DOCTOR'S DATA INC

LAB #:
PATIENT:
ID:
SEX: Male
AGE: 28

CLIENT #:
DOCTOR: , MD
Neurological Research Institute Llc
279 Walkers Mills Rd
Bethel, ME 04217 U.S.A.

Toxic & Essential Elements; Hair

TOXIC METALS		RESULT µg/g	REFERENCE INTERVAL	PERCENTILE 68th 95th
Aluminum	(Al)	10	< 7.0	
Antimony	(Sb)	< 0.01	< 0.066	
Arsenic	(As)	0.026	< 0.080	
Barium	(Ba)	5.2	< 1.0	
Beryllium	(Be)	< 0.01	< 0.020	
Bismuth	(Bi)	0.019	< 2.0	
Cadmium	(Cd)	< 0.009	< 0.065	
Lead	(Pb)	0.26	< 0.80	
Mercury	(Hg)	0.32	< 0.80	
Platinum	(Pt)	< 0.003	< 0.005	
Thallium	(Tl)	0.001	< 0.002	
Thorium	(Th)	< 0.001	< 0.002	
Uranium	(U)	0.60	< 0.060	
Nickel	(Ni)	0.14	< 0.20	
Silver	(Ag)	0.01	< 0.08	
Tin	(Sn)	0.03	< 0.30	
Titanium	(Ti)	0.33	< 0.60	
Total Toxic Representation				

ESSENTIAL AND OTHER ELEMENTS		RESULT µg/g	REFERENCE INTERVAL	PERCENTILE 2.5th 16th 50th 84th 97.5th
Calcium	(Ca)	2450	200- 750	
Magnesium	(Mg)	190	25- 75	
Sodium	(Na)	43	20- 180	
Potassium	(K)	17	9- 80	
Copper	(Cu)	10	11- 30	
Zinc	(Zn)	190	130- 200	
Manganese	(Mn)	0.18	0.08- 0.50	
Chromium	(Cr)	0.45	0.40- 0.70	
Vanadium	(V)	0.078	0.018- 0.065	
Molybdenum	(Mo)	0.045	0.025- 0.060	
Boron	(B)	1.2	0.40- 3.0	
Iodine	(I)	0.62	0.25- 1.8	
Lithium	(Li)	0.025	0.007- 0.020	
Phosphorus	(P)	170	150- 220	
Selenium	(Se)	0.89	0.70- 1.2	
Strontium	(Sr)	21	0.30- 3.5	
Sulfur	(S)	46200	44000- 50000	
Cobalt	(Co)	0.014	0.004- 0.020	
Iron	(Fe)	11	7.0- 16	
Germanium	(Ge)	0.027	0.030- 0.040	
Rubidium	(Rb)	0.012	0.011- 0.12	
Zirconium	(Zr)	1.1	0.020- 0.44	

SPECIMEN DATA

COMMENTS:

Date Collected: 05/13/2016
Date Received: 05/20/2016
Date Completed: 05/23/2016
Methodology: ICP/MS

Sample Size: 0.2 g
Sample Type: Head
Hair Color:
Treatment:
Shampoo: Paul Mitchell

RATIOS

ELEMENTS	RATIOS	RANGE
Ca/Mg	12.9	4- 30
Ca/P	14.4	0.8- 8
Na/K	2.53	0.5- 10
Zn/Cu	19	4- 20
Zn/Cd	> 999	> 800

The Great Plains Laboratory, Inc.

William Shaw, Ph.D., Director | 11813 West 77th Street, Lenexa, KS 66214 | (913) 341-8949 | Fax (913) 341-6207

Requisition #:		**Physician:**	
Patient Name:		**Date of Collection:**	8/18/2016
Patient Age:	29	**Time of Collection:**	12:45 PM
Patient Sex:	M	**Print Date:**	09/[illegible]16

