

Using organic acids to resolve chief complaints and improve quality of life in chronically ill patients

Part IV

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Summer of Work Exposes Medical Students to System's Ills - NYTimes.com Page 1 of 3

The New York Times

September 9, 2009

Summer of Work Exposes Medical Students to System's Ills

By Jacob R. Goffman

SEATTLE — This summer, medical students from the University of Washington took a long look under the hood of the health care system they are about to inherit, and many returned to campus last week with their eyes wide open and their idealism tempered.

Jacob R. Goffman is a senior medical student at the University of Washington. He is the author of "The Tidal Wave of Chronic Illness," a book that will be published by the University of Washington Press in 2010.

Summer of work exposes medical students to system's ills, *The New York Times*, September 9, 2009

The students learned not only to deliver babies and suture wounds, but also to order unnecessary tests as protection against lawsuits, to hector specialists into seeing Medicaid patients, to match patients with prescriptions on Wal-Mart's \$4 list. And they saw firsthand what Mr. Olgard called "a tidal wave of chronic disease" — diabetes, hypertension, obesity, depression — that left many questioning how much any one physician could really accomplish.

"I often wondered what we were actually doing to help people," Mr. Olgard said.

Not surprisingly, the students found the system to be a mess. "You have to be a realist," Mr. Olgard said. "You have to be realistic about the system. It's not perfect, but it's not as bad as you think it is."

"...a tidal wave of chronic illness..."

For 10 years, the University of Washington School of Medicine has dispatched its rising second-year students across the Northwest to provide primary care in rural or underserved areas. The monthlong program is elective, but a record number of students took part this year — 117 from a class of 216.

The school has a cooperative agreement to train medical students from Alaska, Idaho, Montana and Wyoming, none of which has a medical college of its own. The summer program is one of several vehicles, along with loan forgiveness endorsements, designed to lure students back to their home states to practice in areas with a shortage of primary care doctors.

This year, the students set out as negotiations were intensifying in Washington over wholesale changes to the health care system they will soon be joining. Whether there will be improvements to the system remains to be seen, but few students returned thinking there was little to fix.

http://www.nytimes.com/2009/09/09/health/policy/09medschool.html?_r=1&th=&mc=th&... 9/9/2009

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Using the Organic Acids Test – Part 4

Dr. Jeff Moss



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Baracos VE. Overview on metabolic adaptation to stress, pp. 1-13.

“An understanding of the nature of stress is fundamental to the rational design of nutrient mixtures to feed patients whose homeostasis has been altered by one or more stressors.”

“All stresses may be presumed to be associated with characteristic modifications in the metabolism of lipids, carbohydrates, amino acids, and micronutrients.”

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Clinical Nutrition (2004) 23, 1256-1266



ELSEVIER

REVIEW

Acute and “chronic” phase reaction—a mother of disease

Stig Bengmark

Department of Surgery and Liver Institute, UCL, London, UK

Received 23 July 2004

KEYWORDS
Acute phase response;
Chronic phase response;
Metabolic syndrome;
Cellular membranes;
Endothelial cells;
Abdominal obesity;
Insulin resistance;
Growth hormones;
Free fatty acids;
Alzheimer disease;
Parkinson disease;
Neurodegenerative disease;
Atherosclerosis;
Coronary-vascular disease;
Cancer;
Breast cancer;
Colorectal cancer;
Prostatic cancer;
Diabetes;
Cirrhosis;
Fatty liver;
Gallbladder disease;
Chronic renal disease;

Summary The world is in diseases. Almost half of the mortality is due to these chronic diseases. And they are these diseases are associated consumption of calorie and also under-consumption of a function of the innate immune changes induced in response the subsequent development chronic diseases. The endo importance, and the function the function of the underlying cells; their ability to obtain nutrients and antioxidants and to eliminate waste products. The abdominal adipocytes seem to play a key role, as they have the ability to in stressful situations release much of proinflammatory cytokines, PPAR- α and free fatty acids compared to elsewhere in the body. The load on the liver of these various substances in often of greater magnitude than the liver can handle. Some of the most common chronic diseases and their potential association with acute and “chronic” phase responses and metabolic syndrome are discussed separately. The need for studies with lifestyle modifications is especially emphasized.
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Bengmark S. Acute and “chronic” phase reaction – a mother of disease, *Clin Nutr*, Vol. 23, pp. 1256-66, 2004

