

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

Part II

Jeffrey Moss, DDS, CNS, DACBN

jeffmoss@mossnutrition.com

413-530-0858 (cell)

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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 NEVIN BACH

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. Olgard, a second-year medical student, said: "I often wondered what we were actually doing to help people," Mr. Olgard said.

Not surprisingly, 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.

For as young as 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 givers.

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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**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.”

KARGER



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Organic Acids Part 2

Dr. Jeff Moss

Clinical Nutrition (2004) 23, 1256-1266



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

Clinical
Nutrition

www.elsevier.com/locate/clinu

Bengmark S. Acute and "chronic" phase reaction – a mother of disease, *Clin Nutr*, Vol. 23, pp. 1256-66, 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 diseases;
Atherosclerosis;
Coronary-vascular diseases;
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-concentrated food under consumption of a function of the innate immune changes induced in response the subsequent development chronic diseases. The end importance, and 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, P-1 and free fatty acids compared to elsewhere in the body. The load on the liver of these various substances is 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 meta metabolic syndrome are discussed separately. The need for studies with lifestyle modifications is especially emphasized.

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E-mail address: S.bengmark@ucl.ac.uk (S. Bengmark).

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doi:10.1016/j.clnu.2004.07.016

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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	Optimize pH	Optimize fluid and electrolyte balance	Morning urinary pH -pH paper	Potassium bicarbonate -K Alkaline (Moss) Magnesium -Magnesium Glycinate (Vital Nutrients)
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 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: 10.1159/000168182

Received April 26, 2009
Accepted after revision June 5, 2009
Published online February 5, 2010

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 (PMB-LAB), China Medical University and Hospital, Taichung, Taiwan, Stress, Psychiatry and Immunology Laboratory (SPI-LAB), Institute of Psychiatry, King's College London, London, UK

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

Key Words
Major depressive disorder · Depressive acid · Docosahexaenoic acid · Arachidonic acid · Prostaglandins · Thromboxanes · Lipoxygenase A2 · Cyclo-oxygenase

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.

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

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1662-4585/09/1702-0144\$30.00
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Dr. Kuan-Pin Su
Department of Psychiatry
China Medical University Hospital
No. 2, Yuh-Der Road, Taichung 404 (Taiwan)
Tel. +886 4 2205 2171, ext. 1076; Fax +886 4 2205 1230; E-Mail: kshsu@cmuh.org.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 ^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 ^c
Anorexia	13–19	40
Anxiety	13–18 ^d	57
Poor concentration	14 ^d	51

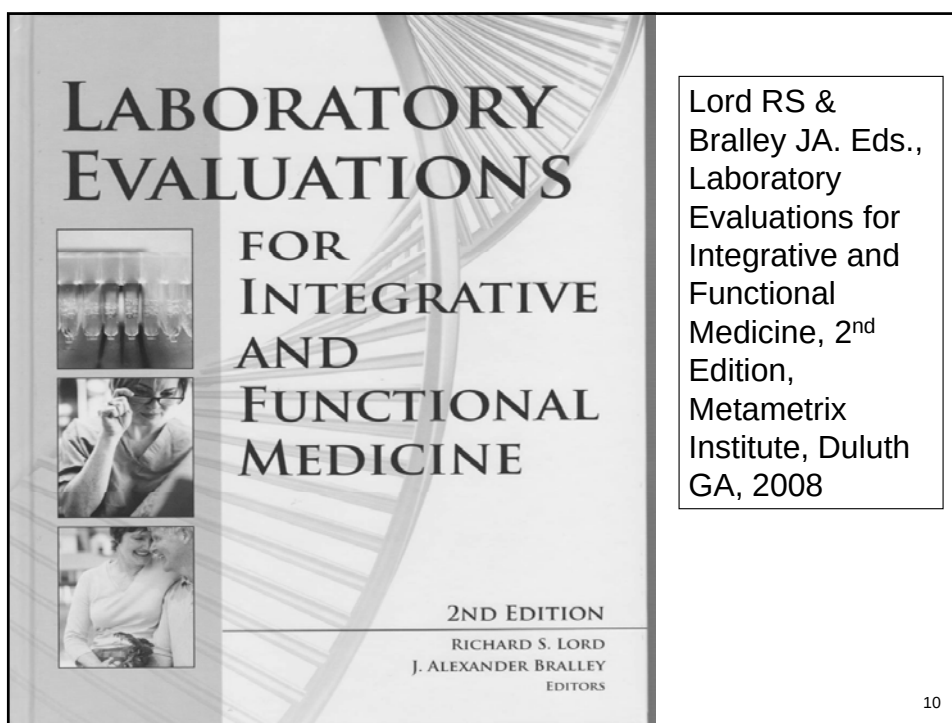
^a [46], unless otherwise specified; ^b [99], unless otherwise specified; ^c [100]; ^d [101]; ^e [102]; ^f [103]; ^g [104].

