

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

Part I

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The New York Times

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September 9, 2009

Summer of Work Exposes Medical Students to System's Ills

13. BEYON BACK

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 B.
more the
eschewed
Olsgaard
treatment

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. Olsgaard 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. Ohsgaard said.

Not sur-
devalue

"You have
reimbursed
me for my

For 35 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.

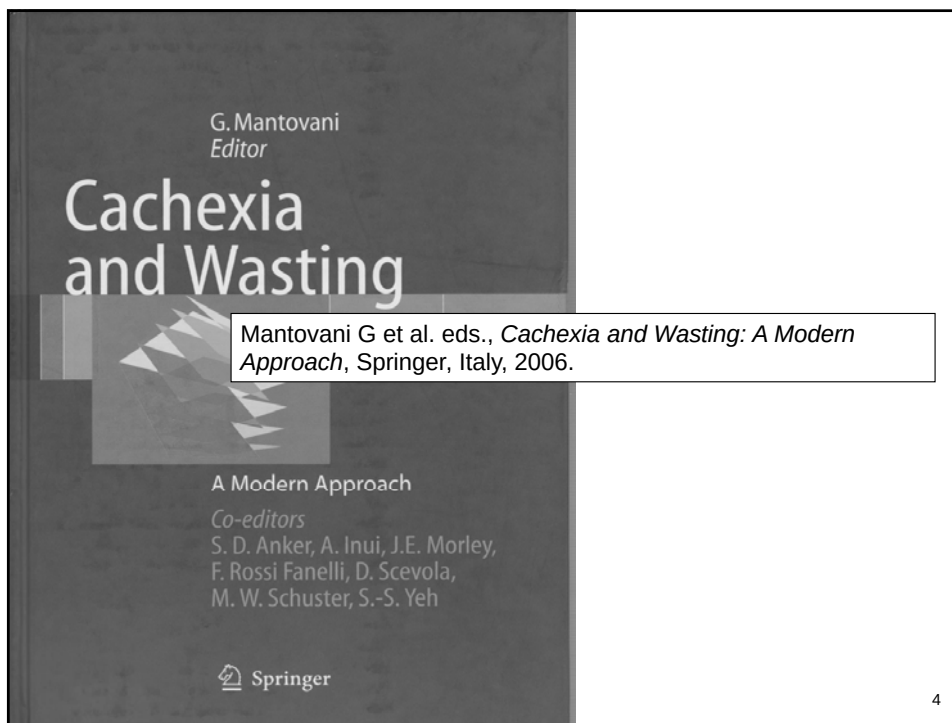
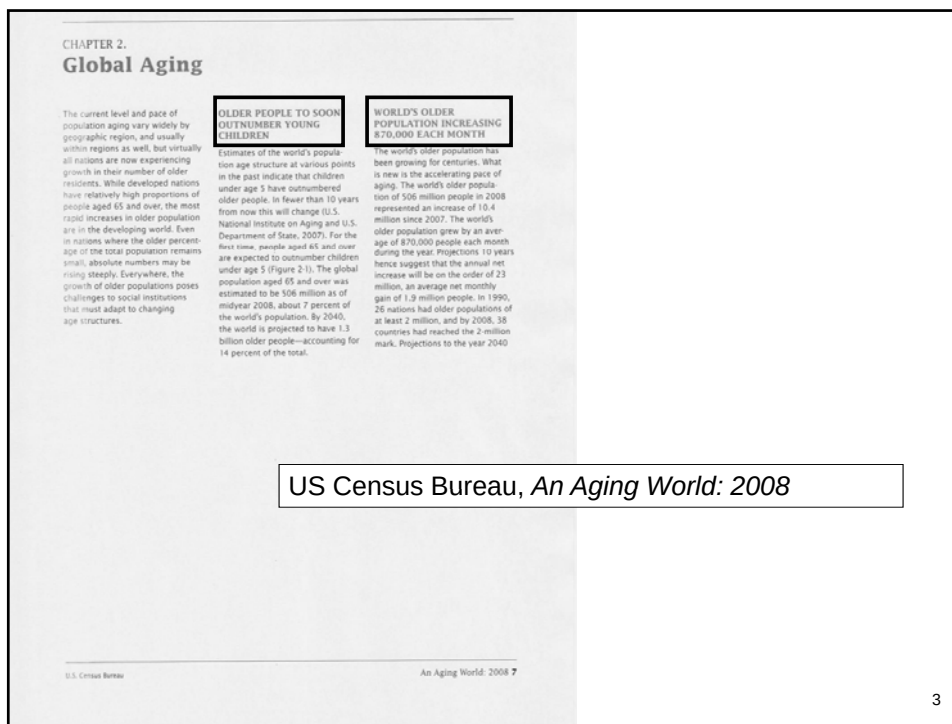
The school has a cooperative agreement to train medical students from Alaska, Idaho, Montana and Wyoming, none of which has a medical college of its own. The summer program is one of several vehicles, along with loan forgiveness enticements, designed to lure students back to their home states to practice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

