stages; the trophozoite stage that is the metabolically active, invasive stage and the cyst stage, which is the vegetative inactive form resistant to unfavorable environmental conditions outside the human host. Helminths are large, multicellular organisms. Like protozoa, helminths can be either free-living or parasitic in nature. In their adult form, helminths cannot multiply in humans.

In general, acute manifestations of parasitic infection may involve diarrhea with or without mucus and or blood, fever, nausea, or abdominal pain. However these symptoms do not always occur. Consequently, parasitic infections may not be diagnosed or eradicated. If left untreated, chronic parasitic infections can cause damage to the intestinal lining and can be an unsuspected cause of illness and fatigue. Chronic parasitic infections can also be associated with increased intestinal permeability, irritable bowel syndrome, irregular bowel movements, malabsorption, gastritis or indigestion, skin disorders, joint pain, allergic reactions, and decreased immune function.

In some instances, parasites may enter the circulation and travel to various organs causing severe organ diseases such as liver abscesses and cysticercosis. In addition, some larval migration can cause pneumonia and in rare cases hyper infection syndrome with large numbers of larvae being produced and found in every tissue of the body.

One negative parasitology x1 specimen does not rule out the possibility of parasitic disease, parasitology x3 is recommended. This exam is not designed to detect Cryptosporidium spp, Cyclospora cayetanensis or Microsproridia spp.

**GIARDIA/CRYPTOSPORIDIUM IMMUNOASSAY**

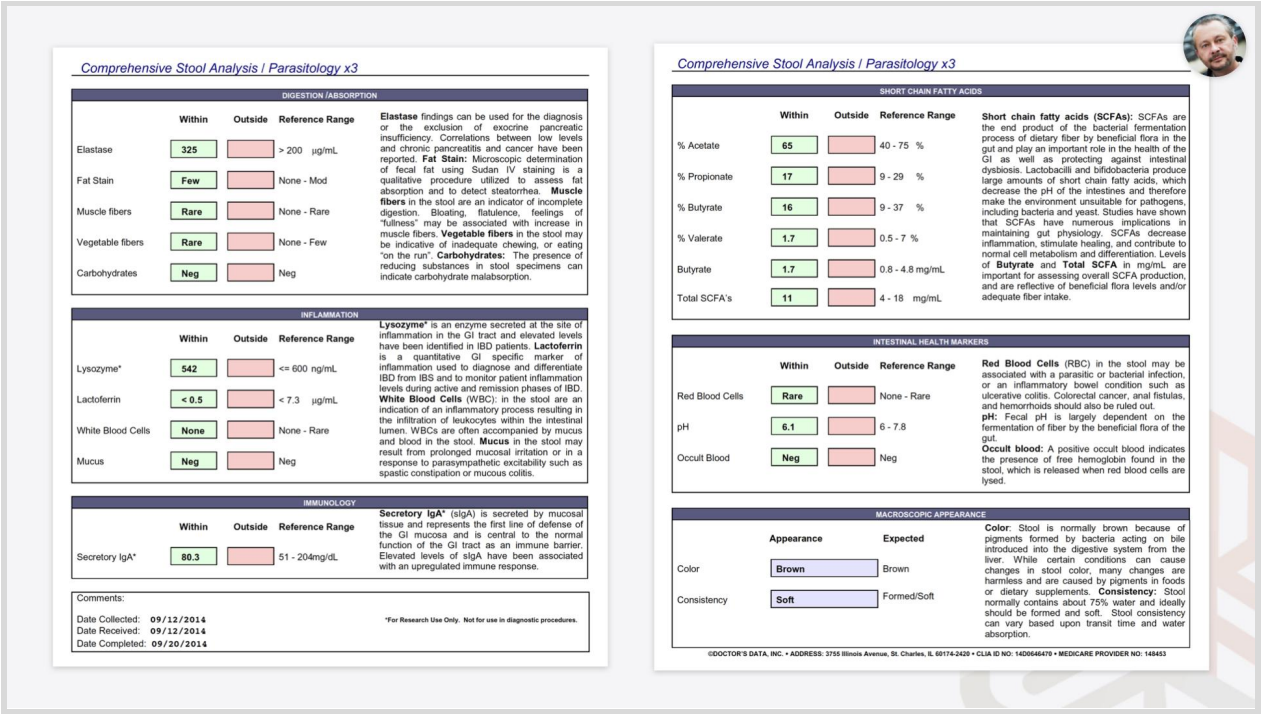
| | Within | Outside | Reference Range |
|---|---|---|---|
| Giardia intestinalis | Neg | | Neg |
| Cryptosporidium | Neg | | Neg |

**Giardia intestinalis** (lamblia) is a protozoan that infects the small intestine and is passed in stool and spread by the fecal-oral route. Waterborne transmission is the major source of giardiasis.
**Cryptosporidium** is a coccidian protozoa that can be spread from direct person-to-person contact or waterborne transmission.

Comments:

Date Collected: 09/12/2014
Date Received: 09/12/2014
Date Completed: 09/20/2014

The Doctor's Data stool test showed pretty significant dysbiosis. They weren't able to grow any *Bifidobacterium*, beneficial [*Escherichia*] *coli*, or *Lactobacillus* in his stool. And he did have some mild fungal overgrowth with few on the microscopic yeast exam. And then he had *Dientamoeba fragilis* in one of the stool specimens. As you recall, that's similar to *Blastocystis hominis*, as its pathogenicity has been somewhat controversial over time.



*Comprehensive Stool Analysis / Parasitology x3*

**DIGESTION /ABSORPTION**

| | Within | Outside | Reference Range |
|---|---|---|---|
| Elastase | 325 | | > 200 µg/mL |
| Fat Stain | Few | | None - Mod |
| Muscle fibers | Rare | | None - Rare |
| Vegetable fibers | Rare | | None - Few |
| Carbohydrates | Neg | | Neg |

**Elastase** findings can be used for the diagnosis or the exclusion of exocrine pancreatic insufficiency. Correlations between low levels and chronic pancreatitis and cancer have been reported. **Fat Stain:** Microscopic determination of fecal fat using Sudan IV staining is a qualitative procedure utilized to assess fat absorption and to detect steatorrhea. **Muscle fibers** in the stool are an indicator of incomplete digestion. Bloating, flatulence, feelings of "fullness" may be associated with increase in muscle fibers. **Vegetable fibers** in the stool may be indicative of inadequate chewing, or eating "on the run". **Carbohydrates:** The presence of reducing substances in stool specimens can indicate carbohydrate malabsorption.

**INFLAMMATION**

| | Within | Outside | Reference Range |
|---|---|---|---|
| Lysozyme* | 542 | | <= 600 ng/mL |
| Lactoferrin | < 0.5 | | < 7.3 µg/mL |
| White Blood Cells | None | | None - Rare |
| Mucus | Neg | | Neg |

**Lysozyme*** is an enzyme secreted at the site of inflammation in the GI tract and elevated levels have been identified in IBD patients. **Lactoferrin** is a quantitative GI specific marker of inflammation used to diagnose and differentiate IBD from IBS and to monitor patient inflammation levels during active and remission phases of IBD. **White Blood Cells** (WBC): in the stool are an indication of an inflammatory process resulting in the infiltration of leukocytes within the intestinal lumen. WBCs are often accompanied by mucus and blood in the stool. **Mucus** in the stool may result from prolonged mucosal irritation or in a response to parasympathetic excitability such as spastic constipation or mucous colitis.

**IMMUNOLOGY**

| | Within | Outside | Reference Range |
|---|---|---|---|
| Secretory IgA* | 80.3 | | 51 - 204mg/dL |

**Secretory IgA*** (sIgA) is secreted by mucosal tissue and represents the first line of defense of the GI mucosa and is central to the normal function of the GI tract as an immune barrier. Elevated levels of sIgA have been associated with an upregulated immune response.

Comments:

Date Collected: 09/12/2014
Date Received: 09/12/2014
Date Completed: 09/20/2014

*For Research Use Only. Not for use in diagnostic procedures.

*Comprehensive Stool Analysis / Parasitology x3*

**SHORT CHAIN FATTY ACIDS**

| | Within | Outside | Reference Range |
|---|---|---|---|
| % Acetate | 65 | | 40 - 75 % |
| % Propionate | 17 | | 9 - 29 % |
| % Butyrate | 16 | | 9 - 37 % |
| % Valerate | 1.7 | | 0.5 - 7 % |
| Butyrate | 1.7 | | 0.8 - 4.8 mg/mL |
| Total SCFA's | 11 | | 4 - 18 mg/mL |

**Short chain fatty acids (SCFAs):** SCFAs are the end product of the bacterial fermentation process of dietary fiber by beneficial flora in the gut and play an important role in the health of the GI as well as protecting against intestinal dysbiosis. Lactobacilli and bifidobacteria produce large amounts of short chain fatty acids, which decrease the pH of the intestines and therefore make the environment unsuitable for pathogens, including bacteria and yeast. Studies have shown that SCFAs have numerous implications in maintaining gut physiology. SCFAs decrease inflammation, stimulate healing, and contribute to normal cell metabolism and differentiation. Levels of **Butyrate** and **Total SCFA** in mg/mL are important for assessing overall SCFA production, and are reflective of beneficial flora levels and/or adequate fiber intake.

**INTESTINAL HEALTH MARKERS**

| | Within | Outside | Reference Range |
|---|---|---|---|
| Red Blood Cells | Rare | | None - Rare |
| pH | 6.1 | | 6 - 7.8 |
| Occult Blood | Neg | | Neg |

**Red Blood Cells** (RBC) in the stool may be associated with a parasitic or bacterial infection, or an inflammatory bowel condition such as ulcerative colitis. Colorectal cancer, anal fistulas, and hemorrhoids should also be ruled out.
**pH:** Fecal pH is largely dependent on the fermentation of fiber by the beneficial flora of the gut.
**Occult blood:** A positive occult blood indicates the presence of free hemoglobin found in the stool, which is released when red blood cells are lysed.

