KRESSER INSTITUTE
FOR FUNCTIONAL AND EVOLUTIONARY MEDICINE

Thyroid Hypofunction I - Part Four

The next patient is a 40-year-old female with chief complaint of chronic inflammation, leaky gut, food sensitivities, and skin conditions.

Marker	Value	Functional Range	Lab Range
Glucose	86	75 – 90	65 - 99
Hemoglobin A1c	5.3	4.4 – 5.4	4.8 - 5.6
Uric Acid	4.5	3.2 - 5.5	2.5 - 7.1
BUN	10	13 – 18	6 - 20
Creatinine	0.88	0.85 – 1.1	0.57 - 1
Sodium	137	135 – 140	134 - 144
Potassium	4.1	4.0 – 4.5	3.5 - 5.2
Chloride	98	100 – 106	97 - 108
C02	25	25 – 30	18 - 29
Calcium	9.3	9.2 – 10.1	8.7 - 10.2
Phosphorus	3.3	3.5 – 4.0	2.5 - 4.5
Magnesium	2.0	2.0 – 2.6	1.6 - 2.6
Protein, total	6.9	6.9 – 7.4	6.0 - 8.5
Albumin	4.5	4.0 – 5.0	3.5 - 5.5
Globulin	2.4	2.4 – 2.8	1.5 - 4.5
A/G ratio	1.9	1.5 – 2.0	1.1 - 2.5
Bilirubin, total	1.0	0.1 – 1.2	0.0 - 1.2
Alkaline Phosphatase	65	42 – 107	39 - 117
LDH	149	140 - 180	119 - 226
AST	19	10 - 30	0 - 40
ALT	20	10 - 22	0 - 32
GGT	13	0 - 28	0 - 60
TIBC	296	250 – 350	250 - 450
UIBC	123	150 - 375	150 - 375
Iron	173	85 – 135	35 - 155
Iron saturation	58	15 – 45	15 - 55
Ferritin	33	15 - 120	15 - 150
Cholesterol, total	160	150 – 250	100 - 199
Triglycerides	67	50 – 100	0 - 149
HDL	68	55 – 85	> 39
LDL	79	0 – 175	0 - 99
T. Chol / HDL Ratio	2.4	< 3	0 - 4.4
Triglycerides / HDL Ratio	0.99	< 2	< 3.8
TSH	23.770	0.5 – 2.5	0.45 - 4.50
T4, total	8.9	6.0 – 12	4.5 - 12.0
T3 Uptake	30	28 - 35	24 - 39
T3, Total	77	100 – 180	71 - 180
Vitamin D, 25-hydroxy	28.6	35 - 60	30.0 - 100.0

Marker	Value	Functional Range	Lab Range
WBC	4.5	5.0 – 8.0	3.4 - 10.8
RBC	4.57	4.4 – 4.9	3.77 - 5.28
Hemoglobin	13.8	13.5 - 14.5	11.1 - 15.9
Hematocrit	40.7	37 - 44	34.0 - 46.6
MCV	89	85 – 92	79 - 97
MCH	30.2	27.7 – 32.0	26.6 - 33.0
MCHC	33.9	32 – 35	31.5 - 35.7
RDW	12.8	11.5 – 15.0	12.3 - 15.4
Platelets	203	150 – 415	150 - 379
Neutrophils	50	40 – 60	
Lymphocytes	40	25 – 40	
Monocytes	9	4.0 – 7.0	
Eosinophils	1	0.0 – 3.0	
Basophils	0	0.0 – 3.0	

Additional Tests:			
T3, Free	2.3	2.5 - 4.0	2 - 4.4
T4, Free	1.28	1 - 1.5	0.82 - 1.77
CRP-hs	0.5	< 1.0	0.00 - 3.00
Homocysteine	7.4	< 9.0	0.0 - 15.0
Vitamin B-12	688	450 – 2000	211 - 946
Copper	110		72 - 166
Zinc	116		56 - 134
Zinc / Copper Ratio	1.05	> 0.85	
Serum Methylmalonic Acid (MMA)	167	0 - 325	0 - 378

As you can see, her TSH is almost 24, but her T4, both total and free, and T3, both total and free, are within normal lab limits. Her total T3 and free T3 are low in the functional range, however. Again, this is technically subclinical hypothyroidism.

