

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.***

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

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Medical News: Walking Speed and Survival Connected - Printable Version

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TODAY

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A | A | A | A

Walking Speed and Survival Connected

By Todd Heale, Staff Writer, MedPage Today
January 04, 2011

MedPage Today Action Points

- Note that this study proposes measurement of gait speed as a standardized, inexpensive, and relatively quick indicator of health and survival in older patients.
- Point out that at present there is no evidence that gait speed improvements are associated with better health-related outcomes, and it would not be reasonable to impose interventions on patients in order to do this.

Review

Walking speed -- an indicator of vitality -- may help clinicians predict how long their older patients will live, a meta-analysis showed.

In a pooled analysis of nine cohort studies, each 1-m/s increase in gait speed was associated with a 12% relative reduction in the risk of death during follow-up (HR 0.88, 95% CI 0.87 to 0.90), according to Stephanie Studenski, MD, MPH, of the University of Pittsburgh, and colleagues.

The risk reduction was consistent across most subgroups -- except for functional status -- with hazard ratios ranging from 0.81 to 0.92 ($P < 0.002$ for all), the researchers reported in the Jan. 5 issue of the *Journal of the American Medical Association*.

If use of gait speed -- a simple indicator of physical function -- as a predictor or survival is implemented in clinical practice, it may help clinicians individualize the treatment of older patients, according to Studenski and her colleagues.

Patients whose gait speed indicates a high probability of living for five to 10 years may be appropriate candidates for preventive interventions that require a long time to work. On the other hand, patients with slow gait speed could be targeted for interventions to ameliorate risks to health and survival.

Additionally, monitoring gait speed over time for deviations may allow for the detection of new health problems that require evaluation.

Finally, the authors wrote, gait speed may be useful in the research setting, by allowing for the evaluation of whether interventions to improve walking speed result in

<http://www.medpagetoday.com/ebp/abstract.cfm?tid=24180>

1/9/2011

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ORIGINAL CONTRIBUTION

Gait Speed and Survival in Older Adults

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Context Survival estimates help individualize goals of care for geriatric patients, but life tables fail to account for the great variability in survival. Physical performance measures, such as gait speed, might help account for variability, allowing clinicians to make more individualized estimates.

Objective To evaluate the relationship between gait speed and survival.

Design, Setting, and Participants Pooled analysis of 9 cohort studies (collected between 1966 and 2000), using individual data from 34 485 community-dwelling older adults aged 65 years or older with baseline gait speed data, followed up for 6 to 21 years. Participants were a mean (SD) age of 73.5 (5.9) years; 59.6%, women; and 79.4%, white, and had a mean (SD) gait speed of 0.92 (0.07) m/s.

Main Outcome Measures Survival rates and life expectancy.

Results There were 17 528 deaths; the overall 5-year survival rate was 84.8% (confidence interval [CI], 79.6%-89.8%) and 10-year survival rate was 59.7% (95% CI, 46.5%-70.6%). Gait speed was associated with survival in all studies (pooled hazard ratio per 0.1 m/s, 0.88; 95% CI, 0.87-0.90; $P < .001$). Survival increased across the full range of gait speeds, with significant increments per 0.1 m/s. At age 75, predicted 10-year survival across the range of gait speeds ranged from 19% to 87% in men and from 30% to 91% in women. Predicted survival was similar in men and women across the range of gait speeds.

Study This study is to evaluate the association of gait speed with survival in older adults and to determine the degree to which gait speed explains variability in survival after accounting for age and sex.

METHODS

Overview We conducted a meta-analysis of data from 9 cohort studies, baseline data for which were collected between 1966 and 2000 (TABLE 1).¹⁻¹⁰ Each study, which included more than 400 older

men, were given a score of 1 if they were monitored survival for at least 5 years. Analyses performed herein were conducted in 2009 and 2010. All studies required written informed consent and institutional review board approval.

Populations All studies recruited community-dwelling older adults. Although some sought representative samples,^{4,11,12,13} others focused on healthier participants,¹⁴ single sex,¹⁵ or older adults from primary care practices.¹⁶ Only

For editorial comment see p 93.

50 JAMA, January 5, 2011 • Vol 305, No. 1 (Reprints)

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EDITORIAL

Editorials represent the opinions of the authors and do not necessarily reflect those of the American Medical Association.

Role of Gait Speed in the Assessment of Older Patients

Matteo Cosset, MD, PhD

OVER THE LAST 3 DECADES, A LARGE AND increasing amount of evidence has documented the importance of physical function in elderly individuals, both as a critical component of clinical assessment and as a useful outcome for interventions.¹ Yet physical function is still not one of other clinical or biochemical time, inadequate space, and it is not one of the obstacles of physical function in the geriatric population. Gait speed has increasingly been proposed as an unique measure of physical performance and as a potential screening tool,^{2,3} but adoption has remained inconsistent.

