KRESSER INSTITUTE
FOR FUNCTIONAL AND EVOLUTIONARY MEDICINE

Dyslipidemia - Part Three

Okay, now let's look at some cases. The first patient is a 59-year-old female with chief complaint of dry, red, hot, painful, and very itchy skin. She had not had any comprehensive blood work for over two decades and was unaware that she had very high cholesterol.

Marker	Value	Functional Range	Lab Range
Glucose	90	75 – 90	65 - 99
Hemoglobin A1c	5.6	4.4 – 5.4	4.8 - 5.6
Uric Acid	4.7	3.2 - 5.5	2.5 - 7.1
BUN	17	13 – 18	6 - 24
Creatinine	0.79	0.85 – 1.1	0.57 - 1
BUN/Creatinine Ratio	22	9 – 23	9 - 23
Sodium	141	135 – 140	134 - 144
Potassium	4.3	4.0 – 4.5	3.5 - 5.2
Chloride	102	100 – 106	97 - 108
C02	24	25 – 30	18 - 29
Calcium	9.3	9.2 – 10.1	8.7 - 10.2
Phosphorus	4.0	3.5 – 4.0	2.5 - 4.5
Magnesium	2.1	2.0 – 2.6	1.6 - 2.6
Protein, total	6.2	6.9 – 7.4	6.0 - 8.5
Albumin	4.1	4.0 – 5.0	3.5 - 5.5
Globulin	2.1	2.4 – 2.8	1.5 - 4.5
A/G ratio	2.0	1.5 – 2.0	1.1 - 2.5
Bilirubin, total	0.3	0.1 – 1.2	0.0 - 1.2
Alkaline Phosphatase	63	42 – 107	39 - 117
LDH	187	140 - 180	119 - 226
AST	20	10 - 30	0 - 40
ALT	16	10 - 22	0 - 32
GGT	7	0 - 28	0 - 60
TIBC	230	250 – 350	250 - 450
UIBC	148	150 - 375	150 - 375
Iron	82	85 – 135	35 - 155
Iron saturation	36	15 – 45	15 - 55
Ferritin	133	MW: 30 - 150	15 - 150
Cholesterol, total	356	150 – 250	100 - 199
Triglycerides	49	50 – 100	0 - 149
HDL	98	55 – 85	> 39
LDL	248	0 – 175	0 - 99
T. Chol / HDL Ratio	3.6	< 3	0 - 4.4
Triglycerides / HDL Ratio	0.50	< 2	< 3.8
TSH	2.270	0.5 – 2.5	0.450 - 4.500
T4, total	6.5	6.0 – 12	4.5 - 12.0
T3 Uptake	33	28 - 35	24 - 39
T3, Total	73	100 – 180	71 - 180
Vitamin D, 25-hydroxy	35.3	35 - 60	30.0 - 100.0

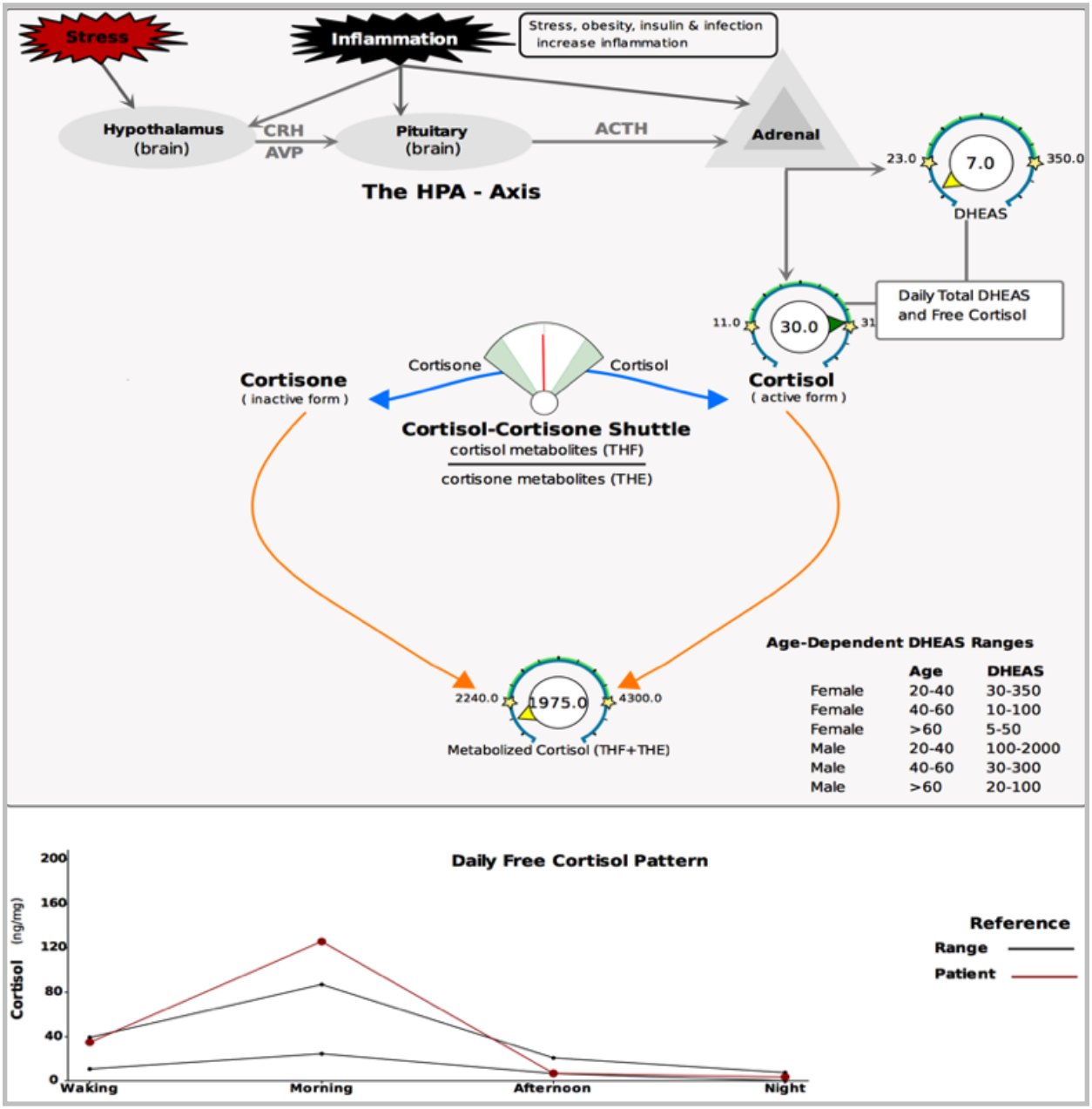
Marker	Value	Functional Range	Lab Range
WBC	5.3	5.0 – 8.0	3.4 - 10.8
RBC	4.27	4.4 – 4.9	3.77 - 5.28
Hemoglobin	13.5	13.5 - 14.5	11.1 - 15.9
Hematocrit	38.5	37 - 44	34.0 - 46.6
MCV	90	85 – 92	79 - 97
MCH	31.6	27.7 – 32.0	26.6 - 33.0
MCHC	35.1	32 – 35	31.5 - 35.7
RDW	13.4	11.5 – 15.0	12.3 - 15.4
Platelets	273	150 – 415	150 - 379
Neutrophils	60	40 – 60	
Lymphocytes	31	25 – 40	
Monocytes	7	4.0 – 7.0	
Eosinophils	1	0.0 – 3.0	
Basophils	1	0.0 – 3.0	
Additional Tests:			
CRP-hs	0.26	< 1.0	0.00 - 3.00
Homocysteine	8.5	< 7.0	0.0 - 15.0
Vitamin B-12	443	450 – 2000	211 - 946
Copper	110		72 - 166
Zinc	105		56 - 134
Zinc / Copper Ratio	0.95	> 0.85	
Serum Methylmalonic Acid (MMA)	137	0 - 325	0 - 378

As you can see, her total cholesterol is 356. Her HDL is also high at 98, but because her total cholesterol is so high, the total cholesterol-to-HDL ratio is still above the optimal range of below three. Hers is 3.6. Given her total cholesterol of 356, LDL of 248, and the fact that she did have a tendon xanthoma, she meets the Simon Broome criteria for FH. Note that her TSH is borderline high, and total T3 is borderline low, so thyroid hypofunction may be playing a role here.

GI Pathogen Screen with H. pylori Antigen - 401H

Parameter	Result
***** Stool Culture *****	
Preliminary Report	Normal flora after 24 hours
Final Report	* Escherichia coli isolated *
Amount of Growth	Abundant
***** Ova & Parasites *****	
Ova & Parasites #1	No Ova/Parasites detected
Ova & Parasites #2	* Blastocystis hominis detected *
Ova & Parasites #3	No Ova/Parasites detected
Ova & Parasites #4	No Ova/Parasites detected
Trichrome Stain	Few protozoan forms of Blastocystis hominis seen on Trichrome Stain
***** Stool Antigens *****	
Cryptosporidium Antigen	Not detected
Giardia lamblia Antigen	Not detected
***** Additional Tests *****	
Fungi	No fungi isolated
C. difficile Toxin A	Not detected
C. difficile Toxin B	Not detected
Yeast	No yeasts isolated
Occult Blood	Not detected
*****Helicobacter Pylori Stool Antigen*****	
H. pylori Antigen	Not detected

This stool analysis determines the presence of ova and parasites such as protozoa, flatworms, and roundworms; Cryptosporidium parvum, Entamoeba histolytica, and Giardia lamblia antigens; bacteria, fungi (including yeasts), and occult blood; and Clostridium difficile colitis toxins A and B. Sensitivity to pathogenic organisms will be reported as necessary.

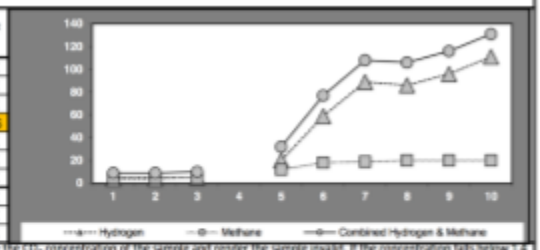


She had Blastocystis hominis on her BioHealth stool test. She had borderline high free cortisol and low metabolized on DUTCH, which strengthens the hypothesis that hypothyroidism is an issue.

Data

H_2 = Hydrogen CH_4 = Methane CO_2 = Measured

	Sample	ppm H_2	ppm CH_4	Total H2 + CH4	CO_2 %*
1	Baseline	4	5	9	OK
2	20 min	4	5	9	OK
3	40 min	5	5	10	OK
4	60 min				QNS
5	80 min	20	12	32	OK
6	100 min	59	18	77	OK
7	120 min	89	19	108	OK
8	140 min	86	20	106	OK
9	160 min	96	20	116	OK
10	180 min	111	20	131	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 =	9	-	≤20ppm
Greatest H_2 increase over the lowest preceding value within first 120 minutes =	85	H	≤20ppm
Greatest CH_4 increase over the lowest preceding value within first 120 minutes =	14	H	≤12ppm
Greatest combined H_2 & CH_4 increase over the lowest preceding value within first 120 minutes =	99	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.	**POSITIVE**
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**

She had an equivocal positive for SIBO. This could be a single peak, but there is a little dip and an increase again, so that could be a double peak. Because of her skin issues, it makes me consider SIBO more carefully, so I would definitely treat in this situation.

