# The Great Plains Laboratory, Inc.

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| | | | |
|---|---|---|---|
| ***Requisition #:*** | 702598 | ***Physician:*** | DAVID JOCKERS |
| ***Patient Name:*** | Ensign Cowell | ***Date of Collection:*** | 06/24/2019 |
| ***Patient Age:*** | 78 | ***Time of Collection:*** | Not Given |
| ***Patient Sex:*** | M | ***Print Date:*** | 07/08/2019 |

## 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 | 2.4 | 2.4 |
| 2 5-Hydroxymethyl-2-furoic | ≤ 18 | | 6.0 | 6.0 |
| 3 3-Oxoglutaric | ≤ 0.11 | | 0 | 0.00 |
| 4 Furan-2,5-dicarboxylic | ≤ 13 | | 5.5 | 5.5 |
| 5 Furancarbonylglycine | ≤ 2.3 | | 1.3 | 1.3 |
| 6 Tartaric | ≤ 5.3 | H | 74 | 74 |
| 7 Arabinose | ≤ 20 | H | 44 | 44 |
| 8 Carboxycitric | ≤ 20 | | 4.3 | 4.3 |
| 9 Tricarballylic | ≤ 0.58 | | 0.13 | 0.13 |
| **Bacterial Markers** | | | | |
| 10 Hippuric | ≤ 241 | H | 304 | 304 |
| 11 2-Hydroxyphenylacetic | 0.03 - 0.47 | | 0.42 | 0.42 |
| 12 4-Hydroxybenzoic | ≤ 0.73 | | 0.41 | 0.41 |
| 13 4-Hydroxyhippuric | ≤ 14 | | 5.7 | 5.7 |
| 14 DHPPA (Beneficial Bacteria) | ≤ 0.23 | | 0.08 | 0.08 |
| **Clostridia Bacterial Markers** | | | | |
| 15 4-Hydroxyphenylacetic *(C. difficile, C. stricklandii, C. lituseburense & others)* | ≤ 18 | | 13 | 13 |
| 16 HPHPA *(C. sporogenes, C. caloritolerans, C. botulinum & others)* | ≤ 102 | | 32 | 32 |
| 17 4-Cresol *(C. difficile)* | ≤ 39 | | 6.9 | 6.9 |
| 18 3-Indoleacetic *(C. stricklandii, C. lituseburense, C. subterminale & others)* | ≤ 6.8 | | 1.1 | 1.1 |

***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; the FDA does not currently regulate such testing.***

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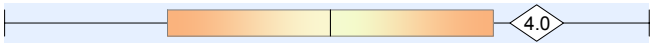
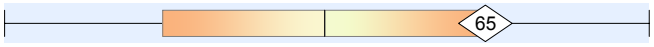
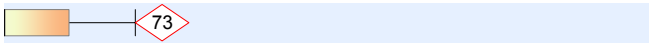
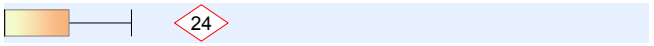
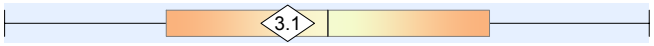
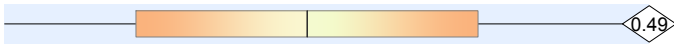
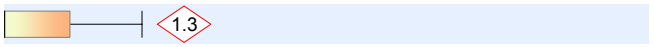
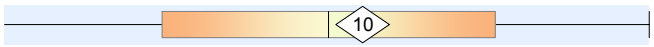
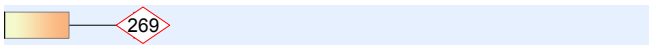
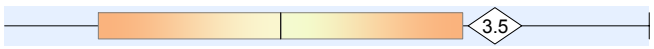
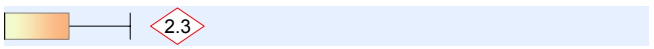
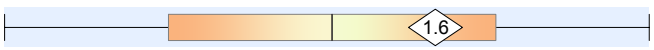
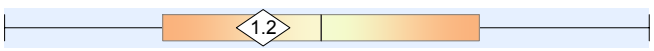
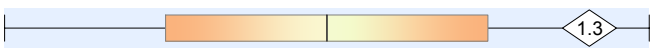
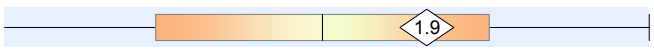
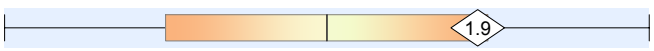
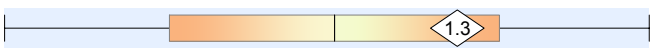
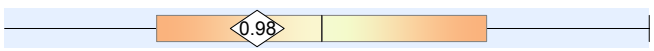
| ***Patient Name:*** | Ensign Cowell | ***Date of Collection:*** | 06/24/2019 |

