

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

Part XI

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Sulfur metabolism – Part II: Diagnostic modalities and metabolic correction

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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.”

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REVIEW

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

Stig Bengmark

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

Received 25 July 2004

KEYWORDS
Acute phase response;
Chronic phase response;
Metabolic syndrome;
Cellular membranes;
Endothelial cells;
Abdominal obesity;
Insulin resistance;
Glycemic homeostasis;
Free fatty acids;
Alzheimer disease;
Parkinson disease;
Neurodegenerative diseases;
Atherosclerosis;
Coronary vascular disease;
Cancer;
Breast cancer;
Colorectal cancer;
Prostate cancer;
Diabetes;
Cystitis;
Fatty liver;
Leukodystrophy;
Chronic myeloid disease.

Summary—The world is in distress. Almost half of the mortality is due to these diseases. These diseases are associated with consumption of calorie-rich diets and underconsumption of fiber. The function of the liver is impaired. Changes in the liver are the subsequent development of chronic diseases. The end of the function of the underlying cells; their ability to obtain nutrients and antioxidants and to eliminate waste products. The endometrial adipocytes seem to play a key role, as they have the ability to in stressful situations release much of proinflammatory cytokines, TNF- α 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 response, and with 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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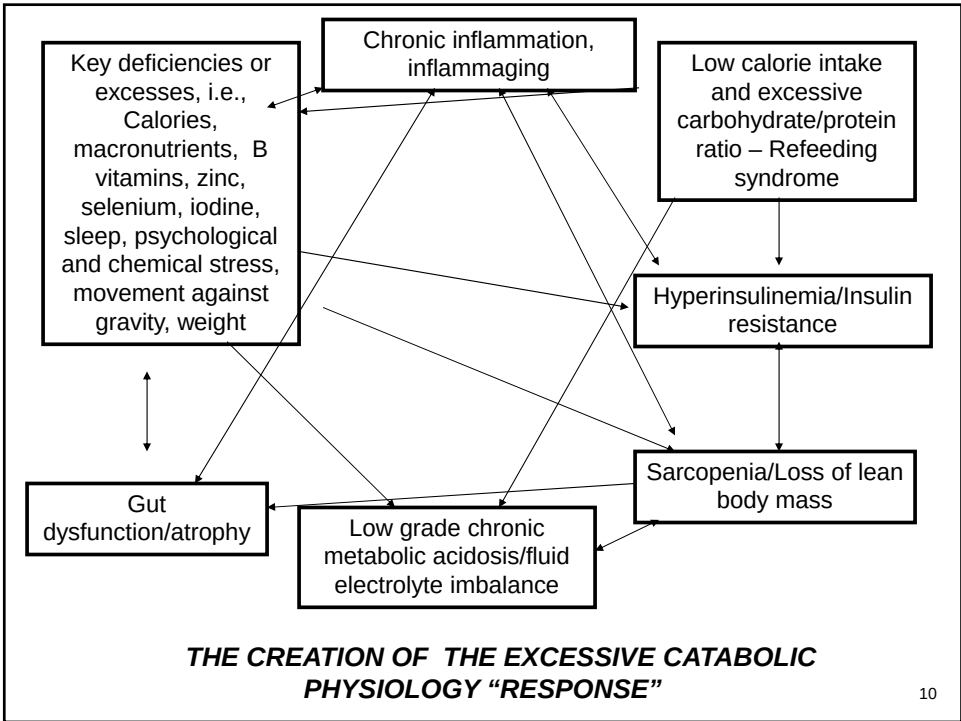
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Considerations for the Catabolic Patient				
Signs & Symptoms	Consider	Treatment Plan	Monitor	Supplementation
Signs & Symptoms: - Feeling "lousy" - Fatigue - Cravings - Depression/anxiety - Poor quality sleep - Poor digestion/absorption/leaky gut - 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 Baste (Metabolic Maint.) Protein powders/bars -UltraLean products (Biogenesis) -Palco products (DFH)
	Reducing inflammation	Reduce inflammation Address food allergies	Inflammatory biomarkers: -hs-CRP -Fibrinogen levels -WBC -Differential	Anti-inflammatory agents -Inflametric (Olympian) -D-Flame (Protocolt) -Pain Guard (Porque) 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 -Palco 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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Invited Review

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Neuro
SIGNALS

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 · Eicosanoid · Prostaglandins · Thromboxanes · Lipid metabolism · Cytosol · Cytochrome

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 etiology 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 behavior. Based on recent evidence, omega-3 polyunsaturated fatty acids (ω-3 PUFA), or ω-3 fatty acids, are enlightening a promising path to discover the unresolved depression sickness behavior and to tell the connection of mind and body. The PUFA are classified into n-3 (ω-3) and n-6 (ω-6) groups. Eicosapentaenoic acid and docosahexaenoic acid, the major bioactive components of ω-3 PUFA, 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 dysfunction of neuronal membrane stability and neurotransmitter

prostaglandin E₂ synthesis, which might be linked to the somatic manifestations and physical comorbidity in depression. The role of n-3 PUFA 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 PUFA 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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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

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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 ^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].