Up to 80 percent of patients who are hypothyroid have normal T4 and T3 in the reference range at least, so this could be an argument for tightening the T4 and T3 reference range. TSH is considered the most sensitive marker of thyroid function, which is why some clinicians, especially in the conventional model, only use TSH for diagnosis and treatment.

Note that this patient's iron levels are also high. Both iron deficiency and overload can adversely affect the HPA axis and contribute to autoimmune thyroid disease and hypothyroidism.

Finally, this case illustrates another common finding, especially in Hashimoto's patients, which is low T4-to-T3 conversion, so she has normal T4 levels and normal free T4 levels, but her total T3 and free T3 are borderline low.

I wrote an entire series on low T3 syndrome, and I'll link to the articles in the resource section, and I would again suggest that you read it for this unit. I'm just going to give you a quick overview here.

The thyroid gland produces thyroxine and triiodothyronine, T4 and T3, which are the primary circulating hormones. The thyroid produces T4 in significantly greater quantities to T3. It's a ratio of 17:1. T3 is approximately five times more biologically active than T4. T4 is converted into the more active T3 by the deiodinase system: D1, D2, and D3 in multiple tissues and organs, but especially in the liver, gut, skeletal muscle, brain, and the thyroid gland itself. The deiodinase D3 converts T3 into an inactive form of thyroid hormone called reverse T3 in the liver. One of the primary causes of low T4-to-T3 conversion is inflammation. For example, interleukin-6 levels are positively correlated with reverse T3, which is the inactive form of T3, and inversely correlated with free T3. Hashimoto's is an inflammatory condition, which is why many patients with Hashimoto's don't convert T4 to T3 well and why monotherapy with levothyroxine is often not effective.

Reverse T3: not a marker of thyroid function

Reverse T3 is another marker on the full thyroid panel. Again, it's the inactive form of T3. In emotional, psychological, or physiological stress, the body will convert excess T4 to reverse T3 as a means of conserving energy for healing and repair. From this perspective, replacing thyroid hormone in these cases may not be beneficial and may even be harmful. On the other hand, in those suffering from long-term chronic illness, high reverse T3 or high reverse T3-to-free T3 ratio may be more reflective of pathology than adaptation, and that group may benefit from T3 supplementation.

The studies on this are mixed. Some show harm. Others show no change, and others show a benefit. The T3 replacement has been shown to be consistently beneficial only in cardiac patients who have recently had surgery, heart failure, or transplant.

TSH					
TSH	**0.017**	**Low**	uIU/mL	0.450 – 4.500	02
DHEA-Sulfate, Serum					
DHEA-Sulfate, LCMS	99		ug/dL		01
Reference Range:					
Adult Females (41 – 50y): <229					
Thyroxine (T4) Free, Direct, S					
T4,Free(Direct)	**1.84**	**High**	ng/dL	0.82 – 1.77	02
Reverse T3, Serum	**32.2**	**High**	ng/dL	9.2 – 24.1	03
EBV Ab VCA, IgG	**183.0**	**High**	U/mL	0.0 – 17.9	02
			Negative	<18.0	
			Equivocal	18.0 – 21.9	
			Positive	>21.9	
Vitamin B12	664		pg/mL	211 – 946	02
Ferritin, Serum	45		ng/mL	15 – 150	02
Triiodothyronine,Free,Serum	4.0		pg/mL	2.0 – 4.4	02

This patient is a 39-year-old female. She has Hashimoto's. She also has reactivated Epstein-Barr and several significant markers of inflammation. She was taking a high dose of levothyroxine, which is common in these patients. They don't convert T4 to T3 well because of inflammation, so the doctor just prescribes higher and higher doses of levothyroxine, and this can lead to facetious hyperthyroidism. Her TSH is effectively zero, and she has high free T4. However, note that her reverse T3 is high, which is indicative of inflammation. Even when free T3 is normal or high-normal, as it was for her, the ratio of reverse T3 to free T3 will still be elevated.