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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- “Cachexia may well represent the devastating flip side of the tremendous achievements of modern medicine, as the incidence of cachexia is also a function of survival of chronic illness.”
- “Many diseases – which rapidly led to death only a few years ago – are now better controlled by new therapies. Even if we cannot cure and eradicate these diseases, their natural history has significantly increased by months and years. Although these new therapeutic strategies represent a remarkable advantage over the previous standards of care, ***it is impossible to ignore the fact that many more patients are now facing the nutritional and metabolic consequences of prolonged immunological and hormonal challenges due to both the illness process itself and the aggressive therapies.***”

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Nestlé Nutrition Workshop Series
Clinical & Performance Program, Vol. 8

**Nutrition and
Critical Care**

Luc Cynober
Frederick A. Moore

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.”

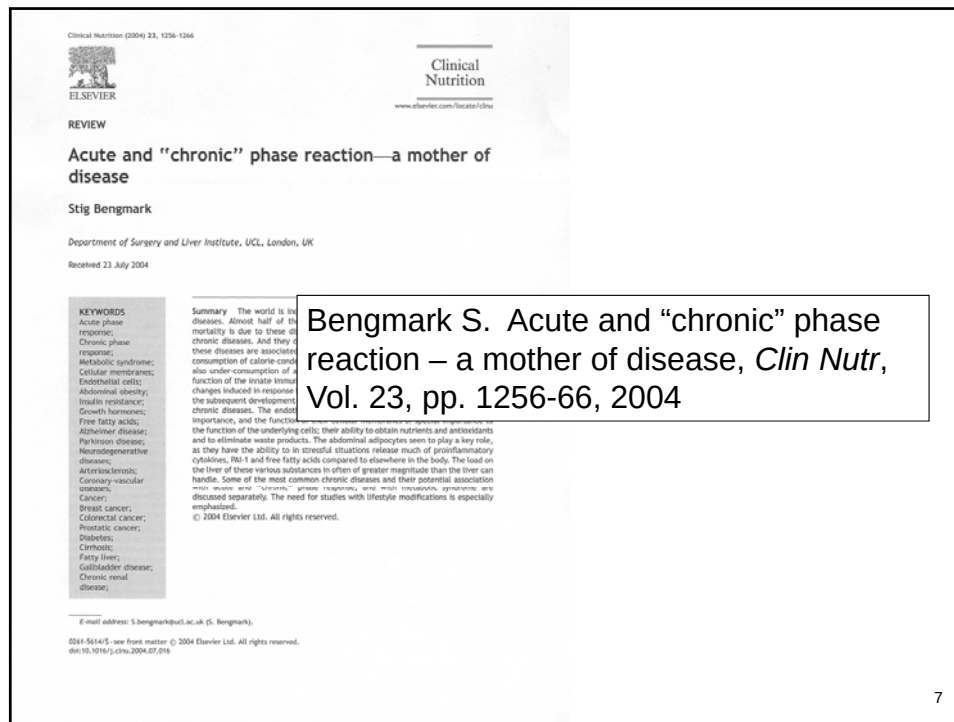
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Signs & Symptoms	Consider	Treatment Plan	Monitor	Supplementation
Signs & Symptoms: - Feeling "lousy" - Fatigue - Cravings - Depression/anxiety - Poor quality sleep - Poor digestion/absorption/leaky gut - Chronic pain/infection	Optimize pH	Optimize 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 -UltraL-can products (Biogenesis) -Palo products (DFH)
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	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 -Palo products (DFH) -UltraL-can (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/imbalances	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) Isole (Moss) Zinc Supreme (DFH)

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NEURO-SIGNALS

Invited Review

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Biological Mechanism of Antidepressant Effect of Omega-3 Fatty Acids: How Does Fish Oil Act as a 'Mind-Body Interface'?

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Key Words
Major depressive disorder · Depression · Docosahexaenoic acid · Arachidonic acid · Prostaglandins · Thromboxanes · Leukotrienes · Cyclo-oxygenase 2 · Cyclo-oxygenase 1

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. Docosahexaenoic acid and arachidonic 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.

Su KP. Biological mechanism of antidepressant effect of omega-3 fatty acids: How does fish oil act as a 'mind-body interface'? *Neurosignals*, Vol. 17, pp. 144-152, 2009

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.

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, noradrenaline and/or dopamine

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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 ^c
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.

