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VIRTUAL LAF CONFERENCE

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SUBJECT: Vitamin D & LAF

Part I – Vitamin D and LAF

The Western World is experiencing an epidemic of obesity and diabetes. Many believe that this is tied to the deficiency of vitamin D in our population. In some studies up to 80% of Americans are deficient in this vitamin. Please visit http://hbomedicine.com/nutrition/Vit_D_deficient-obesity.htm

This latter lists some fascinating circumstantial evidence for the obesity/vitamin D deficiency connection. A summary of that evidence follows:

- Weight increases with higher latitude, lower altitude and in the winter. So does vitamin D.
- When aboriginal populations migrate from high altitude to low altitude, without significantly changing their diet, body fat increases.
- Studies show that populations whose religious customs dictate that the skin be covered, such as Arabs, do not show variations in weight based on altitude. Populations that do not cover the skin, such as central Asians, show variations in weight based on altitude.
- Higher calcium intake is consistently associated with lower body weight. As vitamin D significantly increases calcium absorption, it seems likely that higher intakes of vitamin D would decrease body weight, even if the vitamin itself had no direct effect on weight. Two human feeding studies using calcium (543 mg) and vitamin D (349 units) indicated that the combination of both reduced subsequent spontaneous food intake and increased the metabolism of fat.
- Patients with VDR (vitamin D receptor) polymorphisms have reduced vitamin D activity at their receptors and usually show an increased incidence of vitamin D related diseases. Although VDR polymorphism studies are often contradictory, they tend to show associations with body weight.
- Vitamin D deficiency is associated with elevated parathyroid hormone levels. If vitamin D deficiency caused obesity, than obese patients should have elevated blood parathyroid hormone levels. In fact, the association between obesity and elevated parathormone levels is well known.

Only two studies examined actual vitamin D levels in obese subjects. One found dramatically lower levels in obese subjects. A South Carolina study found all of the obese subjects had levels below 2.2 ng/ml while all of the non-obese subjects had levels above 8 ng/ml. (These are vitamin D levels, not 25(OH)D levels.) Starting in 1981, at least ten studies have shown a linear association, that is, the higher your 25(OH)D levels, the less you weigh. Not only weight, but percentage of body fat, increases as 25(OH)D levels fall. Obese subjects appear to deposit some of their vitamin D in their excessive fatty tissue, thus impairing their ability to raise their 25(OH)D levels. Obesity and vitamin D deficiency are comorbid with numerous diseases such as heart disease, hypertension, diabetes, osteoarthritis, osteoporosis, depression and even periodontal disease. This is consistent with the theory that vitamin D deficiency plays a role in obesity. As obesity is associated with early death, and low vitamin D levels are more likely in the winter, then you are more likely to die in the winter, which is true.

A Norwegian group reported that the more vitamin D and calcium in your diet, the less you weigh.

Just yesterday (5/17/05) I read an article entitled
"Dairy Products May Lower Risk of Type 2 Diabetes in Men"
http://www.medscape.com/viewarticle/504520?rss

Although not mentioned in the article, most dairy products are fortified with vitamin D. Could this be just additional evidence of the importance of adequate vitamin D in preventing diabetes?

So why is there an epidemic of both obesity and vitamin D deficiency? Is it solely or even predominantly due to inadequate intake? Perhaps. But my personal experience on this point, as detailed below, suggests otherwise. There has been tremendous worldwide transmigration. Dark skinned individuals going north and light skinned individuals going south. If sun exposure plays even a small role in the obesity epidemic, then there must be a flip side to this coin. There should also be an epidemic in something related to hypervitaminosis D as well. Could one of those somethings be LAF?

Since relocating to HI my LAF has deteriorated considerably, and I'm now going to try to make an argument involving excess vitamin D in this process.

You may recall that sometime back (late January 2005) I reported on a problem I was having with Vitamin D. Although my blood calcium has always been and continues to be midrange, my blood 25(OH)D was 93 ng/ml on 1/27/05 (normal is 20-57 ng/ml).. In Part II of this report I will reveal why I looked at this. Through January 2005 I was taking 400 IU Vitamin D, a rather puny dose. I immediately stopped this. However, a repeat 25(OH)D drawn on 4/14/05 (2 ½ months later) was still 89 ng/ml. Although it can take months for an elevated blood level to normalize after taking corrective action, at this rate for me it would take years. Since then I've eliminated all dietary Vitamin D and calcium (no egg yolks, no fish, no fortified foods, no mushrooms, etc.). Furthermore, I've reduced my sun exposure by about 95%. A blood 25(OH)D drawn on 5/13/05 after a month of the above lifestyle changes was 71 ng/ml. These modifications appear to be working. I have to believe this is due primarily to sun avoidance. How can elimination of an amount of dietary Vitamin D over a month decrease 25(OH)D by 18 ng/ml, whereas elimination of a similar amount over 2 ½ months decreased it only by 4 ng/ml?

