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SUBJECT: Molybdenum

Hans and All,

I apologize, Hans, for starting a new thread, but all the other posts were getting confusing, and I did NOT want anyone to miss this.

A few points I'd like to bring back up, to refresh our memories concerning Molybdenum (Mo) are:

1) I was very low, and Dr. Gersten pointed out that this could be the most profound finding in all my tests.

2) Fran's sister has esophageal cancer, which deficiencies of Mo are known to be a cause.

3) Mo helps with glutamate toxicity sensitivities. Could this be what Fran is lacking????

4) NEW POINT: Mo is essential for the pathway of acetaldehydes to be broken down into acetic acid, for removal from the body.

To tell the story as it unfolded, I found a site that was a Great Smokies Metabolic Analysis lab test for someone else, with results very similar to mine, almost uncannily so, with the exception of low copper. Pls. see pg. #8 of this link.
http://www.ivfg.nl/VoorbeeldrapportenGSDL/M-010.pdf

On this page it showed an aldehyde oxidase weakness and that Mo is the possible remedy, but to check the RBC test, which indicated abnormally low levels on my test, hence the doctor telling me to take 300mcg. daily.

So I'm thinking, what the heck is aldehyde oxidase, and I begin my search. I came upon this site first that explains the importance of aldehyde oxidase and Mo. Here's what it said:

The Candida/Aldehyde detox pathway and the Molybdenum Connection

As it relates to Candida, those of you who have read the work of Dr. Orion Truss, or who have seen quotes by others from his work, will already have been alerted to his assertion that much of the harm done by Candida results from its waste product, acetaldehyde, which in turn can affect the metabolic, neurological, endocrine, and immune systems. Further, that few chemicals can create so much havoc in the body as acetaldehyde can. It may interfere with the receptors for acetylcholine which is supposedly the major neurotransmitter in the corpus callosum. Formaldehyde, obviously then, is related to acetaldehyde in the aldehyde chain of chemicals.

Dr. Stephen Rochlitz worked with cross-crawl brain integration exercises with dyslexic patients with formaldehyde taped to these patients right brain hemisphere, and sometimes the left.

Acetaldehyde is a fungal waste product.

Dr. Stephen Cooter, in his book "Beating Chronic Disease", ProMotion Publishing, San Diego, California, states that "Candida is responsible for flooding the system with an accumulation of toxic acetaldehydes. Acetaldehydes are known to poison tissues -- accumulating in the brain, spinal cord, joints, muscles and tissues."

Dr. Cooter then goes on to describe how he learned from a chiropractor, Dr. Carol Cooper [this name came up on this List way back] that molybdenum -- a mineral -- not a medication, but a nutrient, had a blanket reputation for breaking down yeast by-products into forms that the body could excrete. Coincidentally, Dr. Cooter read the monogram by Dr. Walter Schmitt "Molybdenum for Candida Albicans Patients and Other Problems" through Dr. Cooper. [Interestingly, these are all chiropractor, Drs. Roschlitz, Cooper, and Schmitt.]

I'm beginning to see a glimmer of some possible connections here. Dr. Roschlitz's work, and Dr. Walter Schmitt's, although slightly different, seems similar to me to the principle of Dr. Nambupridad's work with NAET, and perhaps then, holding the substance, when the body is worked on through one of their modalities, might not seem so strange after all. I think I see a common denominator here. Worth exploring? Perhaps....

Back to Dr. Cooter and Dr. Schmitt: "Molybdenum is chemically responsible for breaking down acetaldehyde into acetic acid. Acetaldehyde cannot be excreted from the body; it accumulates. Acetic acid can be, though, and the body naturally removes it or changes it into acetyl coenzyme A, a major player in the body's energy system.... Acetaldhyde accumulations in tissue are responsible for weakness in muscles, irritation, and PAIN."

And now for the good part (g), directly quoted from Dr. Walter Schmitt:

"Chemical aldehydes are best known as fragrances." [Shall I repeat that?] "Chemical aldehydes are best known as fragrances.... Ethanol, or drinking alcohol, is also processed to acetaldehyde. ...the body has an enzyme which breaks down the aldehydes to less toxic substances. This enzyme is aldehyde oxidase, or sometimes, aldehyde dehydrogenase. Aldehydes encountered dietarily or environmentally or produced in the body must be handled by aldehyde oxidase metabolic pathways.

