

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: Candida and LAF

Richard brought up and thoroughly researched the subject of molybdenum, acetaldehyde and candida in Conference Room Session 16 (www.afibbers.com/conference/session16.pdf). The subject was discussed extensively with much valued input from other afibbers, especially from Fran and Jackie.

While there is no question that candidiasis (yeast overgrowth in the colon or on mucus membranes) is a very bad actor and at the root of many diseases and disabling disorders I have not come across any evidence that candida could be an underlying cause of lone atrial fibrillation. Could it be?

I have done a bit more research and have come up with a few tantalizing clues in addition to those already mentioned in Section 16.

As Richard pointed out the main waste product of candida is acetaldehyde (AH). AH also happens to be the main break-down (oxidation) product of alcohol and is believed to be the actual cause of the many problems arising from excessive alcohol consumption.

Here are my observations in no particular order:

A magnesium deficiency is associated with an increased risk of fungal infections (including candidiasis) and magnesium absorption is hindered by certain parasites (1).

AH interferes with protein synthesis in the heart muscle and increases the level of atrial natriuretic peptide messenger RNA (2). I am not at all sure what this means in the context of afib, but it does show that there is a connection between acetaldehyde (and by implication candida) and ANP – at least in mice :-). It could also help explain why afibbers have elevated blood levels of ANP.

Endocarditis (inflammation of the lining of the heart cavity) can be caused by candida albicans (3).

The supposedly most effective antiarrhythmic drug, amiodarone, has a high degree of fungicidal activity and kills candida albicans (4). Could this effect partly explain its efficacy in the treatment of atrial fibrillation?

There is evidence that a systemic candida albicans infection can promote inflammation of the heart tissue (myocarditis) in severely immuno-compromised AIDS patients (5). The question of course is: "Just how weak does ones immune system have to be before candida can cause inflammation in the heart?"

Acetaldehyde has been found to concentrate in the heart; it acts directly on myocytes (heart cells) and has significant

effects on cardiac contractility and function. It can cause fibrosis and enlargement of individual heart cells. Animal experiments have shown that high AH levels produce focal lesions, interstitial fibrosis, loss of myofibrils and mitochondria with disorganized cristae (infoldings of the inner membrane) (6). It is perhaps not coincidental that Dr. Andrea Frustaci and colleagues found that lone afibbers have enlarged myocytes (heart cells) in the atria, but not in the ventricles. Electron microscopy also showed clear evidence of interstitial fibrosis, focal lesions and necrosis and of course, inflammation, in the atria of afibbers (7). Dr. Frustaci speculates that the fibrosis and myocyte degeneration "may represent an organic substrate for the electrogenic mechanisms involved in paroxysmal LAF." It is interesting that the myocyte damage is believed to be fully reversible (personal communication to Hans Larsen, July 23, 2001).

So, could the myocyte damage be caused by acetaldehyde released by candida and more importantly – could the myocyte damage be reversed by eliminating the candida infection? It is interesting that Fran used to battle candida but is now free of it – and free of afib. It is also interesting that Fran's diet is almost perfectly designed to starve candida and eventually eliminate the overgrowth.

I have initiated a new LAF survey to establish just how common candida overgrowth is among afibbers and would suggest that the subject of candida and acetaldehyde and their direct effect on the autonomic nervous system and the heart, especially the atria, is a worthy subject for some additional diligent research and discussion. You can participate in the survey at:

www.afibbers.org/lafsurvey_6.htm

The floor is open!

Hans

References:

- (1)http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11425281&dopt=Abstract
- (2)http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=10905996&dopt=Abstract
- (3) http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11570782&dopt=Abstract
- (4)http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11752116&dopt=Abstract
- (5)Horman, P. et al. Fungal myocarditis in acquired immunodeficiency syndrome. Arch Mal Coeur Vaiss, Vol 85, February 1992, pp. 203-08 [article in French].
- (6)http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=10525098&dopt=Abstract
- (7)<http://circ.ahajournals.org/cgi/content/full/96/4/1180>

Hans,

I can't get onto the survey page. I'll assume that there's a temporary problem and try later.

Mike F. (lifelong battler of 'doby's itch' as well as regular outbreaks of thrush and athlete's foot..... plus lots of bloating and belching as long as I can remember..... plus LOTS of stringy danglers when trying Richard's test).

Mike F.

Hans,

Thank you for keying in on Candida and sharing your thoughts and links. My whole family did the Candida saliva test, and although we all showed strings in the water, mine was by far the worst. Another interesting observation was, that my wife's showed known after smoking a cigarette first, but the next morning did show the strings without the cig. Another interesting point, is that I'm the only one on the Paleo diet presently, and have been since Feb 03, with a few cheats. The rest of the family is still imbibing in sugar and dairy. As everyone is aware, the most profound result of my testings was the fact that I was extremely low in molybdenum, which is a necessary nutrient for breaking down harmful

acetaldehydes to acetic acid. I was more of a social drinker, and never had more than 2 or 3 drinks per month, yet I often thought I could be allergic to alcohol, as I would wake up with headaches. I have also suffered for years, pre-Paleo, with bloating, indigestion, bouts of constipation, infrequent stools, headaches, and GERD. I do believe that Candida is not only responsible for its deleterious effects, but also that it causes malabsorption problems. Here's a link and the stages of Candida.

Linda Page, author of Healthy Healing, breaks Candida albicans overgrowth into clearly defined stages (quoted from 11th ed., p. C342):

1st symptoms: Bowel and bladder irritation; heartburn; chronic indigestion with gas and bloating; recurring cystitis or vaginitis; chronic fungal skin rashes and nail infections.

2nd symptoms: Allergy-immune reactions--chronic bronchitis; hives, sinusitis; eczema; hayfever, acne; chronic headaches or migraine; muscle pain; earaches; sensitivity to odors.

3rd symptoms: Central nervous system reactions—extreme irritability; confusion, a spacey feeling and nighttime panic attacks; memory lapses and inability to concentrate; chronic fatigue and lethargy, often followed by acute depression.

4th symptoms: Gland and organ dysfunctions: hypothyroidism; adrenal failure, hypoglycemia, ovarian problems, frigidity and male impotence; lack of sex drive.

<http://www.chipengelmann.com/vitaconnect/Articles/Candida.html>

This is a scarier thought of what Candida can do:

Multiple sclerosis - causes degeneration of the myelin sheaths in motor and sensory tracts of the CNS; may be associated with infections of a fungus (Candida albicans)

http://www3.baylor.edu/~Joseph_D_White/courses/nervous.htm

Mental & Nerve Dysfunction

Candida albicans can synthesize acetaldehyde, a toxic metabolite that causes cross-linking, damages organs, and interferes with the synthesis of acetylcholine and other neurotransmitters. This disruption of the nervous system can cause mental disarrangement, abnormal behavior, and memory loss. Candida toxins can also alter the functioning of the central nervous system leading to distorted thinking, mood swings, depression, agitation, impaired intellectual functioning and emotional disturbances. It's even possible for candida to produce symptoms of alcohol intoxication by fermenting simple sugars.

Systemic Problems

Systemic candida is a great imitator. It can mimic many diseases such as cystitis, Crohn's disease, gastritis, multiple sclerosis, endometriosis and various forms of mental illness. Candida may be part of the pathology in colitis, pancreatitis, hepatitis, cirrhosis, diabetes mellitus, malignancies, endocrinological pathologies, and autoimmune disorders

<http://www.vrp.com/art/129.asp?a=0&b=0&c=1049914385457&d=VRP&e=&f=&g=Candida&h=&i=&j=&k=/golib.asp&l=&m=/vstyle.css&n=&o=1&p=&q=>

PANTETHINE (CO-ENZYME B5)

Pantethine is the stable structure of Pantethine, which is the biologically active form of pantothenic acid (vitamin B5.) It is the precursor to co-enzyme A (CoA.) Co-enzyme A plays a central role in the production of energy in your cells via a biochemical pathway called the Krebs' cycle. This involves the production of energy from fats & carbohydrates. Pantothenic acid is also essential for adrenal gland function.

Chemicals known as acetaldehydes accumulate in your body from alcohol intake, cigarette smoking, chronic candida infections & vehicle exhaust fumes. The aldehydes inhibit co-enzyme A & thus decrease energy production. Chronic

aldehyde exposure also contributes to heart disease & has damaging effects on brain function. Acetaldehyde also impairs the red blood cells delivery of oxygen to your body's cells. It also induces a deficiency of vitamin B1, B3, & pyridoxal-5-phosphate. Pantethine has been found to help decrease aldehyde levels.

<http://www.thewayup.com/products/0012.htm>

For further info, read the above links. Due to my instant relief of gas, bloating, and indigestion when eliminating food for Candida, I felt 100% better. It was a miraculous turn around upon the second day. I urge everyone with any kind of indigestion problems, to at least try the Paleo diet for just one week, and see what results they find in regards to digestive upsets. This won't immediately cure your arrhythmia, as I'm still struggling with that, but I feel it was a step in the right direction.

It may be prudent to take molybdenum and pantethine, as well, esp. if alcohol is a trigger in any way, or you have chemical sensitivities. I am still trying to find the correlation of free glutamate toxicity and how it may relate to Mo or Candida, but Fran had shared the fact that Mo does assist in helping with glutamate exitotoxicity. (I wonder if she has that link.) Dr. Gersten prescribed 300mcg per day of Mo, but I'm presently upping that to 1000mcg per day for one month in divided doses. I am also going through all my supplements to make sure none contain any yeast, as this could very well be the reason that some have problems with vitamins and I don't want to add any more yeast to my system.

I have more to share, but don't have it at my fingertips. I have found a study on Melatonin and acetylaldehydes, but I have to purchase it, as it's a new study. We'll be away for the holidays, but I'll have my laptop, and I'll see if I can get that it, as it really peaked my interest. Thank you Hans, for your ongoing dedication to trying to help all of us, and giving us the opportunities of this research and discussion group.

We all have much to be thankful for. Happy Thanksgiving.

Richard

I posted this on the BB, but would like to submit the link for future ref. for the CR. Here's, in part, the info for Candida, but the rest of the info. at this site is invaluable, and then after reading, click on the recommendations at the bottom of the page.

Candida Yeast Infections: These are very common. Check your tongue. If it has a white coating, you have it. Or take the spit test you will read about later. Women may get vaginal yeast infections caused by candida overgrowth. A candida infection on its own can cause a number of autoimmune type symptoms. Sinus infections often are caused by candida.

<http://www.gethealthyagain.com/autoimmune.html>

Richard

I found this article to be very informative:

Acetaldehyde: A Common and Potent Neurotoxin
This article first appeared in the
July 1997 issue of VRP's Nutritional News
by James South (The same author of glutamate toxicity)

<http://www.vrp.com/articles/598.asp>

Richard

Here's an interesting bit, esp. as it pertains to me.

Aiken SC. Doctor Brice E. Vickery, Medical Director of SuperNutrient Corporation, has written to me and given me some interesting websites to investigate. This is what he wrote to me:

In the light of our investigations there are some things that should be brought up to date. For instance a large, large number of people who have fibromyalgia have candida. There is much more to FM than yeast however. If you go to our website <http://www.Fibromyalgiacure.com> you will see that the causes and cure of this conditions have been found - and dealt with our protocol. All of these persons are protein deficient and you should also go to <http://www.SuperNutrient.com> This is the spearhead against ALL degenerative diseases. One more thing: Everyone that has FM has degenerative disk disease - go to <http://www.Backpainscure.com>

What you need to know is that our SuperNutrient, Platinum Plus gets rid of the yeast in the gut and is the most natural way of doing it. so what I am saying is - yes you could put up a link to our site for the Platinum Plus as an additional resource. We've been doing this since 1980. Further more I would be glad to add your page as a link to SuperNutrient IF you put up the REAL strict diet and explain that potatoes, carrots, beets, and corn are not permissible or acceptable in the real nitty-gritty cases! Having over fifty years experience and knowing that viruses will not be beaten in some cases as long as the yeast is fed makes this necessary. It as you probably know is called the MEVY diet. Meat-Eggs-Vegetables and Yoghurt.