What do I mean by elevated? Well, there is no published research on this that I'm aware of. Many advocacy groups and clinicians have come up with a threshold based on clinical and anecdotal experience. If you're using free T3, the suggested cutoff is above 20 for optimal ratio, and if you're using total T3, you'd want it to be above 10 for an optimal ratio. In this case, using free T3, the ratio is 8, which is well below the threshold of 20.

I personally don't put a lot of stock in these ratios. I just use them loosely as indicators of psychological, emotional, or physiological stress, in other words, allostatic load. Remember, a depressed reversed T3-to-free T3 ratio doesn't indicate thyroid dysfunction, per se, and doesn't always or even typically signal a need for thyroid hormone replacement. Even with this high dose of T4, her TSH is almost zero, and she has a high, high-normal free T4 and free T3, and the patient was still symptomatic. This is a great example of why the underlying condition needs to be addressed, and T4 monotherapy as the only treatment for Hashimoto's is woefully inadequate.

Some clinicians and patient thyroid advocates have argued that when the ratio of RT3, or reverse T3, to free T3 is high, even when RT3 is high-normal or normal and T3 is normal or low-normal, the patient should be treated with T3 replacement, especially when the patient has symptoms consistent with hypothyroidism such as cold hands and feet, GI issues, and fatigue.

TSH+T4F+T3Free

TSH	1.670	uIU/mL	0.450 - 4.500	01
Triiodothyronine,Free,Serum	3.1	pg/mL		01

No patient age and/or gender provided

Age	Male	Female
0 - 3 days	2.0 - 7.9	2.0 - 7.9
4 - 30 days	2.0 - 5.2	2.0 - 5.2
31 days - 11 months	1.6 - 6.4	1.6 - 6.4
1 - 5 years	2.0 - 6.0	2.0 - 6.0
6 - 10 years	2.7 - 5.2	2.7 - 5.2
11 - 19 years	2.3 - 5.0	2.3 - 5.0
>19 years	2.0 - 4.4	2.0 - 4.4

T4,Free(Direct)	1.41	ng/dL		01

No patient age and/or gender provided

Age	Male	Female
0 - 3 days	.66 - 2.71	.66 - 2.71
4 - 30 days	.83 - 3.09	.83 - 3.09
31 days - 11 months	.48 - 2.34	.48 - 2.34
1 - 5 years	.85 - 1.75	.85 - 1.75
6 - 10 years	.90 - 1.67	.90 - 1.67
11 - 19 years	.93 - 1.60	.93 - 1.60
>19 years	.82 - 1.77	.82 - 1.77

TESTS	RESULT	FLAG	UNITS	REFERENCE INTERVAL	LAB
Thyroxine (T4)	9.9		ug/dL	4.5 - 12.0	01
Triiodothyronine (T3)	118		ng/dL	71 - 180	01
Reverse T3, Serum	22.3		ng/dL	9.2 - 24.1	04

This is referred to as Wilson's syndrome, a condition that does not exist in the scientific literature, but it's popular in some functional and integrative medicine circles. For example, check out the labs on this slide. The TSH is 1.67, so that's normal. Free and total T4 are normal. Free and total T3 are all normal, but if you do the RT3-to-T3 ratio, you get 5.4 for total and 7.2 for free, both of which are low. This would lead to treatment with T3 of a patient with entirely normal TSH and free and total T4 and T3. I don't really agree with this approach. The symptoms are not specific enough to diagnose a thyroid problem in the absence of supportive labs, and as mentioned, there is not

enough data on RT3. The few studies I have seen suggest that RT3 does not distinguish between hypothyroidism and euthyroid sick syndrome or low T3 syndrome.

