

THE AFIB REPORT
Your Premier Information Resource for Lone Atrial Fibrillation
Publisher: Hans R. Larsen MSc ChE

VIRTUAL LAF CONFERENCE

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SUBJECT: Cardiac Fibrotic Remodeling

Please note that this initial posting is rather long. You may find it more convenient to download it at

www.afibbers.org/fibrosis.pdf

and then print it out for perusal in greater comfort :-)

So here goes!

Cardiac Fibrotic Remodeling – The Role of Fibrosis in LAF

INTRODUCTION

Atrial fibrosis – is it simply the result of AF which then causes more episodes or might it be that fibrotic remodeling as a result of aging leads to AF?

Or is the natural decline of fibrolytic enzymes at fault? Further, might we consider the oxidative stress connection? And let's also examine the evidence suggesting the pro-fibrotic effects of magnesium deficiency.

These questions were posed to me by Erling Waller ex-afibber (75) who thinks there may be a connection. His afib began at 64 and then 10 years later, after some nutritional adjustments, his AF vanished. (See his story in Hans' book.)

Erling has been a regular and prolific contributor of important information and was always generous with his time and support to help others seeking answers. He continues to ponder the origin of AF and finds the fibrosis remodeling connection not only intriguing but also plausible. Erling is tackling another remodeling project at the moment and asked me to present this topic to the BB.

Interrelated factors in the “Fibrotic Remodeling Theory”

- I. Fibrosis mechanism
- II. Mechanisms of AF
- III. Inflammation and C-reactive Protein
- IV. Oxidative Stress and Nitric Oxide/Peroxynitrite production
- V. Magnesium deficiency

BACKGROUND

First, you must go to this site and view the color photo of a fibrotic heart and the quick time video of electrically-stimulated cardiac myocyte contracting. Keep the image of this heart in your memory as you consider fibrosis as a connection to AF.

http://www.clevelandclinic.org/heartcenter/pub/atrial_fibrillation/AFresearch.htm

This paper published by the CCF, states that atrial fibrosis is simply the result of atrial fibrillation (AF). Author and researcher David Van Wagener, PhD, bases his conclusion on research studies. (1)

Let's begin –

I. FIBROSIS

The body's natural scar formation or healing defense-mechanism. When functioning optimally, and in response to injury, too much reparative fibrin is always laid down and then lysed (broken down) by fibrolytic enzymes; then removed from the body via the circulatory system.

Research shows that around age 27, the body begins to build fibrotic tissue because of a decline in fibrolytic enzymes production. In atrial fibrillation, this could explain why younger people are frequently affected and also why the general age group for the onset of AF seems to be around the age of 60 years. Some individuals lack fibrolytic enzyme production ability at earlier ages than others. (Biochemical individuality.)

A logical discussion on the role of fibrosis – in terms of our causative investigation – comes from William Wong PhD, a Classical Naturopath, Exercise Physiologist, Certified Athletic Trainer (AATA), Certified Sports Medicine Trainer (ASMA) in his article, "Fibrosis - Enemy of Life." (2) He says:

"In all of us as we age (after 27), fibrosis grows inside of all of our internal organs diminishing their size and with that shrinkage comes a diminution of function. Med school anatomy teaches this lowering of function is what ultimately leads to us dying as the organs fail due to weakness.

... this leads to the question: Why does this seem to start after 27? At or around 27 our own production of proteolytic enzymes drops.

We make a finite amount of enzymes in a lifetime and use about half of that by 25. (That's the reason why young folks, though they make cancer cells from the first day of life don't usually develop that or most any of the other conditions mentioned, they have an adequate supply of proteolytic enzymes to fight off fibrosis

It is after our supply of proteolytic enzymes drops to be spread through the rest of our lifetime that we begin to develop the fibrosis conditions.

(For you docs out there it's my contention that we can measure a pre morbid state from taking measures of proteolytic enzymes just as we can predict death within 3 days by measuring the levels of Dopamine. Useful diagnostic tool maybe. Nifty research tool certainly).

So if we can deal with the laying down of fibrosis as efficiently as we did as youngsters, then we would avoid or reduce much of what is trying to shorten our lives or at least make us sick or less able.

The most important thing to put back into an aging body is are not vitamins and minerals, not herbs, not the growth hormones but enzymes, the proteolytic enzymes.

Vitamins and minerals are more properly named co enzymes and co factors in other words they are things that help enzymes to work. If the enzymes aren't there to begin with, then the vitamins and minerals have little to work on and little action.

That's the reason why vitamin / mineral supplementation works so well for some and does not do squat for others, they have little of the enzymes they need to work on.

If we put in some of the primary protein-eating enzymes, then the body will cause the "enzyme cascade" creating thousands of new enzymes from the original 4 or 5.

Regarding fibrin.... all proteolytic enzymes eat away at fibrin (fibrinolysis) to some degree but some are considerably stronger at that than others. If the proteolytic enzymes you put back are also very highly fibrinolytic then the scar tissue your body has been creating WILL be taken away.

(This is a secret that plastic surgeons, internists and pulmonologists i.e. lung doctors, are learning about systemic enzymes). The fibrin that is supposed to be there is marked by the body as an endogenous protein, in other words something that is supposed to be part of your structure, but excesses in fibrin, though deposited by the body, are marked as exogenous proteins - or as something not belonging in the body.

Remember excesses in fibrin equal:

- * weak structure, (by not leaving enough space for epithelial tissue to grow through the fibrin matrix),
- * restriction of range of motion (joints and muscles)
- * diminution of size and function (internal organs).

(End of Wong quote.)

Conclusions in "Cardioreparation in Hypertensive Heart Disease" (HHD) (3)

This paper addresses cardiac fibrotic remodeling in general; not resulting from AF but rather, from hypertensive heart disease(HHD). Note the author is addressing structural remodeling and treatment by cardioprotective or cardioreparative strategies which support Wong's findings.

Quote:

The potential to regress cardiac fibrosis by ACE inhibition and to reverse ventricular diastolic dysfunction is supported by experimental studies in rats with genetic hypertension and the recent clinical trial by Brilla et al in patients with HHD.

The importance of pathologic structural remodeling, not simply the control of arterial pressure, needs to be addressed in recognizing that quality, not quantity, of myocardium in HHD is responsible for adverse cardiovascular events, management must no longer solely focus on a regression in left ventricular mass. Far more desirable is a regression of left ventricular mass and fibrosis with correction of ventricular dysfunction.

Moreover, cardioprotective and cardioreparative interventions specifically target such remodeling with the view toward respectively preventing or regressing cardiac fibrosis in HHD and in so doing favorably influencing adverse risk."

This article suggests the adverse accumulation of matrix protein be reduced by proteolytic digestion...a cardioreparative measure. (3) End quote (Be sure to read this article.)

II. MECHANISMS: ATRIAL FIBRILLATION

AF is a progressive disease; numerous lines of evidence suggest that disease progression results from cumulative electrophysiological and structural remodeling of the atria.(5) Ongoing events coupled with fibrotic remodeling perpetuate longer episodes. (4)

In many patients, AF begins with short episodes, typically characterized as "palpitations" (a fluttering sensation in the chest), or "paroxysms."

Over time, there is a tendency for these episodes to become longer. Why does this happen? Once AF has been initiated, the atria undergo a process known as "remodeling." (1)

Structural changes: (remodeling) In time, afib that persists for years, is also accompanied by significant degenerative structural changes. (1)

- Individual muscle cells within the fibrillating atria tend to become elongated and sometimes wider. AF-induced atrial remodeling causes both structural and electrical changes(1)

- Fibrosis: In addition, the space between individual myocytes typically becomes more fibrotic, with fatty infiltration, and the atria is less able to contract. (1)

Electrical changes: (remodeling)

- Fibrillating atria tend to have more complicated patterns of electrical activity. This is due both to the increased fibrosis, and to intrinsic changes in the electrical activity in the atrial myocytes. Research at the CCF has helped to characterize the electrical remodeling process associated with long-standing AF. (1)

- The net result is that in patients with persistent AF, the atria are more able to sustain fibrillatory activity, due to the combined effects of both structural and electrical remodeling. (1)

Atrial Electrical Remodeling (sustains afib) Refer to the video web page.

- The net result of the electrical and structural changes is that the enlarged atria are more likely to sustain fibrillatory activity. Thus, AF can persist for a longer duration in the remodeled atria. (1)

- Calcium ions have an important role in both the electrical and contractile activity of the heart. Thus, calcium overload is implicated as an early event in the electrical remodeling process. (1)

- Persistent AF results in further changes in protein expression, loss of myofibrillar structure, and eventually myocyte death and replacement fibrosis. (1)

Atrial Structural Remodeling

The fibrillating atria are subjected to continuous, high rate electrical activity (with rates up to 500 per minute). This results in impaired atrial contractility, and the initiation of structural changes. (Be sure to look at the picture of a heart exhibiting AF-induced fibrosis and the stained fibrotic-tissue studies.) (1)

- At the macroscopic level, structural remodeling is frequently characterized by increased atrial fibrosis and fatty infiltration, both on the endocardial surface, and between muscle bundles. (1)

- At the microscopic level, fibrosis can isolate muscle bundles. (1)

- (Key Point) It is evident that fibrosis can isolate muscle bundles and that this can alter the pathway of electrical activation, creating a substrate that can promote the persistence of atrial fibrillation. (1)

IONIC REMODELING IN THE HEART

Pathophysiological Significance and New Therapeutic Opportunities for Atrial Fibrillation (Circulation Research. 2000;87:440.) © 2000 American Heart Association, Inc.

Heart disease has long been recognized to alter cardiac electrical function. Detailed studies of disease-induced remodeling of ionic transport processes that underlie ventricular electrophysiological alterations have been performed over the past 10 years, but our knowledge of atrial ionic remodeling is more limited and has emerged much more recently.

(This) review focuses on recent findings regarding ionic remodeling at the atrial level, particularly with respect to two conditions that promote atrial fibrillation (AF) in well-developed clinically relevant animal models: (1) sustained atrial tachycardia and (2) ventricular tachypacing-induced congestive heart failure.

Morphology of atrial myocardium in human pulmonary veins: a postmortem analysis in patients with and without atrial fibrillation. (Pub Med)

..... significant differences in the histology of PVs between the two groups were a higher frequency of discontinuity and hypertrophy and a higher degree of fibrosis of the atrial myocardium in the PVs of patients with AF.

Atrial myocardium was more often present in the PVs of patients with compared with patients without AF. In the first group, the atrial myocardium in the PVs was characterized by more severe discontinuity, hypertrophy, and fibrosis. A marked variation in anatomical dimensions of the PVs existed.

