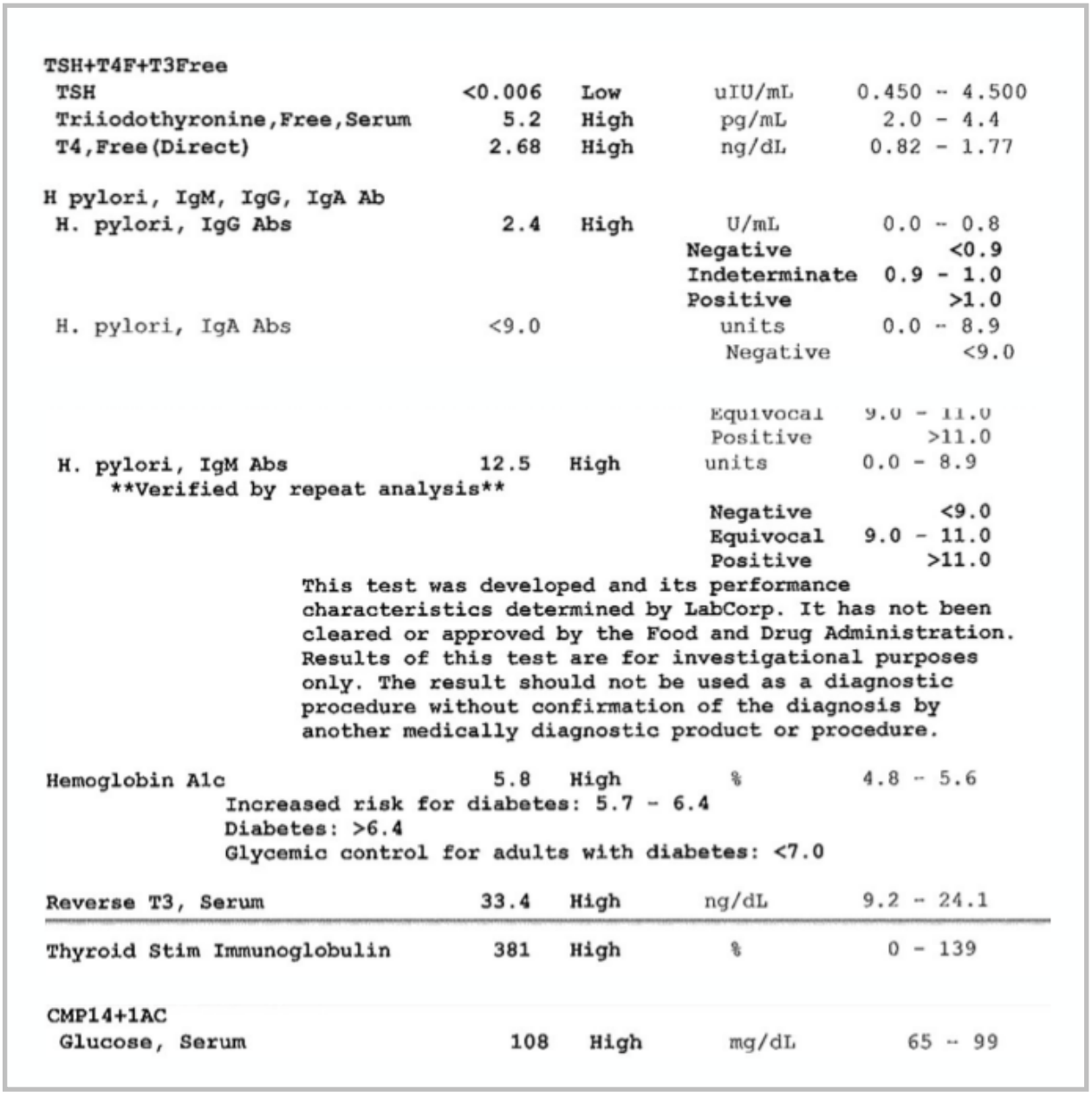
KRESSER INSTITUTE FOR FUNCTIONAL AND EVOLUTIONARY MEDICINE

Thyroid Hyperfunction - Part Two

All right, let's dive into a few cases.