Organic Acids Test - Nutritional and Metabolic Profile

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Value	Reference Population - Males Age 13 and Over
Intestinal Microbial Overgrowth				
Yeast and Fungal Markers				
1 Citramalic	0.11 - 2.0	H	3.4	
2 5-Hydroxymethyl-2-furoic	≤ 18		4.2	
3 3-Oxoglutaric	≤ 0.11	H	0.40	
4 Furan-2,5-dicarboxylic	≤ 13		3.4	
5 Furancarbonylglycine	≤ 2.3		0.54	
6 Tartaric	≤ 5.3	H	8.9	
7 Arabinose	≤ 20	H	155	
8 Carboxycitric	≤ 20		6.8	
9 Tricarballylic	≤ 0.58		0.10	
Bacterial Markers				
10 Hippuric	≤ 241		214	
11 2-Hydroxyphenylacetic	0.03 - 0.47		0.40	
12 4-Hydroxybenzoic	0.01 - 0.73	H	1.0	
13 4-Hydroxyhippuric	≤ 14	H	17	
14 DHPPA (Beneficial Bacteria)	≤ 0.23		0.05	
Clostridia Bacterial Markers				
15 4-Hydroxyphenylacetic *(C. difficile, C. stricklandii, C. lituseburense & others)*	≤ 18		8.2	
16 HPHPA *(C. sporogenes, C. caloritolerans, C. botulinum & others)*	≤ 102		40	
17 4-Cresol *(C. difficile)*	≤ 39		7.0	
18 3-Indoleacetic *(C. stricklandii, C. lituseburense, C. subterminale & others)*	≤ 6.8		2.1	

Testing performed by The Great Plains Laboratory, Inc., Lenexa, Kansas. The Great Plains Laboratory has developed and determined the performance characteristics of this test. This test has not been evaluated by the U.S. FDA; it is used for clinical purposes.

The Great Plains Laboratory, Inc.

Requisition #: *Physician:*

Patient Name: *Date of Collection:*

Metabolic Markers in Urine		Reference Range (mmol/mol creatinine)		Patient Value	Reference Population - Males Age 13 and Over
Oxalate Metabolites					
19	Glyceric	0.21 - 4.9		3.2	
20	Glycolic	18 - 81		46	
21	Oxalic	8.9 - 67	H	450	
Glycolytic Cycle Metabolites					
22	Lactic	0.74 - 19		8.7	
23	Pyruvic	0.28 - 6.7		1.1	
Mitochondrial Markers - Krebs Cycle Metabolites					
24	Succinic	≤ 5.3	H	8.3	
25	Fumaric	≤ 0.49		0.16	
26	Malic	≤ 1.1		0.45	
27	2-Oxoglutaric	≤ 18		0.69	
28	Aconitic	4.1 - 23		11	
29	Citric	2.2 - 260		148	
Mitochondrial Markers - Amino Acid Metabolites					
30	3-Methylglutaric	0.02 - 0.38		0.17	
31	3-Hydroxyglutaric	≤ 4.6		2.2	
32	3-Methylglutaconic	0.38 - 2.0		0.42	
Neurotransmitter Metabolites					
Phenylalanine and Tyrosine Metabolites					
33	Homovanillic (HVA) *(dopamine)*	0.39 - 2.2	H	3.9	
34	Vanillylmandelic (VMA) *(norepinephrine, epinephrine)*	0.53 - 2.2		1.3	
35	HVA / VMA Ratio	0.32 - 1.4	H	3.0	
Tryptophan Metabolites					
36	5-Hydroxyindoleacetic (5-HIAA) *(serotonin)*	≤ 2.9		0.18	
37	Quinolinic	0.52 - 2.4		1.4	
38	Kynurenic	0.12 - 1.8		0.93	
39	Quinolinic / 5-HIAA Ratio	≤ 2.5	H	7.6	

The Great Plains Laboratory, Inc.