	Laboratory Test	Notes	High Risk	Intermediate Risk	Optimal	High Risk Range	Intermediate Risk Range	Optimal Range	Previous Results
Lipids	Total Cholesterol (mg/dL)		379			≥ 240	200 - 239	< 200	
	LDL-C Direct (mg/dL)		261			≥ 130 CHD & CHD risk eq. > 100	100 - 129 CHD & CHD risk eq. 70 - 100	< 100 CHD & CHD risk eq. < 70	
	HDL-C (mg/dL)				105	< 50		≥ 50	
	Triglycerides (mg/dL)				56	> 199	150 - 199	< 150	
	Non-HDL-C (mg/dL) (calculated)		273			≥ 160	130 - 159	< 130	
Lipoprotein Particles and Apolipoproteins	Apo B (mg/dL)		182			≥ 80	60 - 79	< 60	
	LDL-P (nmol/L)[5], by NMR		2640			≥ 1360	1020 - 1359	< 1020	
	Small LDL-P (nmol/L)[5], by NMR			537		> 1000	501 - 1000	< 501	
	sdLDL-C (mg/dL)[5]		59			> 30	21 - 30	< 21	
	Apo A-I (mg/dL)				208	< 130	130 - 150	> 150	
	HDL-P (µmol/L)[5], by NMR				43.2	≤ 34.0	34.1 - 38.0	> 38.0	
	HDL2-C (mg/dL)[5]				55	≤ 12	13 - 16	≥ 17	
	Apo B:Apo A-I Ratio (calculated)		0.87			≥ 0.81	0.61 - 0.80	≤ 0.60	
	Lp(a)-P (nmol/L)[5]				61	> 125	75 - 125	< 75	
Inflammation/ Oxidation	Fibrinogen (mg/dL)				352	< 126 or > 517	438 - 517	126 - 437	
	hs-CRP (mg/L)				< 0.3	> 2.9	1.0 - 2.9	< 1.0	
	Lp-PLA$_2$ (ng/mL)[5]		509			> 383	291 - 383	< 291	
	Oxidized LDL-β$_2$GPI (U/mL)[5]				< 0.1	≥ 0.2 High Risk	0.1 Moderate Risk	< 0.1 Low Risk	

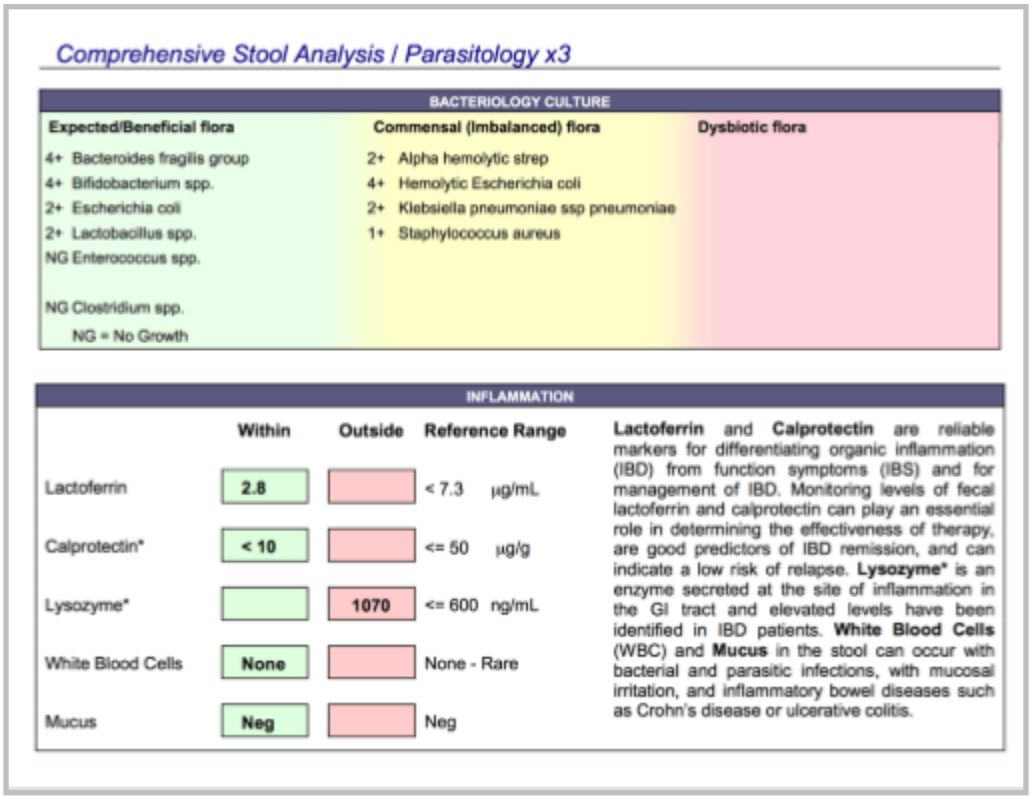
Unfortunately, in this particular case, after addressing her thyroid issues, her gut, SIBO, and HPA axis, she didn't have any environmental toxins or insulin resistance, her total cholesterol was even higher six months later on the retest. It's not clear if the difference between the two results is statistically significant because there is so much intraindividual variation for total cholesterol. It can vary up to two standard deviations, which a standard deviation for total cholesterol is 17 mg/dL, so it can go up or down by about 30 points from test to test without any changes in diet or treatment. We can conclusively say that this is not an improvement. In this case, the patient will need direct intervention to reduce LDL-P because addressing underlying causes did not budge the numbers in her case.

Her LDL-P is 2,640. That puts her in something like the 98th or 99th percentile, if I'm going by memory, maybe the 99th. It's very high. Fortunately, her Lp(a)-P is normal at 61, so this would be much more concerning if the Lp(a) was high. Most of her inflammatory markers are normal, but her Lp-PLA2, which is a specific marker of cardiovascular inflammation, is high at 509.

This patient is a 57-year-old male with a sole complaint of high cholesterol.

	Laboratory Test	Notes	High Risk	Intermediate Risk	Optimal	High Risk Range	Intermediate Risk Range	Optimal Range
Lipids	Total Cholesterol (mg/dL)		293			≥ 240	200 - 239	< 200
	LDL-C Direct (mg/dL)		226			≥ 130 CHD & CHD risk eq. > 100	100 - 129 CHD & CHD risk eq. 70 - 100	< 100 CHD & CHD risk eq. < 70
	HDL-C (mg/dL)				66	< 40		≥ 40
	Triglycerides (mg/dL)				60	> 199	150 - 199	< 150
	Non-HDL-C (mg/dL) (calculated)		228			≥ 160	130 - 159	< 130
Lipoprotein Particles and Apolipoproteins	Apo B (mg/dL)		152			≥ 80	60 - 79	< 60
	LDL-P (nmol/L)§, by NMR		2582			≥ 1360	1020 - 1359	< 1020
	Small LDL-P (nmol/L)§, by NMR			997		> 1000	501 - 1000	< 501
	sdLDL-C (mg/dL)§		44			> 30	21 - 30	< 21
	Apo A-I (mg/dL)				159	< 114	114 - 131	> 131
	HDL-P (µmol/L)§, by NMR			37.3		≤ 34.0	34.1 - 38.0	> 38.0
	HDL2-C (mg/dL)§				24	≤ 8	9 - 11	≥ 12
	Apo B:Apo A-I Ratio (calculated)		0.95			≥ 0.81	0.61 - 0.80	≤ 0.60
	Lp(a)-P (nmol/L)§				< 50	> 125	75 - 125	< 75
Inflammation/ Oxidation	Fibrinogen (mg/dL)				409	< 126 or > 517	438 - 517	126 - 437
	hs-CRP (mg/L)				< 0.3	> 2.9	1.0 - 2.9	< 1.0
	Lp-PLA$_2$ (ng/mL)§		597			> 383	291 - 383	< 291

Total cholesterol is 293. LDL cholesterol is 228. He didn't have any tendon xanthoma and didn't technically meet the Simon Broome criteria, although when you see LDL-P this high, it's quite likely that there is some kind of genetic mutation that is contributing or polymorphism that is contributing. LDL-P is 2,582. That is again a very-high-risk category. Optimally you want it to be below … the THD range is below 1,000 optimal. I say below 1,300 is optimal level, especially if there aren't any other risk factors present. Again, fortunately, his Lp(a)-P is in the optimal range of below 50, and once again, we have a situation where the Lp-PLA2, the specific marker of cardiovascular inflammation, is high.



Comprehensive Stool Analysis / Parasitology x3

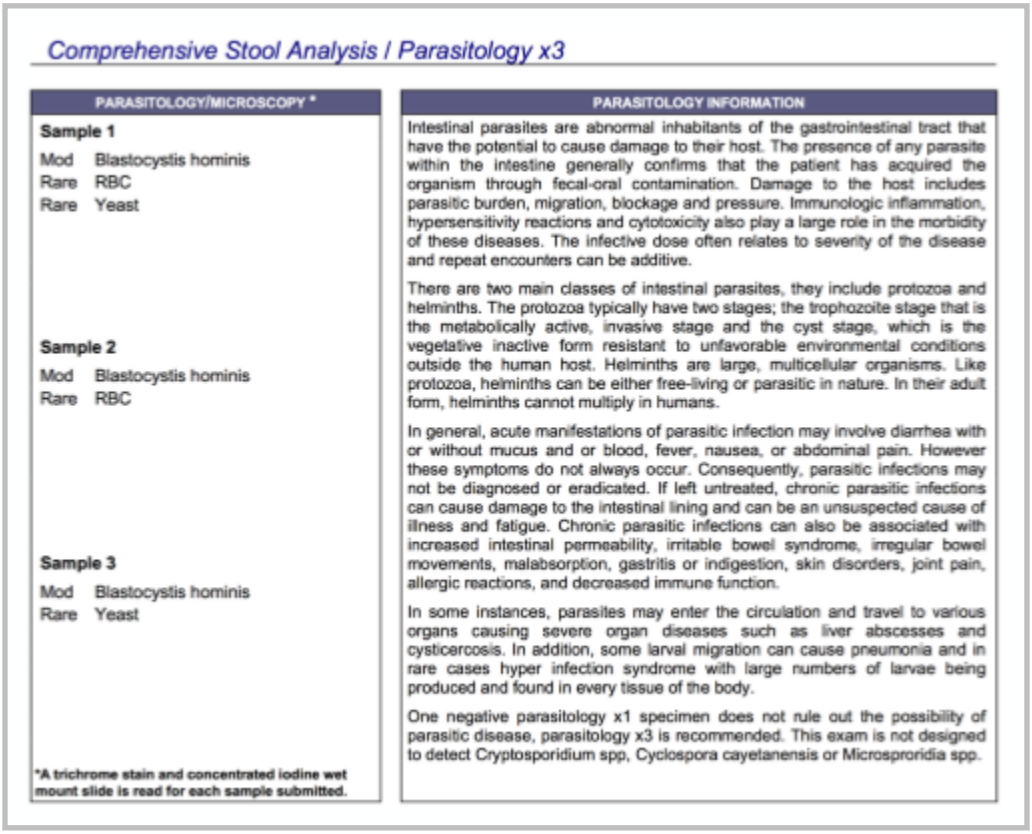
BACTERIOLOGY CULTURE

Expected/Beneficial flora	Commensal (Imbalanced) flora	Dysbiotic flora
4+ Bacteroides fragilis group	2+ Alpha hemolytic strep	
4+ Bifidobacterium spp.	4+ Hemolytic Escherichia coli	
2+ Escherichia coli	2+ Klebsiella pneumoniae ssp pneumoniae	
2+ Lactobacillus spp.	1+ Staphylococcus aureus	
NG Enterococcus spp.		
NG Clostridium spp.		
NG = No Growth		