Acetaldehyde is a particularly toxic substance which, in addition to being produced by threonine and ethanol, is a product of the metabolism (i.e. fermentation) of carbohydrate in yeast -- hence the Candida connection. Acetaldehyde is thought to be the major source of tissue damage in alcoholics rather than ethanol itself. The conversion of acetaldehyde into acetic acid" for this reaction to occur, threonine to acetaldehyde to acetic acid to acetyl coenzyme A, NAD (niacine amide) is required, and aldehyde oxidase is dependent of riboflavin, iron, and molybdenum. These forgoing nutrients could be helpful to Candida albicans patients, and others who are sensitive to various fragrances and airborne odors. Those patients with aldehyde sensitivity are incredibly sensitive to any type of fragrance.

By coincidence, (or is it?) there's a little squibb in the newsletter from the Environmental Health Association of Dallas on fragrance. "Perfume today is not made from flowers but from toxic chemicals..... More than 4,000 chemicals are used in fragrances. Of these, 95 percent are made from petroleum. Some toxic chemicals found in fragrances: toluene, ethanol, acetone, formaldehyde, limonene, benzene derivatives, methylene chloride, and many others known to cause cancer, birth defects, infertility, nervous system damage, or other injuries.... Exposure to scented products can cause exhaustion, weakness, 'hay fever', dizziness, difficulty concentrating, headaches, rashes, swollen lymph glands, muscle aches and spasms, heart palpitations, nausea, stomach cramps, vomiting, asthma attacks, neuromotor dysfunction, seizures, and loss of consciousness." This was reprinted from No Perfume Means Healthier Air brochure, Breath of Fresh Air Battleaxe, Oakland, California.

And from another source comes another connection -- from Dr. Robert Atkins' newsletter: Dr. Atkins is writing about Pantethine which he prescribes to his Crohn's Disease and Colitis patients, with acknowledgement to Dr. Melvin Werbach for Dr. Werbach's study that demonstrated that people with colitis have markedly decreased Coenzyme A activity if the mucosal surface of their colons, even when the blood levels of pantothenic acid are normal. Dr. Atkins concluded, based on his success with these patients of his, that Pantethine bypasses the block in converting Vitamin

B5 (Pantothenic Acid) to Coenzyme A. But also, that Pantethine is a growth factor for lactobacillus bulgaricus and bifidobacterium that we know help control yeast overgrowth (and Dr. Cooter also speaks of it in his book). Candida, according to antibody studies done at the Atkins Center, is involved in more than 80 percent of all cases of Crohn's and Colitis.

And for autoimmune problems, Dr. Atkins states, " For all conditions that a doctor might prescribe prednisone -- allergies, asthma, rheumatoid arthritis, psoriasis, lupus, and other autoimmune diseases, pantethine can be safely, effectively substituted. I routinely use it for all of those conditions on hundreds of my patients, and it's valuable in weaning them off steroidal drugs, or certainly in allowing a lower dose....

By upping body levels of a body enzyme, pantethine counteracts brain fog, certain allergic sensitivities, and some consequences of alcoholism. (And here it is --) ... In people with candidiasis, the enzyme fights off a toxic byproduct called acetaldehyde, which is thought to cause brain fog, often-suffered but rarely diagnosed.... Acetaldehyde also is suspected of being responsible for some symptoms of alcoholism, including alcoholic heart muscle disease. The pantethine-stimulated enzyme also detoxifies formaldehyde, an all too frequent offender for chemically sensitive individuals."

In summary, Dr. Atkins is saying that Pantethine, without toxic consequences, can reduce cholesterol, counteract oxidation, stimulate the growth of friendly bacteria, and fight allergies, inflammation, autoimmune disruptions, and alcoholism.

In case you wondered, Dr. Cooter and Dr. Schmtt suggest 300 micrograms of Molybdenum in three divided doses per day, and further suggests staying on it for at least 4 months.. Dr. Atkins suggests 450 to 900 miligrams daily of Pantethine with an equal amount of Pantethenic Acid.

Send e-mail to author Jann Weiss.
http://www.panix.com/~candida/aldehyde.shtml

Pretty interesting. Here's more on Mo.

Molybdenum is an essential constituent of two enzymes found in humans: xanthine oxidase, which is involved in uric acid formation, and aldehyde oxidase, which catalyzes the chemical oxidation of aldehydes.

