

Entry Level Clinical Nutrition
Part III

Blood chemistry interpretation
from a metabolic point of view

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***There is a problem with the
usual ways of interpreting
blood chemistries.***

2

***There is a lack of an
overriding, big picture
concept.***

3

What is the big picture?

4

***Quality of life issues are the
major concerns more than
ever now.***

5

- ***What appears to be the most important determinant of quality of life?***
- ***Optimal lean body mass.***

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“...gait speed may be considered a new ‘vital sign,’ specifically sensitive for older persons.”

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- ***Why is lean body mass lost?***
- ***Two reasons.***
- ***Aging***
- ***A response to environmental stressors***

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What is the nature of this response?

- ***Where does the protein go?***
- ***Does it go to production of functional tissue such as muscles, gut lining, ligaments, regulatory factors, detox enzymes? (Anabolic)***
- ***Or***
- ***Does it go to production of inflammatory mediators such as acute phase proteins and cytokines and production of energy? (Catabolic)***

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***Aging makes it more difficult
to respond anabolically.***

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Underlying hypotheses of Entry Level Clinical Nutrition:

- Chief complaints in chronically ill patients are not diseases but responses that have gone on too long (Allostatic load).
- The metabolic imbalances that combine to form this response have been well defined by critical care nutritionists.
- Blood chemistry, instead of being used to identify isolated diseases will be used to determine which metabolic imbalances predominate.

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

The Slow Shift

September 9, 2009

Summer of Work Exposes Medical Students to System's Ills

By David G. H. ...

WASHINGTON — 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 emotions raw.

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

The students learned not only to deliver better and safer care, but also to offer emergency-room patients a more humane, less frantic experience. Medical patients, for nearly patients with chronic conditions, had to learn to live with the system's chronic problems — diabetes, hypertension, obesity, depression — that left many questioning how much any one physician could really help.

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

"...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 resulting program is elective, but a recent number of students had gone to the program — or from a class of 100.

The school has a cooperative agreement to train medical students from Alaska, Idaho, Montana and Wyoming, some of which have a critical shortage of doctors. The summer program is one of several initiatives, along with basic business and management, designed to have students back to their home states to practice in areas with a shortage of primary care physicians.

This year, the students set out on assignments with interesting in Washington over wholesale changes to the health care system that will soon be joining. Whether there will be improvements to the system remains to be seen, but the students returned feeling they were 50% in the.

http://www.nytimes.com/2009/09/09/health/policy/09medstud.html?_r=1&ft=health&ft=... 9/9/2009

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Plan now so your later years can be comfortably extended

USA Today, January 11, 2011

Palliative care, in my view, is the future of care for those of us entering the In-Between Stage. Its aim is to treat symptoms and relieve suffering, and it can be offered when we are first diagnosed with a serious illness.

New Passage
By Jeff Moss

Journalist and doctor "call" liberty in the matter of it being otherwise of it, except, including Passage in Cambridge.

There has been an even more dramatic increase among Medicare beneficiaries, who report three or more chronic conditions, and that jump occurred equally across, sex, race, ethnicity and income groups."

"Sixty-five percent of Americans in mid-life (ages 45-64) live with at least one chronic illness, according to a national 2009 study reported in *Health Affairs*. There has been an even more dramatic increase among Medicare beneficiaries, who report three or more chronic conditions, and that jump occurred equally across, sex, race, ethnicity and income groups."

Editor: Please, really, never to, just, of course, improvement of himself.

Some conditions or serious health problems, the need for advance-care planning increases. It should be a routine part of our medical care.

The last thing most of us would want is to be rushed off to the hospital with a stroke or heart attack or emergency surgery and be prepared by the medical staff with questions about what extreme measures we want or don't want, when all we can think about is getting out of pain.