INFLAMMATION

	Within	Outside	Reference Range
Lactoferrin	2.8		< 7.3 µg/mL
Calprotectin*	< 10		<= 50 µg/g
Lysozyme*		1070	<= 600 ng/mL
White Blood Cells	None		None - Rare
Mucus	Neg		Neg

Lactoferrin and **Calprotectin** are reliable markers for differentiating organic inflammation (IBD) from function symptoms (IBS) and for management of IBD. Monitoring levels of fecal lactoferrin and calprotectin can play an essential role in determining the effectiveness of therapy, are good predictors of IBD remission, and can indicate a low risk of relapse. **Lysozyme*** is an enzyme secreted at the site of inflammation in the GI tract and elevated levels have been identified in IBD patients. **White Blood Cells** (WBC) and **Mucus** in the stool can occur with bacterial and parasitic infections, with mucosal irritation, and inflammatory bowel diseases such as Crohn's disease or ulcerative colitis.



Comprehensive Stool Analysis / Parasitology x3

PARASITOLOGY/MICROSCOPY *	
Sample 1	
Mod	Blastocystis hominis
Rare	RBC
Rare	Yeast
Sample 2	
Mod	Blastocystis hominis
Rare	RBC
Sample 3	
Mod	Blastocystis hominis
Rare	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.

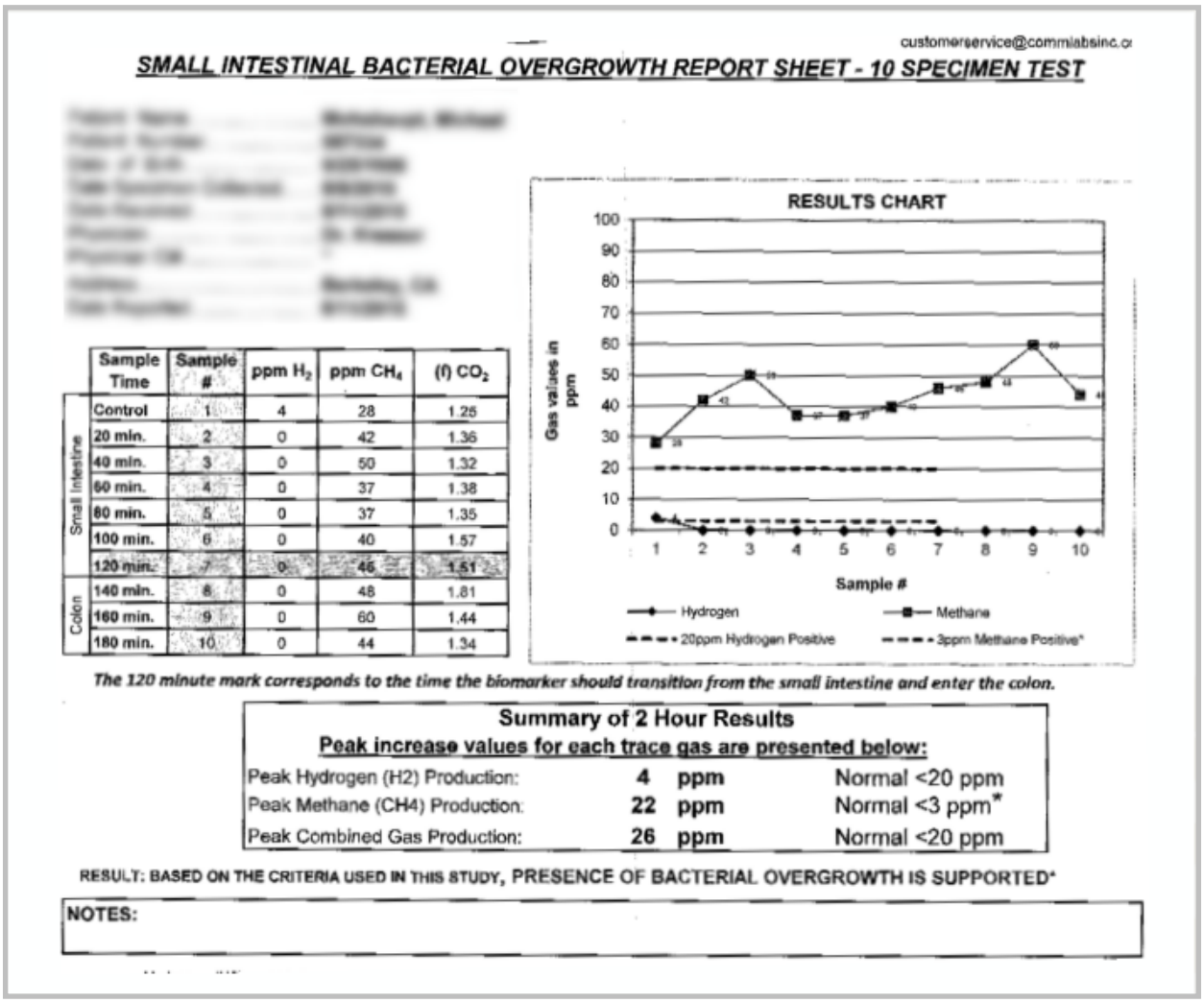
There are two main classes of intestinal parasites, they include protozoa and helminths. The protozoa typically have two stages; the trophozoite stage that is the metabolically active, invasive stage and the cyst stage, which is the vegetative inactive form resistant to unfavorable environmental conditions 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.

This patient had gut dysbiosis, gut inflammation, and Blastocystis hominis.



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SMALL INTESTINAL BACTERIAL OVERGROWTH REPORT SHEET - 10 SPECIMEN TEST

[redacted]

	Sample Time	Sample #	ppm H_2	ppm CH_4	(f) CO_2
Small Intestine	Control	1	4	28	1.25
	20 min.	2	0	42	1.36
	40 min.	3	0	50	1.32
	60 min.	4	0	37	1.38
	80 min.	5	0	37	1.35
	100 min.	6	0	40	1.57
	120 min.	7	0	46	1.51
Colon	140 min.	8	0	48	1.81
	160 min.	9	0	60	1.44
	180 min.	10	0	44	1.34

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

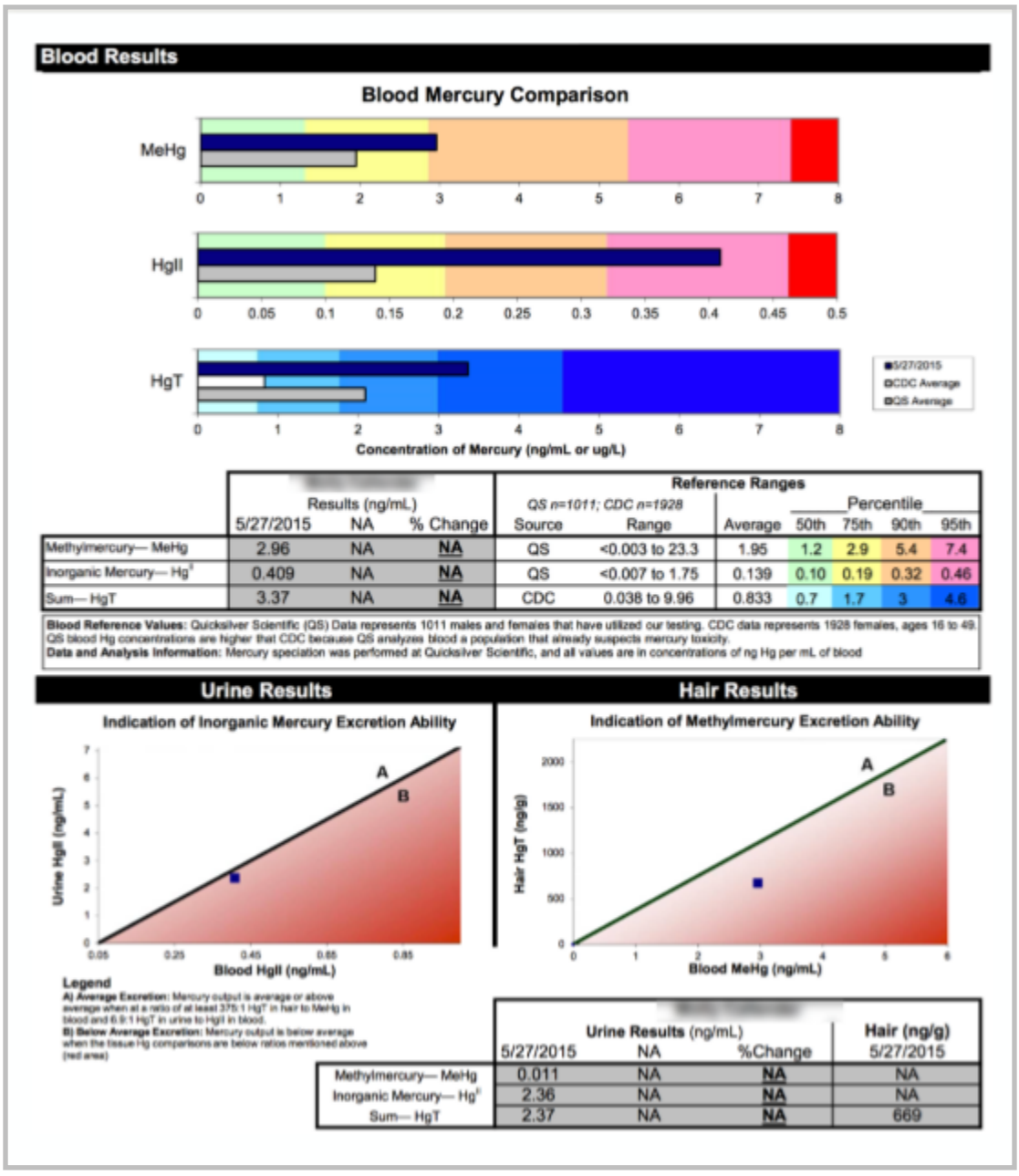
Peak increase values for each trace gas are presented below:

Peak Hydrogen (H2) Production:	**4 ppm**	Normal <20 ppm
Peak Methane (CH4) Production:	**22 ppm**	Normal <3 ppm*
Peak Combined Gas Production:	**26 ppm**	Normal <20 ppm

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

NOTES:

He also had methane-predominant SIBO with a peak value in the first 120 minutes of 46 parts per million.