Atrial fibrillation: the tip of the iceberg. Arch Mal Coeur Vaiss. 2002 Sep; 95(9): 827-32.

Atrial fibrillation (AF) usually results from profound alterations of the functional properties and structure of the atrial myocardium.

The electrical remodeling of diseased atria is most often associated with severe tissular and cellular alterations including: fibrosis, myocyte dystrophy with myolysis and dedifferentiation, apoptosis and gap junction disorganization. These abnormalities could result from a common and non specific adaptive response to changes in the working conditions of the atrial myocardium

Electrical remodeling in atrial fibrillation. Time course and mechanisms. Circulation. 1996 Dec 1; 94(11): 2968-74.

Atrial fibrillation is self-perpetuating, suggesting that the tachyarrhythmia causes electrophysiological changes that contribute to the progressive nature of the disease. In animal models, pacing-induced rapid atrial rates result in sustained atrial fibrillation. This is mediated by shortening of refractory periods termed electrical remodeling. The purpose of the present study was to characterize the time course of electrical remodeling and to define mechanisms of the phenomenon.

Atrial electrical remodeling develops quickly, is progressive, and may be persistent. Shifts in autonomic tone, atrial stretch, or depletion of high-energy phosphates do not contribute significantly to the phenomenon. Results of the study suggest that atrial electrical remodeling is mediated by rate-induced intracellular calcium overload.

III Inflammation and C-Reactive Protein

What about the role of inflammation and afib as indicated by elevated C-reactive protein markers? Now proven to be elevated in people with varying degrees of AF, CRP is not only a marker or risk factor indicating heart disease, but is also common in afib.(6) Which came first? The inflammation or the AF?

C-reactive protein elevation in patients with atrial arrhythmias: inflammatory mechanisms and persistence of atrial fibrillation. Circulation. 2001 Dec 11; 104(24): 2886-91.

Conclusions: CRP is elevated in AF patients. This study is the first to document elevated CRP in non-postoperative arrhythmia patients. These findings are reinforced by stepwise CRP elevation with higher AF burden. Although the cause of elevated CRP levels in AF patients remains unknown, elevated CRP may reflect an inflammatory state that promotes the persistence of AF. (6)

Ascorbate attenuates atrial pacing-induced peroxynitrite formation and electrical remodeling and decreases the incidence of postoperative atrial fibrillation. Circ Res. 2001 Sep 14; 89(6): E32-8.

(Ascorbate equates to Vitamin C – a strong anti-oxidant and anti-inflammatory agent)

We have recently demonstrated that chronic human AF is associated with increased atrial oxidative stress and peroxynitrite formation.

In chronically instrumented dogs, we found that rapid (400 min⁻¹) atrial pacing was associated with attenuation of the atrial effective refractory period (ERP). Treatment with ascorbate, an antioxidant and peroxynitrite decomposition catalyst, did not directly modify the ERP, but attenuated the pacing-induced atrial ERP shortening following 24 to 48 hours of pacing.

Biochemical studies revealed that pacing was associated with decreased tissue ascorbate levels and increased protein nitration (a biomarker of peroxynitrite formation). Oral ascorbate supplementation attenuated both of these changes. Patients receiving ascorbate had a 16.3% incidence of postoperative AF, compared with 34.9% in control subjects.

IV. OXIDATIVE STRESS AND NITRIC OXIDE/PEROXYNITRITE PRODUCTION

In an overview article written by David R. Van Wagoner, PhD, of the CCF, entitled "Molecular Basis of Atrial Fibrillation: A Dream or A Reality," (4) he indicates his published study (5) on this topic focused on the pathways related to oxidant injury and inflammation signaling.

It was found that increased protein tyrosine nitration was evident in atrial tissues from patients in permanent AF, which is highly suggestive of increased peroxynitrite formation (due to avid interaction of nitric oxide and superoxide anion). He observes that oxidative stress may underlie structural and contractile changes in the fibrillating atria. Also observed in animal pacing studies (also published)(6), pretreatment and daily supplementation of dogs with high dose ascorbate (as an antioxidant) was associated with a significant decrease in protein nitration.

He speculates that the left atrium may be preferentially sensitive to oxidative stresses, due to either lower metabolic reserves or increased wall stresses.

What is the Connection Between Oxidative Stress and AF?

(Here is peroxynitrite again. Be sure to review this section in the CCF(1) article.)

- AF is associated with calcium overload. This can increase the production of nitric oxide (NO), and mitochondrial free radical production.
- AF is associated with neurohormonal activation, frequently leading to increased production of Angiotensin II (and superoxide).
- Our research (in collaboration with colleagues at the Ohio State University) has shown that, as a result of increased NO and/or superoxide production, protein nitration is increased during persistent AF, suggesting increased peroxynitrite formation.
- This relationship suggests that, in some circumstances, antioxidants may help to prevent the cellular injury and electrical changes that normally accompany AF. Several studies have been completed to evaluate this hypothesis, and others are ongoing.

Impaired Myofibrillar Energetics and Oxidative Injury During Human Atrial Fibrillation

<http://circ.ahajournals.org/cgi/content/abstract/104/2/174>

Atrial fibrillation (AF) is associated with severe contractile dysfunction and structural and electrophysiological remodeling.

Mechanisms responsible for impaired contractility are undefined, and current therapies do not address this dysfunction. We have found that myofibrillar creatine kinase (MM-CK), an important controller of myocyte contractility, is highly sensitive to oxidative injury, and we hypothesized that increased oxidative stress and energetic impairment during AF could contribute to contractile dysfunction.

The present results provide novel evidence of oxidative damage in human AF that altered myofibrillar energetics may contribute to atrial contractile dysfunction and that protein nitration may be an important participant in this condition.

IV. MAGNESIUM DEFICIENCY

This survey would not be complete without examining the role of magnesium deficiency (MgD).

Pattern of cardiac fibrosis in rabbits periodically fed a magnesium-restricted diet and administered rare earth chloride through drinking water.

It has been postulated that causation of the tropical cardiomyopathy endomyocardial fibrosis (EMF) is linked to magnesium (Mg) deficiency and cardiac toxicity of the rare earth (chloride) element cerium (Ce).

The results suggest that in rabbits, recurrent episodes of Mg deficiency lead to myocardial fibrosis similar to the pattern observed in human EMF.

(This article can be found by title at PubMed Online)

Magnesium deficiency-related changes in lipid peroxidation and collagen metabolism in vivo in rat heart. *Int J Biochem Cell Biol.* 1997 Jan;29(1):129-34.

Magnesium deficiency is known to produce a cardiomyopathy, characterized by myocardial necrosis and fibrosis.

(This) study provides evidence of increased lipid peroxidation and net deposition of collagen in the myocardium in response to dietary deficiency of magnesium.

It is suggested that the increase in cardiac collagen synthesis and fibroplasia associated with Mg deficiency may

represent reparative fibrogenesis, upon oxidative damage to the cardiac muscle, and is mediated by a mechanism independent of changes in cardiac tissue levels of Mg.

Pro-fibrogenic effects of magnesium deficiency in the cardiovascular system. *Magnes Res.* 2002 Dec;15(3-4):307-15.

Magnesium deficiency is known to produce cardiovascular injury. A large body of experimental evidence supports the postulation that an immuno-inflammatory reaction and increased oxidative stress may damage the myocardium and vasculature in magnesium deficiency. Reparative/reactive fibrosis in response to the injury has, however, received little attention.

Recent evidence from a rodent model of acute magnesium deficiency suggests that humoral factors may activate cardiac fibroblasts by a free radical-mediated mechanism and contribute to cardiac fibrogenesis. A similar mechanism may also promote cellular hyperplasia and increased matrix synthesis in the vasculature.

Magnesium Deficiency in the Pathogenesis of Mitral Valve Prolapse (8)

This paper specifically related to Mitral Valve Prolapse (MVP) and MgD, includes important statements:

- MVP contributes to cardiac arrhythmia. Mg therapy provides relief of MVP symptoms.
- MgD hinders the mechanism by which fibroblasts degrade defective collagen, increases circulating catecholamines, predisposes to cardiac arrhythmias, thromboembolic phenomena and dysregulation of the immune and autonomic nervous systems.
- Durlach et al. considered all their patients to be Mg-deficient, based on blood Mg levels. They also suggest that complications of MVP such as cardiac arrhythmia, thromboembolism and neurasthenia may all have the same cause: Mg-D.
- Some important aspects of the MVP syndrome cannot be explained by chronic Mg-D alone: its pathology, which suggests a dyscollagenosis, the association with autoimmune thyroid disorders and the complexity of dysautonomia.
- Mg-deficient animals show enhanced adrenergic responsiveness due to lack of induced down-regulation of P-receptors MVP patients, on the other hand, are extremely sensitive to P-blockade, and may show excessive adrenergic and parasympathetic tone. Experimental Mg-D produces myocardial collagen deposition whereas MVP is associated with valvular collagen dissolution. These paradoxes might be explained by a failure of cAMP-dependent mechanisms.
- Fibroblasts continually produce defective collagen and delete it by a process which is cAMP activated]. Adenylate cyclase is Mg dependent and Mg-D is associated with defective activity of some cAMP-dependent pathways. If MVP were indeed a genetic disorder of connective tissue, its penetrance would be enhanced by any condition which impairs the ability of fibroblasts to delete defective collagen.
- Another means by which chronic Mg-D might affect cAMP activity is impairment of essential fatty acid (EFA) metabolism. Patients with normocalcemic LT show elevation of linoleic acid in plasma phospholipids and decreased levels of its desaturation products.
- PGEI is an important determinant of cellular cAMP activity. It stimulates fibroblast collagen degradation and enhances the maturation of cytotoxic/suppressor lymphocytes PGEI protects against autoimmune phenomena; its deficiency may predispose to autoimmunity and also to fibrosis.

Conclusion

Most features of the MVP syndrome can be attributed to direct physiological effects of MgD or to secondary effects produced by blockade of EFA desaturation. These include valvular collagen dissolution, ventricular hyperkinesis, cardiac arrhythmias, occasional thromboembolic phenomena. autonomic dysregulation and association with LT, pelvic fibrosis, autoimmune disease, anxiety disorders, allergy and chronic candidiasis. (8)

From the Lancet, "Extraordinary unremitting endurance exercise and permanent injury to normal heart"(9) author William J. Rowe, MD, observes:

".....magnesium ion deficiency, which can be induced by exercise, could exacerbate two vicious cycles: severe ischaemia and high catecholamines, the second would be between coronary vasospasm (induced by high catecholamines) and endothelial injury and also contribute to catecholamine-induced thrombogenesis.