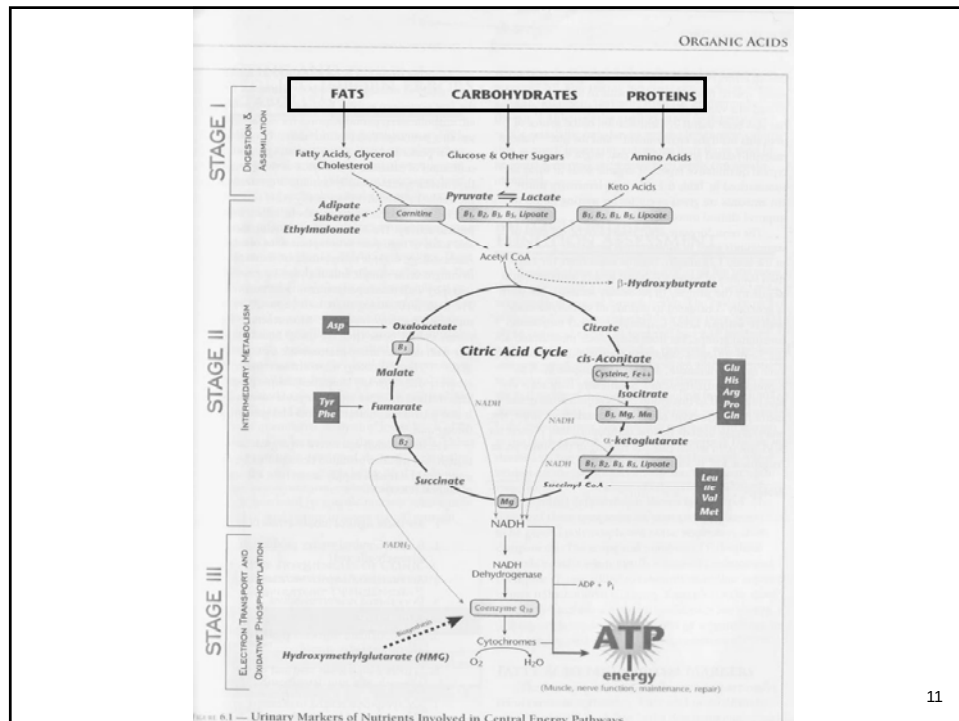
¹ Nausea, vomiting, bowel problems.

² Result from depressed inpatient population.

Organic acids

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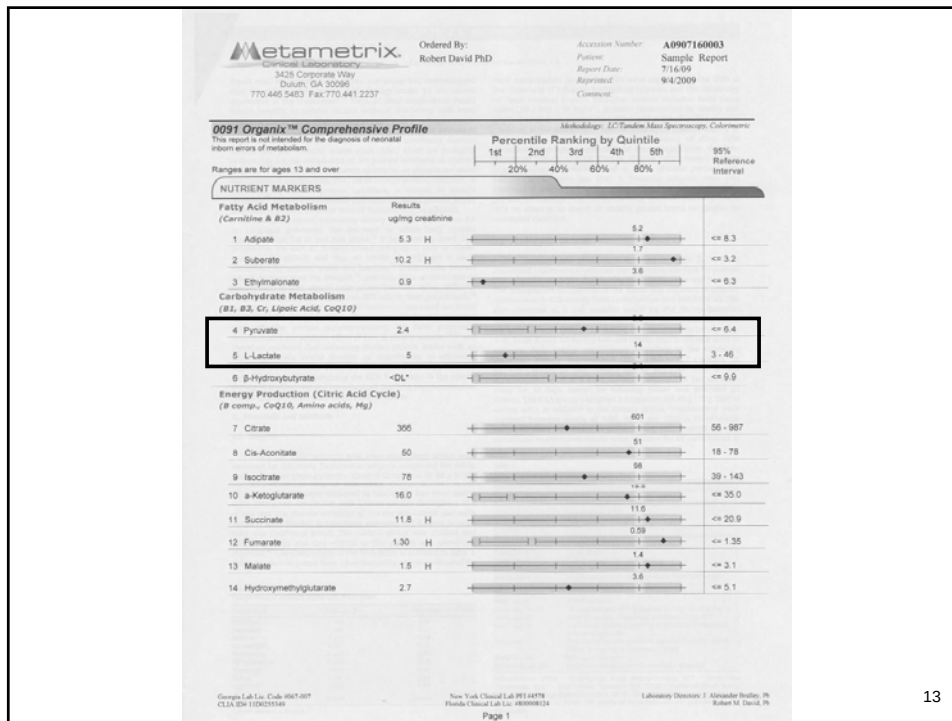


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Pyruvate and lactate as major indicators of catabolic physiology and overall severity of chronic illness