Blood Results

Blood Mercury Comparison

	Results (ng/mL) 5/27/2015	NA	% Change	Reference Ranges QS n=1011; CDC n=1928 Source	Range	Average	Percentile 50th	75th	90th	95th
Methylmercury— MeHg	2.96	NA	NA	QS	<0.003 to 23.3	1.95	1.2	2.9	5.4	7.4
Inorganic Mercury— Hg^{II}	0.409	NA	NA	QS	<0.007 to 1.75	0.139	0.10	0.19	0.32	0.46
Sum— HgT	3.37	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) 5/27/2015	NA	%Change	Hair (ng/g) 5/27/2015
Methylmercury— MeHg	0.011	NA	NA	NA
Inorganic Mercury— Hg^{II}	2.36	NA	NA	NA
Sum— HgT	2.37	NA	NA	669

Normal total mercury levels, but his inorganic mercury levels were above the 95th percentile, high at 0.4. The range here goes up to 1.75, but we typically treat above 0.2. He had slightly impaired detox capacity for inorganic mercury and methylmercury.

TESTS	RESULT	FLAG	UNITS	REFERENCE INTERVAL	LAB
TSH+T4F+T3Free					
TSH	**7.140**	**High**	uIU/mL	0.450 - 4.500	01
Triiodothyronine,Free,Serum	2.0		pg/mL	2.0 - 4.4	01
T4,Free(Direct)	1.23		ng/dL	0.82 - 1.77	01
Renal Panel (10)					
Glucose, Serum	83		mg/dL	65 - 99	01
BUN	16		mg/dL	8 - 27	01
Creatinine, Serum	0.86		mg/dL	0.57 - 1.00	01
eGFR If NonAfricn Am	72		mL/min/1.73	>59	
eGFR If Africn Am	83		mL/min/1.73	>59	
BUN/Creatinine Ratio	19			11 - 26	
Sodium, Serum	139		mmol/L	134 - 144	01
Potassium, Serum	4.1		mmol/L	3.5 - 5.2	01
Chloride, Serum	100		mmol/L	97 - 108	01
Carbon Dioxide, Total	23		mmol/L	18 - 29	01
Calcium, Serum	9.2		mg/dL	8.7 - 10.3	01
Phosphorus, Serum	4.0		mg/dL	2.5 - 4.5	01
Albumin, Serum	4.0		g/dL	3.6 - 4.8	01

He had a high thyroid-stimulating hormone level and borderline low free T3, which is indicative of poor thyroid function probably contributing.

	Laboratory Test	Notes	High Risk	Intermediate Risk	Optimal	High Risk Range	Intermediate Risk Range	Optimal Range	Previous Results 1/29/2016
Lipids	Total Cholesterol (mg/dL)				190	≥ 240	200 - 239	< 200	293
	LDL-C Direct (mg/dL)			122		≥ 130 CHD & CHD risk eq. > 100	100 - 129 CHD & CHD risk eq. 70 - 100	< 100 CHD & CHD risk eq. < 70	226
	HDL-C (mg/dL)				62	< 40		≥ 40	66
	Triglycerides (mg/dL)				65	> 199	150 - 199	< 150	60
	Non-HDL-C (mg/dL) (calculated)				128	≥ 160	130 - 159	< 130	228
Lipoprotein Particles and Apolipoproteins	Apo B (mg/dL)		90			≥ 80	60 - 79	< 60	152
	LDL-P (nmol/L)[5], by NMR		1511			≥ 1360	1020 - 1359	< 1020	2582
	Small LDL-P (nmol/L)[5], by NMR			649		> 1000	501 - 1000	< 501	997
	sdLDL-C (mg/dL)[5]			22		> 30	21 - 30	< 21	44
	Apo A-I (mg/dL)				171	< 114	114 - 131	> 131	159
	HDL-P (µmol/L)[5], by NMR				38.4	≤ 34.0	34.1 - 38.0	> 38.0	37.3
	HDL2-C (mg/dL)[5]				23	≤ 8	9 - 11	≥ 12	24
	Apo B:Apo A-I Ratio (calculated)				0.53	≥ 0.81	0.61 - 0.80	≤ 0.60	0.95
	Lp(a)-P (nmol/L)[5]				< 50	> 125	75 - 125	< 75	< 50
Inflammation/ Oxidation	Fibrinogen (mg/dL)				420	< 126 or > 517	438 - 517	126 - 437	409
	hs-CRP (mg/L)				0.4	> 2.9	1.0 - 2.9	< 1.0	< 0.3
	Lp-PLA$_2$ (ng/mL)[5]				256	> 383	291 - 383	< 291	597
	Myeloperoxidase (pmol/L)[5]			281		≥ 332	256 - 331	≤ 255	263
	Oxidized LDL-β$_2$GPI (U/mL)[5]				< 0.1	≥ 0.2 High Risk	0.1 Moderate Risk	< 0.1 Low Risk	

In this case, after addressing all of the core mechanisms, look at the improvement we got in the test results. Total cholesterol went from 293 to 190. LDL cholesterol went from 226 to 122. LDL-P went from 2,582 to 1,511, which is still high but an incredible reduction. Lp-PLA2 went from 597 to 256, which took it from being high into the normal range. The only marker that didn't change significantly was myeloperoxidase, which is actually a little bit higher on this follow-up test but unlikely to be statistically significantly higher.

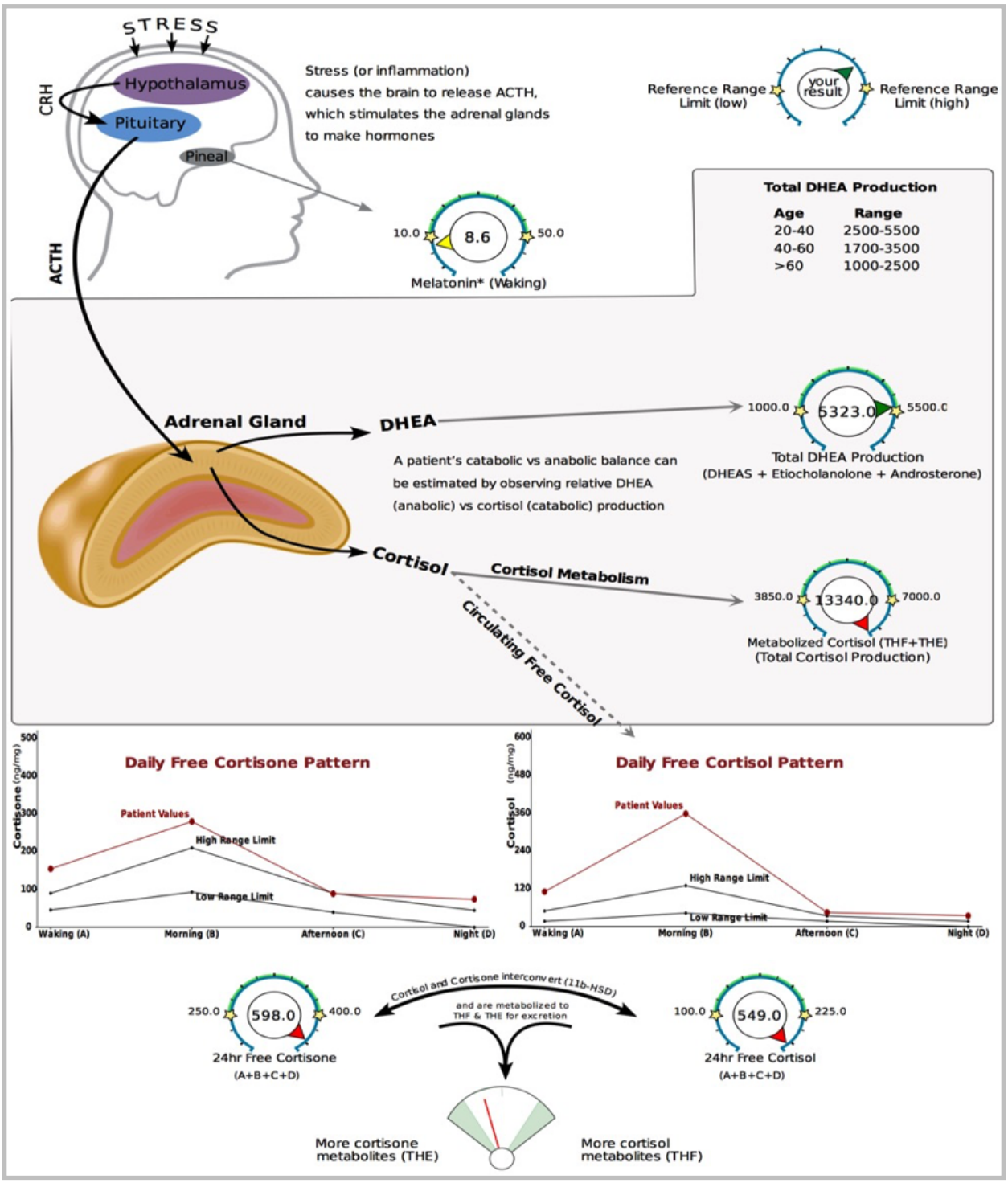
This is one of the most dramatic improvements I've seen from addressing underlying causes versus the last case, which was rare in that markers didn't improve at all. I figured I'd start with giving you the extremes on both ends of the spectrum. Usually you don't see an improvement this big just from addressing underlying causes, and usually you don't see no improvement at all, so these next cases will be more typical of what you would see.

The next patient is a 39-year-old male with a chief complaint of high cholesterol. He is the founder and CEO of a well-known tech company in Silicon Valley. He also had insomnia, fatigue, and high stress, but these weren't his primary concerns, and he viewed them as situational, but they certainly could have been contributing to his lipid numbers.