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Using the Organic Acids Test – Part 4
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Considerations for the Catabolic Patient				
Signs & Symptoms	Consider	Treatment Plan	Monitor	Supplementation
Signs & Symptoms: - Feeling "lousy" - Fatigue - Cravings - Depression/anxiety - Poor quality sleep - Poor digestion/absorption/leaky gut - Chronic pain/infection Clinical Findings: - High % body fat - Low bone density - High fasting insulin - Heavy metal burden - Calcium to phosphorus ratio 3:1 - High normal globulin - Cholesterol (220-280) - High LDL, low HDL - High triglycerides - Low normal serum potassium, high normal serum sodium - High normal BUN with normal creatinine - Low urinary sulfate - Genomic variations	Optimize pH	Optimize fluid and electrolyte balance	Morning urinary pH -pH paper	Potassium bicarbonate -K Alkaline (Moss) Magnesium -Magnesium Glycinate (Vital Nutrients)
	Optimize muscle mass (reduce sarcopenia) and amino acid levels	Pay special attention to protein and amino acids	Body composition: -Bioelectric impedance scale Grip strength: -dynamometer	Amino acids -Custom Amino Base (Metabolic Maint.) Protein powders/bars -UltraLean products (Biogenesis) -Paleo products (DFH)
	Reducing inflammation	Reduce inflammation Address food allergies	Inflammatory biomarkers: -HS-CRP -Fibrinogen levels -WBC -Differential	Anti-inflammatory agents -Inflametric (Olympian) -D-Flame (Protocols) -Pain Guard (Perque) Fish Oils -Ultra Pure Fish oil (Vital Nutrients) Food allergy blends -AllerGzymes (DFH)
	Improving insulin sensitivity	Watch carbohydrate/protein ratio	Fasting glucose Fasting insulin Cravings	Metabolic Synergy (DFH)
	Optimize dietary carbohydrate/protein ratios	Replete protein deficiencies and reduce carbohydrate intake along with improvements in dietary quality	Diet diary Optimal protein intake -1.0 – 1.2 g/kg body weight	Protein powders/bars -Paleo products (DFH) -UltraLean (Biogenesis)
	Optimize GI function	Dietary changes, supplementation of digestive aids	Patient signs and symptoms	Betaine HCl (Vital Nutrients) Digestzymes (DFH) Intestinal Repair Complex (Biogenesis)
	Optimize micronutrient deficiencies/imbbalances	Supplementation of multis Possible emphasis on vitamin D, iodine, and zinc	Serum 25 OH vitamin D Fatigue/serum thyroid indicators Zinc taste test	Multi Nutrients (Vital Nutr) Vitamin D (Vital Nutr) Iosol (Moss) Zinc Supreme (DFH)
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Neuro-SIGNALS Invited Review Neurosignals 2009;17:144-152 DOI: 10.1159/000188162 Neurosign April 20, 2009 Accepted after revision June 5, 2009 Published online February 5, 2009	
Biological Mechanism of Antidepressant Effect of Omega-3 Fatty Acids: How Does Fish Oil Act as a 'Mind-Body Interface'? Kuan-Pin Su Department of General Psychiatry and Mind-Body Interface Laboratory (MBI-LAB), China Medical University and Hospital, Taichung, Taiwan; Stress, Psychiatry and Immunology Laboratory (SPI-LAB), Institute of Psychiatry, King's College London, London, UK	
Key Words Major depressive disorder · Depressive acid · Docosahexaenoic acid · Arachidonic acid · Prostaglandins · Thromboxanes · Lipid Phospholipase A2 · Cyclo-oxygenase	Su KP. Biological mechanism of antidepressant effect of omega-3 fatty acids: How does fish oil act as a 'mind-body interface'? <i>Neurosignals</i>, Vol. 17, pp. 144-152, 2009
Abstract The unsatisfactory results of monoamine-based antidepressant therapy and the high occurrence of somatic symptoms and physical illness in patients with depression imply that the serotonin hypothesis is insufficient to approach the aetiology of depression. Depressive disorders with somatic presentation are the most common form of depression. Somatization, the bodily symptoms without organic explanation, is similar to cytokine-induced sickness behaviour. Based on recent evidence, omega-3 polyunsaturated fatty acids (n-3 PUFAs, or n-3 fatty acids) are enlightening a promising path to discover the unsolved of depression, sickness behaviour and to link the connection of mind and body. The PUFAs are classified into n-3 (or omega-3) and n-6 (or omega-6) groups. Eicosapentaenoic acid and docosahexaenoic acid, the major bioactive components of n-3 PUFAs, are not efficiently synthesized in humans and should therefore be obtained directly from the diet, particularly by consuming fish. Docosahexaenoic acid deficiency is associated with dysfunctions of neuronal membrane stability and transmission	prostaglandin E₂ synthesis, which might be linked to the somatic manifestations and physical comorbidity in depression. The role of n-3 PUFAs in immunity and mood function supports the promising hypothesis of psychoneuroimmunology of depression and provides an excellent interface between 'mind' and 'body'. This review is to provide an overview of the evidence about the role of n-3 PUFAs in depression and its common comorbid physical conditions and to propose mechanisms by which they may modulate molecular and cellular functions. Copyright © 2009 S. Karger AG, Basel Introduction Major depressive disorder (MDD) is a serious psychiatric illness with a high lifetime prevalence rate [1]. However, the current treatment for this high burden disease is not satisfactory. Less than 50% of patients achieve full remission with optimized medication treatment [2] despite that more than 40 antidepressants with mechanisms related to serotonin, norepinephrine and/or dopamine
KARGER © 2009 S. Karger AG, Basel 1662-4547/09/1702-0144\$30.00 Fax: +41 76 30 12 14 E-Mail: karger@karger.ch www.karger.com	Dr. Kuan-Pin Su Department of Psychiatry China Medical University Hospital No. 2, Yuh-Der Road, Taichung 40452 (Taiwan) Tel.: +886 4 2206 2121, ext. 1070; Fax: +886 4 2206 1200; E-Mail: kalsu@cmu.edu.tw