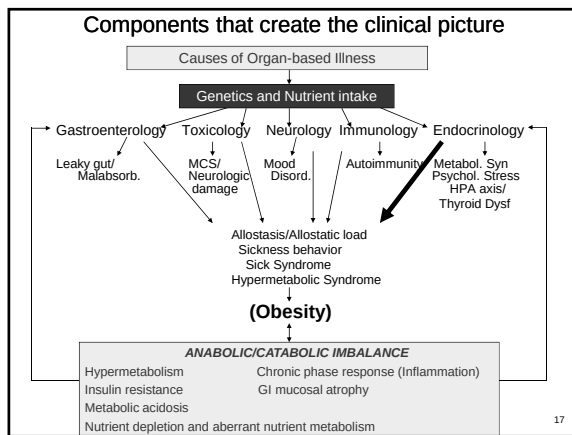
Palliative care, in any sense, is the future of care for those of us entering the In-Between Stage. Its aim is to treat symptoms and relieve suffering, and it can be offered when we are first diagnosed with a serious illness. It's not the same as hospice.

Simply with a range of choices, including, for example, pneumonia and chemotherapy," says Diane Shiras, a geriatrician at the Mount Sinai School of Medicine.

Sadly, the more common route is a meandering one from hospital to rehab to rehabilitation, which is especially troubling for older people. They can't wait long enough. They can't fight antibiotic-resistant infections. They can't talk back to a compromised staff. The sense of isolation and total loss of control has an adverse effect on the immune system.

By contrast, groundbreaking research from Harvard and other research centers shows that palliative care may actually prolong life.

One breakthrough that you're likely to see "terminal" stages have advance care plans. Healthcare providers are talking to patients, and, eventually, the



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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."

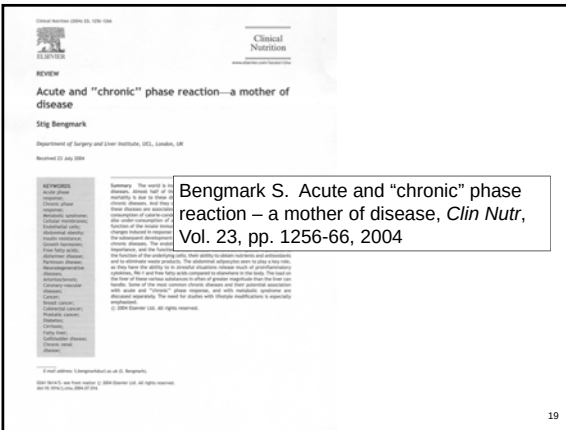
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Nestlé NUTRITION

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A simplified approach to helping patients feel better

- Understanding the true nature of chronic illness: Excessive allostatic load
- Simple diagnostic tools
- A simplified menu to improve patient quality of life

Signs & Symptoms	Consider	Treatment Plan	Monitor	Supplementation
Signs & Symptoms: - Feeling "tired" - Fatigue - Cravings - Depression/anxiety - Poor quality sleep - Poor digestive absorption/food gut - Chronic pain/infection Clinical Findings: - High % body fat - Low bone density - High fasting insulin - Heavy metal burden - Calcium to phosphorus ratio 1:1 - High normal globulin - Cholesterol (LDL-200) - 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+ Chloride (Moss) Magnesium Glycinate (Vital Nutrients)
	Optimize muscle mass (reduce sarcopenia) and amino acid levels	Pay special attention to protein and amino acids	Body composition Bioelectrical impedance scale Grip strength dynamometer	Amino acids Creatine Anionic Base (Metabolic Matter) Protein powders (Pure Protein products) Amino acid products (Biotinics) Pulse products (DFFB)
	Reducing inflammation	Reduce inflammation Address food allergies	Inflammatory biomarkers: HbA1c-CRP Inflammatory levels "WBC" Eosinophil	Anti-inflammatory agents Inflammix (Oxygent) D-Fenox (Pharmacia) Pain Guard (Purpex) Fish Oil Aqua Pure Fish oil (Vital Nutrients) Food allergy blends —MoffGut (DFFB)
	Improving insulin sensitivity	Watch carbohydrate/protein ratio	Fasting glucose Fasting insulin C-peptide	Metabolic Synergy (DFFB)
	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 (Pure Protein products) Amino acid (Biotinics)
	Optimize GI function	Dietary changes supplementation of digestive aids	Patient signs and symptoms	Enzyme BCI (Vital Nutrients) Digestion (DFFB) Intestinal Repair Complex (Biotinics)
	Optimize micronutrient deficiencies/imbalances	Supplementation of nutrients Pay special emphasis on vitamins D, iodine, and zinc	Serum 25 OH vitamin D Erythrocyte folic acid indicators Zinc, ferritin test	Multi Nutrients (Vital Nut) Vitamin D (Vital Nut) Iodine (Moss) Zinc, Selenium (DFFB)

This is a relatively easy, inexpensive way to help most patients feel better early on during the course of therapy no matter what their “disease” or chief complaint.