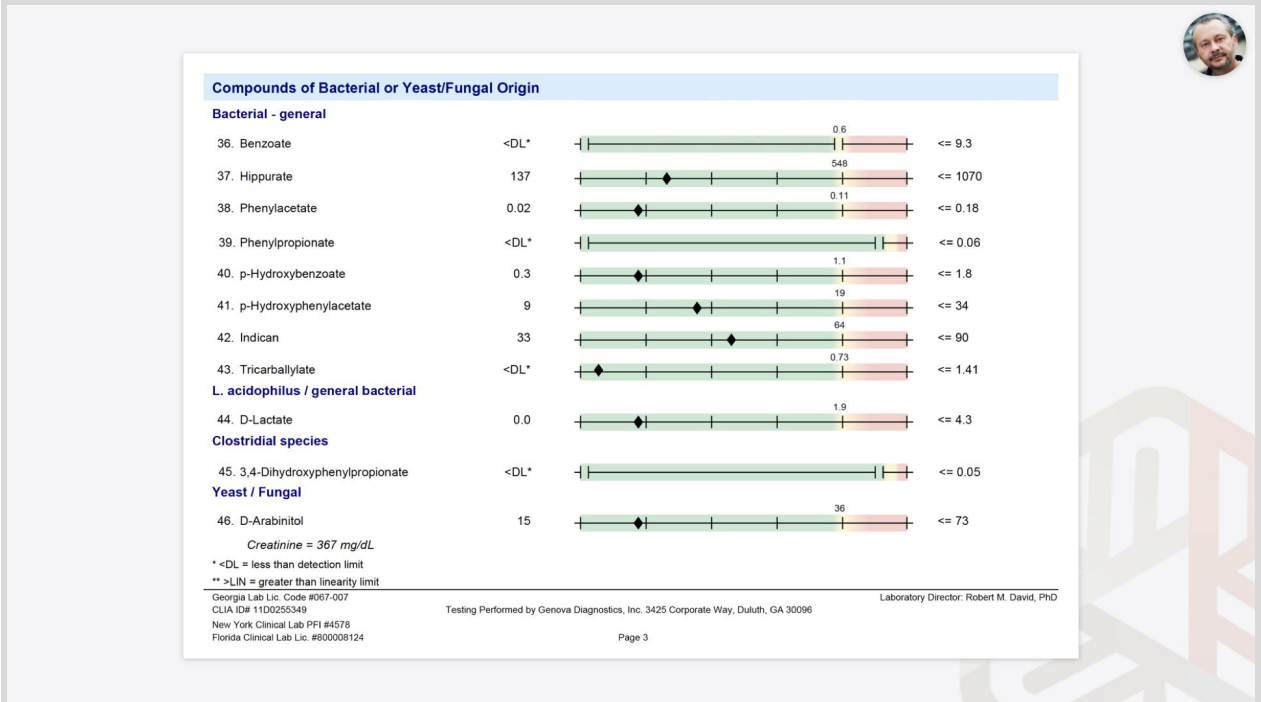
**MACROSCOPIC APPEARANCE**

| | Appearance | Expected |
|---|---|---|
| Color | Brown | Brown |
| Consistency | Soft | Formed/Soft |

**Color:** Stool is normally brown because of pigments formed by bacteria acting on bile introduced into the digestive system from the liver. While certain conditions can cause changes in stool color, many changes are harmless and are caused by pigments in foods or dietary supplements. **Consistency:** Stool normally contains about 75% water and ideally should be formed and soft. Stool consistency can vary based upon transit time and water absorption.



Here [are] the next couple of pages. Nothing really happening in [the] digestion or inflammation or immunology sections or in the short-chain fatty acids or intestinal health. So [he had] a pretty normal result on his stool test.



**Compounds of Bacterial or Yeast/Fungal Origin**

| | | | |
|---|---|---|---|
| **Bacterial - general** | | | |
| 36. Benzoate | <DL* | 0.6 | <= 9.3 |
| 37. Hippurate | 137 | 548 | <= 1070 |
| 38. Phenylacetate | 0.02 | 0.11 | <= 0.18 |
| 39. Phenylpropionate | <DL* | | <= 0.06 |
| 40. p-Hydroxybenzoate | 0.3 | 1.1 | <= 1.8 |
| 41. p-Hydroxyphenylacetate | 9 | 19 | <= 34 |
| 42. Indican | 33 | 64 | <= 90 |
| 43. Tricarballylate | <DL* | 0.73 | <= 1.41 |
| **L. acidophilus / general bacterial** | | | |
| 44. D-Lactate | 0.0 | 1.9 | <= 4.3 |
| **Clostridial species** | | | |
| 45. 3,4-Dihydroxyphenylpropionate | <DL* | | <= 0.05 |
| **Yeast / Fungal** | | | |
| 46. D-Arabinitol | 15 | 36 | <= 73 |

*Creatinine = 367 mg/dL*

* <DL = less than detection limit
** >LIN = greater than linearity limit

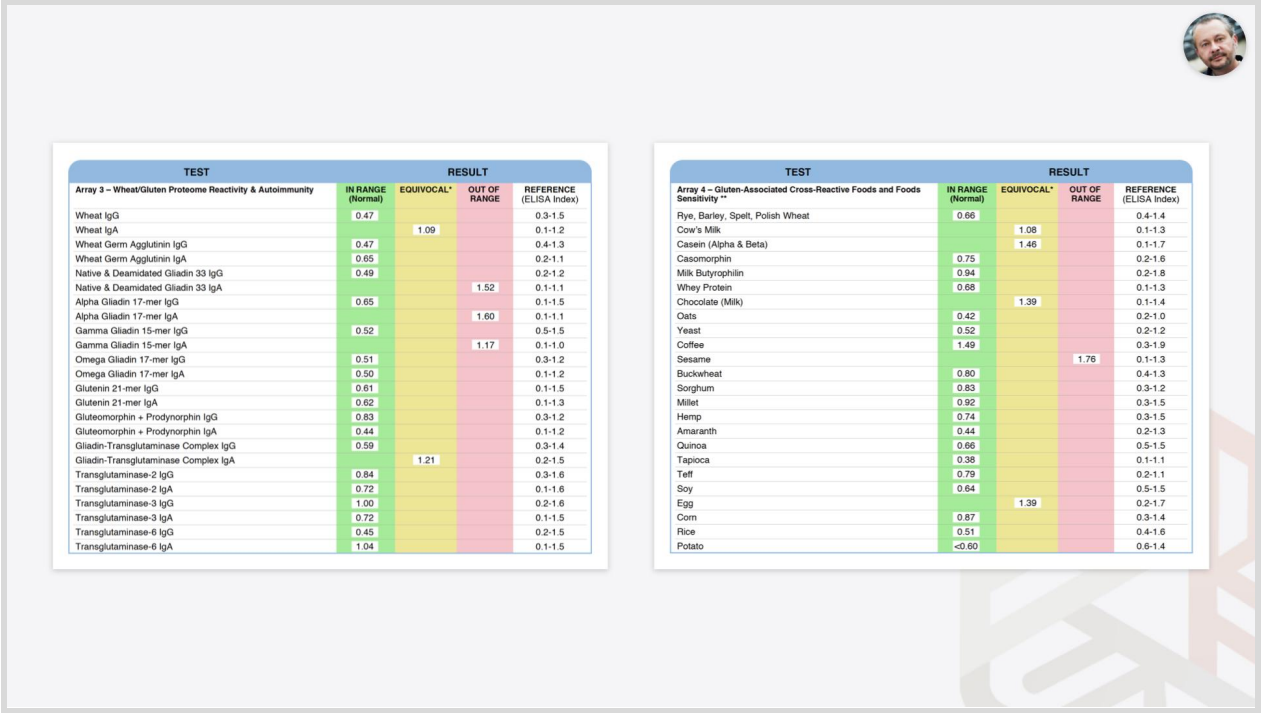
Georgia Lab Lic. Code #067-007
CLIA ID# 11D0255349
New York Clinical Lab PFI #4578
Florida Clinical Lab Lic. #800008124

Testing Performed by Genova Diagnostics, Inc. 3425 Corporate Way, Duluth, GA 30096

Laboratory Director: Robert M. David, PhD

Page 3

And here's the urine organic acids [test], [which is] also pretty much normal.



| TEST | RESULT | | | |
|---|---|---|---|---|
| Array 3 – Wheat/Gluten Proteome Reactivity & Autoimmunity | IN RANGE (Normal) | EQUIVOCAL* | OUT OF RANGE | REFERENCE (ELISA Index) |
| Wheat IgG | 0.47 | | | 0.3-1.5 |
| Wheat IgA | | 1.09 | | 0.1-1.2 |
| Wheat Germ Agglutinin IgG | 0.47 | | | 0.4-1.3 |
| Wheat Germ Agglutinin IgA | 0.65 | | | 0.2-1.1 |
| Native & Deamidated Gliadin 33 IgG | 0.49 | | | 0.2-1.2 |
| Native & Deamidated Gliadin 33 IgA | | | 1.52 | 0.1-1.1 |
| Alpha Gliadin 17-mer IgG | 0.65 | | | 0.1-1.5 |
| Alpha Gliadin 17-mer IgA | | | 1.60 | 0.1-1.1 |
| Gamma Gliadin 15-mer IgG | 0.52 | | | 0.5-1.5 |
| Gamma Gliadin 15-mer IgA | | | 1.17 | 0.1-1.0 |
| Omega Gliadin 17-mer IgG | 0.51 | | | 0.3-1.2 |
| Omega Gliadin 17-mer IgA | 0.50 | | | 0.1-1.2 |
| Glutenin 21-mer IgG | 0.61 | | | 0.1-1.5 |
| Glutenin 21-mer IgA | 0.62 | | | 0.1-1.3 |
| Gluteomorphin + Prodynorphin IgG | 0.83 | | | 0.3-1.2 |
| Gluteomorphin + Prodynorphin IgA | 0.44 | | | 0.1-1.2 |
| Gliadin-Transglutaminase Complex IgG | 0.59 | | | 0.3-1.4 |
| Gliadin-Transglutaminase Complex IgA | | 1.21 | | 0.2-1.5 |
| Transglutaminase-2 IgG | 0.84 | | | 0.3-1.6 |
| Transglutaminase-2 IgA | 0.72 | | | 0.1-1.6 |
| Transglutaminase-3 IgG | 1.00 | | | 0.2-1.6 |
| Transglutaminase-3 IgA | 0.72 | | | 0.1-1.5 |
| Transglutaminase-6 IgG | 0.45 | | | 0.2-1.5 |
| Transglutaminase-6 IgA | 1.04 | | | 0.1-1.5 |