Requisition #: *Physician:*

Patient Name: *Date of Collection:*

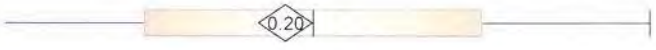
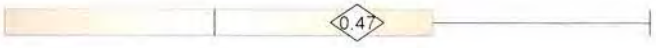
	Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Value	Reference Population - Males Age 13 and Over
Pyrimidine Metabolites - Folate Metabolism					
40	Uracil	≤ 6.9		0.95	
41	Thymine	≤ 0.36		0	
Ketone and Fatty Acid Oxidation					
42	3-Hydroxybutyric	≤ 1.9		1.0	
43	Acetoacetic	≤ 10		0	
44	4-Hydroxybutyric	≤ 4.3		2.9	
45	Ethylmalonic	0.13 - 2.7		1.2	
46	Methylsuccinic	≤ 2.3		1.4	
47	Adipic	≤ 2.9		2.0	
48	Suberic	≤ 1.9	H	9.0	
49	Sebacic	≤ 0.14	H	1.1	
Nutritional Markers					
Vitamin B12					
50	Methylmalonic	≤ 2.3		1.1	
Vitamin B6					
51	Pyridoxic (B6)	≤ 26		12	
Vitamin B5					
52	Pantothenic (B5)	≤ 5.4	H	42	
Vitamin B2 (Riboflavin)					
53	Glutaric	≤ 0.43		0.18	
Vitamin C					
54	Ascorbic	10 - 200		13	
Vitamin Q10 (CoQ10)					
55	3-Hydroxy-3-methylglutaric *	≤ 26		8.3	
Glutathione Precursor and Chelating Agent					
56	N-Acetylcysteine (NAC)	≤ 0.13		0	
Biotin (Vitamin H)					
57	Methylcitric	0.15 - 1.7		1.2	

* [illegible] may indicate a deficiency of this vitamin

The Great Plains Laboratory, Inc.

Requisition #: *Physician:*

Patient Name: *Date of Collection:* 8/18/2016

Metabolic Markers in Urine		Reference Range (mmol/mol creatinine)	Patient Value	Reference Population - Males Age 13 and Over
Indicators of Detoxification				
Glutathione				
58	Pyroglutamic ✱	5.7 - 25	18	
59	2-Hydroxybutyric ✱	≤ 1.2	0.85	
Ammonia Excess				
60	Orotic	≤ 0.46	0.20	
Aspartame, salicylates, or GI bacteria				
61	2-Hydroxyhippuric	≤ 0.86	0.47	
Amino Acid Metabolites				
62	2-Hydroxyisovaleric	≤ 0.41	0	
63	2-Oxoisovaleric	≤ 1.5	0	
64	3-Methyl-2-oxovaleric	≤ 0.56	0	
65	2-Hydroxyisocaproic	≤ 0.39	0.03	
66	2-Oxoisocaproic	≤ 0.34	0	
67	2-Oxo-4-methiolbutyric	≤ 0.14	0.05	
68	Mandelic	≤ 0.09	0	
69	Phenyllactic	≤ 0.10	0.07	
70	Phenylpyruvic	0.02 - 1.4	0.89	
71	Homogentisic	≤ 0.23	0.01	
72	4-Hydroxyphenyllactic	≤ 0.62	0.46	
73	N-Acetylaspartic	≤ 2.5	0.59	
74	Malonic	≤ 9.9	1.9	
Mineral Metabolism				
75	Phosphoric	1 000 - 4 900	3 189	

✱ *A high value for this marker may indicate a Glutathione deficiency.*

76	*Creatinine	94	mg/dL

*The creatinine test is performed to adjust metabolic marker results for differences in fluid intake. Urinary creatinine has limited diagnostic value due to variability as a result of recent fluid intake. Samples are rejected if creatinine is below 20 mg/dL unless the client requests results knowing of our rejection criteria.

Explanation of Report Format

The reference ranges for organic acids were established using samples collected from typical individuals of all ages with no known physiological or psychological disorders. The ranges were determined by calculating the mean and standard deviation (SD) and are defined as ± 2SD of the mean. Reference ranges are age and gender specific, consisting of Male Adult (≥13 years), Female Adult (≥13 years), Male Child (<13 years), and Female Child (<13 years).

There are two types of graphical representations of patient values found in the new report format of both the standard Organic Acids Test and the Microbial Organic Acids Test.