In this issue of JAMA, the study by Studenski et al⁴ fills an important research gap and paves the way to a broader adoption of gait speed assessment. Their findings from a pooled analysis of 9 major cohort studies confirm gait speed as a predictor of mortality in older persons^{4,5} and also provide the statistical foundations to estimate expected survival at different ages based only on gait speed. Several barriers limiting the clinical use of physical performance measures might thus be considered overcome. First, the study establishes the validity of a test that is easy to perform and can be used in a wide range of settings, including in the home.

logically aged) from geriatric (biologically aged) patients. This very ability to screen frail geriatric patients vs old individuals is an urgent matter in geriatrics but increasingly urgent in other specialties. For example, oncologists seek objective methods to identify patients eligible for standard and more aggressive interventions because they are chronologically old but not biologically old. Also, certain surgeons

vided it facilitates clinical decisions on the basis of a "hard" outcome such as life expectancy.

The study by Studenski et al⁴ may also be of interest for investigators involved in clinical research on aging. Researchers and clinicians seek results that are clinically, not just statistically, significant. Because of the established association of gait speed with survival, it could be used as a surrogate for survival outcomes in clinical trials of older persons.

Gait speed should not be regarded solely as a measure of lower extremity function. Gait speed has been associated with clinical (eg, comorbidities^{6,7}) as well as subclinical conditions (eg, atherosclerosis⁸ or inflammatory status⁹) and is able to predict several health-related events even apparently unrelated to physical function (eg, cognitive impairment, hospitalization, institutionalization). Gait speed may serve as a marker of physiological reserve and potentially could quantify overall health status."

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"...gait speed may be considered a new 'vital sign,' specifically sensitive for older persons."

- ***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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ELCN Part 3
Dr. Jeff Moss

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 KEVIN GACK

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. move on
exposed
Olgard
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. Olgard called "a tidal wave of chronic disease" — diabetes, hypertension, obesity, depression — that left many questioning how much any one physician could really accomplish.

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

Not sur
devalue

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

"You had
reimbur
treatments they can absorb early instead of waiting until there's a catastrophic surgery, it's pretty simple."

For its part, 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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Plan now so your later years can be comfortably extended

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.

USA Today, January 11, 2011

New Passages
By Gail Sheehy

Journalist and lecturer Gail Sheehy is the author of 16 books about adult life stages, including *Passages in Caregiving*.

There has been an even more dramatic increase among Medicare beneficiaries, who report three or more chronic conditions.

"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."

what they really want is individualized treatment if desired

chronic 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 peppered by harried staff with questions about what extreme measures we want or don't want, when all we can think about is getting out of pain.

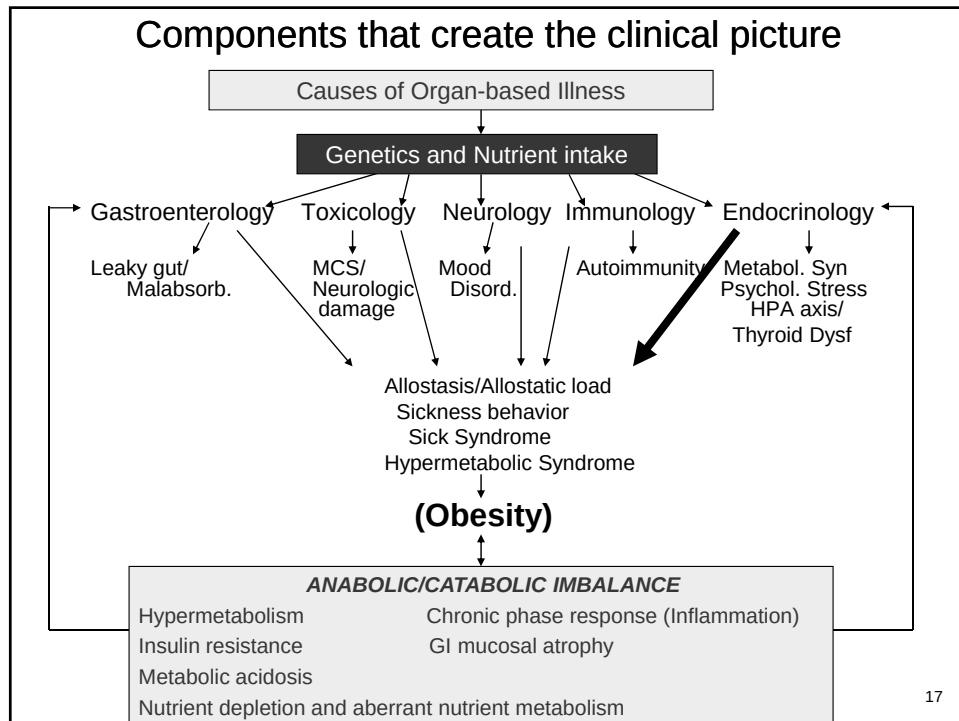
Palliative care, in my view, is the future of care for those of us entering the In-Between Stage. Its aim is to treat symptoms and relieve suffering, and it can be offered when we are first diagnosed with a serious illness. It's not the same as hospice, which is for those who are nearing the end of life.

