

December 1997 Issue | Morrison C. Bethea, M.D.

<http://jeffreybland.com/knowledgebase/december-1997-issue-morrison-c-bethea-m-d/>

[DOWNLOAD AUDIO](#) |

Welcome to *Functional Medicine Update*™ for December, 1997. By this time, you should have received the announcement of the Fifth International Symposium on Functional Medicine. The Symposium will be held May 2 through 7, 1998, at the beautiful Orchid at Mauna Lani, on the island of Hawaii.

The focus of the symposium will be Functional Medicine Applications to Disorders of Gene Expression. The three principal days of the symposium will deal with things like homocysteine, inflammation and modifying coronary heart disease, arthritis, and dementia. The second day will deal with nutrient modulation of adverse side effects of drugs, and the third day will focus on nutrient modulation of cancer genes.

We will have presenters who are at the forefront of research and clinical application in those topic areas, and some good workshops to integrate these concepts into effective clinical practice. Over the next few months, you will hear more about the topic areas, and this will be part of the focus in *Functional Medicine Update*™.

This month, I want to focus on the theme of modulating the aspects of premature, age-related diseases by recognizing certain functional risk factors. These risk factors extend to functional aspects of cellular physiology, interrelationships of organ systems, and the web-like pattern we have been describing in previous issues of *FMU*. That web-perspective allows recognition of individualized patterns of health or age-related risk factors, which can then be modified. We are talking about modifying risk factors by asking the right questions and getting different answers.

We will focus on some of those modifiable areas and try to bring them into clinically meaningful applications. First, we will look at mitochondrial DNA relationships to dysfunction as a central functional medicine theme that is emerging from the literature. Second, we will look at the molecular medicine modification of those risks. We will discuss what Dr. Linus Pauling and Dr. Roger Williams described years ago as the molecular milieu, trying to adjust this milieu in the cell to promote better function and reduce the risk to even a genetically encoded disease.

We will focus on pathogenesis of the red cell, looking at thalassemias and sickle cell disorders as a model. Next, we will look at adverse reactions to the environment, including adverse reactions to drugs. We will move from that discussion to endotoxicity-related reactions, with specific emphasis on chronic fatigue syndrome. Last, we will discuss some age-related phenomena associated to dysglycemia, or the poor management of blood sugar and insulin levels.

Let's begin by looking at the mitochondrion, the site of energy production in cells. The clinical symptoms of fatigue and pain (the two symptoms that most often bring patients to the offices of health providers) are interesting in their origin. We can look at them as whole-organism problems of functional disability, problems of organ systems (organs and tissues), and finally in terms of the cells and the subcellular milieu.

What is the origin of fatigue and pain? The immediate answer is that both conditions relate to a myriad of interacting messenger substances and cell physiological dysfunction that ultimately give rise to fatigue and pain. We cannot pinpoint a specific cause. We can't say the CPT code, or the diagnostic code, for fatigue and pain is "blank," and then fill in the blank with a single explanation. What we can say is that there is an emerging view that many of the conditions associated with fatigue and pain are a consequence of alterations in mitochondrial function and energy transport.

If the mitochondrion is the organelle in the cell that is the responsible site for energy production and transmission, then the root origin of some of the problems can be traced back to mitochondrial dysfunction. As molecular techniques have improved in power and precision, people are starting to look at mitochondrial DNA as a record of a person's genetic history.

The mitochondrion is the only site in the cell that is known to contain genetic material -- polydeoxyribonucleic DNA -- that is outside of the cell nucleus and the traditional chromosomes. Presumably, the form of mitochondrial DNA is very different from nuclear DNA. It is circular, and it is not bound up with histone and non-histone proteins. Therefore, it is very clearly exposed to oxidant stress, and it doesn't have the coats of protection that nuclear DNA has.

According to Dr. Bruce Ames at the University of California at Berkeley, levels of oxidative damage to mitochondrial DNA is at least 10-fold higher than those of nuclear DNA. That means mitochondrial DNA is susceptible to mutagenic chromosomal or, in this case, genetic damage.

INTERVIEW TRANSCRIPT

Morrison C. Bethea, M.D.
1633 Napoleon Avenue, Suite 1001
New Orleans, Louisiana 70115

To order Dr. Bethea's book, *Sugar Busters*, call or fax (504) 897-6770

JB: We have the pleasure to have with us for the first time as our Clinician of the Month on *Functional Medicine Update*[™] a vascular and thoracic surgeon. Dr. Morrison Bethea has had many years of experience in the surgical theater, and he has an interest and a demonstrated knowledge of nutrition and its relationship to his patients. That is an interesting blend, but it's not a common combination.

We are fortunate to have Dr. Bethea with us today. He is a graduate of Davidson College and Tulane University School of Medicine. He completed his postgraduate training in thoracic and cardiac surgery at the Columbia Presbyterian Medical Center in New York and practices thoracic, cardiac, and vascular

surgery in New Orleans. He is a medical consultant to Freeport-McMoran worldwide operations and is on the Taylor Energy and Advisory Board of Mercy Hospital in New Orleans. We welcome you, Dr. Bethea, to *Functional Medicine Update*™.