TSH+T4F+T3Free				
TSH	<0.006	Low	uIU/mL	0.450 - 4.500
Triiodothyronine,Free,Serum	5.2	High	pg/mL	2.0 - 4.4
T4,Free(Direct)	2.68	High	ng/dL	0.82 - 1.77
H pylori, IgM, IgG, IgA Ab				
H. pylori, IgG Abs	2.4	High	U/mL	0.0 - 0.8
			Negative	<0.9
			Indeterminate	0.9 - 1.0
			Positive	>1.0
H. pylori, IgA Abs	<9.0		units	0.0 - 8.9
			Negative	<9.0
			Equivocal	9.0 - 11.0
			Positive	>11.0
H. pylori, IgM Abs	12.5	High	units	0.0 - 8.9
Verified by repeat analysis				
			Negative	<9.0
			Equivocal	9.0 - 11.0
			Positive	>11.0

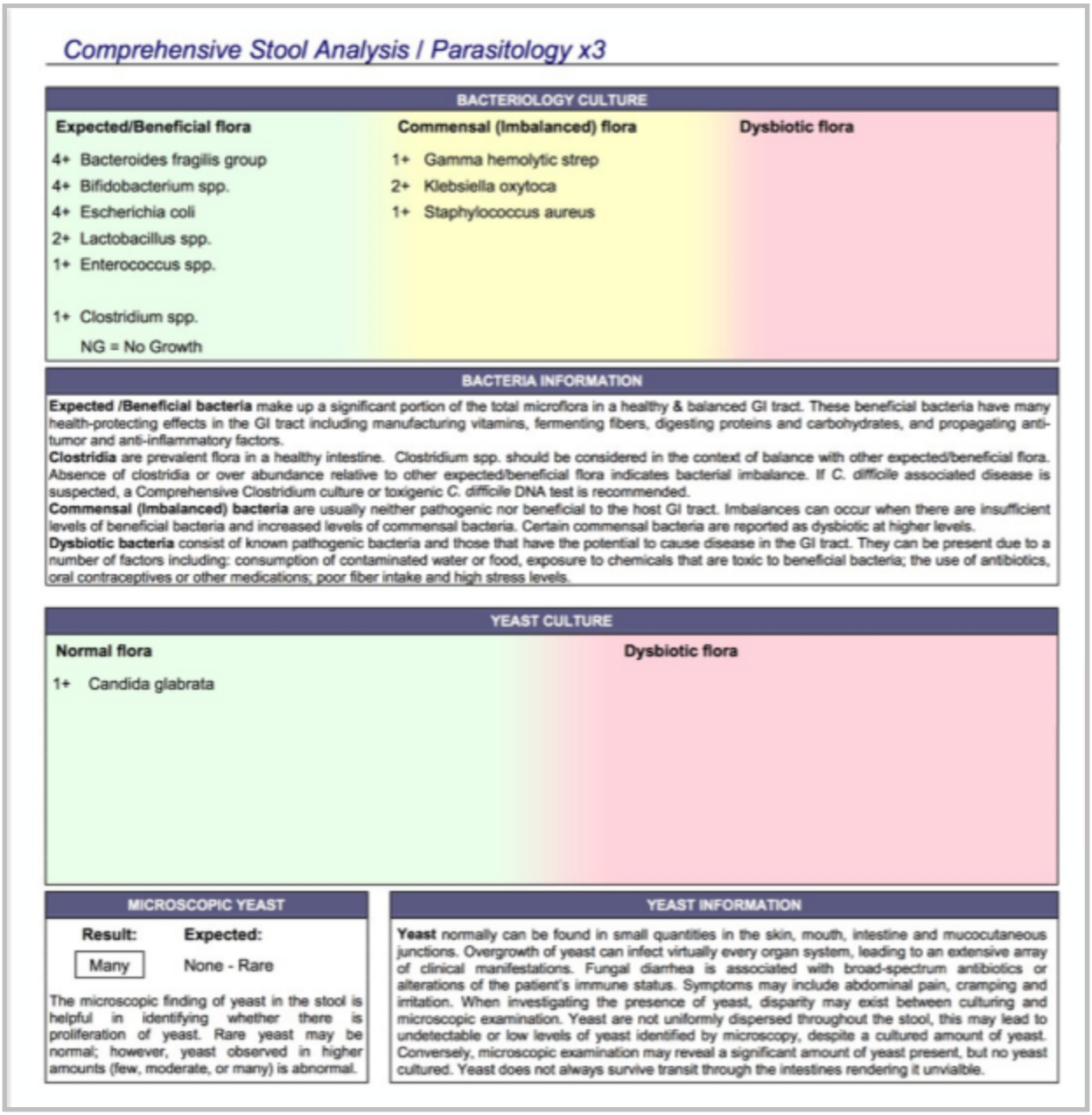
This test was developed and its performance characteristics determined by LabCorp. It has not been cleared or approved by the Food and Drug Administration. Results of this test are for investigational purposes only. The result should not be used as a diagnostic procedure without confirmation of the diagnosis by another medically diagnostic product or procedure.

