



Patient: **SAMPLE**
REPORT

DOB: November 1, 1966
Sex: M

Order Number: A2123456

Completed: June 17, 2008
Received: June 11, 2008

Genova Diagnostics Europe
Referring Laboratory
Parkgate House
356 West Barnes Lane
New Malden, Surrey KT3 6NB
Great Britain and Northern Ireland

Malabsorption and Dysbiosis Markers

Reference Range

Malabsorption Markers		mmol/mol creatinine
1. Indoleacetic Acid (IAA)	2.1	<= 9.0
2. Phenylacetic Acid (PAA)	<dl	<= 0.0
3. Dihydroxyphenylpropionic Acid (DHPPA)	0.8	<= 2.2
4. Succinic Acid	4.6	<= 20.0

Bacterial Dysbiosis Markers		mmol/mol creatinine
5. Citramalic Acid	2.8	<= 7.0
6. Indoleacetic Acid (IAA)	2.1	<= 9.0
7. Phenylacetic Acid (PAA)	<dl	<= 0.0
8. Dihydroxyphenylpropionic Acid (DHPPA)	0.8	<= 2.2
9. Benzoic / Hippuric Acids Ratio	0.00	<= 0.02
10. Succinic Acid	4.6	<= 20.0

Yeast / Fungal Dysbiosis Markers		mmol/mol creatinine
11. Arabinose	16.7	<= 42.3
12. Tartaric Acid	<dl	<= 14.1
13. Citramalic Acid	2.8	<= 7.0

Neurotransmitter Metabolites

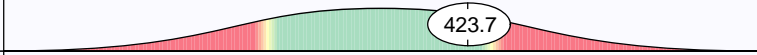


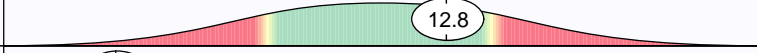
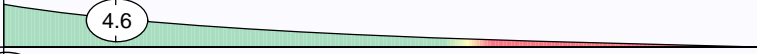
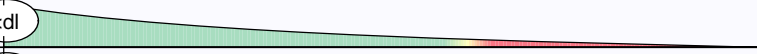
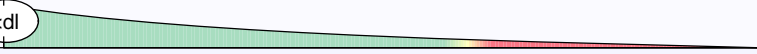
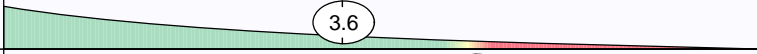
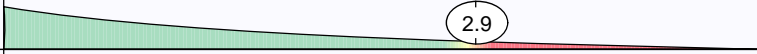
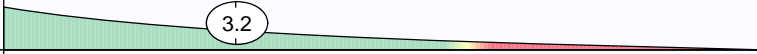
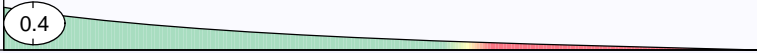
		mmol/mol creatinine
14. Vanilmandelic Acid (VMA)	1.8	1.2-5.9
15. Homovanillic Acid (HVA)	4.0	0.9-4.4
16. 3-Methyl-4-OH-phenylglycol (MHPG)	4.3	<= 16.7
17. 5-OH-Indoleacetic Acid (5-HIAA)	1.6	1.1-6.5

Cellular Energy and Mitochondrial Metabolites

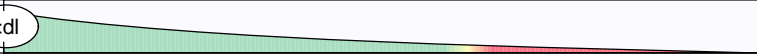


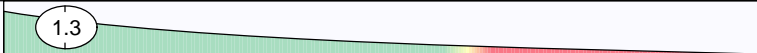

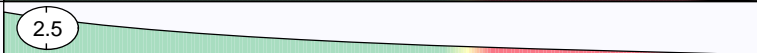
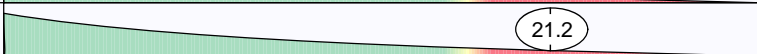
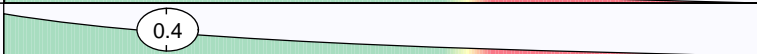




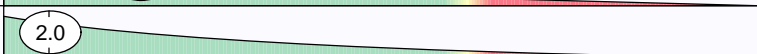
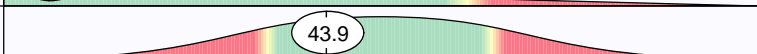
Glycolysis Metabolites		mmol/mol creatinine
18. Lactic Acid	5.6	6.3-36.4
19. Pyruvic Acid	9.1	1.1-15.4

Cellular Energy and Mitochondrial Metabolites cont.