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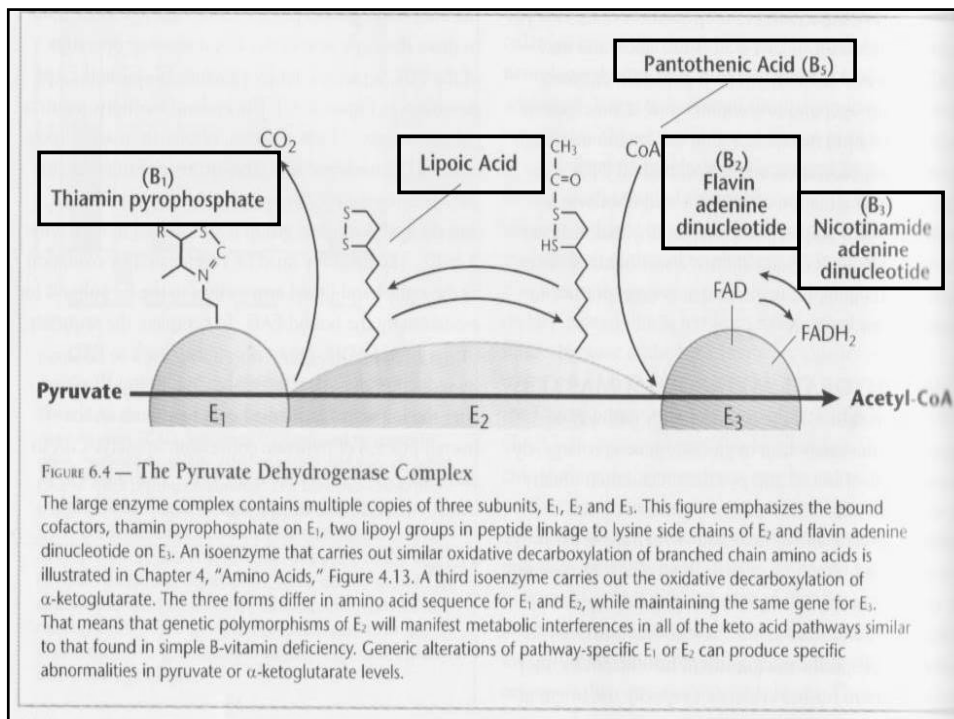
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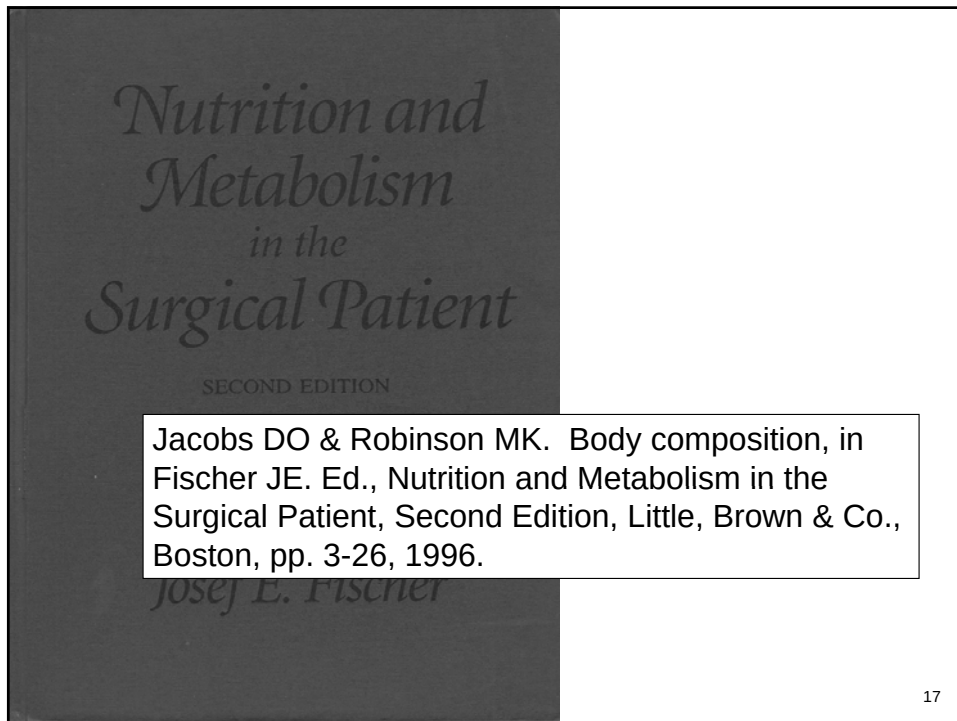
- “Abnormalities of urinary excretion of pyruvate and lactate provide useful insight to basic metabolic factors due to their position in the energy production process.”
- “Pyruvate and lactate are anaerobic breakdown products of glucose.”
“Under conditions favoring anaerobic metabolism, pyruvate is reduced to lactate by the action of lactate dehydrogenase.”