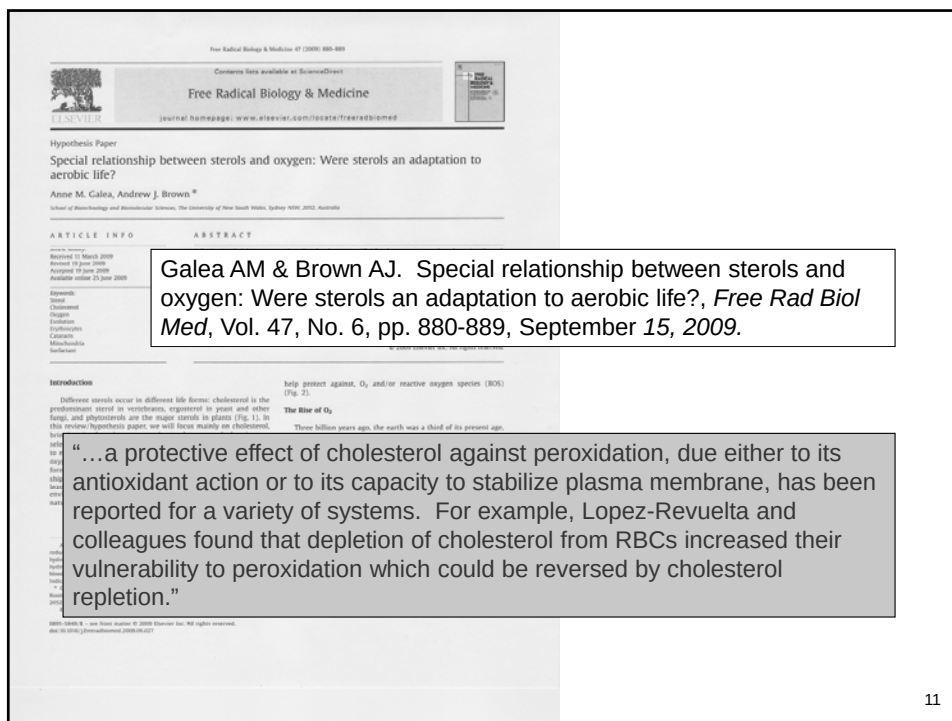
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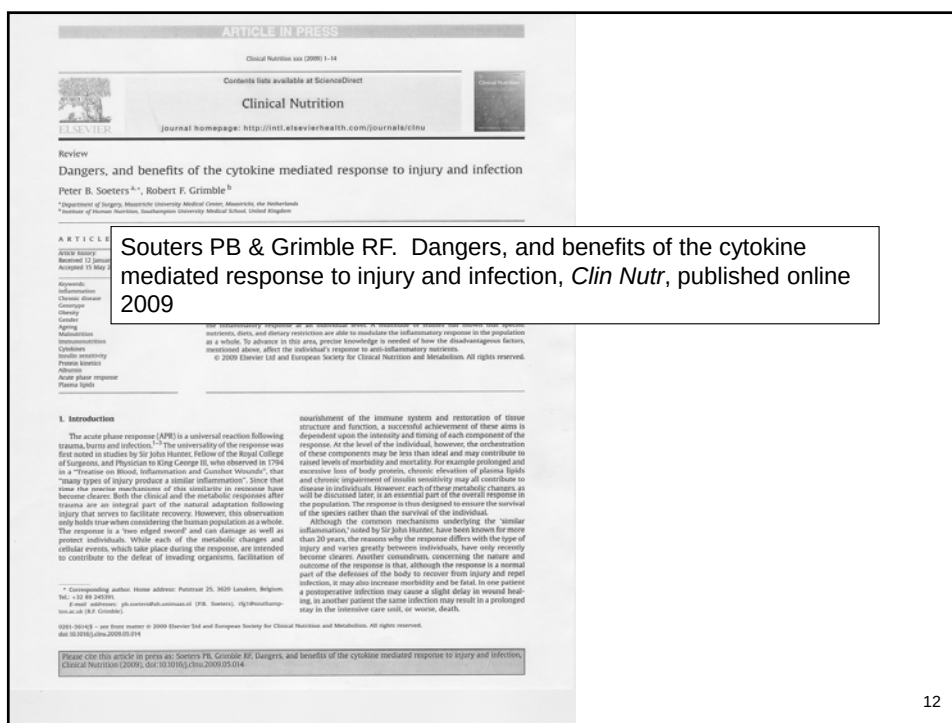
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- “Endotoxin, via stimulation of pro-inflammatory cytokine production, induces increased plasma lipid concentrations due to increased synthesis and secretion of triglyceride-rich lipoproteins by the liver and inhibition of lipoprotein lipase.”
- “This effect leads to hyperlipoproteinemia and hypertriglyceridemia, which increases the availability of substrate for the immune system and wound healing, but can also bind and neutralize LPS.”
- *“It has therefore been postulated that triglyceride-rich lipoproteins (very low-density lipoproteins and chylomicrons) are also components of an innate, non-adaptive host immune response to infection.”*