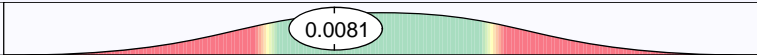
Reference Range

Citric Acid Cycle Metabolites		mmol/mol creatinine
20. Citric Acid		21.9-475.1
21. Cis-Aconitic Acid		1.4-76.8
22. Isocitric Acid		3.7-87.4
23. a-Ketoglutaric Acid (AKA)		0.5-16.0
24. Succinic Acid		<= 20.0
25. Fumaric Acid		<= 1.4
26. Malic Acid		<= 2.4
Ketone and Fatty Acid Metabolites		mmol/mol creatinine
27. Adipic Acid		<= 5.2
28. Suberic Acid		<= 3.0
29. b-OH-b-Methylglutaric Acid (HMG)		<= 6.7
30. b-OH-Butyric Acid (BHBA)		<= 6.4

Organic Acids for Cofactor Need

		mmol/mol creatinine
31. a-Ketoisovaleric Acid (AKIV)		<= 2.0
32. a-Ketoisocaproic Acid (AKIC)		<= 2.0
33. a-Keto-b-Methylvaleric Acid (AKBM)		<= 2.0
34. Kynurenic Acid		<= 10.0
35. Formiminoglutamic Acid (FIGlu)		<= 9.0
36. 3-Hydroxyproprionic Acid (3HPA)		<= 27.5
37. Methylmalonic Acid (MMA)		<= 19.0
38. 2-Hydroxyphenylacetic Acid (2-HPAA)		<= 1.2
39. 4-Hydroxyphenylpyruvic Acid (4-HPPA)		<= 24.7
40. Homogentisic Acid		<= 2.0
41. a-Ketoadipic Acid (AKAA)		<= 0.8
42. Glutaric Acid		<= 2.5
43. Orotic Acid		<= 20.7
44. Pyroglutamic Acid		21.7-105.3