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- “Conditions favoring aerobic metabolism increase the clearance of pyruvate by oxidation to acetyl-CoA, avoiding a buildup of lactate.”
- “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.”

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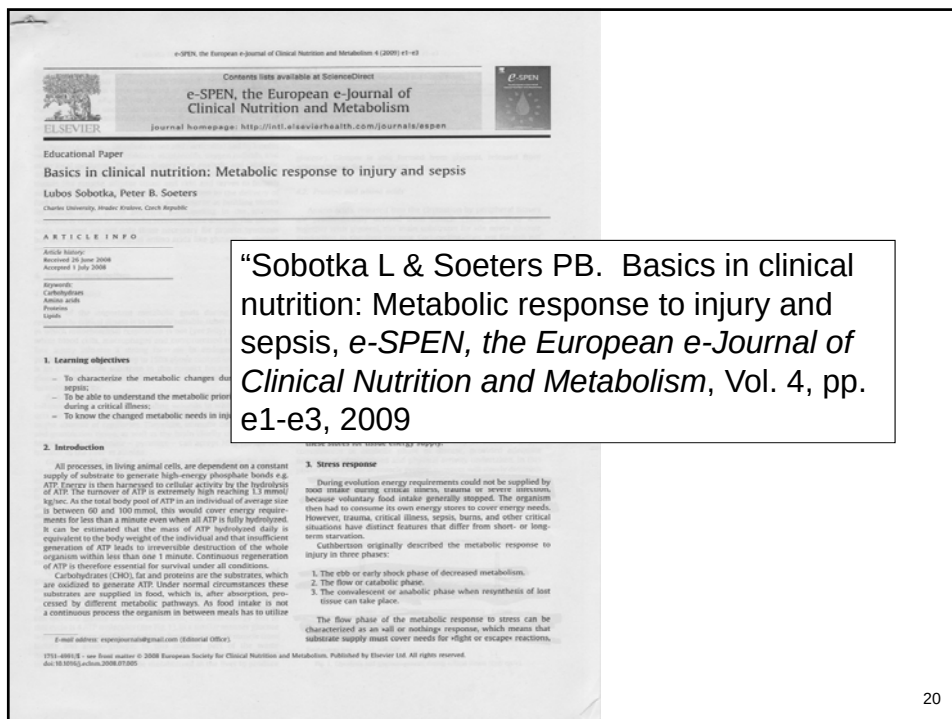


- "...within the first 24 hours of illness, glycogen stores are rapidly depleted, and thus muscle amino acids, glycerol, lactate, and pyruvate released from the periphery are used as precursors for accelerated hepatic gluconeogenesis."
- "The magnitude of increase in glucose production is proportional to the extent of illness."
- "The glucose produced is used by the central nervous system, which specifically requires carbohydrate to meet its energy needs, although its rate of utilization is relatively normal."
- "The remainder of the glucose is used for reparative processes in wounds or in other injured tissues."

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- “Injured tissue abounds with inflammatory cells, erythrocytes, and fibroblasts.”
- “Cells populating such tissue undergo anaerobic metabolism and produce large quantities of lactate.”
- “The lactate is then transported back to the liver for resynthesis of glucose that can then be reused by tissues undergoing repair (the Cori cycle).”