people with a range of cancers, scleroderma, emphysema and shortness of breath," says Diane Meier, a geriatrician at the Mount Sinai School of Medicine.

Sadly, the more common route is a revolving door from hospital to rehab to hospitalization, which is especially debilitating for older people. They can't tell day from night. They can't fight antibiotic-resistant infections. They can't talk back to overworked 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.

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**Luc Cynober
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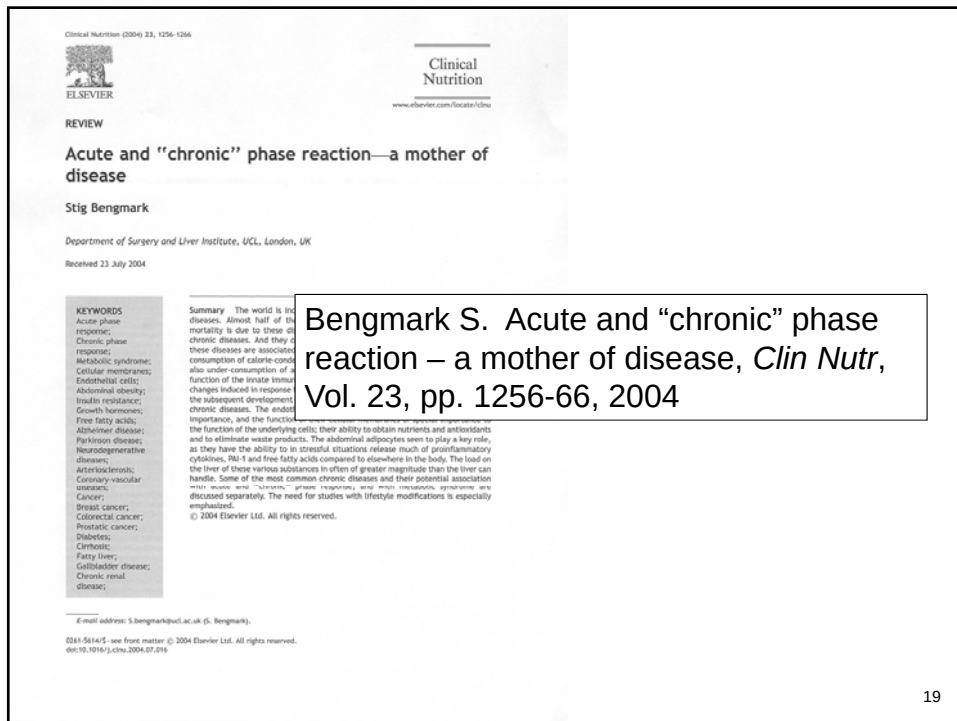
KARGER

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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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