I have heard anecdotal reports of improvement from people who have taken replacement T3 hormone when their RT3-to-T3 ratio is off, even with normal thyroid labs, but I'm concerned about overriding the body's attempt to downregulate thyroid metabolism as a means of energy conservation and repair in these conditions. My approach is when there is low T4-to-T3 conversion, high reverse T3, or a high RT3-to-free T3 or -total T3 ratio, I would look at that as not a sign of a thyroid problem but a sign of inflammation, HPA axis dysfunction, or nutrient imbalance, and I would address those conditions rather than giving thyroid hormone.

Marker	Value	Functional Range	Lab Range
Glucose	97	75 - 90	65 - 99
Hemoglobin A1c	5.7	4.4 – 5.4	4.8 - 5.6
Uric Acid	5.5	3.7 – 6.0	3.7 - 8.6
BUN	12	13 – 18	6 - 24
Creatinine	1.02	0.85 – 1.1	0.76 - 1.27
Sodium	138	135 – 140	134 - 144
Potassium	4.7	4.0 – 4.5	3.5 - 5.2
Chloride	99	100 – 106	97 - 108
C02	23	25 – 30	18 - 29
Calcium	9.4	9.2 – 10.1	8.7 - 10.2
Phosphorus	4.2	3.5 – 4.0	2.5 - 4.5
Magnesium	2.0	2.0 – 2.6	1.6 - 2.6
Protein, total	7.1	6.9 – 7.4	6.0 - 8.5
Albumin	4.8	4.0 – 5.0	3.5 - 5.5
Globulin	2.3	2.4 – 2.8	1.5 - 4.5
A/G ratio	2.1	1.5 – 2.0	1.1 - 2.5
Bilirubin, total	0.4	0.1 – 1.2	0.0 - 1.2
Alkaline Phosphatase	66	42 – 107	39 - 117
LDH	93	140 - 180	121 - 224
AST	28	10 - 30	0 - 40
ALT	17	10 - 29	0 - 44
GGT	22	0 - 40	0 - 65
TIBC	364	250 – 350	250 - 450
UIBC	282	150 - 375	150 - 375
Iron	82	85 – 135	40 - 155
Iron saturation	23	15 – 45	15 - 55
Ferritin	43	30 - 150	30 - 400
Cholesterol, total	245	150 - 240	100 - 199
Triglycerides	66	50 – 100	0 - 149
HDL	108	55 – 85	> 39
LDL	124	0 – 175	0 - 99
T. Chol / HDL Ratio	2.3	< 3	0 - 5.0
Triglycerides / HDL Ratio	0.61	< 2	< 3.8
TSH	3.260	0.5 – 2.5	0.45 - 4.50
T4, total	4.3	6.0 – 12	4.5 - 12
T3 Uptake	29	30 - 38	24 - 39
T3, Total	120	100 – 180	71 - 180
Vitamin D, 25-hydroxy	56.9	35 - 60	30.0 - 100.0

Marker	Value	Functional Range	Lab Range
WBC	5.2	5.0 – 8.0	3.4 - 10.8
RBC	5.34	4.4 – 4.9	4.14 - 5.8
Hemoglobin	16.1	14 - 15	12.6 - 17.7
Hematocrit	48.4	40 - 48	37.5 - 51.0
MCV	91	85 – 92	79 - 97
MCH	30.1	27.7 – 32.0	26.6 - 33.0
MCHC	33.3	32 – 35	31.5 - 35.7
RDW	14.1	11.5 – 15.0	12.3 - 15.4
Platelets	240	150 – 415	150 - 379
Neutrophils	44	40 – 60	
Lymphocytes	38	25 – 40	
Monocytes	14	4.0 – 7.0	
Eosinophils	2	0.0 – 3.0	
Basophils	1	0.0 – 3.0	

Additional Tests:			
CRP-hs	0.43	< 1.0	0.00 - 3.00
Homocysteine	7.4	< 9.0	0.0 - 15.0
Vitamin B-12	849	450 – 2000	211 - 946
Copper	104		72 - 166
Zinc	149		56 - 134
Zinc / Copper Ratio	1.43	> 0.85	
Serum Methylmalonic Acid (MMA)	121	0 - 325	0 - 378