Table 1. Overlapping of symptoms of acute sickness behaviour associated with IFN- α therapy and the somatic symptoms in MDD

Symptoms	Prevalence in IFN- α therapy ^a , %	Prevalence in MDD ^b , %
Fatigue/asthenia	39–90	73
Headache	27–67	33 ^e
Gastrointestinal symptoms	50 ^{c, 1}	34–47 ^f
Psychomotor slowing	40 ^c	59–65 ^f
Insomnia	20–39 ^d	63
Irritability	35 ^d	50
Arthralgia	9–36	31 ^e
Musculoskeletal pain	26–32	62–80 ^{g, 2}
Abdominal pain	15–20	21 ^e
Anorexia	13–19	40
Anxiety	13–18 ^d	57
Poor concentration	14 ^d	51

^a [46], unless otherwise specified; ^b [99], unless otherwise specified; ^c [100]; ^d [101]; ^e [102]; ^f [103]; ^g [104].

¹ Nausea, vomiting, bowel problems.

² Result from depressed inpatient population.

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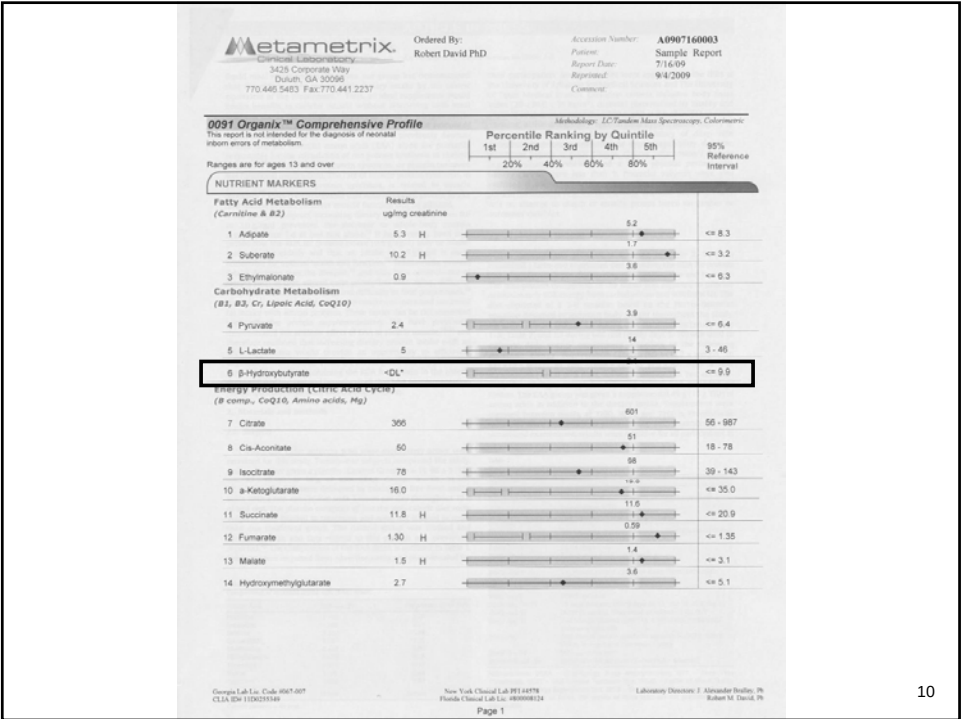
Organic Acids