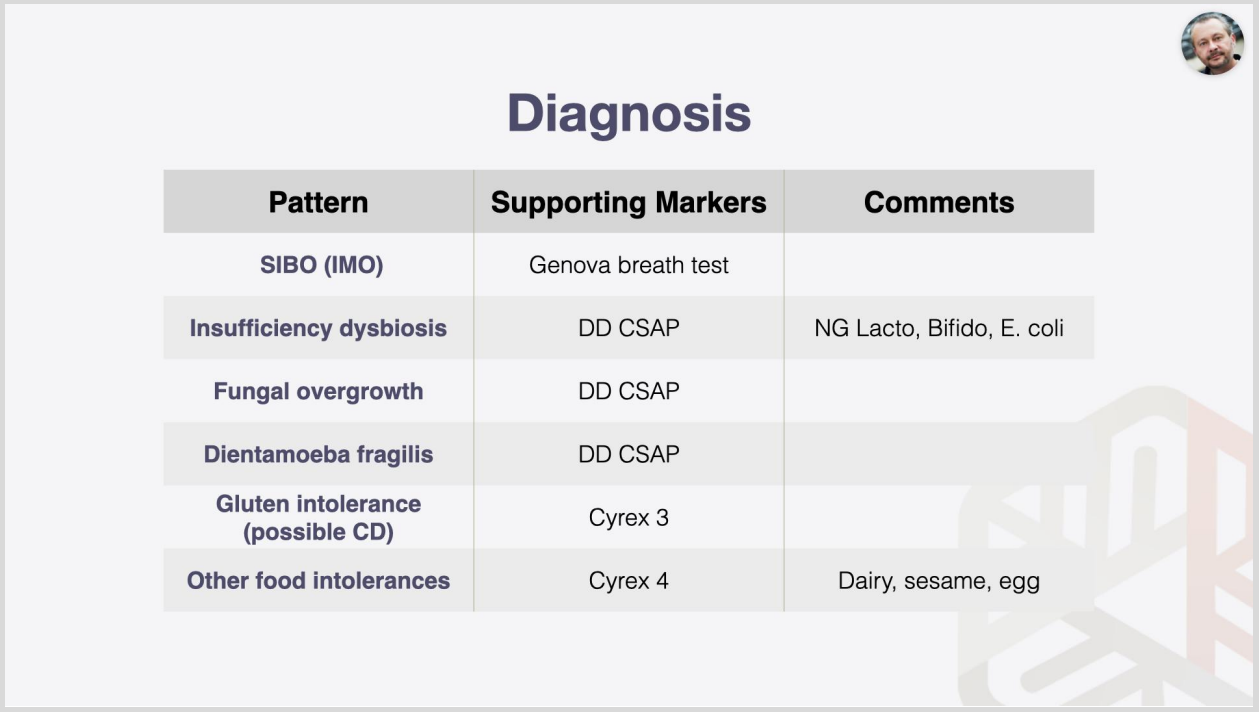
| TEST | RESULT | | | |
|---|---|---|---|---|
| Array 4 – Gluten-Associated Cross-Reactive Foods and Foods Sensitivity ** | IN RANGE (Normal) | EQUIVOCAL* | OUT OF RANGE | REFERENCE (ELISA Index) |
| Rye, Barley, Spelt, Polish Wheat | 0.66 | | | 0.4-1.4 |
| Cow's Milk | | 1.08 | | 0.1-1.3 |
| Casein (Alpha & Beta) | | 1.46 | | 0.1-1.7 |
| Casomorphin | 0.75 | | | 0.2-1.6 |
| Milk Butyrophilin | 0.94 | | | 0.2-1.8 |
| Whey Protein | 0.68 | | | 0.1-1.3 |
| Chocolate (Milk) | | 1.39 | | 0.1-1.4 |
| Oats | 0.42 | | | 0.2-1.0 |
| Yeast | 0.52 | | | 0.2-1.2 |
| Coffee | 1.49 | | | 0.3-1.9 |
| Sesame | | | 1.76 | 0.1-1.3 |
| Buckwheat | 0.80 | | | 0.4-1.3 |
| Sorghum | 0.83 | | | 0.3-1.2 |
| Millet | 0.92 | | | 0.3-1.5 |
| Hemp | 0.74 | | | 0.3-1.5 |
| Amaranth | 0.44 | | | 0.2-1.3 |
| Quinoa | 0.66 | | | 0.5-1.5 |
| Tapioca | 0.38 | | | 0.1-1.1 |
| Teff | 0.79 | | | 0.2-1.1 |
| Soy | 0.64 | | | 0.5-1.5 |
| Egg | | 1.39 | | 0.2-1.7 |
| Corn | 0.87 | | | 0.3-1.4 |
| Rice | 0.51 | | | 0.4-1.6 |
| Potato | <0.60 | | | 0.6-1.4 |

Here's a Cyrex Array 3 and [an] Array [4] for him. As a reminder, the Cyrex [Array 3] panel is now the 3X, and it has been updated to include more markers. So for this patient, he wasn't eating a lot of gluten, but he was still eating it occasionally when he traveled and ate out. So he wanted to find out whether it was a problem for him. And sure enough, it was. You can see here he's got [immunoglobulin A] (IgA) antibodies to native and deamidated gliadin, and IgA antibodies to alpha gliadin and IgA antibodies to gamma gliadin, and then also IgA antibodies to gliadin transglutaminase complex and to wheat. Interestingly enough, this patient's producing exclusively IgA antibodies rather than [immunoglobulin G] (IgG). So what you could expect, given the native deamidated gliadin antibodies and alpha gliadin antibodies and the transglutaminase complex, suspecting celiac [disease] is worth consideration.

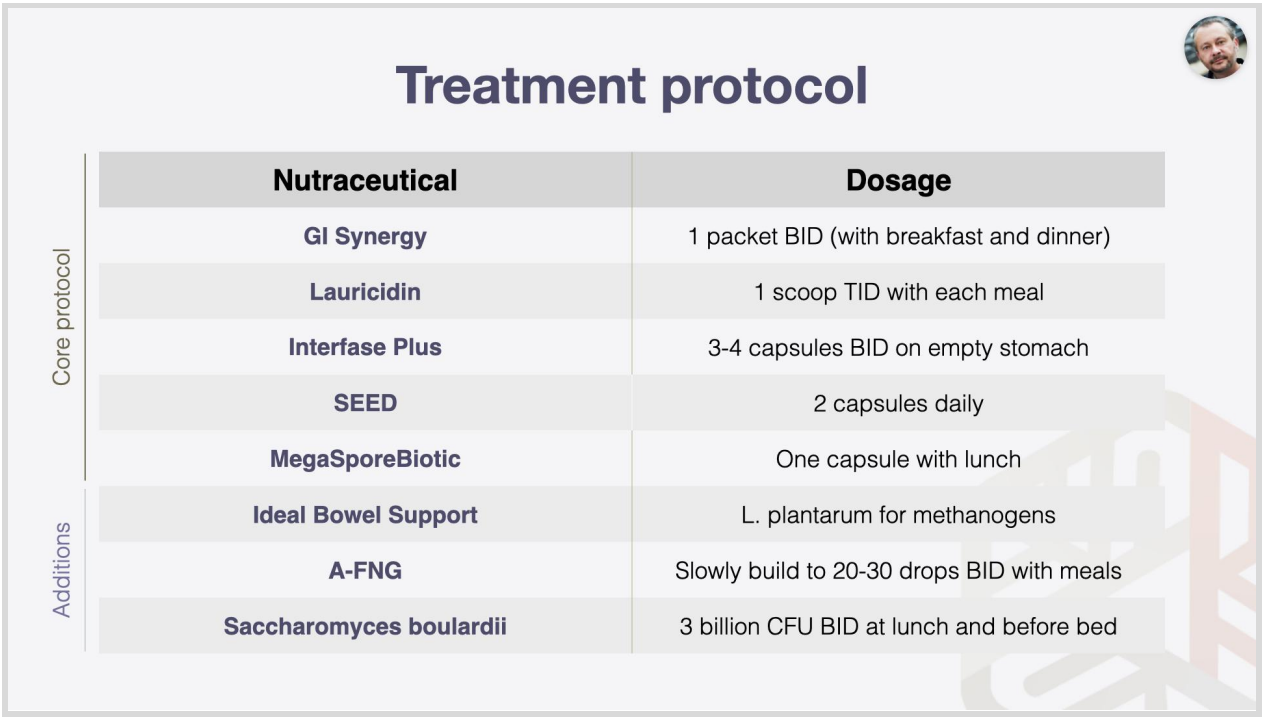
The patient [agreed] to completely cut out gluten. He didn't really see the need [to get] a follow-up or further testing for celiac [disease] because he was fine with completely removing wheat and any of the other potential cross-reactive proteins.

On [Cyrex] Array 4, he tested positive for dairy, cow's milk, casein and chocolate milk, and then sesame and egg. So he [removed] those from his diet, as well, as part of the elimination and

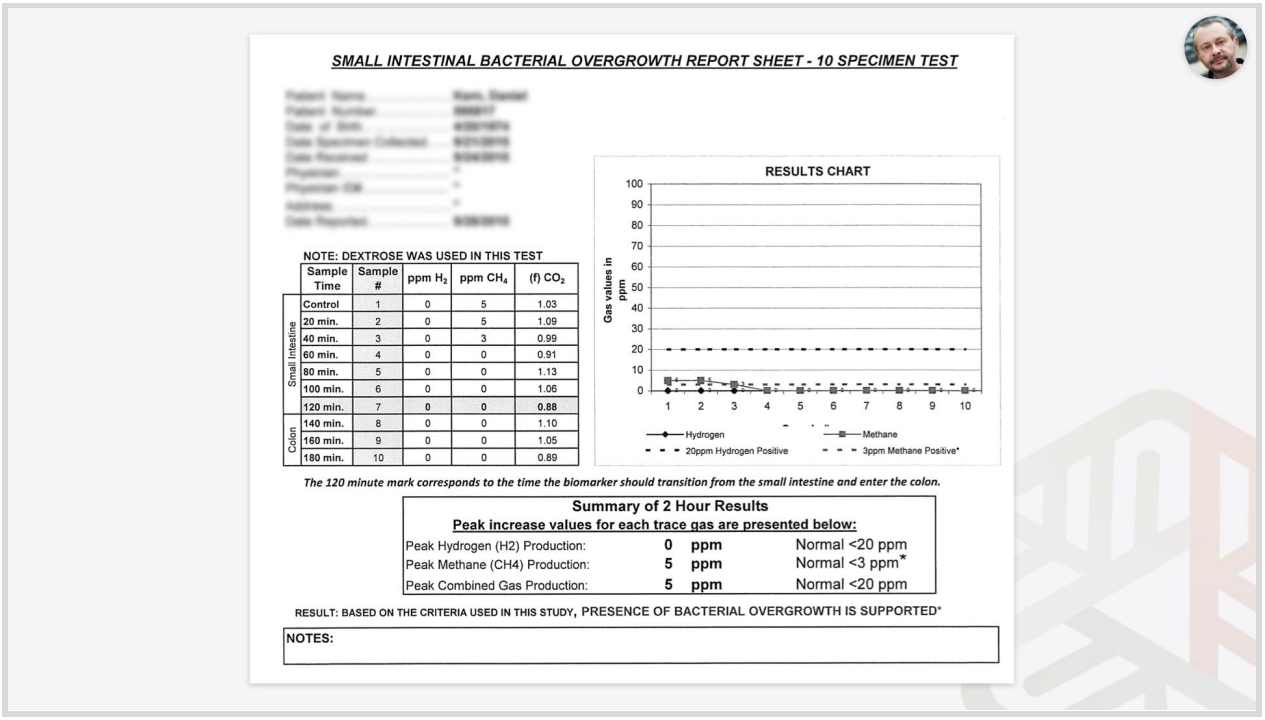
experimentation phase. In reviewing this later, as our clinical practice evolves, we now run immunoglobulin panels along [with] this test for IgA and IgG production to ensure proper production and proper interpretation.