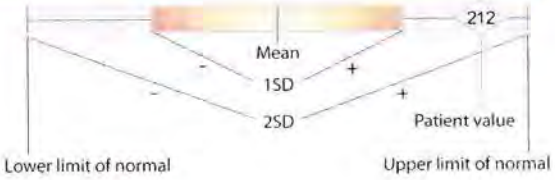
The first graph will occur when the value of the patient is within the reference (normal) range, defined as the mean plus or minus two standard deviations.

The second graph will occur when the value of the patient exceeds the upper limit of normal. In such cases, the graphical reference range is "shrunk" so that the degree of abnormality can be appreciated at a glance. In this case, the lower limits of normal are not shown, only the upper limit of normal is shown.

In both cases, the value of the patient is given to the left of the graph and is repeated on the graph inside a diamond. If the value is within the normal range, the diamond will be outlined in black. If the value is high or low, the diamond will be outlined in red.

Example of Value Within Reference Range

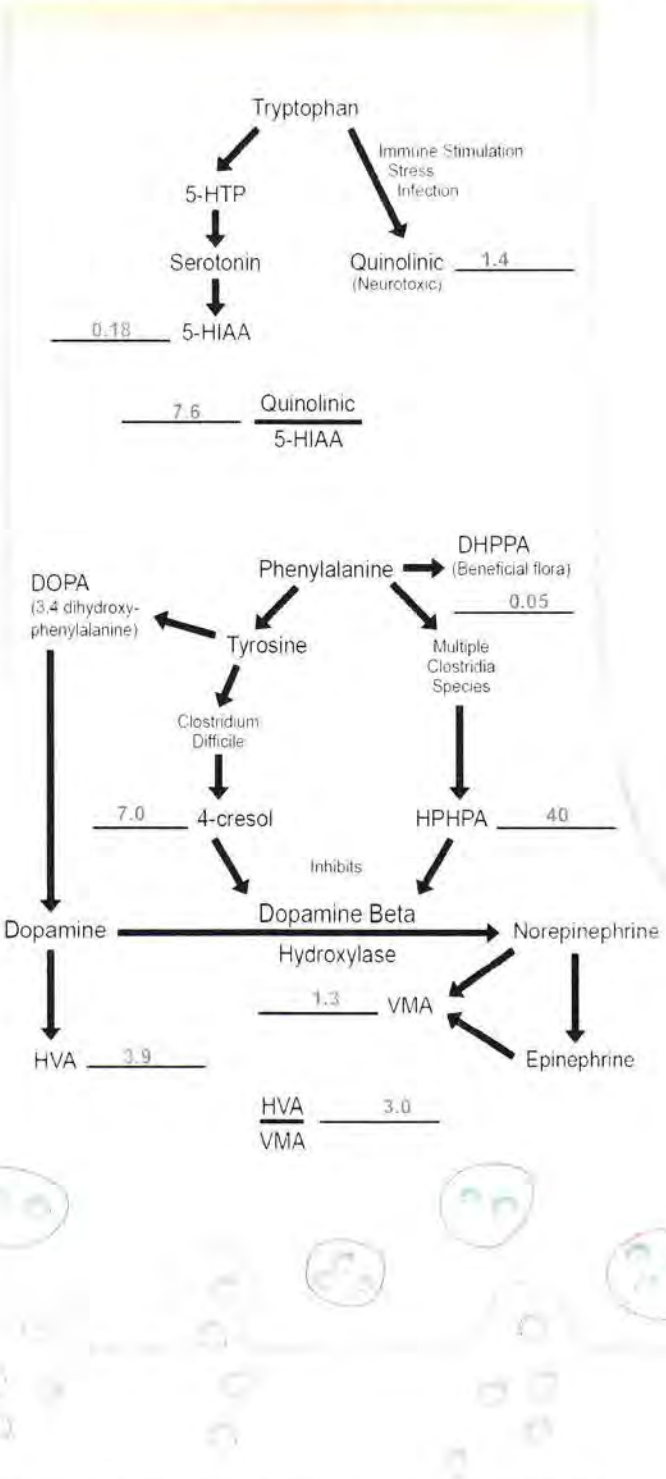
Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Result	Reference Range - Males Age 13 and Under
Intestinal Microbial Overgrowth				
HPHPA (Clostridia marker)	<	219.9	212	



Example of Elevated Value

Metabolic Markers in Urine	Reference Range (mmol/mol creatinine)		Patient Result	Reference Range - Males Age 13 and Under
Intestinal Microbial Overgrowth				
HPHPA (Clostridia marker)	<	219.9	H 3894	

Neurotransmitter Metabolism Markers



The diagram contains the patient's test results for neurotransmitter metabolites and shows their relationship with key biochemical pathways within the axon terminal of nerve cells. The effect of microbial byproducts on the blockage of the conversion of dopamine to norepinephrine is also indicated.