Tests	Result	Flag	Units	Reference Interval
CMP12+LP+6AC+CBC/D/Plt+PSA+...				
ALT (SGPT)	44		IU/L	0-44
GGT	50		IU/L	0-65
Iron, Serum	89		ug/dL	40-155
Cholesterol, Total	**337**	**High**	mg/dL	100-199
Triglycerides	87		mg/dL	0-149
HDL Cholesterol	72		mg/dL	>39
	According to ATP-III Guidelines, HDL-C >59 mg/dL is considered a negative risk factor for CHD.			
LDL Cholesterol Calc	**248**	**High**	mg/dL	0-99
Comment:				
T. Chol/HDL Ratio	4.7		ratio units	0.0-5.0
				T. Chol/HDL Ratio Men Women 1/2 Avg.Risk 3.4 3.3 Avg.Risk 5.0 4.4 2X Avg.Risk 9.6 7.1 3X Avg.Risk 23.4 11.0
Estimated CHD Risk	0.9		times avg.	0.0-1.0
				T. Chol/HDL Ratio Men Women 1/2 Avg.Risk 3.4 3.3 Avg.Risk 5.0 4.4 2X Avg.Risk 9.6 7.1 3X Avg.Risk 23.4 11.0
	The CHD Risk is based on the T. Chol/HDL ratio. Other factors affect CHD Risk such as hypertension, smoking, diabetes, severe obesity, and family history of premature CHD.			
Homocyst(e)ine, Plasma	11.5		umol/L	0.0-15.0
Prostate Specific Ag, Serum	1.6		ng/mL	0.0-4.0
	Roche ECLIA methodology.			
	According to the American Urological Association, Serum PSA should decrease and remain at undetectable levels after radical prostatectomy. The AUA defines biochemical recurrence as an initial PSA value 0.2 ng/mL or greater followed by a subsequent confirmatory PSA value 0.2 ng/mL or greater. Values obtained with different assay methods or kits cannot be used interchangeably. Results cannot be interpreted as absolute evidence of the presence or absence of malignant disease.			
Free Testosterone(Direct)	8.6		pg/mL	6.8-21.5
DHEA-Sulfate	375.0		ug/dL	71.6-375.4
Estradiol	25.9		pg/mL	7.6-42.6
	Roche ECLIA methodology			
C-Reactive Protein, Cardiac	0.39		mg/L	0.00-3.00
				Relative Risk for Future Cardiovascular Event Low <1.00 Average 1.00 - 3.00

NMR LipoProfile				
LDL-P	**2247**	**High**	nmol/L	<1000
				Low < 1000 Moderate 1000 - 1299 Borderline-High 1300 - 1599 High 1600 - 2000 Very High > 2000
LDL-C	**249**	**High**	mg/dL	0-99
				Optimal < 100 Above optimal 100 - 129 Borderline 130 - 159 High 160 - 189 Very high > 189
	LDL-C is inaccurate if patient is non-fasting.			
HDL-C	71		mg/dL	>39
Triglycerides	91		mg/dL	0-149
Cholesterol, Total	**338**	**High**	mg/dL	100-199
HDL-P (Total)	32.6		umol/L	>=30.5

Total cholesterol is 337. LDL is 248. HDL is 72. Total cholesterol-to-HDL ratio is 4.7, which is well above the 3.0 cut point. LDL-P is 2,247, which puts him in the very-high-risk category, and his homocysteine is also above what I like to see. It's 11.5, so it's in the lab range but above the optimal range. His free testosterone is low, and in men, testosterone is protective against heart disease, and then his DHEA is high, and that's consistent with the stress response.



Not surprisingly, his DUTCH test results were a mess given his stress and job: high 24-hour free cortisol and cortisone, over two times the upper end of the lab range; very high metabolized cortisol; high DHEA; and low melatonin.

Comprehensive Stool Analysis / Parasitology x3

BACTERIOLOGY CULTURE

Expected/Beneficial flora	Commensal (Imbalanced) flora	Dysbiotic flora
3+ Bacteroides fragilis group	2+ Alpha hemolytic strep	
3+ Bifidobacterium spp.	1+ Gamma hemolytic strep	
2+ Escherichia coli	2+ Hemolytic Escherichia coli	
1+ Lactobacillus spp.	1+ Providencia rettgeri	
1+ Enterococcus spp.		
NG 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+ Zygosaccharomyces bailii	

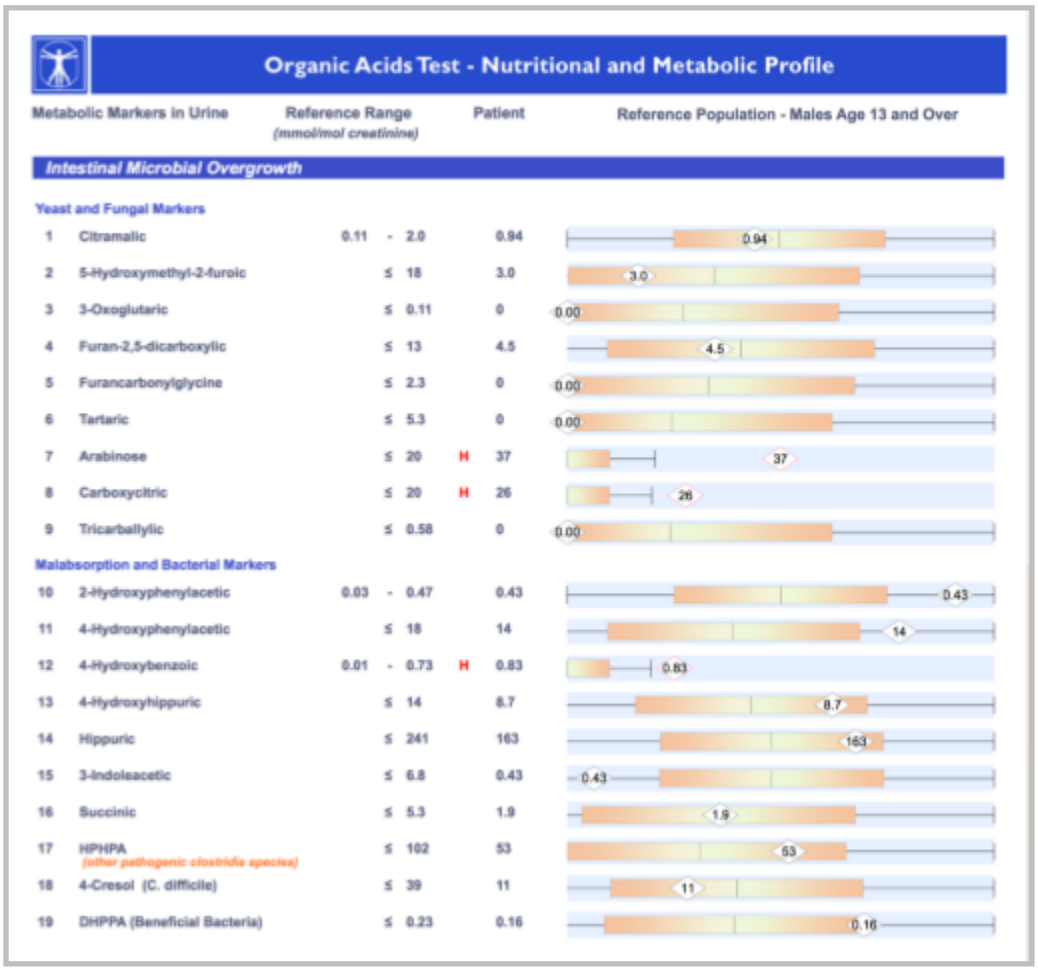
MICROSCOPIC YEAST

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

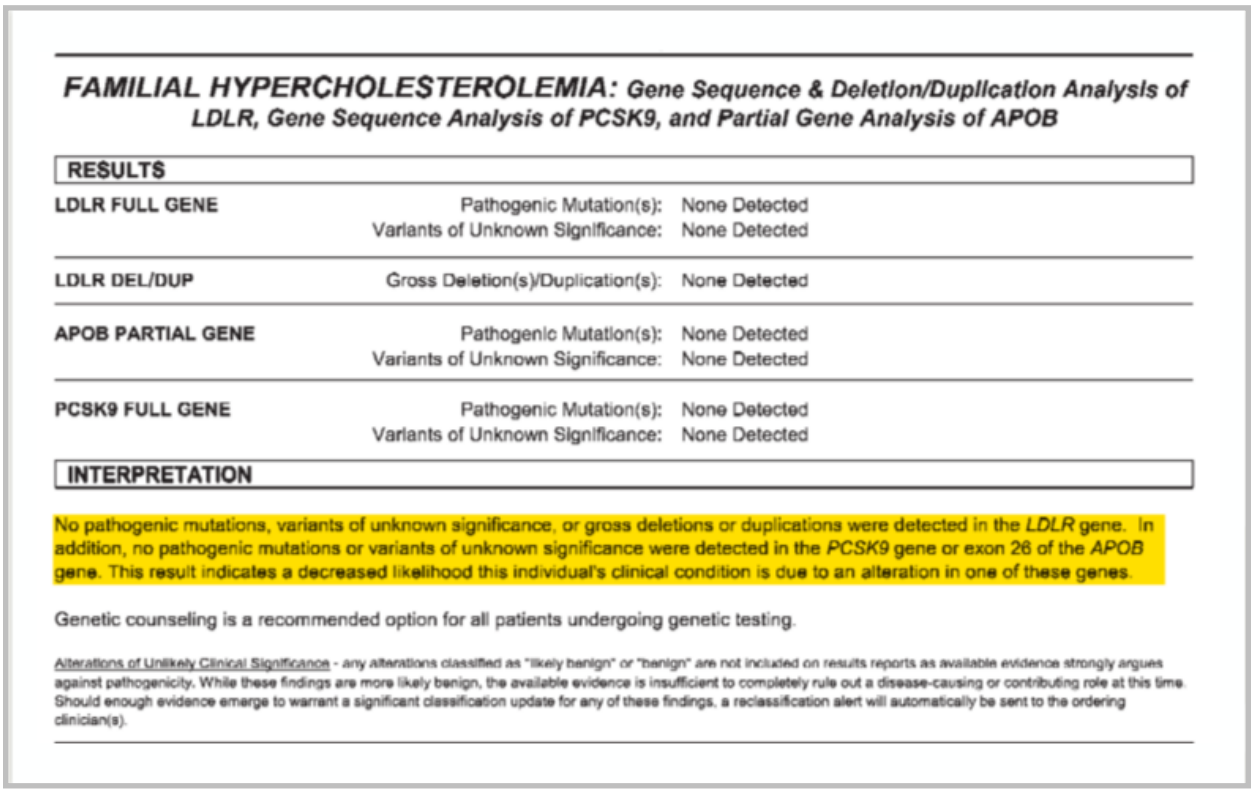
Yeast normally can be found in small quantities in the skin, mouth, intestine and mucocutaneous junctions. Overgrowth of yeast can infect virtually every organ system, leading to an extensive array of clinical manifestations. Fungal diarrhea is associated with broad-spectrum antibiotics or alterations 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.



Organic Acids Test - Nutritional and Metabolic Profile

	Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient
Intestinal Microbial Overgrowth				
Yeast and Fungal Markers				
1	Citramalic	0.11 - 2.0		0.94
2	5-Hydroxymethyl-2-furoic	≤ 18		3.0
3	3-Oxoglutaric	≤ 0.11		0
4	Furan-2,5-dicarboxylic	≤ 13		4.5
5	Furancarbonylglycine	≤ 2.3		0
6	Tartaric	≤ 5.3		0
7	Arabinose	≤ 20	H	37
8	Carboxycitric	≤ 20	H	26
9	Tricarballylic	≤ 0.58		0
Malabsorption and Bacterial Markers				
10	2-Hydroxyphenylacetic	0.03 - 0.47		0.43
11	4-Hydroxyphenylacetic	≤ 18		14
12	4-Hydroxybenzoic	0.01 - 0.73	H	0.83
13	4-Hydroxyhippuric	≤ 14		8.7
14	Hippuric	≤ 241		163
15	3-Indoleacetic	≤ 6.8		0.43
16	Succinic	≤ 5.3		1.9
17	HPHPA (other pathogenic clostridia species)	≤ 102		53
18	4-Cresol (C. difficile)	≤ 39		11
19	DHPPA (Beneficial Bacteria)	≤ 0.23		0.16

Despite no GI symptoms, he had moderate fungal overgrowth and dysbiosis on both Doctor's Data and Great Plains Lab organic acids tests.