Hemoglobin A1c	5.8	High	%	4.8 - 5.6
Increased risk for diabetes: 5.7 - 6.4				
Diabetes: >6.4				
Glycemic control for adults with diabetes: <7.0				
Reverse T3, Serum	33.4	High	ng/dL	9.2 - 24.1
Thyroid Stim Immunoglobulin	381	High	%	0 - 139
CMP14+1AC				
Glucose, Serum	108	High	mg/dL	65 - 99

This is a 36-year-old patient. Chief complaint was autoimmune thyroid; exophthalmos, which is a protrusion of the eyes that is common with Graves'; digestive distress with gas, bloating, and diarrhea; abdominal fat; and eczema. Notice that her TSH is essentially zero. Free T3 is 5.2, which is high. Free T4 is 2.68, which is also high. Reverse T3 is very high at 33.4, and TSI is

high at 381 percent. Also notice that she is positive for H. pylori and IgG and IgM antibodies, indicating possible current H. pylori infection. You'd want to confirm that with a stool or breath test. There is an association between H. pylori and Graves' in the scientific literature, so this is worth noting. Also note that her fasting glucose and A1c are high, and Graves' is associated with both hyperglycemia and hypoglycemia. Graves' patients are more likely to develop antibodies to insulin and a condition known as Hirata disease, which leads to a very low fasting glucose, but they are also more likely to enhance endogenous glucose production via several mechanisms, which can lead to high blood sugar, so you can see either high or low blood sugar in Graves' patients.

TEST	RESULT			
Array 3 – Wheat/Gluten Proteome Reactivity & Autoimmunity	**IN RANGE (Normal)**	**EQUIVOCAL***	**OUT OF RANGE**	**REFERENCE** (ELISA Index)
Wheat IgG	0.89			0.3-1.5
Wheat IgA	0.64			0.1-1.2
Wheat Germ Agglutinin IgG	0.81			0.4-1.3
Wheat Germ Agglutinin IgA		1.05		0.2-1.1
Native & Deamidated Gliadin 33 IgG	0.74			0.2-1.2
Native & Deamidated Gliadin 33 IgA	0.60			0.1-1.1
Alpha Gliadin 17-mer IgG	0.69			0.1-1.5
Alpha Gliadin 17-mer IgA		1.06		0.1-1.1
Gamma Gliadin 15-mer IgG	0.67			0.5-1.5
Gamma Gliadin 15-mer IgA	0.29			0.1-1.0
Omega Gliadin 17-mer IgG	0.55			0.3-1.2
Omega Gliadin 17-mer IgA	0.47			0.1-1.2
Glutenin 21-mer IgG			1.57	0.1-1.5
Glutenin 21-mer IgA	0.55			0.1-1.3
Gluteomorphin + Prodynorphin IgG			1.82	0.3-1.2
Gluteomorphin + Prodynorphin IgA		0.92		0.1-1.2
Gliadin-Transglutaminase Complex IgG		1.18		0.3-1.4
Gliadin-Transglutaminase Complex IgA	0.48			0.2-1.5
Transglutaminase-2 IgG	0.90			0.3-1.6
Transglutaminase-2 IgA	1.05			0.1-1.6
Transglutaminase-3 IgG	0.77			0.2-1.6
Transglutaminase-3 IgA	0.77			0.1-1.5
Transglutaminase-6 IgG	0.57			0.2-1.5
Transglutaminase-6 IgA	0.54			0.1-1.5



Comprehensive Stool Analysis / Parasitology x3

BACTERIOLOGY CULTURE

Expected/Beneficial flora	Commensal (Imbalanced) flora	Dysbiotic flora
4+ Bacteroides fragilis group	1+ Gamma hemolytic strep	
4+ Bifidobacterium spp.	2+ Klebsiella oxytoca	
4+ Escherichia coli	1+ Staphylococcus aureus	
2+ Lactobacillus spp.		
1+ Enterococcus spp.		
1+ 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 glabrata	

MICROSCOPIC YEAST

Result:	Expected:
Many	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.