Molybdenum is concentrated primarily in the liver, kidney, bone, and skin. There is estimated to be approximately nine milligrams of molybdenum in the adult human body.

Molybdenum is an antagonist to copper absorption, as is copper to molybdenum absorption. Excess molybdenum intake can induce copper deficiencies with the subsequent symptoms.

Method of Action

Molybdenum is an important constituent of aldehyde oxidase and xanthine oxidase. Aldehyde oxidase catalyzes the oxidation of an aldehyde functional group to the corresponding carboxylic acid. Xanthine oxidase catalyzes the oxidation of xanthine to uric acid for excretion. Xanthine is a product formed in the chemical degradation of purine nucleotides found in DNA and RNA.

Molybdenum, in the presence of inorganic sulfate, tends to reduce copper absorption and retention. Several theories for why this antagonistic relationship occurs have been postulated. There is evidence that copper and molybdenum form an insoluble complex called lingrenite, which cannot be absorbed easily. Other theories include the postulated interference of ceruplasm synthesis: ceruplasm is a protein necessary for copper transport into blood.

Molybdenum absorption occurs readily in gastrointestinal tract, and excretion occurs primarily via the urine.

Properties and Uses

Molybdenum has been implicated as a possible contributor to decreased incidence of dental carries, a reduced

incidence of cancer, and a bringing into balance of female hormones for the control of premenstrual syndrome.

Consequences of Deficiency

Molybdenum deficiencies in humans have not been conclusively linked to any specific set of symptoms; however, one source indicates that a high incidence of cancer of the esophagus may be the result of low molybdenum intake. http://www.springboard4health.com/notebook/min_molybdenum.html

And more:

Widespread cellular distribution of aldehyde oxidase in human tissues found by immunohistochemistry staining.
Moriwaki Y, Yamamoto T, Takahashi S, Tsutsumi Z, Hada T.
Third Department of Internal Medicine, Hyogo College of Medicine, Nishinomiya, Japan.

Aldehyde oxidase (EC 1.2.3.1) is a xenobiotic metabolizing enzyme that catalyzes a variety of organic aldehydes and N-heterocyclic compounds. However, its precise pathophysiological function in humans, other than its xenobiotic metabolism, remains unknown. In order to gain a better understanding of the role of this enzyme, it is important to know its exact localization in human tissues. In this study, we investigated the distribution of aldehyde oxidase at the cellular level in a variety of human tissues by immunohistochemistry. The enzyme was found to be widespread in respiratory, digestive, urogenital, and endocrine tissues, though we also observed a cell-specific localization in the various tissues studied. In the respiratory system, it was particularly abundant in epithelial cells from the trachea and bronchium, as well as alveolar cells. In the digestive system, aldehyde oxidase was observed in surface epithelia of the small and large intestines, in addition to hepatic cells. Furthermore, the proximal, distal, and collecting tubules of the kidney were immunostained with various intensities, while glomerulus tissues were not. In epididymus and prostate tissues, staining was observed in the ductuli epididymidis and glandular epithelia. Moreover, the adrenal gland, cortex, and notably the zona reticularis, showed strong immunostaining. This prevalent tissue distribution of aldehyde oxidase in humans suggests some additional pathophysiological functions besides xenobiotic metabolism. Accordingly, some possible roles are discussed.
http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=11510964&dopt=Abstract

Per the above, acetaldehydes are used in perfumes, and I have not been able to wear cologne, as it gives me rashes, and also, acetaldehydes have something to do with moths and their pheromones for mating. I don't know if this has anything to do with my problem, but my wife use to say that I sometimes had a peculiar smell, that reminded her of mothballs. No, Mike, not the ones between their tiny legs. She even tried to search out information on that once, thinking it might be a clue. Just a strange thought I felt like mentioning. Then I came to this study. Now, I know none of us are alcoholics, at least I don't think anyone here is, but this study showed how acetaldehydes damage the heart.

Here's pertinent information:
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 (acetaldehyde) by ADH 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 (EMPHASIS ON ALTER) the activity of xanthine oxidase (MO IS ALSO IMPORTANT FOR THIS ENZYME), which generates free radicals. Another pathway of ethanol metabolism is the microsomal ethanol oxidizing system (MEOS), which catalyzes the conversion of ethanol to ACA.