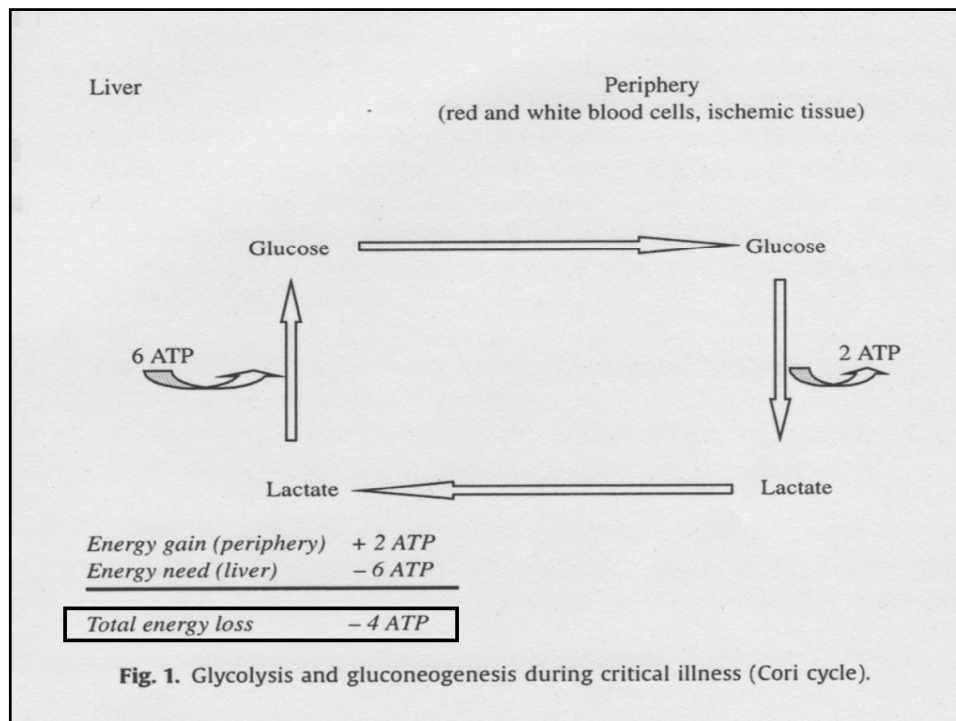
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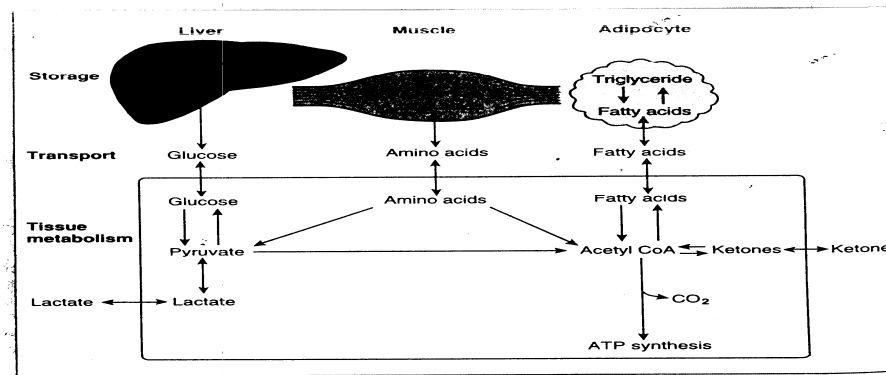
- “Quantitatively, lactate is the most important precursor of gluconeogenesis.”
- “As this substrate is the result of anaerobic glucose metabolism, the glucose carbons circulate between peripheral tissues and liver (Cori cycle).”
- “Under normal conditions about 150 g of lactate is metabolized in the liver, but this amount can increase greatly in stress conditions.”
- “*The total energy loss in this cycle is 4 ATP molecules.*”

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Organic Acids Part 2
Dr. Jeff Moss

Interconversion of Metabolic Fuels



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

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Lactic Acidosis [Print] - eMedicine Critical Care

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eMedicine
eMedicine Critical Care

eMedicine Specialties > Critical Care > Medical Topics

Lactic Acidosis

Kyle J Gunnerson, MD, Associate Professor, Departments of Anesthesiology/ICM and Emergency Medicine, Virginia Commonwealth University; Director of Critical Care Anesthesiology, Co-Director of Cardiac Surgery ICU, Virginia Commonwealth University Health System
 Sat Sharma, MD, FRACP, Professor and Head, Division of Pulmonary Medicine, Department of Internal Medicine, University of Manitoba, Site Director, Respiratory Medicine, St Boniface General Hospital
 Updated: Jul 22, 2009

Introduction

Background

By the turn of the 20th century, many physicians recognized that that patients who are critically ill could exhibit

Gunnerson KJ & Sharma S. Lactic acidosis, eMedicine Critical Care, Medscape.com.

moderate (2-5 mmol/L) persistent increase in blood lactate concentration without metabolic acidosis, whereas lactic acidosis is characterized by persistently increased blood lactate levels (usually > 5 mmol/L) in association with metabolic acidosis.

Pathophysiologic classification of lactic acidosis

Lactic acidosis generally occurs in the setting of impaired tissue perfusion, altered buffering systems, and inadequate renal excretion. Lactic acidosis is associated with major metabolic derangements. Acute lactic acidosis is often the result of severe hypoxia, leading to anaerobic metabolism and subsequent lactic acid production. Other causes include severe liver disease, which impairs the ability of the liver to clear lactate, and renal failure, which impairs the ability of the kidneys to excrete lactate. Lactic acidosis can also be caused by a variety of drugs, including metformin, which can cause lactic acidosis in patients with renal impairment. Other causes include severe liver disease, which impairs the ability of the liver to clear lactate, and renal failure, which impairs the ability of the kidneys to excrete lactate. Lactic acidosis can also be caused by a variety of drugs, including metformin, which can cause lactic acidosis in patients with renal impairment.

Compensated lactic acidosis is necessary to reduce effects of acidosis, such as effects on cardiovascular, pulmonary, and renal systems. In severe lactic acidosis, the body's compensatory mechanisms are overwhelmed, and the resulting acidosis can lead to multi-organ dysfunction and death. Lactic acidosis is a medical emergency, and prompt treatment is essential to prevent complications and improve outcomes.