Creatinine Concentration

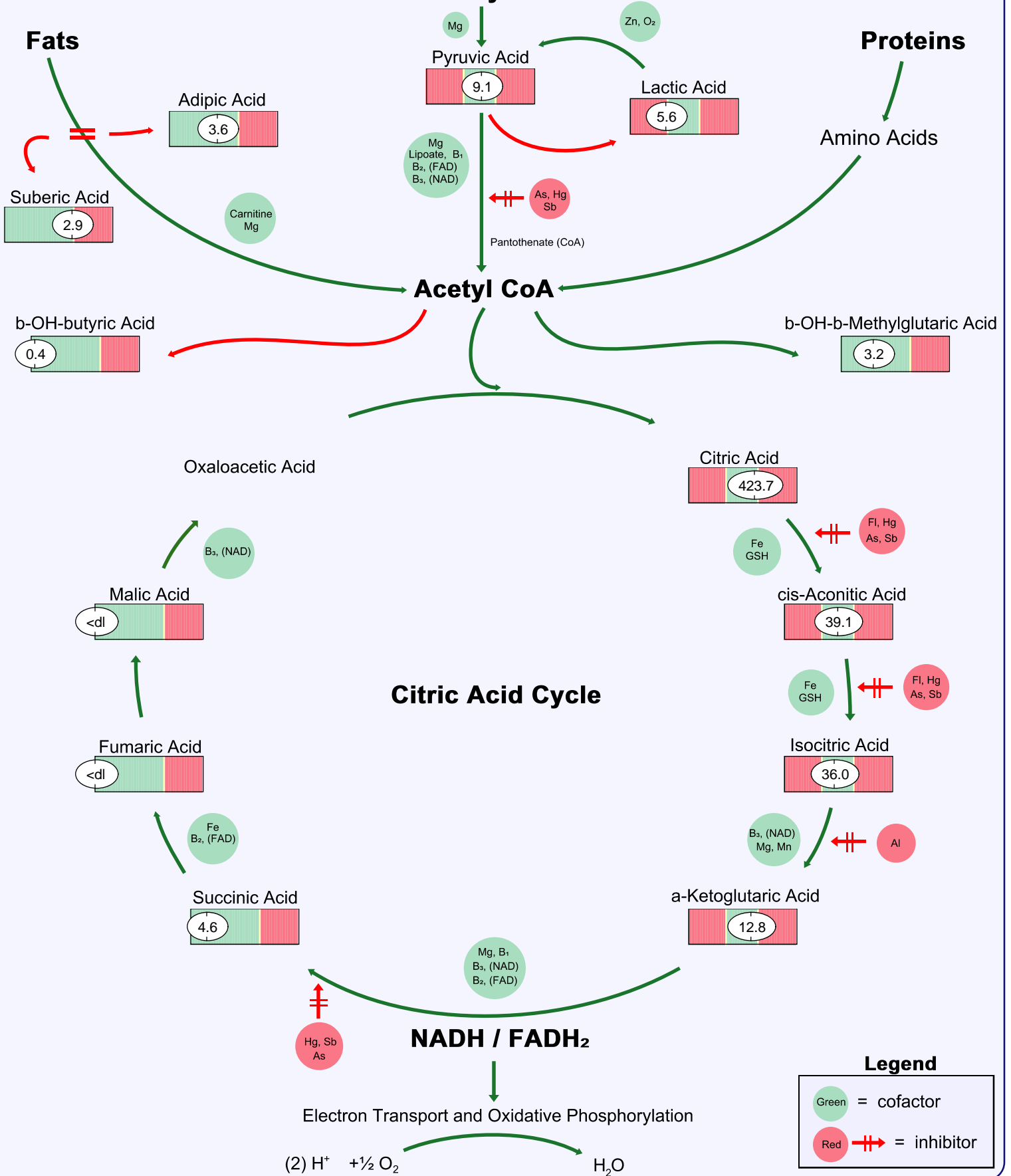
		mol / L
45. Creatinine Concentration		0.0031-0.0195

Lab Comments

This test has been developed and its performance characteristics determined by Genova Diagnostics, Inc. It has not been cleared or approved by the U.S. Food and Drug Administration.