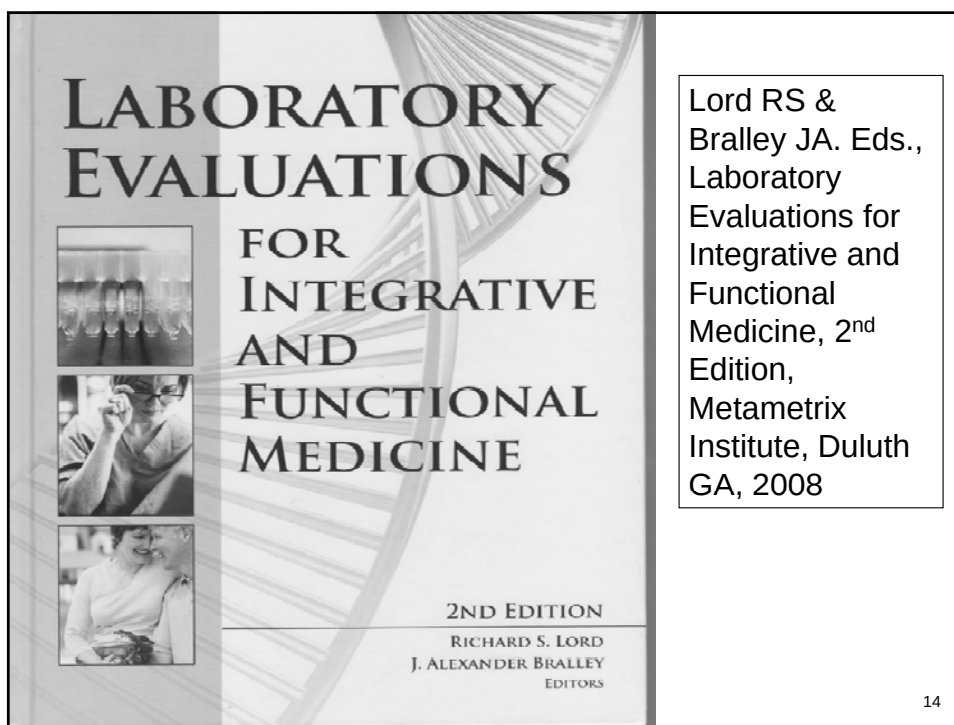
¹ Nausea, vomiting, bowel problems.

² Result from depressed inpatient population.

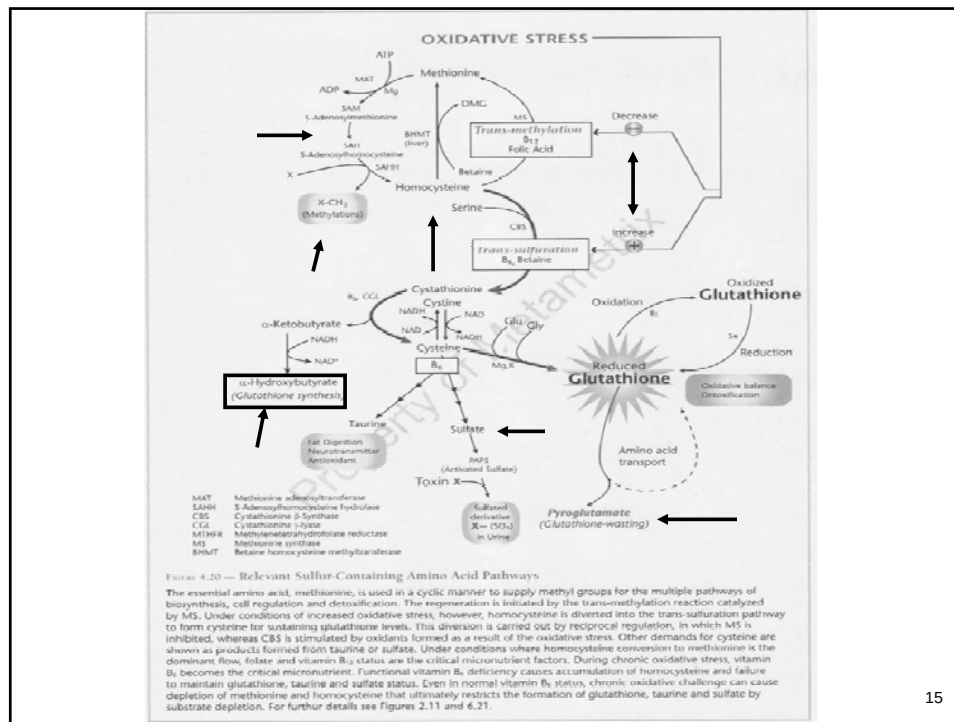
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Sulfur metabolism: The metabolic “big picture”