In addition to ischaemia, there are several mechanisms, including the effect of free fatty acids liberated by the lipolytic effect of high catecholamines, that could cause direct myocardial injury.

Magnesium ion deficiency is a further possible complication of long exercise, some deficiency may still be present 3

months later. Exposure to heat also contributes to magnesium ion deficiency. The increase in catecholamine concentration may persist until the second day after a marathon. It is noteworthy that in a group of 20 patients with vasospastic (variant) angina showed that almost half had magnesium ion deficiency that is often unrecognized. Accompanying this paper is a flow chart describing biochemical reactions in very long endurance exercise. It is worth noting where High Shear Stress Turbulence fits in with Endothelial injury. See this chart on the last page before references. (9)

A collaboration of researchers in a paper on the Role of Magnesium in Aging, (10) concludes:

- Aging leads to a higher requirement for magnesium.
 - Cells lose magnesium mono exponentially with age.
 - One of the biological changes associated with aging is an increase in free radical formation with subsequent damage to cellular processes.
 - The consequences of stress susceptibility, defective membrane functions and perturbation of intracellular calcium metabolism, inflammation, cardiovascular diseases including atherosclerosis and ischaemia/reoxygenation injury, diabetes, fibrosis, immune dysfunction and other diseases associated with aging
- Superoxide-mediated activation of cardiac fibroblasts by serum factors in hypomagnesemia. *Free Radic Biol Med.* 2001 Oct 1;31(7):882-6.

Magnesium deficiency is known to produce myocardial fibrosis in different animal models.

The findings are consistent with the postulation that serum factors may activate cardiac fibroblasts via a superoxide-mediated mechanism and contribute to the fibrogenic response in the heart in magnesium deficiency.

Model of cardiovascular injury in magnesium deficiency. *Med Hypotheses.* 2001 Jan;56(1):110-3.

Magnesium deficiency is known to produce cardiovascular lesions. It is, however, not clear as to what constitutes magnesium deficiency - reduced serum levels, reduced tissue levels or reduced intracellular levels of the ionic form of the element. This article cites evidence in support of a hypothesis that a fall in serum magnesium levels may trigger a temporal sequence of events involving vasoconstriction, hemodynamic alterations and vascular endothelial injury to produce pro-inflammatory, pro-oxidant and pro-fibrogenic effects, resulting in initial perivascular myocardial fibrosis which, in turn, would cause myocardial damage and replacement fibrosis. Further, angiotensin II may be the prime mover of the pathogenetic cascade in magnesium deficiency. Importantly, such a mechanism of cardiovascular injury would be independent of a reduction in myocardial or vascular tissue levels of magnesium.

CONCLUSIONS

If you have followed along this fibrosis path and examined the relationships of possible contributing factors, the question remains.....What are the connections? What ties it all together?

Taking PC's summary of the Pall papers presented in the CR on the NMDA receptor, the production of nitric oxide and the resultant cytotoxic product, peroxynitrate, might we hypothesize that this production (from whatever stimulus) leads to irritation and fibrosis?

Dr. Wong tells us that Fibromyalgia and CFS share a common etiology in excess nitric oxide and peroxynitrite. He says FM pain originates because ... "Growing too much fibrin (scar tissue) on contractile (muscle) tissue binds down that tissue causing a localized ischemia.(7)

He relates that years back, "while Max Wolf, MD, Ph.D. (x7) was teaching at Fordham and researching at Columbia, he came to an interesting discovery most of us are only partially familiar with. This author of the first medical textbook on endocrinology found that old age begins at 27. This is triggered by a down turn in the body's production of proteolytic enzymes. Aside from their familiar but secondary role in digestion, proteolytic enzymes have four primary functions in mammals:

- First line of defense against inflammation. Enzymes cleave Circulating Immune Complexes it sees as being excessive in number or exogenous to the body.
- Balances the bodies repair mechanism preventing excessive fibrin from being deposited in wounds, fractures and

across joints or moving parts.

- Cleans the blood of necrotic debris and excesses of fibrin.
- Modulates immune function as an adaptogen.” (7)

Can we state there is a connection between lack of fibrolytic enzymes and the creating of fibrosis in the body – specifically in the heart and this is the cause of AF? Certainly studies are indicating that fibrosis prolongs or perpetuates it.

Is the Oxidative Stress factor significant enough of an irritant to suggest that fibrosis is the result of the body's natural defense mechanism to an injury or insult.... the laying down of fibrin.... and if one is lacking sufficient production of the proteolytic enzymes that break down excess fibrin, then does the person end up with cardiac fibrosis?

Obviously, we would like to connect the dots to these plausible, causative factors associated with atrial fibrillation.

However, this survey is just that... a collection of relevant studies and papers ...just a few out of a huge amount of resources.

What do you think? Is Erling on to something here?

I'm sure he'll be reading your opinions with interest. For now, I'm going with the lack of fibrolytic enzymes theory. I may change my mind after I read your comments.

Sorry this is so lengthy, but I tried to spare you going back and forth to the Internet with reference articles to allow the flow of this hypothesis..... you will need to read many of the featured studies in detail, however, to get the total concept.

I'm looking forward to your response.

Jackie

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Jackie,

How do you find the time?

Another well researched treatise.

IMHO fibrosis is the culprit in those that develop AF in association with cardiovascular disease, i.e., not LAF.

LAF, predominantly vagal according to Hans survey by about 2:1 over adrenergic, is a horse of another color.

I think LAF that typically develops around age 50 is genetic but would otherwise go unexpressed, if it were not for a dietary shortfall. Coumel described his first case of VMAF in 1986. It's hard for me to believe that a disease with such a dramatic presentation would have gone unnoticed for so many years, if it had previously existed.

So what's changed? Have we suddenly stopped producing adequate proteolytic enzymes? Has there been an acute increase over the last decade or two in the production of reactive oxygen species (ROS)? I think not. Endurance sports have been around for centuries. I don't think Phidipedes died of VMAF.

My money's on magnesium. However, I'm sure that there are other key nutrients that we are not getting in our fast food/processed food diet. Why is the incidence of osteoporosis highest in countries with the highest intake of calcium and the lowest intake of magnesium. This is just another manifestation of the National Academy of Sciences finding that 83% of Americans are deficient in magnesium.

If there's one thing readers of the BB and CR should take home, it's eat your fruits and vegetables. Mg and K go hand in hand.

Thank you for once again highlighting magnesium and its role in inflammation and fibrosis.

PC

I humbly agree with PC. I think this is a dead end topic for LAF'ers. I don't think it will lead to any meaningful suggestions as to how to deal with LAF in a more effective manner.

In my case, I went from no AF to frequent episodes almost overnight. Did I suddenly develop this fibrotic heart? I know I haven't. Just instinct and the issue is a waste of time for me. No offense intended.

Jon

Hi Jackie,

Thanks for bringing this topic up. Most interesting. I'll be honest, I haven't read all of this in detail. One thing that came to my attention was the part about proteolytic enzymes. I checked google and saw all the different companies which have their formulations claiming theirs to be superior, etc. I have taken from time to time digestive enzymes, but not the full regimen of proteolytic enzymes-don't know much about them-thanks for bringing this to my attention. This may possibly be the reason why I notice no effect from taking vitamins & minerals, but I take them anyway believing they must be of some benefit. Proteolytic enzymes will over time really eliminate scar tissue??? Possibly even fibrotic scars causing remodeling??

Which "brand", or company do you use when it comes to proteolytic enzymes?

Jim

PC - thanks for your input. It's winter here.... subzero temps, ice storms, etc so I find plenty of time inside for projects like this. If it were summer, it never would have happened.

Actually, I thought the connections were all interesting and I was particularly interested in the papers indicating the heart becomes fibrotic after prolonged duration of afib. I still think that could be a contributing factor.... but I, like you, feel magnesium is the key.

Once I my cells became superstaturated with magnesium, the arrhythmia breakthrough was gone. That tells me something very important. Of course I did other fine tuning and I hope to address that in another post at a later date.

And your point about a dietary shortfall is right on. Erling added fish oil to his diet which healthied up the lipid layer of cells allowing his magnesium supplements to finally get in the cells...along with other good nutrients.

I totally agree about diet, but if the phospholipid layers of cells are so damaged from the wrong dietary fat, nutrients from good foods won't enter the cells. So first, repair is in order.

I do find merit in the depleted fibrolytic enzyme theory from Dr.Wong...and the fact it is being suggested as a method "cardioreparative" in the HHD article. Enzymes could become an important topic.

However, the only enzymes I was taking when I began my "calming " protocol prior to ablation were digestive enzymes - although they did contain bromelain, papain, amalyse, peptase, etc.... and I did step up the quantity in the hopes of reducing inflammation. I had not read any of the material I've just presented at that time.

Based on this project, I have begun following his regimen for reducing the fibrotic tissue said to be responsible for Fibromyalgia since mine has revved up again after the ablation. Time will tell. I have nothing to lose but a few dollars and the fibrotic buildup.

You wrote: Endurance sports have been around for centuries. I don't think Phidipedes died of VMAF.

Interesting, but what was the average life span at that time? Today we are talking the onset about age 50? did you say? Most of those sports warriors were dead and gone by 50.

I'm happy to see the response to Erling's theory. I hope he finds time to lurk and note it as well.

Jackie

Jackie,

Thanks for the posting.

Please have a look at this product available in the UK and let me know what you think of its suitability as a proteolytic enzyme supplement.

http://www.vitalsupplements.co.uk/acatalog/Bio_Support_Joint_Care_19.html

What do you think of PC's take on this - particularly as regards VMAF in middle age as not likely being connected to fibrosis??

A quick search on google for fibrolytic enzymes shows that many animal feed formulators and manufacturers are aware of their importance..... No mention of the importance of fibrolytic enzymes in human diet anywhere though.....

Mike F.

Mike F - The serrapeptase enzyme is one that is included in the product Dr. Wong endorses called Vitalzym.

http://www.beautywalk.com/lifestyle/august03/Totality_of_Being.htm

Try going to this site and following the threads of the Dr.Wong articles.... he has several interesting commentaries about enzymes in general. He likes Vitalym because of the serrapeptase properties.

Also if you do a google on William Wong, you will come up with some websites that have audio tapes by Dr. Wong on a variety of topics that make for interesting listening.

I've decided to try Vitalzym.

The AHA - article on HHD does mention the use of fibrolytic enzymes to reduce the fibrosis factor in that condition.

Jackie

Jim - Note what I've posted to Mike F. Yes, the proper enzymes can reduce the fibrotic tissue. Dr. Wong mentions a patient with lung fibrosis who regained full breathing capacity with the use of enzymes.