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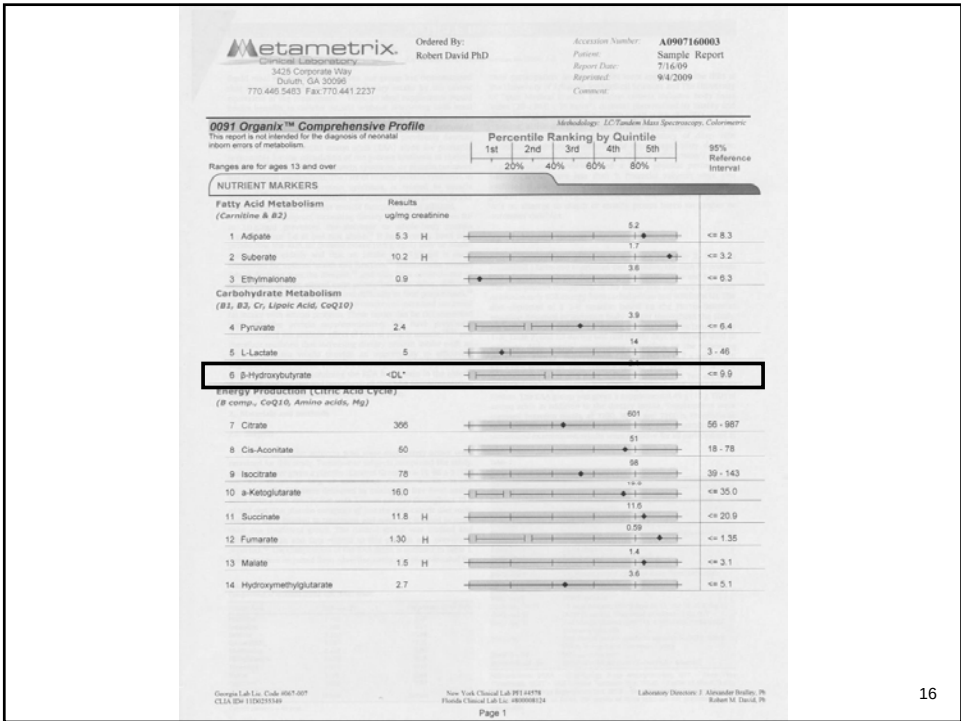
- ***“In experimental settings it is has indeed been demonstrated that lipoproteins can bind LPS, protect against LPS-induced toxicity, and play an important role in host response to LPS.”***