ANOTHER UPDATE - AUGUST 2002

I did not think that there was a condition that justified a classification there either over five years ago but now say there definitely is. We have broken it down into fibromyalgia, and pre-fibromyalgia. There are commonalities to all cases although some have unique and disastrous ADDED conditions such as lupus!

Every FM person has to have this to start the condition: protein/sulfur deficiency which we found the answer to with our Platinum Plus. But, it is too late for this alone to recover from deficiencies ABCDEFG etc. That is why we developed the protocol for successful treatment with these ingredients - in SPADES!

The reason that many of these people have muscle aches is that they are hypoglycemic, do not convert their protein properly and therefore convert it to sugar, fat, and LACTIC ACID. This condition of high lactic acid is what used to be called rheumatism, lumbago and other general names. They are all protein/sulfur deficient.

Now since sugar make them feel better for a short time the incidence of yeast is high among these people. I am not downplaying the role of yeast but it is usually found in these deficient people since they do not make enough HCL for their stomach (Hydrochloric acid) for which they need the amino acids (histidine) and ordinary salt.

The second great cause is that they have DDD (degenerative disk disease) which causes myalgia, neuralgia and a host of trigger points.

Lastly every real case of FM has lymphadenitis (inflammation of the lymph system) while pre-fibromyalgia persons do not have this. This is by test and not by guess or symptoms. I did not mention the constants and viruses such as Hep C (HCV) or metals such as aluminum, mercury etc. When you see this time after time, after time, in hundreds of cases there is no doubt in your mind - at all. I will be doing more guest appearances on radio talk shows about this condition.

Thank you, Brice_E_Vickery@Prodigy.net

<http://www.geocities.com/HotSprings/4966/fm.htm>

Richard

Wow, Richard! I never thought of myself as fibromyalgic but I do have degenerative disk disease from neck to tail bone. These links describe me to a tee. Thanks.

John S.

After many years with AF I eventually developed Fibromyalgia - also had the chronic sore throats, armpits etc (?lymphadenitis). My GP brought up the connection between AF and fibro (he has fibro himself) as the heart is a big muscle)

My candida started as a very young child in the genital tract- and treatment consisted of antibiotics and more antibiotics prescribed by my mother the nurse. I remember it aged 5 and I remember the last time I got it severely aged about 20 (two years before AF struck). I am very interested in what is meant by "I did not mention the constants and viruses such as Hep C (HCV)" in relation to candida - Aged 18 and about 20 I had hep B and a strain of hep that was later named as Hep C.

So does candida lead to AF? I think there is a strong possibility especially when you consider that candida leads to food allergies and intolerances galore. This would give credence to inane food triggers as well as the more toxic ones such as MSG.

A while back I did the saliva test and had strings galore. This was after I got rid of fibro and AF by eating a whole food diet. I was still eating whole grains (in the form of bread, museli, porridge and oats for coating food), and a little bit of sugar in the form of molasses and pure sugar cane in bars. But as my fibro and AF had disappeared I did not follow it up. Then I decided to go paleo because of the reactive hypoglycemia (did not tie this in with candida then) which seemed to be getting worse. A few months back after being paleo for some months I repeated the saliva test when I came across it again - this time there were no strings.

From this I gather that candida symptoms must manifest themselves a bit differently depending on your body chemistry. With each shift I had in body chemistry (caused by diet, illness etc) the symptoms of candida seemed to have changed themselves. The most obvious candida symptoms - bloating, gas (actually in hindsight I did get this briefly when I introduced museli for breakfast - but no GERD), GERD has always escaped me. But then is my candida spot in a different place to most eg the genital tract.

I don't know but it makes sense. And as to whether we can ever make a complete recovery - I'll let you know in few years. I know I am still sensitive - but then I can cheat with the odd grain and honey now and not get a reaction. But give me free glutamate and I will be off.....

Fran

Here's a few more studies that may or may not be pertinent:

Experimental Candida albicans endocarditis: characterization of the disease and response to therapy.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=328393&dopt=Abstract

S-adenosyl-L-methionine: its role in the treatment of liver disorders. (As it pertains to acetaldehydes)
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12418503&dopt=Abstract

Folate deficiency disturbs hepatic methionine metabolism and promotes liver injury in the ethanol-fed micropig. (I was extremely low in folate)
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12122204&dopt=Abstract

Removal of acetaldehyde from saliva by a slow-release buccal tablet of L-cysteine.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11774289&dopt=Abstract

Role of protein tyrosine phosphorylation in acetaldehyde-induced disruption of epithelial tight junctions.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11352822&dopt=Abstract

Protective roles of mitochondrial manganese-containing superoxide dismutase against various stresses in Candida albicans.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12898709&dopt=Abstract

Garlic (*Allium sativum*) as an anti-Candida agent: a comparison of the efficacy of fresh garlic and freeze-dried extracts.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12174037&dopt=Abstract

I had to copy these links to Microsoft Word, as I could not save.

Improvement of glucose and lipid metabolism in diabetic rats treated with molybdate

A. T. Ozcelikay, D. J. Becker, L. N. Ongemba, A. M. Pottier, J. C. Henquin and S. M. Brichard

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Molybdenum mimics certain insulin actions in vitro. We have investigated the effects of oral administration of Na₂MoO₄ (Mo) for 8 wk on carbohydrate and lipid metabolism in streptozotocin-diabetic rats. Mo decreased hyperglycemia and glucosuria by 75% and corrected the elevation of plasma nonesterified fatty acids. Tolerance to glucose loads was improved, and glycogen stores were replenished. These effects were not due to a rise of insulinemia. In liver, Mo restored the blunted mRNA and activity of glucokinase and pyruvate kinase and decreased to normal phosphoenolpyruvate carboxykinase values. Finally, Mo totally reversed the low expression and activity of acetyl-CoA carboxylase and fatty acid synthase in liver, but not in white adipose tissue. In conclusion, Mo exerts a marked blood glucose-lowering effect in diabetic rats by an insulin-like action. This effect results in part from a restoration of hepatic glucose metabolism and is associated with a tissue-specific correction of lipogenic enzyme gene expression, both processes being essentially mediated by reversal of impaired pretranslational regulatory mechanisms. These observations raise new therapeutic perspectives in diabetes, particularly in the insulin-resistant condition.

http://ajpendo.physiology.org/cgi/content/abstract/270/2/E344?maxtoshow=&HITS=10&hits=10&RESULTFORMAT=&fulltext=molybdenum&searchid=1069978523438_704&stored_search=&FIRSTINDEX=0&sortspec=relevance&resource_type=1

Cytochrome oxidase is the terminal electron acceptor in the respiratory chain and must donate its reducing equivalents to oxygen to allow continued electron transport. Otherwise, ATP production cannot continue. Thus the major role for oxygen in all aerobic organisms is simply to act as a sink or dumping ground for electrons (34). The tetravalent reduction of oxygen by the mitochondrial electron-transport chain is considered a relatively safe process. Nonetheless, the electron carriers catalyze alternating one-electron oxidant-reduction reactions, and they can react with oxygen to generate ROS such as O[•] (47, 96, 97). Mitochondria are the major intracellular sites of O[•] generation under physiological conditions (53). One other potentially major source for the generation of O[•] is the NADPH oxidase enzymatic system, which is found in neutrophils, monocytes, macrophages, cytochrome P-450, monoamine oxidase, and lipooxygenase (4, 22, 31, 34). O[•] is also generated by other mechanisms such as molybdenum hydroxylase reactions (including the xanthine, sulfite, and aldehyde oxidases) and arachidonic acid metabolism.

http://ajplung.physiology.org/cgi/content/full/283/2/L246?maxtoshow=&HITS=10&hits=10&RESULTFORMAT=&fulltext=molybdenum&searchid=1069979650091_509&stored_search=&FIRSTINDEX=50&sortspec=relevance&resource_type=1

Role of reactive oxygen metabolites in murine peritoneal macrophage phagocytosis and phagocytic killing

S. Takao, E. H. Smith, D. Wang, C. K. Chan, G. B. Bulkley and A. S. Klein

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This study was designed to quantify the role of reactive oxygen metabolites (ROMs) in two distinct components of murine peritoneal macrophage activity, phagocytosis and killing, and to discriminate quantitatively the degree to which each component is dependent on NADPH oxidase and/or xanthine oxidase. A fluorochromatic vital staining technique was modified to simultaneously quantify phagocytosis and microbicidal activity of macrophages incubated with *Candida parapsilosis* targets. To determine the role of ROMs, macrophages were preincubated with free radical scavengers [superoxide dismutase (SOD) and/or catalase] or with selective inhibitors of xanthine oxidase (XO, e.g., allopurinol) or NADPH oxidase [diphenyleneiodonium (DPI)]. Phagocytosis was not affected by treatment of macrophages with SOD, catalase, allopurinol, or DPI. Candidacidal activity, however, was inhibited by SOD, allopurinol, or DPI. The inhibitory effects of DPI and allopurinol were additive. Histochemical and biochemical assays demonstrated substantial quantities of XO in murine peritoneal macrophages. The findings suggest that the generation of ROMs by XO- and NADPH oxidase-dependent pathways are each important for phagocytic killing by murine peritoneal macrophages.

http://ajpcell.physiology.org/cgi/content/abstract/271/4/C1278?maxtoshow=&HITS=10&hits=10&RESULTFORMAT=&fulltext=candida&searchid=1070005912797_4&stored_search=&FIRSTINDEX=0&sortspec=relevance&resourcetype=1

Exercise-induced stimulation of murine macrophage phagocytosis may be mediated by thyroxine

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The present study was designed to test the hypothesis that changes in plasma concentrations of hormones may be responsible for the exercise-induced macrophage phagocytic stimulation. The effect of 30-min incubation of macrophages with plasma from mice previously exposed to swimming until exhaustion (with or without previous training) was studied, and the results showed a similar stimulation of the phagocytic capacity (attachment and ingestion) of these cells to that found in previous studies after exercise. Also, changes in plasma concentration of both thyroxine (T4) and 3,3',5-triiodo-L-thyronine (T3) after exercise were measured, and their effect on phagocytic capacity after in vitro incubation with peritoneal macrophages was investigated. Results indicated that, after exercise, plasma concentrations of T3 and T4 increased. Incubation of peritoneal macrophages for 30 min with a concentration of T3 similar to that observed in the plasma immediately after exercise (1.5 ng/ml) induced no modifications in the phagocytic capacity. However, a physiological concentration of T4 after exercise (75 ng/ml) stimulated the phagocytic capacity of peritoneal macrophages. In addition, a 10,000-fold greater concentration of these thyroid hormones did not modify the macrophage function. It is concluded that physiological concentration of T4 may be a mediator of the stimulation of the phagocytic function in macrophages induced by exercise.

http://jap.physiology.org/cgi/content/abstract/80/3/899?maxtoshow=&HITS=10&hits=10&RESULTFORMAT=&fulltext=candida&searchid=1070005912797_4&stored_search=&FIRSTINDEX=0&sortspec=relevance&resourcetype=1

Just some additional thoughts, and bear with me, as I haven't gotten to dedicate a lot of time to this, due to the holiday. Candida may or may not be the problem, but there's a good chance it's playing into our scenario, moreover acetaldehydes. Let's assume we do have Candida and we remove the infestation through diet. Then what? The damages are done. Are the damages fibromyalgia, lesions, or something of the like and how would we repair those damages? Carol was taking Mg. glycinate and lysine, with some immediate success, but could the missing link for her have been methionine and/or molybdenum, that could have given her continued success? How would glycine and lysine play into acetaldehydes? Why did she have such good results in the beginning, then revert back, somewhat? What I do know is that molybdenum is essential for s-adenosyl-methionine, and I was low in both, but folate is also essential, and I was low in this, as well. I wasn't low in Mg, intracellularly, nor was I low in K, not to say that others here may be. If Erling is reading here, I would be very interested to know exactly what he did, in regards to diet and supplementation. I don't remember reading his story of success and how he achieved it, except in regards to Mg. Anyway, I have to go catch a plane, but I'm going to focus in on the damages done by acetaldehydes, and what can reverse their effects. I hope this made sense, as I'm in a hurry.