Try to follow some of the threads in the directions I gave to Mike F. if you are at all interested in learning more.

These enzymes have to be enteric coated so they pass from the small intestine to the blood stream and then perform their lysing task.

I've decided to try the Vitalzym that Dr. Wong likes I ordered mine from www.iherb.com

In one of his sites, he has a dosing schedule for the condition and how much to take. If you are interested, email me and I'll send you the web sites.

If you have some spare time, listening to his audio tapes online is very enlightening..... also, note his credentials. He seems to be very well respected in the field of sports medicine.

Jackie

Jackie and Erling,

I found this post very thought provoking, and am in complete agreeance with the importance of enzymes. For anyone who would like to read more about enzymes, you can go here:

<http://www.enzymestuff.com>

As for fibrosis of the heart, I'm still pondering that, as well as, polymorphisms. Maybe I'm in denial. I just find it very strange, that I only have episodes when ingesting triggers, such as MSG. I am on flecainide, however, and maybe that masks the fibrosis or polymorphism. My situation is a bit different in that I have flutter, but why did betas cause AF for me? Did they just exaggerate what was already lurking. I was a classic case of VMAF when taking them. Also, in regards to polymorphisms, I can see where Lynn would have one, because she is in chronic AF, but I can't see where that would come into play if AF/flutter was intermittent. Am I not understanding something?

Also, why does Mg not set too well with me? I've switched to Mg. taurate, but am only taking 1 cap per day, and that keeps me pretty regular. If I take more, then the floodgates open. It's not that I haven't worked up to it, because I've been taking it now for about 5 mths. (1 tab of Glycinate before). For me, Mg may have not been an issue, and my intracellular levels indicated that.

I've started taking lithium orotate (5 mg elemental) daily, and haven't noticed any adverse or good effects so far, and will up it, eventually, to 15 mg. per day.

Anyway, thank you Jackie and Erling for an excellent post. I plan on re-reading it again, and doing some more studying. Just one more comment. There sure does seem to be a lot of diseases out there that all relate to the NMDA receptors. A LOT!!

Richard

Richard - Thanks for your response. I agree that undoubtedly many afibbers find a connection in the NMDA receptor issue which is most likely connected to the polymorphisms, gene expression, etc. and that damage.

However, after reading the studies on fibrosis as a result of long-standing events of fibrillation, I am still intrigued by the plausibility of that phenomonea.

A dietary deficiency could be the enabler of the events at the onset, as could the oversensitivity to excitotoxins, etc... but I keep wondering that unless the heart gets calm enough for long enough, might not the fibrotic tissue enable the "circuits" to continue the afib habit or pattern.

This would seem reasonable to me sense, in time, and without nutritional intervention, afib seems to become progressively worse.

Just musing out loud.

I believe I read in one study, acknowledgement that there is no "one-size fits all" solution to treating afib. Everyone seems to be so different.

Undoubtedly, this is why we are all here looking at all the possibilities.

But..... if you go to that picture of a fibrotic heart..... one cannot deny the fibrosis factor. I think that's very graphic.

Best regards,

Jackie

I found this very thought provoking and also think it is a probable cause but have not read the studies in detail yet. It also supports in part (without me thinking about it) my own observations and thoughts on AF. Erling and Jackie you both make wonderful researchers.

I also agree with PC about his thoughts on the role of processed foods. PC also said "So what's changed? Have we suddenly stopped producing adequate proteolytic enzymes? Has there been an acute increase over the last decade or two in the production of reactive oxygen species (ROS)? I think not. Endurance sports have been around for centuries."
"

I agree with this - but it is only recently that endurance sportsmen have been eating and drinking the food we have to day - especially these isotonic drinks full of aspartame and energy bars full of hidden MSG!! As someone who got no benefit from Mg alone think it goes much deeper - especially when you realise the effect of excess free glutamate and nitric oxide - which mg didn't seem to touch for me and come to think of it neither did glutamate blocking drugs.

I have a question to PC. Why do you think that fibrosis in the heart has to manifest itself as heart disease. Could it not be something slightly different - such as in fibromyalgia - which does not show as a different tissue structure - but certainly causes damage and pain. Could it not just be a variant symptom shown up as AF in those of us who are susceptible, I begin to think that so many diseases are caused by the same thing but manifest themselves differently depending where the weak spot is. I mean even Mg deficiency can show up with different symptoms in different

people.

Unfortunately I was not someone who started aging at 27. It must have happened much younger in me (why do I look younger than I am?). AF started when I was 22. But I did work and play to the extreme. No matter what I was doing I would push myself to the limit of my abilities, be it swimming, gymnastics, ballet, research, problem solving, essay writing, or drinking someone under the table. You could say that as a teenager I burnt the candle at both ends. So should I have been surprised. Absolutely no.

So if the theory is correct and I did age myself prematurely due to diminishing proteolytic enzymes then if the enzymes were replaced then in theory I should get better - slowly. Well I did. By eating no more processed food, eating loads of good quality proteins - which have the ability to repair tissue and loads of antioxidants etc in the form of veggies I have hit a winner (I hope). I also learnt how to not take things to the extreme and not push myself. IF AF was due to a build up of something akin to fibro through oxidative stress in the heart or pulmonary veins then I must have reversed it, but then I am reminded that I still can't eat free glutamate - so maybe it is not scarring but back to the plain old NMDA receptor. I am reminded of Richards scar which disappeared when he took some enzyme but he still has AF or flutter. I hope this continues so we can understand more.

Fran

To clarify a little of what I've already posted.

VMAF and AF secondary to measureable cardiovascular disease appear to me to be two ends of the AF spectrum. Adrenergic LAF may be somewhere in the middle.

On the one hand, some forms of ALAF may also be genetic. However, on the other hand, as Lam points out in his book, the ability of modern medicine to measure what's happening biochemically at the cellular level is crude. No doubt we all are undergoing subtle cellular deterioration that eludes detection. Some ALAFers probably fall into this camp. God knows they're mostly over 27.

There must also be a spectrum within VMAF. The more frequent and longer the episodes the more likely there is to be some early fibrosis. And then it becomes an AF begets AF situation, as Jackie has indicated.

Whether inflammation causes AF or the reverse is the chicken v. the egg question. Sure there's oxidative damage to the cell membranes that might result in cell death and slight elevation of CRP. The efficacy of ablation around the PV ostia, the location of the first heart cells to be exposed to ROS from the lungs is persuasive for this argument. But AF itself causes an increase in intracellular calcium, which is cytotoxic and can do the same thing. Furthermore, ablation efficacy may be also related to the denervation of vagal nerve fibers that enter the heart from the lungs around the PV ostia, prolonging refractory period.

And Fran and Jackie absolutely correct. People who are magnesium deficient manifest such differently - migraines, constipation, insomnia, toxemia of pregnancy, arrhythmias, muscle cramps, msg sensitivity, etc. And if we are Mg deficient because of the modern diet, what other vital ingredients are also missing in sufficient quantities - B vitamins, C, E, fish oils, etc.

Most importantly, this dietary shortfall (AFer or LAFer) is a wakeup call.

PC

It's hard for me to swallow (pardon the pun!) the use of oral fibrolytic protein supplements to "rebuild" cellular fibrolytic enzymes. First of all, these are proteins, and will be treated like proteins in the digestive system -- i.e., they will be degraded to their amino acids (even enterically-coated, because most of this degradation takes place in the small intestine anyway). And second, even if they did survive the proteolytic enzymes of the stomach and intestine, they can't be absorbed from the intestine to the blood stream. Only the remnants of proteins (amino acids and very small peptides -- combinations of amino acids) can be absorbed through the intestinal cells.

Dick

Jackie,

Great treatise with lots of thought-provoking stuff. I must admit though that I am a little confused about the enzymes. Are fibrolytic enzymes just a souped up version of standard proteolytic enzymes (proteolytic enzymes + serrapeptase)?

In any case I firmly believe that fibrosis of the myocardium is a dynamic process at any age. In other words, once the causative factor (oxidative stress, inflammation, aldosterone/cortisol excess, etc) is brought under control the myocardium will shed the fibrosis and regenerate itself - with or without fibrolytic enzymes.

Hans

Hans - thanks for your comments....

It is certainly thought provoking and the most provocative article, to me, was the AHA piece on cardioreparative strategies in the terms of fibrosis and hypertensive heart disease.

The fact they are indicating the use of enzymatic interventions gave validity to my fibrosis search, in general.

While we are concerned with the heart, and I do hope you are correct that once influencing factors are eliminated, the heart repairs itself, fact remains that fibrosis elsewhere is not resolved by a natural mechanism... apparently.

One of the many, many articles I read indicated during autopsy and while looking for heart specimens, it was difficult to find a heart that was not either fibrotic or rock-hard from calcification. That's a scary thought.

Included in that generalization was the fact that brains, hearts, and other organs all become smaller with age due shrinkage and fibrotic overgrowth. Smaller organs/ dysfunctional systems/ aging and death. Sounds reasonable.

If enzymes will prevent the fibrotic takeover of organs, then perhaps it is worth considering.... even if our hearts are able to overcome fibrosis.

I'm glad to have had the opportunity to be forced to investigate a topic I probably would not have looked at otherwise. Thanks Erling!

Jackie

Hi again Jackie & Folks,

Well, I've been reading about Vitalzym-the capsule that seems to be quite the rage on the internet-proteolytic enzymes touted by Dr. Wong (who is he anyway?). It all seems pretty reasonable & good, but what do I know? Hey, I was big into fiber optic tech stocks in 2000. What a fun year!! Anyway, a 100 bucks for 450 capsules. I bought some, why not, certainly is a heck of a lot cheaper than those tech stocks were. And, who is to say it won't work. Not good for those on coumadin (like me), but since my INR is usually low, I'm going to try it. Just maybe all those vitamins & minerals I am taking will start working better & I just might be one of those success stories PC is looking for. But then again, what is that line about a sucker being born every minute? Stay tuned, we'll see.....

Jim

Jim - we can compare notes on Vitalzym results. You are correct - much less expensive than a flyer on Wall Street. May those days never darken my portfolio again!!!!

The interesting side benefit I expect to gain in addition to eliminating fibrosis in muscle tissue is the increase in immunity. Vitalzym is said to prevent viral infection because it does denature the tough viral coating and prevent replication.

I'm keeping my mind open on this one.

Jackie

Dr. Wong -who is he anyway?

Dr. William Wong is a Texas State Naturopathic Medical Association professional member, World Sports Medicine Hall of Fame member, a Classical Naturopath, a Ph.D. Exercise Physiologist, a Certified Athletic Trainer (AATA), a Certified Sports Medicine Trainer (ASMA), and a Health/Fitness Consultant.