| 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 | | 4.0 |   |
| 20 | Glycolic | 18 - 81 | | 65 |   |
| 21 | Oxalic | 8.9 - 67 | H | 73 |   |
| **Glycolytic Cycle Metabolites** | | | | | |
| 22 | Lactic | 0.74 - 19 | H | 24 |   |
| 23 | Pyruvic | 0.28 - 6.7 | | 3.1 |   |
| **Mitochondrial Markers - Krebs Cycle Metabolites** | | | | | |
| 24 | Succinic | ≤ 5.3 | | 1.2 |   |
| 25 | Fumaric | ≤ 0.49 | | 0.49 |   |
| 26 | Malic | ≤ 1.1 | H | 1.3 |   |
| 27 | 2-Oxoglutaric | ≤ 18 | | 10 |   |
| 28 | Aconitic | 4.1 - 23 | | 14 |   |
| 29 | Citric | 2.2 - 260 | H | 269 |   |
| **Mitochondrial Markers - Amino Acid Metabolites** | | | | | |
| 30 | 3-Methylglutaric | 0.02 - 0.38 | H | 0.63 |   |
| 31 | 3-Hydroxyglutaric | ≤ 4.6 | | 3.5 |   |
| 32 | 3-Methylglutaconic | 0.38 - 2.0 | H | 2.3 |   |
| **Neurotransmitter Metabolites** | | | | | |
| **Phenylalanine and Tyrosine Metabolites** | | | | | |
| 33 | Homovanillic (HVA) *(dopamine)* | 0.39 - 2.2 | | 1.6 |   |
| 34 | Vanillylmandelic (VMA) *(norepinephrine, epinephrine)* | 0.53 - 2.2 | | 1.2 |   |
| 35 | HVA / VMA Ratio | 0.32 - 1.4 | | 1.3 |   |
| **Tryptophan Metabolites** | | | | | |
| 36 | 5-Hydroxyindoleacetic (5-HIAA) *(serotonin)* | ≤ 2.9 | | 1.9 |   |
| 37 | Quinolinic | 0.52 - 2.4 | | 1.9 |   |
| 38 | Kynurenic | 0.12 - 1.8 | | 1.3 |   |
| 39 | Quinolinic / 5-HIAA Ratio | ≤ 2.5 | | 0.98 |   |

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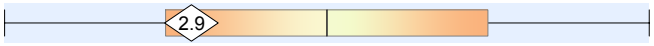
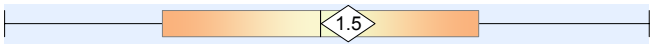
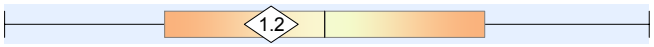
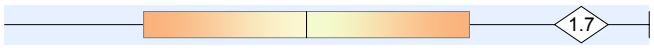
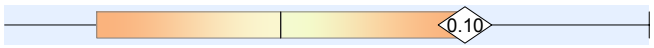
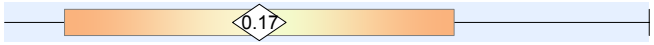
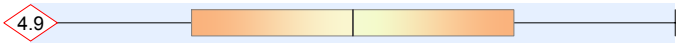
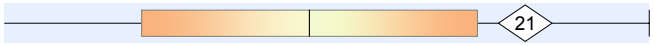
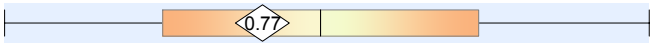
| ***Requisition #:*** | 702598 | ***Physician:*** | DAVID JOCKERS |
| ***Patient Name:*** | Ensign Cowell | ***Date of Collection:*** | 06/24/2019 |