As previously mentioned, aldehyde oxidase and xanthine oxidase are both involved in the metabolism of ethanol and ACA, and result in oxidative stress leading to cardiac mechanical dysfunction. (REMEMBER THESE ENZYMES WERE ALTERED) As indicated in Fig. 2, both aldehyde oxidase and xanthine oxidase may be intervened pharmacologically with menadione and allopurinol (YEAH, MORE DRUGS), respectively.

Synopsis
Alcohol abuse continues to remain as a prevalent social problem contributing to severe heart problems in alcoholics. It is a current opinion that the toxic effects of alcohol on the heart are attributable, in large part, to the first metabolic

product of ethanol - ACA. ACA is formed from the breakdown of ethanol in cells and is far more toxic and reactive than ethanol. In order to better understand the adverse repercussions of chronic alcohol abuse, assessment of cardiac toxicity of ACA using our described methods should lead to useful clues regarding the pathogenesis of alcoholic cardiomyopathy.
http://www.biologicalprocedures.com/bpo/arts/1/41/m41.htm

Soooooo, what do you think?????

By the way, Carlson's make Moly-B glycinate, Albion process. Oh boy, more glycinate.

Richard

Richard said: " Now, I know none of us are alcoholics, at least I don't think anyone here is, but this study showed how acetaldehydes damage the heart. "

Whispers I am a recovered alcoholic, I drank for 4 years and quit Oct. 15 th 1979, so 24 years sober, was dx with afib in '93 but had symptoms since '89.
I now also have multi chemical sensitivities.

Ella

Ella,

I'm glad to hear you kicked the habit so long ago. Have you ever had your molybdenum, mineral or amino levels checked? I certainly think it's worth asking your doctor about the Mo. You don't have to whisper, Ella, even though that was a bit funny, as we've probably all had our drinking days, and I think some still do. And yes, even though I've not been a heavy drinker, would sometimes like to indulge in just one, but I don't.

Thanks for the confession.
Richard

Richard,

What do I think?? I think that you're a VERY diligent and determined researcher..... and yes, I KNOW what moth balls are.... as opposed to moth's balls!!

Interesting about aftershave.... I've NEVER been able to wear it.... and I've tried at the behest of lady friends who dig it..... but it always makes me feel slightly sick.

And my blood urate is always top of or above normal reference range..... And I've always had problems with candida such as Doby's/Dobies itch, athletes' foot, and thrush - never severe or chronic, just mildish and on-and-off. I've often wondered whether my digestive tract troubles could have led to leaky gut contributing to candida in the blood. Wow, does all of this stuff tie in or what..... Keep it up Richard... it's interesting and much-appreciated.

Mike F.

OK Richard

You have got me convinced. I would prefer to try and get molybdenum through diet and am off to do some research on the levels in our soil here and near abouts where I eat from. Trouble is the USDA database and fitday don't mention this nutrient in their food databases. So it might be quite hard to get the info I am looking for. I assume that the best

places if the animal isn't deficient, would be the organs?

Fran

Liver bacon and onions for tea, lamb chops, rocket and tomato for lunch and herring for breakfast. Not joshing. But did find myself looking for additive free hagis today when I was shopping, I thought that once I could get away with the oats. It's made from the lungs etc and is our traditional food. Waste not, want not... I could only find vegetarian ones!!

Discovered that I live relatively near a place with high molybdenum levels and famous slate. Still can't discover the level here, but due to the acidic soil think it might be relatively low in Cu and molybdenum. However, I think that liming with shell sand ups the molybdenum levels - or at least the bioavailability of it. Sometime early next year I will get soil testing done. This land has never been intensively farmed. Do you know if there is molybdenum in seaware?

I don't know if you posted this Linus Pauling link on molybdenum but I thought it was good

http://lpi.oregonstate.edu/infocenter/minerals/molybdenum/

Fran

Hans, Richard,
Perhaps I can bring it back into the focus on copper. Dr. Balch states
In addition to copper promoting Fe, and Zn it also promtoes the absorption of Mo and S. Interesting enough Mo is inhibited by N.
This information was found in the interaction of minerals in Third Edition of Prescritipn for Nutritional Healing.

FWIW,
Joe

Richard,
Isn't this contrary to what you suggested as Cu being an antagonist to Mo? This is an interesting conundrum....