Dr. Wong has more than 23 years of professional experience in natural health, as applied to sports medicine and rehabilitation, with the last 12 devoted almost exclusively to chronic fatigue and fibromyalgia, Dr. Wong has authored books on natural healing and has taught Physical Medicine at the South West College of Naturopathic Medicine in Arizona.

His shorter writings have appeared in such diverse magazines as G.Q., Black Belt, Survival Guide, The Townsend Letter for Doctors, and Well Being Journal. In 1993, he was also inducted into the Martial Arts Hall of Fame as Wing Chun Kung Fu Instructor of the Year.

Dr. Wong has been a guest on over 500 national and local radio programs, as well as appearing on the nationally acclaimed PBS series Healthy Living hosted by Jane Seymour.

In November 2002, Dr. Wong appeared on the Heartbeat of America show hosted by William Shatner. Currently, using a blend of movement, nutrition, exercise and spirituality, Dr. Wong is specializing in developing programs for longevity and virility to help people overcome the effects of aging and the after effects of chronic debilitating conditions.

Dr. William Wong and his wife Michele are devoted to bringing forth information on new and effective natural treatments for chronic illnesses, teaching little known information about exercise and spreading their philosophy on what they've found to be the cornerstones of a healthy and active life.

Physical Education and Sports Medicine, S. Brooklyn College. The Sports Medicine program was headed by the late Prof. William Chisolm. 1978

Classical Naturopath. (NOT a Naturopathic Physician as they also use drugs and surgery, while Classical Naturopaths cling to the founding principles of the art).

Classical Naturopaths and Board Certified Naturopaths belong to the ANMA, while Naturopathic Physicians belong to the AANP.

N.D. 1981 (Doctorate of Naturopathic Medicine) Brantridge Forest, Sussex, England; US program at Long Island University, Brooklyn Campus, tutored by Dr. Charles W. Turner DC, ND, DO, D.Sc. (Brantridge closed on the death of the director Dr. Bruce Copen in 1986. Dr. Turner died in 1983). 1

Exercise Physiologist Ph. D. In Martial Arts for the dissertation "The Application of Biomechanics and Motor Learning to the Modern Teaching of the Martial Arts" presented to the Soke (Grand Masters) Council, Shorinji Temple School, Hokkaido Japan through the US branch of the school at Cleveland, Ohio. Also given the title of Professor and the rank of Hanshi (9th Degree). 1994

Teacher of Physical Medicine, Southwest College of Naturopathic Medicine, Tempe, AZ. Taught: Introduction to Physical Medicine, Sports Medicine and Therapeutic Modalities. 1994 -1997

World Sports Medicine Hall of Fame Inductee 1993

World Martial Arts Hall of Fame Inductee (Wing Chun Kung Fu Instructor of the Year). Dr. Wong was the 25th disciple of the Wing Chun Grand Master Moy Yat. 1993

ASMA/AATA Arcadia, CA. Certified Sports Medicine Trainer 1995

AATA Arcadia, CA. Certified Athletic Trainer 1978

Professional Member Texas State Naturopathic Medical Association. (Governing body of Naturopathic Medicine for the State of Texas). 2002

AATA Athletic Trainer of the Year 1992

Brooklyn College Sports Medicine Alumni Award 1986

Author 10 Natural Treatments You Haven't Heard of Until Now 2000

The Best Natural Sports Medicine Book Ever 2003

Jackie,

Golly, Golly, Golly.....what credentials. Everyone should read this. I'm hooked....so we will see & compare notes. Since I am still taking coumadin, I will start off slower checking my INR levels. Assuming I have no more episodes (nothing since that last bigeminy on the 23rd of Jan) I'm taking myself off in another month anyway. However AF being the way it can be-after an ablation if not successful-I will not be surprised if something happens again around 20-24th of this month. We'll see. Keeping my fingers crossed.

Thanks for this post. What a wealth of information you are!!

Jim

Jim - I secured an audio tape of one of Dr. Wong's presentations.... he is an interesting man. Very likeable and very "into" his subject. What I like is the sports medicine angle. He can't be too far wrong and remain successful in giving that type of advice and treatment. Interesting as well that he had fibromyalgia so he understands the condition, the pain and the remedy.

Well, I can't take credit for finding Dr. Wong. Erling found him and became fascinated with the potential of using systemic enzymes.

But I will say, for me, it's opened up a whole new consideration for treatment - and not just afib.

The fact that "real" medical doctors have researched all this and used it in the past lends even more credibility to the potentials.

I'm about 5 days into taking Vitalzyme. Hans says he's interested in the "field trials" so keep a log, Jim, and we can compare notes.

Jackie

Jackie,

Thanks for the info. It appears that I can't readily get Vitalzym here in the UK (Only the serrapeptase I mentioned previously). In any event, I notice on some US sites that 'Naticor' is now taking over from Vitalzym. Do a google on

Naticor and let me know if you concur with its producers as to its superiority over Vitalzym.

Mike F.

Hi Mike F - I finally got time to check out the web page and it does appear that this product is the same as one of the ingredients in Vitalzym.

They list all the uses - sports injury, inflammation, etc. And yes, it is the silk worm enzyme. So, if that's all you can get in the UK, it would undoubtedly be helpful. I think the Vitalzyme relies on at least four other enzymes which you may be able to get in another product.

Wobenzyme is a German product and was the one Dr. Wong originally used for his treatment...but he likes the serrapeptase alot. If you can get both Wobenzyme and also this one.... I'd say you'd have a super enzyme package.

If you go to <http://www.iherb.com> - and type in both Vitalzym and Wobenzyme, and click on the hyperlink once you have it...you can see the label comparisons of each and the quantities of each component. That may be of assistance to you.

Let me know if I can help more.

Jackie

Actually Fran, I had not attributed the scar disappearance to enzymes, but I was taking them then. Hmm?? I had figured it was due to the amino acids I was taking. At that time, I was taking proline, taurine, lysine (which was what I thought did it), alpha lipoic acid, and n-acetyl-carnitine. Enzymes, however, are made from amino acids, but then that's a "catch 22", because if one is low in enzymes, then the aminos can't be made into the enzymes. My indigestion, followed by GERD, was the first indicator, and symptom, for lack of enzymes. My other symptom that I had alongside indigestion were headaches, which worsened over the years, just as my indigestion did. This tells me two things. MSG and lack of enzymes. Over time, I insulted my body further, by reducing stomach acid, hence making my pancreas work even harder.

No matter what has been the end results of years of insults, we now know several important things.

- 1) We are a Mg deficient society.
- 2) We must avoid MSG at all cost, healthy or not.
- 3) We must eat lots of fruits and vegetables, with protein.
- 4) We would probably be served well, by taking enzymes, since we are still eating a lot of our foods cooked. From what Bastyr's told me, the pancreas will reabsorb these enzymes for reuse.
- 5) We should try to eliminate as many chemical and pesticide exposures, as we possibly can.
- 6) If one cannot eliminate wheat or dairy, then extra doses of enzymes for help in these areas would be beneficial.
- 7) And for me, I will always continue to take sulfur aminos, because the body cannot keep up with the assaults our society now faces. These assaults were not meant to be in the bigger scheme of things.

When I step back and think about it, enzymes are vital for every function of our body, and without the first line of enzymes being there to break down the food components for absorption, then how could the rest of the body function or absorb the nutrients without them.

Richard

Dick - Your observation is apparently a common one since Dr. Wong addresses exactly your argument in his text and audio tapes. The controversy has been around for a long time, but the results from Europe where systemic enzymes have been used successfully in this manner for over 30 years are being used here successfully in sports medicine.

In one section, Dr. Wong points out that we agree that Salmonella gets into the blood stream and I think he says the size of Salmonella is 5 times greater than the enzyme...so why wouldn't the enzyme pass through.

There is a lot of use and research news on these systemic enzymes - not digestive enzymes - that bears review before coming to conclusion....

Personally, I think the enzymes have merit. After the ablation, my fibromyalgia flared up again and I'm going to try it to see if I get relief. If it works, I'll be pleased.

And even if it doesn't, there are still the studies pointing to afib and fibrosis.

Something needs to stop the fibrosis....and it isn't in the just heart...it's the brain and all other organs. Personally, I'm going to be thrilled if I find something that will stop the progression of decreasing organ mass due to aging and fibrosis.

I think it is a fascinating topic. And I'm interested in the input everyone is providing.

Jackie

Jackie,

The topic of absorption of complex molecules (whether or not it can happen) is frequently encountered in alternative medicine.

In medical school we were all taught that digestion and absorption is a two-step process, as Dick described. It seems intellectually untenable that something as fragile as an enzyme could survive such a process as digestion. Not only must the molecule remain intact but its three dimensional configuration must as well. It's not a size thing, although that's part of it (more linkages that are exposed to the cleaving enzymes). Salmonella and most infectious bacteria have a very tough capsule that can withstand digestion and a lot worse.

However, the proof of the pudding is in the eating and I will be all eyes when it comes to reading proteolytic enzyme therapy success stories.

PC

This is rather depressing---at the age of 27 our bodies begin to build fibrotic tissue, declining fibrolytic enzymes--I find it amazing that there are people that live into their nineties without afib.

A question about CRP---if the levels are higher in AF patients, after a PV ablation does the CRP drop?

My last CRP test was 0.7--lab value <0.8 MG/DL, so I was okay at that time, I suppose it could be lower.

Liz

Liz - Yes, depressing to think at such an early age, our bodies could begin aging. But, in fact that's exactly what happens.

I've just done a mental survey of what was going on in my life -healthwise - around age 25..... First, I was developing fibroids of the uterus about that time. Hemorrhagic monthly bleeding. By age 38, I developed severe fibrocystic breast disease. The Fibromyalgia came much later.... 58.... but the first afib event surfaced at age 59.

Logically, for me, I can entertain the notion that I was building fibrotic tissue and did not have the fibrolytic enzymes to break it down.

About your CRP question.... I'll be having that test done soon. Let you know. I know they did a baseline at the CCF at ablation time and it was .9 if I recall correctly. Way too high. They want it as close to zero as possible. I've previously reported that my CRP's continued to rise every time I was tested...they started out at an acceptable level... like .6 - think and were over 1 by the time I enrolled for ablation.

CRP indicates inflammation. A healthy body should not be showing signs of inflammation.

Now with all my antiinflammatory protocols, and no afib for five months duration, I'm anxious learn if it has normalized.

Jackie

PC - I understand what you are saying. While I didn't go to medical school, I do have a health-science education and I do understand what was taught and is still taught. The controversy on this topic abounds.