This patient was still consuming gluten despite having significant gluten intolerance, as you can see with the Cyrex Array 3. She traveled a lot for work and didn't restrict her intake while she was away from home. She has antibodies to glutenin and gluteomorphin and equivocal antibodies to gliadin transglutaminase, WGA, and alpha gliadin. She also had fungal overgrowth and elevated lysozyme and lactoferrin, which is indicative of gut inflammation. The prevalence of celiac is much higher in patients with autoimmune thyroid disease, and patients with both autoimmune thyroid disease and celiac disease have common genetic backgrounds. Studies have shown that antigliadin antibodies are more likely to be present in patients with autoimmune thyroid disease, and, in fact, the connection is so strong that some researchers have suggested

that all patients with autoimmune thyroid disease be screened for celiac and non-celiac gluten sensitivity.

Marker	Value	Functional Range	Lab Range
Glucose	97	75 – 90	65 - 99
Hemoglobin A1c	5.1	4.4 – 5.4	4.8 - 5.6
Uric Acid	4.0	3.2 - 5.5	2.5 - 7.1
BUN	6	13 – 18	6 - 20
Creatinine	0.63	0.85 – 1.1	0.57 - 1
BUN/Creatinine Ratio	10	9 – 23	8 - 20
Sodium	137	135 – 140	134 - 144
Potassium	4.3	4.0 – 4.5	3.5 - 5.2
Chloride	103	100 – 106	97 - 108
C02	22	25 – 30	18 - 29
Calcium	9.8	9.2 – 10.1	8.7 - 10.2
Phosphorus	3.4	3.5 – 4.0	2.5 - 5.3
Magnesium	1.9	2.0 – 2.6	1.6 - 2.6
Protein, total	7.1	6.9 – 7.4	6.0 - 8.5
Albumin	4.0	4.0 – 5.0	3.5 - 5.5
Globulin	3.1	2.4 – 2.8	1.5 - 4.5
A/G ratio	1.3	1.5 – 2.0	1.1 - 2.5
Bilirubin, total	<0.2	0.1 – 1.2	0.0 - 1.2
Alkaline Phosphatase	53	42 – 107	39 - 117
LDH	150	140 - 180	119 - 226
AST	21	10 - 30	0 - 40
ALT	26	10 - 22	0 - 32
GGT	13	0 - 28	0 - 60
TIBC	431	250 – 350	250 - 450
UIBC	376	150 - 375	150 - 375
Iron	55	85 – 135	35 - 155
Iron saturation	13	15 – 45	15 - 55
Ferritin	25	15 - 120	15 - 150
Cholesterol, total	185	150 – 250	100 - 199
Triglycerides	143	50 – 100	0 - 89
HDL	79	55 – 85	> 39
LDL	77	0 – 175	0 - 109
T. Chol / HDL Ratio	2.3	< 3	0 - 4.4
Triglycerides / HDL Ratio	1.81	< 2	
TSH	0.014	0.5 – 2.5	0.450 - 4.500
T4, total	12.6	6.0 – 12	4.5 - 12.0
T3 Uptake	20	28 - 35	24 - 39
T3, Total	241	100 – 180	71 - 180
Vitamin D, 25-hydroxy	50.3	35 - 60	30.0 - 100.0