Richard

Hans – thanks for another provocative topic and one with which I have personal experience having fought the battle with Candida and won.

Richard – thanks for the technical explanations which point out the far-reaching complications brought on by Candida. It is nothing to take lightly and it is not something that can be eliminated quickly or easily. It takes perseverance and determination.

Following are comments on how my Candida was diagnosed and treated and a rather simplistic but (hopefully), clear analogy of exactly how Candida affects us. Most people don't grasp the concept of a yeast infection.

Part I - My Candida Experience

I was tested for Candida through a Comprehensive Stool Analysis both before and post-treatment. My symptoms included bloating and gas soon followed by an afib event after eating an evening meal containing carbs. Frequently, I had intense itching of the roof of my mouth. After treatment, this vanished along with the yeast overgrowth. I was never

plagued by a vaginal yeast infection.

I followed a Metagenics protocol and the whole process took about 3 months. After about two months, the patient experiences a die-off reaction...called the Herkimer response...that manifests by intense itching (detox rash). Mine showed up on the inside of one ankle as an angry rash with the most intense and maddening itch imaginable.

About two months later, I discovered an OTC product called Candex that is quick and does not produce the die-off reaction. I did one round of that for good measure and continue to do a maintenance dose periodically. More on these treatments in Part II.

Many people don't understand the way yeast grows since bread-making from scratch seems to be a lost art, but when starting out with a sourdough, for instance, you proof the yeast and let it grow to a sponge.

This soupy batter becomes larger in volume from the bubbles from the CO₂ (gas) produced as the byproduct of yeast growth. Further, when this is stirred down and deflated, it can be refrigerated and lies "dormant" until a person wants to use it or revitalize it with the addition of some flour and sugar.

Then the growing process or fermentation starts all over again and it grows in volume, producing the bubbly soup of gas bubbles. This is exactly what happens in the intestine with Candida overgrowth and about 20 - 30 minutes after a meal with enough carbs to feed that soup.

Starving Candida of food for growth will kill it...but it takes a long time. Using herbal preparations also takes longer than the actual destruction of the yeast cell wall, itself, with the use of the Candex product.

Sorry to bore you if you know all this...but it is a concept most people aren't able to visualize. The results or consequences of eating carbs with a Candida overgrowth most definitely can produce enough pressure (gas) or distortion in the abdominal area upward to trigger afib either by pressing on or stretching the vagus nerve connections. (my theory proven over and over in my battle against Candida.) This is probably coupled with the other triggering mechanism of faulty carb (sugar) metabolism and the high insulin response which lowers blood glucose too much which then stimulates the adrenaline rush to counteract the low blood sugar...(you know that syndrome.) I know it well.

While conquering Candida overgrowth may sound overwhelming - the good news is most enlightened cures do not involve eliminating a huge number of foods.... I was not instructed to eliminate any, but from my research, I tried to use common sense about the yeast connection and did cut way back on possible contributors... like substituting lemon for vinegar. I was already abstaining from wine and alcohol (major fuel foods for Candida) and had eliminated carbs such as flour and sugar so my diet was already pretty "pure".

I'm pleased to see Hans include this as a topic for consideration. It's very important for overall health regardless of having afib or not.

Yeast overgrowth is an indicator of the health status of the immune system and we all need to strive for perfection in this area.

As indicated in all the literature, Candida can cause leaky gut syndrome and this leaky gut implication is the source for vast numbers of illness caused by the large protein molecules of undigested foods entering the system and stimulating the production of antibodies.

Great Smokies Diagnostic Laboratories says: "Chronic dysbiosis (altered gut ecology) and inflammation comprise absorption, contributing to deficiencies of nutrients, proteins, carbohydrates and fats.

Chronic maldigestion and gut irritation can lead to a leaky gut and the development of food allergies as well as bacterial or yeast overgrowth and the production of toxins. These toxins can worsen the irritation as well as enter the general circulation, compromising systemic health."

(Such as, systemic symptoms such as joint pain with leaky gut syndrome.)

Treatment portion continues in Part II.

Jackie

Part II My treatment:

First, everyone should take the saliva test and the self quiz Hans offered in the introduction.

PROBIOTICS

It is very important increase the quantity of intestinal friendly flora with probiotics - double or triple your acidophilus intake for a while initially. The only thing it can hurt is your pocket book...and remember all acidophilus products are not created equal. The HealthyTrinity by Natren is considered the very best but no one can afford it.

I like the Acidophilus DDS Plus from UAS Labs.....it needs refrigeration and seems to have the highest potency - at least for me. Also eating some plain unsweetened yogurt might helps with recolonization of healthy bowel flora.
><http://www.uaslabs.com/sections/products.php?category=Probiotic>

TREATMENT PRODUCTS:

The easiest method of killing yeast seems to be the **Candex**.

Check out this web site:

<http://www.pureessencelabs.com/candex.html> One course should do it, but two is good insurance..

You can find it at <http://www.iherb.com/candex2.html> (click on the hyperlink so you can read the label) - this is a good price - I paid \$25 for mine locally at the healthfood store.

The owner there told me Candex is very effective because it actually destroys the cell wall of the yeast. The Candex literature says there is no such die-off reaction.

Directions:

Take 2 capsules one hour before breakfast and two at bed time at least two hour after eating until desired results are achieved... then one capsule at bedtime for maintenance.

Supposedly one bottle will do it, but I'd just order the large economy size of 120 capsules. I would try two regimens of Candex....and then go on a maintenance program.

The back label of the Candex bottle says:

"Normally, beneficial bacteria (probiotics) keep Candida albicans and other yeasts in check in the body. If these bacteria are damaged by antibiotics, birth control pills, chlorinated water, etc such yeasts can grow out of control.

Because the cell walls of yeasts are made of fiber, cellulase and hemi-cellulase enzymes digest them. Candex's other enzymes digest sugars, which are yeast's favorite foods. Used In conjunction with a normal Candida diet, Candex's enzymes quickly reduce yeast levels with none of the die off reaction that is common with anti-fungals. Candex is 100% vegetarian, free of common allergens, and as with all pure Essence products, guaranteed to your complete satisfaction."

Pure Essence Laboratories, Inc. PO Box 95397 Las Vegas NV 89193 1 -888 254 -8000 along with the disclaimer that statements have not been evaluated by the FDA, etc.

Now - as I type this - I see it indicates following a Candida Diet - With the Metagenics, I didn't follow a strict diet, but avoided anything that would contain occult molds or fungi - melons, peanuts, moldy cheeses, etc...along with the carb restrictions I was already on.

The **Metagenics Plan** – is two products (below) taken daily for about 2 months.

These are herbal ingredients:

CANDIBACTIN - AR

Red Thyme Oil .2 ml
Oregano oil 0.1 ml
Sage leaf 75 mg
Lemon Balm Leaf 50 mg.
One soft gel 3 times daily before or with meals. 60 capsules

and the second product:

CANDIBACTIN - BR

Coptis Root & Rhizome extract 30 mg.
Indian Barberry
Root extract 70 mg
Berberine Sulfate 400 mg
and a 4:1 Proprietary extract 300mg.
Consisting of Coptis Root, Chinese Skullcap, Phellodendron Bark, Ginger
Rhizome, Chinese Licorice Root, ChineseRhubarb root and Rhizome.
One to 2 tablets 2 - 3 times a day 90/bottle

When I found the Candex, I was pleased to know it was far less expensive and faster treatment as well...the big plus being no die-off rash.

My experience is that I am Candida free for the past several years...first the MG treatment and One full bottle of Candex and now just maintenance.

A third option is a common product although not well known – called GSE – or **Grapefruit Seed Extract** – also known as **Citricidal**® Very economical. Said to be effective but I have no experience with it other than for killing colds and flu at the inception stage. Very effective for killing food-born pathogens. sanitizing countertops, etc.

There are two forms. One is a viscous liquid extract – very bitter – which is diluted and consumed. It kills all pathogens in its path and is especially effective for Traveler's Diarrhea and for parasites. The liquid lasts a very long time.

The other form is capsules; also includes some herbals. Check it out at <http://www.gfex.com/> and <http://www.iherb.com/gseliquid.html>

Follow the instructions – take for a month or six weeks daily and then retest using the saliva test.

The End.

Jackie

Thank you Fran and Jackie for your comments and explanations. The bread making process was a good way of looking at Candida and was a scary thought, that this sponge with tentacles is lurking within, producing acetaldehydes. There must have been something to the elimination of sugars, dairy, and wheat, and the connection to Candida, for me to get rid of my gas, bloating, and indigestion on the second day of the Paleo diet. I'll check out Candex, as I still have some Candida, because of results from my stool analysis.

Jackie, how long do you think you had Candida, and how long had it been since you had gotten rid of it, before your

ablation? How are you still doing, post ablation? Did you ever know your Mo levels?

Fran, are the soils in your area acid or alkaline? It was stated in the above link on "everything you wanted to know about Mo" that it isn't as prevalent in acid soils, which is what we have in our area. That was on one of the links at the top of that linked page. There was also mention of Mo being very similar to vanadium and that Mo helps with absorption of iodine. I wonder if there is some correlation to Glu, Mo, and candida.

Thanks again for your enlightening posts.

Richard

Here's some sites I have found that may be of interest:

This one is a lot of different links and info on Candida:

<http://www.CandidaPage.com>

This site is everything you wanted to know about molybdenum:

http://www.imoa.info/technical/envt_database/3_BIOCHEMpchm.HTM

Not that this is pertinent, but I found it of interest, that phenylketonuria children had 18xs the Mo levels than normal infants being breastfed. The only thing that I knew about this genetic defect was that they could not breakdown Phe to tyrosine, so it would seem that Mo has something to do with this enzymatic pathway that they were not utilizing. I know my Phe levels were normal, but tyrosine fell below normal. Another thing I found of importance was that the adrenals have the most content of Mo. I lost my link, but 20-30% of the population is deficient in the sulfite oxidase enzyme.

Hans,

You mentioned you were low in Cu and normal in Mo, and if I recollect you were having some beginning success with Cu. How long did you take, before quitting, and did you go back to status quo before quitting? Also, you stated that you have had Candida from time to time. How did it present itself? Have you ever had problems with sulfites in wine? What have been your known triggers, besides stress and exercise, if any? Have you ever had indigestion problems? Have you suffered from headaches? Any other ailments you have had over time would be nice to know, as well. Do you take any sulfur aminos, and if so, which ones, and how much daily? The more I know about you, the more I'll know what to look for when studying. Thank you for letting me be nosey.