This patient is a 46-year-old male who developed health problems after extensive travel in China and South America, primarily digestive distress but also brain fog, poor memory, and weak immune function. His TSH was within the normal reference range, but T4 is lab low. Interestingly, T3 is normal, even in the functional range. This case illustrates an important point. Sometimes hypothyroidism is the underlying cause of the patient's symptoms, but other times, it is more of a symptom of a deeper problem, and this is especially true with subclinical hypothyroidism. In these cases, it makes more sense, in my opinion, to address the underlying pathology first and then see what happens with thyroid unless they are in really bad shape, and supporting thyroid right away is necessary. This is a functional medicine approach to hypothyroidism. We're always peeling the layers of the onion back to try to get to the underlying cause.

Comprehensive Stool Analysis / Parasitology x3

BACTERIOLOGY CULTURE

Expected/Beneficial flora	Commensal (Imbalanced) flora	Dysbiotic flora
4+ Bacteroides fragilis group	1+ Staphylococcus aureus	4+ Citrobacter farmeri
2+ Bifidobacterium spp.		4+ Enterobacter cloacae complex
3+ Escherichia coli		3+ Proteus mirabilis
2+ Lactobacillus spp.		
4+ Enterococcus spp.		
4+ 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
No yeast isolated	

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

Mod	Blastocystis hominis
Rare	Dientamoeba fragilis trophs
Rare	Endolimax nana cysts
Few	Endolimax nana trophs
Rare	WBC

Sample 2

Mod	Blastocystis hominis
Rare	Dientamoeba fragilis trophs
Few	Endolimax nana cysts
Few	Endolimax nana trophs
Few	Yeast

Sample 3

Mod	Blastocystis hominis
Rare	Dientamoeba fragilis trophs
Rare	Endolimax nana cysts
Rare	Endolimax nana trophs

***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.

Thyroid Antibodies

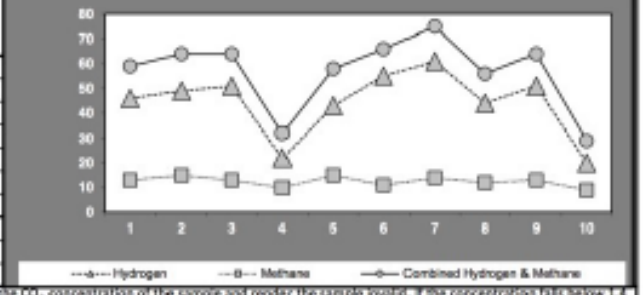
Thyroid Peroxidase (TPO) Ab	**184**	**High**	IU/mL	0 - 34	01
Thyroglobulin, Antibody	<1.0		IU/mL	0.0 - 0.9	01
Please Note:					01

Low positive Thyroglobulin antibodies are seen in a portion of the asymptomatic populations.
Antithyroglobulin antibodies measured by Beckman Coulter Methodology

Data

H_2 = Hydrogen CH_4 = Methane CO_2 = Measured

	Sample	ppm H_2	ppm CH_4	Total H2 + CH4	CO_2 %*
1	Baseline	46	13	59	OK
2	20 min	49	15	64	OK
3	40 min	51	13	64	OK
4	60 min	22	10	32	OK
5	80 min	43	15	58	OK
6	100 min	55	11	66	OK
7	120 min	61	14	75	OK
8	140 min	44	12	56	OK
9	160 min	51	13	64	OK
10	180 min	20	9	29	OK