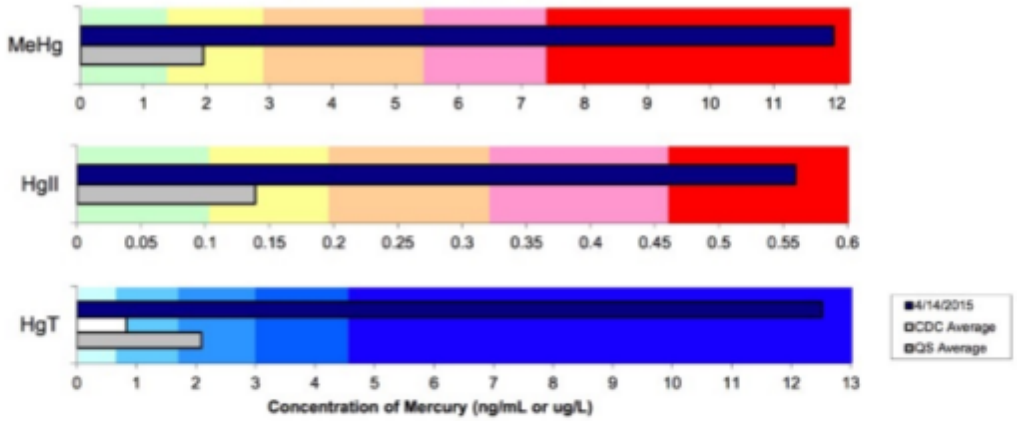
Marker	Value	Functional Range	Lab Range
WBC	5.7	5.0 – 8.0	3.4 - 10.8
RBC	4.89	4.4 – 4.9	3.77 - 5.28
Hemoglobin	14.0	13.5 - 14.5	11.1 - 15.9
Hematocrit	40.8	37 - 44	34.0 - 46.6
MCV	83	85 – 92	79 - 97
MCH	28.6	27.7 – 32.0	26.6 - 33.0
MCHC	34.3	32 – 35	31.5 - 35.7
RDW	14.9	11.5 – 15.0	12.3 - 15.4
Platelets	379	150 – 415	150 - 379
Neutrophils	41	40 – 60	
Lymphocytes	46	25 – 40	
Monocytes	11	4.0 – 7.0	
Eosinophils	1	0.0 – 3.0	
Basophils	1	0.0 – 3.0	

Additional Tests:			
T3, Free	4.5	2.5 - 4.0	2.3 - 5
T4, Free	1.28	1 - 1.5	0.93 - 1.6
CRP-hs	4.77	< 1.0	0.00 - 3.00
Homocysteine	5.7	< 7.0	0.0 - 15.0
Vitamin B-12	517	450 – 2000	211 - 946
Copper	192		72 - 166
Zinc	76		56 - 134
Zinc / Copper Ratio	0.40	> 0.85	
Serum Methylmalonic Acid (MMA)	95	0 - 325	0 - 378

Okay, the next patient is a 39-year-old female with infertility, hot flashes, waking up sweating at night, anxiety, and loose stools. TSH is again nearly zero. T4 is high at 12.6. T3 is high at 241. Free T3 is out of the functional range at 4.5. Free T4 is totally normal. T3 uptake is low, and this can be caused by high estrogen, and the patient was taking an oral contraceptive, which is one of the main causes of low T3 uptake that I see. Note that her C-reactive protein and copper are both high, and she has a very low zinc-to-copper ratio, which suggests inflammation is present. She is also iron deficient. Iron saturation is at 13 percent. TIBC and UIBC are high. Serum iron and ferritin are borderline low. Her fasting glucose and triglycerides are high. As I mentioned on the last case, you can see both low and high blood sugar with Graves', but I much more commonly see high blood sugar in cases of hyperthyroidism. ALT is elevated out of the functional range, which is consistent with what we talked about before, as ALT and AST being potential consequences of hyperthyroidism.

Blood Results

Blood Mercury Comparison

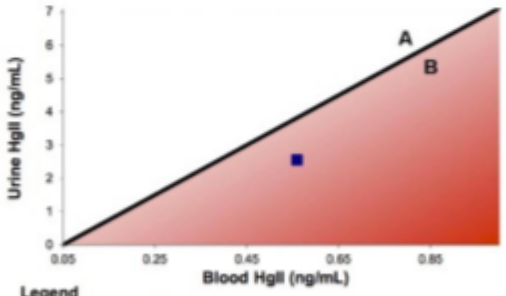


	Results (ng/mL)			Reference Ranges						
				QS n=1011; CDC n=1928			Percentile			
	4/14/2015	NA	% Change	Source	Range	Average	50th	75th	90th	95th
Methylmercury— MeHg	12.0	NA	NA	QS	<0.003 to 23.3	1.95	1.2	2.9	5.4	7.4
Inorganic Mercury— Hg^{II}	0.560	NA	NA	QS	<0.007 to 1.75	0.139	0.10	0.19	0.32	0.46
Sum— HgT	12.5	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