Considerations for the Catabolic Patient				
Signs & Symptoms	Consider	Treatment Plan	Monitor	Supplementation
Signs & Symptoms: - Feeling "lousy" - Fatigue - Cravings - Depression/anxiety - Poor quality sleep - Poor digestion/absorption/leaky gut - Chronic pain/infection Clinical Findings: - High % body fat - Low bone density - High fasting insulin - Heavy metal burden - Calcium to phosphorus ratio 3:1 - High normal globulin - Cholesterol (220-280) - High LDL, low HDL - High triglycerides - Low normal serum potassium, high normal serum sodium - High normal BUN with normal creatinine - Low urinary sulfate - Genomic variations	Optimize pH	Optimize fluid and electrolyte balance	Morning urinary pH -pH paper	Potassium bicarbonate -K Alkaline (Moss) Magnesium -Magnesium Glycinate (Vital Nutrients)
	Optimize muscle mass (reduce sarcopenia) and amino acid levels	Pay special attention to protein and amino acids	Body composition: -Bioelectric impedance scale Grip strength: -dynamometer	Amino acids -Custom Amino Base (Metabolic Maint.) Protein powders/bars -UltraLean products (Biogenesis) -Paleo products (DFH)
	Reducing inflammation	Reduce inflammation Address food allergies	Inflammatory biomarkers: -HS-CRP -Fibrinogen levels -WBC -Differential	Anti-inflammatory agents -Inflametric (Olympian) -D-Flame (Protocols) -Pain Guard (Perque) Fish Oils -Ultra Pure Fish oil (Vital Nutrients) Food allergy blends -AllerGzymes (DFH)
	Improving insulin sensitivity	Watch carbohydrate/protein ratio	Fasting glucose Fasting insulin Cravings	Metabolic Synergy (DFH)
	Optimize dietary carbohydrate/protein ratios	Replete protein deficiencies and reduce carbohydrate intake along with improvements in dietary quality	Diet diary Optimal protein intake -1.0 - 1.2 g/kg body weight	Protein powders/bars -Paleo products (DFH) -UltraLean (Biogenesis)
	Optimize GI function	Dietary changes, supplementation of digestive aids	Patient signs and symptoms	Betaine HCl (Vital Nutrients) Digestzymes (DFH) Intestinal Repair Complex (Biogenesis)
	Optimize micronutrient deficiencies/imbbalances	Supplementation of multis Possible emphasis on vitamin D, iodine, and zinc	Serum 25 OH vitamin D Fatigue/serum thyroid indicators Zinc taste test	Multi Nutrients (Vital Nutr) Vitamin D (Vital Nutr) Iosol (Moss) Zinc Supreme (DFH)
Presented by: MOSS NUTRITION 800-851-5444 mossnutrition.com Tools for True Healing™				

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.

NEURO-SIGNALS

Invited Review

Neurosignals 2009;17:144-152
DOI: 10.1159/000168182

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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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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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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].

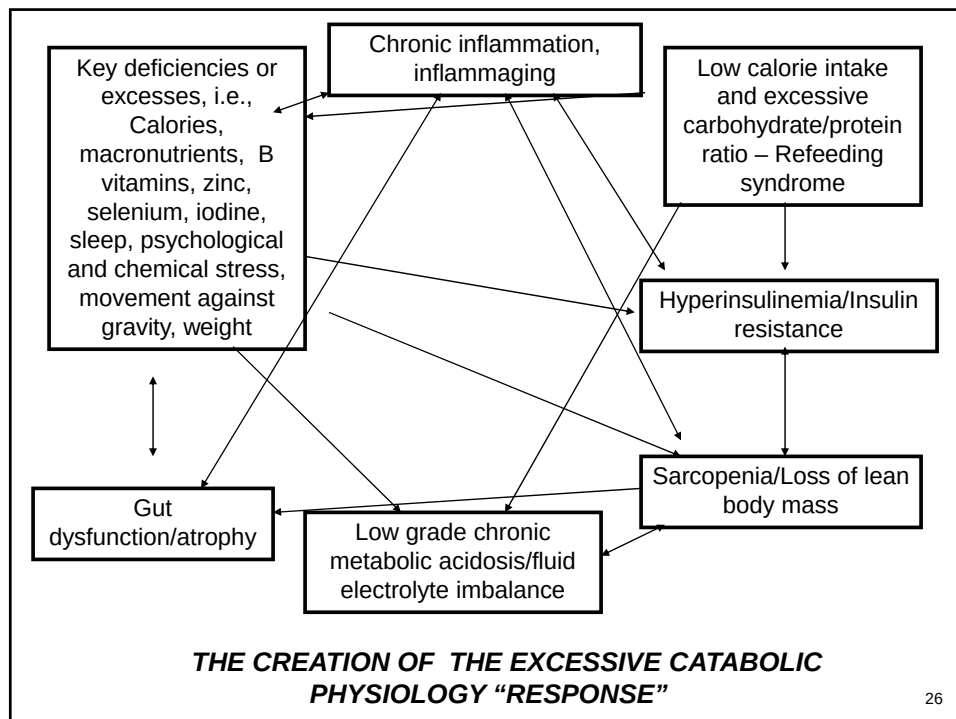
¹ Nausea, vomiting, bowel problems.

² Result from depressed inpatient population.