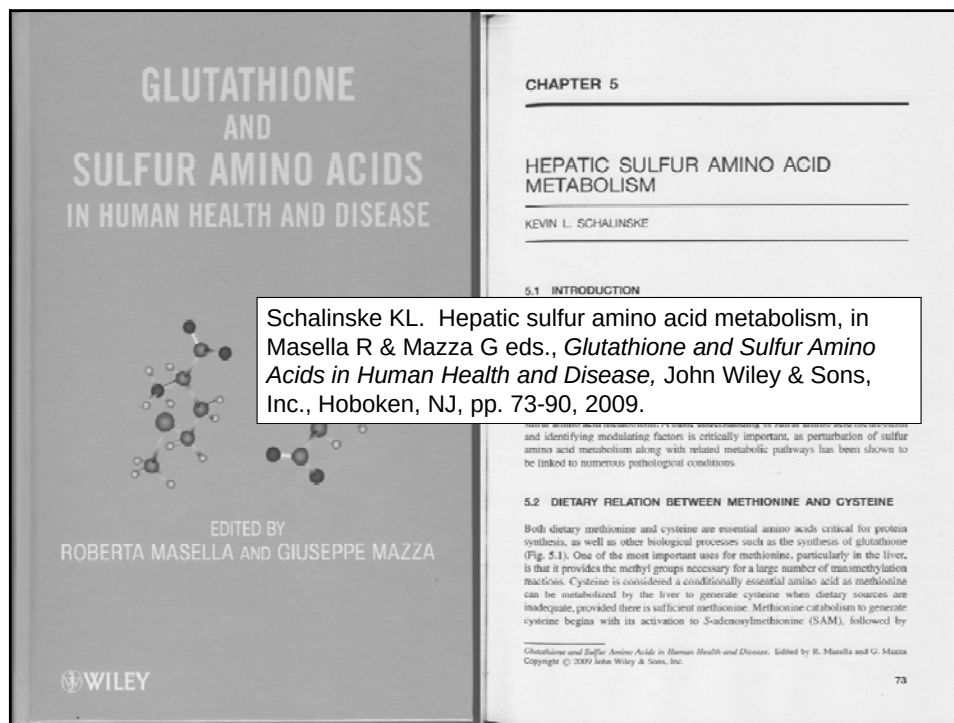
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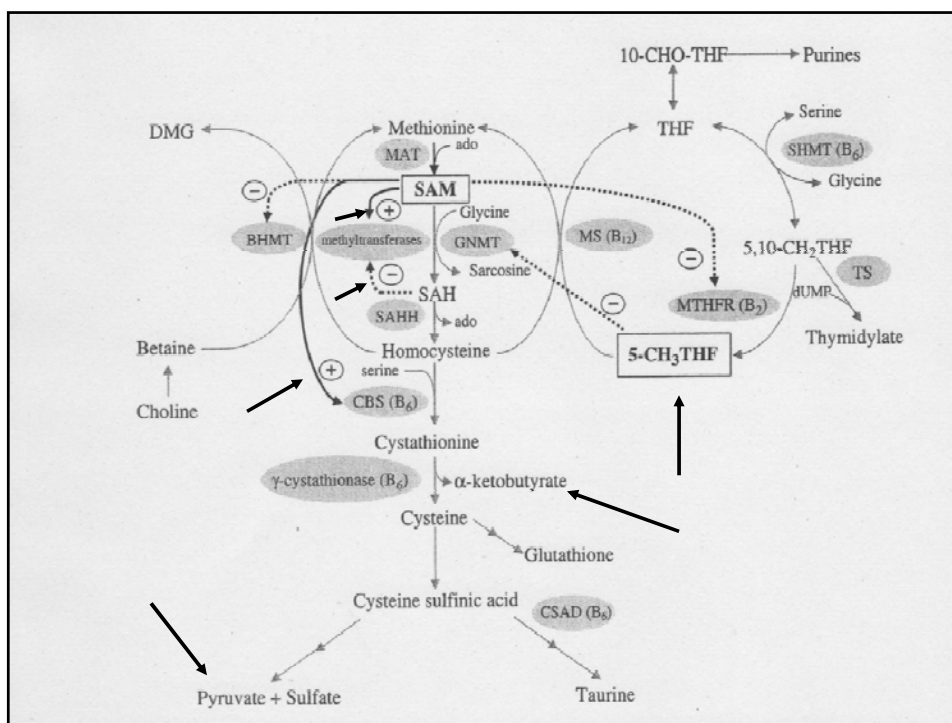


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Diagnosis of glutathione
status:
 α -Hydroxybutyrate

- “Although there is little direct evidence to date, the dominant source of urinary α -hydroxybutyrate (AHB) seems to be the conversion of cystathionine to cysteine.”
- “The activity of this pathway is highly variable, changing in response to demands for protection against oxidative stress.”
- “As oxidative stress increases, the flow of homocysteine shifts away from transmethylation to methionine toward transsulfuration to cystathionine in order to increase the flux of cysteine into glutathione synthesis.”

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“Thus, AHB production can be directly related to the rate of hepatic glutathione synthesis.”

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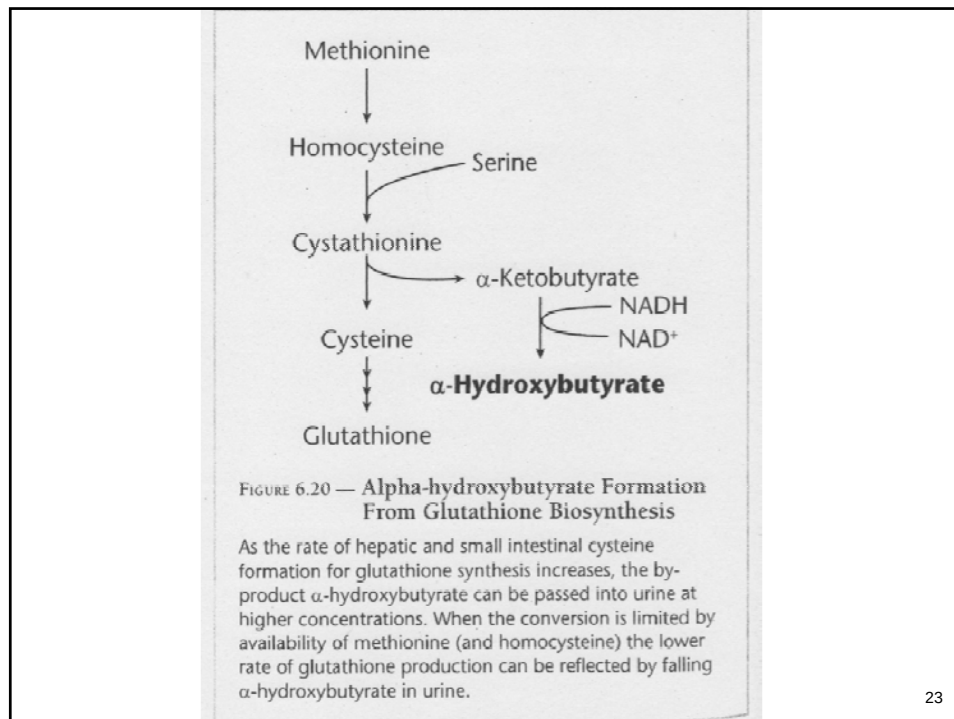
Clinical conditions associated with elevated urinary AHB