Markers for vitamins B1, B2,
B3, B5 and lipoic acid

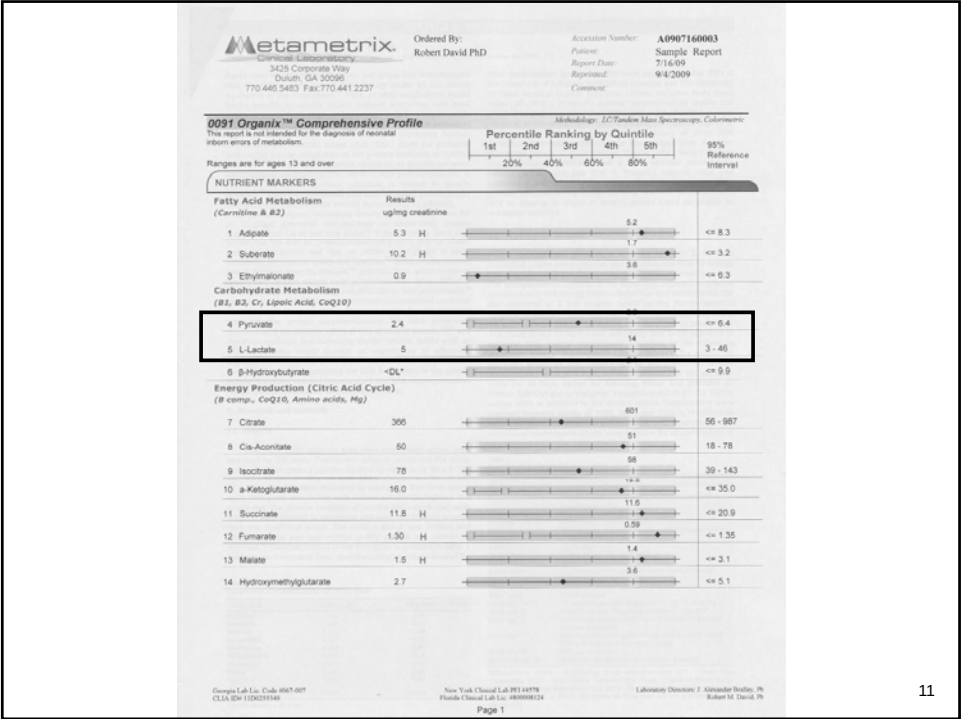
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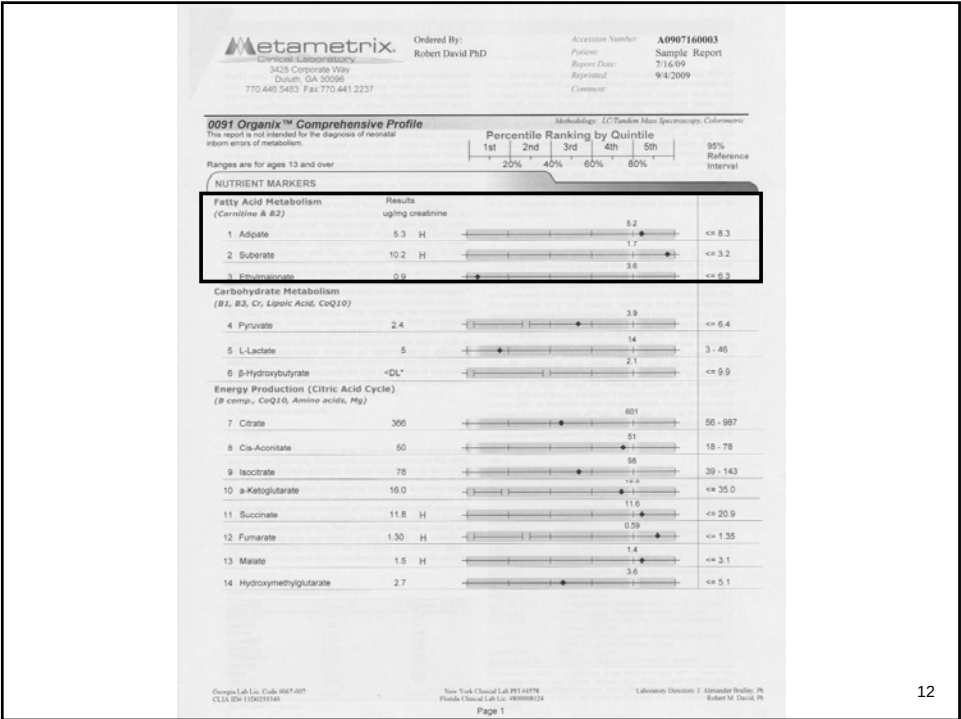
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Signs & Symptoms	Consider	Treatment Plan	Monitor	Supplementation
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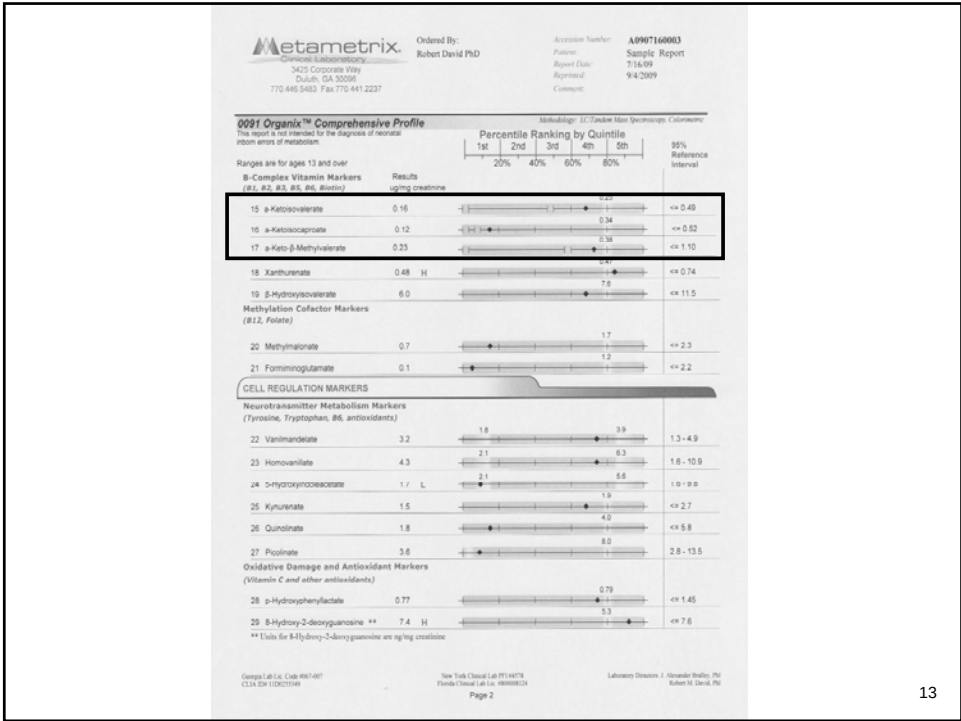