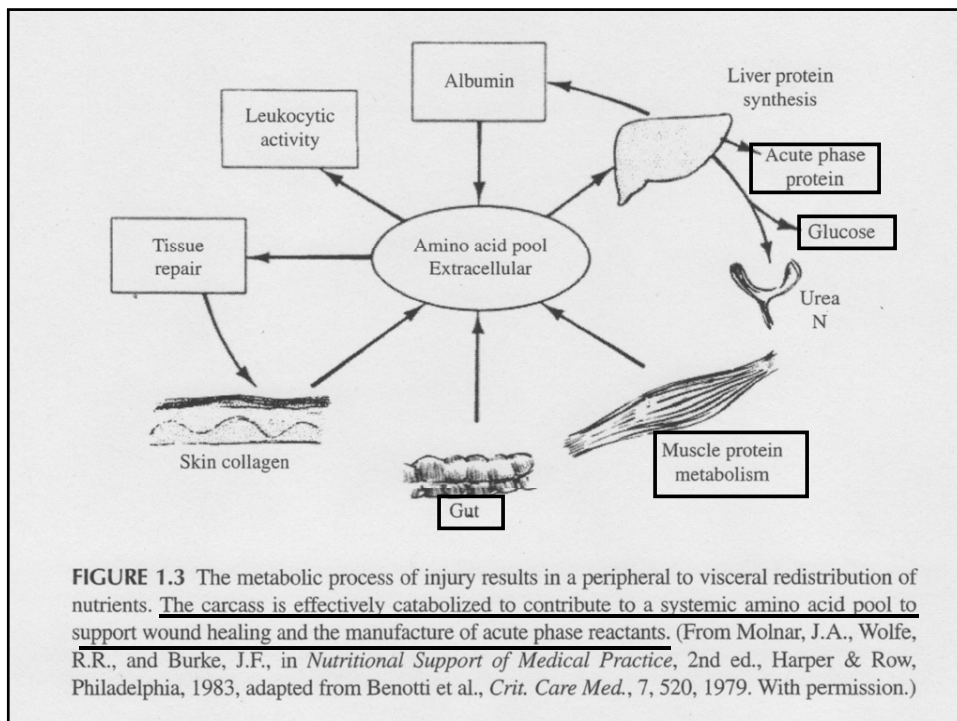
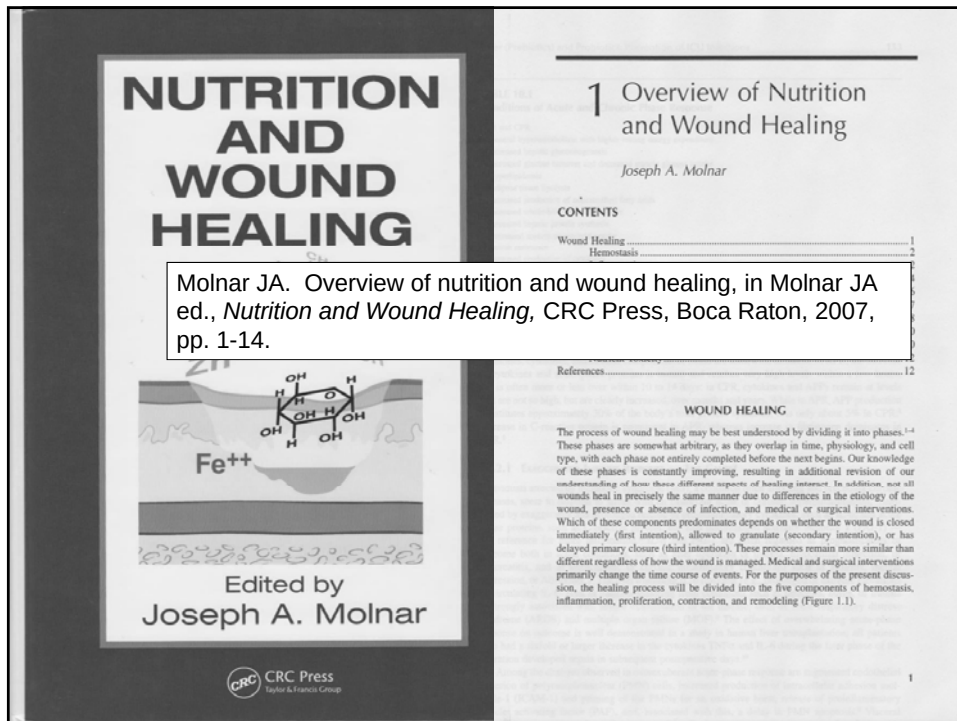
Key metabolic imbalances seen with the acute phase response