There are rare reports in the literature of markedly elevated 25(OH)D due to sun exposure alone, although the literature will tell you otherwise. I've been communicating with Krispin Sullivan, a Vitamin D expert highly touted by Dr. Mercola, who agrees with my approach to lowering 25(OH)D and has treated several patients with hypervitaminosis D secondary to sun exposure alone.

According to her and the recent medical literature, although Vitamin D (1,25(OH)2D) is the active form, 25(OH)D also exhibits activity. Blood calcium need not be increased for this activity to occur. The primary risk posed by elevated 25(OH)D is coronary arterial calcification, renal stones, bone loss and soft tissue calcification, even in the presence of normocalcemia. These risks have been documented with levels starting at 67 ng/ml (bone loss) and 75 ng/ml (heart disease). LAF may be the least of your worries.

Hypervitaminosis D, according to the literature, can cause "irregular heartbeat, hypoglycemia, weight loss, loss of appetite". I have developed the latter two and the former two have worsened, since relocating. Vitamin D can also lower BP. Mine has dropped significantly since relocation to HI (I know many of you may think there is another more likely explanation for this). Vitamin D is insulinotropic and enhances the action of insulin. Fran (hope you're doing OK) once said that LAF was kind of the opposite of diabetes. Perhaps excess vitamin D is the missing link. In addition Vitamin D through Vitamin D Receptors (VDRs) affect phospholipids in cell membranes by increasing permeability to Ca++. Since VDRs are present in the heart, perhaps this may play a role in "irregular heartbeat". We all know that increased intracellular Ca++ is primarily responsible for the "AF begets AF" phenomenon. Since relocating to this sunny state my AF has markedly deteriorated. This could be due to either the hypoglycemia or the increased Ca++ permeability both caused by hypervitaminosis D. But I think there is another reason (see Part II below).

The gene encoding for VDR is polymorphic and sensitivity to vitamin D has been show to vary depending on genotype. Not only has our departure from the Paleolithic diet of our ancestors wreaked havoc with our general health but also global relocation and hence sun exposure have created additional adverse health consequences wrt the hormone we

call vitamin D.

Additionally vitamin D dysregulation (please visit http://members.aol.com/SynergyHN/vitd) appears to be much more common in the U.S community than previously suspected. Elevated levels are now thought to play a role in Chronic Fatigue Syndrome and Fibromyalgia.

Although blacks can certainly develop LAF (e.g., the famous American basketball player Akeem Olajuwon), why do blacks appear to be less likely to do so? Could their increased melanin pigment make hypervitaminosis D less likely and vitamin D deficiency more likely? Could this also explain in part why obesity and type 2 diabetes are more prevalent amongst blacks than whites? I would also be greatly surprised to find the incidence of LAF in Asians to be anywhere near its prevalence in whites, also due to increased skin pigmentation in Asians.

In one of Hans' LAF surveys he reported that diabetes was absent amongst those that responded to the survey, but that hypoglycemia was present in 24%. This is definitely at odds with their prevalence in the general population.

While I continue to believe that LAF is primarily due to the presence of rogue cells in the PVs near their entry into the left atrium (?P cells), perhaps elevated Vitamin D accelerates the expression of LAF by these cells. It might play less of a role in triggering episodes and more of a role in prolonging them.

Perhaps you ought to test your blood 25(OH)D, especially if you live in the sunbelt.

Part II – Vitamin D and Disopyramide

Furthermore, I've developed a severe adhesive capsulitis AKA frozen shoulder (FS) of both shoulders (left >right). This has slowly developed over the past 11 months from the time just after my arrival in HI.

It is clearly unrelated to any recent physical activity and I have experimented mightily with my daily dietary and supplement intake in order to discover the cause, all to no avail.

Because it is fat soluble, is increased by sun exposure and directly impacts bone and soft tissue, I thought that perhaps vitamin D might be causing my orthopedic problem and hence the request for a blood 25(OH)D level.

Disopyramide may also be playing a role in all this. I've spent several months at a time in HI (two months every summer for the three years prior to relocating) and have had no shoulder problems. I commenced significant daily disopyramide intake in October of 2003. Perhaps disopyramide, 25(OH)D (really a prohormone) and sunlight somehow are working in concert to create the problem. The Norpace product insert describes no adverse reaction even close to what I was experiencing.

So, I went to my favorite website google and worked it up. It turns out that disopyramide is metablized in the liver by a specific enzyme in the Cytochrome P450 system called 3A4. This is one of about 50 such enzymes in "CYP 450". It also turns out that 3A4 is a hydroxylase enzyme and figures prominently in the 25 hydroxylation of cholecalciferol (D3). CYP27 can also perform this hydroxylation.