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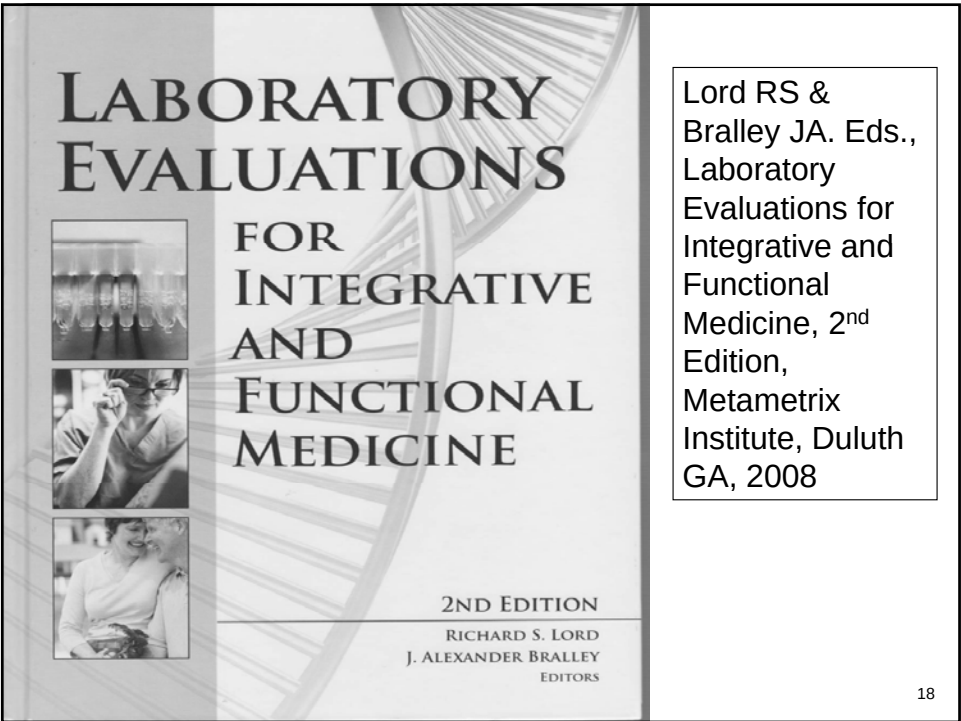
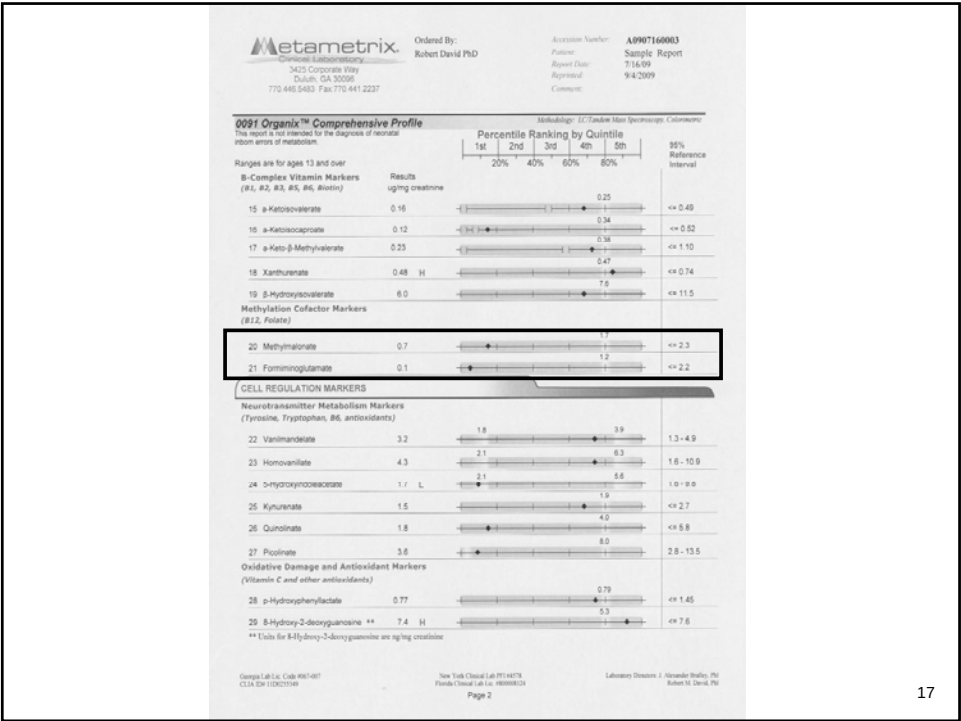
Organic Acids

Introduction

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- “The term ‘organic acid’ refers to a broad class of compounds used in fundamental metabolic processes of the body.”
- “Chemically, organic acids share the common features of water solubility, acidity, and ninhydrin negativity (no primary or secondary amines).”
- “The term is generally considered to include all carboxylic acids, with or without keto-, hydroxyl-, or other non-amino functional groups, but does not include most amino acids.”