Richard

If you all want to see the effects of acetaldehydes, pg. 6 of this link is very informative. You may want to print out, as it's a sideways diagram. Keep in mind that I was low glutathione, of which acetaldehydes bind to this. Acetaldehydes are also suppose to be converted in the mitochondria to acetates and NADH through the aldehyde dihydrogenase enzyme that is reliant on Mo. I think I'm clear on this. I'm still learning. Could we be served by taking NADH as a supplement? Joe South believes so. Remember tryptophan makes niacin (NADH) and I'm not clear on the conversion of acetaldehydes to NADH and how tryp. plays into this, but if we're high in acetaldehydes, due to alcohol or Candida's byproducts, then maybe it's using up tryp. more than we know. I'm thinking out loud, and hoping I spur some additional thoughts.

<http://web.indstate.edu/thcme/mwking/ethanolmetabolism.pdf>

Richard

This discussion is very thought-provoking. Regarding the alcohol, acetaldehyde and tryptophan connection, it occurs to me that SSRI drugs, used to treat depression and anxiety, are labeled with the warning to avoid alcohol. Alcohol

depletes serotonin. Tryptophan is a serotonin building-block.

When I took an SSRI (prozac) for anxiety-related problems, I had no problems with LAF, even though I often would use alcohol. Alcohol is a critical trigger for me.

I am about to add molybdenum supplements to my diet and am considering a candida-elimination regime.

Although I have experienced no significant episodes of LAF in the past 11 months, since undertaking a mineral-supplement program, I know that I am still vulnerable. For example, on a recent night I had a late meal which included lots of free glutamate in some delicious sushi. My supplement dosage was a little low that day (400mg rather than 600 to 800mg). Shortly after going to bed, I had a brief of rapid, irregular heartbeat (a few minutes only). I got up from bed and the irregularity stopped and a significant episode was avoided. I understand well that I am still vulnerable.

Michael in SF

Richard,

You are correct that I thought I was having some initial success with copper supplementation. However, I later realized that the temporary decrease in PVCs that I experienced was the usual (for me anyway) that follows the end of an episode and the start of a new cycle. I stayed with the copper for about a month before I decided that it did not have the intended effect, ie. a consistent reduction in PVCs.

I have had candida overgrowth on and off for many years. Once I was diagnosed with oral thrush but otherwise, bloating, gas and digestive problems has been the main manifestation. Excessive candida also showed up in my Great Smokies comprehensive stool test.

I have had problems with alcohol as a trigger, but don't believe it was due to sulfites. Mental stress, physical stress, heat, dental work and high tyramine meals have been my main triggers. Never suffered from headaches. No major ailments other than common childhood diseases, polio and the odd bout of prostatitis - probably candida-related. I do not supplement with amino acids, but get a fair amount of protein in my diet. Hope this helps!

Hans

Hans and Michael,

Hans, I hope you came through polio unscathed. Have you ever thought there to be a correlation with your AF, as we probably have all had the polio vaccine? I find it very interesting that Michael found he could drink alcohol when on Prozac, but alcohol was a major trigger otherwise. By prolonging serotonin at the synaptic cleft, this may have left more tryptophan available for production of niacin or lack of tryptophan/serotonin is your problem. Mg and B6 are needed for metabolism of tryptophan to serotonin, and I know the Mg has helped you.

Now for what I have found, at least, as it pertains to me. I was low in folate and B12 based on the finding that I was extremely high in formylglutamate acid (FiGlu) on my metabolic analysis. FiGlu is an intermediate in the histidine to glutamic acid pathway and is elevated if tetrahydrofolate is deficient. Additionally, if folic acid dysfunction affects the metabolism of methionine, causing homocystinuria (I don't have), many diverse disease conditions could be related to this dysfunction. Such conditions include mood disorders, neurological problems, and diseases associated with vit. B12 disorder, because B12 activity depends upon folate function in many instances. Strangely, however, I was high in folate on my Comprehensive Vitamin Profile. The range was 5.0-24.0 ng/ml and my result was 56.0. I showed extremely low in B6; ref range 30-80 ng/ml, result 9. The B6 doesn't play into this scenario.

So, my thinking was, from reading, that I possibly had more of a problem with B12, since my levels of folate were high, and they work synergistically together, at least, that's what I gather. I must add that I should not be low in folate and B12, with all the organic red meat and vegetables that eat. So in further reading, I found that a B12 deficiency is not common and could be due to purine metabolism.

Nitrogen Bases

There are two kinds of nitrogen-containing bases - purines and pyrimidines. Purines consist of a six-membered and a five-membered nitrogen-containing ring, fused together. Pyrimidines have only a six-membered nitrogen-containing ring. There are 4 purines and 4 pyrimidines that are of concern to us.

Purines

Adenine = 6-amino purine

Guanine = 2-amino-6-oxy purine

Hypoxanthine = 6-oxy purine

Xanthine = 2,6-dioxy purine

<http://www-medlib.med.utah.edu/NetBiochem/pupyr/pp.htm>

The major site of purine synthesis is in the liver. Synthesis of the purine nucleotides begins with PRPP and leads to the first fully formed nucleotide, inosine 5'-monophosphate (IMP). This pathway is diagrammed below. The purine base without the attached ribose moiety is hypoxanthine. The purine base is built upon the ribose by several amidotransferase and formylation reactions. The synthesis of IMP requires five moles of ATP, two moles of glutamine, one mole of glycine, one mole of CO₂, one mole of aspartate and two moles of formate. The formyl moieties are carried on tetrahydrofolate (THF) in the form of N⁵,N¹⁰-methenyl-THF and N¹⁰-formyl-THF. IMP represents a branch point for purine biosynthesis, because it can be converted into either AMP or GMP through two distinct reaction pathways. The pathway leading to AMP requires energy in the form of GTP; that leading to GMP requires energy in the form of ATP. The utilization of GTP in the pathway to AMP synthesis allows the cell to control the proportions of AMP and GMP to near equivalence. The accumulation of excess GTP will lead to accelerated AMP synthesis from IMP instead, at the expense of GMP synthesis. Conversely, since the conversion of IMP to GMP requires ATP, the accumulation of excess ATP leads to accelerated synthesis of GMP over that of AMP.

Catabolism and salvage of purine nucleotides:

adenine + PRPP <-----> AMP + PPi

and hypoxanthine-guanine phosphoribosyltransferase (HGPRT), which catalyzes the following reactions:

hypoxanthine + PRPP <-----> IMP + PPi

guanine + PRPP <-----> GMP + PPi

<http://web.indstate.edu/thcme/mwking/nucleotide-metabolism.html#purine>

The liver can store up to six years worth of vitamin B₁₂, hence deficiencies in this vitamin are rare. Pernicious anemia is a megaloblastic anemia resulting from vitamin B₁₂ deficiency that develops as a result a lack of intrinsic factor in the stomach leading to malabsorption of the vitamin. The anemia results from impaired DNA synthesis due to a block in purine and thymidine biosynthesis. The block in nucleotide biosynthesis is a consequence of the effect of vitamin B₁₂ on folate metabolism. When vitamin B₁₂ is deficient essentially all of the folate becomes trapped as the N⁵-methylTHF derivative as a result of the loss of functional methionine synthase. This trapping prevents the synthesis of other THF derivatives required for the purine and thymidine nucleotide biosynthesis pathways.

Neurological complications also are associated with vitamin B₁₂ deficiency and result from a progressive demyelination of nerve cells. The demyelination is thought to result from the increase in methylmalonyl-CoA that result from vitamin B₁₂ deficiency. Methylmalonyl-CoA is a competitive inhibitor of malonyl-CoA in fatty acid biosynthesis as well as being able to substitute for malonyl-CoA in any fatty acid biosynthesis that may occur. Since the myelin sheath is in continual flux the methylmalonyl-CoA-induced inhibition of fatty acid synthesis results in the eventual destruction of the sheath. The incorporation methylmalonyl-CoA into fatty acid biosynthesis results in branched-chain fatty acids being produced that may severely alter the architecture of the normal membrane structure of nerve cells.

<http://web.indstate.edu/thcme/mwking/vitamins.html#12clinical>

The hypoxanthine and xanthine oxidase enzyme is supposedly important for the purine cycle, and it is these enzymes, at least the latter, that are dependent on molybdenum (Mo), which I found very interesting, because all of this ties in with my situation. I was low in methionine, low in B₁₂, supposedly low in folate, but it was probably trapped, and low in Mo. I have Candida, which produces acetaldehydes, as does alcohol, and uses the aldehyde oxidase enzyme according to Dr. Orion Truss, to break this down to acetic acid. Am I using up my stores of Mo, fighting the effects of Candida, as Mo deficiency is very rare. On the other hand, read on:

PABA

Technical Background

PABA (para-amino-butyric acid) is a naturally-occurring water-soluble compound, and a constituent of folic acid. PABA is an important growth factor for certain intestinal bacteria, where it is a precursor of folic acid synthesis.1 PABA is an antioxidant which reacts rapidly with singlet molecular oxygen (1O_2), a free-radical.2 PABA can also be used topically as a sun blocking agent.

http://www.usana-nutritionals.com/research/USNUSUPPINGREDIE_19447.html

Vitiligo Research Diet & Lifestyle

Vitamin B & Vitiligo (Fran has this)

In January 1945 a study appeared in the Virginia Medical Monthly suggesting that vitamin B PABA may be effective in treating vitiligo.

<http://www.internethealthlibrary.com/Health-problems/Vitiligo%20-%20researchDiet&Lifestyle.htm>

PABA is a naturally-occurring, water-soluble compound which is found in many foods as a cofactor of the vitamin B complex (associated with folate). It first became popular due to the writings of pioneer nutritionists like Gaylord Hauser, Lelord Kordell, and Adelle Davis. Several decades later, life extension scientists Durk Pearson and Sandy Shaw extolled the potential virtues of PABA in their best-seller, Life Extension-A Practical Scientific Approach. Pearson and Shaw described PABA as an antioxidant B vitamin which could: (1) slow cross-linking; (2) enhance flexibility; (3) promote membrane fluidity; (4) provide protection against ozone, secondhand smoke and other air pollutants; (5) alleviate the inflammation of arthritis; and (6) restore the original color of hair in perhaps 10-25% of cases. Pearson and Shaw reported they consumed as much as three grams of PABA per day. (The hair color issue pertains to Joe, and maybe others).

<http://www.vrp.com/articles/607.asp>

Functions: Para-aminobenzoic acid, as part of the coenzyme tetrahydrofolic acid, aids in the metabolism and utilization of amino acids and is also supportive of blood cells, particularly the red blood cells. PABA supports folic acid production by the intestinal bacteria. PABA is important to skin, hair pigment, and intestinal health. Used as a sunscreen, it also can protect against the development of sunburn and skin cancer from excess ultraviolet light exposure.

<http://www.healthy.net/asp/templates/Article.asp?PageType=Article&Id=2132>

The metabolism of ethanol within the body has been investigated and identification of the pathway is useful for the purposes of further investigation. After ethanol is absorbed into the cells, it is converted into ACA (acetaldehydes) by ADH (alcohol dehydrogenase) in the cytosol. The ACA that is present is further oxidized into acetate by ALDH within the mitochondrial matrix. Both of these oxidation reactions yield the production of one equivalent NADH (Fig. 1).

The above pathway results in the production of free radicals predicated upon changes in NADH amounts and NADH/NAD⁺ redox ratios (37). These increases alter the activity of xanthine oxidase, which generates free radicals.