CYP3A4 Is a Vitamin D-24- and 25-Hydroxylase
http://jcem.endojournals.org/cgi/content/abstract/90/2/1210

CYP3A4 is a Human Microsomal Vitamin D 25-Hydroxylase
http://www.jbmr-online.org/abstracts/01904/JBMR0190406800_abs.html

Cholecalciferol is created primarily in the skin from 7-dehydrocholesterol by the action of UV-B light.

So, my thinking is that the continuous presentation to the liver of significant quantities of disopyramide has resulted in increased production of the required metabolizing enzyme 3A4. Since my relocation to HI in June 2004 there has been increased quantities of cholecalciferol (due to sun exposure) that are now available as additional substrate for 3A4 (which was already increased due to disopyramide). The result is an elevated blood 25(OH)D. The flip side to this is faster metabolism of disopyramide. Since relocating, I've had to gradually increase my dosage regimen in order to

achieve efficacy, i.e., more does less. It is now to the point where disopyramide is nearly useless. Consequently, I'm on the docket in Bordeaux for PVI.

This may seem far fetched and perhaps it is. But the biochemical and chronologic coincidences are hard for me to overlook.

Many cases of adhesive capsulitis are associated with physical activity (throwing, swimming, serving in tennis), but many cases are not in any way so associated. They have no known etiology. The peculiar thing is that nearly all cases spontaneously improve with or without therapy but only after a year or two (?after stopping some medication). Perhaps this is just one heretofore unknown manifestation of another complex drug interaction that may be further camouflaged by "biologic individuality". For example, some people may be homozygous for absence of 3A4 and develop rickets (Vitamin D deficiency).

At this point you may ask how does hypervitaminosis D cause a frozen shoulder. According to Krispin Sullivan, two transitional symptoms have been noted by persons optimizing vitamin D therapy, midday sleepiness and a "reawakening of old injuries" with associated discomfort. I have the former but have difficulty differentiating it from "Hawaiian sleeping sickness" (ha-ha). The latter may hold the explanation for my FS. I am left handed and have occasionally over-rotated my left shoulder playing baseball and while wrestling during my collegiate years.

Here again, you may think this far fetched and perhaps it is. The big questions are will my shoulders improve now that my 25(OH)D is headed down and will my disopyramide become as effective as it once was. I have high hopes for the former, but seriously doubt the latter (too much intracellular calcium).

If you're on disopyramide and live in the sunbelt, then you have additional reason to test your blood 25(OH)D.

PC

PC - Hans is correct, this certainly is interesting reading and it would definitely take someone with your background to ferret it out.

Did I interpret this correctly... too much vitamin D allows excess intracellular calcium and which is excitatory to the heart cells and promotes AF?

Because of living here on the North Coast Ohio, we get far less sunshine than we need. In spite of taking 400 IU (which I know is puny) of vitamin D for years, I still tested low in D. Since taking a more aggressive supplementation, I've increased my levels from 18 to 30 which is still low on the lab range of 20 - 100. So, I was just the opposite of your situation when I was having the acceleration of afib that prompted my ablation decision... low vitamin D and minimal sunshine even though I accumulated as much as possible when we had sun and I could golf, etc.

Also interesting about the reactivation of the frozen shoulder. I would have chalked it up to increasing fibrotic tissue.

I'm really sorry to read of all your problems. One would think retiring to the land of hulas and sunshine would be the epitome of health utopia. But... since you've made this discovery, perhaps between now and the time that your PVI date comes up, you'll enjoy a significant improvement. I'm certainly hoping that for you.

Thanks for this. I'm looking forward to the next instalment.

Best regards,

Jackie

Maybe either too much or too little vit. D leads to afib? Works that way with other things.

PeggyM

PC,

I'm very sorry to hear that you're having more problems since your move to Hawaii. I guess you can still do the hula by moonlight:-)) I think your theory on the Vit D issue is very intriguing, especially since my problems seem to become increasingly bad come November, and tend to lighten up around April/May. On the other hand, strangely, I went out of rhythm quite a bit when I went to the Big Island at the Feb. time of year, and I was more debilitated there. That could have been due to more activity, however. I think I had just started my dietary changes at that time too.

I found this piece of info a bit interesting, esp. since you probably still drink WWater and that increases your pH.