High yeast/fungal metabolites (Markers 1,2,3,4,5,6,7,8) indicate a yeast/fungal overgrowth of the gastrointestinal tract. Prescription or natural (botanical) anti-fungals, along with supplementation of high potency multi-strain probiotics (20-50 billion cfu's), may reduce yeast/fungal levels.

High 4-hydroxybenzoic acid and/or 4-hydroxyhippuric acid (Markers 12,13) may be due to bacterial overgrowth of the GI tract, intake of fruits such as blueberries rich in polyphenols (anthocyanins, flavonols, and hydroxycinnamates), or may be from paraben additive exposure. Parabens are 4-hydroxybenzoic acid alkyl esters with antimicrobial properties. 4-Hydroxybenzoic acid may be excreted as its glycine conjugate 4-hydroxyhippuric acid. High levels of these paraben metabolites in urine (>10 mmol /mol creatinine) may result from excessive exposure to parabens. Parabens are common preservatives allowed in foods, drugs, cosmetics and toiletries, but they also have a long history of use in a variety of pharmaceutical products for injection, inhalation, oral, topical, rectal or vaginal administration. Some individuals experience skin reactions as most parabens are readily and completely absorbed through the skin and the GI tract. Parabens have been considered safe because of their low toxicity profile and their long history of safe use; however, recent studies challenge this view. In 1998, Routledge *et.al.*, (Toxicol.Appl.Pharmacol. **153**,12-19), reported parabens having estrogenic activity *in vitro*. A number of *in vivo* studies have further elucidated potential endocrine disruption by parabens affecting reproduction or promote tumor growth. Parabens have been found at high levels in breast cancer biopsies, although a definitive relationship with breast cancer has not been demonstrated. Parabens may contribute to mitochondrial failure by uncoupling oxidative phosphorylation and depleting cellular ATP. 4-Hydroxyhippuric acid has been found to be an inhibitor of Ca2+- ATPase in end-stage renal failure. Eliminate all sources of parabens. To accelerate paraben excretion, use sauna therapy, the Hubbard detoxification protocol employing niacin supplementation, or glutathione supplementation (oral, intravenous, transdermal, or precursors such as N-acetyl cysteine [NAC]).

High oxalic with or without elevated glyceric or glycolic acids (Markers 19,20,21) may be associated with the genetic hyperoxalurias, autism, women with vulvar pain, fibromyalgia, and may also be due to high vitamin C intake. However, kidney stone formation from oxalic acid was not correlated with vitamin C intake in a very large study. Besides being present in varying concentrations in most vegetables and fruits, oxalates, the mineral conjugate base forms of oxalic acid, are also byproducts of molds such as *Aspergillus* and *Penicillium* and probably *Candida*. If yeast or fungal markers are elevated, antifungal therapy may reduce excess oxalates. High oxalates may cause anemia that is difficult to treat, skin ulcers, muscles pains, and heart abnormalities. Elevated oxalic acid is also the result of anti-freeze (ethylene glycol) poisoning. Oxalic acid is a toxic metabolite of trichloroacetic acid and other environmental pollutants. In addition, decomposing vitamin C may form oxalates during transport or storage.

Elevated oxalate values with a concomitant increase in glycolic acid may indicate genetic hyperoxaluria (type I), whereas increased glyceric acid may indicate a genetic hyperoxaluria (type II). Elevated oxalic acid with normal levels of glyceric or glycolic metabolites rules out a genetic cause for high oxalate. However, elevated oxalates may be due to a new genetic disorder, hyperoxaluria type III.