- 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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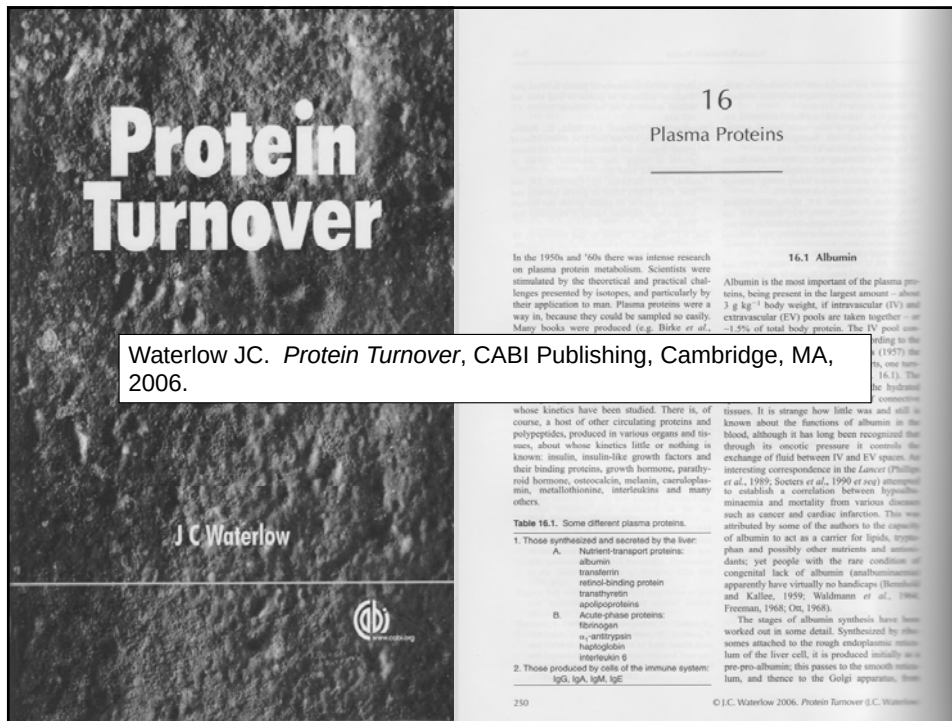


- “During periods of metabolic stress, the body is able to effectively catabolize the carcass (muscle, skin, bone) to support visceral protein synthesis of acute phase proteins, immunoglobulins, inflammatory cells, and collagen, needed to fight infection and heal the wound.”
- “In the case of proteins, 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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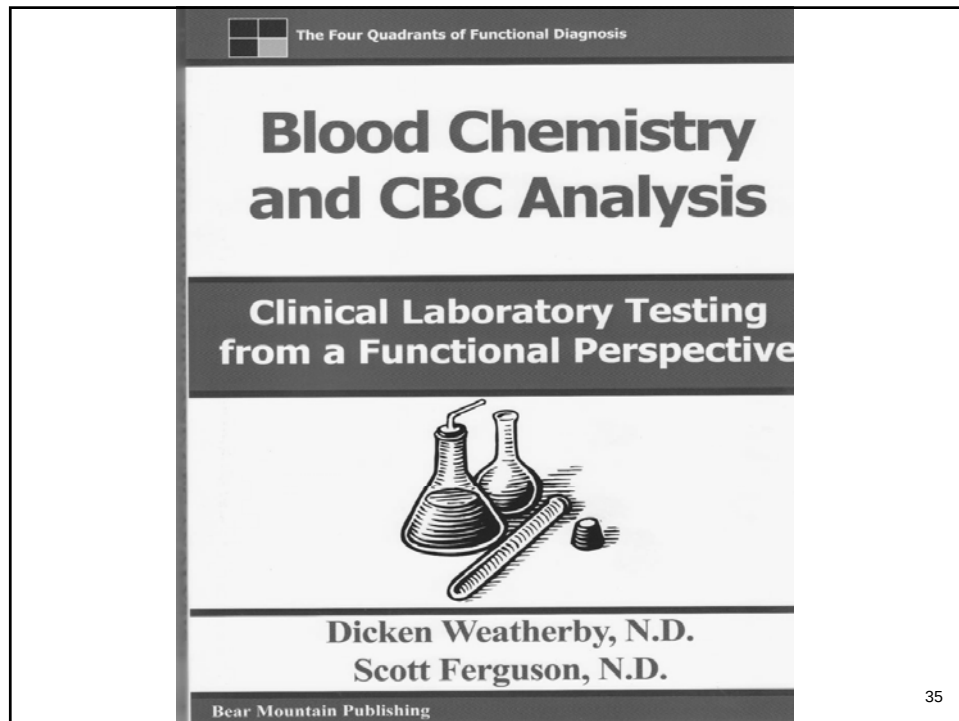
- “In many patients plasma albumin concentrations are low and it has been suggested that the acute phase proteins compete with albumin for the amino acids required for synthesis.”
- “An interesting hypothesis was proposed by Reeds *et al.* to relate the acute phase protein response to the muscle wasting that occurs in so many clinical conditions.”