- Smoking
- Poor diet
- Lack of exercise
- Infectious diseases such as measles
- Birth asphyxia

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***“All of the conditions that have been
associated with increased AHB
excretion may be related to
increased rates of hepatic
glutathione synthesis from
methionine.”***

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“When the conversion is limited by availability of methionine (and homocysteine) the lower rate of glutathione production can be reflected by falling AHB in urine.”

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- “Elevated AHB, thus, shows increased flow through the transsulfuration pathway as required during times of increased demand for glutathione for meeting oxidative stress or for detoxification functions.”
- “Other signs, such as urinary pyroglutamate and sulfate...must be assessed to determine a patient’s ability to sustain this flow.”

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- “It is also possible to find some patients in late stages of chronic glutathione and methionine depletion who have such a low capacity to generate the transsulfuration flow that their AHB is normal.”
- “These patients generally show low plasma methionine and taurine.”

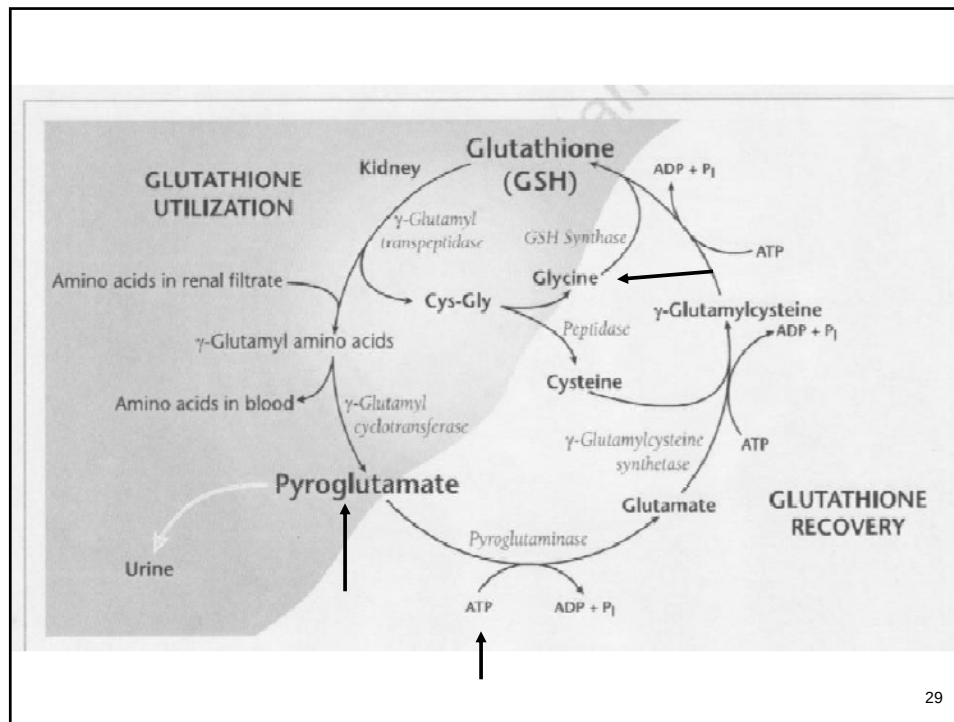
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Diagnosis of glutathione status: Pyroglutamate

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- “Pyroglutamate is created in the γ -glutamyl cycle (GGC), a pathway that is highly active in renal tubules and anywhere there is a high demand for glutathione.”
- “In healthy individuals, a very modest amount of pyroglutamate is spilled in the urine because the rate of pyroglutamate conversion to glutamate for glutathione reformation keeps pace with the rate of γ -glutamyl amino acid formation.”

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- “As glutathione fails to be recovered in the γ -glutamyl pathway, a concurrent reduction in total body sulfate may be found.”
- “Studies showing an age-related decline in expression of glutathione synthesis in rat liver suggests that this problem may be more prevalent in older patients.”
- “If any compromise in energy production occurs, an increase in pyroglutamate could theoretically be noted.”

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- “Alternatively, drugs that require glutathione conjugation can cause failure to sustain glutathione adequacy. This effect has been reported with acetaminophen toxicity.”
- “Burn patients excrete higher than normal amounts of pyroglutamate, whereas their blood levels of glutathione are lowered and their rate of glycine synthesis is decreased.”