A high circulating level of the biologically active form of vitamin D (1,25(OH)2 vitamin D [1,25(OH)2D) is known to inhibit formation of cancer in the prostate. Eating a diet high in meat and milk and low in fruit reduces the level of this anti-prostate cancer vitamin. "High intakes of calcium and phosphorus, largely from dairy products, lower circulating 1,25(OH)2D level, and sulfur-containing amino acids from animal protein lower blood pH, which also suppresses 1,25(OH)2D production."

http://www.futurepundit.com/archives/002558.html

Personally, I did extremely well with high red meat intake coupled with plenty of salads/vegetables and not so much on the fruit side. That was also the diet prescribed by Dr. Gonzalez for low sympathetic tone. I was not consuming dairy, and still rarely do, but I haven't been following my diet quite as well lately, but I TRY. I did start taking fish oil again in the November season, and am now wondering if that could be part of my problem. I'm not consuming a lot, but I may not need any. I'll have to look over my testings and see if I was ever tested for D.

The sulfur part of the above quote is interesting to me, especially since I was really low in ALL the sulfur aminos, as you may well remember, which I still maintain is crucial to our dilemma as well. If you remember, all the aminos that we seem to think are so important to our heart health, are either a sulfur amino, or are manufactured in the body from a sulfur amino. And the homocysteine marker also has to do with the sulfur/methylation pathway.

Anyway, I appreciate and enjoy reading your ongoing research, and I do hope that you are enjoying your retirement and life in Hawaii despite your increased activity of AF. It's late, so I hope this made sense.

I wish you the very best, PC...

Richard

Richard - So nice to see you posting again. Regarding the D content of fish oil..... if you consume Omega 3 fish oil from body oil, there won't be any natural content of vitamin D. The D portion comes from the liver...as in Cod Liver Oil....

We really do need Omega 3's and quite a significant quantity to maintain the cellular lipid envelope layer integrity... fluidity. Also, studies have been recently issued indicating Omega 3s prevent arrhythmia and others show it helps with blood thinning....certainly something every afibber needs.

Miss your prolific research and posting.

Best regards,

Jackie

Jackie,
VDRs increase cell membrane permeability to both Ca++ and Mg++. But there is usually much more of the former, ergo … In the vitamin D mediated GI absorption of these two cations, calcium is favored over magnesium.

Peggy,
I think you are quite right about the Goldilocks nature to vitamin D.

Richard,
Regarding vitamin D and prostate cancer, it is well known that a deficiency of the former increases the latter. However, it is less well known that too much vitamin D can do the same thing.

Too Much Vitamin D Can Cause [Prostate] Cancer
http://www.mercola.com/2004/jan/28/vitamin_d_cancer.htm

I'm kinda ignorant wrt methylation, but I have no doubt of its tremendous importance.

LAF is clearly multifactorial and very complex. Your seasonal experience with it certainly mirrors that in the medical literature. I thought about this same exception to my argument before I posted it. That's why I stated that vitamin D excess may be more of a player in determining length of episode and persistent v. permanent rather than actually triggering an episode. There is a difference between temperature and actual sun exposure, i.e., cold can exist with sun exposure, e.g., at higher altitudes. Perhaps the cold is more of a player than sufficient vitamin D. Or perhaps Peggy's Goldilocks approach is the answer.

Many on this BB in particular are quite attuned (and rightfully so) to the detriment caused by our relatively recent changes to the Paleolithic diet of our ancestors. The thrust of my post was in general meant to underscore the deleterious effects of transmigration and this same genetic "directive", a point less appreciated.
If the obesity epidemic is in any way related to this transmigration then vitamin D would appear to be the link. Perhaps even more subtle is the point that if some are vitamin D deficient because of this transmigration, then some have to have an excess. Consequently there have to be other diseases, also becoming epidemic in proportion, that are caused by this excess of vitamin D. I think I could make a strong argument that osteoporosis is one. I was just suggesting that perhaps LAF in some is another such manifestation.

PC

Jackie,

Thank you so much for that reminder. I had forgotten about the differences, and yes I was consuming Cod Liver Oil. Thank you for missing me, and I miss all of you, as well. Hopefully when things settle down I can get back to research. My life, and my family's, changed drastically this past summer when our teens discovered new friends (and boyfriend) and my business went in a different direction. I do try to visit from time to time and see what's going on. I'm sure everyone is very thankful for your ongoing dedication to this matter, and I hope everything is going very well for you, and you are AF free.

God bless you Jackie.....

Richard

PC,

Since nothing seems to be working now (my summary of your post), have you tried getting off everything for a while & seeing what happens?

As an aside, with regard to our discussion on HR monitors, I don't trust any reading unless I sit very quietly during the sample time. If I move my arms above the elbow, artefacts are surely to occur.

George

Hi George,

Yes, I've tried that. My episodes gradually started lengthening out and then I became concerned with increasing difficulty to convert. In my pre diso/flec days I'd have episodes that would terminate only after almost 48 hours. I don't want to go there again.

At this point I just take a rather large dose of disopyramide (550mg) on demand and I invariably convert within two hours whether the episode started at night or during the day. It's just that they occur much more frequently than before.