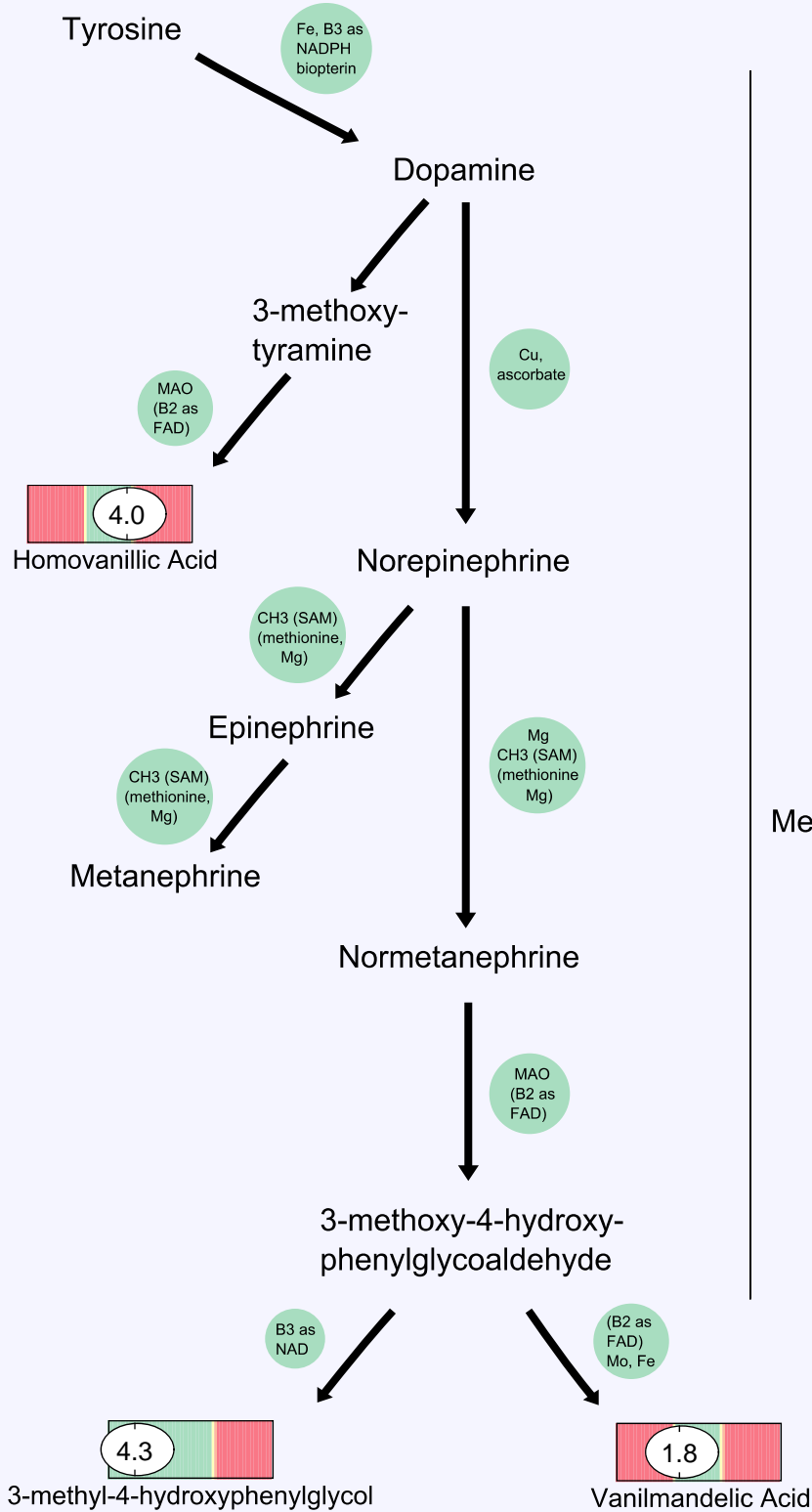
Krebs Cycle at a Glance

Carbohydrates

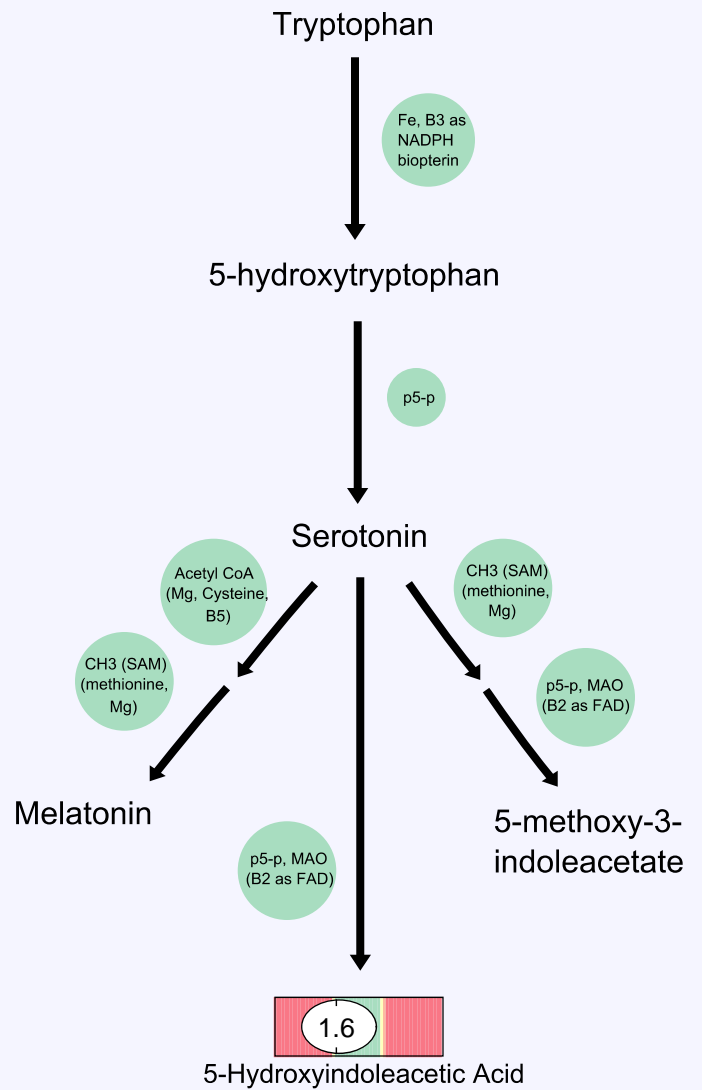


Neurotransmitter Metabolism

Catecholamine Metabolism



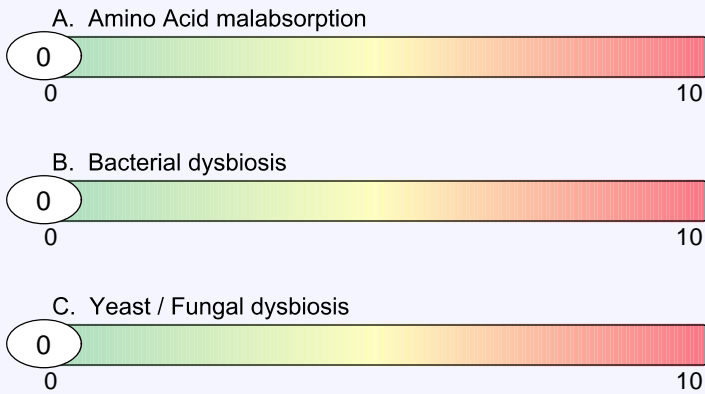
Serotonin Metabolism



Interpretation at a Glance