I just re-read an article by Michael T. Murray, ND who is head of research for Natural Factors (he left Enzymatic Therapies). Dr. Murray is very well known and respected in the alternative medicine field...written many books, etc. I've heard his seminars and talked personally to him about fibromyalgia. I tend to believe what he writes more often than just some obscure name I just happened across from a google search.

<http://www.doctormurray.com/articles/Penzymes.htm>

Because I'm fascinated with the proteolytic enzyme possibilities for many reasons, I'm delving into it further. Another of Dr. Wong's audio tapes online mentioned that the enzymes had been "tracked" through a radioactive coating or procedure assay method and was indeed found to enter the blood stream. Does that make sense? Sounds dangerous...he didn't say if it was human or animal studies.

Anyway, I'm the human guinea pig. I've begun taking Vitalzym the proteolytic enzyme containing the Serrapeptase and I'll be reporting when I have a transformation. I'm a good test case because right now I'm having a significant problem with one area affected by FM....if that diminishes, it may be that the enzymes are doing what is claimed.

Certainly, they aren't harmful.... as I said...maybe just a waste of \$.

Jackie

Hello All,

In doing some searching on proteolytic enzymes, I ran across this article that I found extremely interesting, esp as it pertains to the ANS. Here's some excerpts, but pls. read the article in its entirety. Dr. Gonzalez has also been granted funding for clinical trials through the alternative branch of the govt. to test his regimen on pancreatic cancer.

It is clear from our extensive clinical experience that pancreatic proteolytic enzymes have a profound anti-neoplastic effect, but we do not know how they work. We have not had the resources to support basic science research, but with appropriate funding we do not believe it would difficult to set up animal models to explore the molecular action of the enzymes against cancer cells.

We have found that specific nutrients and foods have specific, precise and predictable effects on the autonomic nervous system. For example, a vegetarian diet emphasizes fresh fruits and vegetables, particularly leafy greens, and contains large doses of minerals such as magnesium and potassium. It has been shown in many studies that magnesium suppresses sympathetic function, while potassium stimulates parasympathetic activity. Furthermore, a largely vegetarian diet tends to be very alkalinizing, and the neurophysiologic research documents that in an alkalinizing environment, sympathetic activity is reduced and parasympathetic activity increased. So, whatever other effect a vegetarian diet has, in terms of autonomic nervous system function, such a diet will reduce sympathetic activity and stimulate the parasympathetic system.

A meat diet is loaded with minerals such as phosphorous and zinc, which tend to have the opposite effect. A high-meat diet stimulates the sympathetic system and tones down parasympathetic activity. Furthermore, such a diet is loaded with sulfates and phosphates that in the body are quickly converted into free acid, that in turn stimulates the sympathetic nervous system while suppressing parasympathetic activity.

NJG: All of our patients, whether they have cancer or some other problem, consume specific combinations of vitamins, minerals, trace elements, amino and fatty acids, and animal-derived glandular and organ concentrates. We use such supplements very specifically, in very precise doses and combinations as we use diet, to manipulate autonomic function and to bring about balance to an imbalanced system. Certain vitamins, minerals and trace elements, such as many of the B vitamins and, as mentioned above, magnesium and potassium, tone down the sympathetic nervous system and stimulate the parasympathetic nerves. Other nutrients, particularly calcium, phosphorous and zinc, stimulate the sympathetic system but weaken the parasympathetic system. By the use of precise combinations of vitamins, minerals and trace elements, along with diet, we are able to bring about balance to the autonomic system. And, again, when the autonomic branches come into balance, the patients, whatever the underlying disease, do better.

http://www.dr-gonzalez.com/clinical_pearls_txt.htm

There are some links at the bottom of this article for further reading. This article even further explains why I don't do well on Mg. I need to build up my sympathetic side, hence why I felt so much better on my protein based diet.

Evaluation of pancreatic proteolytic enzyme treatment of adenocarcinoma of the pancreas, with nutrition and detoxification support.

Gonzalez NJ, Isaacs LL.

Historically, large doses of proteolytic enzymes, along with diet, nutritional supplements, and "detoxification" procedures, have been used in alternative therapies to treat all forms of cancer, without formal clinical studies to support their use. A 2-year, unblinded, 1-treatment arm, 10-patient, pilot prospective case study was used to assess survival in patients suffering inoperable stage II-IV pancreatic adenocarcinoma treated with large doses of orally ingested pancreatic enzymes, nutritional supplements, "detoxification" procedures, and an organic diet. From January 1993 to April 1996 in the authors' private practice, 10 patients with inoperable, biopsy-proven pancreatic adenocarcinoma were entered into the trial. After one patient dropped out, an 11th patient was added to the study (however, all 11 are considered in the data tabulation). Patients followed the treatment at home, under the supervision of the authors. As of 12 January 1999, of 11 patients entered into the study, 9 (81%) survived one year, 5 (45%) survived two years, and at this time, 4 have survived three years. Two patients are alive and doing well: one at three years and the other at four years. These results are far above the 25% survival at one year and 10% survival at two years for all stages of pancreatic adenocarcinoma reported in the National Cancer Data Base from 1995. This pilot study suggests that an aggressive nutritional therapy with large doses of pancreatic enzymes led to significantly increased survival over what would normally be expected for patients with inoperable pancreatic adenocarcinoma. http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=10368805&dopt=Abstract

Here's link for further info on enzymes:

<http://www.doctormurray.com/articles/Penzymes.htm>

Richard

In case you all don't read the above link, I failed to post this excerpt:

Dr. Beard believed enzymes had to be injected to prevent destruction by hydrochloric acid in the stomach. However, recent evidence demonstrates that orally ingested pancreatic proteolytic enzymes are acid stable, pass intact into the small intestine and are absorbed through the intestinal mucosa into the blood stream as part of an enteropancreatic recycling process.

Richard

Dear Richard,

Regarding the information from Dr. Gonzalez about diet and supplements effecting the autonomic nervous system that you posted on 2/10 , does this mean that the first plan that is heavily vegetarian with magnesium and potassium be contraindicated for vagal afibbers and beneficial for adrenergic afibbers?

And the second plan, a protein diet, is better for a vagal afibber or do I have this turned around?

Carol

Carol,

You have it turned around. The protein diet stimulates the symp. side, which would be good for vagals and the vegetarian diet stimulates the parasymp. side, which would be good for adrenergics, unless I'm missing something here. It also would seem that Mg and K are used to stimulate the vagal side, so the supplement issue is contradictory to what we have been learning. I'm still very confused about all this. Hans says vagals should not take beta blockers, and I understand this, because this blocks adrenalin, therefore suppresses the sympathetic side. I can attest to this, because betas at higher doses were devastating for me. Also, the protein diet was really good for me, as well, because it relieved my 20+ yrs of GERD, and made me feel so much better, which apparently was stimulating my symp side. On the other hand, I can hardly tolerate more than 1 tab. of Mg, of which I've been taking for 5 mths. now and I can't so beyond that. My intracellular levels of Mg were slightly above ref range and my K was normal inside the cell, as well. Fran mentioned in a post in the last conf. room topic, that she did not believe that Ca was the problem, but more that MSG/free glutamate was the problem that causes Ca to influx into the cell. I have to agree with her, as it pertains to me. We are all so different, however. You had good results for awhile on Mg, but went back to AF, yet Jackie had continued good results, but she was also on flec. that could have been masking the AF altogether. PC did not have continued good results with Mg either, and when he was doing MG IV's, that actually put him into AF. Sometimes I believe we cannot see the forest for the trees. There is no doubt that I go into flutter after MSG, and others have had the same problem. I do believe that Mg is an essential mineral that we could have been lacking in to some degree, but I think we may be overemphasizing it for vagally dominated people. Pls. someone correct me if I'm wrong, as I feel like I'm the only one that is seeing this or I'm missing something. I feel we need to be building up our failing sympathetic side.

Another question I have is this. If one were vagally dominant, and the parasympathetic side is what controls digestion, then why would one have contents that sit in the stomach for such long periods creating gas buildup and GERD. I would think that the gastric emptying would occur much faster, due to overstimulation of the parasymp. side. I'm not sure how the symp side fits into this scenario, however. I guess I always looked at GERD as a problem with histadine, or taurine and bile.

I really do believe Fran is right about a lot of this, and she's one of the lucky ones to be AF free. Even though some of my supps. have Ca in them, I tested more Ca and Phosphorous last night with no problems. I'll continue to do so, and see what happens, with strict avoidance of MSG. Also, if PC's reading this, remember that there was the possibility of the phosphorous in Coke converting us. Phosphorous converted me on several occasions; in Coke and in a doctor prescribed drink. My Pho levels were a bit low

Here's the excerpt about supplementation again for refreshment of memory.

We use such supplements very specifically, in very precise doses and combinations as we use diet, to manipulate autonomic function and to bring about balance to an imbalanced system. Certain vitamins, minerals and trace elements, such as many of the B vitamins and, as mentioned above, magnesium and potassium, tone down the sympathetic nervous system and stimulate the parasympathetic nerves. Other nutrients, particularly calcium, phosphorous and zinc, stimulate the sympathetic system but weaken the parasympathetic system.

So could someone help me see what I may be missing here. Maybe I can't see the forest for the trees, because I'm brain locked.

Richard

Richard - I can't "argue" the biochemistry of this, but everything I read says that Mg and K are relaxing... rather than stimulating as is calcium and sodium.

I don't doubt your experiences. While I was reading the research studies for this piece, there was a tremendous amount of reference to calcium overload as the culprit.

In simplistic terms - Dr. Wong said the reason for arrhythmia is that too much calcium floods the space that should be reserved for magnesium and so instead of getting a contract/relax response, one gets a contract/contract response and hence the arrhythmia.

I'd like to know the mechanism whereby the free glutamate allows calcium into the cells.....does it imbalance magnesium first? This could be the whole key right here. Have you run across a biochemical explanation for this?

Additionally, food that sits too long in the stomach and creates GERD is mainly traced to insufficient stomach acid and enzymes to break down food in a timely manner.... if it lies there long enough, it will create gas and stomach distension which will then affect the vagus nerve.

About the phosphorous in Coke.... might it just be the sugar content? Over the years when nothing else worked on self-converting, lots of times I'd just give up and eat something I didn't normally eat - like a cookie or something sweet... and within 20 minutes, I'd convert. This happened over and over... but it wasn't a consistent remedy. Just when the afib had gone a long time - over 20 hours or so. I always felt it had to do with satisfying the need for quick glucose.