Lactic acidosis may not necessarily produce problems in a patient. The development of lactic acidosis depends on the underlying cause of the acidosis, the buffering capacity of the body, and the presence of other conditions that produce acidosis. In some cases, lactic acidosis may be a benign finding, while in other cases it may be a sign of a serious underlying condition. The clinical significance of lactic acidosis depends on the underlying cause and the patient's overall health status.

Further Readings

Therapeutic management of lactic acidosis involves the treatment of the underlying cause of the acidosis and the use of bicarbonate to correct the acidosis. In some cases, dialysis may be necessary to remove lactate from the body. The goal of treatment is to reduce lactate levels and restore normal acid-base balance.

http://emedicine.medscape.com/article/167027-print

2/14/2010

Pathophysiological classification of lactic acidosis	
HYPOXIC	NON-HYPOXIC
Ischemia	Delayed Clearance
Shock, severe anemia, cardiac arrest	Renal or hepatic dysfunction
Global Hypoxia	→ Pyruvate Dehydrogenase Dysfunction
Carbon monoxide poisoning	→ Sepsis, thiamine deficiency, catecholamine excess, alcoholic and diabetic ketoacidosis
Respiratory Failure	Uncoupling of Oxidative Phosphorylation
Severe asthma, COPD, asphyxia	→ Cyanide, salicylates, methanol & ethylene glycol metabolites, anti-retroviral drugs, valproic acid, biguanides, INH
Regional Hypoperfusion	Accelerated Aerobic Glycolysis
Limb or mesenteric ischemia	→ Increased effort, sepsis, seizures, large fructose loads, malignancies

Pathophysiologic classification of lactic acidosis.

- "...hyperlactemia in patients with sepsis is a marker of the severity of stress response."
- Hyperlactemia may possibly develop as a byproduct of overall acceleration in glycolysis in severe sepsis."
- "This may well be an adaptive host mechanism designed to provide efficient generation of energy in response to severe stress."

Lactate versus non-lactate metabolic acidosis: a retrospective outcome evaluation of critic... Page 1 of 1

MEDLINE Abstract

Lactate versus non-lactate metabolic acidosis: a retrospective outcome evaluation of critically ill patients.
Crit Care. 2006; 10(1):R22 (ISSN: 1466-609X)

Gunnerson KJ ; Saul M ; He S ; Kellum JA
Virginia Commonwealth University Reanimation Engineering Shock Center Laboratory, Department of Anesthesiology/Critical Care, Medical College of Virginia/Virginia Commonwealth University, 1200 East Broad Street, Richmond, VA 23298, USA.

INTRODUCTION: Acid-base abnormalities are common in the intensive care unit (ICU). Differences in outcome exist between respiratory and metabolic acidosis in similar pH ranges. Some forms of metabolic acidosis (for example, lactate) seem to have worse outcomes than others (for example, chloride). The relative incidence of each type of disorder is unknown. We therefore designed this study to determine the nature and clinical significance of metabolic acidosis in critically ill patients. METHODS: An observational, cohort study of critically ill patients was performed in a tertiary care hospital. Inpatient mortality of the ICU of 5.8 days. RESULTS: January 2001 and 30. of arterial lactate level disorder was absent. A a metabolic acidosis (for those with no meta predominant anion pre (56%); for strong ion g logistic regression mo CONCLUSION: In cri strong independent pr with metabolic acidosis were nearly twice as likely to die as patients without metabolic acidosis.

PreMedline Identifier:16507145

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- “In critically ill patients in which a measurement of lactate level was ordered, lactate and strong anion gap were strong independent predictors of mortality when they were the major source of metabolic acidosis.”
- “Overall, patients with metabolic acidosis were nearly twice as likely to die as patients without metabolic acidosis.”

REVIEW

Biology of Cachexia

Michael J. Tisdale*

About half of all cancer patients show a syndrome of cachexia, characterized by loss of adipose tissue and skeletal muscle mass. Such patients have a decreased survival time, compared with the survival time among patients without weight loss, and loss of total body protein leads to substantial impairment of respiratory muscle function. These changes cannot be fully explained by the accompanying malnutrition and anorexia, which is a wasting process. Despite a falling caloric intake, cachectic patients frequently show an elevated expenditure as a result of increases in Cori cycle conversion of lactic acid to glucose, and triglyceride-fatty acid cycling, and glucose turnover of cytokines, including tumor necrosis factor-1 and 6, interferon γ , and leukemia-inhibitory factor. However, the results of a number of clinical studies suggest that the action of the cytokines is able to explain the complex mechanism of wasting in cancer patients. In addition, cachexia has been observed in some xenograft models even without a cytokine involvement, suggesting that other factors may be involved. These probably include catabolic factors, which act directly on skeletal muscle and adipose tissue and the presence of which has been associated with the clinical development of cachexia. A polyunsaturated fatty acid, docosahexaenoic acid, attenuates the action of such catabolic factors and has been shown to stabilize the process of wasting and resting energy expenditure in patients with pancreatic cancer. Such a pharmacologic approach may provide new insights into the treatment of cachexia. (*J Natl Cancer Inst* 1997;89:1763-73)