| 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 | | 2.3 |   |
| 41 | Thymine | ≤ 0.36 | | 0.14 |   |
| **Ketone and Fatty Acid Oxidation** | | | | | |
| 42 | 3-Hydroxybutyric | ≤ 1.9 | H | 3.4 |   |
| 43 | Acetoacetic | ≤ 10 | | 2.9 |   |
| 44 | 4-Hydroxybutyric | ≤ 4.3 | | 3.3 |   |
| 45 | Ethylmalonic | 0.13 - 2.7 | | 1.5 |   |
| 46 | Methylsuccinic | ≤ 2.3 | | 1.3 |   |
| 47 | Adipic | ≤ 2.9 | | 1.2 |   |
| 48 | Suberic | ≤ 1.9 | | 1.7 |   |
| 49 | Sebacic | ≤ 0.14 | | 0.10 |   |
| **Nutritional Markers** | | | | | |
| **Vitamin B12** | | | | | |
| 50 | Methylmalonic ✱ | ≤ 2.3 | | 1.3 |   |
| **Vitamin B6** | | | | | |
| 51 | Pyridoxic (B6) | ≤ 26 | | 2.3 |   |
| **Vitamin B5** | | | | | |
| 52 | Pantothenic (B5) | ≤ 5.4 | | 2.1 |   |
| **Vitamin B2 (Riboflavin)** | | | | | |
| 53 | Glutaric ✱ | ≤ 0.43 | | 0.17 |   |
| **Vitamin C** | | | | | |
| 54 | Ascorbic | 10 - 200 | L | 4.9 |   |
| **Vitamin Q10 (CoQ10)** | | | | | |
| 55 | 3-Hydroxy-3-methylglutaric ✱ | ≤ 26 | | 21 |   |
| **Glutathione Precursor and Chelating Agent** | | | | | |
| 56 | N-Acetylcysteine (NAC) | ≤ 0.13 | | 0 |   |
| **Biotin (Vitamin H)** | | | | | |
| 57 | Methylcitric ✱ | 0.15 - 1.7 | | 0.77 |   |

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

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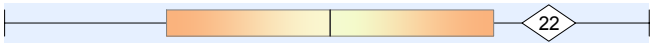
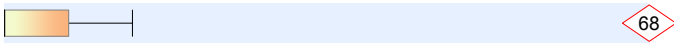
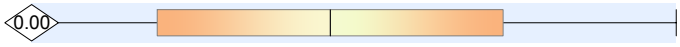
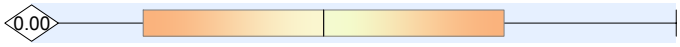
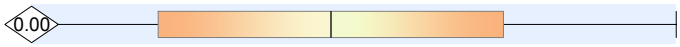
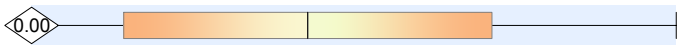
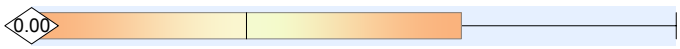
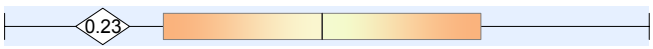
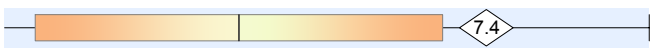
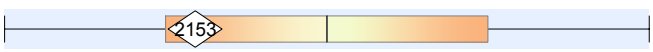
| ***Patient Name:*** | Ensign Cowell | ***Date of Collection:*** | 06/24/2019 |

| 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 | | 22 |   |
| 59 | 2-Hydroxybutyric ✱ | ≤ 1.2 | H | 2.4 |   |
| **Ammonia Excess** | | | | | |
| 60 | Orotic | ≤ 0.46 | | 0.22 |   |
| **Aspartame, salicylates, or GI bacteria** | | | | | |
| 61 | 2-Hydroxyhippuric | ≤ 0.86 | H | 68 |   |