I'm not sure how long this approach will last, but I'm just trying to limp home and make my date in Bordeaux before going persistent or permanent.

Your advice on getting accurate Polar readings is much appreciated. Do you have kids? I'm working with mine wrt measuring their genetic risk for developing LAF. I'm hoping that I can solicit the participation of some LAFers with kids (?any age) that might be interested. All the data could be obtained without travel or lab tests. But this is only in the formative stages.

PC

PC,

I have a 17 year old son & 15 year old daughter. We can participate.

I've already suggested to them to increase their minerals. My son's diet is poor, but at least I've got him using KCl instead of NaCl at the table. At this stage in life, their ears are pretty deaf to my advice.

George

PC,

I guess my point to that little clip above, was that sulfur may help reduce excess Vit D, so maybe you're lacking in the sulfur dept., but you may also be lacking in Ca, which could be driving up your D. And then I also know you were raising your pH through the WWater. Could any of these be part of the problem? Who knows?

Richard

Richard,

As usual you are most perceptive.

I stopped the waller water because I became concerned about the possibility of Milk Alkali syndrome (high milk intake combined with an alkaline antacid). I don't drink milk (except a teeny weeny amount on occasion), but there were other sources of calcium in my diet and the ww provides the alkalinity. Milk Alkali syndrome causes soft tissue calcification and renal stones. By decreasing my dietary calcium blood PTH (parathormone) levels are reflexively increased. This results in calcium loss from bone to address the calcium shortfall. This also stimulates the conversion of 25(OH)D to 1,25(OH)2D, the active form. The latter was/is my goal. And, of course, this goal is undermined, if I were to continue to

ingest dietary vitamin D.

How would you suggest that i address any possible shortfall in the sulfur dept?

Thanks much.

A belated welcome back.

PC

Hi PC,

Sulfur Food Sources: (best sources onions and garlic) Protein….Cabbage….Asparagus….Fruits and vegetables….Eggs….Fish….Dried beans….Milk….Cheese

See: http://www.chemicalbalance.com/sulfur.htm

George

PC - consider MSM - you can look up the properties. Is a quick way to add sulfur. Extremely therapeutic in natural health therapies.

Jackie

Jackie and George,

Thanks for the suggestions.

Most of those on your list (eggs, fish, dairy), George, I have to avoid for the time being in order to lower my 25(OH)D.

And, Jackie, I already take one tab of Nature Made Triple Flex everyday (1500mg glucosamine, 1200mg chondroitin and 250mg MSM).

Thank you all

PC

Here is a recent article on Vitamin D & cancer: http://www.mariettatimes.com/news/story/new22_520200581812.asp

I've pulled out the following from it:

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The new and growing controversy pits proponents of “safe sun,” and those who advise people to stay out of the sun altogether, or coat themselves with sunscreen blocks, at all times, out of doors.

Discussion stems from a recent address to the American Association for Cancer Research by Dr. Edward Giovannucci, a Harvard University professor of medicine and nutrition, suggesting that vitamin D might help prevent 30 deaths for each one caused by skin cancer.

The talk was so impressive that the American Cancer Society is reviewing its sun protection guidelines.
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It would seem that without getting a blood test, you really wouldn't know which camp you are in -- too little, too much or just right.

Here is another article on the same topic (too little Vit. D). Obviously, in PC's experience, #10 on the list is untrue.

George

http://www.newstarget.com/z003069.html

George - thanks for this URL... in yesterday's paper, there was an article about the author, Michael Holick, MD, PhD, vitamin D researcher.... it said once his book came out...the UV Advantage....he lost his job because what he presented was such a departure from conventional thinking.

Going down that list... #11 does it for me. I was D deficient and am still very low.... and it hurts to press on my sternum and has for many years.

I always concluded it was just another fibromyalgia symptom.

Actually, if you type in Michael Holic MD on Google, you get many really good references to his work.

This is important info for all of us. Thanks.

Jackie

PC,

Have you had your cellular levels of minerals tested recently?

By the way, my own approach to the sun (living in a sunny climate), is to go out without sunscreen if I'm out for < 1 hour. If I'm going to be out all day with intense exposure, I lather up (i.e. skiing, hiking, beach & etc.).

I may ask my GP to prescribe another unusual blood test. He tells my wife that I'm an entertaining patient!

One more thought - why are thin, Caucasian women more likely to have osteoporosis (too much vit D)?

George

George,

You're entertaining for us too.

Regarding Vitamin D and osteoporosis, there are so many variables. I think the Goldilocks argument on Vitamin D is really applicable. Too much or too little and one can see bone loss.

While I certainly agree with you on the thin/osteoporosis connection, some of this has got to do with weight. Weight bearing has always been recommended as a tonic for osteoporosis and there isn't much of that if you're thin.