Joe

Mike,

Thank you for those links. I found them quite interesting, especially in regards to asking myself, why I have a Mo deficiency. It is known to be rare. My Cu levels are normal in the packed erythocyte test, but extremely high in my hair analysis, top of the chart compared to the rest of the minerals. I hope David S. doesn't mind me discussing his hair analysis, but I know he's shared the info in the past. David's results of Cu are right at the lowest of the normal range and have dropped some since his last hair analysis, which was just a bit low to normal. He said he was having a bit more problems. His Mo levels were more normal to high in his first test, and then dropped to the low/normal range. If these two minerals compete, then why did both his levels drop, w/Mo dropping the most. David's sulfur levels in hair fell into a normal range, with a bit of a drop since his last test, whereas mine were to the high side, past the green normal area. But you know what? My levels of the sulfur aminos were at the bottom, in the amino urine analysis, and methionine, taurine, and cysteine were all low in serum. Hair analysis of sulfur would be somewhat useless, in that hair is made up mostly of cysteine. But with David's sulfur results being lower than mine, I would think he has a worse sulfur content in his body than I do, and that's pretty bad. The only other thing that stood out between our two hair analysis (I'm not sure how to make this word plural), was lithium (Li). David fell slightly, but that put him on the edge of the green/yellow line, so he's teetering on low, whereas mine was below normal. I might need to do some searching on Li. David did well on mercury, but I was a bit high. He's also high in Ca and Mg. I showed normal on Mg in hair, but high in packed ethrocyte. I showed low/normal in Ca hair, but I don't have a serum test for Ca. David was also high in

strontium (Sr), but I don't know much about that, and that wasn't measured on any of my tests. Manganese and chromium were both in the low normal range for me, but high for David, esp. manganese, but his levels had dropped from his previous test. So in summary:

We need SULFUR!!!!! I think this somehow plays into Cu and Mo. They both seem to be elements that have an antioxidant effect, but without sulfur they can't do their jobs, or they're wasted somehow. The sulfur somehow stabilizes these minerals, and maybe others. In one of your articles, Mike, a chromium deficiency showed a crusty yeast on the skin, due to Candida. Was that just because of the chromium deficiency or was it because of lack of sulfur, the main component of Glutathione, our major antioxidant. The one that gets used up constantly, because of alcohol, Candida, pollution, chemical sprays, chemicals in deodorants, shampoos, animal dander, pollen, and eating sick animals, who don't have enough stores of their own sulfur. I bet you all are getting tired of me saying that last one, even though it's true. I think we would all be served to take more sulfur, by way of methionine, taurine, cysteine or N-acetyl cysteine, alpha lipoic acid or reduced glutathione.

I have been prescribed by Dr. Gersten, taurine 3000mg day divided in 3 doses, N-acetyl cysteine 1500mg day divided 3x's day, and 75mg of reduced glutathione, in which the latter is combined in some of my supplements and 145mg alpha lipoic acid in combo of vitamins. He has hit me big with the B vitamins, 3x's per day. Some in coenzymated forms and some in normal forms. B6, riboflavin, folic acid, Vit E, and at Path Med. Clinic, they always give selenium with the sulfur aminos, and feel that Mg. zinc, and vandium may also enhance glutathione synthesis.

Here's a bit of interest from "The Healing Nutrients Within". In treating mania, a manifestation of bipolar disorder, Chouinard from McGill Univ. considers tryptophan to be as effective as lithium and even more effective than the antipsychotic chlorpromazine (Thorazine). One mechanism by which lithium works is by promoting serotoninergic neuron transmission. Tryptophan enhances the benefit of lithium.

Another bit for PC. Modlinger and colleagues from the VA Hosp in E. Orange, NJ, showed that 2-10mg of tryptophan daily could stimulate aldosterone, renin, and cortisol, steroid hormones produced by the adrenals. Tryp. can also lower high blood pressure.

I need to study more on sulfur's enhancements in the body, along with getting a better understanding of lithium.

Richard

J. Pisano wrote:

Richard,
Isn't this contrary to what you suggested as Cu being an antagonist to Mo? This is an interesting conundrum....

There appear to be many such conundrums: just like one lot of experts saying taking lots of vit C is good, and another lot (including Mercola) saying lots of C is bad/unnecessary.