***FAMILIAL HYPERCHOLESTEROLEMIA:** Gene Sequence & Deletion/Duplication Analysis of LDLR, Gene Sequence Analysis of PCSK9, and Partial Gene Analysis of APOB*

RESULTS

LDLR FULL GENE	Pathogenic Mutation(s):	None Detected
	Variants of Unknown Significance:	None Detected
LDLR DEL/DUP	Gross Deletion(s)/Duplication(s):	None Detected
APOB PARTIAL GENE	Pathogenic Mutation(s):	None Detected
	Variants of Unknown Significance:	None Detected
PCSK9 FULL GENE	Pathogenic Mutation(s):	None Detected
	Variants of Unknown Significance:	None Detected

INTERPRETATION

No pathogenic mutations, variants of unknown significance, or gross deletions or duplications were detected in the *LDLR* gene. In addition, no pathogenic mutations or variants of unknown significance were detected in the *PCSK9* gene or exon 26 of the *APOB* gene. This result indicates a decreased likelihood this individual's clinical condition is due to an alteration in one of these genes.

Genetic counseling is a recommended option for all patients undergoing genetic testing.

Alterations of Unlikely Clinical Significance - any alterations classified as "likely benign" or "benign" are not included on results reports as available evidence strongly argues against pathogenicity. While these findings are more likely benign, the available evidence is insufficient to completely rule out a disease-causing or contributing role at this time. Should enough evidence emerge to warrant a significant classification update for any of these findings, a reclassification alert will automatically be sent to the ordering clinician(s).

In this case, since money wasn't an issue, the patient wanted genetic testing, and as you can see, no known pathogenic mutations were detected in the LDL receptor, apoB, or PCSK9 gene, yet he almost certainly still has some genetic contributing factor. As I mentioned before, there are many genetic factors that contribute to high LDL-P and Lp(a)-P, and we can't test for them all yet. We still need to address the high LDL-P regardless of whether we know which genes are contributing or not contributing. Again, I question the value of this testing. Certainly it can help some patients to know that they have a genetic susceptibility, and if they are willing to pay for the testing, I don't have a problem with it, but it doesn't often change the treatment plan.

	Laboratory Test	Notes	High Risk	Intermediate Risk	Optimal	High Risk Range	Intermediate Risk Range	Optimal Range	Previous Results
Lipids	Total Cholesterol (mg/dL)		264			≥ 240	200 - 239	< 200	
	LDL-C Direct (mg/dL)		180			≥ 130 CHD & CHD risk eq. > 100	100 - 129 CHD & CHD risk eq. 70 - 100	< 100 CHD & CHD risk eq. < 70	
	HDL-C (mg/dL)				71	< 40		≥ 40	
	Triglycerides (mg/dL)				94	> 199	150 - 199	< 150	
	Non-HDL-C (mg/dL) (calculated)		193			≥ 160	130 - 159	< 130	
Lipoprotein Particles and Apolipoproteins	Apo B (mg/dL)		122			≥ 80	60 - 79	< 60	
	LDL-P (nmol/L)[5], by NMR		2184			≥ 1360	1020 - 1359	< 1020	
	Small LDL-P (nmol/L)[5], by NMR			667		> 1000	501 - 1000	< 501	
	sdLDL-C (mg/dL)[5]		39			> 30	21 - 30	< 21	
	Apo A-I (mg/dL)				175	< 114	114 - 131	> 131	
	HDL-P (µmol/L)[5], by NMR				43.2	≤ 34.0	34.1 - 38.0	> 38.0	
	HDL2-C (mg/dL)[5]				22	≤ 8	9 - 11	≥ 12	
	Apo B:Apo A-I Ratio (calculated)			0.70		≥ 0.81	0.61 - 0.80	≤ 0.60	
	Lp(a)-P (nmol/L)[5]		316			> 125	75 - 125	< 75	
Inflammation/ Oxidation	hs-CRP (mg/L)			1.0		> 2.9	1.0 - 2.9	< 1.0	
	Lp-PLA$_2$ (ng/mL)[5]			328		> 383	291 - 383	< 291	
	Oxidized LDL-β$_2$GPI (U/mL)[5]				< 0.1	≥ 0.2 High Risk	0.1 Moderate Risk	< 0.1 Low Risk	

We addressed the underlying causes and started him on some supplements that can reduce LDL-P and normalize lipid profiles, which we'll talk about in the treatment section, and here are his follow-up results. Total cholesterol went from 338 to 264. LDL-C went from 248 to 180, very big changes. LDL-P did drop a little bit from 2,247 to 2,184 but not nearly as significantly as total cholesterol and LDL cholesterol.

This is, unfortunately, a challenge with currently available treatments, whether we're talking about pharmaceuticals or supplements and botanicals. Statins are much more effective at reducing LDL cholesterol than they are at lowering LDL particle number, especially in some patients, and the same is true for the natural treatments. LDL particle number should be the target of treatment given what we know about it as a risk factor. Unfortunately, many clinicians still treat to LDL-C, which is a mistake.

Notice that his Lp(a) in this case is very high. It was 316 nmol/L, and ideally you want it to be below 75. He also has borderline high C-reactive protein and high Lp-PLA2, so there is some inflammatory process happening here. Along with that high Lp(a) and still high LDL-P, he is still at significantly elevated risk of cardiovascular disease.

The next patient is a 57-year-old male with a sole complaint of high cholesterol and family history of heart disease.

Lipids	Total Cholesterol (mg/dL)		346			≥ 240	200 - 239	< 200
	LDL-C Direct (mg/dL)		228			≥ 130 CHD & CHD risk eq. > 100	100 - 129 CHD & CHD risk eq. 70 - 100	< 100 CHD & CHD risk eq. < 70
	HDL-C (mg/dL)				100	< 40		≥ 40
	Triglycerides (mg/dL)				61	> 199	150 - 199	< 150
	Non-HDL-C (mg/dL) (calculated)		245			≥ 160	130 - 159	< 130
Lipoprotein Particles and Apolipoproteins	Apo B (mg/dL)		156			≥ 80	60 - 79	< 60
	LDL-P (nmol/L)§, by NMR		2433			≥ 1360	1020 - 1359	< 1020
	Small LDL-P (nmol/L)§, by NMR				238	> 1000	501 - 1000	< 501
	sdLDL-C (mg/dL)§		40			> 30	21 - 30	< 21
	Apo A-I (mg/dL)				176	< 114	114 - 131	> 131
	HDL-P (µmol/L)§, by NMR			37.2		≤ 34.0	34.1 - 38.0	> 38.0
	HDL2-C (mg/dL)§				53	≤ 8	9 - 11	≥ 12
	Apo B:Apo A-I Ratio (calculated)		0.89			≥ 0.81	0.61 - 0.80	≤ 0.60
	Lp(a)-P (nmol/L)§		219			> 125	75 - 125	< 75
Inflammation/ Oxidation	Fibrinogen (mg/dL)			442		< 126 or > 517	438 - 517	126 - 437
	hs-CRP (mg/L)				0.4	> 2.9	1.0 - 2.9	< 1.0
	Lp-PLA$_2$ (ng/mL)§		496			> 383	291 - 383	< 291
	Oxidized LDL-β$_2$GPI (U/mL)§				< 0.1	≥ 0.2 High Risk	0.1 Moderate Risk	< 0.1 Low Risk

Renal	Result	Flag	Reference Interval
Microalbumin (urine) (mg albumin/g of creatinine)	7		≤ 29
Creatinine, urine (mg/dL)	76		20 - 400

Anemia	Result	Flag	Reference Interval
Iron (µg/dL)	80		59 - 158
Direct TIBC (µg/dL)	325		250 - 450
Transferrin Saturation (%) (calculated)	24		15 - 50
Ferritin (ng/mL)	226		30 - 400

Others	Result	Flag	Reference Interval
Myeloperoxidase (pmol/L)§	244		< 557

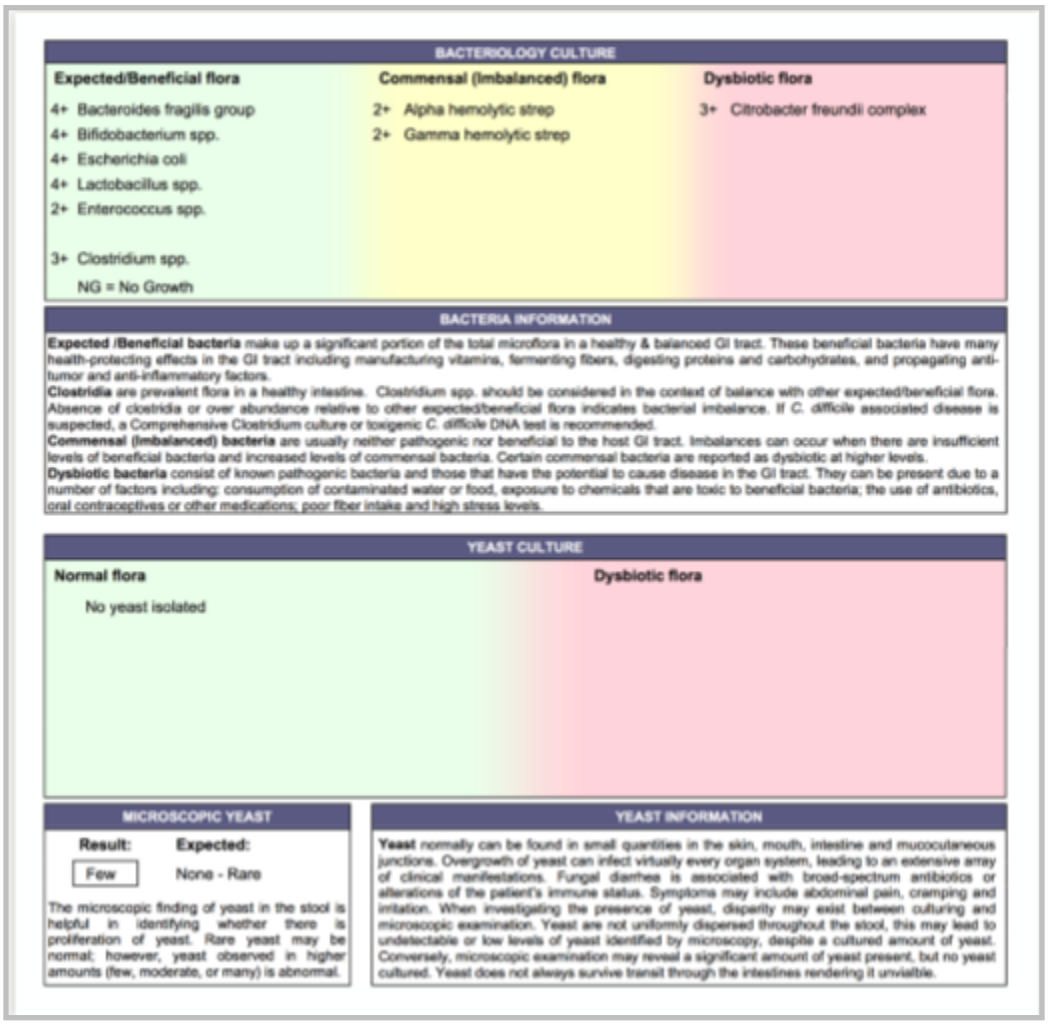
Thyroid	Result	Flag	Reference Interval
TSH (µIU/mL)	3.81		0.27 - 4.20
T4 (µg/dL)	9.4		4.5 - 11.7
T4, free (ng/dL)	1.53		0.93 - 1.70
T3 (ng/dL)	95		80 - 200
T3, free (pg/mL)	2.8		> 19 yrs - 2.0 - 4.4

Total cholesterol is 346. LDL is 228. HDL is very good at 83, but because total cholesterol is so high, the TC-to-HDL ratio is not optimal at 3.5. LDL-P is 2,433, which is very high. Lp(a) is also high at 219, and then Lp-PLA2 is high at 496, and fibrinogen is borderline high at 442. Again, this is a concerning profile, a significant risk because of the very high LDL-P and Lp(a)-P.