- “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

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 T_3 and/or T_4
- 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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Serum antioxidants and inflammation predict red cell distribution width in older women: ... Page 1 of 2

Clinical Nutrition

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Articles in Press

Serum antioxidants and inflammation predict red cell distribution width in older women: The Women's Health and Aging Study I

Richard D. Semba^{1,2,3,4}, Kuehling V. Patel¹, Luigi Ferrucci¹, Kai Sun¹, Cindy N. Roy¹, Jack M. Guralnik¹, Linda P. Fried¹

Received 19 November 2009; accepted 1 March 2010; published online 08 March 2010.
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Summary

ABSTRACT

FULL TEXT PDF (145 KB)

KEYWORDS

RELATED ARTICLES

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Semba RD et al. Serum antioxidants and inflammation predict red cell distribution width in older women: The Women's Health and Aging Study, *Clin Nutr*, published online ahead of print March 8, 2010

Methods

RDW was measured at baseline, 12 months, and 24 months follow-up in 796 moderately to severely disabled community-dwelling women, aged 65 years, in the Women's Health and Aging Study I in Baltimore, Maryland.

“Red cell distribution width (RDW), a measure of heterogeneity in the size of circulating erythrocytes, is associated with some chronic diseases and predicts mortality.”

Serum selenium is an independent predictor of RDW and may potentially mediate effects on RDW through IL-6.

Keywords: Carotenoids, Interleukin-6, Oxidative stress, Red cell distribution width, Selenium, Vitamin E

“Serum selenium is an independent predictor of RDW and may potentially mediate effects on RDW through IL-6.”

<http://www.clinicalnutritionjournal.com/article/PIIS0261561410000427/abstract> 9/23/2010 43

Medical News Today News Article - Printer Friendly Page 1 of 1

Medical News Today

www.medicalnewstoday.com

BUSM Researchers Uncover Cellular Mechanism Responsible For Chronic Inflammation, Type 2 Diabetes

31 Dec 2010 [Click to Print](#)

Researchers from Boston University School of Medicine (BUSM) have demonstrated that certain T cells require input from monocytes in order to maintain their pro-inflammatory response in people with type 2 diabetes (T2D). The study also showed, for the first time, how a loss in homeostasis in this group of T cells most likely promotes chronic inflammation associated with T2D.

Barbara Nikolajczyk, PhD, an associate professor of microbiology and medicine at BUSM, is the senior author of the study, which is currently featured in an online edition of the *Journal of Immunology*.

T2D is a chronic inflammatory disease in which the body has high levels of glucose in the blood due to the lack of insulin or the body's inability to use insulin efficiently. The incidence of T2D continues to rise at alarming rates in both children and adults in the United States.

Previous research done in mice has shown that T cells play a critical role in the development of insulin resistance in response to a high fat diet, often leading to T2D. Additional findings indicate that T cells exhibit a pro-inflammatory response more often than an anti-inflammatory response.

Working with human T cells, the team observed that in order for T cells to exhibit the pro-inflammatory response, they required constant interaction with monocytes, indicating that monocytes play an indirect role in chronic inflammation and T2D.

While it is not known what the homeostatic balance levels are between pro-inflammatory and anti-inflammatory T cells, this study indicates the need to restore a balance in order to halt chronic inflammation and T2D.

"The true importance of our observations is the indication that altering balance among immune system cells could be a fundamentally novel treatment for T2D-associated inflammation and perhaps insulin resistance," said Nikolajczyk.

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Elevated Proinflammatory Cytokine Production by a Skewed T Cell ... <http://jimmunol.org/>

Elevated Proinflammatory Cytokine Production by a Skewed T Cell Compartment Requires Monocytes and Promotes Inflammation in Type 2 Diabetes

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Abstract

An appropriate balance between proinflammatory (Th17 and Th1) and anti-inflammatory (regulatory T cells [Tregs] and Th2) subsets of T cells is critical to maintain homeostasis and avoid inflammatory disease. Type 2 diabetes (T2D) is a chronic inflammatory disease promoted by changes in immune cell function. Recent work indicates T cells are important mediators of inflammation in a mouse model of T2D. These studies identified an elevation in the inflammatory cells from signs of inflammation in a mouse model of T2D. These studies identified an elevation in the inflammatory cells from signs of inflammation in a mouse model of T2D. These studies identified an elevation in the inflammatory cells from signs of inflammation in a mouse model of T2D.

Jagannathan-Bogdan M et al. Elevated proinflammatory cytokine production by a skewed T cell compartment requires monocytes and promotes inflammation in type 2 diabetes, *J Immunol*, Published online ahead of print December 2010.