I wonder if thin little old Asian ladies aren't even more prone to osteoporosis. Due to their increased skin pigmentation they would be even more likely to become short of Vitamin D.

Clearly #10 is wrong. I know for a fact that I'm not the only person with this problem. Krispin Sullivan (Naked at Noon, Understanding Sunlight and Vitamin D - due out soon) is about the only one beating the sun exposure/possible Vitamin D excess drum. That same newswire article was in Saturday's paper here in the Islands.

Jackie and Joyce,

Thank you for your comments. I can't tuck in my shirt with my left hand and only barely with my right. A bra would be out of the question (for fastening that is).

PC

George, i always thought that was just a part of what i might call general puniness. On reflection, being of very robust build myself, i believe i think of thin women as less sturdy than i am, and their having more osteoporosis as just an aspect of that. This is a form of prejudice, i will readily admit, but it has a kernel of truth in it, in that the greater stresses on the bones of bigger women like myself tend to make those bones denser and stronger. Fat cells manufacture estrogen at a steady rate, too, even after menopause, and that too plays a part in stronger bones for heavier women. So maybe it isn't that thin women have more osteoporosis, but that fat women have less osteoporosis. It all depends on how you look at things.

PeggyM

Peggy - it's written that thin women have a greater risk of osteoporosis because they don't have the weight to act as an antagonist for the ligament attachments on bones. This action of the pulling at the insertion points stimulates osteoblasts which in turn produce new bone deposition.

They also recommend that thin women use weights and do weight-bearing exercise to give them this stimulation. (swimming doesn't do that)

Heavier women already are carrying around their own weights. :)

That's the mechanical side of it. Then there is there is the biochemical side which involves nutrients to support bone building and a diet that allows the calcium to stay in the bones rather than be pulled from it to buffer an acidic system.

In addition to the obvious minerals involved in bone building,(calcium, magnesium, phosphorous) are critical vitamins K and D and other nutrients like strontium and boron.

Several great books come to mind when discussing osteoporosis...

Preventing and Reversing Osteoporosis by Alan R. Gaby MD and

Strong Women Stay Young by Miriam Nelson, PhD

Better Bones, Better Body by Susan E Brown, PhD

Jackie

PC,

Sorry I disappeared, but wasn't it you that had purchased SAMe, and did you begin taking it, and if so when?

Another good form of sulfur, in my opinion, is n-acetyl-cysteine, but as we all know, B12, folic acid, B6 and Mg are required along with this.

My wife has occasional hip pain, and she takes methionine, n-acetyl cysteine (NAC), and Mg taurate coupled with the other above mentioned vitamins, and her hip pain completely disappears. A side benefit is that her skin tone evens out and there is a more youthful appearance to the skin. The fountain of youth???? Taking NAC alone did not give her the same results. This only happened with the addition of methionine.

FWIW, methionine is much cheaper than SAMe. I know it's a bit scary to take methionine, because of homocysteine, but in my opinion, if all of the other nutrients are taken together with it, then all should be OK.

As a reminder, my molecular tests only indicated a B6 shortage. My intracellular levels of K and Mg were normal to high, as well as my B12 and folic acid levels. My methionine, cysteine, taurine, and glutathione were all below normal. ....And this was after eating a really good diet for 6 mths. prior to testing. I still don't know what my Vit D levels were.

Richard

Hi Richard,

I don't know how you remember these things!

My intake of SAMe has been sporadic, but I do take N-Acetyl Cysteine on a daily basis.

I'm looking at my half full box of SAMe now and promise to be more compliant.

Thanks for the consult.

PC

PC - My mission is creating awareness and now, I really want to thank you for introducing this topic and creating awareness for me even though my focus does not lie with AF at this point, but your post gave me a basis to explore my own experience with my vitamin D levels. I was found to be low 18 in a range of 20-100 and was instructed to take 1200IU vitamin D drops in a micelized form for better absorption. I am at the opposite of the spectrum from you but thanks to you, my awareness level is now where it should be about the symptoms of D deficiency.

Initially, within a few weeks of adding the supplement, much of my stiffness and muscle pain about which I had been complaining on and off for years, (diagnosed as Fibromyalgia), began to diminish considerably. Because I was also doing a liver detox as directed by my FM MD, I thought I had reached the breakthrough point. As I began reintroducing certain foods, I noticed a relapse in wellness and increase in FM symptoms, again. I was instructed to go back on the more strict detox menu complete with the liver support/cleanse nutrients.

And week by week, I became much worse.... and I felt the detox concoction was the culprit, so I stopped completely and but still followed the dietary restrictions.... no dairy, eggs, certain foods that showed up as allergens, etc.

About the time I began experiencing almost continual pain and muscle aches and now, weakness, (I was unable to force myself to workout at my usual routine)...I decided to go back to the point when I had the breakthrough and felt great. What was the difference? It was the addition of the additional vitamin D....I had always taken just 400 IU from Fall to Spring.