So here's a diagnosis: intestinal methanogen overgrowth borderline. Again, pathogenicity [is] a little unclear here. Insufficiency dysbiosis on [the] Doctor's Data stool panel along with fungal overgrowth and *Dientamoeba fragilis*, gluten intolerance, and possible celiac [disease] and some other food intolerances on the Cyrex [Array] 4 panel.




Here's a summary of the treatment plan we went with. I've gone back and forth, tweaked this slightly to reflect updated and current product recommendations. So we decided to do the antimicrobial protocol based on the borderline SIBO, the *D. fragilis*, and the fungal overgrowth. If it was just insufficiency dysbiosis, I probably wouldn't have done antimicrobials and would have gone right to [the] prebiotic and probiotic rebuilding of the gut phase. But in this case, because of the SIBO, *D. fragilis*, and fungal overgrowth, we decided to do 30 to 60 days on that antimicrobial protocol. And his main complaint was high cholesterol. And since we know that gut issues can actually be a major contributor to cholesterol metabolism—and we wanted to approach high cholesterol from a functional perspective instead of just taking statins—he was motivated to address some of these underlying causes to see if that brought down his cholesterol levels. So I added a few things based on his presentation, like Ideal Bowel Support, which is *Lactobacillus plantarum*, which degrades methane because he had primarily elevated methane levels for the SIBO test. He didn't want to do any pharmaceuticals to begin with, so we started here [with] A- FNG for the fungal overgrowth and *Saccharomyces boulardii* for fungal overgrowth and also for *D. fragilis*. As you know from the treatment protocol section, *Saccharomyces boulardii* can be a helpful antiparasitic agent.



**SMALL INTESTINAL BACTERIAL OVERGROWTH REPORT SHEET - 10 SPECIMEN TEST**

[redacted]

NOTE: DEXTROSE WAS USED IN THIS TEST

| | Sample Time | Sample # | ppm $H_2$ | ppm $CH_4$ | (f) $CO_2$ |
|---|---|---|---|---|---|
| Small Intestine | Control | 1 | 0 | 5 | 1.03 |
| | 20 min. | 2 | 0 | 5 | 1.09 |
| | 40 min. | 3 | 0 | 3 | 0.99 |
| | 60 min. | 4 | 0 | 0 | 0.91 |
| | 80 min. | 5 | 0 | 0 | 1.13 |
| | 100 min. | 6 | 0 | 0 | 1.06 |
| | 120 min. | 7 | 0 | 0 | 0.88 |
| Colon | 140 min. | 8 | 0 | 0 | 1.10 |
| | 160 min. | 9 | 0 | 0 | 1.05 |
| | 180 min. | 10 | 0 | 0 | 0.89 |

RESULTS CHART



*The 120 minute mark corresponds to the time the biomarker should transition from the small intestine and enter the colon.*

**Summary of 2 Hour Results**

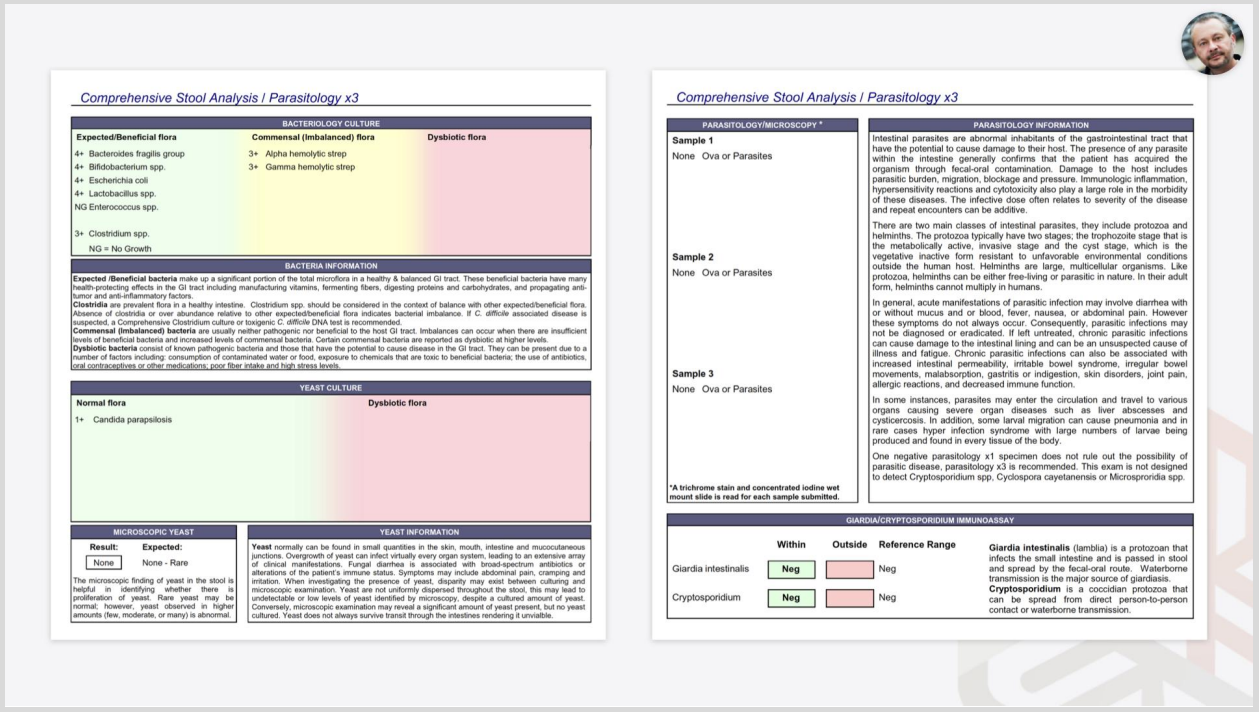
**Peak increase values for each trace gas are presented below:**

| | | |
|---|---|---|
| Peak Hydrogen (H2) Production: | **0 ppm** | Normal <20 ppm |
| Peak Methane (CH4) Production: | **5 ppm** | Normal <3 ppm* |
| Peak Combined Gas Production: | **5 ppm** | Normal <20 ppm |

RESULT: BASED ON THE CRITERIA USED IN THIS STUDY, PRESENCE OF BACTERIAL OVERGROWTH IS SUPPORTED*

NOTES:

We did a follow-up with Commonwealth Labs. Again, we aren't using them in practice anymore, but here you can see methane is negative for the new North American Consensus. But at the time of the test, Commonwealth was using [inaudible]'s criteria of more than three parts per million as positive. So that's why it's marked positive here. [It's] just important to remember that interpretation of the SIBO test has changed over the years, and you may have people [who] bring you old tests. So it's important to have that context. As we've discussed, when both hydrogen and methane are at zeros, it can be representative of hydrogen sulfide excess. This presentation is a little bit more tricky because you do have some methane, and it's right after an antimicrobial treatment protocol. So considering his lack of symptoms, we didn't really continue to pursue additional treatments at this time.



*Comprehensive Stool Analysis / Parasitology x3*

**BACTERIOLOGY CULTURE**

| Expected/Beneficial flora | Commensal (Imbalanced) flora | Dysbiotic flora |
|---|---|---|
| 4+ Bacteroides fragilis group | 3+ Alpha hemolytic strep | |
| 4+ Bifidobacterium spp. | 3+ Gamma hemolytic strep | |
| 4+ Escherichia coli | | |
| 4+ Lactobacillus spp. | | |
| NG Enterococcus spp. | | |
| 3+ Clostridium spp. | | |
| NG = No Growth | | |

**BACTERIA INFORMATION**

**Expected /Beneficial bacteria** make up a significant portion of the total microflora in a healthy & balanced GI tract. These beneficial bacteria have many health-protecting effects in the GI tract including manufacturing vitamins, fermenting fibers, digesting proteins and carbohydrates, and propagating anti-tumor and anti-inflammatory factors.
**Clostridia** are prevalent flora in a healthy intestine. Clostridium spp. should be considered in the context of balance with other expected/beneficial flora. Absence of clostridia or over abundance relative to other expected/beneficial flora indicates bacterial imbalance. If *C. difficile* associated disease is suspected, a Comprehensive Clostridium culture or toxigenic *C. difficile* DNA test is recommended.
**Commensal (imbalanced) bacteria** are usually neither pathogenic nor beneficial to the host GI tract. Imbalances can occur when there are insufficient levels of beneficial bacteria and increased levels of commensal bacteria. Certain commensal bacteria are reported as dysbiotic at higher levels.
**Dysbiotic bacteria** consist of known pathogenic bacteria and those that have the potential to cause disease in the GI tract. They can be present due to a number of factors including: consumption of contaminated water or food, exposure to chemicals that are toxic to beneficial bacteria; the use of antibiotics, oral contraceptives or other medications; poor fiber intake and high stress levels.

**YEAST CULTURE**

| Normal flora | Dysbiotic flora |
|---|---|
| 1+ Candida parapsilosis | |

**MICROSCOPIC YEAST**

| Result: | Expected: |
|---|---|
| None | None - Rare |

The microscopic finding of yeast in the stool is helpful in identifying whether there is proliferation of yeast. Rare yeast may be normal; however, yeast observed in higher amounts (few, moderate, or many) is abnormal.

**YEAST INFORMATION**

**Yeast** normally can be found in small quantities in the skin, mouth, intestine and mucocutaneous junctions. Overgrowth of yeast can infect virtually every organ system, leading to an extensive array of clinical manifestations. Fungal diarrhea is associated with broad-spectrum antibiotics or alterations of the patient's immune status. Symptoms may include abdominal pain, cramping and irritation. When investigating the presence of yeast, disparity may exist between culturing and microscopic examination. Yeast are not uniformly dispersed throughout the stool, this may lead to undetectable or low levels of yeast identified by microscopy, despite a cultured amount of yeast. Conversely, microscopic examination may reveal a significant amount of yeast present, but no yeast cultured. Yeast does not always survive transit through the intestines rendering it unviable.

*Comprehensive Stool Analysis / Parasitology x3*

**PARASITOLOGY/MICROSCOPY ***

**Sample 1**
None Ova or Parasites

**Sample 2**
None Ova or Parasites

**Sample 3**
None Ova or Parasites

***A trichrome stain and concentrated iodine wet mount slide is read for each sample submitted.**

**PARASITOLOGY INFORMATION**

Intestinal parasites are abnormal inhabitants of the gastrointestinal tract that have the potential to cause damage to their host. The presence of any parasite within the intestine generally confirms that the patient has acquired the organism through fecal-oral contamination. Damage to the host includes parasitic burden, migration, blockage and pressure. Immunologic inflammation, hypersensitivity reactions and cytotoxicity also play a large role in the morbidity of these diseases. The infective dose often relates to severity of the disease and repeat encounters can be additive.

There are two main classes of intestinal parasites, they include protozoa and helminths. The protozoa typically have two stages; the trophozoite stage that is the metabolically active, invasive stage and the cyst stage, which is the vegetative inactive form resistant to unfavorable environmental conditions outside the human host. Helminths are large, multicellular organisms. Like protozoa, helminths can be either free-living or parasitic in nature. In their adult form, helminths cannot multiply in humans.