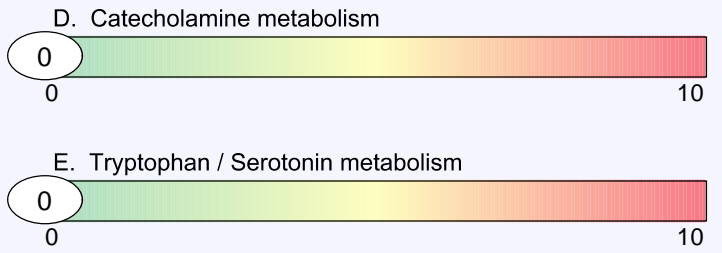
Malabsorption and Dysbiosis Markers

Relative Probability of Disorder

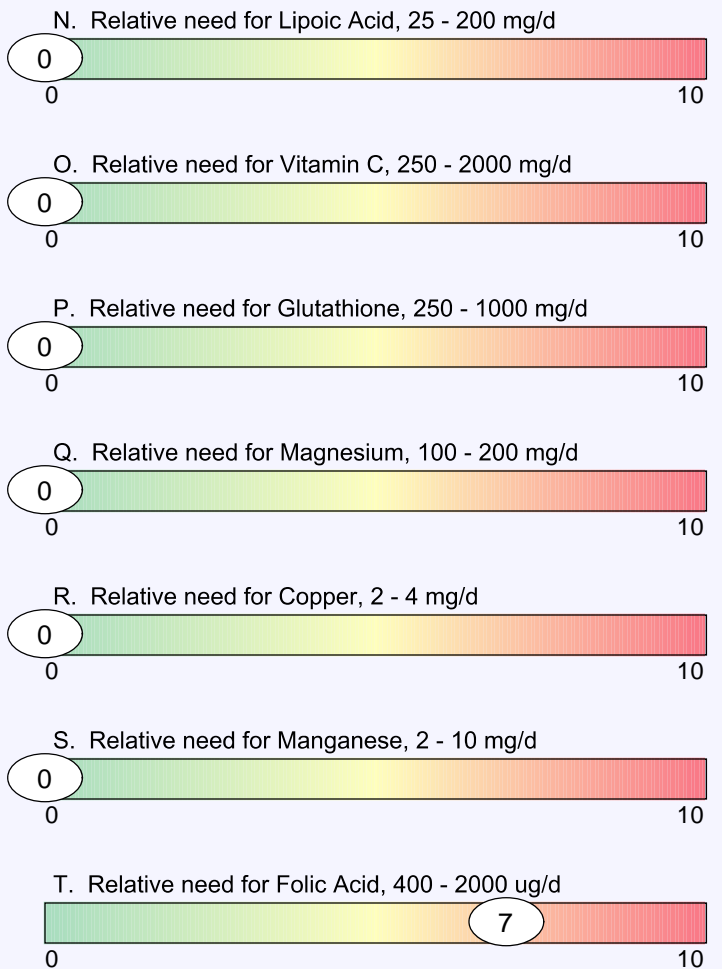
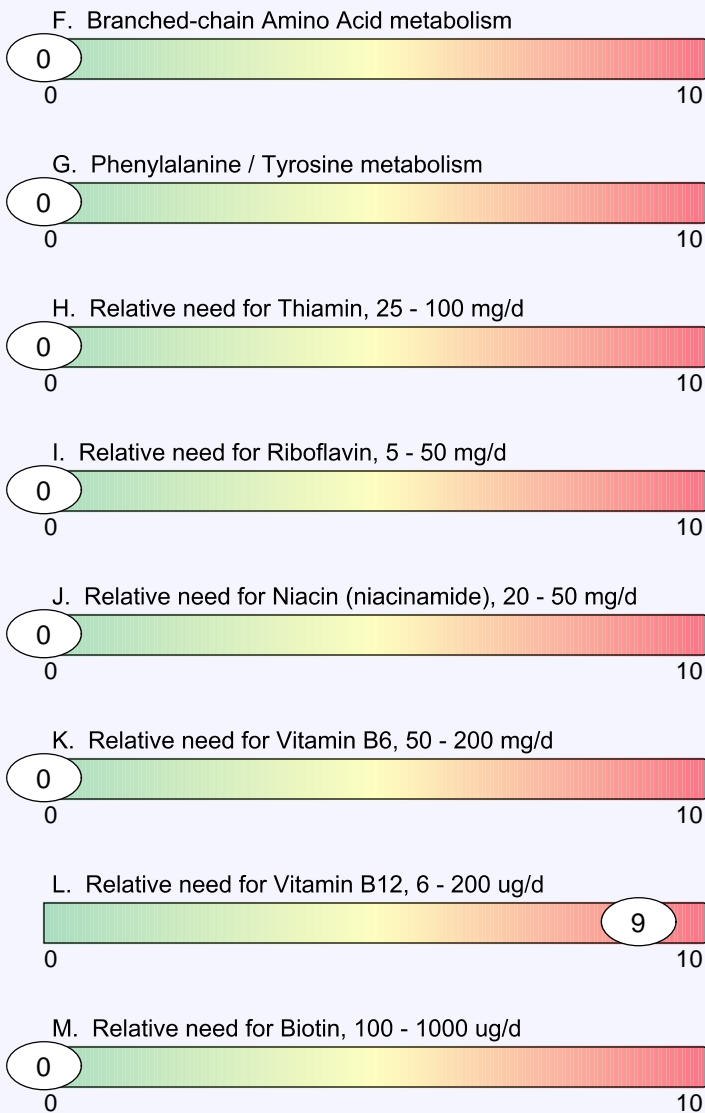


Neurotransmitter Metabolites

Relative Probability of Disorder



Vitamins and Minerals, Miscellaneous Cofactors



Commentary

Please note that Beta-ketoglutaric Acid has been removed from the Metabolic Analysis Profile. Beta-ketoglutaric Acid is a minor product of yeast/fungal metabolism. Other markers of yeast dysbiosis continue to be available on the Metabolic Analysis Profile.