I re-read your introductory post here with other additions and the post from George giving the URL on vitamin D...to which I commented about pain in the sternum being a sign of deficiency....and decided I had to do something pretty drastic on my own. My doctor is out of the office for another week and I wasn't going to wait for her permission.

Based on information at Dr. Mercola's site, Dr. Gordon and several other writers including reports by researchers in the vitamin D field and also Krispin's info from several sites, I decided to increase my daily dose to 4000 IU which is what

Dr. Mercola says should probably be the RDA.

Now, this is difficult to believe, but within a few hours, I noticed the pain was starting to subside. Placebo effect? I decided to wait and see what the next day produced. Another dose of 4000IU, and I was moving around and feeling at least 50% less pain. I had gotten to the point where even using pruning shears or digging with a trowel caused pain in my fingers and hands and I was alarmed that it might be a sign of some auto immune problem or worse. By day three... today... The pain and stiffness in my neck and shoulders is reduced by about 90%. I can hardly wait for tomorrow.

By next week, I'll contact the doctor for another blood draw to watch the increase if any in the blood levels. My most recent draw took the level up to 30 but apparently that isn't enough to keep the symptoms away.

The most interesting research I found was that D deficiency is frequently mis-diagnosed as FM. And the most thought provoking comment came from one writer who offered a comment that many people are in nursing homes unnecessarily due to this muscle weakness and pain. Many are in wheelchairs and using walkers. What if it is all 'just' a vitamin D deficiency? While I don't consider myself 'older' I guess those numbers don't lie and so at 69, I guess I qualify as one of the people mentioned.

This is really an eye opener. I hope it's just this simplistic.

I'm grateful for your post, and I wish you a speedy recovery coming in from the opposite direction.

Best regards,
Jackie

Are Older Folk at Risk of Vitamin D Deficiency?
Irene Berman-Levine, PhD, RD
March 12, 2003

Muscle weakness and bone pain in adults can be a symptom of vitamin D deficiency. Dr. Holick believes that many people are mistakenly diagnosed with fibromyalgia - they really have chronic vitamin D deficiency because it also can cause similar symptoms. If someone is presenting with muscle weakness and bone pain it is important to evaluate risk of vitamin D deficiency, particularly if there is adequate exposure to sunlight.

Just imagine the huge numbers of home-bound individuals or those in long term care who are rarely exposed to the sun. Senior adults are at greater risk for vitamin D deficiency because aging can cause a four-fold decline in the synthesis of vitamin D.

Higher risk in seniors also occurs because of low exposure to the sun. Dr. Holick indicated that having seniors sit in the sun for 15-20 minutes a day significantly improve the synthesis of vitamin D. He said that in some countries, such as Great Britain, they are regularly exposing residents in long term care to special ultraviolet lights indoors, to encourage adequate vitamin D status.

http://www.healthandage.com/Home/gid2=2312

And from another article:

Patients with severe vitamin D deficiency and hypocalcemia present with classic findings of neuromuscular irritability, including numbness, paresthesias, muscle cramps, laryngospasm, Chvostek's sign, Trousseau's phenomenon, tetany, and seizures.1

By contrast, patients with mild vitamin D deficiency present with more subtle complaints such as muscle weakness or pain. Finding only a modest reduction in a patient's calcium or phosphate level should not reassure the physician that all is well. When vitamin D deficiency is the cause of hypocalcemia or hypophosphatemia, replacing calcium or phosphate alone does not restore the body to homeostasis.2,3

Vitamin D deficiency is diagnosed by finding a low 25-hydroxyvitamin D level.
Ionized hypocalcemia has been found in 15 to 50 percent of patients being treated in intensive care units (ICUs) and is associated with increased mortality and disease severity.4,5 However, chronically ill patients only rarely develop true tetany and hemodynamic instability.5,6

Prolonged asymptomatic hypocalcemia from deficient vitamin D production or absorption stimulates the release of parathyroid hormone (PTH). If vitamin D is not provided, secondary hyperparathyroidism develops with increased bone turnover and decreased bone mineralization.2

The adult patient with severe vitamin D depletion develops osteomalacia and presents with localized bone pain, antigravity muscle weakness, difficulty rising from a chair or walking, and pseudofractures.3,7

Undiagnosed Vitamin D Deficiency in the Hospitalized Patient
http://www.aafp.org/afp/20050115/299.html
American Family Physician

Hi Jackie,

Coming from opposite ends of the Vitamin D spectrum, I hope we don't collide in the middle of the normal range for 25(OH)D.

I'm definitely improved wrt my orthopedic problem and have managed to regain my appetite. There is no doubt in my mind that this was/is Vitamin D excess related.