Regardless of its source, high oxalic acid may contribute to kidney stones and may also reduce ionized calcium. Oxalic acid absorption from the GI tract may be reduced by calcium citrate supplementation before meals. Vitamin B6, arginine, vitamin E, chondroitin sulfate, taurine, selenium, omega-3 fatty acids and/or N-acetyl glucosamine supplements may also reduce oxalates and/or their toxicity. Excessive fats in the diet may cause elevated oxalate if fatty acids are poorly absorbed because of bile salt deficiency. Unabsorbed free fatty acids bind calcium to form insoluble soaps, reducing calcium's ability to bind oxalate and increase its absorption. If taurine is low in a plasma amino acid profile, supplementation with taurine (1000 mg/day) may help stimulate bile salt production (taurocholic acid), leading to better fatty acid absorption and diminished oxalate absorption.

High levels of oxalates are common in autism. Malabsorption of fat and intestinal *Candida* overgrowth are probably the major causes for elevated oxalates in this disorder. Even individuals with elevated glyceric or glycolic acids may not have a genetic disease. To rule out genetic diseases in those people with abnormally high markers characteristic of the genetic diseases, do the following steps: (1) Follow the nutritional steps indicated in this interpretation for one month; (2) If *Candida* is present, treat *Candida* for at least one month; (3) Repeat the organic acid test after abstaining from vitamin C supplements for 48 hours; (4) If the biochemical markers characteristic of genetic oxalate disorders are still elevated in the repeat test, consider DNA tests for the most common mutations of oxalate metabolism. DNA testing for type I hyperoxaluria is available from the Mayo Clinic, Rochester, MN as test #89915 "*AGXT* Gene, Full Gene Analysis" and, for the p.Gly170Arg mutation only, as # 83643 "Alanine:Glyoxylate Aminotransferase [*AGXT*] Mutation Analysis [G170R], Blood"). Another option to confirm the genetic disease is a plasma oxalate test, also available from the Mayo Clinic (Phone 507.266.5700). Plasma oxalate values greater than 50 micromol/L are consistent with genetic oxalate diseases and may serve as an alternate confirmation test.

Bone tends to be the major repository of excess oxalate in patients with primary hyperoxaluria. Bone oxalate levels are negligible in healthy subjects. Oxalate deposition in the skeleton tends to increase bone resorption and decrease osteoblast activity.

Oxalates may also be deposited in the kidneys, joints, eyes, muscles, blood vessels, brain, and heart and may contribute to muscle pain in fibromyalgia. Oxalate crystal formation in the eyes may be a source of severe eye pain in individuals with autism who may exhibit eye-poking behaviors. High oxalates in the GI tract also may significantly reduce absorption of essential minerals such as calcium, magnesium, zinc, and others.

A low oxalate diet may also be particularly useful in the reduction of body oxalates even if dysbiosis of GI flora is the major source of oxalates. Foods especially high in oxalates include spinach, beets, chocolate, soy, peanuts, wheat bran, tea, cashews, pecans, almonds, berries, and many others. A complete list of high oxalate foods is available online at

High succinic acid (Marker 24) may indicate a relative deficiency of riboflavin and/or coenzyme Q10 (cofactors for succinic dehydrogenase in the Krebs cycle). Supplementation with a minimum of 20 mg riboflavin (which could be provided through a high quality multivitamin) and/or 50 mg/day of coenzyme Q10 is recommended. Clinical observation suggests that succinic acid levels also decrease after treatment for GI dysbiosis.

High HVA (Marker 33) may result from toxic metal exposure (including lead, aluminum, manganese, and mercury), presumably due to increased release of dopamine from neurons. Heavy metal testing (blood or hair) might be useful to determine if such exposure is significant. Homovanillic acid (HVA), a dopamine metabolite, is often elevated due to stress-induced catecholamine output from the adrenal gland which depletes vitamin C. Supplementation with vitamin C (ascorbate) may be helpful in such cases.