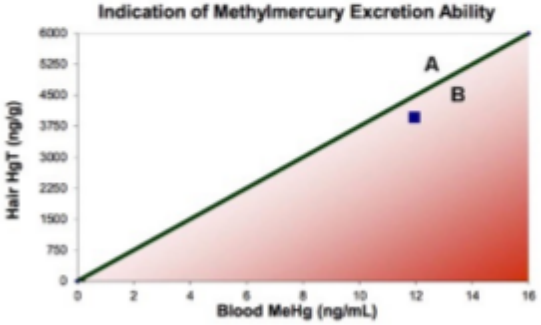
Indication of Inorganic Mercury Excretion Ability



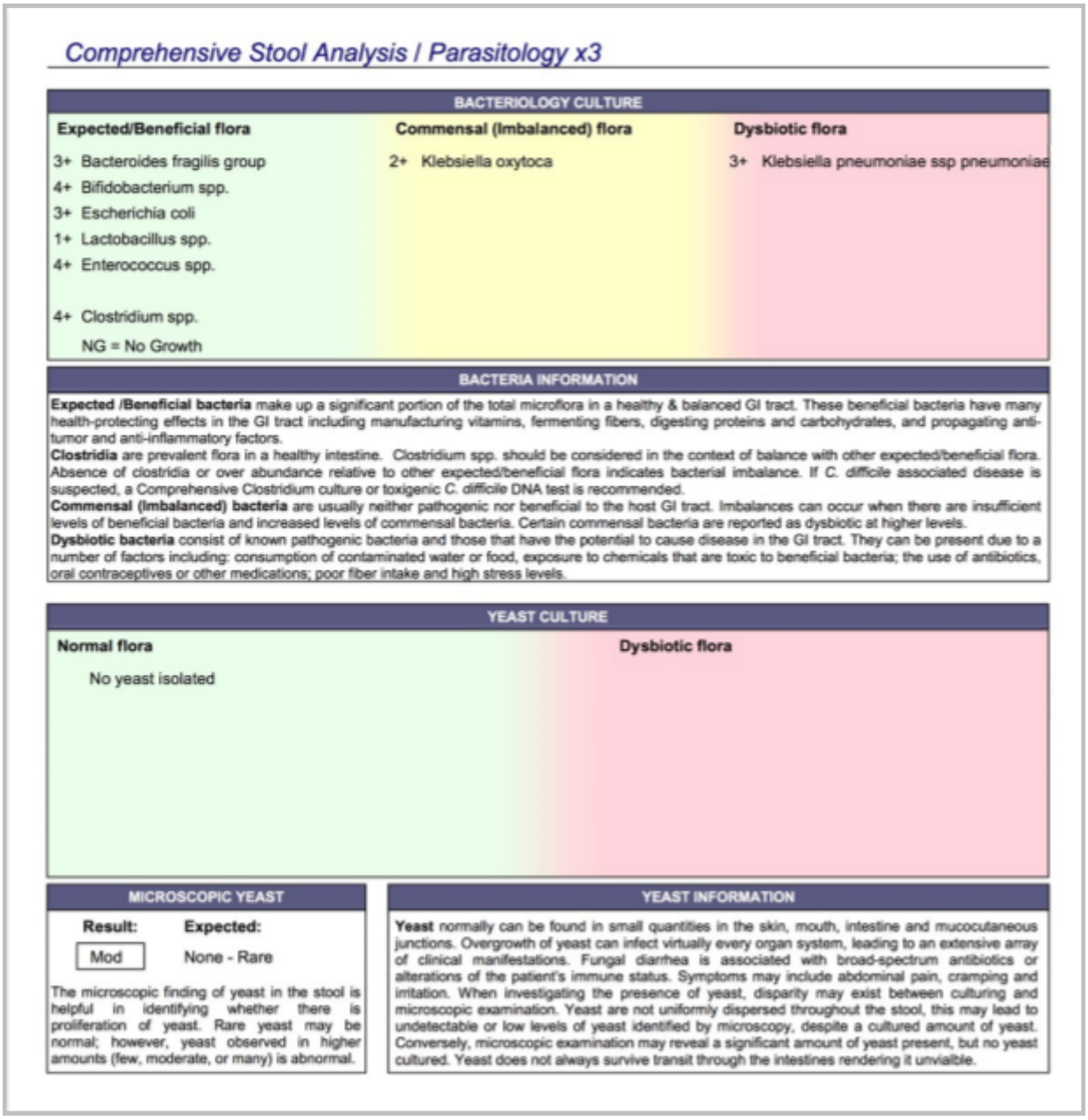
Legend
A) Average Excretion: Mercury output is average or above average when at a ratio of at least 375:1 HgT in hair to MeHg in blood and 6.9:1 HgT in urine to HgII in blood.
B) Below Average Excretion: Mercury output is below average when the tissue Hg comparisons are below ratios mentioned above (red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