You may have noticed that HDL is high, and triglycerides are normal in all of these patients that look like familial hypercholesterolemia patients, and that is typical. This pattern is referred to as

pure hypercholesterolemia, where you have high total cholesterol, high LDL, and then triglycerides and HDL are normal. This is different than dyslipidemia, which usually involves high triglycerides, low HDL, normal or high total cholesterol, and normal or high LDL cholesterol, and that pattern is caused by metabolic dysfunction, whereas FH and pure hypercholesterolemia are often caused by genetics and some of the other things that we mentioned before.

TSH here was slightly elevated at 3.8, but free T3 and free T4 were in the optimal range, so it's unclear whether the thyroid was contributing, but I decided to do a very low dose of natural desiccated thyroid, given the relationship with LDL receptors and cardiovascular disease in general.



BACTERIOLOGY CULTURE

Expected/Beneficial flora	Commensal (Imbalanced) flora	Dysbiotic flora
4+ Bacteroides fragilis group	2+ Alpha hemolytic strep	3+ Citrobacter freundii complex
4+ Bifidobacterium spp.	2+ Gamma hemolytic strep	
4+ Escherichia coli		
4+ Lactobacillus spp.		
2+ 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
No yeast isolated	

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.

Organix

0091 Organix® Comprehensive Profile - Urine

Methodology: LC/Tandem Mass Spectroscopy, Colorimetric

Summary of Abnormal Findings

	Findings	Intervention Options	Common Metabolic Association
Fatty Acid Metabolism			
No Abnormality Found			
Carbohydrate Metabolism			
No Abnormality Found			
Energy Production Markers			
a-Ketoglutarate	Very High	CoQ10, Lipoic Acid, B1, B2, B3, B5	Citric acid cycle
B-Complex Vitamin Markers			
No Abnormality Found			
Methylation Cofactor Markers			
No Abnormality Found			
Neurotransmitter Metabolism Markers			
No Abnormality Found			
Oxidative Damage and Antioxidant Markers			
No Abnormality Found			
Detoxification Indicators			
No Abnormality Found			
Bacterial - General			
Tricarballylate	High	Probiotics	Intestinal Bacterial Overgrowth
L. acidophilus / general bacteria			
No Abnormality Found			
Clostridial Species			
No Abnormality Found			
Yeast/Fungal			
No Abnormality Found			

He had mild fungal overgrowth and dysbiosis on Doctor's Data stool test and on Organix. DUTCH test was normal. No metabolic dysfunction. No metal toxicity. Diet and lifestyle were dialed in. In this case, the cause of hypercholesterolemia was almost certainly genetic.

Laboratory Test		Notes	High Risk	Intermediate Risk	Optimal	High Risk Range	Intermediate Risk Range	Optimal Range	Previous Results 11/16/2015
Lipids	Total Cholesterol (mg/dL)		240			≥ 240	200 - 239	< 200	346
	LDL-C Direct (mg/dL)		142			≥ 130 CHD & CHD risk eq. > 100	100 - 129 CHD & CHD risk eq. 70 - 100	< 100 CHD & CHD risk eq. < 70	228
	HDL-C (mg/dL)				83	< 40		≥ 40	100
	Triglycerides (mg/dL)				64	> 199	150 - 199	< 150	61
	Non-HDL-C (mg/dL) (calculated)			157		≥ 160	130 - 159	< 130	245
Lipoprotein Particles and Apolipoproteins	Apo B (mg/dL)		103			≥ 80	60 - 79	< 60	156
	LDL-P (nmol/L)[1], by NMR		1722			≥ 1360	1020 - 1359	< 1020	2433
	Small LDL-P (nmol/L)[1], by NMR				290	> 1000	501 - 1000	< 501	238
	sdLDL-C (mg/dL)[1]			25		> 30	21 - 30	< 21	40
	Apo A-I (mg/dL)				180	< 114	114 - 131	> 131	176
	HDL-P (µmol/L)[1], by NMR			38.0		≤ 34.0	34.1 - 38.0	> 38.0	37.2
	HDL2-C (mg/dL)[1]				37	≤ 8	9 - 11	≥ 12	53
	Apo B:Apo A-I Ratio (calculated)				0.57	≥ 0.81	0.61 - 0.80	≤ 0.60	0.89
	Lp(a)-P (nmol/L)[1]		143			> 125	75 - 125	< 75	219
Inflammation/ Oxidation	Fibrinogen (mg/dL)				387	< 126 or > 517	438 - 517	126 - 437	442
	hs-CRP (mg/L)				< 0.3	> 2.9	1.0 - 2.9	< 1.0	0.4
	Lp-PLA$_2$ (ng/mL)[1]				251	> 383	291 - 383	< 291	496
	Myeloperoxidase (pmol/L)[1]				205	≥ 332	256 - 331	≤ 255	244
	Oxidized LDL-β$_2$GPI (U/mL)[1]					≥ 0.2 High Risk	0.1 Moderate Risk	< 0.1 Low Risk	< 0.1

Renal	Result	Flag	Reference Interval
Microalbumin (urine) (mg albumin/g of creatinine)	8		≤ 29
Creatinine, urine (mg/dL)	106		20 - 400

Anemia	Result	Flag	Reference Interval
Iron (µg/dL)	70		59 - 158
Direct TIBC (µg/dL)	332		250 - 450
Transferrin Saturation (%) (calculated)	21		15 - 50
Ferritin (ng/mL)	130		30 - 400

Thyroid	Result	Flag	Reference Interval
TSH (µlU/mL)	2.82		0.27 - 4.20
T4 (µg/dL)	7.6		4.5 - 11.7
T4, free (ng/dL)	1.39		0.93 - 1.70
T3 (ng/dL)	90		80 - 200
T3, free (pg/mL)	2.7		> 19 yrs - 2.0 - 4.4

After treatment with supplements to address dyslipidemia, which again we'll discuss in the treatment section, here are his results. Total cholesterol dropped from 346 to 240. LDL cholesterol dropped from 228 to 142. Non-HDL cholesterol dropped from 245 to 157. LDL-P decreased by 30 percent in this case from 2,433 to 1,722, and Lp(a)-P dropped from 219 to 143. The numbers are still high, but these are remarkable changes without pharmaceuticals, especially with Lp(a)-P. There are currently no approved drugs to lower Lp(a)-P, and this is one reason why it's often not measured or tracked. It's not clear why some patients see a drop in Lp(a)-P after addressing underlying causes and using natural treatments while others don't. Note that TSH dropped, but free T4 and T3 didn't increase, so you could consider upping the NDT dose until TSH is optimized to see if that helps even more because it is still a little bit high at 2.8.

Also note that these improvements that we saw in total cholesterol, LDL-C, non-HDL-C, and LDL-P are equivalent or greater than what you might expect to see in statins, depending on the dose, so pretty remarkable.

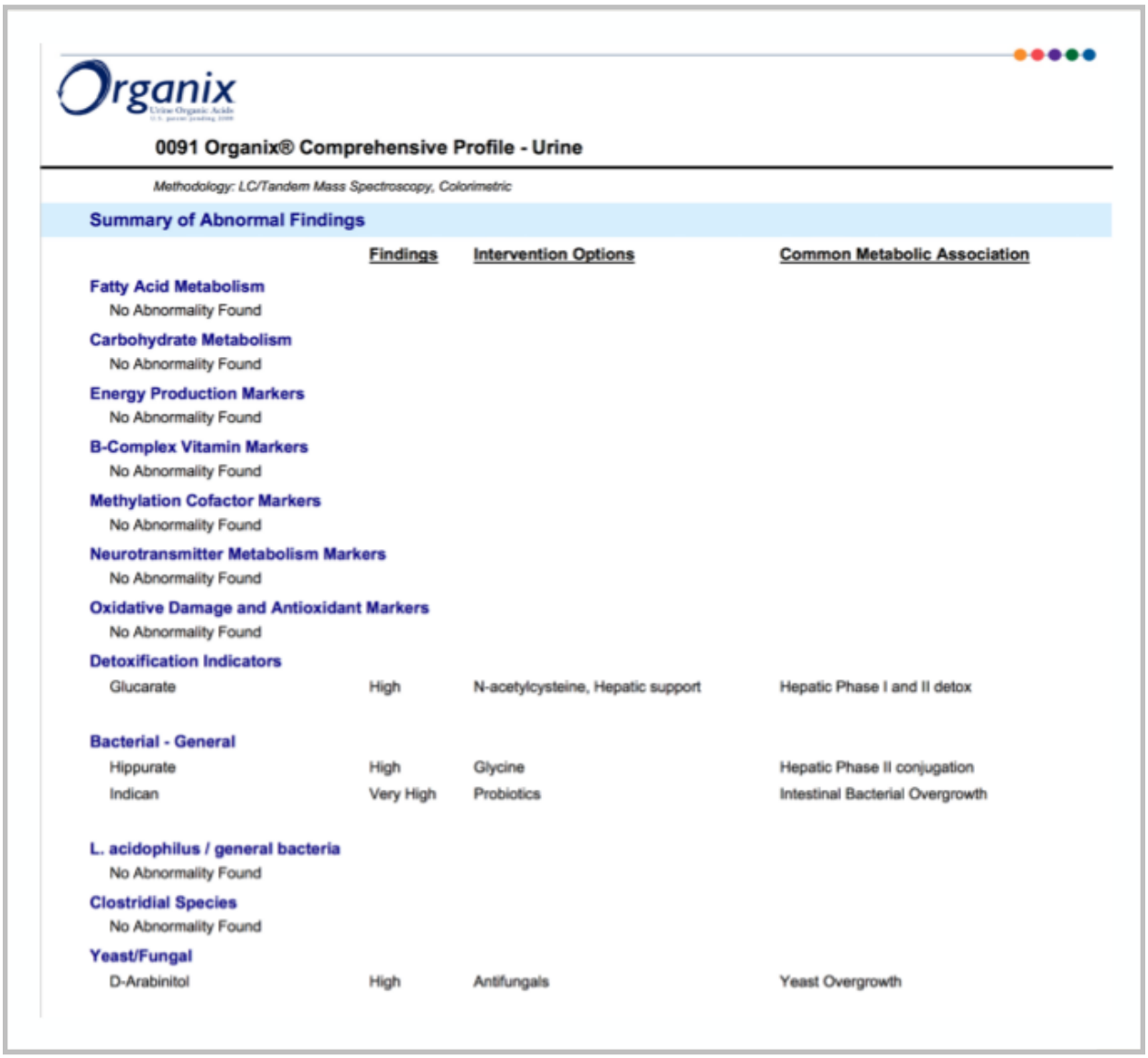
The next patient is a 51-year-old female with chief complaint of mild fatigue, Hashimoto's, eczema, and other skin issues.