The word "cachexia" is derived from the Greek "kakos" meaning "bad" and "hexis" meaning "condition." It occurs in a number of disease states, including cancer, acquired immunodeficiency syndrome (AIDS), major trauma, surgery, malabsorption, and severe sepsis. Cachexia is characterized by weight loss involving depletion of both adipose tissue and skeletal muscle mass. Weight loss in cancer patients differs from that found in simple starvation. During the first few days of starvation, glucose utilization by the brain and erythrocytes necessitates depletion of liver and muscle glycogen and an increased glucose production by the liver, using gluconeogenic amino acids derived from catabolism of muscle. This early phase is replaced in long-term starvation by the use of fat as a fuel, in which free fatty acids released from adipose tissue are converted into ketone bodies, which are utilized for energy by peripheral tissues and eventually to a great extent by the brain. This leads to conservation of muscle mass. In anorexia nervosa, more than three quarters of the weight loss arises from fat and only a small amount from muscle. In contrast, in cancer cachexia, there is equal loss of both fat and muscle, so that for a given degree of

Tisdale MJ. Biology of cachexia, *J Nat Cancer Inst*, Vol. 89, No. 23, pp. 1763-73, December 3, 1997

cancer patients through dietary counseling failed to reverse the cachexia (8). Trials of total parenteral nutrition in cachectic cancer patients also failed to show benefit in terms of increased median survival time or long-term weight gain (9). Although a short-term weight gain was observed, this weight was subsequently lost, suggesting the retention of water (7). Analysis of body composition indicated that patients receiving total parenteral nutrition temporarily maintained body fat stores, but there was no evidence for preservation of lean body mass. Thus, the cause of wasting in cancer cachexia is more complex than that in simple starvation.

Anorexia and Cachexia

Although anorexia alone is unlikely to be responsible for the wasting seen in cancer patients, it may be a contributing factor. In addition, its presence is an extremely distressing syndrome because appetite and the ability to eat have been reported to be the most important factors in the physical and psychological aspects of a patient's quality of life (10). Anorexic cancer patients often report early satiety, which, together with a reduced appetite has been postulated to be caused by the production of factors by the tumor that exert their effects by acting on the hypothalamus.

*Correspondence to: Michael J. Tisdale, PhD, D.Sc., Pharmaceutical Sciences Institute, Aston University, Birmingham B4 7ET, U.K. E-mail: m.j.tisdale@aston.ac.uk

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REVIEW 1763

Journal of the National Cancer Institute, Vol. 89, No. 23, December 3, 1997

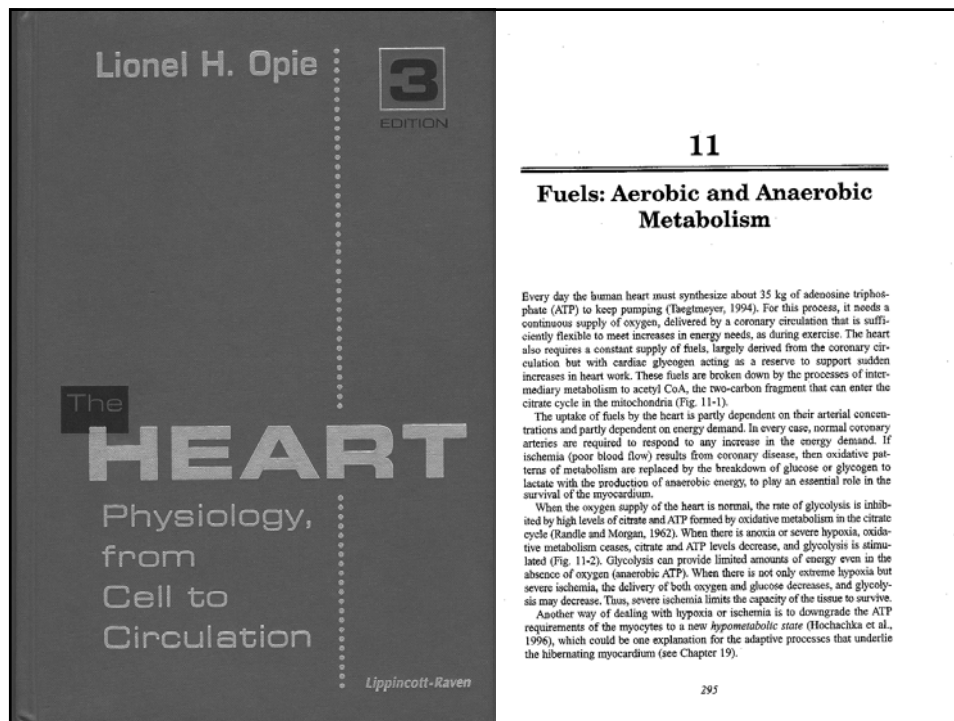
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- “Patients with progressive cancer have been shown to have an increased glucose synthesis not only from lactate, but also from alanine and glycerol.”
- “The Cori cycle normally accounts for 20% of glucose turnover, but it was shown to be increased to 50% in cachectic cancer patients, accounting for the disposal of 60% of the lactate produced.”