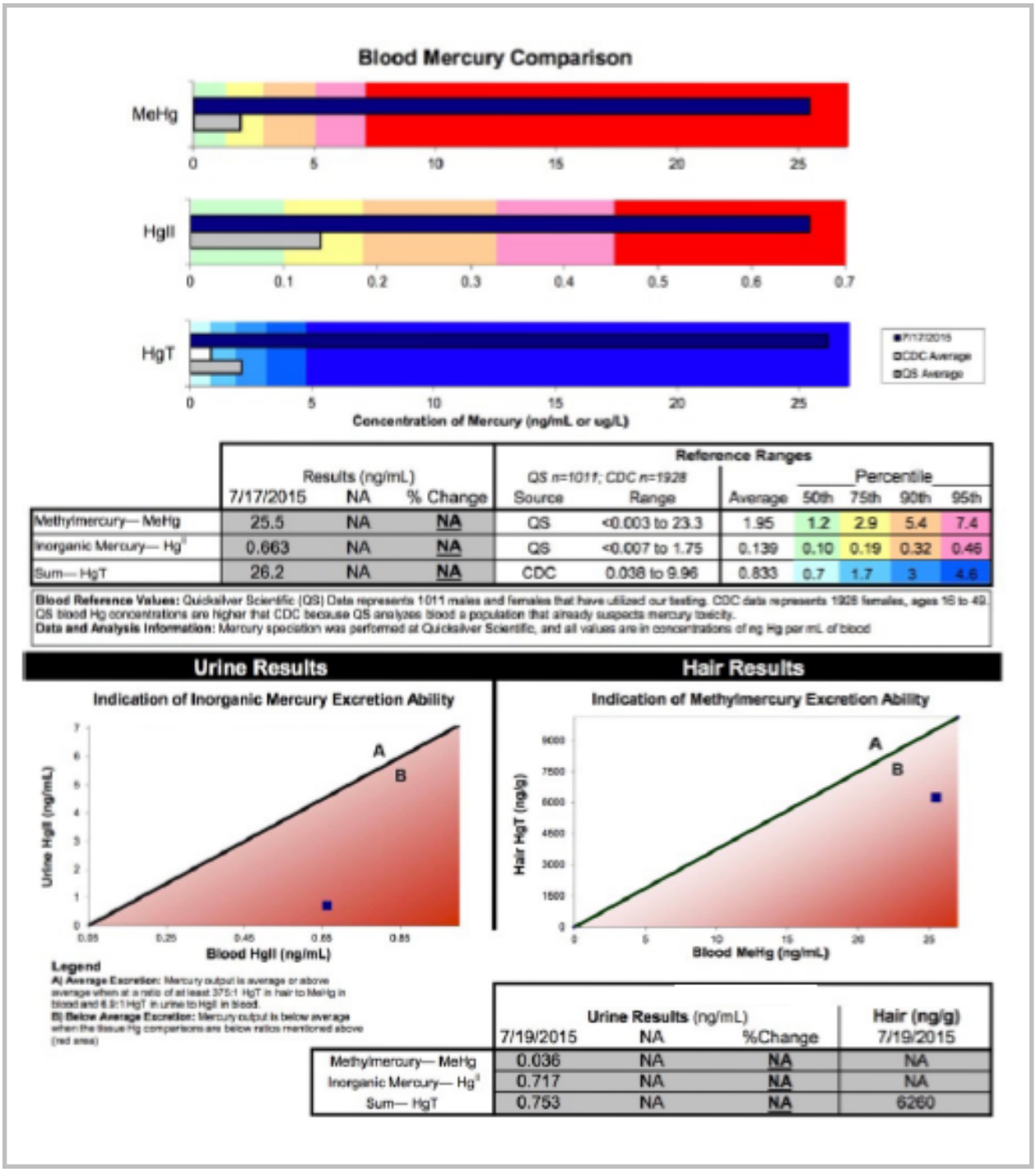
*Dilutions of the sample may occur during the sampling procedure which may decrease the CO_2 concentration of the sample and render the sample invalid. If the concentration falls below 1.4 %, the entry for CO_2 will be marked as Quantity Not Sufficient (QNS) and the entries for H_2 and CH_4 will be highlighted. If the sample is otherwise unusable the entry for CO_2 will be marked as Not Available (N/A) and the entries for H_2 and CH_4 will be highlighted. See notes section for details if cells are highlighted and blank or highlighted and contain N/A or QNS.