JB: Let me start by asking a question that our listeners are probably wondering about. How would a vascular, thoracic surgeon ever come to be interested in something related to nutrition? It seems that it's not a traditional part of a surgeon's interest.

MB: Well, it's not a traditional part of a surgeon's interest, but I think it's a very important part that maybe we have overlooked. First, I'd like to thank you for having me on *Functional Medicine Update*™. I've enjoyed your tapes and have gleaned a lot of useful information from them. So, to have an opportunity to participate with you is an added pleasure.

JB: Thank you so much. You are one of the co-authors of a book that Barbara Schiltz, who works in our clinical research center, had read and was very impressed with. She told me I had to read this book because it is so understandable and so motivating for patients to get some insight into their nutrition status. It's called *Sugar Busters - Cut Sugar to Trim Fat*. I found it a well-written, insightful book. You've taken a lot of sophisticated information and distilled it down into an effective learning system for patients. Tell me how this book came about and how it relates to what you do as a medical professional.

MB: It came about as a result of an experience of one of my co-authors, Leighton Steward. While visiting in Paris, he came across a book written by Michel Martignac, a Frenchman. The title of the book is *Dine Out and Lose Weight*. Michel had noticed that obesity is increasing at an alarming rate throughout the world, especially in developed countries. He made the observation that the French people, by and large, have less obesity. Indeed, if one looks at World Health Organization studies, you would see that the French, as a population, have lower cholesterol and, on an age-adjusted basis, fewer strokes and heart attacks than those of other developed countries.

Martignac wrote a book outlining specifically how the French have eaten for decades. As you know, they eat wonderful food. They are gourmets, but they eat lean and trimmed meat, high-fiber vegetables, and whole-grain cereals and bread. The higher fiber content has a tremendous effect on metabolic processes.

Leighton brought this book back and asked me what I thought about it. I told him I thought it was very logical and practical, but if people were going to be compliant, they needed to understand why it worked. So, as we began to look into the concept that was later mirrored in the book *Sugar Busters*, we saw that the bottom line was basically controlling insulin secretion -- modulating it downward while at the same time increasing glucagon secretion.

And just this year on your program, you had several discussions about the importance of insulin, the importance of glucagon, and the importance of maintaining blood sugars in an acceptable range. That's what *Sugar Busters* is doing.

Obviously, all of these factors have a tremendous influence on the cardiovascular system. The most common question asked me in my practice today is, "Doctor, how can I avoid getting arteriosclerosis, atherosclerosis, or hardening of the arteries?" My answer is that by using healthy lifestyle habits they can decrease the progression of it.

Arteriosclerosis is an aging process. As we get older, we will all be victims of these changes, but by using healthy lifestyle habits in the form of nutritional support, vitamins, and some other things like antioxidants, we can decrease the progression of this process. We can slow our aging. So that's where my interest developed, because I saw an area that we have really not taken advantage of that can have significant ramifications to our benefit in the cardiovascular arena

JB: That is very insightful. I was interested to see that one of the co-authors of the book is Dr. Samuel Andrews, a graduate of the Louisiana State University School of Medicine who works in the endocrinology area, and another was Dr. Luis Balart, also a graduate of Louisiana State Medical University, who works at the New Orleans Oshner Clinic in gastroenterology. It appears you've got an interesting team, along with Leighton Steward, in the way that you've viewed this. Do you four have the chance to sit down and discuss how these concepts translate across your disciplines?

MB: We certainly have and we continue to do so. More and more information substantiating our basic concept of controlling insulin secretion is coming to the forefront. Sam Andrews, an endocrinologist, has a large practice of diabetic patients, and on our *Sugar Busters* concept he has been able to reduce, if not eliminate, insulin injections for many of his patients. Luis is also head of the liver transplant program at LSU Medical Center and has a superb knowledge of hepatic metabolism.

As you know, the liver is our metabolic computer. It orchestrates what happens to everything we consume in a day's time. We felt that by combining expertise in the cardiovascular area with the expertise of an endocrinologist and a hepatologist, we would have all the expertise we needed to be able to confidently put together what we have done in *Sugar Busters*. Leighton, being the CEO, has been our manager. He's the one who keeps everyone on focus and moving toward our objective.