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Fuels of the heart Pyruvate

- “Pyruvate stands at the crossroads of glycolysis and oxidative metabolism in the citrate cycle. It is formed from lactate taken up by the heart and from glycolysis.”
- “In the anaerobic heart, pyruvate forms lactate. In the aerobic heart, the major fate of pyruvate is oxidative decarboxylation and entry into the citrate cycle, which requires the activity of pyruvate dehydrogenase.”

CHAPTER 11, FUELS: AEROBIC & ANAEROBIC METABOLISM 303

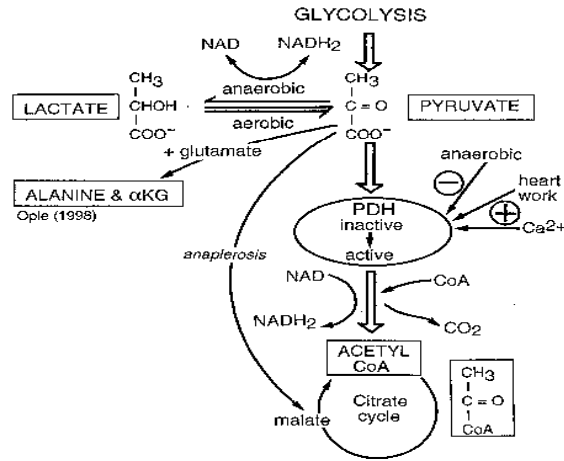
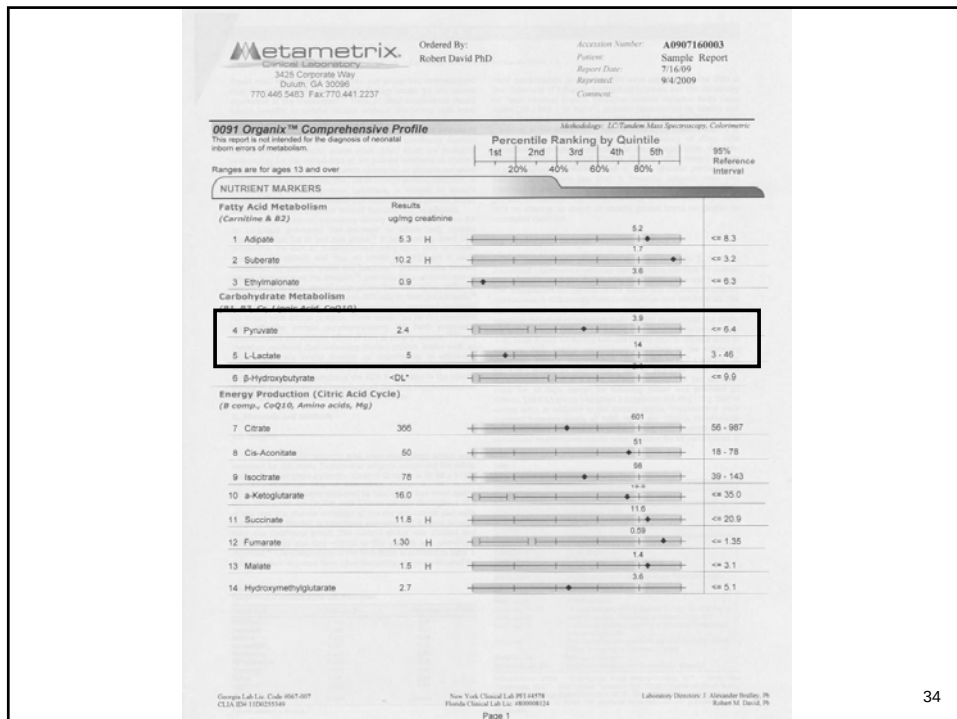


FIG. 11-6. The major fates of pyruvate. In the normal aerobic heart, lactate is taken up and converted to pyruvate, the major part of which enters the citrate cycle. Transamination is a pathway of minor significance. In the anaerobic heart, lactate is produced from pyruvate derived from glycolysis. There is also formation of alanine and the possibility of ATP production via GTP as succinate is ultimately formed. During postischemic reperfusion, pyruvate can be converted to malate to help replenish the citrate cycle intermediates by anaplerosis. α KG, α -ketoglutarate; PDH, pyruvate dehydrogenase.



Organic Acids Part 2

Dr. Jeff Moss

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/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) Iosol (Moss) Zinc Supreme (DFH)

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Thank you!!

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