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CHAPTER 6

TABLE 6.1 — SUMMARY OF ABNORMALITIES FOR ORGANIC ACIDS IN URINE

Name	Potential Intervention	Metabolic Pathway
Fatty Acid Oxidation		
Adipate	H: L-Carnitine, 500-1000 mg TID	Fatty acid oxidation
Suberate	H: L-cysteine (if low), 500 mg TID; B2, 100mg BID	
Ethylmalonate	H: See text for other interventions in genetic disorders	
Carbohydrate Metabolism		
Pyruvate	H: B ₁₂ up to 100mg TID with B complex support; For concurrent H Lactate: lipoic acid, 500mg TID	Aerobic/anaerobic energy production
Lactate	H: Coenzyme Q ₁₀ , 50 mg TID	
β-Hydroxybutyrate	H: Chromium picolinate, 200 µg BID	Balance of fat and CHO metabolism
Energy Production (Citric Acid Cycle)		
Citrate	H: Arginine, 1-3 gm/day	
	L: Aspartic acid, 500 mg; magnesium citrate, 500 mg	
Cit-aconitate	H: Cysteine, 1000 mg BID; Check for iron deficiency	Renal ammonia clearance
Isocitrate	H: Lipoic acid, 25 mg/kg/day	
	Magnesium, 400 mg; manganese, 20 mg	
α-Ketoglutarate	L: α-KG, 300 mg; arginine, 1000 mg; glutamine, 1-3 g	
	H: B-complex, 1 TID; lipoic acid 100 mg	
Succinate	L: Botulinum, 1000 mg TID; valine, 1000 mg TID	
	H: CoQ ₁₀ , 50 mg TID; magnesium, 300 mg	
Fumarate	L: Tyrosine, 1000 mg BID; phenylalanine, 500 mg BID	
Malate	H: CoQ ₁₀ , 50 mg TID; B ₁₂ , 100 mg TID	
Hydroxymethylglutarate	L/H: CoQ ₁₀ , 50 mg TID	(L) Substrate-limited CoQ ₁₀ synthesis (H) HMG-CoA reductase inhibition
B-Complex Vitamin Metabolism		
α-Ketoglutarate	H: B-complex, 1 TID; lipoic acid 100 mg	Valine catabolism
α-Ketoglutarate	H: B-complex, 1 TID; lipoic acid 100 mg	Leucine catabolism
α-Keto-β-methylvalerate	H: B-complex, 1 TID; lipoic acid 100 mg	Isoleucine catabolism
Xanthurenate	H: Vitamin B ₆ , 100 mg/d	Tryptophan catabolism (hepatic)
β-Hydroxyisovalerate	H: Biotin, 5 mg/day; magnesium, 100 mg BID	Isoleucine catabolism
Methylmalonate or Propionate	H: B ₁₂ , 1000 µg TID	Valine or odd-chain fatty acid catabolism
Formiminoglutamate	H: Folic acid, 400 µg/d	Histidine catabolism
Neurotransmitter Metabolism		
Vanilmandelate	L/H: Tyrosine, 1000 mg BID-TID, between meals and phenylalanine hydroxylase cofactors as needed	(L) Tyrosine-limited or (H) Tyrosine-depleting epinephrine & norepinephrine catabolism
Homovanillate	L/H: Contraindicated for patients taking MAO inhibitors	(L) Tyrosine-limited or (H) Tyrosine-depleting DOPA catabolism
5-Hydroxyindolacetate	L/H: 5-Hydroxytryptophan, 50-100 mg TID; Magnesium, 300 mg; vitamin B ₆ , 100 mg (5-HTP may be contraindicated with 5HTF)	(L) Tryptophan-limited or (H) Tryptophan-depleting Serotonin catabolism
Kynurenate	H: B ₆ , 100 mg; magnesium, 300 mg	Inflammation-stimulated macrophage and astrocyte kynurenine pathway activity
Quinolinate	H: Magnesium, 300 mg	

Table 6.1 continued on following page

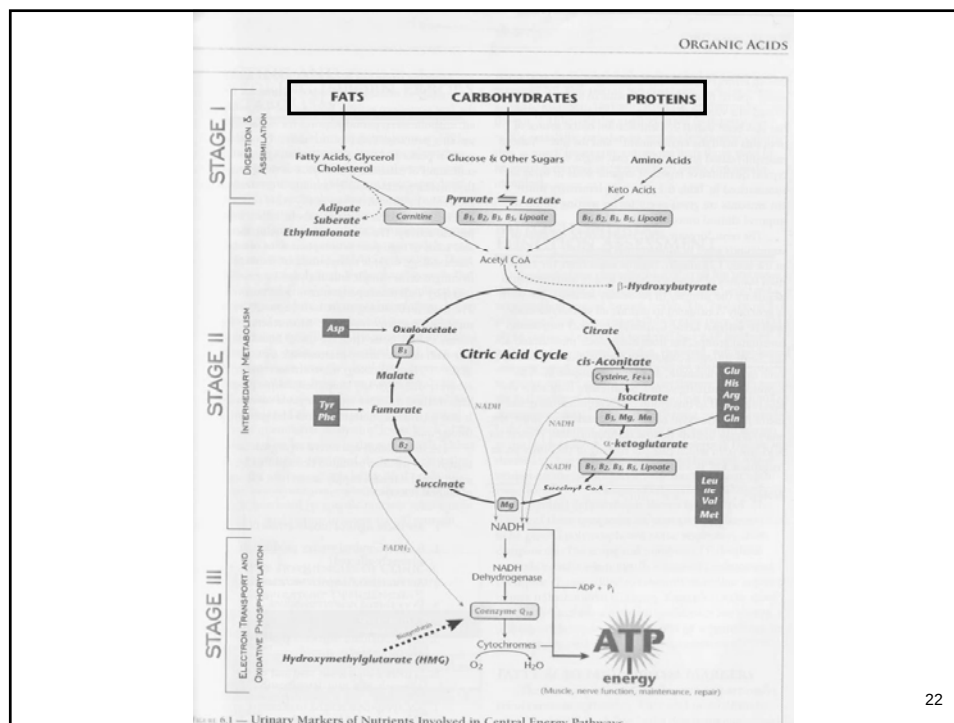
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Big Picture concepts

Organic acids are “acids.”