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- “Urinary pyroglutamic aciduria has been reported as a marker for glycine deficiency.”
- “If glycine conjugation demand concurrently is increased in healthy human subjects by methionine loading, then pyroglutamate excretion increases because of lowered availability of glycine.”
- “Under these conditions urinary pyroglutamate is positively related to urinary sulfate in a linear manner over the entire sulfate concentration range.”

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- “Small amounts of pyroglutamate are always present in overnight urine because it is produced as an intermediate in a cycle used in the active transport of amino acids in renal tubules.”
- “Since the appearance of a micromole of pyroglutamate in urine is accompanied by the recovery of a micromole of amino acids into the renal blood supply, we might think of the situation as one where glutathione is wasted in order to prevent massive essential amino acid loss.”

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“Up to one-third of the glutathione circulating in blood may be used in this amino acid recovery process.”

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- “Since pyroglutamate may be formed by heating of foods that contain high amounts of glutamic acid, urinary pyroglutamate may have dietary origins.”
- “Foods high in glutamic acid include artificial diets where glutamate is used as a flavor enhancer and high-protein foods, such as meats eggs and dairy products.”

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Diagnosis of glutathione
status:
Sulfate

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- “Sulfate is the ionic form of an inorganic rather than organic acid.”
- “It may be included on panels of urinary organic acids because of the important information it provides about sulfur metabolism.”
- “For example, the protein synthesis disruption of zinc deficiency causes decreased incorporation of cystine into proteins with concurrent large increases in urinary sulfate and taurine from cysteine degradation.”

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“The rise in sulfate parallels the depth of zinc deficiency, indicating that, if other sulfate sources are normalized, urinary sulfate can be a metabolic marker of the severity of zinc deficiency.”

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- “Severe depletion of organic sulfur sources will cause simultaneous high pyroglutamate and low sulfate excretion.”
- “High pyroglutamate with normal sulfate indicates inadequate organic sulfur sources for production of cysteine required for glutathione synthesis.”
- “Only organic sulfur in the form of compounds such as N-acetylcysteine or methionine along with adequate glycine will restore normal glutathione levels.”

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- “Normal urinary pyroglutamate with low sulfate levels can occur in individuals with impaired sulfate activation.”
- “In these cases, rapid replenishment of hepatic sulfate may be accomplished with either sulfur donors like N-acetylcysteine or inorganic sulfate such as sodium sulfate.”
- “On the other hand, a well nourished patient under temporary metabolic stress of detoxification from, for example, use of acetaminophen, may have elevated α -hydroxybutyrate, signaling the increased rate of hepatic glutathione synthesis, but have no need for amino acid or glutathione therapy due to normal sulfate level.”

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- “An individual with limited ability to produce glutathione may show the glycine depletion sign of high pyroglutamate and low sulfate.”
- “This patient is a candidate for glutathione administration along with glycine and N-acetylcysteine or methionine, and their taurine status should be monitored, since depletion of other sulfur-containing compounds is likely.
- “Urinary sulfate has also been shown to reflect intake of sulfiting agents widely used as food additives.”

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Treatment options for optimizing glutathione status

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Sulfur amino acid metabolism limits the growth of children living in environments of poor sanitation

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ABSTRACT

Environmental enteropathy has been identified as a cause of poor growth in children living in low-income countries. It is characterized by chronic inflammation of the small intestine, malabsorption, and growth retardation. The pathogenesis of environmental enteropathy is unknown, but it is believed to be caused by ingesting enteric bacteria while living in an environment of poor sanitation. The growth suppression associated with environmental enteropathy is believed to result from repartitioning of dietary nutrients away from pathways that result in growth, toward processes related to host defense and catabolism.

Introduction

Child undernutrition is highly prevalent in low- and middle-income countries. It is estimated that 12% of the 550 million preschool children in underdeveloped countries are stunted and another 20% are underweight [1]. Childhood malnutrition leads to long-term cognitive deficits, poorer performance in school and fewer years of completed schooling, and ultimately results in lower economic productivity during adulthood [2]. Moreover, one-third of all children living in settings of poor sanitation

permeability was associated with impaired growth [3]. Plasma concentrations of endotoxin and anti-endotoxin antibodies were also elevated and related to both growth and measures of mucosal enteropathy. There was also chronic immunostimulation, as evidenced by abnormally elevated white blood cells, lymphocytes, and platelets, and frequently raised plasma concentration of C reactive protein. The etiology of environmental enteropathy is unknown, but is assumed to be caused from ingesting enteric bacteria while living in an environment of poor sanitation. The growth suppression associated with environmental enteropathy is believed to result from repartitioning of dietary nutrients away from pathways that result in growth, toward processes related to host defense and catabolism.