Hair Results

Indication of Methylmercury Excretion Ability



	Urine Results (ng/mL)			Hair (ng/g)
	4/14/2015	NA	%Change	4/14/2015
Methylmercury— MeHg	0.011	NA	NA	NA
Inorganic Mercury— Hg^{II}	2.55	NA	NA	NA
Sum— HgT	2.56	NA	NA	3960



Comprehensive Stool Analysis / Parasitology x3

BACTERIOLOGY CULTURE

Expected/Beneficial flora	Commensal (Imbalanced) flora	Dysbiotic flora
3+ Bacteroides fragilis group	2+ Klebsiella oxytoca	3+ Klebsiella pneumoniae ssp pneumoniae
4+ Bifidobacterium spp.		
3+ Escherichia coli		
1+ 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:
Mod	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.

This patient did have several possible immune triggers present. She had mercury toxicity. Her total mercury was 12, and her inorganic mercury was 0.56, which puts her probably in the 99th percentile for inorganic mercury. She is over the 95th percentile for total mercury and organic mercury as well, so even though this isn't out of the lab range, it is at a level that we would certainly treat. She also had Klebsiella and moderate fungal overgrowth on Doctor's Data stool panel.

TESTS	RESULT	FLAG	UNITS	REFERENCE INTERVAL	LAB
TSH+T4F+T3Free					
TSH	**0.010**	**Low**	uIU/mL	0.450 - 4.500	01
Triiodothyronine,Free,Serum	4.9		pg/mL	2.3 - 5.0	01
T4,Free(Direct)	1.32		ng/dL	0.93 - 1.60	01
Thyroid Stim Immunoglobulin	40		%	0 - 139	02
Thyroid Antibodies					
Thyroid Peroxidase (TPO) Ab	7		IU/mL	0 - 26	01
Thyroglobulin Antibody	<1.0		IU/mL	0.0 - 0.9	01
Thyroglobulin Antibody measured by Beckman Coulter Methodology					

Follow-up testing revealed negative thyroid antibodies, TPO, and thyroglobulin, but persistent hyperthyroidism. TSH was again effectively zero. Free T3 was actually a little higher. In this case, it was 4.9, almost out of the lab range. Again, free T4 was normal. TSI was completely normal at 40, and they are highly sensitive, so a false negative is unlikely but again possible. TPO and thyroglobulin are a lot less sensitive, so it is possible she has Hashitoxicosis. At this point, I might refer out to an endocrinologist who can test for toxic multinodular goiter, adenoma, and some of the less common causes of hyperthyroidism. In the meantime, you can also focus on addressing the underlying mechanisms, the mercury toxicity and the gut, to see if that helps, but when T3 is high, TSH is nearly zero, and the cause is unclear, I would definitely suggest referring out for further workup.

Ca+PTH Intact

Calcium, Serum	10.2	mg/dL	8.7 - 10.2
PTH, Intact	24	pg/mL	15 - 65

Intact PTH

Interpretation	Intact PTH (pg/mL)	Calcium (mg/dL)
Normal	15 - 65	8.6 - 10.2
Primary Hyperparathyroidism	>65	>10.2
Secondary Hyperparathyroidism	>65	<10.2
Non-Parathyroid Hypercalcemia	<65	>10.2
Hypoparathyroidism	<15	< 8.6
Non-Parathyroid Hypocalcemia	15 - 65	< 8.6

TSH	**0.287**	**Low**	uIU/mL	0.450 - 4.500

Thyroxine (T4) Free, Direct, S

T4,Free(Direct)	1.64	ng/dL	0.82 - 1.77

Vitamin D, 25-Hydroxy	**26.9**	**Low**	ng/mL	30.0 - 100.0

Vitamin D deficiency has been defined by the Institute of Medicine and an Endocrine Society practice guideline as a level of serum 25-OH vitamin D less than 20 ng/mL (1,2). The Endocrine Society went on to further define vitamin D insufficiency as a level between 21 and 29 ng/mL (2).

1. IOM (Institute of Medicine). 2010. Dietary reference intakes for calcium and D. Washington DC: The National Academies Press.
2. Holick MF, Binkley NC, Bischoff-Ferrari HA, et al. Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. JCEM. 2011 Jul; 96(7):1911-30.