Then again... might the phosphoric acid lower a pH problem that was existing at the time of afib. Lots of mysteries yet unsolved.

Thanks for using up your brain power.

Jackie

Jackie,

I'll see if I can find the biochemistry behind the glutamate issue. It's quite a complicated process.

As for converting with phosphorous, I also converted a few times on the Pho drink prescribed by my EP. It didn't work everytime, however. Coke worked 3 or 4x's. When first converting with Coke, I believed it was the caffeine, but later believed that it was possibly the phosphoric acid, hence why the doctor prescribed the Pho drink, after taking a closer look at my charts.

As for the enzymes and digestion, I completely agree that enzymes are a real issue here, but they only helped some when quitting Prevacid. It was when I changed to a high protein and salad/vegetable diet, that all GERD completely disappeared on the second day. Somehow, the combination of taking enzymes and eating protein turned my situation around. My wife worried that I was eating too much protein, but the good feelings that I got from this diet, told me I was doing the right thing. Somehow, eliminating grains and dairy played into this scenario, however, and I had already eliminated sugar and bad fats several weeks before.

The body tightly regulates Ca within the blood. If there's not enough, then it robs it from the bones, and I would suppose if there was too much then it would use the excess to rebuild the bones or excrete it. Every test I took while in the hospital on numerous occasions, indicated my Ca levels were either normal, or a bit to the low normal side. They never indicated being high or high normal. I do believe the real issue here is not Ca overload, but the cells being made to allow Ca entry by a neurotransmitter faultiness, i.e. glutamate. The best way, to my knowledge, of knowing the status of Ca within the body is to do a bone scan. Several here are suffering from osteoporosis, so that would indicate

a shortfall of Ca.

Jackie, pls. don't get me wrong, as I feel Mg is very important, and may be critical for many here. The food tables indicate the importance of Mg, as well, with an average 1:1 ratio in foods that we should be eating, with eggs being about 5:1. We should also realize and take into consideration the abundance of osteoporosis in this nation, even though we are high dairy consumers. Pasteurizing the milk could also release free glutamate, causing a double-edged sword, but also know it destroys the enzymes. In Angus' post on being AF free for two years, since elimination of dairy, could be that the opioids in dairy were his problem, which have an effect on the NMDA receptors. But then again, it could have been Ca overload. I would like to know the biochemistry behind what regulates Ca homeostasis in the body.

For me, I cannot dispute the fact that overload of glutamate causes Ca to influx into the cell, causing me arrhythmia, and my levels of Mg were normal. Paint fumes put me out of rhythm, as well, and also had an effect on Lynn. Has anyone else here had an intracellular test, indicating their levels of Mg? I would really like to know what those results were. And David S., if you're out there, I know you have had good success with Mg and K, but you also have had reactions to MSG. Can you comment?

I'll get the biochemistry info for you Jackie, and I'll search and see what I find on Ca regulations in the body. I'm taking more enzymes now, which include Metagest from Metagenics w/Betaine HCl, Pepsin, and herbal bitters, Azeo-Pangen Extra Strength from Metagenics from porcine pancreatic concentrate, and Digestzyme from Transformation w/ all the -ases. I'm going to order what you recommended, as well. I certainly believe that enzymes are very important and what many are lacking.

Richard

Richard,

I tested low on mag:

1. Bottom of range red blood cell mag;
2. Mildly deficient in urine retention test (24hr mag (IV) loading).

None-the-less, mag ALWAYS (glycinate and taurate) gives me increased ectopy. I'm currently trying Concen-Trace mineral drops (188mg colloidal mag per 30 drops) - I go for 60-80 drops per day just to make sure I'm at least getting RDA of mag that way. After 1 week of this? No probs so far.

Mike F.

Hi Richard - Oh - I don't misunderstand about magnesium and your needs or levels... on the contrary..... I'm just musing along here and wondering what the exact reaction is that selectively will allow calcium to flood the cells.

I don't doubt for a minute that you, Lynn, Fran and others find that it is the free glutamate that causes afib. Fran's proved it, Lynn's on her way...and your experience certainly supports it as well.

I'll be interested if you learn of a system that regulates calcium other than the buffering system that relies mainly on calcium to lower pH for kidney health and function. That's the main reason for osteoporosis... a high acid-ash forming diet.

I agree pasteurized milk is a bad actor on many levels.

Excess calcium (free calcium) is deposited in soft tissue... as in atherosclerotic plaque and heart tissue. It helps form the matrix....it also makes kidney stones and bone spurs. The chemistry for getting calcium into the bone matrix heavily on the other minerals such as magnesium, boron, strontium...and some trace others that escape me as I type. Calcium alone won't do it.

Hence there the osteoporosis epidemic.... people are taking supplemental calcium and drinking milk and it isn't the right chemistry. Plus the high consumption of cola beverages with phosphoric acid uses up the Ca stores.

You know the story about countries who get no dairy and have no osteoporosis because the calcium and magnesium etc are supplied from the plants... but their ground isn't over-farmed or over managed with commercial fertilizers as ours is here.

Since you asked the question, I had my intracellular magnesium checked and it was in the normal range, but I was still magnesium deficient.... since my experiment of flooding the cells got me out of breakthrough on Flecainide when nothing else did. The previous 4 years on Flecainide did not keep me out of arrhythmia.

Thanks for your input.... I'll be very interested to learn more about the biochemistry of the free glutamate receptors and the control of calcium. I think this is a very crucial issue for many afibbers.

Jackie

Jackie

This is what I have been trying to argue for ages. It is the mechanism of free glutamate that allows calcium influx (and sodium) and hence excitability and AF etc etc. To stop it means avoiding free glutamate.....

To understand the mechanism go to this site for a diagram.

http://www.memantine.com/en/studies/preclinical_data/signal-to-noise_hypothesis/

NMDA receptors are transiently activated by mM concentrations of glutamate (Clements et al., 1992). In order to prevent excessive influx, the ion channel is blocked by a magnesium under resting conditions. If the NMDA receptor is activated by glutamate and the postsynaptic neuron is depolarized at the same time, magnesium leaves the ion channel and calcium can flow in, which is important for learning processes.

And here is my old favourite again. Be sure to read it in its entirety.

<http://smart-drugs.net/ias-excitotoxins.htm>

Looking at the NMDA receptor diagram it shows that there are receptor sites for chemicals other than glutamate. The zinc site can be occupied by the zinc ion, and this will block the opening of the ion channel. The PCP site can be occupied by the drug PCP ("angel dust"), an animal tranquilizer; ketamine, an anesthetic; MK-801, an experimental NMDA antagonist; or the previously mentioned meantime. When the PCP is occupied, the opening of the ion channel is blocked, even when glutamate occupies its receptor site. (1-3) The mineral magnesium (Mg) can occupy a site near to, or perhaps identical with, the PCP site. Magnesium blocks the NMDA channel in a "voltage dependent manner." This means that as long as the neuron is able to maintain its normal resting electrical potential of -90 millivolts, the magnesium blocks the ion channel even with glutamate in its receptor.

However, if for any reason (e.g. not enough ATP energy to maintain the resting potential) the surface membrane electrical charge of the cell drops to -65 millivolts, allowing the neuron to fire, the magnesium block is overcome, and the channel opens, allowing the sodium and calcium to flood the neuron. (1-3) After the neuron has fired, membrane pumps then pump the excess sodium and calcium back outside the neuron. (15) This is necessary to return the neuron to its resting, non-firing state. Neurons in a resting state prefer to keep calcium inside the cell at a level only 1/10,000 of that outside, with sodium levels 1/10 as high as outside the neuron (15) These pumps require ATP energy to function, and if neuronal energy production is low for any reason (hypoglycemia, low oxygen, damaged mitochondrial enzymes, serious B vitamin or CoQ10 deficiency, etc.), the pumps may, gradually fail, allowing excessive calcium/sodium build up inside the cell. This can be disastrous. (1-3)

CALCIUM, THE EXCITOTOXIC "HIT MAN"

Normal levels of calcium inside the neuron allow normal functioning, but when excessive calcium builds up inside

neurons, this activates a series of enzymes, including phospholipases, proteases, nitric oxide synthases and endonucleases.(1,3) Excessive intraneuronal calcium can also make it impossible for the neuron to return to its resting state, and instead cause the neuron to "fire" uncontrollably. (1,3) Phospholipase A2 breaks down a portion of the cell membrane and releases arachidonic acid, a fatty acid. Other enzymes then convert arachidonic acid into inflammatory prostaglandins, thromboxanes and leukotrienes, which then damage the cell. (1,3) Phospholipase A2 also promotes the generation of platelet activating factor, which also increases cell calcium influx by stimulating release of more glutamate. (3) And whenever arachidonic acid is converted to prostaglandins, thromboxanes, and leukotrienes, free radicals, including superoxide, peroxide and hydroxyl, are automatically generated as part of the reaction (1-3, 16). Excessive calcium also activates various proteases (protein-digesting enzymes) which can digest various cell proteins, including tubulin, microtubule-proteins, spectrin, and others. (1,3) calcium can also activate nuclear enzymes (endonucleases) that result in chromatin condensation, DNA fragmentation and nuclear breakdown, i.e. apoptosis, or "cell suicide". (3) Excessive calcium also activates nitric oxide synthase which produces nitric oxide. When this nitric oxide reacts with the superoxide radical produced during inflammatory prostaglandin/leukotriene formation, the supertoxic peroxynitrite radical is formed (3,17). Peroxynitrite oxidizes membrane fats, inhibits mitochondrial ATP-producing enzymes, and triggers apoptosis (17). And these are just some of the ways glutamate -NMDA stimulated intracellular calcium excess can damage or kill neurons!

Fran

Thanks Fran - I've printed out your post - will study it and go to the sites you've listed so I can get this straight in my mind.

Looks like you're off the hook, Richard. Fran has all the research.

Jackie

Hi Richard

Forgive me. Whenever I think of enzymes I associate them with amino acids because enzymes are made from the amino acids. Hence when you eat protein it is broken down into the amino acids and then made into enzymes which build your own proteins.

My thinking on all this comes from a slightly different angle to yours and others as I work with whole foods rather than supplements. So rather than take a supplement I go for the whole food from.

Fran

Fran - whole foods must be broken down into micro particles (by enzymes) before they can be utilized in the body. Foods not broken down by the powerful enzymes secreted in the stomach and pancreas will simply pass through the digestive tube and out the body; and contribute nothing to nutrition.

After foods are broken down by enzymes - they become micronutrients in the form of amino acids, fatty acids, vitamins and minerals, etc. Vitamins and minerals actually function as co-enzymes for enzymatic process, but they can't be utilized until the whole foods are broken down.