BIBLIOGRAPHY

1. Bãnhgyi G, Braun L, Csala M, Puskàs F, Mandl J. Ascorbate metabolism and its regulation in animals. *Free Rad Biol Med.* 1997;23(5):793-803.
2. Bianchi GP, Marchesini G, Fabbri A, et al. Vegetable versus animal protein diet in cirrhotic patients with chronic encephalopathy: a randomized cross-over comparison. *J Intern Med.* 1993;233:385-392.
3. Braun L, Puskàs F, Csala M, Mészáros G, Mandl J, Bãnhgyi G. Ascorbate as a substrate for glycolysis or gluconeogenesis: evidence for an interorgan ascorbate cycle. *Free Rad Biol Med.* 1997;23(5):804-808.
4. Bunn HF. Pathogenesis and treatment of sickle cell disease. *N Engl J Med.* 1997;337(11):762-767.
5. Charlton MR. Branched chains revisited. *Gastroenterol.* 1996;111:252-255.
6. Freese R, Mutanen M. α -linolenic acid and marine long-chain n-3 fatty acids differ only slightly in their effects on hemostatic factors in healthy subjects. *Am J Clin Nutr.* 1997;66(3):591-598.
7. Girodon F, Blache D, Monget AL, et al. Effect of a two-year supplementation with low doses of antioxidant vitamins and/or minerals in elderly subjects on levels of nutrients and antioxidant defense parameters. *J Am Coll Nutr.* 1997;16(4):357-365.
8. Grant KE, Chandler RM, Castle AL, Ivy JL. Chromium and exercise training: effect on obese women. *Med Sci Sports Exercise.* 1997;29(8):992-998.

9. Hambly RJ, Rumney CJ, Cunninghame M, Fletcher JM, Rijken PJ, Rowland IR. Influence of diets containing high and low risk factors for colon cancer on early stages of carcinogenesis in human flora-associated (HFA) rats. *Carcinogenesis*.1997;18(8):1535-1539.
10. Hechter O, Grossman A, Chatterton RT. Relationship of dehydroepiandrosterone and cortisol in disease. *Med Hypotheses*. 1997;49:85-91.
11. Ito S, Miyaji H, Azuma T, et al. Hyperammonaemia and Helicobacter pylori. *Lancet*.1995;346:124-125.
12. Jaradat ZW, Marquardt RR. L-arginine as a therapeutic approach for the verotoxigenic *Escherichia coli*-induced hemolytic uremic syndrome and thrombotic thrombocytopenic purpura. *Med Hypotheses*. 1997;49:277-280.
13. Linday LA. Trivalent chromium and the diabetes prevention program. *Med Hypotheses*. 1997;49:47-49.
14. Macbeth WA, Kass EH, McDermott WV Jr. Treatment of hepatic encephalopathy by alteration of intestinal flora and Lactobacillus acidophilus. *Lancet*. 1965;1:399-403.
15. McGregor NR, Dunstan RH, Butt HL, Roberts TK, Klineberg IJ, Zerbes M. A preliminary assessment of the association of SCL-90-R psychological inventory responses with changes in urinary metabolites in patients with chronic fatigue syndrome. *J Chronic Fatigue Syndrome*. 1997;3(1):17-37.
16. More evidence for a mitochondrial DNA defect in Parkinson's disease. *Parkinson's Disease Update*. 1997;13:460-461.
17. O'Grady JG. Paracetamol hepatotoxicity: how to prevent. *J Royal Soc Med*.1997;90(1):368-370.
18. Read AE, McCarthy CF, Heaton KW, Laidlaw J. *Lactobacillus acidophilus* (Enpac) in treatment of hepatic encephalopathy. *BMJ*. 1966;1:1267-1269.
19. Reddy PL, Bowie LJ, Callistein S. Binding of nitric oxide to thiols and hemes in hemoglobin H: implications for α -thalassemia and hypertension. *Clin Chem*.1997;43(8):1442-1447.
20. Reznick AZ, Han D, Packer L. Cigarette smoke induced oxidation of human plasma proteins, lipids, and antioxidants; selective protection by the biothiols dihydrolipoic acid and glutathione. *Redox Rpt*. 1997;3(3):169-174.
21. Riordan SM, Williams R. Treatment of hepatic encephalopathy. *N Engl J Med*.1997;337(7):473-479.
22. Rivabene R, Straface E, Giammarioli AM, Rainaldi G, Malorni W. Combined effect of 3-aminobenzamide and N-acetylcysteine on HIV replication in chronically infected U937 cells. *Redox Rpt*. 1997;3(3):145-151.
23. Scott MD, Yang L, Ulrich P, Shupe T. Pharmacologic interception of heme: a potential therapeutic strategy for the treatment of β thalassemia? *Redox Rpt*. 1997;3(3):159-167.
24. Serjeant GR. Sick-cell disease. *Lancet*. 1997;350(9079):725-730.
25. Shipman P. Does mtDNA saw Neandertal limb off human family tree? *J NIH Res*.1997;9:30-31.
26. Silvestri G. Age-related macular degeneration: genetics and implications for detection and treatment. *Mol Med Today*. 1997;3(2):84-91.
27. Weinberg ED. The lactobacillus anomaly: total iron abstinence. *Perspect Biol Med*.1997;40(4):578-583.
28. Wiedmann B, Kircheis G, Klauk S, Günther L, Malferheiner P. The influence of Helicobacter pylori (Hp) on protein-induced hyperammonemia (0.5 g/kg B.W.) in cirrhotic patients without hepatic encephalopathy. *Hepatology*.1996;24(suppl453A):abstract.
29. Zemel MB. Dietary pattern and hypertension: the DASH Study. *Nutr Rev*.1997;55(8):303-305.

p>