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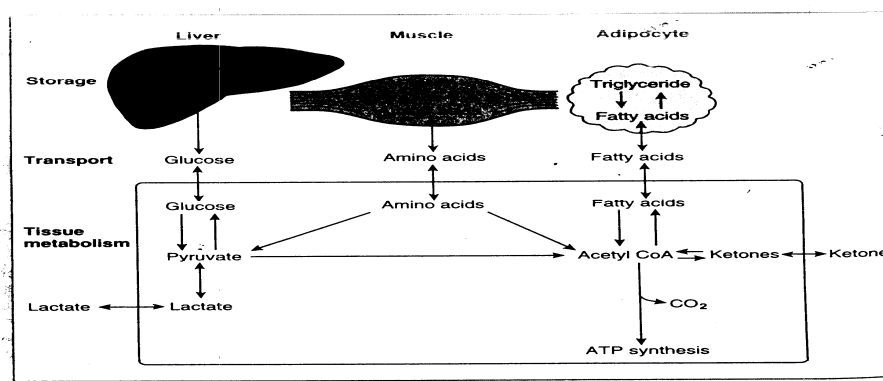


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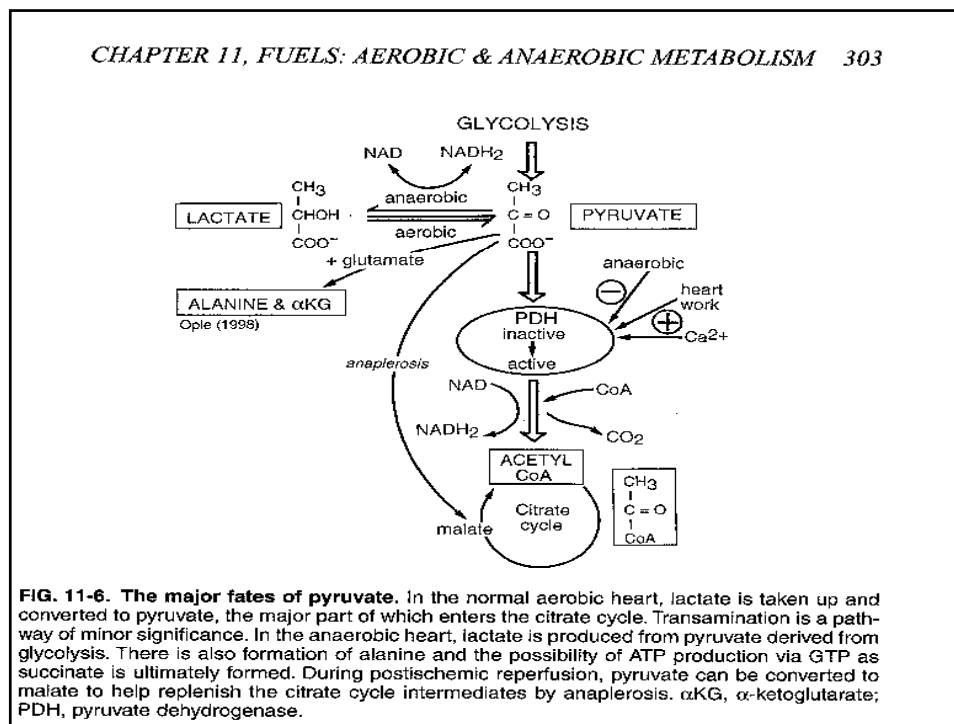
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Interconversion of Metabolic Fuels



Interconversion of metabolic fuels, Coffee CJ. Metabolism, Fence Creek Publ., Madison, CT, 1998, p. 102