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Invited Review

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

Karen P. Su

Department of Clinical Psychology and Health Behavior, University of Illinois at Chicago, Chicago, Illinois, USA; Center for Health Systems Research and Analysis, University of Illinois at Chicago, Chicago, Illinois, USA

Key Words: Major depressive disorder; Omega-3 fatty acids; Antidepressant; Fish oil; Neuroinflammation; Neuroplasticity; Phospholipase A2; Cytokines

Abstract: The neurobiology of depressive disorder involves complex interactions between genetic, environmental, and neurobiological factors. Omega-3 fatty acids have been shown to have antidepressant effects in both animal and human studies. This review examines the biological mechanisms of the antidepressant effect of omega-3 fatty acids, focusing on the role of these fatty acids in modulating neuroinflammation, neuroplasticity, and the gut-brain axis. The review also discusses the clinical implications of these findings for the treatment of major depressive disorder.

Introduction: Major depressive disorder (MDD) is a serious mental health condition characterized by persistent feelings of sadness, loss of interest, and changes in appetite and sleep. The exact biological mechanisms of MDD are not fully understood, but research suggests that neuroinflammation and neuroplasticity play significant roles. Omega-3 fatty acids, found in fish oil, have been shown to have antidepressant effects in both animal and human studies. This review examines the biological mechanisms of the antidepressant effect of omega-3 fatty acids, focusing on the role of these fatty acids in modulating neuroinflammation, neuroplasticity, and the gut-brain axis. The review also discusses the clinical implications of these findings for the treatment of major depressive disorder.

Neuroinflammation: Neuroinflammation is a key feature of MDD, characterized by increased levels of pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α). Omega-3 fatty acids have been shown to reduce levels of these cytokines, thereby exerting anti-inflammatory effects. This reduction in inflammation is thought to be one of the mechanisms by which omega-3 fatty acids exert their antidepressant effects.

Neuroplasticity: Neuroplasticity refers to the brain's ability to reorganize itself by forming new neural connections. Omega-3 fatty acids have been shown to promote neuroplasticity by increasing the levels of brain-derived neurotrophic factor (BDNF), a protein that supports the growth and survival of neurons. This promotion of neuroplasticity is thought to be another mechanism by which omega-3 fatty acids exert their antidepressant effects.

Gut-Brain Axis: The gut-brain axis is a bidirectional communication system between the gut and the brain. Omega-3 fatty acids have been shown to modulate the gut-brain axis by increasing the levels of short-chain fatty acids (SCFAs) produced by gut bacteria. SCFAs have been shown to have anti-inflammatory and neuroprotective effects, which may contribute to the antidepressant effects of omega-3 fatty acids.

Clinical Implications: The findings of this review suggest that omega-3 fatty acids may be a promising adjunctive treatment for MDD. However, more research is needed to determine the optimal dosage and duration of omega-3 fatty acid supplementation for the treatment of MDD. Additionally, it is important to note that omega-3 fatty acids should not be used as a substitute for standard antidepressant therapy.

Conclusion: Omega-3 fatty acids exert their antidepressant effects through multiple mechanisms, including the reduction of neuroinflammation, promotion of neuroplasticity, and modulation of the gut-brain axis. These findings suggest that omega-3 fatty acids may be a promising adjunctive treatment for MDD. However, more research is needed to determine the optimal dosage and duration of omega-3 fatty acid supplementation for the treatment of MDD.

References:

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2. Kasper S, Lin A, Lin A, et al. Omega-3 fatty acids in the treatment of major depressive disorder: A meta-analysis. *Journal of Clinical Pharmacy and Therapeutics*. 2010;35(1):1-10.