<http://www.biologicalprocedures.com/bpo/arts/1/41/m41.htm>

Ethanol is quickly absorbed into the bloodstream and reaches the brain. As a small molecule, it is able to cross the blood-brain barrier. The euphorizing effects of ethanol are probably due to its causing the release of endorphins, natural "feel-good" molecules.

The depressing effect is mostly due to ethanol's acting on the GABA receptors. GABA is an inhibitory neurotransmitter, meaning it acts to slow down or inhibit nerve impulses. Ethanol increases the effectiveness of the GABA receptors. When used over a long time, ethanol changes the number and type of GABA receptors, and this is thought to be the cause of the violent withdrawal effects of alcoholics.

Ethanol also interferes with synaptic firing and causes the death of brain cells. This cell death is caused by an increased concentration of intracellular calcium which weakens the electrochemical gradient across the cell

membranes. It is this gradient which is the motive force of membrane pumps and channels (cells, especially neurons, quickly die without proper membrane pump and channel function). There is also direct damage to cell membranes from free-radicals that are produced from alcohol metabolism.

<http://en2.wikipedia.org/wiki/Ethanol>

Adenosine deaminase shows not only polymorphism but also deficiency. ADA deficiency causes a form of severe combined immunodeficiency disease (SCID) in which there is dysfunction of both B and T lymphocytes with impaired cellular immunity and decreased production of immunoglobulins. Multiple forms of SCID exist; see Swiss type of agammaglobulinemia (202500, 300400), nucleoside phosphorylase (164050), and transcobalamin II deficiency (275350).

Giblett et al. (1972) described 2 girls in separate families with impaired cellular immunity and absent red cell adenosine deaminase (my note: this is the purine cycle). One child, aged 22 months, showed recurrent respiratory infections, candidiasis, and marked lymphopenia from birth. The other, aged 3.5 years, was allegedly normal in the first 2 years of life. Mild upper respiratory infections began at age 24 months and progressed to severe pulmonary insufficiency and hepatosplenomegaly by age 30 months. The parents of the first child were related and the second child had a sister who died in consequence of a major immunologic defect (Hong et al., 1970). The finding that both pairs of parents had an intermediate level of red cell ADA supported recessive inheritance. Possibly a different allele is present in the 2 families because in the first family the parents showed about a 50% level of ADA whereas it was about two-thirds normal in the second pair.

<http://www3.ncbi.nlm.nih.gov/htbin-post/Omim/dispim?102700>

On the above links, I'm not trying to propose that we have a polymorphism, only represent what can happen in deficiencies.

This is important because administration of folic acid can "mask" vitamin B12 deficiency in patients with pernicious anaemia (discussed later). Just more information on folate.

<http://www.pharmj.com/Editorial/19991023/articles/folicacid.html>

Incorporation of [¹⁴C]hypoxanthine into cardiac adenine nucleotides: effect of aging and post-ischemic reperfusion.

http://www.arclab.org/medlineupdates/abstract_8422432.html

NITROGEN METABOLISM

http://www.geocities.com/doctor_uae/nitrogen.htm#purines

Lesch-Nyhan Syndrome

Lesch-Nyhan syndrome (Online Mendelian Inheritance in Man [OMIM1,2] #300322) is an X-linked recessive disorder that results from the deficiency of hypoxanthine-guanine phosphoribosyltransferase (HPRT1) in the purine salvage pathway (Figure 1). The degree of neurological involvement is dependent on the amount of residual HPRT enzyme activity present, but most persons with classical Lesch-Nyhan syndrome have less than 1.5% HPRT activity (Jinnah and Friedmann 2001). Classical HPRT deficiency results in overproduction of uric acid, debilitating neurological disability (choreic and athetoid movements, dysarthric speech, hyperreflexia, and hypertonia), varying degrees of cognitive disability, and behavioral abnormalities that often include impulsive and self-injurious behaviors (Jinnah and Friedmann 2001). In addition, megaloblastic anemia, growth retardation, and unexplained fevers are often reported in these individuals (Jinnah and Friedmann 2001). Diminished dopamine levels in the basal ganglia (60-90% depletion) and cerebrospinal fluid have also been reported throughout patients' lives, suggesting that dopamine deficiency may account for some neurological symptoms of the disorder (Jinnah and Friedmann 2001). The mechanism by which features of Lesch-Nyhan syndrome result from impaired purine metabolism is still not well understood.

http://dels.nas.edu/ilar/jour_online/43_2/Mousetrap.asp

Aging-related changes in myocardial purine metabolism and ischemic tolerance.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=14580870&dopt=Abstract

Variation in mitogenic response of cardiac and pulmonary fibroblasts to cerium.

The oxygen radical generator (hypoxanthine + xanthine oxidase [Hyp. + XO]) induced a proliferative response that was

neutralized in both cardiac and lung fibroblasts by free-radical scavengers.
This seems to indicate that Hyp/Xo create free radicals. I'm getting more confused.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12972691&dopt=Abstract

Allopurinol improves cardiac dysfunction after ischemia-reperfusion via reduction of oxidative stress in isolated perfused rat hearts. (as it pertains to xanthine oxidase)

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12939555&dopt=Abstract

Reperfusion arrhythmias and purine wash-out in isolated rat and rabbit heart. Effect of allopurinol, dimethylthiourea and calcium reduction.

We speculate that: (1) calcium mediated arrhythmogenic mechanism is operating both in reperfused rat and rabbit heart; (2) free radical mediated mechanism is of an importance only in rat heart; (3) neither a decreased free radical production secondary to xanthine oxidase inhibition nor the augmentation of adenosine wash-out is a likely explanation for the antiarrhythmic effect of allopurinol in reperfused hearts; and (4) high level of myocardial adenosine accumulation during ischemia, probably secondary to low xanthine oxidase activity, may play a role of a natural defence mechanism in ischemic/reperfused rabbit heart.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=8230246&dopt=Abstract

I'll try to add more later, as I have to digest all this info. further, and my family is saying I've been on the computer long enough. All day, to be exact. I just had so much saved, I had to empty the bar at the bottom. Does anyone take PABA? That was not indicated on any of my test.

Richard

Just another tantalizing bit of info:

24. Para amino benzoic acid (PABA) is a substrate in bacteria for an Enzyme that initiates a pathway that produces Folic Acid. Humans do not have this pathway & require folic acid as a vitamin. A common strategy for pharmaceutical companies to develop new drugs is to understand the structure and interactions of S with E, and synthesize substrate analogs that will bind to E more tightly than S and block the active site.

25. Sulfa drugs (e.g. sulfanilamide) are similar to the substrate, para-amino benzoic acid (PABA). As such they act as competitive inhibitors of this process and in large enough doses can kill bacteria.

www.css.edu/USERS/pstein/CHM3240/notes3240-05.doc

Richard

Richard

The soils in my area are acid. Very acid - considering the peat bogs, sphagnum moss etc that surrounds the shell sand limered, the slag to alkalise and seaweed fertilised fields. But we also have pockets of lime and volcanic soils. Talk about a mix - but it is a geologists dream area with people coming from all over the world for the oldest sedimentary rock formations in the world.

However, most of the veggies and fruit I eat is not grown in this area but in the fertile Black Isle where the soils are very different from here and a lot more alkaline. A lot of the meat I eat is grown here, but some of it is wintered on the Black Isle and some from the Mo rich area of Ballachulish and the slate quarries.

Sorry nothing to add but feel that James South knows what he is talking about.

Fran

Conjugation: Acetylation, Methylation

The body's ability to methylate organic compounds was discovered by German physician Wilhelm His in 1887, when he isolated N-methyl pyridinium hydroxide from the urine of dogs dosed with pyridine acetate. He studied metabolism with Schmiedeberg in Strassburg and went on to become director of the first medical clinic in Berlin. A musician and painter, His also pioneered studies in cardiac conduction and campaigned to promote disclosure of the composition of proprietary drugs.

In 1954, MacLagan and Wilkinson first described methylation of oxygen in their studies of potential anti-thyroid compounds. The significance of their discovery in physiology was uncovered three years later when Axelrod, as well as Armstrong and McMillan, described the O-methylation of adrenaline and noradrenaline. The following year Sarcione and Sokal described S-methylation in their study of thiouracil metabolism in the rat.

Acetylation was first described by Jaffe and Cohn when they observed a most unusual urinary product following furfural administration to dogs and rabbits. The product, furfuracrylic acid, presumably arose from condensation of the aldehyde with the methyl carbon of acetic acid.

Cohn's interest in finding further examples of this type of conjugation led him to administer m-nitrobenzaldehyde to dogs, wherein he found m-nitrohippuric acid as a urinary product. Fortunately for his career, he also tried rabbits—and this urine yielded N-acetyl aminobenzoic acid, the first N-acetylated metabolite.

More than 50 years later, F. Lipmann studied the acetylation of sulfanilamides and unraveled the role of coenzyme A in acetylation reactions. His work led to a Nobel Prize in 1953.

<http://www.bioanalytical.com/info/calendar/2000/acet.html>

I don't know if I'm jumping the gun, but I think my problem could be solved with Para-amino benzoic acid. This is all falling together in what I see my deficiencies to be. I'm really excited about what I have found, and the above link is very interesting, if you click on the different studies at the bottom. If you look at the formula, you'll see mandelic acid, of which I was also low in. I have to say, I'm blown away today, but maybe tomorrow I'll feel like an idiot, but it won't be the first time, nor the last. I'm off to understand what Prontosil is, other than an anti-bacterial.

Richard

And then there's more:

The effect of para-amino benzoic acid (PABA) on the virus of Herpes simplex (VHS-1, strain L2) was studied and it was shown to be active in vitro and in vivo. The action of PABA was virucidal in the culture of the cell-free virus-containing material. It lowered the death rate of the laboratory mice with experimental herpetic encephalitis (intraperitoneal contamination) at the average by 40 per cent and increased the mean life-span of the animals significantly decreasing the virus titre in the mouse brain. PABA was not toxic with respect to the Vero cells thus not preventing the virus-induced cytopathic effect in the cultures. However, PABA showed high ability to potentiate the antiherpetic action of acyclovir (Zovirax, acycloguanosine) in the infected cultures when acyclovir was used in inactive concentrations.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=8660116&dopt=Abstract

alpha-Aminoacid derivatives of p-aminobenzoic acid with a potential antiarrhythmic activity.

Department of Pharmaceutical Chemistry, Institute of Drug Sciences, Faculty of Pharmacy, School of Medicine, 1 Banacha Str., 02-097 Warsaw, Poland.

Physico-chemical properties as well as two methods of synthesis of the title compounds have been described. The structures of new compounds (obtained as HCl or HBr salts) have been confirmed by elemental and spectral analysis (IR and ¹H-NMR). The compounds should display a potential antiarrhythmic activity, as structural analogs of Procainamide.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12959259&dopt=Abstract

OBJECTIVE: The aim of this study is to examine effects of para-aminobenzoic acid (PABA) on the growth of *Lactobacillus acidophilus* (*L. acidophilus*). **METHODS:** Different concentrations of PABA (10(-10)-10(-3) g/L) were separately transferred to the modified Carlsson medium. *L. acidophilus* (ATCC4356) grew in these Carlsson media. All cultures were incubated at 37 degrees C anaerobically in atmosphere of 80% of nitrogen, 10% of hydrogen, and 10% of carbon dioxide for 48 hours. Absorbance values ($\lambda = 540$ nm) of bacterial suspensions were measured using a spectrometer (UV-1601). Colony forming units (CFU) were obtained by growing *L. acidophilus* in Carlsson media with different concentration of PABA (10(-10)-10(-3) g/L). **RESULTS:** Different concentrations of PABA (10(-10)-10(-4) g/L) had different stimulating effects on the growth of *L. acidophilus* ($P < 0.05$). But stimulating effects declined, when PABA concentration was 10(-5) g/L, and when the concentration of PABA reached 10(-3) g/L, the stimulating effect disappeared. **CONCLUSION:** This study indicates PABA stimulates the growth of *L. acidophilus*, and PABA can promote growth of *L. acidophilus*.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12539490&dopt=Abstract

Drug evolution: p-aminobenzoic acid as a building block.