In general, acute manifestations of parasitic infection may involve diarrhea with or without mucus and or blood, fever, nausea, or abdominal pain. However these symptoms do not always occur. Consequently, parasitic infections may not be diagnosed or eradicated. If left untreated, chronic parasitic infections can cause damage to the intestinal lining and can be an unsuspected cause of illness and fatigue. Chronic parasitic infections can also be associated with increased intestinal permeability, irritable bowel syndrome, irregular bowel movements, malabsorption, gastritis or indigestion, skin disorders, joint pain, allergic reactions, and decreased immune function.

In some instances, parasites may enter the circulation and travel to various organs causing severe organ diseases such as liver abscesses and cysticercosis. In addition, some larval migration can cause pneumonia and in rare cases hyper infection syndrome with large numbers of larvae being produced and found in every tissue of the body.

One negative parasitology x1 specimen does not rule out the possibility of parasitic disease, parasitology x3 is recommended. This exam is not designed to detect Cryptosporidium spp, Cyclospora cayetanensis or Microsproridia spp.

**GIARDIA/CRYPTOSPORIDIUM IMMUNOASSAY**

| | Within | Outside | Reference Range |
|---|---|---|---|
| Giardia intestinalis | Neg | | Neg |
| Cryptosporidium | Neg | | Neg |

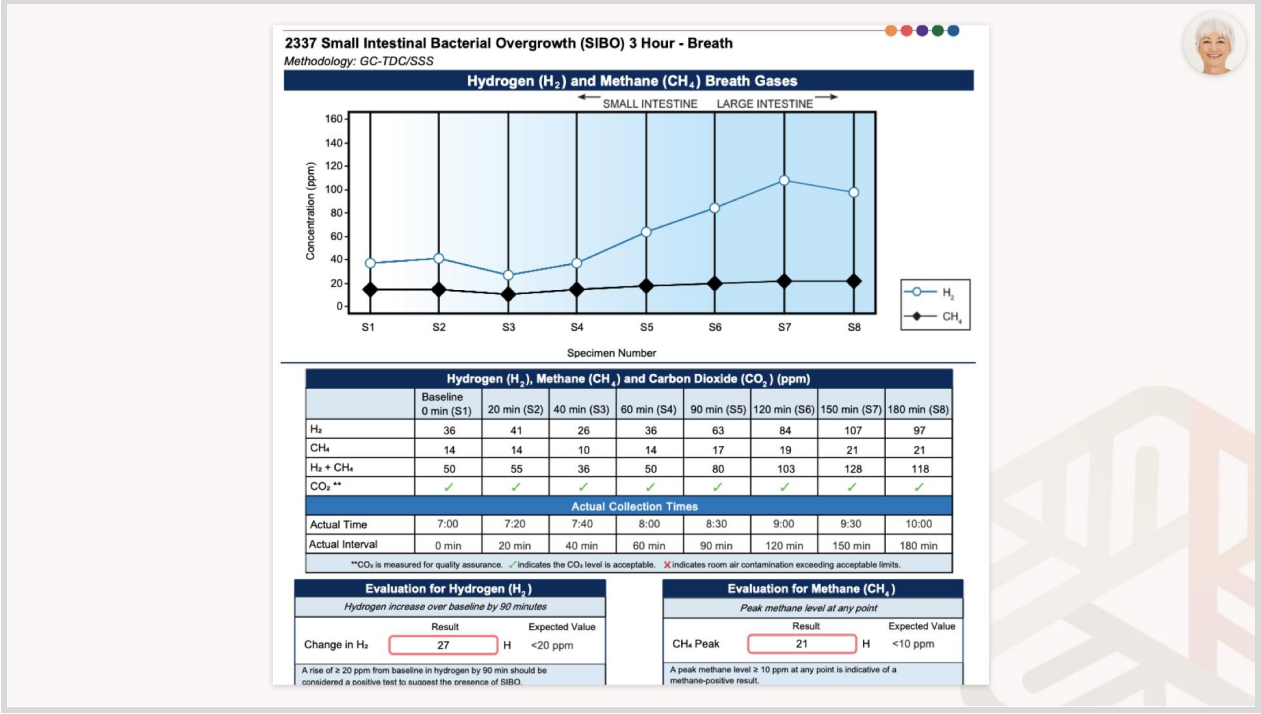
**Giardia intestinalis** (lamblia) is a protozoan that infects the small intestine and is passed in stool and spread by the fecal-oral route. Waterborne transmission is the major source of giardiasis.
**Cryptosporidium** is a coccidian protozoa that can be spread from direct person-to-person contact or waterborne transmission.

Follow-up on the Doctor's Data panel showed significant improvement in beneficial bacteria. That's because, as you saw and as you know from the treatment protocols, part of the botanical antimicrobial protocol involves probiotics, so that can help. The fungal overgrowth, the microscopic exam went down from few to none. There is a 1+ for *Candida* still showing, but that could just be normal flora and no parasites showing on the Doctor's Data parasitology section. So, in this case, unfortunately, the patient's cholesterol didn't come down after addressing the gut and some other issues. His cholesterol was still very high, over 300 for total cholesterol. When you see it that high—and [it] often doesn't respond to addressing underlying problems like nutritional deficiencies, imbalanced diet, [and] gut infections—it's most likely genetic in origin. So probably, this patient did have some familial hypercholesterolemia. On the other hand, his physical and mental performance did improve. He lost some weight and felt better overall. So I think the treatment was still successful from that perspective.

## CASE #4: 62-YEAR-OLD FEMALE

All right, [the] next patient is a 62-year-old female. Her chief complaints were gastrointestinal symptoms, bloating, gas, abdominal pain, cognitive impairment, fatigue, hair loss, joint pain, and anxiety. She had a history of extensive antibiotic use as a child and adult. She had tried a number of diets, including [gut and psychology syndrome] (GAPS), that did not work, and raw

vegan, which helped only for a short period of time, but then symptoms returned and worsened. She was diagnosed with gallstones, had her gallbladder removed, but [it] did not make any difference in her symptoms. So she came to us on a low-FODMAP [(fermentable oligosaccharides, disaccharides, monosaccharides, and polyols)] diet and a history of antacid and [proton pump inhibitor] use that didn't seem to really make a difference in symptoms. She also noted that her symptoms worsened after a food poisoning incident in her 40s and feels that she got sick often when traveling.



**2337 Small Intestinal Bacterial Overgrowth (SIBO) 3 Hour - Breath**

*Methodology: GC-TDC/SSS*

**Hydrogen ($H_2$) and Methane ($CH_4$) Breath Gases**



**Hydrogen ($H_2$), Methane ($CH_4$) and Carbon Dioxide ($CO_2$) (ppm)**

| | Baseline 0 min (S1) | 20 min (S2) | 40 min (S3) | 60 min (S4) | 90 min (S5) | 120 min (S6) | 150 min (S7) | 180 min (S8) |
|---|---|---|---|---|---|---|---|---|
| $H_2$ | 36 | 41 | 26 | 36 | 63 | 84 | 107 | 97 |
| $CH_4$ | 14 | 14 | 10 | 14 | 17 | 19 | 21 | 21 |
| $H_2 + CH_4$ | 50 | 55 | 36 | 50 | 80 | 103 | 128 | 118 |
| $CO_2$ ** | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ | ✓ |
| **Actual Collection Times** | | | | | | | | |
| Actual Time | 7:00 | 7:20 | 7:40 | 8:00 | 8:30 | 9:00 | 9:30 | 10:00 |
| Actual Interval | 0 min | 20 min | 40 min | 60 min | 90 min | 120 min | 150 min | 180 min |

**$CO_2$ is measured for quality assurance. ✓ indicates the $CO_2$ level is acceptable. X indicates room air contamination exceeding acceptable limits.

**Evaluation for Hydrogen ($H_2$)**

*Hydrogen increase over baseline by 90 minutes*

| | Result | | Expected Value |
|---|---|---|---|
| Change in $H_2$ | 27 | H | <20 ppm |

A rise of ≥ 20 ppm from baseline in hydrogen by 90 min should be considered a positive test to suggest the presence of SIBO.

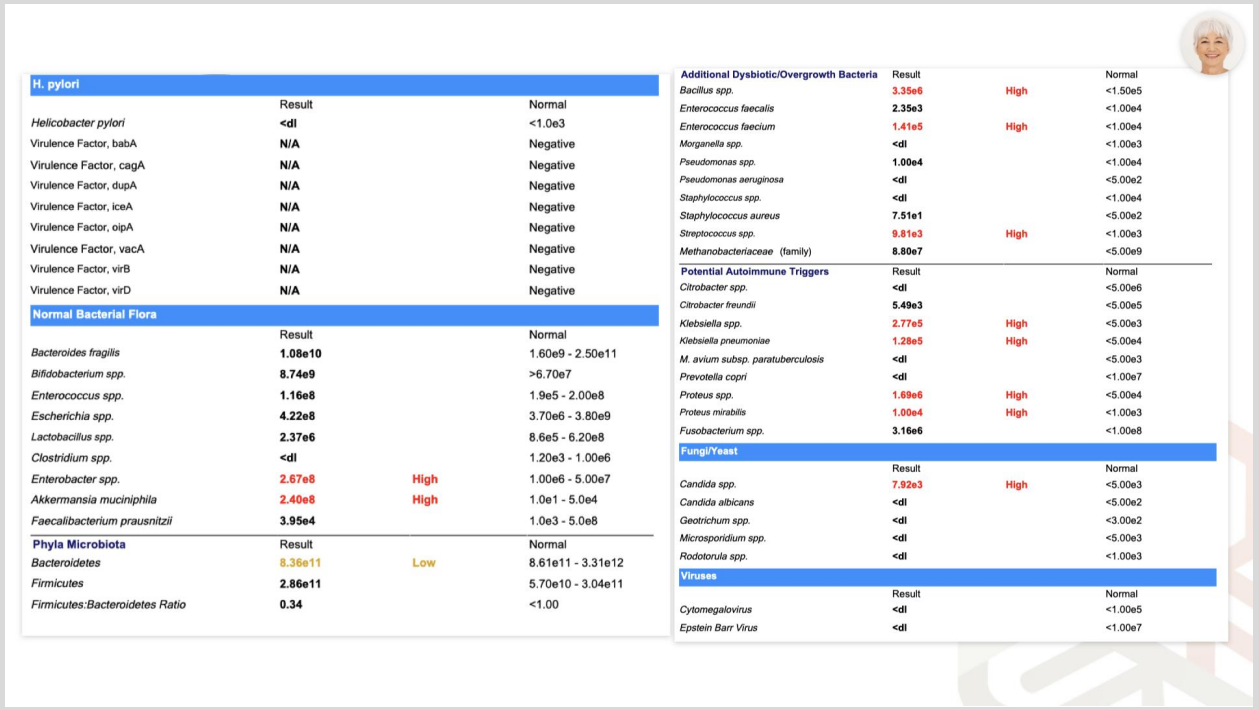
**Evaluation for Methane ($CH_4$)**

*Peak methane level at any point*

| | Result | | Expected Value |
|---|---|---|---|
| $CH_4$ Peak | 21 | H | <10 ppm |

A peak methane level ≥ 10 ppm at any point is indicative of a methane-positive result.