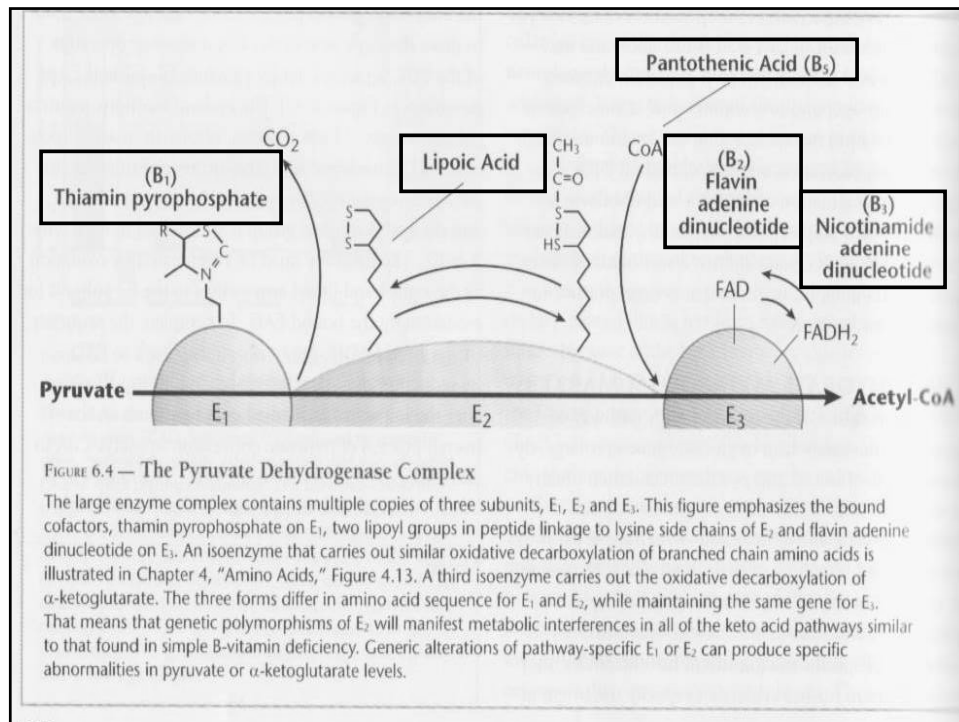
LABORATORY EVALUATIONS

FOR INTEGRATIVE AND FUNCTIONAL MEDICINE

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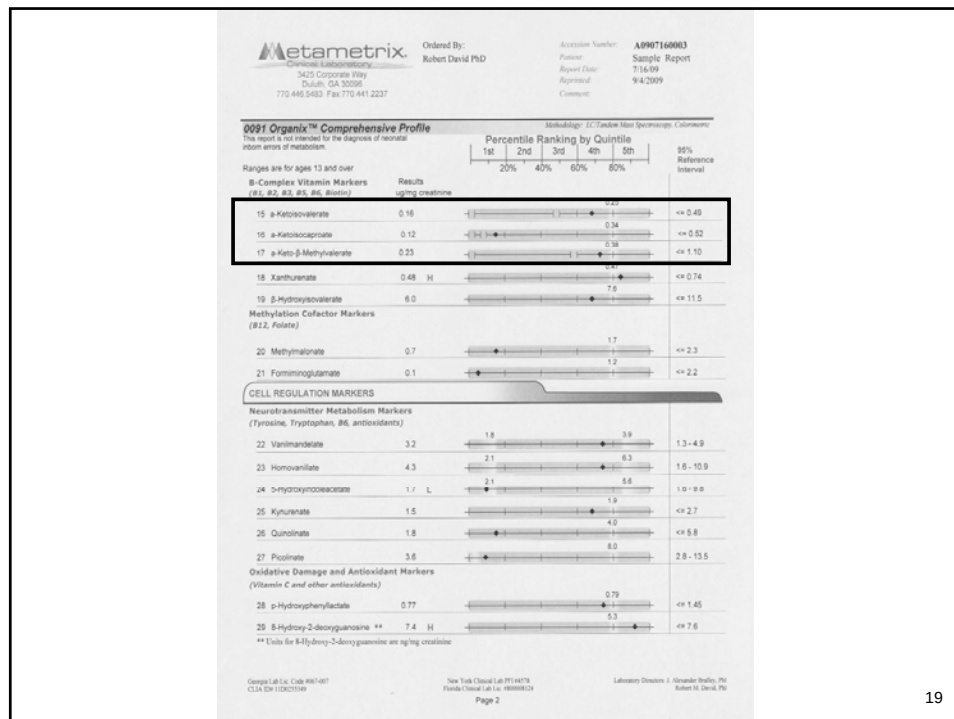
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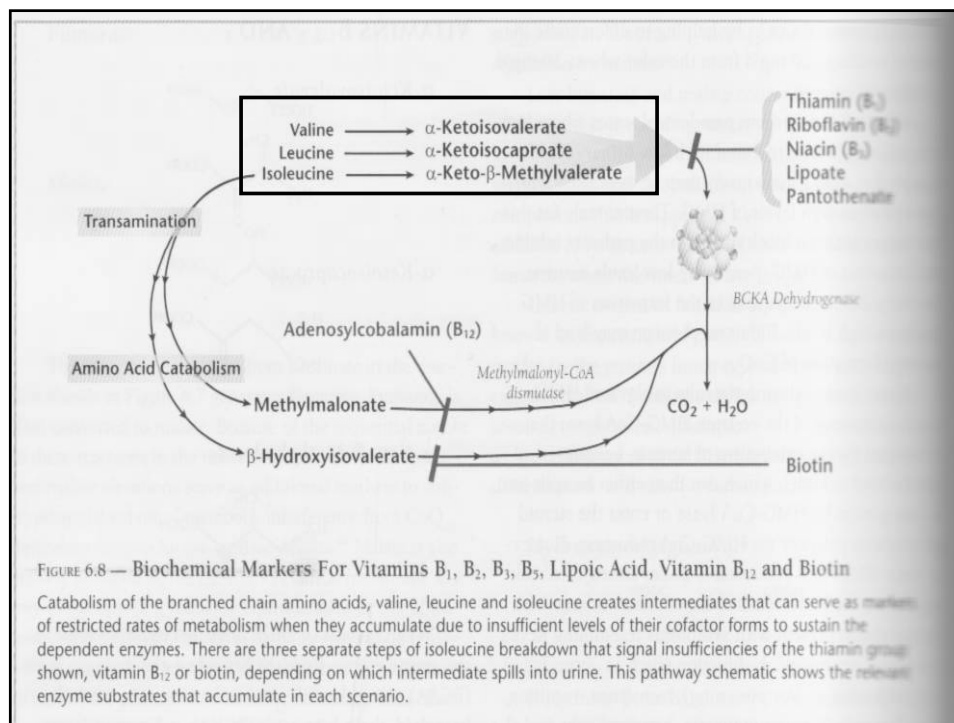
- "Mitochondrial conversion of pyruvate to acetyl-CoA requires the pyruvate dehydrogenase complex (PDC), the enzymatic gatekeeper for generation of acetate units to drive the citric acid cycle."
- "The three-step operation of the PDC is carried out by enzymes E1, E2, and E3..."
- "The cycle of reactions requires cofactors derived from thiamin, riboflavin, niacin, lipoic acid and pantothenic acid (the dietary essential precursor of coenzyme A)."