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

| | | | | | |
|---|---|---|---|---|---|
| **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 |   |
| 66 | 2-Oxoisocaproic | ≤ 0.34 | | 0.21 |   |
| 67 | 2-Oxo-4-methiolbutyric | ≤ 0.14 | | 0 |   |
| 68 | Mandelic | ≤ 0.09 | | 0 |   |
| 69 | Phenyllactic | ≤ 0.10 | | 0 |   |
| 70 | Phenylpyruvic | 0.02 - 1.4 | | 0.23 |   |
| 71 | Homogentisic | ≤ 0.23 | | 0.05 |   |
| 72 | 4-Hydroxyphenyllactic | ≤ 0.62 | | 0.38 |   |
| 73 | N-Acetylaspartic | ≤ 2.5 | | 0.70 |   |
| 74 | Malonic | ≤ 9.9 | | 7.4 |   |
| **Mineral Metabolism** | | | | | |
| 75 | Phosphoric | 1,000 - 4,900 | | 2,153 |   |

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| *Patient Name:* | Ensign Cowell | *Date of Collection:* | 06/24/2019 |

## *Indicator of Fluid Intake*

| | | | |
|---|---|---|---|
| 76 | *Creatinine | 69 | 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.

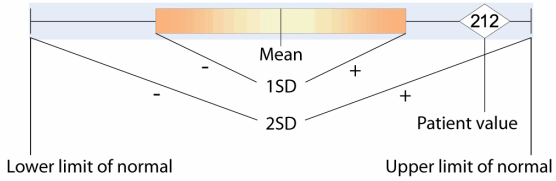
The first graph will occur when the value of the patient is within the reference (normal) range, defined as the mean plus or minus two standard deviations.

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.

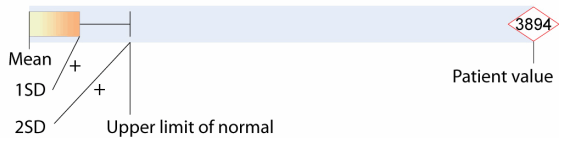
### Example of Value Within Reference Range