Here's the Genova three-hour breath test with a positive hydrogen and methane result. You'll notice that she had high baseline hydrogen on this test, so it's possible she didn't [prepare] for the test well enough. But ultimately, based [on] her symptoms and the methane results, we moved forward with considering this a positive test result and not having her repeat the test. We ran a Diagnostic Solutions Lab[oratory] GI-MAP for her, and there were no major infectious pathogens identified on the first page of the report. I didn't show that here just because the report is so long.



| H. pylori | Result | | Normal |
|---|---|---|---|
| *Helicobacter pylori* | **<dl** | | <1.0e3 |
| Virulence Factor, babA | **N/A** | | Negative |
| Virulence Factor, cagA | **N/A** | | Negative |
| Virulence Factor, dupA | **N/A** | | Negative |
| Virulence Factor, iceA | **N/A** | | Negative |
| Virulence Factor, oipA | **N/A** | | Negative |
| Virulence Factor, vacA | **N/A** | | Negative |
| Virulence Factor, virB | **N/A** | | Negative |
| Virulence Factor, virD | **N/A** | | Negative |

| Normal Bacterial Flora | Result | | Normal |
|---|---|---|---|
| *Bacteroides fragilis* | **1.08e10** | | 1.60e9 - 2.50e11 |
| *Bifidobacterium spp.* | **8.74e9** | | >6.70e7 |
| *Enterococcus spp.* | **1.16e8** | | 1.9e5 - 2.00e8 |
| *Escherichia spp.* | **4.22e8** | | 3.70e6 - 3.80e9 |
| *Lactobacillus spp.* | **2.37e6** | | 8.6e5 - 6.20e8 |
| *Clostridium spp.* | **<dl** | | 1.20e3 - 1.00e6 |
| *Enterobacter spp.* | **2.67e8** | **High** | 1.00e6 - 5.00e7 |
| *Akkermansia muciniphila* | **2.40e8** | **High** | 1.0e1 - 5.0e4 |
| *Faecalibacterium prausnitzii* | **3.95e4** | | 1.0e3 - 5.0e8 |

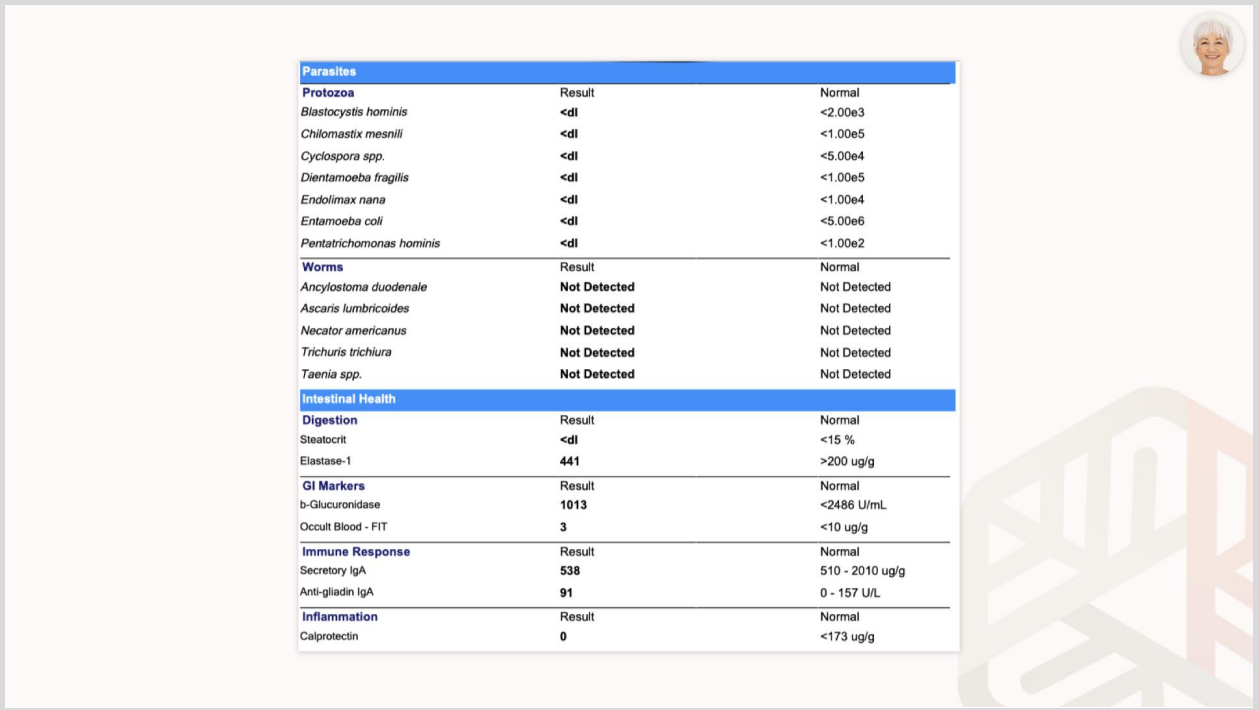
| Phyla Microbiota | Result | | Normal |
|---|---|---|---|
| *Bacteroidetes* | **8.36e11** | **Low** | 8.61e11 - 3.31e12 |
| *Firmicutes* | **2.86e11** | | 5.70e10 - 3.04e11 |
| *Firmicutes:Bacteroidetes Ratio* | **0.34** | | <1.00 |

| Additional Dysbiotic/Overgrowth Bacteria | Result | | Normal |
|---|---|---|---|
| *Bacillus spp.* | **3.35e6** | **High** | <1.50e5 |
| *Enterococcus faecalis* | **2.35e3** | | <1.00e4 |
| *Enterococcus faecium* | **1.41e5** | **High** | <1.00e4 |
| *Morganella spp.* | **<dl** | | <1.00e3 |
| *Pseudomonas spp.* | **1.00e4** | | <1.00e4 |
| *Pseudomonas aeruginosa* | **<dl** | | <5.00e2 |
| *Staphylococcus spp.* | **<dl** | | <1.00e4 |
| *Staphylococcus aureus* | **7.51e1** | | <5.00e2 |
| *Streptococcus spp.* | **9.81e3** | **High** | <1.00e3 |
| *Methanobacteriaceae* (family) | **8.80e7** | | <5.00e9 |

| Potential Autoimmune Triggers | Result | | Normal |
|---|---|---|---|
| *Citrobacter spp.* | **<dl** | | <5.00e6 |
| *Citrobacter freundii* | **5.49e3** | | <5.00e5 |
| *Klebsiella spp.* | **2.77e5** | **High** | <5.00e3 |
| *Klebsiella pneumoniae* | **1.28e5** | **High** | <5.00e4 |
| *M. avium subsp. paratuberculosis* | **<dl** | | <5.00e3 |
| *Prevotella copri* | **<dl** | | <1.00e7 |
| *Proteus spp.* | **1.69e6** | **High** | <5.00e4 |
| *Proteus mirabilis* | **1.00e4** | **High** | <1.00e3 |
| *Fusobacterium spp.* | **3.16e6** | | <1.00e8 |

| Fungi/Yeast | Result | | Normal |
|---|---|---|---|
| *Candida spp.* | **7.92e3** | **High** | <5.00e3 |
| *Candida albicans* | **<dl** | | <5.00e2 |
| *Geotrichum spp.* | **<dl** | | <3.00e2 |
| *Microsporidium spp.* | **<dl** | | <5.00e3 |
| *Rodotorula spp.* | **<dl** | | <1.00e3 |

| Viruses | Result | Normal |
|---|---|---|
| *Cytomegalovirus* | **<dl** | <1.00e5 |
| *Epstein Barr Virus* | **<dl** | <1.00e7 |

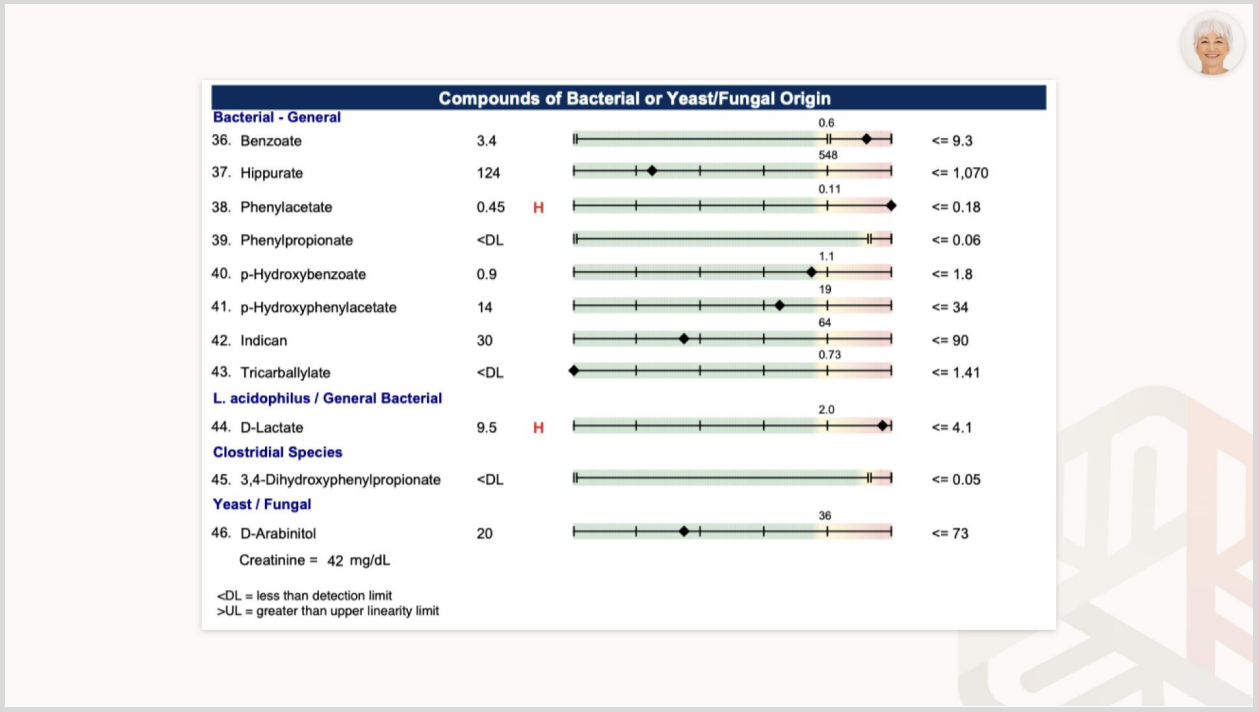


| Parasites | | |
|---|---|---|
| **Protozoa** | Result | Normal |
| *Blastocystis hominis* | **<dl** | <2.00e3 |
| *Chilomastix mesnili* | **<dl** | <1.00e5 |
| *Cyclospora spp.* | **<dl** | <5.00e4 |
| *Dientamoeba fragilis* | **<dl** | <1.00e5 |
| *Endolimax nana* | **<dl** | <1.00e4 |
| *Entamoeba coli* | **<dl** | <5.00e6 |
| *Pentatrichomonas hominis* | **<dl** | <1.00e2 |
| **Worms** | Result | Normal |
| *Ancylostoma duodenale* | **Not Detected** | Not Detected |
| *Ascaris lumbricoides* | **Not Detected** | Not Detected |
| *Necator americanus* | **Not Detected** | Not Detected |
| *Trichuris trichiura* | **Not Detected** | Not Detected |
| *Taenia spp.* | **Not Detected** | Not Detected |

| Intestinal Health | | |
|---|---|---|
| **Digestion** | Result | Normal |
| Steatocrit | **<dl** | <15 % |
| Elastase-1 | **441** | >200 ug/g |
| **GI Markers** | Result | Normal |
| b-Glucuronidase | **1013** | <2486 U/mL |
| Occult Blood - FIT | **3** | <10 ug/g |
| **Immune Response** | Result | Normal |
| Secretory IgA | **538** | 510 - 2010 ug/g |
| Anti-gliadin IgA | **91** | 0 - 157 U/L |
| **Inflammation** | Result | Normal |
| Calprotectin | **0** | <173 ug/g |