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- “When hepatic or small intestinal enzymes remove the amino groups from the branched-chain amino acids (BCAA) valine, leucine, and isoleucine, they form the branched-chain keto acids (BCKA), α -ketoisovalerate, α -ketoisocaproate, and α -keto- β -methylvalerate, respectively.”
- “...the enzymes require cofactors derived from five B-complex vitamins: B₁, B₂, B₃, B₅, and lipoic acid.”
- “Elevations of the branched-chain keto acids provide functional assessments of the sufficiency of these vitamins, especially thiamin.”

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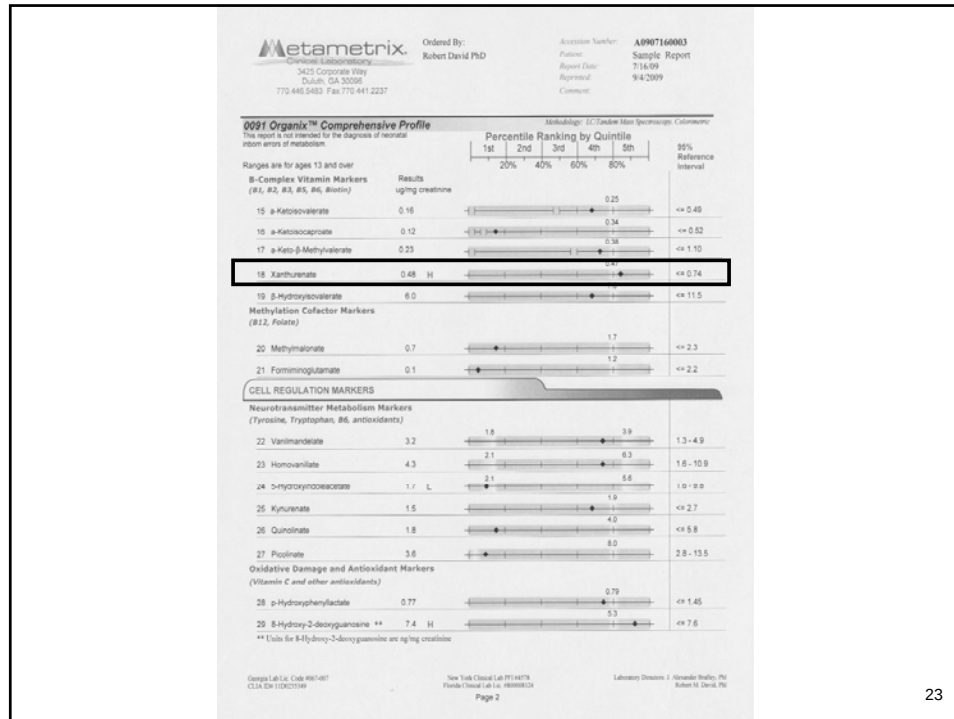
Organic Acids

Marker for vitamin B6

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- “Vitamin B₆ insufficiency leads to elevated excretion of xanthurenate and kyurenate because of a pyridoxal-5-phosphate dependent step in this metabolic pathway, called the kyurenin pathway.”
- “Dietary deficiency of B₆ quickly manifests as slower rates of the B₆-dependent step, with accumulation of 3-hydroxykynurenin, which causes accumulation of kynurenin.”
- “These compounds are rapidly converted to xanthurenate and kynurenate, respectively, which appear at elevated concentrations in urine.”

- “In most individuals the urinary xanthurenate rises to the higher concentrations, sometimes appearing as the only positive marker.”
- “Elevated kynurenate provides biochemical confirmatory evidence of B₆ insufficiency, especially when quinolinate is not high.”
- “Although the excretion of both kynurenate and xanthurenate are increased when vitamin B₆ is insufficient to maintain this function, xanthurenate is the principal product that appears in most cases. This is due to the further metabolism of kynurenate to citric acid cycle intermediates in the liver.”

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- “Elevated urinary xanthurenate may be produced from dietary tryptophan even when there is no extra tryptophan challenge.”
- “In such cases, vitamin B₆ insufficiency is significant enough to interfere with even the normal conversion of dietary tryptophan.”