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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 ^d –1	34–47 ^f
Psychomotor slowing	40 ^e	59–65 ^f
Insomnia	20–39 ^d	63
Irritability	35 ^d	50
Arthralgia	9–36	31 ^e
Musculoskeletal pain	26–32	62–80 ^{e,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.

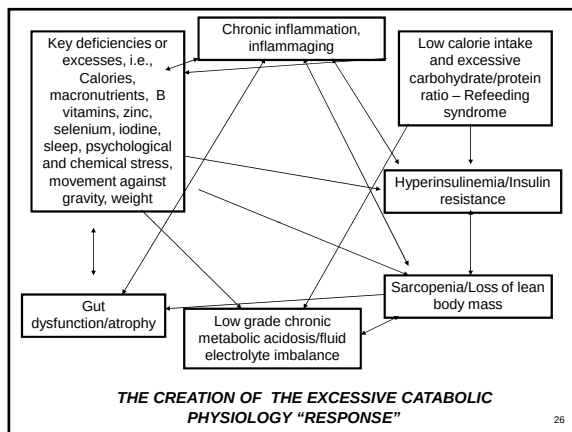
² Result from depressed inpatient population.

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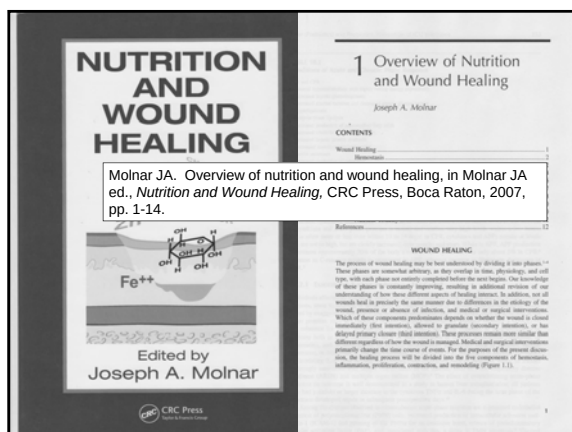
Key metabolic imbalances seen with the acute phase response

- Metabolic acidosis
- Loss of lean body mass (sarcopenia)
- Insulin resistance
- Inflamm-aging (Increased innate immunity and decreased adaptive immunity)
- Suboptimal caloric intake and carbohydrate:protein ratio (Refeeding syndrome)
- Gastrointestinal dysfunction/gut atrophy
- Deficiencies of key micronutrients such as zinc, selenium, and vitamin D

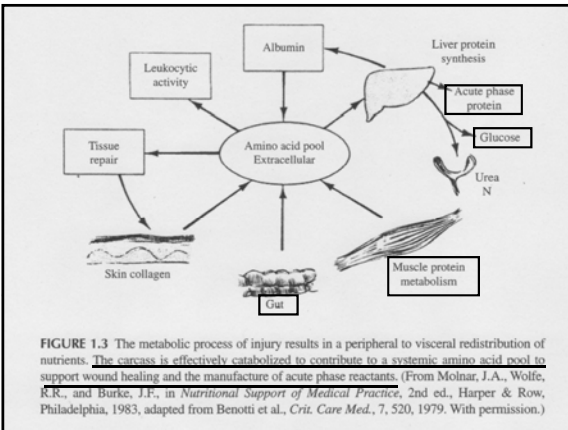
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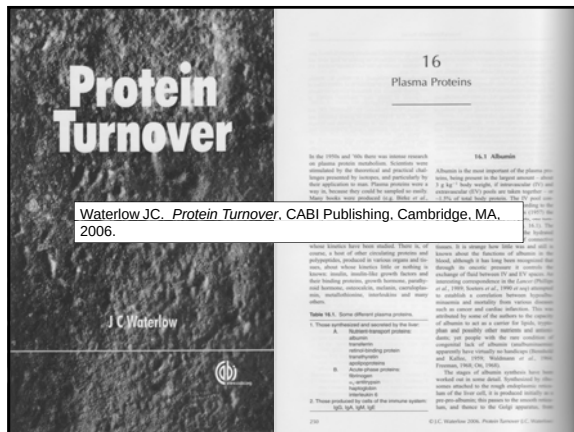


- “During periods of metabolic stress, the body is able to effectively catabolize the carcass (muscle, skin, bone) to support visceral protein synthesis of acute phase proteins, immunoglobulins, inflammatory cells, and collagen, needed to fight infection and heal the wound.”
- “In the case of proteins, intricate shuttling mechanisms have developed to allow redistribution of the substrate from the periphery to the viscera.”