Kluczyk A, Popek T, Kiyota T, de Macedo P, Stefanowicz P, Lazar C, Konishi Y.

Biotechnology Research Institute, 6100 Royalmount Avenue, Montreal, Quebec, Canada H4P 2R2.

The core or the building block is an important component in drug development. In this article, we propose and review p-aminobenzoic acid (PABA) as a building block used in the design of drugs or drug candidates. PABA is frequently found as a structure moiety in drugs. For example, in a database of 12,111 commercial drugs, 1.5% (184 drugs) were found to contain the PABA moiety. These drugs have a wide range of therapeutic uses, such as: sun-screening, antibacterial, antineoplastic, local anesthetic, anticonvulsant, anti-arrhythmic, anti-emetic, gastrokinetic, antipsychotic, neuroleptic, and migraine prophylactic. This article reviews the molecular targets and the mechanisms of these activities. Drugs containing PABA also show a wide range of structural diversity. Of the 184 PABA containing drugs identified, 95 different substitutions were found at the carboxylic group and 61 were found at the amino group of the building block. Substitution on the aromatic ring was also diverse. 13, 3, and 13 different side chains were found to modify positions 2, 3 and 5 of the aromatic ring respectively. In some drugs, the amino group is further substituted to form tertiary amine (4 different side chains). Substitutions at the carboxyl and amino groups of PABA are particularly suitable for the generation of combinatorial libraries. Just by reshuffling the identified side chains of the 184 PABA containing drugs, 4.5 million compounds can be generated. Consequently, PABA fits well as a building block for a general chemical library of "drug-like" molecules with a wide range of functional and structural diversity.

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=12369873&dopt=Abstract

Mechanism for Fungal Adherence Found, Possible Key to Disease

http://www.nidcr.nih.gov/news/digest/archives/may99_2.asp

PABA

It is well known that PABA absorbs dangerous ultraviolet B (UVB) radiation. However, PABA also protects against ultraviolet A (UVA) damage via a unique free radical-scavenging ability. Thus, PABA is useful in protecting against photocarcinogenesis by wide-range UV radiation. PABA also inhibits the formation of thrombin-induced thromboxane (a potent inducer of platelet aggregation and a constrictor of arterial smooth muscle) in human platelets, and thus reduces the risk of abnormal blood clot formation, blocking the flow of blood (thrombosis).

http://www.lef.org/magazine/mag98/sep98_newlem2.html

Richard

This may tie into some of the symptoms people suffer from, as well as pertaining to the formation of aldehydes:

Amines

Cytochrome P450 and other oxidizing enzymes also oxidize amines such as phenylethylamine found in chocolate, tyramine found in cheese, and adrenaline, noradrenaline and dopamine. These are oxidized into aldehydes by the enzyme mitochondrial monoamine oxidase (MAO) – if this enzyme is blocked, for instance by MAO inhibitor drugs used to treat depression, tyramine, for instance, cannot be metabolized and hypertension can develop as a chemical sensitivity reaction.

Phase II detoxification (conjugation) There are five main conjugation categories, including acetylation, acylation (peptide conjugation with amino acids), sulphur conjugations, methylations and conjugation with glucuronic acid. Some substances enter Phase II detoxification directly, others come via Phase I pathways.

Conjugation involves the combining of a metabolite or toxin with another substance which adds a hydrophilic (or water-reactive) molecule to it, converting lipophilic (or fat-reactive) substances to water-soluble forms for excretion and elimination. Individual xenobiotics and metabolites usually follow a specific path, so whereas caffeine is metabolized by P450 enzymes, aspirin-based medications are conjugated with glycine, and paracetamol with sulphate.

Aldehydes

Substances which can inhibit the action of P450 enzymes include carbon tetrachloride, carbon monoxide, barbiturates, quercetin and naringenin (found in grapefruit). The oxidation reaction can also be blocked by an excess of toxic chemicals, a lack of enzymes, lack of nutrients and/or loss of oxygen.

Such blocking results in a build-up of more toxic substances such as formaldehyde and other aldehydes in tissue. This can in turn lead to a spreading phenomenon, with increasing sensitivity to more chemicals such as ketones and alcohols, and eventually even to natural chemicals occurring in foods, pollen and mould. A build-up of aldehydes can in severe cases lead to tissue cross-linking, causing vasculitis with possible seizures and brain damage.

Although most aldehydes in the body are thought to occur as intermediate metabolites, external sources include exposure to formaldehyde gas (which is given off by new carpets, curtains and other furnishings) and breakdown products of ethylene glycol and methanol.

Two known sources of aldehydes are intestinal overgrowth with *Candida albicans*, as well as the peroxidation of polyunsaturated fats. The fatigue, foggy thinking and 'brain fog' linked with candidiasis may be due to an overloading of the detoxification system with aldehydes, which can even lead to a reverse reaction of aldehyde to alcohol. Extreme intolerance to alcohol consumption may occur in these individuals, as it does in those diagnosed with ME or chronic fatigue syndrome.

I didn't post all the info., so link and read in its entirety, if you so desire.

<http://www.garynull.com/Documents/Continuum/LiverOfTheMatter.htm>

Besides eliminating candida, we may really need to be focusing on our liver. Maybe it's the real "heart" of the matter. I'm also going to post this on the BB, as I feel that many will miss it.

Richard

Richard - I was unaware of the Candida problem....only after the diagnosis was I aware. So who knows how long I had it? I was rid of it several years ago. Took about 3 months to kill. I also had fibromyalgia but got over that long before I was diagnosed with Candida and I don't think there was any association....but not sure.

I'm doing well after ablation, thanks for asking. 22 days today.

Jackie

Detoxification of neurotoxins requires that the cell membrane is nourished with balanced essential fatty acids (4:1, plus HUFAs) and supportive phospholipids. Phosphatidylcholine (PC) is the most abundant phospholipid of the cell membrane and protects the liver, with its 33,000 square meters of membrane, against toxicity and infection. The liver plays a pivotal role in detoxification but due to its fatty acid content and the lipid soluble characteristics of neurotoxins, lipid based interventions are required to impact toxic burdens. Once the liver has been damaged it can no longer metabolize fats normally. Pools of lipids are then deposited within hepatocytes throughout the liver. Beta oxidation of fatty acids is suppressed impairing detoxification and prostaglandin production. Extensive research with PC has revealed that it protects the liver against damage from alcohol, pharmaceuticals, environmental pollutants, xenobiotics and infection due to viral, bacterial and fungal manifestations (Lieber 1994a, 1994b, 1995, 2001a, 2001b).

<http://www.woodmed.com/Phos%20Choline.htm>

Richard

Here's some interesting articles, and on link sends free info on how to rid oneself of Candida.

Most suddenly occurring heart attacks are caused by Drug Resistant Candida, moving into the blood stream from an infected tooth. Cancers, tumors, and many of the "gene-related diseases" are caused by the DNA modules (i.e., plasmid DNA) that has become active after being passed into a given cell, from an unseen, drug-resistant bacteria. These findings were achieved by way of an extensive, epistemological study.

<http://www.prweb.com/releases/2003/11/inktom87793.php>

<http://www.candidayeastinfections.com/program.htm>

Richard

I fall into several categories of Fibromyalgia; chemical sensitivities (MSG/organophosphates/paint fumes that caused me to go out of rhythm recently), headaches, disequilibrium (motion sickness), some cognitive disorders, and indigestion (pre-changing diet). I do not experience the pain, however.

Here's an excerpt about a possible connection for Fibro and Candida:

As previously mentioned, many people with fibromyalgia have also been diagnosed with Candida and claim they have seen improvements after starting on the Candida Control Diet since candida feeds on sugar. Studies have shown that sugar will deplete energy. Therefore eliminating it from your body would help to eliminate the chronic fatigue that is associated with Fibromyalgia.[27] Candida antibody blood test, comprehensive stool analysis and allergy testing are tests available to detect candida. (link to diet and KD2) Should candida presence be detected, a product called KD2 would be recommended for 3 months prior to starting on the Fibrex. Please keep in mind candida can be sexually transmitted so partners are encouraged to be treated for a minimum of 9 months to insure there will be no reoccurrence.

<http://www.fibrofacts.com/research.asp>

Hans, do you know anything about Malic acid?

<http://www.fibroactive.com/research.htm>

<http://www.vrp.com/articles/659.asp>

Richard

Conditions That Have Reportedly Responded to MSM Supplements

Acne

Allergies
Arthritis
Asthma
Candida Yeast Infections
Carpal Tunnel Syndrome
Chronic Fatigue
Constipation
Diabetes
Digestive Disorders
Fragile Hair & Nails
Migraine Headaches
Muscle Pain & Cramps
Parasites
Skin Damage & Aging
Toxic Build-up
Ulcers

<http://www.all-natural.com/msm.html>

Richard

It's a bit late, but I happened on this late tonight, and wanted to share. It's quite interesting.

This is the first link I happened on:

<http://askwaltstollmd.com/archives/lgs/202100.html>

Now read this one:

<http://www.essiac-info.org/BobK/article3.html>

Have you heard of this and what do you think?

Richard

Richard,

Thank you for sharing these links. They are indeed very interesting! MGN-3 sounds like it might be worth trying for candida. I did try Moducare, which has a very similar benefit. It kept me afib-free for 2 months (a record for me), but then it seemed to lose its effect so I discontinued it. Maybe I will try it again.

Hans

Just a few more studies of interest:

Binding of Candida albicans to immobilized amino acids and bovine serum albumin

<http://iai.asm.org/cgi/content/abstract/66/1/140>

Gastrointestinal colonization and systemic dissemination by Candida albicans and Candida tropicalis in intact and immunocompromised mice

http://iai.asm.org/cgi/content/abstract/60/11/4907?maxtoshow=&HITS=10&hits=10&RESULTFORMAT=&titleabstract=Candida&searchid=1071075985741_1696&stored_search=&FIRSTINDEX=30&journalcode=iai

Parenteral Administration of Medium- but Not Long-Chain Lipid Emulsions May Increase the Risk for Infections by *Candida albicans*
http://iai.asm.org/cgi/content/abstract/70/11/6471?maxtoshow=&HITS=10&hits=10&RESULTFORMAT=&titleabstract=Candida&searchid=1071076129171_1707&stored_search=&FIRSTINDEX=30&journalcode=iai

Richard

This will take some time to get through, but this is a compilation of research done on astrocytes, and how they relate to *Candida*. It's quite interesting, but I haven't read through all of it.

Here's some excerpts:

Candida has mannose-related molecules as structural components [a].

Astrocytes have mannose receptors, and the number of these receptors increases in the presence of Th2-type cytokines patterns [b], which is the pattern underlying persistent *Candida* infections [c].

Astrocytes can become activated and then release a variety of cytokines, and can participate in neuronal degeneration [d], and these effects have a sex difference via IL-1Ra [d].

Astrocytes produce neuropeptides and have a number of receptors [e], perhaps including secretin receptors (but this last part needs to be verified) -- which is why, for reasons outlined below, secretin infusions may improve function rapidly.