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- “Vitamin B₆ deficiency produces elevated xanthurenate that, in turn, produces increased oxidative stress.”
- “This means, that, in addition to indicating a need for therapy with vitamin B₆, elevated xanthurenate leads to suspicion of antioxidant insufficiency.”
- “Interpretation of elevated urinary xanthurenate should take into account kynurenin pathway modulation by steroid hormones and bacterial endotoxins, plus decreased flux of tryptophan through the pathway for individuals on low-protein intake.”

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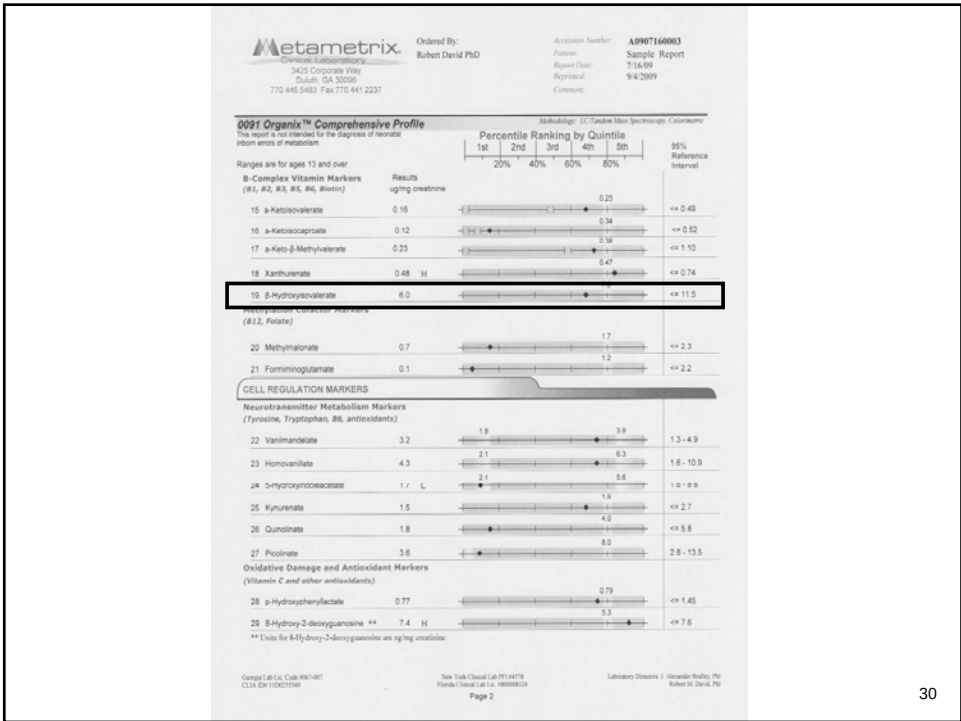
- “Xanthurenic acid forms complexes with insulin, decreasing circulating concentrations and reducing activities of the hormone.”
- “Significantly elevated excretion of xanthurenate has been found in diabetic patients and animal models of diabetes.”
- “In pregnant women with gestational diabetes, excessive amounts of urinary xanthurenate were found.”
- “Treatment with 100 mg of pyridoxine daily for 14 to 23 days restored the urinary xanthurenic acid to normal in all patients tested. In women who received pyridoxine for 14 days oral glucose tolerance also improved.”

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Organic Acids

Marker for biotin

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What is biotin?

- “Biotin is a cofactor in the acetyl-CoA carboxylase, pyruvate carboxylase, methylcrotonyl-CoA carboxylase, and propionyl-CoA carboxylase. The biochemical function of biotin-requiring enzymes is the insertion of carboxyl.”

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Function of biotin-dependent enzymes

- “The carboxylase enzymes have critical roles in major pathways for the utilization of energy from amino acids (where β -hydroxyisovalerate is formed), the synthesis of fatty acids for cell membrane replacement, and the maintenance of blood glucose via gluconeogenesis.”

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How the biomarker is formed

- “Because leucine turnover amounts to many grams per day, its catabolism is a high-flux process that offers a biochemical marker of biotin deficiency.”
- “The product formed after the first three steps of the pathway, β -methylcrotonyl-CoA, requires a biotin-dependent carboxylation to allow the flow to continue.”

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- “Deficiency of biotin causes accumulation of the this biotin-dependent intermediate, and its hydrated product, β -hydroxyisovalerate, spills in urine.”
- “ β -hydroxyisovaleric aciduria appears early in people who are made biotin deficient by consuming the biotin-binding protein, avidin.”

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- “Even antibiotic-induced disruptions of gastrointestinal bacterial biotin synthesis can produce biotin insufficiency detectable by elevated β -hydroxyisovalerate.”
- “In addition to increased β -hydroxyisovalerate, biotin deficiency may produce elevations of lactate and alanine in urine and accumulations of odd-chain fatty acids (C15:0 – C29:0) in plasma or red blood cell membranes.”
- “The lactate and alanine effect results form decreased rates of gluconeogenesis in biotin deficiency.”

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Thank you!!

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