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- “This could also apply to micronutrients. Because a large percentage of the body zinc stores are found in the skin, it would not be surprising that if the skin proteins are catabolized, this might allow mobilization of skin zinc to provide cofactors for enzymatic activity in other parts of the body.”

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Waterlow J.C., *Protein Turnover*, CABI Publishing, Cambridge, MA, 2006.

- “In many patients plasma albumin concentrations are low and it has been suggested that the acute phase proteins compete with albumin for the amino acids required for synthesis.”
- “An interesting hypothesis was proposed by Reeds *et al.* to relate the acute phase protein response to the muscle wasting that occurs in so many clinical conditions.”

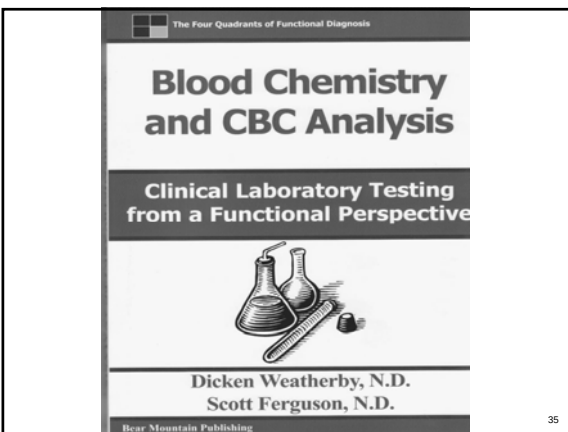
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- “At its peak total synthesis of APPs after a stress, from the data of Fleck *et al.*, could amount to as much as 0.85 g of protein per kg per day.”
- “The APPs contain larger amounts of the aromatic amino acids than muscle, and Reeds calculated that it would require the breakdown of 2 g of muscle protein to provide amino acids for the synthesis of 850 mg of APPs.”
- “The other amino acids that are produced in excess from muscle in order to meet the need of aromatics will be oxidized and the nitrogen excreted in the urine.”

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“This would account for a large part of the ‘catabolic’ loss after trauma or in septic states.”

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How do you know if your patient is catabolic? Routine laboratory and home/office analyses...

- First morning salivary and urine pH
 - Ideal 6.4 – 7.0
- Fasting glucose
 - Ideal <90
- Low normal serum potassium, high normal serum sodium
- Low or low normal serum CO₂ (18-22)
- High normal BUN (24-26) with normal creatinine

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How do you know if your patient is catabolic? Routine laboratory and home/office analyses...

- Calcium to phosphorus ratio
Ideal 10:4
Catabolic physiology 3:1 (Ca 9.3-9.5, P 2.8-2.9)
- High normal globulin (3.6-3.8) (Possible food allergies)
- Low total protein
- High uric acid
- Total cholesterol (220 -280)
- High LDL, low HDL
- High triglycerides

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How do you know if your patient is catabolic? Routine laboratory and home/office analyses...

- Cholesterol to triglyceride ratio
Ideal 2:1
- High liver enzymes (ALT, AST, GGT)
- High WBC, high polys, high lymphs
- High CRP
- High or low TSH
Ideal 2.0
- High normal or low normal T₃ and/or T₄
- High blood pressure

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How do you know if your patient is catabolic? Routine laboratory and home/office analyses...

- High percent body fat
(Visceral adiposity)

Non-athlete male ideal 12% - 18%
Non-athlete female ideal 22% - 28%

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How do you know if your patient is catabolic? Non-routine laboratory analysis...

- High fasting insulin
- High or low salivary cortisol
- Elevated urinary catecholamine metabolites
- Low bone density
- High salivary estradiol to progesterone ratio
- High salivary testosterone to estradiol ratio

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How do you know if your patient is catabolic? Functional laboratory analysis...