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Methylmalonate as an indicator of vitamin B12 deficiency

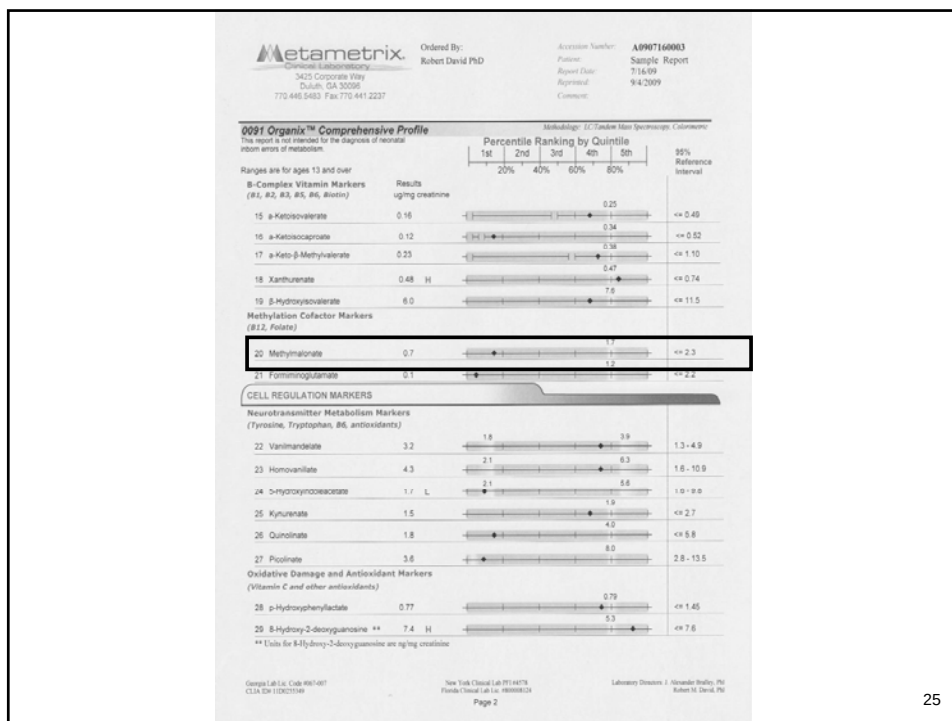
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- “Methylmalonyl-CoA is produced in the multistep process of breaking down the amino acids isoleucine, valine, methionine, and threonine, or from catabolism of odd-chain fatty acids.”
- “Methylmalonate (MMA) is converted into succinic acid by a vitamin B12-dependent enzyme, methylmalonyl-CoA mutase.”
- “The lack of vitamin B12 impairs this conversion as well as the one in which methionine is recovered from homocysteine (tHCYS), leading to accumulation of both tHCYS and MMA.”
- “Thus, an increase in MMA or tHCYS, can signal a vitamin B12 deficiency.”

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Formiminoglutamate (FIGLU)
as an indicator of folate
deficiency

- “Formiminoglutamic acid (FIGLU) is an intermediate in the deamination of the amino acid histidine.”
- “Folic acid is the cofactor required by the enzyme formiminotransferase that converts FIGLU to glutamic acid.”

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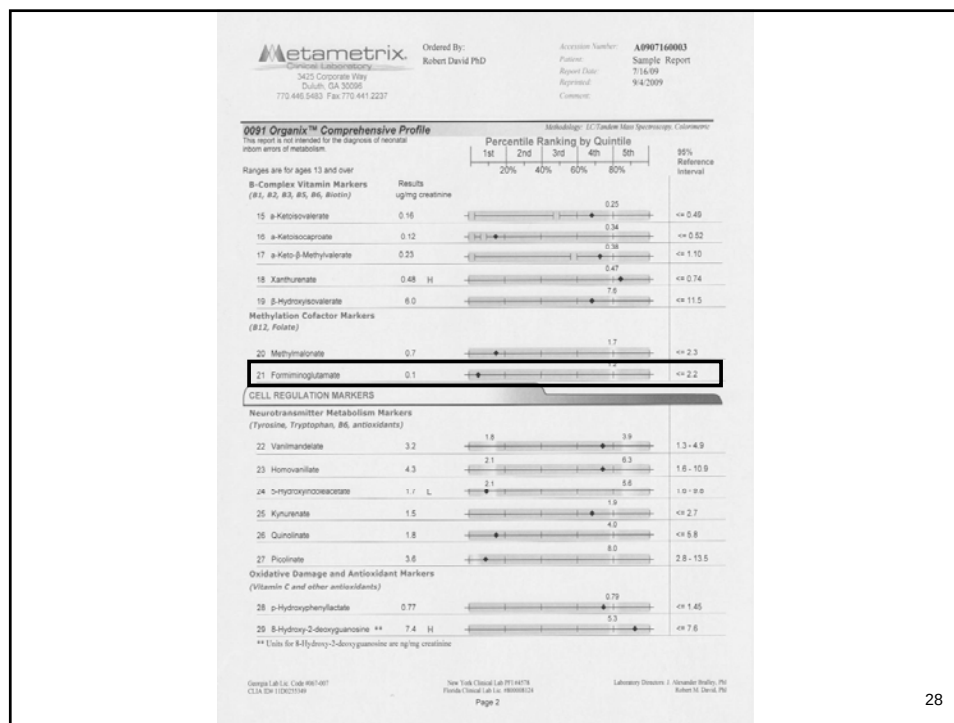