Elevated HVA may also result from the intake of L-DOPA, dopamine, phenylalanine, or tyrosine. If values are more than double the upper limit of normal, the possibility of catecholamine-secreting tumors can be ruled out by 24- hour VMA and/or HVA testing in urine. Even in this subgroup, the incidence of tumors is extremely rare. High HVA may be associated with *Clostridia* or toxoplasmosis infection. If HVA is elevated and VMA is normal, avoid supplementation with phenylalanine or tyrosine until *Clostridia* or toxoplasmosis is treated.

VMA levels below the mean (Marker 34) may indicate lower production of the neurotransmitter norepinephrine or the hormone adrenaline, perhaps due to low dietary intake of the amino acid precursors phenylalanine or tyrosine. Vanylmandelic acid (VMA) is a metabolite of norepinephrine or adrenaline. Low VMA may also result from blocked conversion of dopamine to norepinephrine by *Clostridia* metabolites. Supplementation with phenylalanine or tyrosine may be beneficial. Enzyme cofactors magnesium, B6 (pyridoxine) or biopterin may also be deficient and respond to supplementation.

High HVA/VMA ratio (Marker 35) The most common reason for an elevation of the HVA/VMA ratio is the decreased conversion of dopamine to norepinephrine and epinephrine. The enzyme responsible for this conversion, dopamine betahydroxylase, is copper and vitamin C dependent, so an elevated ratio could be due to deficiencies of these cofactors. Another common factor is inhibition of this enzyme by *Clostridia* byproducts. A high HPHPA, 4-Cresol, or other elevations of metabolites would be consistent with the latter explanation.

5-hydroxyindoleacetic acid (5-HIAA) levels below the mean (Marker 36) may indicate lower production of the neurotransmitter serotonin. 5-hydroxy-indoleacetic acid is a metabolite of serotonin. Low values have been correlated with symptoms of depression. Supplementation with the precursor 5-HTP (5-hydroxytryptophan) at 50-300 mg/day may be beneficial. Supplementation with tryptophan itself may form the neurotoxic metabolite quinolinic acid, however, 5- HTP is not metabolized to quinolinic acid. Excessive tryptophan supplementation has been associated with eosinophilia myalgia syndrome.

High quinolinic acid / 5-HIAA ratio (Marker 39) indicates an imbalance of these organic acids and may be a sign of neural excitotoxicity. Quinolinic acid is an excitotoxic stimulant of certain brain cells that have NMDA-type receptors. Overstimulated nerve cells may die. Brain toxicity due to quinolinic acid has been implicated in Alzheimer's disease, autism, Huntington's disease, stroke, dementia of old age, depression, HIV-associated dementia, and schizophrenia. However, quinolinic acid is derived from the amino acid tryptophan and is an important intermediate that the body uses to make the essential nutritional cofactor nicotinamide adenine dinucleotide (NAD), which can also be derived from niacin (B3).

An elevated ratio is not specific for a particular medical condition and is commonly associated with excessive inflammation due to recurrent infections. If quinolinic acid is not elevated, low 5- HIAA from serotonin may be the source of the imbalance. Supplementation with 5-HTP may increase serotonin levels, but 5-HTP is not metabolized to quinolinic acid. Immune overstimulation, excess adrenal production of cortisol due to stress, or high exposure to phthalates may also increase the quinolinic acid/5-HIAA acid ratio.

The drug deprenyl or the dietary supplements carnitine, melatonin, capsaicin, turmeric (curcumin) and garlic may reduce brain damage caused by quinolinic acid. Niacin (nicotinic acid) and niacinamide may also reduce quinolinic acid production by decreasing tryptophan shunting to the quinolinic acid pathway. Inositol hexaniacinate as an adult dose of 500-1000 mg does not cause niacin flush.

High ethylmalonic, methylsuccinic, adipic, suberic, or sebacic acids (Markers 45,46,47,48,49) may be due to fatty acid oxidation disorders, carnitine deficiency, fasting, or to increased intake of the medium-chain triglycerides found in coconut oil, MCT oil, and some infant formulas. The fatty acid oxidation defects are associated with hypoglycemia, apnea episodes, lethargy, and coma. [An acyl carnitine profile (Duke University Biochemical Genetics Laboratory, http://medgenetics.pediatrics.duke.edu) can rule out fatty acid oxidation defects.] Regardless of cause, supplementation with L-carnitine or acetyl-L-carnitine (500-1000 mg per day) may be beneficial.