| 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 | |

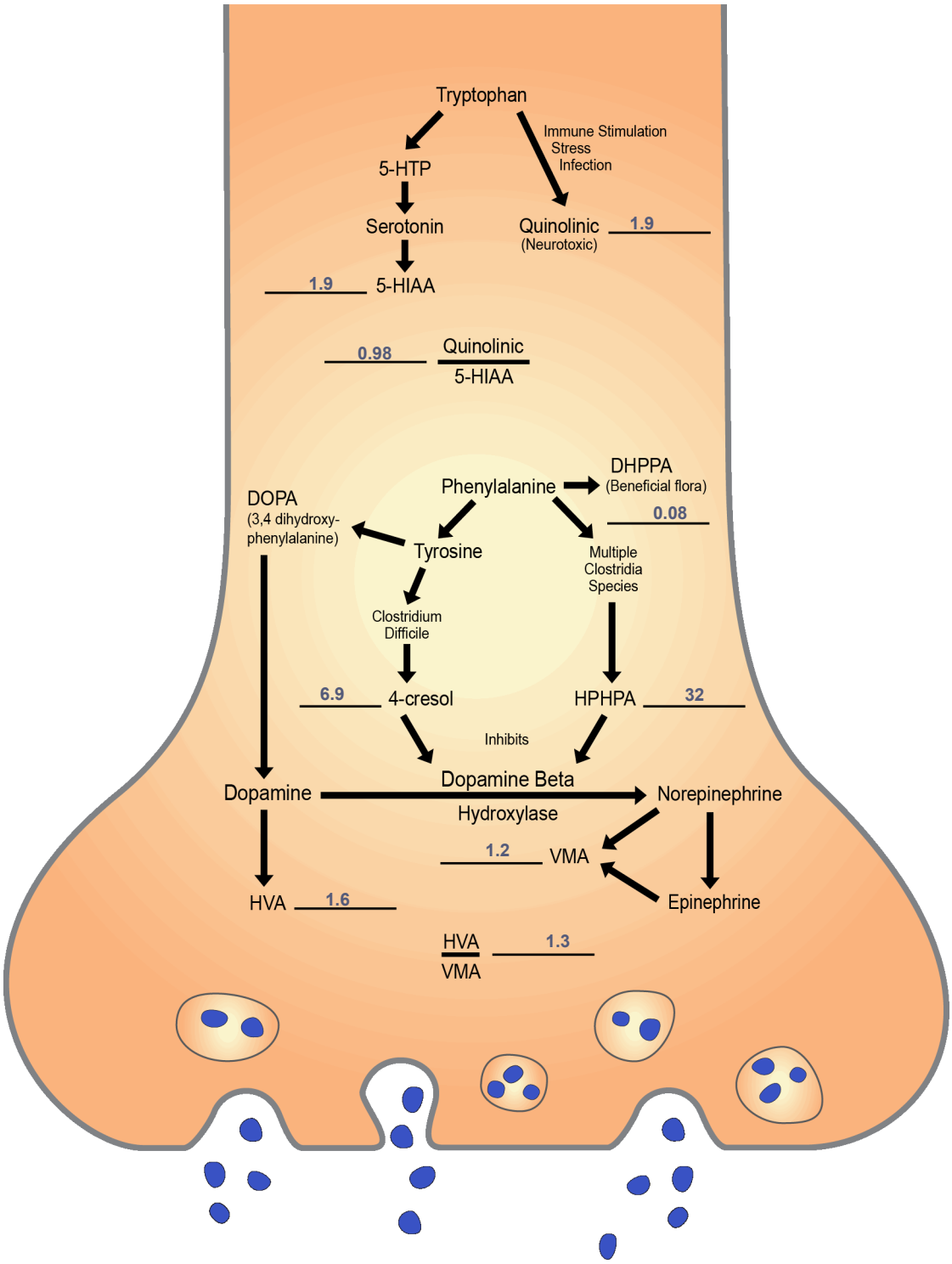



### Example of Elevated Value

| 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.

## Interpretation

***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 hippuric acid (Marker 10)*** may derive from food, GI bacterial activity, or exposure to the solvent toluene. Hippuric acid is a conjugate of glycine and benzoic acid formed in the liver. Most hippuric acid in urine is derived from microbial breakdown of chlorogenic acid to benzoic acid. Chlorogenic acid is a common substance in beverages and in many fruits and vegetables, including apples, pears, tea, coffee, sunflower seeds, carrots, blueberries, cherries, potatoes, tomatoes, eggplant, sweet potatoes, and peaches. Benzoic acid is present in high amounts in cranberry juice and is a food preservative. The workplace is the most common source of toluene exposure, but toluene may be absorbed from outgassing of new carpets and other building materials, or absorbed during recreational abuse of solvents such as glue-sniffing. Because most hippuric acid in urine is from GI sources, this marker is a poor indicator of toluene exposure and is being replaced by other markers in occupational safety testing. Bacterial overgrowth can be treated with natural anti-bacterial agents and/or probiotics (30-50 billion cfu's) that include *Lactobacillus rhamnosus*.

***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 <http://www.greatplainslaboratory.com/home/eng/oxalates.asp>.

***High lactic acid and/or high pyruvic acid (Markers 22,23)*** may be caused by many nonspecific factors, such as vigorous exercise, bacterial overgrowth of the GI tract, shock, poor perfusion, anemia, mitochondrial dysfunction or damage, and many other causes. Conversion of pyruvic acid to acetyl-CoA requires the cofactors coenzyme A (derived from pantothenic acid), lipoic acid, FAD derived from riboflavin, and thiamine. However, the possibility of an inborn error of metabolism increases as the value exceeds 300 mmol/mol creatinine. Values greater than 1000 mmol/mol creatinine indicate a much higher likelihood of an inborn error of metabolism. There are many inborn errors of metabolism that present elevated lactic acid, including disorders of sugar metabolism and pyruvate dehydrogenase deficiency.

***High malic acid (Marker 26)*** indicates a greater requirement for nutrients such as niacin (25-50mg) and coenzyme Q-10 (300-600mg). If malic acid is simultaneously elevated with citric, fumaric and alpha-ketoglutaric acids, a possible Cytochrome C Oxidase deficiency would strongly indicate mitochondrial energy pathway dysfunction.