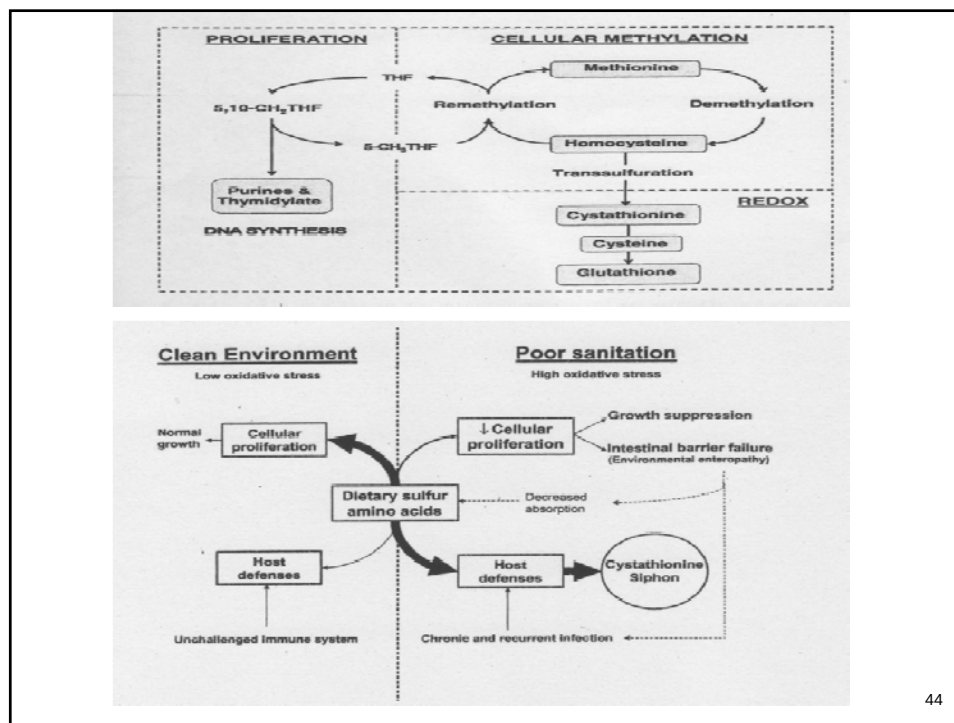
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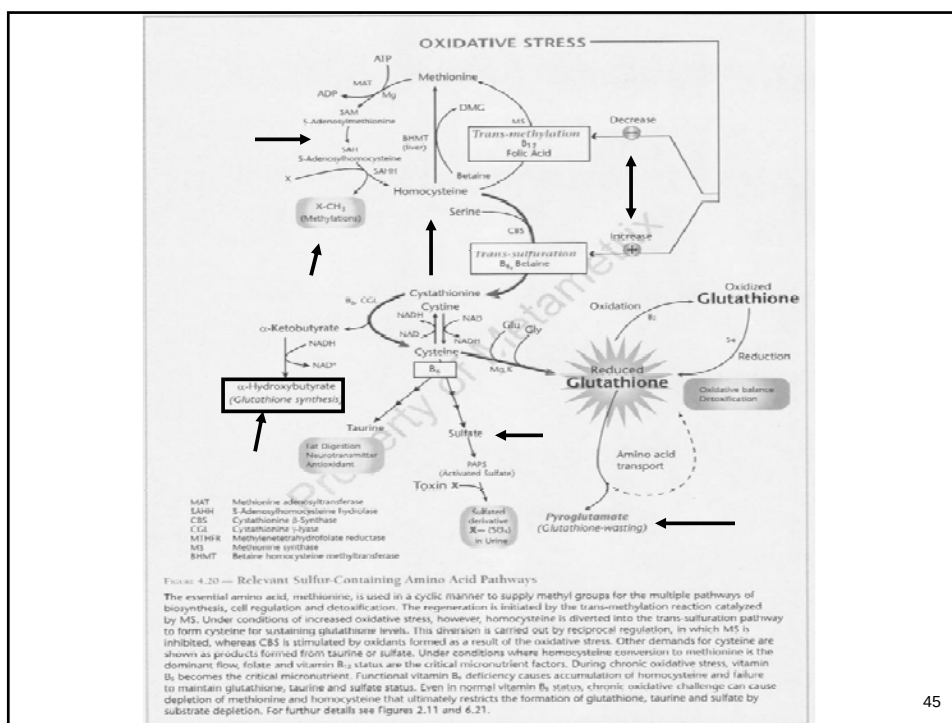
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Optimize anabolic/catabolic
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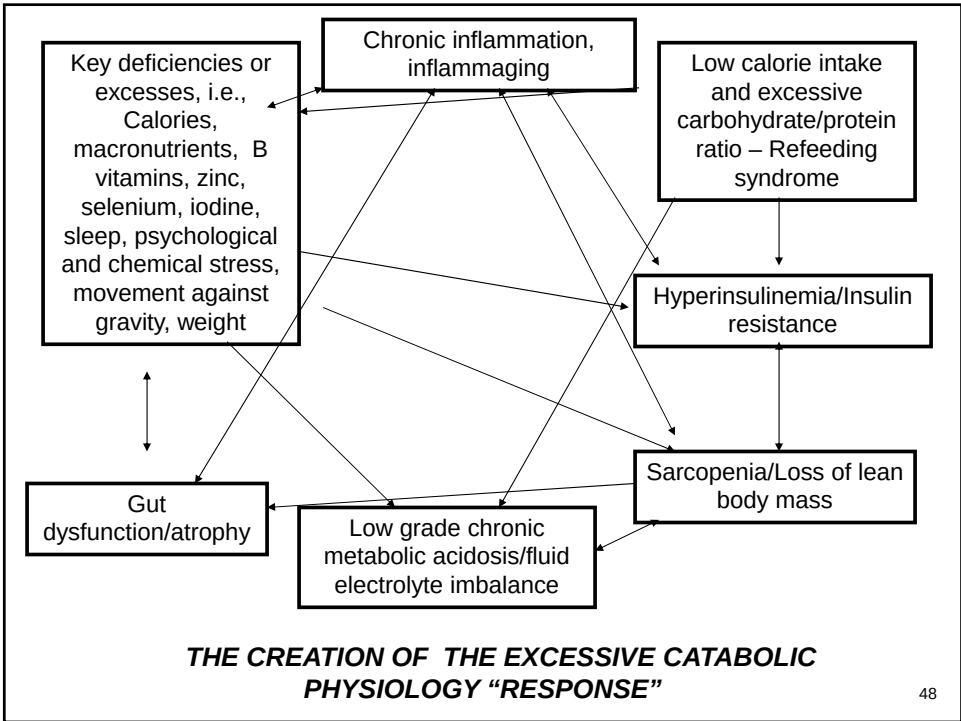
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Considerations for the Catabolic Patient				
Signs & Symptoms	Consider	Treatment Plan	Monitor	Supplementation
Signs & Symptoms: - Feeling "lousy" - Fatigue - Cravings - Depression/anxiety - Poor quality sleep - Poor digestion/absorption/leaky gut - 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) -Palco products (DFH)
	Reducing inflammation	Reduce inflammation Address food allergies	Inflammatory biomarkers: -hs-CRP -Fibrinogen levels -WBC -Differential	Anti-inflammatory agents -Inflametric (Olympian) -D-Flame (Protocolt) -Pain Guard (Porque) 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)
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	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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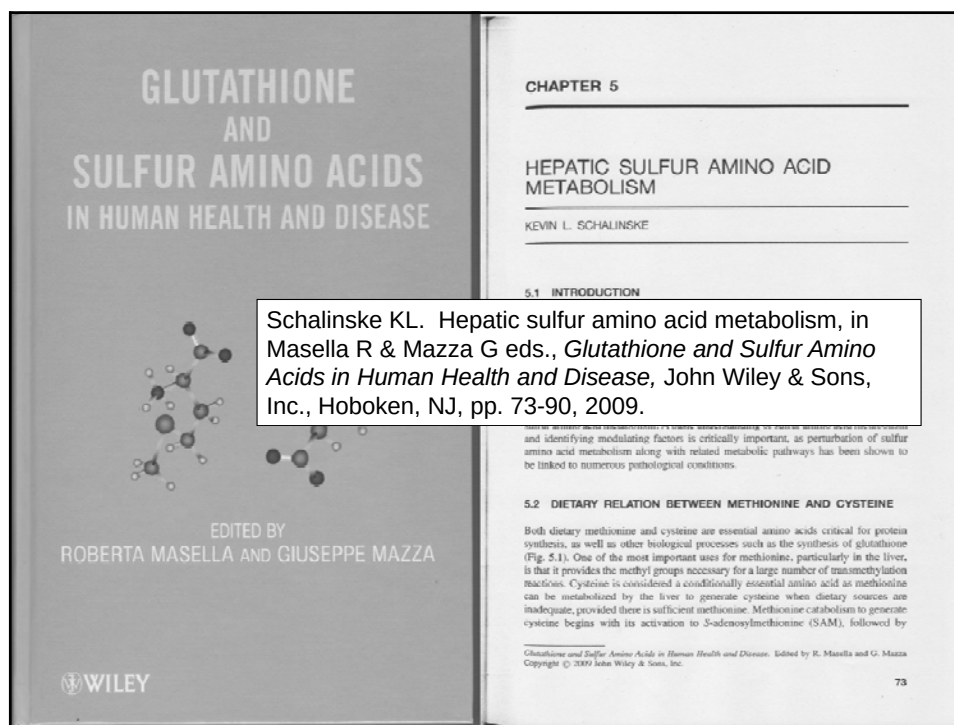
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Supplementation