Analysis

Combined baseline total =	59	H	≤20ppm
Greatest H_2 increase over the lowest preceding value within first 120 minutes =	39	H	≤20ppm
Greatest CH_4 increase over the lowest preceding value within first 120 minutes =	5	-	≤12ppm
Greatest combined H_2 & CH_4 increase over the lowest preceding value within first 120 minutes =	43	H	≤15ppm

Interpretation

SIBO Suspected - Elevated Hydrogen	Increases of hydrogen greater than 20ppm over the lowest preceding value within the first 120 minutes (+/- 5min deviation) are indicative of bacterial overgrowth.	**POSITIVE**
SIBO Suspected - Elevated Methane	Increases of methane greater than 12ppm over the lowest preceding value within the first 120 minutes (+/- 5min deviation) are indicative of bacterial overgrowth.	**NEGATIVE**
SIBO Suspected - Elevated Combined Hydrogen & Methane Gasses	Increases in combined hydrogen and methane gas values greater than 15ppm over the lowest preceding value are indicative of bacterial overgrowth.	**POSITIVE**



	Results (ng/mL) 7/17/2015	NA	% Change	Reference Ranges QS n=1011; CDC n=1928 Source	Range	Average	Percentile 50th	75th	90th	95th
Methylmercury— MeHg	25.5	NA	NA	QS	<0.003 to 23.3	1.95	1.2	2.9	5.4	7.4
Inorganic Mercury— Hg^{3}	0.663	NA	NA	QS	<0.007 to 1.75	0.139	0.10	0.19	0.32	0.46
Sum— HgT	26.2	NA	NA	CDC	0.038 to 9.96	0.833	0.7	1.7	3	4.6

Blood Reference Values: Quicksilver Scientific (QS) Data represents 1011 males and females that have utilized our testing. CDC data represents 1928 females, ages 16 to 49. QS blood Hg concentrations are higher that CDC because QS analyzes blood a population that already suspects mercury toxicity.
Data and Analysis Information: Mercury speciation was performed at Quicksilver Scientific, and all values are in concentrations of ng Hg per mL of blood

Urine Results

Legend
A) Average Excretion: Mercury output is average or above average when at a ratio of at least 375:1 HgT in hair to MeHg in blood and 6.9:1 HgT in urine to HgII in blood.
B) Below Average Excretion: Mercury output is below average when the tissue Hg comparisons are below ratios mentioned above (red area)

Hair Results

	Urine Results (ng/mL) 7/19/2015	NA	%Change	Hair (ng/g) 7/19/2015
Methylmercury— MeHg	0.036	NA	NA	NA
Inorganic Mercury— Hg^{3}	0.717	NA	NA	NA
Sum— HgT	0.753	NA	NA	6260

Not surprisingly, he had significant dysbiosis, as well as Blastocystis hominis and Dientamoeba fragilis. His TPO antibodies were high at 184. He also had pretty significant SIBO and elevated mercury levels, 25.5 for methylmercury and 0.663 for inorganic mercury, for a total mercury of 26.2, which is one of the highest I've ever seen on the Quicksilver lab.

Note that some studies have shown a link between elevated mercury levels and thyroid antibody production, and removal of dental amalgams has been shown to reduce thyroid antibody levels.

TESTS	RESULT	FLAG	UNITS	REFERENCE INTERVAL	LAB
TSH+T4F+T3Free					
TSH	0.546		uIU/mL	0.450 - 4.500	01
Triiodothyronine,Free,Serum	3.3		pg/mL	2.0 - 4.4	01
T4,Free(Direct)	1.39		ng/dL	0.82 - 1.77	01

After treating for gut issues and heavy metal toxicity, we retested his thyroid panel, and as you see, the numbers completely normalized and are now in the optimal range. This doesn't always happen. Sometimes we still need to address the thyroid directly, but it happens often enough that I will typically address underlying issues in cases of subclinical hypothyroidism and low T4-to-T3 conversion first before treating the thyroid directly.