Phosphorus, Serum	4.0	mg/dL	2.5 - 4.5
Triiodothyronine,Free,Serum	3.4	pg/mL	2.0 - 4.4

Thyroid Antibodies

Thyroid Peroxidase (TPO) Ab	12	IU/mL	0 - 34
Thyroglobulin Antibody	<1.0	IU/mL	0.0 - 0.9

Thyroglobulin Antibody measured by Beckman Coulter Methodology

Thyroid Stim Immunoglobulin	17	%	0 - 139

Here is a different presentation. This is subclinical hyperthyroidism. TSH is low, but free T4 and free T3 are normal. Vitamin D is on the low end, but look at PTH. It's below 30, which as you now know suggests he is not biologically deficient. This patient is a 48-year-old male with chief complaint of anxiety, OCD, and difficulty with word recall and memory, as well as low libido. This could be a transient situation where we just caught a low TSH, which isn't even all that low—it's 0.287—or this patient may just have a low TSH setpoint. There is not much evidence that

slightly low TSH with normal T4 and T3 is problematic. Thyroid antibodies were normal. In this case, I would just address the other issues that we find in the case review and then retest the full thyroid panel after that or in six months or so.

Thyroid				
TSH	**<0.006**	**Low**	uIU/mL	0.450 - 4.500
Thyroxine (T4)	8.8		ug/dL	4.5 - 12.0
T3 Uptake	30		%	24 - 39
Free Thyroxine Index	2.6			1.2 - 4.9
Triiodothyronine (T3)	**193**	**High**	ng/dL	71 - 180
Immunoassay				
Vitamin D, 25-Hydroxy	**13.9**	**Low**	ng/mL	30.0 - 100.0

The next patient has again extremely low TSH, 0.006, effectively zero. T4 is normal, but T3 is high, which again is more common in Graves'. Vitamin D is low, and when it is this low, there is really no need to test PTH. It is 13.9. That is pretty much deficiency for everybody. As we discussed in the hypothyroidism presentation, in the context of Hashimoto's, vitamin D plays an important role in T-regulatory cell production and differentiation, and low vitamin D will put patients at risk for developing autoimmunity and also exacerbate existing autoimmunity.

TSH	0.573		uIU/mL	0.450 - 4.500
Thyroxine (T4)	***<0.5***	***Alert***	ug/dL	4.5 - 12.0
Verified by repeat analysis				
T3 Uptake	**23**	**Low**	%	24 - 39
Free Thyroxine Index	**<.1**	**Low**		1.2 - 4.9
Triiodothyronine (T3)	**209**	**High**	ng/dL	71 - 180

Here is an example of facetious hyperthyroidism, and we talked about this a bit in the last unit. This is caused by excess thyroid hormone replacement. Notice that the TSH is still normal at 0.573. It's low, but it is still normal. T4, on the other hand, is almost zero. It's being completely suppressed by negative feedback. It is less than 0.5. They couldn't even detect it, and they underlined it and put it in red to alert me. Then T3, total T3, is high at 209. This is a situation where the body is attempting to protect itself from excess thyroid hormone by reducing endogenous T4 output.

The question is whether facetious hyperthyroidism is a cause for concern. Many clinicians, including her prescribing doctor, believe that it is not and are not particularly worried when they see a presentation like this.

Beware of falsely low TSH with thyroid hormone replacement.

I believe that that is a mistake, and there is a way of determining whether facetious hyperthyroidism is a problem. If TSH is suppressed, even significantly below the lab range, but T4 and T3 are normal, it's probably not a concern provided that TSH is above 0.04, so again, that is very low. It's close to zero, but studies have shown that at that level, if T4 and T3 are normal, there is not a significantly increased risk of osteoporosis and other conditions that are associated with hyperthyroidism. This was, to be frank, news to me. I was under that impression until I did the really deep research for this unit.

Research has shown that the pituitary gland has receptors for TSH, and they recognize TSI as if they were TSH molecules. This causes a reduction of TSH production because the body thinks there is already enough, and this can lead to falsely low TSH levels. However, if the patient has a very low TSH while taking thyroid hormone replacement, and their T4 and especially T3 are elevated, that patient is most definitely at additional risk for bone and cardiovascular disease, and that should be addressed. That was the case with the patient on the last slide.