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- “A number of strategies have evolved to raise levels of glutathione in depleted individuals.”
- “There are at least three potential ways of enhancing cellular GSH content: administration of the three amino acids (cysteine, glutamic acid, and glycine) which comprise the tripeptide, either singly or in various combinations; administration of cofactors for the metabolic pathways leading to GSH production, that is, vitamin B6, riboflavin, and folic acid; and administration of synthetic compounds which become converted to precursors of GSH.”

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- “In HIV positive patients a whey protein formulation was clearly demonstrated to increase plasma GSH concentrations.”
- “N-acetyl cysteine (NAC) rapidly enters the cell and is speedily deacylated to yield L-cysteine.”
- “...randomized studies on asymptomatic HIV positive patients in the presence and absence of antiretroviral therapy (ART) have shown that NAC can raise blood GSH, increase natural killer cell activity, and enhance stimulation indices of T cells incubated with mitogen or tetanus toxin.”

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- “Interestingly the rise in T cell function was accompanied by a fall in plasma IL-6 in subjects receiving ART as well as the drug.”
- “Furthermore studies have shown that survival time was improved in HIV+ patients who maintained high concentrations of GSH in CD4+ T lymphocytes.”
- “It could therefore be surmised that improved T cell function and reduced inflammation are modulated by improvement in antioxidant status in these patients.”

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- “Alpha-lipoic acid provides a further means of enhancing tissue GSH content.”
- “The compound is reduced to dihydrolipoic acid, which converts cystine to cysteine.”
- “Cysteine, on gaining entry to the immune cells, is rapidly converted to GSH.”
- “Flow cytometric analysis of freshly prepared human peripheral blood lymphocytes shows that lipoic acid is able to normalize a subpopulation of cells with severely compromised thiol status rather than increasing the level in all cells above normal values.”