TESTS	RESULT	FLAG	UNITS	REFERENCE INTERVAL	LAB
NMR LipoProfile					
LDL Particle Number					01
LDL-P	**2410**	**High**	nmol/L	<1000	01
			Low	< 1000	
			Moderate	1000 - 1299	
			Borderline-High	1300 - 1599	
			High	1600 - 2000	
			Very High	> 2000	
Lipids					01
LDL-C	**277**	**High**	mg/dL	<100	01
LDL-C is inaccurate if patient is nonfasting.					
			Optimal	< 100	
			Above optimal	100 - 129	
			Borderline	130 - 159	
			High	160 - 189	
			Very high	> 189	
HDL-C	100		mg/dL	>=40	01
Triglycerides	**235**	**High**	mg/dL	<150	01
Cholesterol, Total	**424**	**High**	mg/dL	<200	01
LDL and HDL Particles					01
HDL-P (Total)	44.3		umol/L	>= 30.5	01
Small LDL-P	< 90		nmol/L	<= 527	01
LDL Size	22.5		nm	> 20.5	01

TSH+T4F+T3Free			
TSH	4.090	uIU/mL	0.450 - 4.50
Triiodothyronine,Free,Serum	2.3	pg/mL	2.0 - 4.4
T4,Free(Direct)	1.12	ng/dL	0.82 - 1.77

Her LDL-P was 2,410. This is an NMR LipoProfile that you're looking at, so it was before I was using True Health Diagnostics* and testing for Lp(a)-P. TSH is high-normal at 4.0. Free T4 and free T3 are low-normal, so it's possible that thyroid is contributing here.

* **Note:** *True Health Diagnostics is no longer in business. See* **this post** *for the latest updates.*



Organix

Urine Organic Acids

0091 Organix® Comprehensive Profile - Urine

Methodology: LC/Tandem Mass Spectroscopy, Colorimetric

Summary of Abnormal Findings

	Findings	Intervention Options	Common Metabolic Association
Fatty Acid Metabolism			
No Abnormality Found			
Carbohydrate Metabolism			
No Abnormality Found			
Energy Production Markers			
No Abnormality Found			
B-Complex Vitamin Markers			
No Abnormality Found			
Methylation Cofactor Markers			
No Abnormality Found			
Neurotransmitter Metabolism Markers			
No Abnormality Found			
Oxidative Damage and Antioxidant Markers			
No Abnormality Found			
Detoxification Indicators			
Glucarate	High	N-acetylcysteine, Hepatic support	Hepatic Phase I and II detox
Bacterial - General			
Hippurate	High	Glycine	Hepatic Phase II conjugation
Indican	Very High	Probiotics	Intestinal Bacterial Overgrowth
L. acidophilus / general bacteria			
No Abnormality Found			
Clostridial Species			
No Abnormality Found			
Yeast/Fungal			
D-Arabinitol	High	Antifungals	Yeast Overgrowth

Organix comprehensive showed dysbiosis and fungal overgrowth.

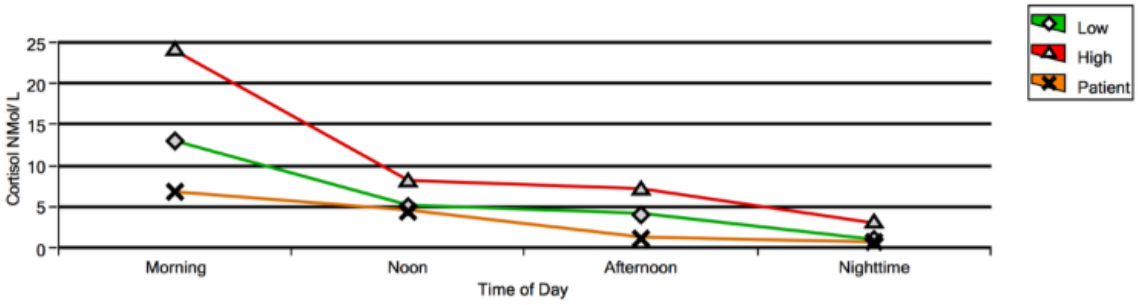
GI Pathogen Screen with H. pylori Antigen - 401H

Parameter	Result
***** Stool Culture *****	
Preliminary Report	Normal flora after 24 hours
Final Report	* Klebsiella species isolated *
Amount of Growth	Light
***** Ova & Parasites *****	
Ova & Parasites #1	No Ova/Parasites detected
Ova & Parasites #2	No Ova/Parasites detected
Ova & Parasites #3	No Ova/Parasites detected
Ova & Parasites #4	No Ova/Parasites detected
Trichrome Stain	No Ova/Parasites detected
***** Stool Antigens *****	
Cryptosporidium Antigen	Not detected
Giardia lamblia Antigen	Not detected
***** Additional Tests *****	
Fungi	Light growth of Aspergillus species isolated
C. difficile Toxin A	Not detected
C. difficile Toxin B	Not detected
Yeast	No yeasts isolated
Occult Blood	Not detected
*****Helicobacter Pylori Stool Antigen*****	
H. pylori Antigen	* Detected *

Fungal overgrowth also showed up on the BioHealth stool test as well as H. pylori, so we've got infections and GI issues as well as thyroid as contributing causes so far.

Functional Adrenal Stress Profile - 201

Parameter	Result	Reference Range	Units
Cortisol - Morning (6 - 8 AM)	**6.8***	13.0 - 24.0	nM/L
Cortisol - Noon (12 - 1 PM)	**4.4***	5.0 - 8.0	nM/L
Cortisol - Afternoon (4 - 5 PM)	**1.1***	4.0 - 7.0	nM/L
Cortisol - Nighttime (10 PM - 12 AM)	**0.7***	1.0 - 3.0	nM/L
Cortisol Sum	**13.0***	23.0 - 42.0	nM/L
DHEA-S Average	**0.35***	2.0 - 10.0	ng/mL
Cortisol/DHEA-S Ratio	**37.2***	5.0 - 6.0	Ratio



The saliva test, which I was running at this point, suggested HPA axis dysregulation with low free cortisol and a blunted cortisol rhythm. We see here several underlying causes of dyslipidemia: poor thyroid function, gut dysbiosis, infection, and HPA axis dysfunction.

TESTS	RESULT	FLAG	UNITS	REFERENCE INTERVAL
NMR LipoProfile				
LDL Particle Number				
LDL-P	**1761**	**High**	nmol/L	<1000
			Low	**< 1000**
			Moderate	**1000 - 1299**
			Borderline-High	**1300 - 1599**
			High	**1600 - 2000**
			Very High	**> 2000**
Lipids				
LDL-C	**187**	**High**	mg/dL	0 - 99
			Optimal	**< 100**
			Above optimal	**100 - 129**
			Borderline	**130 - 159**
			High	**160 - 189**
			Very high	**> 189**
	****Effective October 26, 2015 the reference interval****			
	for LDL-C will be changing to:			
			0 - 19 years	**0 - 109**
			>19 years	**0 - 99**
Comment:				
LDL-C is inaccurate if patient is non-fasting.				
HDL-C	85		mg/dL	>39
Triglycerides	89		mg/dL	0 - 149
	****Effective October 26, 2015 the reference interval****			
	for Triglycerides will be changing to:			
			0 - 9 years	0 - 74
			10 - 19 years	0 - 89
			>19 years	0 - 149
Cholesterol, Total	**290**	**High**	mg/dL	100 - 199

After addressing those mechanisms, LDL-P dropped from 2,400 to the mid-1,700s, which is still a little elevated but a significant and meaningful reduction. The next step in this case would be to use supplements and botanicals to further reduce LDL-P.

The last patient is a 25-year-old male with prediabetes, overweight, and insulin resistance. We don't have follow-up labs on him yet, but I wanted to show you this case to highlight an important principle we discussed earlier, which is that it is possible to have normal and even low total cholesterol and high LDL particle number. This happens most often in patients with metabolic syndrome.

	Laboratory Test	Notes	High Risk	Intermediate Risk	Optimal	High Risk Range	Intermediate Risk Range	Optimal Range	Previous Results
Lipids	Total Cholesterol (mg/dL)				164	≥ 240	200 - 239	< 200	
	LDL-C Direct (mg/dL)			122		≥ 130 CHD & CHD risk eq. > 100	100 - 129 CHD & CHD risk eq. 70 - 100	< 100 CHD & CHD risk eq. < 70	
	HDL-C (mg/dL)		38			< 40		≥ 40	
	Triglycerides (mg/dL)				56	> 199	150 - 199	< 150	
	Non-HDL-C (mg/dL) (calculated)				126	≥ 160	130 - 159	< 130	
Lipoprotein Particles and Apolipoproteins	Apo B (mg/dL)			79		≥ 80	60 - 79	< 60	
	LDL-P (nmol/L)§, by NMR		1505			≥ 1360	1020 - 1359	< 1020	
	Small LDL-P (nmol/L)§, by NMR			859		> 1000	501 - 1000	< 501	
	sdLDL-C (mg/dL)§			22		> 30	21 - 30	< 21	
	Apo A-I (mg/dL)			121		< 114	114 - 131	> 131	
	HDL-P (µmol/L)§, by NMR		31.5			≤ 34.0	34.1 - 38.0	> 38.0	
	HDL2-C (mg/dL)§			9		≤ 8	9 - 11	≥ 12	
	Apo B:Apo A-I Ratio (calculated)			0.65		≥ 0.81	0.61 - 0.80	≤ 0.60	
	Lp(a)-P (nmol/L)§				< 50	> 125	75 - 125	< 75	
Inflammation/ Oxidation	Fibrinogen (mg/dL)				379	< 126 or > 517	438 - 517	126 - 437	
	hs-CRP (mg/L)			1.2		> 2.9	1.0 - 2.9	< 1.0	
	Lp-PLA$_2$ (ng/mL)§				102	> 383	291 - 383	< 291	

If you look at his total cholesterol, it is 164, and if you only were to look at that number, you would get an A-plus. That's a pretty low cholesterol number, but look at the HDL. That's low at 38, which is a sign of metabolic dysfunction. Triglycerides in this case are normal. They would often be high in this kind of metabolic pattern, but for him, they are normal, so don't let that throw you off. If you look at LDL-P, it's 1,500, not super high like some of the 2,500 values we've seen but still elevated. Small LDL-P is in the intermediate-risk category range at 859. HDL-P, HDL particle number, also probably a more important marker for HDL, just as LDL-P is more important than LDL-C, that is low at 31.5. This test really illustrates why it is so important to run LDL particle number, particularly if patient has any evidence of metabolic abnormalities, if they are overweight or have other signs and symptoms of insulin resistance.