- Dysbiosis
- Leaky gut
- Chemical toxicity - Abnormal phase I & II detox pathways
- High heavy metal levels (Hair, urine, serum)
- Low or high urinary sulfate
- Genomic variations (Folate metabolism, low glutathione-S-transferase, etc.)

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How do you know if your patient is catabolic? Signs and symptoms...

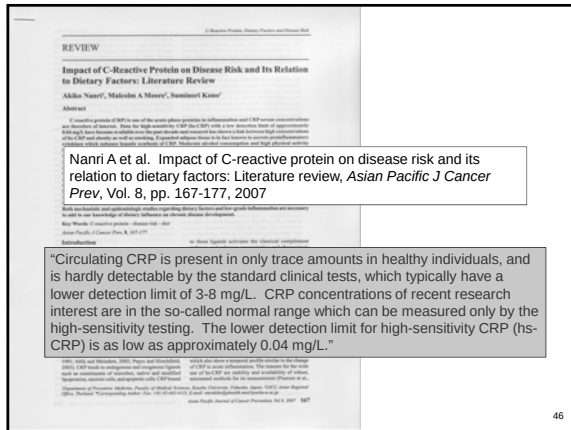
- Fatigue
- Cravings
- Depression and/or anxiety
- Poor diet – high in acid-based, refined foods
- Lack of sleep or poor quality of sleep
- Too little or too much quality exercise
- Signs and symptoms of poor digestion and absorption
- Pain, trauma, infection

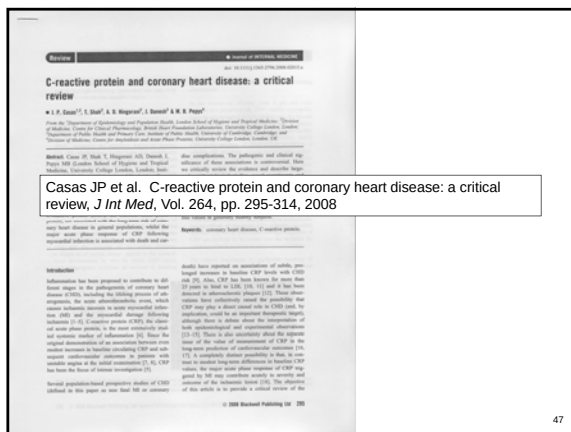
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- "In healthy normal subjects CRP is a trace plasma protein. In the first substantial study of such individuals, the median value in 468 British Caucasian volunteer blood donors aged 18-63 years was 0.8 mg/L, the nineteenth percentile of the distribution was 3 mg/L and the ninety-ninth percentile was 10 mg/L."
- "Similar distributions have been reported from the general adult population of the USA and northern Europe."
- "In USA 50% of individuals have baseline CRP greater than 2 mg/L and 33% are between 3 and 10 mg/L."

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- "The term 'high-sensitivity' or 'highly sensitive' CRP, abbreviated as hs-CRP, has been widely promoted in recent literature."
- "It refers to measurement of CRP in serum or plasma samples using immunoassay methods with sufficient sensitivity to quantify CRP throughout its normal range in contrast to older less sensitive commercial assays which had detection limits in the range 2-10 mg/L and were suitable for measurements of acute phase responses of CRP rather than baseline values."

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- "It is very important to recognize that the analyte designated as hs-CRP is just CRP itself, not anything new or different and in particular is not a novel analyte with any special relationship to cardiovascular disease."
- "hs-CRP is the same exquisitely sensitive and entirely nonspecific systemic marker of infection, inflammation, tissue damage and/or almost any form of adverse nonphysiological stress as the CRP, which has been extensively studied and used clinically for over 75 years."

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- "Precisely the same constraints apply to use of its values, notable that these cannot be interpreted in the absence of full clinical information on the individual patient at the time the sample was taken."
- "An individual with an apparent baseline CRP value of, say 7 mg/L, should not just be told that they have increased risk of cardiovascular disease and be advised to alter their lifestyle accordingly, when the increased CRP concentration might reflect any of a very wide range of other serious diseases which have not yet declared themselves, for example, Hodgkin's disease or renal carcinoma or any other condition which is often occult for some time before clinical presentation."

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Thank you!!

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