Commentary is provided to the practitioner for educational purposes, and should not be interpreted as diagnostic or treatment recommendations. Diagnosis and treatment decisions are the responsibility of the practitioner.

Analyte Histogram Changes: The shape of the histograms for many of the analytes on this test have changed. The new histograms more accurately reflect the distribution of results for the reference population and for expected, normal levels. No reference range changes have been made.

MARKERS CHARACTERISTIC OF INTESTINAL MALABSORPTION AND/OR DYSBIOSIS

Three of these chemical markers are formed by yeast/fungal organisms, usually but not necessarily in the gut: arabinose, tartaric acid and citramalic acid. Citramalate can also be formed by anaerobic bacteria. The remaining markers of this section are the result of malabsorption, gut bacterial action, and in some cases, hepatic detoxication of chemicals produced by dysbiotic flora.

NEUROTRANSMITTER METABOLITES

These metabolites are end products of neurotransmitter metabolism, either the adrenal catecholamines or serotonin (5-HIAA). Abnormal levels correlate with mood swings, mental dysperceptions, anxiety, or depressive disorders.

All of these metabolites are within their reference ranges; there are no abnormalities.

ANALYTES CHARACTERISTIC OF CELLULAR ENERGY AND MITOCHONDRIAL FUNCTION

These markers are metabolites from four important biochemical pathways in the body, all of which significantly impact the production and availability of energy at the cellular level: glycolysis, the citric acid cycle (Krebs cycle) and both beta-oxidation and omega-oxidation of fatty acids. These analytes provide unique insight into macronutrient catabolism and mitochondrial function in cells. Abnormal levels may be associated with fatigue, malaise, myalgia, headache, muscle weakness, myopathy, hypotonia, or acid-base imbalance. This test is intended to be a diagnostic aid for acquired disorders in these pathways. It is not intended for diagnosis of inborn errors of organic acid metabolism, as this would require extensive molecular genetics testing. However, significantly abnormal findings could be consistent with such inborn errors.

If significant abnormalities persist after removal of toxics, supplementation of appropriate nutrients, dietary and hormonal adjustments, and correction of intestinal dysbiosis or infection, it is suggested that the patient be referred to a medical center with capabilities for diagnosis and treatment of congenital metabolic defects.

Lactic Acid , or lactate, is measured to be low. Lactate is formed from pyruvate in anaerobic or oxygen starved (hypoxic) circumstances to allow for ongoing production of ATP in these anaerobic conditions. There are no known clinical problems associated with low lactic acid. Low levels are usually a result of reduced amounts of its precursor, pyruvic acid.

Commentary

COFACTOR-DEPENDENT AND METABOLITES FROM AMINO ACID CATABOLISM

These analytes are formed from essential and protein amino acids via amino group transfer or by other enzymatic transformations. Many are sensitive to vitamin functions as coenzymes and to minerals as enzyme activators. Excesses or deficiencies may lead to various conditions depending upon the particular metabolic imbalance, including fatigue, headaches, myalgias, metabolic acidoses, dietary intolerances, neurological problems, and cognitive disorders.

Formiminoglutamic Acid "FIGlu" is elevated in the urine. FIGlu stands for formiminoglutamic acid, a substance produced in body tissue from the dietary amino acid histidine. FIGlu needs tetrahydrofolate (THF), a reduced form of folic acid, to be changed into forms that are metabolically useful.