The *Helicobacter pylori* was negative, and overall, her beneficial bacteria flora was adequate. And I wasn't overly worried about the slightly elevated levels of *Enterobacter* and *Akkermansia* on their own. She did have some high levels of dysbiotic flora and potential autoimmune triggers. [inaudible] gets my attention just a little bit here, considering its connection to

autoimmunity, but I haven't personally found the *Proteus* marker to be helpful on this test. She did have some elevated *Candida* here, also, that you can see. There weren't any parasites or worms, and her markers of intestinal health are pretty good, considering her symptoms.



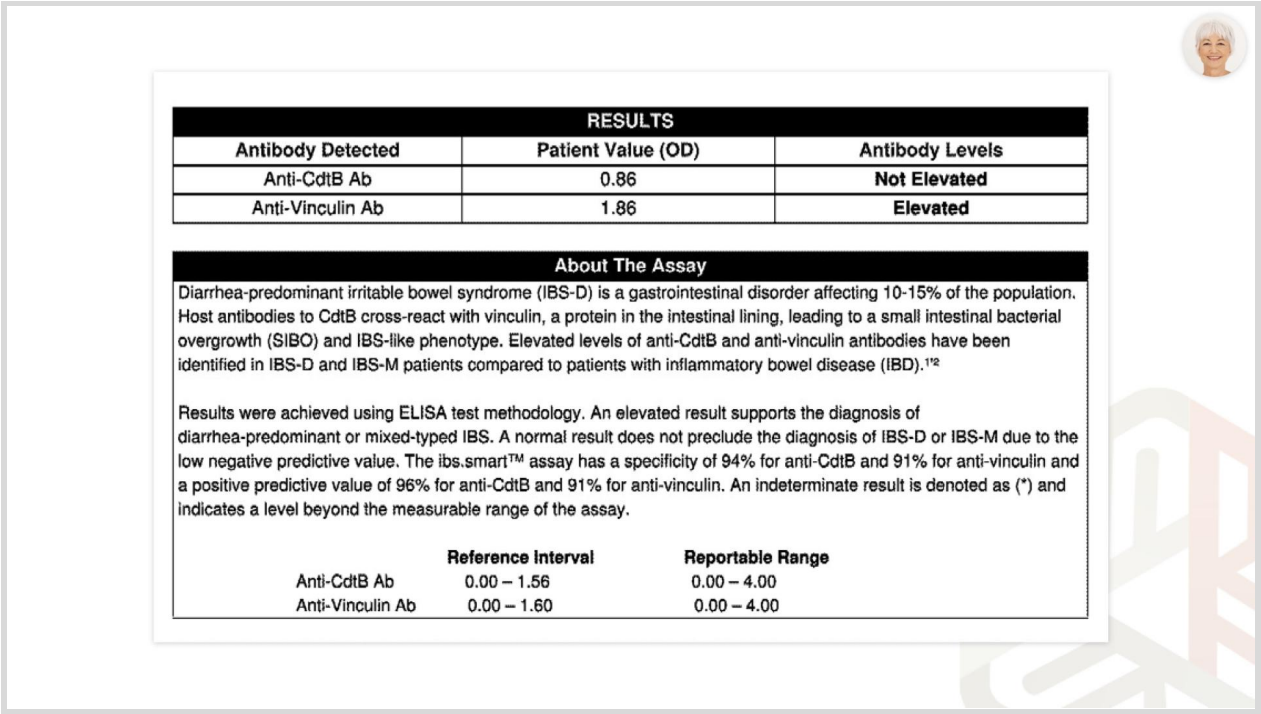
**Compounds of Bacterial or Yeast/Fungal Origin**

| | | | | |
|---|---|---|---|---|
| **Bacterial - General** | | | | |
| 36. Benzoate | 3.4 | | 0.6 | <= 9.3 |
| 37. Hippurate | 124 | | 548 | <= 1,070 |
| 38. Phenylacetate | 0.45 | H | 0.11 | <= 0.18 |
| 39. Phenylpropionate | <DL | | | <= 0.06 |
| 40. p-Hydroxybenzoate | 0.9 | | 1.1 | <= 1.8 |
| 41. p-Hydroxyphenylacetate | 14 | | 19 | <= 34 |
| 42. Indican | 30 | | 64 | <= 90 |
| 43. Tricarballylate | <DL | | 0.73 | <= 1.41 |
| **L. acidophilus / General Bacterial** | | | | |
| 44. D-Lactate | 9.5 | H | 2.0 | <= 4.1 |
| **Clostridial Species** | | | | |
| 45. 3,4-Dihydroxyphenylpropionate | <DL | | | <= 0.05 |
| **Yeast / Fungal** | | | | |
| 46. D-Arabinitol | 20 | | 36 | <= 73 |

Creatinine = 42 mg/dL

<DL = less than detection limit
>UL = greater than upper linearity limit

Her organic acids test showed high levels of phenylacetate and dilactate. The phenylacetate levels here are not astronomically high, which you might see in phenylketonuria (PKU). A reminder, if you see very high levels of PKU in a young child, especially if they have cognitive-behavioral neurological problems, I would suggest referring out to a specialist [in] inherited diseases just to confirm. And mild levels like this are more indicative of dysbiosis often and a high dilactate, another potential marker of dysbiosis, and increased metabolic activity of certain bacterial species like *Lactobacillus acidophilus*. Studies show that symptoms that are associated with elevated dilactate include GI distress but also can include neurological and cognitive symptoms because dilactate is a neurotoxin.



| RESULTS | | |
|---|---|---|
| **Antibody Detected** | **Patient Value (OD)** | **Antibody Levels** |
| Anti-CdtB Ab | 0.86 | **Not Elevated** |
| Anti-Vinculin Ab | 1.86 | **Elevated** |

**About The Assay**

Diarrhea-predominant irritable bowel syndrome (IBS-D) is a gastrointestinal disorder affecting 10-15% of the population. Host antibodies to CdtB cross-react with vinculin, a protein in the intestinal lining, leading to a small intestinal bacterial overgrowth (SIBO) and IBS-like phenotype. Elevated levels of anti-CdtB and anti-vinculin antibodies have been identified in IBS-D and IBS-M patients compared to patients with inflammatory bowel disease (IBD).[1][2]

Results were achieved using ELISA test methodology. An elevated result supports the diagnosis of diarrhea-predominant or mixed-typed IBS. A normal result does not preclude the diagnosis of IBS-D or IBS-M due to the low negative predictive value. The ibs.smart™ assay has a specificity of 94% for anti-CdtB and 91% for anti-vinculin and a positive predictive value of 96% for anti-CdtB and 91% for anti-vinculin. An indeterminate result is denoted as (*) and indicates a level beyond the measurable range of the assay.

| | **Reference Interval** | **Reportable Range** |
|---|---|---|
| Anti-CdtB Ab | 0.00 – 1.56 | 0.00 – 4.00 |
| Anti-Vinculin Ab | 0.00 – 1.60 | 0.00 – 4.00 |

For this patient, I did run an ibs-smart test, considering her history of what sounds like infectious gastroenteritis and persistent symptoms. You can see here that her anti-[cytolethal distending toxin B] (CdtB) was low, but her anti-vinculin was high at 1.6, indicating a post-infectious trigger for her [irritable bowel syndrome] (IBS) and SIBO.

## Diagnosis

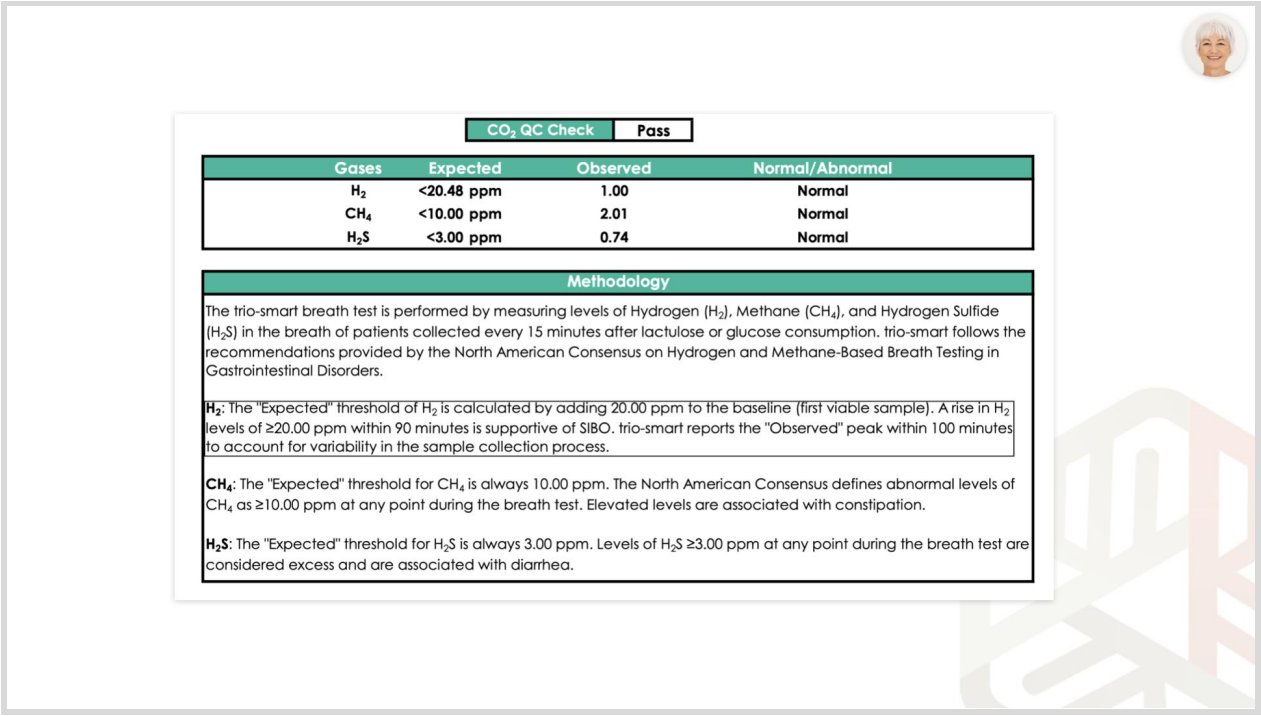
| Pattern | Supporting Markers | Comments |
|---|---|---|
| SIBO | Genova SIBO | IMO and H2 |
| Dysbiosis with fungal overgrowth | GI MAP & Organix | Klebsiella, Candida, OAT markers |
| Post-infectious IBS | IBS-Smart | Anti-vinculin 1.86 |

We have intestinal methanogen overgrowth and hydrogen SIBO indicated on the Genova SIBO test. And we also have markers of dysbiosis and post-infectious IBS.