If the lumen of the small intestine is damaged, clogged or inflamed, the absorption process is hindered.

After absorption into the blood stream, nutrients are then assimilated into the body's cellular network.

The point of mentioning enzymes in this post about fibrosis is the fact that the naturally produced enzymes have limited production in the body starting at the ages of 27 - 35 but the demands for them are not diminished.

In order to insure the body is able to process and absorb foods, the stronger proteolytic enzymes need to be

supplemented. Organ aging due to fibrosis is a natural phenomena....organ size diminishes up to 2/3rd the size of original and becomes hard or fibrotic. Brain, liver, heart, arteries.... nothing is missed.

The supplemental proteolytic enzymes actually dissolve or eat away at fibrotic tissue already in place.

Again, the point of this post was the development of fibrotic tissue, how it forms, and since it is found in the heart and is associated with afib, does it cause afib or perpetuate it once fibrosis is in place.

A remedy or what was termed "cardioreparative" measure by using supplemental enzymes came about as a result of an American Heart Association study suggesting intervention with supplemental enzymes to reduce fibrosis in hearts and arteries of people suffering from hypertensive heart disease.

There is no support for taking supplements and no food. Whole foods are vital to survival.

The supplements to which we are referring replace an enzymatic deficiency which is documented to be in the decline starting between ages 27 and 35 and a list of symptoms indicating the effects of this deficiency is in the literature.

I hope you didn't think this CR topic was about supplements. :)

Jackie

Jackie,

Thanks for that - I've just ordered up:

1. Lipase, Amylase, Protease, Amoglucoside, Peppermint;
2. Bromelaine, Papaine;
3. Serrapeptase.

Show me a bandwagon..... (-:

Any basic rules I should follow as regards what (out of my list as as previously posted) to ingest and when??

Cheers,

Mike F.

Mike F - Regarding dosing: go to <http://www.drwong.info/fromdr.htm>

Additionally, what I wanted to mention was that in listening to Dr. Wong's audio tapes online, it is mentioned that one of the most important enzymes needed to start the enzyme cascade is chymotrypsin.... which is included in the product called Wobenzyme.... but I don't see it on the Vitalzym, although I believe they say it is included in the category of protease.

To be sure you are covered in the enzymes necessary - just compare the labels (online) of Vitalzym and Wobenzyme and shoot for a combination of those. Dr. Wong says that without a doubt, the Serrapeptase is the strongest enzyme of all...so you are undoubtedly well covered with what you've ordered.

If you go to <http://www.iherb.com> you can type in both Wobenzyme and Vitalzym and then click on the name to view the ingredients from the label. Quick way to make a comparison.

Jackie

Hi Jackie

Oh dear I do seem to be causing some confusion. My post you were referring to was in answer to Richard as he reminded me it was amino acids he was taking when his scar disappeared and not enzymes. I flippantly put a post together giving my take on enzymes. So I suppose I better go a bit deeper now.

I know the topic of this post is not about supplements, but it does indicate the need to take enzyme supplements to be sure of breaking your food down. The fact that by the age of 27 most people stop producing these enzymes and start aging means to me that what we have eaten prior to this age means that we have not been eating the right foods - hence there are no ingredients to make the enzymes from. It is the fuel we put in our bodies that indicates what enzymes we produce, unless we have an inborn error of metabolism. For instance my sister in law cannot tolerate oily fish or fish oil supplements. They also come back terribly on her. This indicates that her body is not producing lipase the enzyme that breaks down fat. The fact that she eats a high carb, low fat diet and has done so for many, many years means her body does not have the ingredients to make lipase. We know this because as a child she could eat as much fat as she wanted - in fact her diet was herring, fish and mutton and bread was a luxury item as the delivery van only came once a week, it was only made rarely and deep freezes had not become the common household furniture it is today. What I am saying is that a body will not make something it does not have the ingredients for. So to produce this enzyme she needs to slowly introduce animal fats and suffer the consequences whilst the body kick starts itself again. Similar to a vegetarian starting to eat meat again. If she takes an oral supplement the body will have no need to make those enzymes and to eat fat she will always need to take an enzyme pill. The body can be incredibly lazy when it wants to be, it will not produce something that is coming in orally - hence why when taking supplements you should only take them for a week or so - stop for a bit and then start again - to stop the body becoming dependant.

As for inborn errors of metabolism. I cannot take casein the protein found in milk. This is an inborn error of metabolism (maybe - but maybe just be a convenient label). I know this because as a baby I had projectile vomiting on cows milk and I hated it until I was 5 but became quite fond of it after that. My father and son both could/cannot tolerate casein either- we are/were fine with lactose though?? All of us became addicted to the opiates in dairy too but it gives us phlegm. Now I could take an oral enzyme which could clear this up, but I would become dependent on them every time I wanted to drink milk as my body just will not make it. I cannot help but wonder what the long term effects of taking something my body does not produce would have and how it would impact on other things. I would prefer not to so try to avoid it.

So it would seem to me that the majority of people start aging in accordance with the type of diet they have consumed. Now we can never stop aging as it is as natural as birth and death, but we can prolong it. I turned my body clock off and back just through diet, and at the bottom of this is enzymes and amino acids. Others could choose supplements to replace their enzymes - but in my mind they will never be as good as the ones your body makes. But I am not saying no-one should do this. Who knows further down the line I might feel a need too if I start having digestive problems.

I'm not poo pooing the research you have done. I said further up I thought it good. Only taking it a step further. Sometimes I wish I could read something and believe it implicitly, but I always see faults in supporting 'evidence' which often hold up the main argument, things that are accepted without any question- aging starts at a certain age because of a lack of enzymes - but whoever asked why there was a lack of enzymes and why do we assume we are born with a limited amount - where did the ones we were born with come from - was it not from food the same as our bones and flesh. I aged a lot quicker than the accepted 27 (or at least my organs did) due to bad diet and chemicals, some people at 93 (my old friend) have the body of a 40 year old and the mind of a teenager - with never a days illness in their lives. He still climbs mountains and puts many a young person to shame. Some of it may be in the genes but the fact that he eats fresh food every day, always included a bit of organ meat and never eats anything processed says it all for me... He also smoked for 40 years of his life

Only my take

Fran

Fran, Mike and Jackie,

Thank you Fran for letting me off the hook. I had some really good sites to share on this, but my daughter unplugged my laptop, and I lost them all in my saved area. I haven't been around much since Frid AM, so I have a bit of catching up to do. Thanks Mike for telling me of your Mg levels. That's intriguing that you show low, yet Mg bothers you. Could it be that additional Mg is helping the production of more serotonin, but because of the SSRI's, your body perceives that there is already enough? It could be playing into this scenario somehow, unless you were off SSRI's for any period of time, allowing clearance, and then had the same effects with taking Mg. Has that been the case?

Fran, I agree with your post below, on our eating habits up to the age of 27-35, and the diminishing effects of enzymes, due to eating dead foods. The one question that swayed my wife's decision to not vaccinate our children was this: "Are we bankrupting our immune system?" Maybe the same could hold true for our enzymes, but a bit differently, in that we need to replace these on a daily basis, through alive foods that contain the workers of our system. Of course, we aren't going to adhere to a completely raw diet, esp. with meats, so I personally feel that enzymes could be very helpful. I wonder if free glutamate can be helped by some enzyme we are not aware of, that is destroyed in the cooking process. I do not believe that would apply to MSG, however.

Jackie,

You mentioned below that the most important enzyme was chymotrypsin. Interestingly, this enzyme is made from the amino serine. Here's an excerpt from "The Healing Nutrients Within".

"Serine is a highly reactive amino acid that is found in high concentrations in all cell membranes. It is a hydroxy-amino acid with glycogenic qualities, which enables it to play an active role in the metabolic pathways involved in the proper metabolism of carbohydrates, and fats and fatty acids.

Serine is important in the manufacture of creatine, porphyrins (nonprotein nitrogenous tissue constituents), and purines and pyrimidines (essential constituents in DNA synthesis), and serves as a supplier of methyl groups when needed for DNA synthesis. Serine helps in the formation of ethanolamine, choline, phospholipids, and sarcosine, substances that are needed to produce neurotransmitters and cell membranes, and aids in the production of immunoglobins and antibodies, substances that are essential to maintain a healthy immune system.

Serine can be converted to pyruvate, a common compound in carbohydrate metabolism, which enables it to contribute to the breakdown of carbohydrates for energy and to the exchange of glycogen to glucose in the process of gluconeogenesis. Serine also combines with carbs to form glycoproteins, which help to build basic structural proteins such as hormones, enzymes, and immunologically active molecules. Serine proteases, including trypsin and chymotrypsin, are catalysts for the hydrolysis of peptide bonds in proteins, and assist in the process digestion.

Serine can be made in the body from threonine and glycine. Its conversion from glycine, however, requires adequate quantities of B6, niacin, and folic acid. The critical enzyme involved in this conversion is serine hydroxymethyl transferase, which utilizes B6 and niacin. Glycine is also a precursor to serine, and the two are interconvertible.

Serine is not a well-absorbed amino. In dietary protein, L-serine must first be converted to glycine to be used. In supplement form, it is available as L-serine, but most often it is used in the form of its derivative phosphatidyl serine (PS).

PS and certain enzymes increase opiate binding action in neurons. Serine enhances the effects of opiates such as morphine; therefore, serine supplements may be useful for augmenting pain-relief therapies, especially in cases of chronic pain.

Recent research suggests a link between low levels of serine and conditions of chronic fatigue syndrome (CFS).

Suspiciously high blood concentrations of certain amino acids are often found in schizophrenic patients and individuals with slight neuropsychological alterations. It appears that NMDA receptors are activated in schizophrenia as they are after stroke..... Thus, there may be some parallels in the biochemistry of schizophrenia and stroke. Excess serine lacks the destructive clout of the glutamic and aspartic acid cascade following stroke, but it nevertheless appears to have a neurotoxic effect....

It goes onto to say that there are high serine levels in poor quality foods, such as luncheon meats and sausage. Foods that cause cerebral allergy for example, gluten, soy, and peanuts are also high in serine.

As all this pertains to me, I was high, but not out of ref range in threonine, but was low in serine. I'm pretty sure that it was PS that caused my arrhythmia within 15 mins. of taking. I was originally low in B6, and my folate was trapped, but shouldn't be now, unless I have some kind of genetic problems. Could it be that reactions to sulfites and nitrites in luncheon meats/hotdogs are more to do with serine, or both? It's also interesting that serine is responsible for production of trypsin and chymotrypsin. If there is a metabolic problem with serine, then that's all the more reasons for taking proteolytic enzymes.

Richard