High pantothenic acid (B5) (Marker 52) indicates high recent intake of pantothenic acid. Pantothenic acid is an essential B vitamin. Since some individuals may require very high doses of pantothenic acid, high values do not necessarily indicate the need to reduce pantothenic acid intake.

Ascorbic acid (vitamin C) levels below the mean (Marker 54) may indicate a less than optimum level of the antioxidant vitamin C. Suggested supplementation is 1000 mg/day of buffered vitamin C, divided into 2-3 doses.

Low values for amino acid metabolites (Markers 62-74) indicate the absence of genetic disorders of amino acid metabolism. These markers are deamination (ammonia removed) byproducts that are very elevated only when a key enzyme has low activity; slight elevations may indicate a genetic variation or heterozygous condition which may be mitigated with diet or supplementation. Low values are not associated with inadequate protein intake and have not been proven to indicate specific amino acid deficiencies

High quality nutritional supplements can be purchased through your practitioner or at New Beginnings Nutritionals, , or call 877-575-2467.

Hair test 1200

QUOTE
Once he has given you your unique hair test number forward the below questions through to the **Frequent Dose Chelation group**, inform them of your hair test number and ask for help.
UNQUOTE

from "Mercury Poisoning: The Undiagnosed Epidemic" (p. 312-313). David Hammond. Kindle Edition.

At the time of his **hair test (5/13/16,** see attachment) Martin had just been tested for blood and urine mercury. There was nothing to speak of.

MERCURY, BLOOD **04/29/2016**
MERCURY, BLOOD
Result <4
Range
<OR=10

FASTING:NO

Urine 24-hour test was also negligible
.<2 mcg/L flag range <21 mcg/L

QUESTIONS

• **What are his current symptoms and health history?**

Good health now except for what the psyche meds (negative schizophrenia) do to blood sugar and all the rest, in his case there is tachycardia from clozapine.

Weight is 177, 5'11" tall

Now eating very healthy (soft leafy green salads, beans, hummus, fruit and fruit like veggies). Losing weight slowly as needed. Takes a probiotic (Lactobacillus GG - 30 billion CFU/day)

SYMPTOMS:
negative schizophrenia

• **Dental history (wisdom teeth removed? First root canal placed? Braces? First amalgam etc…**

Plastic(?) braces a few years ago on front upper teeth only, now uses a nighttime retainer.

• What dental work does he currently have in place? What part of the dental cleanup have he completed?

Nighttime retainer only

No amalgams.

• What dentistry did his mother have at any time before or during pregnancy?

She had 4 large molar amalgams at time of birth and while breastfeeding for 2 years afterwards..

• What vaccinations did he have and when (including flu and especially travel shots)?

Had the entire CDC protocol of shots (measles, mumps, diphtheria, etc etc etc) starting as a baby, sometimes multiple shots on same day.

No flu shots or travel shots.

• Supplements and medications (including dosages) taken at time of hair test, or for the 3-6 months before the sample was taken.

Before hair test taking:

clozapine 175 mg/day
olanzapine 15mg, then 7mg, recently 0 mg
metformin 500 mg/day
gemfibrozil 600 mg/day

L-serine 1000-2000 mg/day
D3,
B12
vit C (1-2 g/day)
vit E (400 IE/day)
probiotic Lactobacillus GG 30-45 billion units

After hair test (5/13/16) he added recommended chelation vitamins and minerals, but at lower amounts:

Mg malate 400 mg/day
Zinc citrate 50 mg/day
Selenium 100 mcg/day
Omega 3 (synthetic) 1 g/day
vit B50 complex 1/day

• Other information you feel may be relevant?

Possible sources of mercury poisoning:

Mother's amalgams *(in utero)*
Breastfed for 2 years (mother had amalgams)
Vaccines from birth (1987 to present)

Lead from smoggy air (lives in Los Angeles area)

• What is your location – city & country (so that we can learn where certain toxins are more prevalent).

Since birth living in Orange County, CA, mostly near the beach.