## Treatment protocol

| Nutraceutical | Dosage |
|---|---|
| GI Synergy | 1 packet BID (with breakfast and dinner) |
| A-FNG | Slowly build to 20-30 drops BID with meals |
| Biofilm Defense | 2 capsules, BID on an empty stomach |
| Atrantil | Two capsules, TID at beginning of meals |
| MegaSporeBiotic | One capsule with lunch |
| Low Dose Erythromycin | 50 mg at night |
| MotilPro | 2 capsules, BID in between meals |

For this patient, I started with the core protocol and layered in [inaudible] for the methanogens and impaired mobility as a result of the post-infectious autoimmune process. So we have GI-Synergy and Biofilm Defense. I think I ended up using this biofilm disruptor because Interfase Plus was on backorder, and there was something going on with that patient. And a reminder that you can see the supplement substitutes and alternatives in the preferred supplement list under Tools. I added A, F, and G for the *Candida* here, as well, and I started her on low-dose erythromycin at 50 milligrams nightly. If you're unable to prescribe, then you could start with Modapro, two capsules BID in between meals. I have been known to do both of those together if the patient's symptomatic enough with dysmotility.



| $CO_2$ QC Check | Pass |
|---|---|

| Gases | Expected | Observed | Normal/Abnormal |
|---|---|---|---|
| $H_2$ | <20.48 ppm | 1.00 | Normal |
| $CH_4$ | <10.00 ppm | 2.01 | Normal |
| $H_2S$ | <3.00 ppm | 0.74 | Normal |

**Methodology**

The trio-smart breath test is performed by measuring levels of Hydrogen ($H_2$), Methane ($CH_4$), and Hydrogen Sulfide ($H_2S$) in the breath of patients collected every 15 minutes after lactulose or glucose consumption. trio-smart follows the recommendations provided by the North American Consensus on Hydrogen and Methane-Based Breath Testing in Gastrointestinal Disorders.

**$H_2$**: The "Expected" threshold of $H_2$ is calculated by adding 20.00 ppm to the baseline (first viable sample). A rise in $H_2$ levels of ≥20.00 ppm within 90 minutes is supportive of SIBO. trio-smart reports the "Observed" peak within 100 minutes to account for variability in the sample collection process.

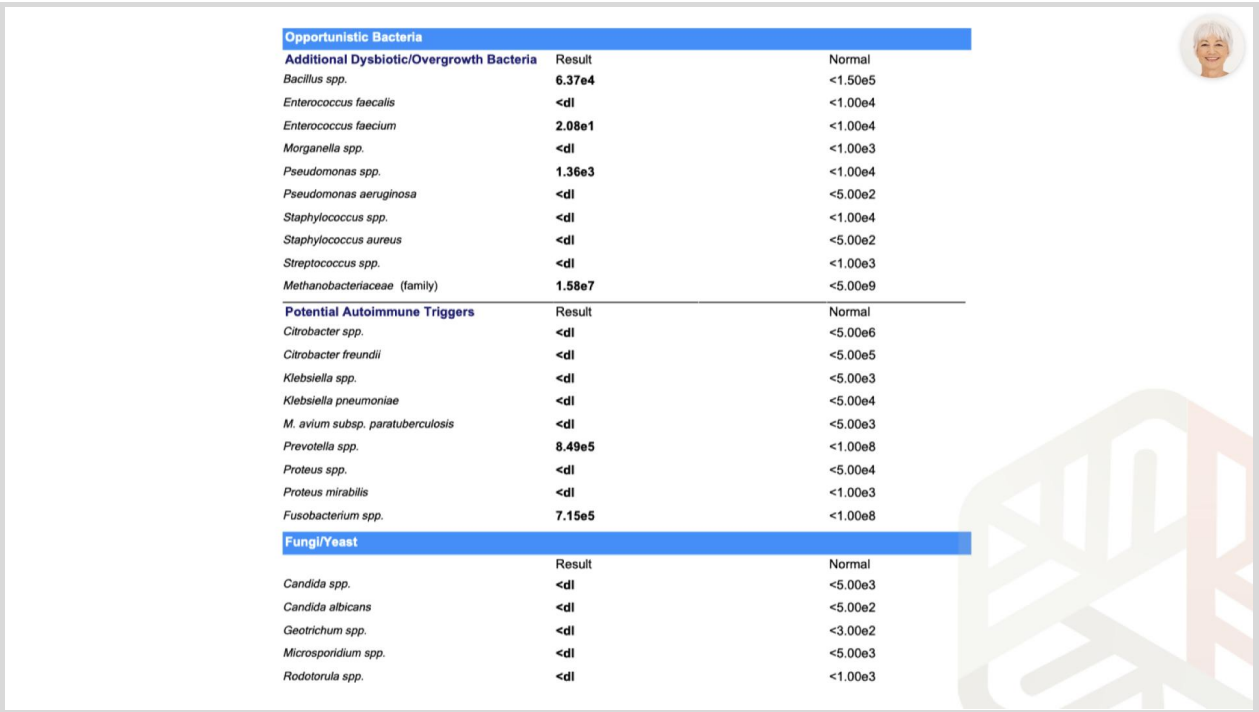
**$CH_4$**: The "Expected" threshold for $CH_4$ is always 10.00 ppm. The North American Consensus defines abnormal levels of $CH_4$ as ≥10.00 ppm at any point during the breath test. Elevated levels are associated with constipation.

**$H_2S$**: The "Expected" threshold for $H_2S$ is always 3.00 ppm. Levels of $H_2S$ ≥3.00 ppm at any point during the breath test are considered excess and are associated with diarrhea.

We ended up retesting this patient with the trio-smart breath test for follow-up, and her SIBO results were all normal after the initial antimicrobial protocol. Both methane and hydrogen [are] normalizing.

| Normal Bacterial Flora | | | |
|---|---|---|---|
| | Result | | Normal |
| *Bacteroides fragilis* | **3.54e9** | | 1.60e9 - 2.50e11 |
| *Bifidobacterium spp.* | **1.29e9** | | >6.70e7 |
| *Enterococcus spp.* | 1.58e4 | Low | 1.9e5 - 2.00e8 |
| *Escherichia spp.* | 3.13e6 | Low | 3.70e6 - 3.80e9 |
| *Lactobacillus spp.* | 7.03e5 | Low | 8.6e5 - 6.20e8 |
| *Clostridia (class)* | 2.58e6 | Low | 5.00e6 - 5.00e7 |
| *Enterobacter spp.* | **2.38e6** | | 1.00e6 - 5.00e7 |
| *Akkermansia muciniphila* | 1.74e5 | High | 1.00e1 - 5.00e4 |
| *Faecalibacterium prausnitzii* | **1.43e3** | | 1.00e3 - 5.00e8 |
| **Phyla Microbiota** | Result | | Normal |
| *Bacteroidetes* | 1.50e11 | Low | 8.61e11 - 3.31e12 |
| *Firmicutes* | 2.37e10 | Low | 5.70e10 - 3.04e11 |
| *Firmicutes:Bacteroidetes Ratio* | **0.16** | | <1.00 |

Here's her follow-up GI-MAP. You can see that we saw quite a big dip in beneficial bacteria species on the retest. That's something to pay attention to, and that's notable.



| Opportunistic Bacteria | | |
|---|---|---|
| **Additional Dysbiotic/Overgrowth Bacteria** | Result | Normal |
| *Bacillus spp.* | **6.37e4** | <1.50e5 |
| *Enterococcus faecalis* | **<dl** | <1.00e4 |
| *Enterococcus faecium* | **2.08e1** | <1.00e4 |
| *Morganella spp.* | **<dl** | <1.00e3 |
| *Pseudomonas spp.* | **1.36e3** | <1.00e4 |
| *Pseudomonas aeruginosa* | **<dl** | <5.00e2 |
| *Staphylococcus spp.* | **<dl** | <1.00e4 |
| *Staphylococcus aureus* | **<dl** | <5.00e2 |
| *Streptococcus spp.* | **<dl** | <1.00e3 |
| *Methanobacteriaceae* (family) | **1.58e7** | <5.00e9 |
| **Potential Autoimmune Triggers** | Result | Normal |
| *Citrobacter spp.* | **<dl** | <5.00e6 |
| *Citrobacter freundii* | **<dl** | <5.00e5 |
| *Klebsiella spp.* | **<dl** | <5.00e3 |
| *Klebsiella pneumoniae* | **<dl** | <5.00e4 |
| *M. avium subsp. paratuberculosis* | **<dl** | <5.00e3 |
| *Prevotella spp.* | **8.49e5** | <1.00e8 |
| *Proteus spp.* | **<dl** | <5.00e4 |
| *Proteus mirabilis* | **<dl** | <1.00e3 |
| *Fusobacterium spp.* | **7.15e5** | <1.00e8 |
| **Fungi/Yeast** | | |
| | Result | Normal |
| *Candida spp.* | **<dl** | <5.00e3 |
| *Candida albicans* | **<dl** | <5.00e2 |
| *Geotrichum spp.* | **<dl** | <3.00e2 |
| *Microsporidium spp.* | **<dl** | <5.00e3 |
| *Rodotorula spp.* | **<dl** | <1.00e3 |

But we also saw improvement in the opportunistic bacteria, *Klebsiella* and *Candida*, so there did appear to be some casualties of this protocol, but overall, the targeted organisms were eliminated. This would really be a great time to then follow up with a restoration protocol with probiotics, prebiotics, and general microbial and mucosal support, which is what we did right after we got these results back. Overall, this patient saw about 50% improvement in gastrointestinal symptoms, which are bloating, gas, and abdominal pain. She did have some improvement [in] cognitive impairment and fatigue but not as much as she likes, so we would continue moving down the process.