***High citric acid (Marker 29)*** may be due to increased intake of citric acid-containing foods or as a result of intestinal yeast that either produce citric acid or perhaps inhibit the human citric acid cycle. Increased citric acid may also indicate depletion of glutathione, which is required for the enzyme aconitase to metabolize both aconitic and citric acids. If pyroglutamic acid is also high, consider supplements of reduced glutathione, n-acetyl cysteine (NAC), or lipoic acid.

***High 3-methylglutaric and/or high 3-methylglutaconic acids (Markers 30,32 )*** may be due to reduced capacity to metabolize the amino acid leucine. This abnormality is found in the genetic disease methylglutaconic aciduria and in mitochondrial disorders in which there are severe deficiencies of the respiratory complexes (Complex I, NADH ubiquinone oxidoreductase and complex IV, cytochrome c oxidase.). Small elevations may be due to impairment of mitochondrial function and may respond to the recommended supplements below. Typical results found in genetic defects are above 10 mmol/mol creatinine. A few non-genetic conditions including pregnancy and kidney failure may also produce elevation of these organic acids in urine. Confirmation of the genetic disease requires enzymes and/or DNA testing. Multiple genetic defects can cause the biochemical abnormality. Confirmation of mitochondrial disorder usually requires tissue biopsy for mitochondria testing. Symptoms differ within different types of genetic disorders, but in severe cases may include speech delay, delayed development of both mental and motor skills (psychomotor delay), metabolic acidosis, abnormal muscle tone (dystonia), and spasms and weakness affecting the arms and legs (spastic quadriparesis). Recommendations include supplementation with coenzyme Q-10 (300-600 mg), NAD 25-50mg, L-carnitine and acetyl-L-carnitine (1000-2000 mg), riboflavin (40-80 mg), nicotinamide (40-80 mg), biotin (4-8 mg), and vitamin E (200-400 IU's) per day.

***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 3-hydroxybutyric and/or acetoacetic acids (Markers 42, 43)*** indicate increased metabolic utilization of fatty acids. These ketones are associated with diabetes mellitus, fasting, dieting (ketogenic or SCD diet), or illness such as nausea or flu, among many other causes. Regardless of cause, supplementation with L-carnitine or acetyl-L-carnitine (500-1000mg per day) may be beneficial.

***Pyridoxic acid (B6) levels below the mean (Marker 51)*** may be associated with less than optimum health conditions (low intake, malabsorption, or dysbiosis). Supplementation with B6 (20 - 50 mg/day) or a multivitamin may be beneficial.

***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.

***High 2-hydroxybutyric acid (Marker 59)*** This organic acid is elevated when there is increased production of sulfur amino acids derived from homocysteine. The reasons for an increase can be due to the following reasons (which are not mutually exclusive):

1. There is increased need for glutathione to detoxify a host of toxic chemicals, resulting in increased shunting of homocysteine into the production of cysteine for glutathione. This is the most common reason.
2. There are genetic variants of the DNA such that methylation of homocysteine by betaine homocysteine methyl transferase or methionine synthase is impaired.
3. There are nutritional deficiencies of betaine, methylcobalamin, or methyltetrahydrofolate that reduce the enzyme activities of the enzymes in #2 above.
4. There is a genetic variant in cystathionine beta synthase (CBS) enzyme such that there is excessive shunting of homocysteine into cysteine production that results in excessive 2-hydroxybutyric acid formation.

***High 2-hydroxyhippuric acid (Marker 61)*** may result after ingestion of aspartame (Nutrasweet®) or salicylates (aspirin), or from GI bacteria converting tyrosine or phenylalanine to salicylic acid. 2-Hydroxyhippuric acid is a conjugate of hydroxybenzoic acid (salicylic acid) and glycine.

***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, www.NBNUS.com <http://www.NBNUS.com> , or call 877-575-2467.**

***The nutritional recommendations in this test are not approved by the US FDA. Supplement recommendations are not intended to treat, cure, or prevent any disease and do not take the place of medical advice or treatment from a healthcare professional.***