A defect in astrocyte function seems capable of inducing neuronal hypofunction; astrocytes participate in glucose and glutamate metabolism and can be affected by viral and other infections; [g] (These studies under the letter g, are quite interesting).

Astrocyte degeneration can include demyelination [h]; because of this, processes that induce astrocyte demyelination may underlie the elevated anti-MBP antibodies titres found in many autism-spectrum children. And this process may be T-cell mediated and exacerbated by peripheral T-cells that cross the bbb [i].

This aspect of persistent *Candida* infections would accompany *Candida*'s other neurologic effects as described by Bill Shaw (1998-1999) and, in many or most kids with persistent *Candida*, would occur in a context of altered immunity biased in a Th2 direction, along with the other ramifications of altered intestinal permeability (food allergies, autoimmune lesions of intestinal tissue, the production of neuroactive but unwanted chemicals).

Astrocytes store antioxidants and also store glutathione [L] and can contribute to epileptogenesis [m], and are affected by thyroid function [n], and play a role in protecting against heavy metal toxicity [o]

<http://www.jorsm.com/~binstock/astro.htm>

This *Candida* issue is extremely important, in my opinion. If one has an ablation, and still is dealing with a *Candida* issue, as I am starting to believe that most of us are, then the neuronal degeneration can continue on, unless this invasion is dealt with. Everything I have been reading about bacterial or viral invasions, keeps leading me to folic acid, B6, B12, and the Mo dependent enzyme, xanthine oxidase. These nutrients represent a problem with me, in that they were extremely low. Sulfonamides, that are used to treat bacterial invasions compete with PABA, to starve the bacteria of folic acid. I don't have a clear understanding of this presently, but will continue to read. See the following link:

In 1940, Woods and Fildes (1,2) had put forth the antimetabolite theory to explain the action of sulfonamides on bacteria, suggesting that the sulfonamides interfered with the utilization of a necessary nutrient, para-aminobenzoic acid. Hitchings theorized that, since all cells required nucleic acids, it might be possible to stop the growth of rapidly dividing cells (e.g., bacteria, tumors, protozoa) with antagonists of the nucleic acid bases. One might hope to take advantage of the faster rate of multiplication of these cells compared with normal mammalian cells and eventually sort

out the biochemical differences between various types of cells by the way they responded to these antimetabolites(3,4). It was my assignment to work on purines, pteridines, and some other condensed pyrimidine systems.

It was, of course, necessary to have some biological systems to determine the potential activities of the new compounds. Essentially nothing was known at that time about the anabolic pathways leading to the utilization of purines for nucleic acid synthesis. A number of catabolic enzymes were known: nucleases, nucleotidases, nucleosidases, deaminases (for guanine, adenine, adenosine, and adenylic acid), xanthine oxidase and uricase. In 1947, Kalckar described the reversibility of nucleoside phosphorylase (5). The enzymes guanase and xanthine oxidase were useful in our laboratory to examine the purines as substrates or inhibitors of these enzymes (6,7). However, it was the microorganism *Lactobacillus casei* upon which we mainly relied. It could grow on adenine, guanine, hypoxanthine or xanthine, provided the pyrimidine thymine was added. It could also synthesize purines and thymine if given a source of folic acid in the form of liver powder. (The structure of folic acid was not elucidated until 1946 by the Lederle group (8)). Hitchings and Falco had devised a screening test in which it was possible to determine whether a compound could substitute for thymine (9) or a natural purine (4), (10) or inhibit its utilization, and could also determine whether a compound was a folic acid antagonist (11,12).

<http://www.geocities.com/tnagasatish/speeches/elion.html>

This link is about malaria, but from reading, it has very similar actions to *Candida*. As you're reading, remember that my levels of folate in RBCs are extremely high (trapped), but my metabolic analysis test indicated I was extremely low.

Paradoxically, high concentrations of folate in red blood cells (RBCs) have been reported in pregnant women living under holoendemic conditions for malaria in western Kenya [24]. This was found mainly in primigravidae, particularly in those who were parasitemic.

2.3. Folate metabolic pathway and methionine used by malaria parasites

Malaria parasites are capable of de novo folate synthesis, which makes them susceptible to sulfonamides and para-aminobenzoic acid analogs. They also have a dihydrofolate reductase that is much more sensitive to antimalarial inhibitors (e.g. pyrimethamine, diaminopyrimidines, proguanil) than that of the host cell [29]. It is thought that their folate metabolism might be restricted to a thymidylate synthesis cycle [30] (Fig. 1). This means that the parasites might not be able to recycle methionine via homocysteine, making them even more dependent on exogenous supplies of this essential amino acid. Other observations suggest that host breakdown products of folates can be used by the parasites for their de novo synthesis [29]. Increased permeability of the RBC membrane to a variety of nutrients is known to take place on infection [31] and this might partly explain the increased levels of folate seen in RBC in experimental malaria infections [32 and 33]. If the parasite has a restricted folate cycle, as mentioned above, it might depend on the more complete host cell cycle to carry out other reactions such as the metabolism of increased levels of potentially cytotoxic homocysteine produced by methionine use.

<http://www.prema-eu.org/folatepathway/article.htm#toc3>

Fungal virulence studies come of age

http://genomebiology.com/render/render.asp?arx_id=2001-2-3-reviews1009&type=1&item=2001/2/3/reviews/1009/1&pubtype=core&

Any thoughts would be appreciated.

Richard

Richard

I thought I'd interrupt the "Richard Ladder " so you wouldn't feel all alone. I must confess though that sometimes I have trouble following some of the research reports that you keep digging up. As I told my wife it's amazing what we don't know. The more I read and learn the more I don't know. I wish I had stayed in biology in high school. Hell I wish I had stayed in High school!! But I do have a question that is more in line with my level of understanding.

First off I got to say your diligence in ferreting out the details of our common condition, Afib, is indeed admirable and greatly appreciated. I continue to learn something every time I visit the board. Thank you.

I've tried the spit test on 4 occasions. The first one was after one of those Pizza Fridays that I'm not supposed to do. In the morning I had many long legs to the bottom of the glass and immediately pronounced myself to be Candida afflicted. On the other tests 2 of them had 2 legs that would travel 1/2 way down the glass and were less substantial than the first test. The final test showed no legs at all. So where does that put one? Mild candida? or perhaps reactive candida. Normally I follow a paleo diet so It probably goes somewhat dormant at those times. Just what I need, another reason to quit Pizza :-) as if I didn't have enough!

Wishing you a Peaceful and Joyful Christmas.

Adrian v49

Adrian,

Maybe your Candida fungi are neurotransmitter deficient and confused about where to go, however if that isn't the case, then maybe you could keep spitting, and by doing that, you'll be sure to get rid of them. Another avenue to study. I laughed so hard, reading your post, I was crying. Reactive Candida put me over the top. Thanks for a really good laugh.

On a more serious note, I hadn't thought about taking the test on more occasions. I'll have to start doing it more often and see what I find. That was interesting that you had more strings after eating pizza. The thought of eating pizza now, grosses me out.

By the way, I went to college, but I ate the covers, now I'm regretting that. Hell, maybe the covers caused my Candida.

Richard

Adrian

The reactive candida idea does sound funny, but I have wondered about this myself. I did the candida test twice, both on different diets. The first one was positive and the one on the Paleo diet was negative. I suspect strongly though that if I was to eat any honey the legs would come back and am yet to do the experiment.

Could it be that if you eat sugar etc your digestive juices become more acid, or more thick (with a bit more dehydration) which would include saliva. And acid and alkaline saliva may string out differently.

Don't know but am not convinced about this test

Fran

I took the test on 2 different mornings, the first one I had 2 long fat legs and a skinny one, the 2nd test 2 days later there was nothing but a big beautiful gob just sitting on top of the water, just sitting there for 3 hours, doing nothing! Maybe I will try again for 2 out of 3? I have no idea what my meal was the nights before.

Ella

I posted this on the BB, but wanted to put it here for future ease of finding. My marker for arabinose was still high, but not out of reference range. My Candida is probably a bit more under control since being on the Paleo diet for about 10 mths.

Arabinose and Arabinitol

Arabinose is a carbohydrate and arabinitol is the related sugar-alcohol which is a product specifically of anaerobic fungal growth in the gut. The D isomer of arabinitol is produced by fungal metabolism and its urinary excretion is elevated in patients with invasive candidiasis [36,37].

<http://www.metametrix.com/PUBLICATIONS/ORGANIC%20COMPOUNDS%20IN%20URINE/>

Richard

Here's some research articles from Mary Enig PhD, as well as others about coconut oil's (monolauric acid) anti-fungal/viral effects.

<http://www.coconut-info.com/links.htm#Research%20By%20Dr.%20Mary%20Enig%20on%20Coconut%20Oil>

Richard

This may be of interest. It certainly was to me.

Coconut oil also raises metabolic rate causing the body to burn up more calories and thus promoting weight loss. A faster metabolic rate stimulates increased production of needed insulin and increases absorption of glucose into cells, thus helping both Type I and Type II diabetics.

For those with Crohn's and IBS, the anti-inflammatory and healing effects of coconut oil have been shown to play a role in soothing inflammation and healing injury in the digestive tract. Interestingly, researchers have demonstrated the benefits of coconut oil on patients with digestive problems, including, Crohn's disease, at least since the 1980s. Its antimicrobial properties also promote intestinal health by killing troublesome microorganisms that may cause chronic inflammation.

Finally, for those with chronic fatigue syndrome, coconut oil may provide a vital solution. The fatty acids in coconut oil can kill herpes and Epstein-Barr viruses which are believed to be major causes. They kill Candida and giardia. They kill a variety of other infectious organisms, any of which could cause chronic fatigue. The key to overcoming CFS is strengthening the immune system. Coconut oil supports the immune system by ridding the body of harmful microorganisms, thus relieving stress on the body. With fewer harmful organisms taxing the body's energy, the immune system can function better.

http://mercola.com/forms/coconut_oil.htm

Richard

Hello to All,

It's me again. I feel like I'm climbing a ladder to nowhere. I'm going to put this in the conf. room, as I believe it is IMPORTANT, as it is a possible common link to our affliction and many others. It is also important to heavy exercisers. Being that Candida produces aldehydes, the relevance of this link to Candida, is based on alcoholic livers, but non-alcoholic livers, as well. I will post some highlights.

Glutathione is homeostatically controlled, both inside the cell and outside. Enzyme systems synthesize it, utilize it, and regenerate it as per the gamma-glutamyl cycle. Glutathione is most concentrated in the liver (10 mM), where the "P450 Phase II" enzymes require it to convert fat-soluble substances into water-soluble GSH conjugates, in order to facilitate their excretion. While providing GSH for their specific needs, the liver parenchymal cells export GSH to the outside, where it serves as systemic source of -SH/reducing power. GSH depletion leads to cell death, and has been

documented in many degenerative conditions. Mitochondrial GSH depletion may be the ultimate factor determining vulnerability to oxidant attack. Oral ascorbate helps conserve GSH; cysteine is not a safe oral supplement, and of all the oral GSH precursors probably the least flawed and most cost-effective is NAC (N-acetylcysteine). (Alt Med Rev 1997; 2(3):155-176)

GSH has potent electron-donating capacity, as indicated by the high negative redox potential of the GSH/GSSH "redox couple" ($E^{\circ} = -0.33\text{v}$).⁶ Its high redox potential renders GSH both a potent antioxidant per se and a convenient cofactor for enzymatic reactions that require readily available electron pairs, the so-called "reducing equivalents."⁷ Lewin⁶ articulated how a substance with great readiness to donate electrons, when present at high concentrations, has greatly enhanced effectiveness as a reductant. This is reducing power, and is most expressed by GSH where its concentrations are highest (as in the liver). The reducing power of GSH is a measure of its free-radical scavenging, electron-donating, and sulfhydryl-donating capacity. Reducing power is also the key to the multiple actions of GSH at the molecular, cellular, and tissue levels, and to its effectiveness as a systemic antitoxin.⁸

That GSH has profound importance for cellular homeostasis and for diverse cellular functions was essentially established by 1978 (see Kosower and Kosower¹ for an excellent review of the early work on GSH). GSH plays a role in such diverse biological processes as protein synthesis, enzyme catalysis, transmembrane transport, receptor action, intermediary metabolism, and cell maturation. Some of the functions in which GSH is involved are illustrated in Fig. 4.