Hope your path to improvement continues.

Now if only LAF was as easy to address and correct.

Looking forward to Bordeaux.

PC

P.S. Thank you for all the awareness you create. Your patience and dedication is a model for us all.

PC - thank you for your kind words. I, too, hope we don't collide, am looking forward to a prompt resolution of this problem. I'm noticing a gradual improvement daily so I must be on the right path. I'll go in for a test soon just to insure I don't over-correct.

Sigh, I agree about the cure for AF. So near and yet so elusive for some of us. But, we gave it a good run and learned much along the way. I have no regrets.

My single-most observation, which I think is key to turning around AF by oneself...is catching it at the onset and boosting all the nutrients involved early on. That way, the damage or pattern or whatever it is doesn't become entrenched. Probably it isn't that simplistic, but I know several of the newbies have had remarkable results with the electrolytes and dietary mindfulness. Maybe they aren't 'cured' but the absence of AF is remarkable.

I'm readying the angels and prayers for you and I can hardly wait to read your report.

Be well and be calm until the big day.

Best regards,
Jackie

P.C.

What is meant by a prohormone as when you say "Perhaps disopyramide, 25(OH)D (really a prohormone)?"

Can this hormone interact with estrogen?

Carol

Jackie said:

My single-most observation, which I think is key to turning around AF by oneself...is catching it at the onset and boosting all the nutrients involved early on. That way, the damage or pattern or whatever it is doesn't become entrenched. Probably it isn't that simplistic, but I know several of the newbie's have had remarkable results with the electrolytes and dietary mindfulness. Maybe they aren't 'cured' but the absence of AF is remarkable.

Hi Jackie and PC,

This has nothing much to do with PC's Vit D but I thought I would add to Jackie's paragraph.

Over the past months to years I have "corrected" I think ! my nutrient and dietary shall I say habits, in as much that I try to keep myself hydrated with ww, keep to my somewhat inclined low-ish carb diet. I have my banana first thing. Eat a variable breakfast ie one morning a carbie serial the next a protein breakfast with my lsV8, fish oil, Mg., vit E, vit C. My main meal is
now mid day. Snacks are fruit, nuts, or a light something on toast. I don't eat any simple carbs after two to three pm, have a light evening meal at around five to six pm with my fish oil, vit C, and Taurine. I salt any meal or snack with KC1.

To date I have had three short af's always early am, around a couple of hours at most, about a month apart (being sixty seven I don't think it is male menopause !) these I have "possibly" converted using lsV8, Mg, and Taurine. I have been taking NK at onset eight hours apart 2 x 3 times after any af. I have Flec if needed but after the known after effects I am loath to even try to shorten af with Flec.

Have just started cycling again with a warm up/down before/after, and a close eye on my max hr. So far so good.

So to this I think Jackie is right as usual with her summery above. I will look into vit D with a serum test after our winter. I usually have a check up as our summer season starts.

Thanks PC or a thought provoking post.

David S. vlaf 6 yy

Aloha Carol,

Krispin Sullivan and a few other experts refer to 25(OH)D and 1,25(OH)2D as prohormones, because they control so many physiologic processes. The term vitamin doesn't begin to do it justice. The prefix "pro" simply indicates that it is a precursor.

I'm sure that Vitamin D interacts with estrogen and many other hormones -anything that impacts calcium metabolism. But I'm not an endocrinologist and can't be more specific.

PC

G'day David,

Sorry to hear that AF has reared its ugly head.

Couldn't agree with you more on the earlier the better approach to controlling AF. There are all kinds of receptor sites in the atria that are upregulated/downregulated by AF. Most notably angiotensin II type 1 receptors are downregulated and angiotensin II type 2 receptors are upregulated.

Those with high vagal tone hyperrespond with renin after orthostatic challenge. One can only imagine what is going on in the heart wrt to this up/down regulation when participating in endurance sports.

At this point I think that P cells are not damaged, but the atrial substrate is and becomes more receptive over time to the PACs that are constantly being produced by those with "misplaced" P cells. At some AF manifests. Electrolyte control and tweaking is helpful at first but the floodgates of the RAAS system have done their damage and continue to do so.

PC

Jackie,

I've been out of town, so just catching up here (I wanted a stress test of my supplement program, so I took 8 teenagers to the beach in Mexico for a HS graduation trip -- they survived & I stayed in NSR, a happy event on all accounts!). I agree that an early and agreesive approach to rectifying an mineral issues certainly gives someone a better chance at beating afib than letting it get entrenched. However Fran certainly had an entrenched case & she beat it with diet.

Thanks for posting on your results with D supplementation. My wife has some of the issues that you speak of & I will recommend that she have her 25(OH)D levels tested.

Now that it is several weeks later, how are you responding?

Regards!

George