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“Hence lipoic acid may also prove to be a useful clinical agent for restoring cellular GSH concentrations in immunocompromised subjects.”

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The amino acid requirements of disease

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Abstract—It is now clear that accidental injury, surgery, infection, cancer and psychosocial stress activate new metabolic pathways that consume amino acids. For example, immune activation appears to alter glutamine and arginine metabolism, acute phase protein synthesis demands the increased provision of the aromatic and sulfur amino acids, while an increased rate of glutathione turnover increases the cysteine requirement of the body. Critically, under every stressful condition, the accompanying demands mean that these new demands have to be met from the patient's protein stores. This, in turn, demands a replacement of these amino acids in the diet. This paper reviews the evidence for these demands and discusses the implications for clinical practice.

Reeds PJ & Jahoor F. The amino acid requirements of disease, *Clin Nutr*, Suppl 1, pp. 15-22, 2001.

Key words: amino acids; requirements; malnutrition; trauma; infection; acute phase response

Introduction

There is no doubt that the changes in cytokines, immune activation and stress hormones that accompany infection, injury and malignant disease set in train a disruption of the normal regulation of metabolism in general, and protein turnover and amino acid metabolism in particular. It is now nearly 50 years since the first report of metabolic measurements of the effect of

examining whether there are reasons to suppose that the diseased state imposes particular demands on amino acid needs. We will, as far as possible, consider the circumstances of relatively uncomplicated disease rather than the circumstances that surround multiple organ failure, because under these more severe circumstances metabolic regulation is so disrupted that severe illness, sepsis, hypotension, cardiac and renal under-perfusion and acidosis become dominating influences and lead to a situation that is impossible to interpret in simple nutritional terms.

Although the title of this paper emphasizes the amino acid requirements associated with disease (and by

“...it is of interest that supplementation of HIV patients and of non-insulin dependent diabetics with N-acetylcysteine leads to an improvement of N-acetyl cysteine.”

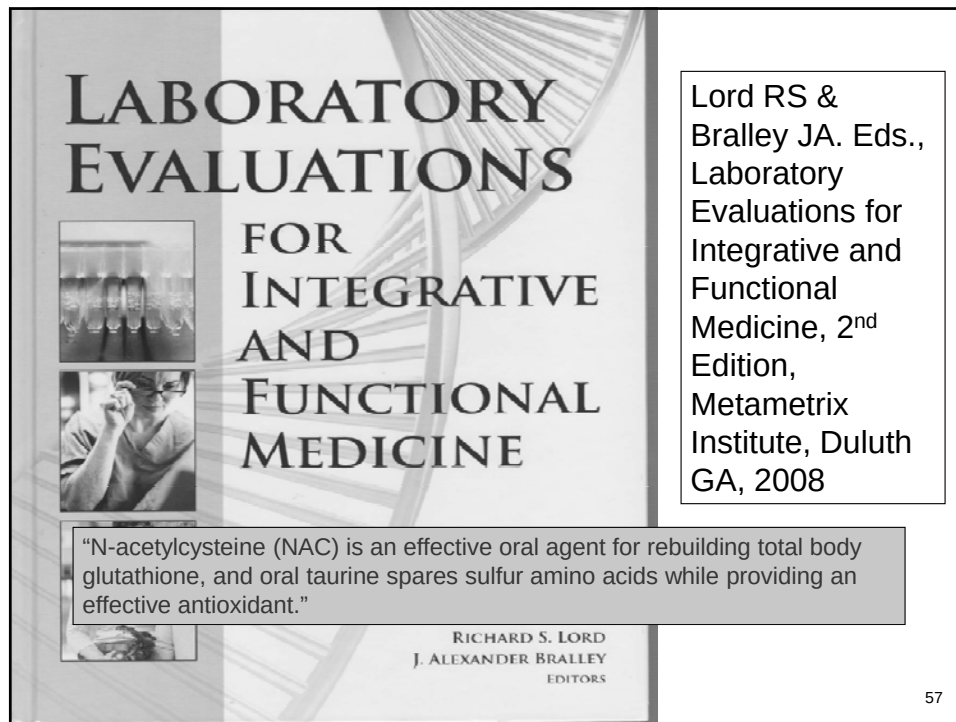
extension, stress), their prolonged activation has pathological effects and predisposes the infected or traumatized individual to malnutrition.

It seems axiomatic that the development of appropriate nutritional strategies directed towards the amelioration of these adverse physiological effects should start from an understanding (both quantitative and mechanistic) of the nature of the metabolic changes that are initiated by stress. The purpose of this paper is to discuss the nature of these responses to stress, and to

this loss (Table 1) are not only similar among a variety of stresses, but also among similar stresses of different severity. These observations imply the following: first, that the general metabolic response is uniform, irrespective of the nature of the insult; second, that the eventual deleterious effects of the different insults are primarily a reflection of the duration of the response rather than its initial magnitude. In other words, the response to the insult (Figures 2) while presumably of immediate benefit, leads to other metabolic and physiological changes that

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Dosing recommendations

- N-acetylcysteine – 1500 mg/day with meals
- Lipoic acid – 300 mg/day with meals
- R-lipoic acid – 150 mg/day with meals
- Taurine – 1-2 g per day with meals

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Thank you!!

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