Footnotes

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“These data indicate that T cells in T2D patients are naturally skewed toward proinflammatory subsets that likely promote chronic inflammation in T2D through elevated cytokine production.”

T2D
Type 2 diabetes
Treg
regulatory T cell.

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C-Reactive Protein, Dietary Factors and Disease Risk

REVIEW

Impact of C-Reactive Protein on Disease Risk and Its Relation to Dietary Factors: Literature Review

Akiko Nanri¹, Malcolm A Moore¹, Suminori Kono¹

Abstract

C-reactive protein (CRP) is one of the acute-phase proteins in inflammation and CRP serum concentrations are therefore of interest. Data for high-sensitivity CRP (hs-CRP) with a low detection limit of approximately 0.04 mg/L have become available over the past decade and research has shown a link between high concentrations of hs-CRP and obesity as well as smoking. Expanded adipose tissue is in fact known to secrete proinflammatory cytokines which enhance hepatic synthesis of CRP. Moderate alcohol consumption and high physical activity

Nanri A et al. Impact of C-reactive protein on disease risk and its relation to dietary factors: Literature review, *Asian Pacific J Cancer Prev*, Vol. 8, pp. 167-177, 2007

Both mechanistic and epidemiologic studies regarding dietary factors and low-grade inflammation are necessary to add to our knowledge of dietary influence on chronic disease development.

Key Words: C-reactive protein - disease risk - diet

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Introduction

to these ligands activates the classical complement

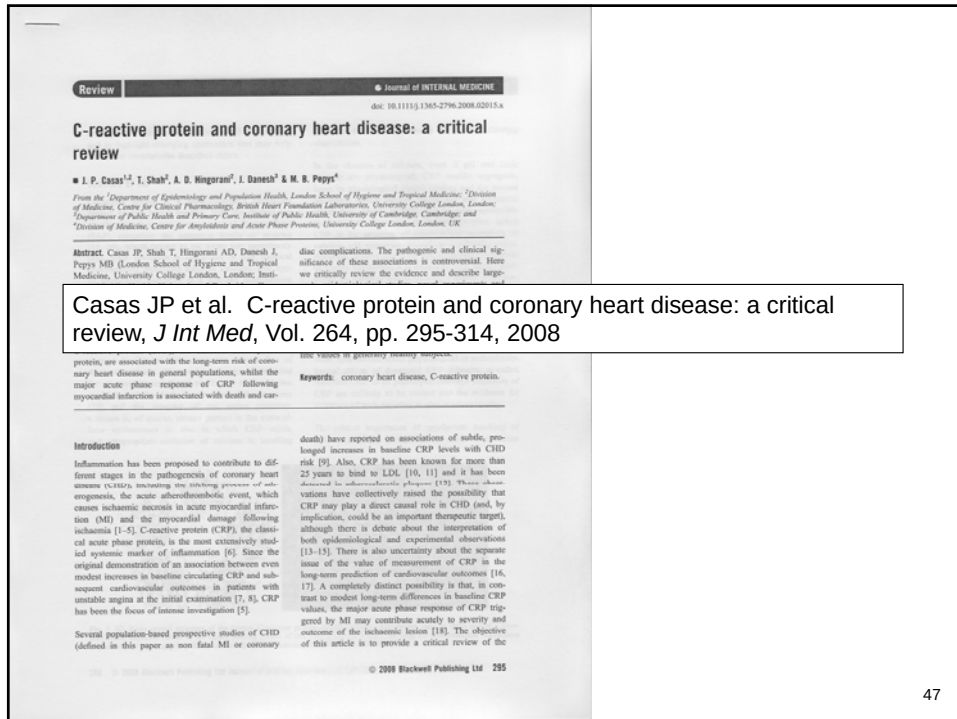
“Circulating CRP is present in only trace amounts in healthy individuals, and is hardly detectable by the standard clinical tests, which typically have a lower detection limit of 3-8 mg/L. CRP concentrations of recent research interest are in the so-called normal range which can be measured only by the high-sensitivity testing. The lower detection limit for high-sensitivity CRP (hs-CRP) is as low as approximately 0.04 mg/L.”

1991; Ahli and Meisner, 2002; Peppy and Hirschfeld, 2003). CRP binds to endogenous and exogenous ligands such as constituents of microbes, native and modified lipoproteins, necrotic cells, and apoptotic cells. CRP-bound which also show a temporal profile similar to the change of CRP in acute inflammation. The reasons for the wide use of hs-CRP are stability and availability of robust, automated methods for its measurement (Pearson et al., 2002).

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Asian Pacific Journal of Cancer Prevention, Vol 8, 2007 167

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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.”

- “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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