TABLE 6.7 — DIFFERENTIATING B₁₂, FOLATE, AND B₆ DEFICIENCY

Scenario	Markers	Potential Deficiency
1	Homocysteine ↑ with no other test result	Either B ₁₂ , folate or B ₆ , or methyl donor compounds
2	Methylmalonate ↑ with Homocysteine and FIGLU normal	B ₁₂
3	Homocysteine ↑ with normal methylmalonate	B ₆ or folate
4	Both Homocysteine ↑ and methylmalonate ↑	B ₁₂ , folate, and B ₆
5	Homocysteine ↑ with MMA and FIGLU normal	B ₆ and possible methyl donor compounds
6	HCys, MMA and FIGLU all ↑	B ₆ , B ₁₂ , folate and methyl donor compounds

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**β-Hydroxybutyrate as an
indicator of glucose/insulin
dysregulation**

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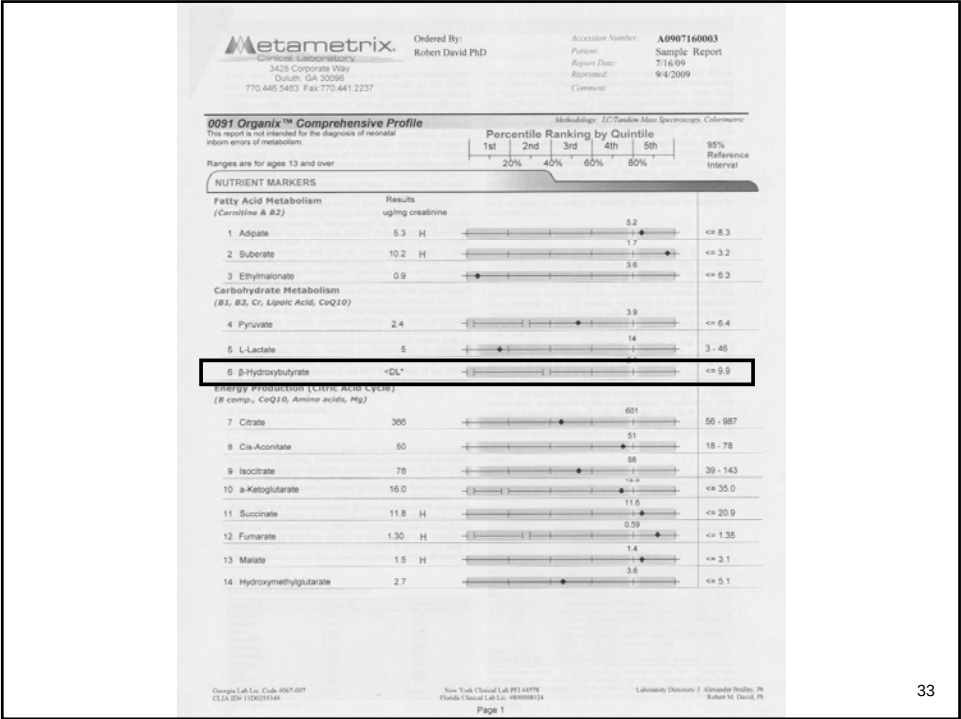
- “ β -Hydroxybutyrate is a ketone body.”
- “Ketone bodies are a feature of metabolic acidosis due to failure of glucose utilization as with diabetes.”
- “Ketone body production increases in diabetes because the oxidation of free fatty acids is stimulated, and excess acetyl-CoA is converted to the four-carbon organic acid, β -hydroxybutyrate.”

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- “Elevations of β -hydroxybutyrate in an overnight urine collection may indicate inefficient utilization or mobilization of glucose.”
- “Restriction of dietary carbohydrate, a common dietary approach to weight loss, produces elevated β -hydroxybutyrate.”

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Thank you!!

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