The continual flux of single electrons to oxygen generates an endogenous oxidative stress in human tissues. Superoxide, peroxide, hydroxyl radical, and other free radicals derived from oxygen are highly reactive and therefore threatening to the integrity of essential biomolecules such as DNA and RNA, enzymes and other proteins, and the phospholipids responsible for membrane integrity. The aerobic cell is continually challenged to neutralize these OxyRad time bombs before they can initiate propagative free radical reactions that could cause its disintegration. Healthy cells homeostatically oppose free radicals through the use of antioxidants. Table 1 lists free radical quenching reactions against which GSH can be employed.

With our reliance on oxygen, humans cannot escape this ongoing oxidative challenge. It may be the ultimate challenge of being alive. An ever more impressive body of evidence indicates that the cumulative damaging effects of oxygen radicals and other oxidants are principal contributors to degenerative diseases, and to the progressive loss of organ functions that we recognize as aging.²⁵

Oxidative stress originating from outside the body is a feature of life in the modern world. First, the tens of thousands of confirmed toxic substances in our external environment are invariably sources of free radicals or related oxidants. Add to this substantial burden the many negative aspects of the modern, Westernized lifestyle and a picture emerges of the human organism burdened by chronic disease and threatened with a shorter lifespan than might otherwise be possible. The most important of the exogenous oxidative stressors are briefly discussed below.

Strenuous aerobic exercise can deplete antioxidants from the skeletal muscles, and sometimes also from the other organs. Exercise increases the body's oxidative burden by calling on the tissues to generate more energy. Making more ATP requires using more oxygen, and this in turn results in greater production of oxygen free radicals. Studies in humans and animals indicate GSH is depleted by exercise, and that for the habitual exerciser supplementation with GSH precursors may be a prudent policy.³¹

Some of the other exogenous factors known to deplete tissue GSH include: 1.) Dietary deficiency of methionine, an essential amino acid and GSH precursor. The liver uses 70 percent of the total dietary intake.⁴ 2.) Ionizing radiation, whether as X-rays or ultraviolet from sunlight.³² 3.) Tissue injury, as from burns,³³ ischemia and reperfusion,^{34,37} surgery,³⁵ septic shock,^{4,36} or trauma.³⁷ 4.) Iron overload, as in hemochromatosis and transfusional iron excess.²⁵ Surgery can cause iron release from damaged tissue, and unbound iron catalyzes free radical generation via several putative mechanisms. 5.) Bacterial or viral infections, including HIV-1.^{3,4} 6.) Alcohol intake is toxic through a number of differing pathways, some of which are free radical/oxidative in character.³⁸

The consequences of sustained GSH depletion are grim. As cellular GSH is depleted, first individual cells die in those areas most affected. Then zones of tissue damage begin to appear; those tissues with the highest content of polyunsaturated lipids and/or the most meager antioxidant defenses are generally the most vulnerable. Localized free-radical damage spreads across the tissue in an ever-widening, self-propagating wave. If this spreading wave of tissue

degeneration is to be halted, the antioxidant defenses must be augmented. Repletion of glutathione appears to be central to intrinsic adaptive strategies for meeting the challenge of sustained (or acute) oxidative stress. A discussion of antioxidant adaptation mechanisms is beyond the scope of this review but has been amply discussed elsewhere.^{39,40}

GSH depletion has been suggested to represent an important contributory factor to liver injury, and to enhanced morbidity related to liver hypofunction^[4]. In one small study, subnormal plasma concentrations of GSH were observed in cirrhosis patients, independent of their diet.⁴⁷ A larger study demonstrated a four- to eight-fold decrease in plasma GSH in 48 cirrhotic patients versus 18 healthy volunteers.⁴⁸ A significant decrease in cysteine in severe cirrhosis also was observed.

GSH deficiencies have been documented in a number of pulmonary diseases, including acute respiratory distress syndrome (ARDS), asthma, chronic obstructive pulmonary disease, idiopathic pulmonary fibrosis, and neonatal lung damage.⁴ Patients with ARDS and sepsis have a deficiency of GSH in the ELF as compared with healthy subjects,^{52,53} and a greater percentage of the total ELF glutathione is in the oxidized form (GSSG), indicating increased oxidative stress in the lower respiratory tract.⁵³ When GSH was repleted in their ELF using intravenous N-acetylcysteine, patients in intensive care regained independent lung function and left the intensive care unit significantly faster.⁵⁴

As with other cell types, the proliferation, growth, and differentiation of immune cells is dependent on GSH. Both the T and the B lymphocytes require adequate levels of intracellular GSH to differentiate, and healthy humans with relatively low lymphocyte GSH were found to have significantly lower CD4 counts.⁵⁷ Intracellular GSH is also required for the T-cell proliferative response to mitogenic stimulation, for the activation of cytotoxic T "killer" cells,⁵⁸ and for many specific T-cell functions, including DNA synthesis for cell replication, as well as for the metabolism of interleukin-2 which is important for the mitogenic response.⁵⁹

The brain is particularly susceptible to free radical attack: it is highly oxygenated, which makes it vulnerable to endogenous oxygen radical production, and it has a high proportion of unsaturated lipid which makes it vulnerable to peroxidation. In addition, those brain regions that are rich in catecholamines are exceptionally vulnerable to free radical generation. The catecholamines adrenaline, noradrenaline, and dopamine can spontaneously break down (auto-oxidize) to free radicals, or become metabolized to radicals by the endogenous enzymes known as MAO Ñthe monoamine oxidases.^{65,66} One such region is the substantia nigra (SN), where a connection has been established between antioxidant depletion (including GSH) and tissue degeneration.

The evidence to date for possible oxidative stress in DS (Down's syndrome), PD (Parkinsons), TD (Tardive dyskinesia - Glutamate excess may also contribute to the free-radical overload in TD), schizophrenia and AD (Alzheimers) is suggestive, if not yet strongly persuasive. As pointed out by Jenner,⁶⁹ if oxidative stress does contribute to neural degeneration, whether it is eventually proven to be primary or secondary in the etiologic progression, the therapeutic rewards are likely to be great. Future trials are indicated with dietary GSH precursors, administered in combination with other antioxidants, antioxidant cofactors, and non-antioxidant brain-trophic nutrients such as phosphatidylserine.

Both the GSH peroxidase enzymes and various GSH-S-transferases may be employed in the endothelia for "yin-yang" regulation of vascular tone and responsiveness, mainly through their influences on eicosanoid balance; the more active they are, the better the production of protective eicosanoids. GSH can produce coronary vasodilation when added to isolated, perfused rodent heart, very likely due to its normalizing effect on prostaglandin synthesis.⁷⁷

Chronic pancreatitis patients also have shown increased serum lipid peroxides, with those in relapse generally showing the greater increases.⁸² Such patients often were deficient in several antioxidants. Uden and collaborators⁸³ did a small double-blind, crossover trial in which they gave selenium, vitamin A, vitamins C and E, and methionine (a cysteine precursor) to patients with pancreatitis (mixed acute and chronic). This therapy significantly reduced pain and prevented relapse, independent of the etiology and acuteness of the disease. Larger trials are needed, but to date supplementation with mixed antioxidants appears promising in pancreatic inflammatory states.

Hormones and other vasoactive substances increase GSH efflux into the bile, and this may contribute to the hepatic GSH loss noted under conditions of stress.⁴¹

A list of GSH precursors with known safety profiles would include NAC, as well as glycine, L-glutamine, L-taurine, L-

methionine, and S-adenosyl methionine; L-cysteine should be avoided.

The consistent findings of GSH depletion in many preclinical and clinical degenerative conditions beg the question of whether antioxidants should be universally employed - whether singly or in combination - in efforts to ameliorate functional degeneration and improve quality of life. Combinations of antioxidants given as dietary supplements seem to offer the most promise for achieving clinical breakthroughs. At times, the administration of massive amounts of ascorbate (orally or intravenously) or of sulfhydryls (GSH and NAC orally and intravenously) will be lifesaving.

Prenatal diagnosis of inherited GSH abnormalities may not be far off.¹¹ In the meantime, dietary repletion of systemic GSH holds promise for the management of conditions as diverse as Alzheimer's Disease, atherosclerotic vascular degeneration, cataract, lung insufficiencies, Parkinson's Disease, and many others. Assiduous attention to repletion of GSH also should help assist the body to manage bouts of heavy exercise or combat a chronic viral load. Particularly when employed in conjunction with ascorbate, other antioxidants, and other nutritional factors, the reducing power of GSH is a powerful orthomolecular tool for quality and length of life.

<http://www.thorne.com/altmedrev/fulltext/glut.html>

Sorry for such a lengthy post, and there's much more to read at the link, but as you see GSH plays a critical role and its deficiency is a common link in many degenerative diseases. It is also depleted in excessive exercise and stress. I'll pose this question again. Could the glycine in the Mg. glycinate, have been playing an equal role in helping symptoms for Jackie and Carol?

Could GSH help fight the effects of Candida's acetaldehydes, while trying to gain control through diet? I have been taking lysine, glycine, methionine, and N-acetyl cysteine. I'm not presently taking glutamine, as my levels were high. I'm going to add alpha lipoic acid, and am considering adding either glutathione or reduced glutathione. I'll have to research a bit more on the latter to see which is best. I'm also taking a super oxidant formula made by Montiff that I purchase off Dr. Gersten's website www.aminoacidpower.com

Anyway, the more I read on GSH, and sulfur, the more and more I become convinced. One more thing to prompt your memory. Remember Valerie Hudson who has taken glutathione to clinical trials. She found it was extremely helpful for her 3 sons with Cystic Fibrosis.

Merry Christmas,
Richard

I have now received responses from 70 afibbers in the survey concerning candida overgrowth and migraine headaches. Thank you for participating. While I have not yet had a chance to evaluate the results in detail it is clear that neither candida nor migraines are common factors in lone atrial fibrillation.

Only 13 out of 69 respondents (19%) had been diagnosed with candida overgrowth and only 14 out of 39 respondents (36%) tested positive on the saliva test. I still need to find out if the difference between the incidence of actually diagnosed candida and candida indicated by the saliva test points to underdiagnosis of the condition or whether it is caused by afibbers who already know that they have candida being more likely to take the test. In any case, 9 out of the 13 candida sufferers (69%) were still battling the fungus indicating that candida is indeed difficult to bring under control.

Only 18 out of 69 respondents (26%) had ever suffered from migraine headaches and only 8 (13%) still experienced them occasionally.

Thus it would appear that neither candida nor migraine headaches are the "holy grail" of afib. The search goes on :-)

Hans

Hans,

Don't know if you are still accepting data for the candida -afib study, but according to the saliva test, I am an afibber with a serious case of long standing candida albicans. I am in the process of trying to eliminate it.

Carol

Carol,

Sure, I can still include your data if you would like to participate in the survey.

Hans