Elevated urine FIGlu can occur with several circumstances. Dietary deficiency of folic acid or severe oxidant stress that limits biologic reduction of folic acid to the THF form can cause this elevation. Histidine as a supplemented nutrient can contribute to urine FIGlu levels, especially if taken in amounts that exceed 50 mg/Kg body weight. Metabolism of folic acid can be impaired if vitamin B12 is insufficient or if its metabolism is disordered. So, elevated FIGlu also can mean that some form of B12 or cobalamin is needed. The enzyme that promotes processing of FIGlu and THF requires pyridoxal 5-phosphate as a coenzyme, and vitamin B6 deficiency also may contribute to elevated FIGlu. Finally, there are rare disorders in purine synthesis that impair normal utilization of folate forms that come from FIGlu and THF. Abnormal levels of uric acid, succinylpurines, inosine or adenosine may be investigated if FIGlu levels remain elevated despite folate, cobalamin, pyridoxine and antioxidant therapy.

Elevated FIGlu can be coincident with homocystinuria and predisposition to cardiovascular disease. In children, elevated FIGlu and folate and/or vitamin B12 dysfunctions may be associated with mental retardation, autism, growth failure and seizures. Folate and/or vitamin B12 insufficiencies can be secondary to gastrointestinal disorders or poor quality diet, and deficiencies of both have been noted in elderly populations.

Methylmalonic Acid (MMA) is measured to be high in the urine. MMA comes from propionyl-CoA via methylmalonyl-CoA, and leads to succinyl-CoA (needed by the citric acid cycle). Major dietary sources of propionyl-CoA include valine, isoleucine, methionine, threonine (from dietary protein) and odd-carbon-chain fatty acids.

The most common cause of methylmalonic aciduria is cobalamin (vitamin B12) insufficiency, since the nutrient is needed for its metabolism. Expected symptoms of B12 dependent methylmalonic aciduria may be those of B12 deficiency, including fatigue, ataxia, sensory losses, paresthesias and other neurological problems. Anemia may or may not be present. Vitamin B12 administration is the most appropriate therapy when either MMA or 3-HPA is high. Nutritional adequacy of vitamin B12 is the amount needed for cobalamin in its coenzyme forms, such that coenzyme activity does not limit metabolism.

Less common causes of elevated MMA include deficiency or dysfunction of the apoenzyme, methylmalonyl-CoA mutase or deficiency of the coenzyme, deoxyadenosylcobalamin, needed for cobalamin synthesis. In such cases, megadoses of vitamin B12 as hydroxycobalamin are appropriate: 1000-2000 mcg per day (intramuscular) for 2-3

Commentary

days, followed by periodic high doses of oral B12. The frequency for both of these genetic defects is 1 in 20,000.

INTERPRETATION AT A GLANCE



Urine Lipid Peroxides



Patient: **SAMPLE REPORT**

Order Number: A2123456

Completed: June 17, 2008

Received: June 11, 2008

Sex: M

Genova Diagnostics Europe

Referring Laboratory

Parkgate House

356 West Barnes Lane

New Malden, Surrey KT3 6NB

Great Britain and Northern Ireland

Urine Lipid Peroxides

	Inside Range	Outside Range	Reference Range
Urine Lipid Peroxides	4.9		<=10.0 micromol/g Creatinine

Commentary

The performance characteristics of all assays have been verified by Genova Diagnostics, Inc. Unless otherwise noted with ♦ as cleared by the U.S. Food and Drug Administration, assays are For Research Use Only.

Commentary is provided to the practitioner for educational purposes, and should not be interpreted as diagnostic or treatment recommendations. Diagnosis and treatment decisions are the responsibility of the practitioner.

Urine lipid peroxides is a marker of free radical damage in the body. An elevated level may reflect excess free radical production and/or insufficient antioxidants. Free radical damage is thought to underlie many processes such as atherosclerosis, chronic fatigue syndrome, cancer, cardiovascular disease, Parkinson's disease, Alzheimer's, and aging.