Just like Cu assisting absorption of Mo and in other ways not doing so - must be that there are so many biochemical processes involved with so many nutrients that in one scenario A helps with the absorption of B and another scenario where A hinders the absorption of B. Seems like we need to understand and account for all such scenarios for all major nutrients and come up with a balanced strategy catering for all such scenarios..... Wow: a tall order indeed. Just kinda blundering in with a load of one nutrient is accordingly NOT - in the majority of cases (perhaps with the exception of magnesium) likely to work very well - at least in the medium and long term. Short term benefit may be gained, but once the systems involved become overly bombarded with one nutrient, then other imbalances occur which throw balances out in other scenarios thus cancelling out any short-term benefit. As with so many lines of thought/reasoning, I'm often led back to Fran's take on all of this. OK, so Fran's approach might only work REALLY well for Fran, but at the end of the day I can really think of only one type of supplement which has had millions of years invested in its formulation so as to be suitable in terms of providing a suitable balance of all necessary nutrients in the correctly balanced amounts for us mammals, and that my amigos is good wholesome organic nutritious food - although I obviously accept that most soils are now somewhat depleted in certain key nutrients and some judicious supplementation may be required - particularly mag and selenium, and quite possibly moly too. So..... back to my

abundantly stocked supplement cabinet?? Jeez. Notwithstanding all of the above-written which might appear a little negative as regards the supplementation approach (and it is not really meant to), I just want to say again how much I appreciate and enjoy reading the fruits of the endeavours of hard researching individuals such as Carol, Fran, Hans, Jackie, Joe, PC, Richard and others.

Respect,

Mike F.

Thank you, Mike. You know that wine is producing acetaldehydes. I like to give you a hard time. Anyway, a bit more that I found on acetaldehydes, and this one is even more interesting than the first one, about Candida and molybdenum.

How to prevent the damaging effects of smoking, alcohol consumption, and air pollution

Acetaldehyde - A Common and Potent Neurotoxin
Acetaldehyde is hardly a household word in America, yet it is one of the most common neurotoxins in the lives of tens of millions of people. It is a simple substance its chemical formula is CH3CHO yet acetaldehyde insidiously promotes damage to brain structure and function through numerous pathways.
Sources of Acetaldehyde
There are four main routes that bring acetaldehyde (abbreviated here as "AH") into the human brain. These are alcohol consumption, Candida "the yeast syndrome," exhaust from cars and trucks, and cigarette smoking.

Ethanol (more commonly known as alcohol) is the chemical contained in beer, wine, liquor and liqueurs that gets people drunk. These beverages serve as carriers to get ethanol into the drinker's brain, promoting some degree of intoxication. Once in the body, alcohol is broken down into carbon dioxide and water. However, this process takes time and occurs in several steps. The first step occurs primarily in the liver, although other organs such as the brain and kidney can also perform this stage of alcohol detoxification to a slight extent. An enzyme called "alcohol dehydrogenase" converts alcohol into AH. Then another enzyme "aldehyde dehydrogenase" must break the AH down into acetate. Acetate can then serve as a fuel in cellular energy production. (Acetate is a form of acetic acid, the acid that makes vinegar sour.)

However, the conversion of AH to acetate does not always occur quickly or smoothly and therein lies the problem. Research over the last several decades has shown that alcoholics tend to rapidly convert alcohol to AH, but then convert AH to acetate very slowly, thus giving AH a chance to work its mischief in the body.1 And depending on a person's genetics, nutritional status, and exposure to other chemicals such as formaldehyde, which also utilize aldehyde dehydrogenase for their detoxification, even non-alcoholics may have difficulty rapidly detoxifying AH.

The second major route of AH into the brain is through its production by a yeast called Candida albicans. Candida is known to occur in the intestinal tract of virtually all humans to some degree. When present only in small amounts, being kept in check by a healthy immune system and the so-called "friendly flora," such as Acidophilus and Bifidus bacteria, Candida is relatively harmless. Yet due to the modern overuse of antibiotics, birth control pills, and cortisone/prednisone drug therapy, as well as excessive stress (which naturally produces excess cortisone in the body), sugar consumption and malnutrition, millions of Americans now suffer from an excessive growth of Candida in their intestines the so-called "yeast syndrome."2 Candida lives by fermenting sugars to produce energy. Unfortunately for the humans who harbour large colonies of Candida in their gut, the waste by-product of this sugar fermentation by Candida is AH.3 Biochemical research has shown that this AH may combine with red blood cells, proteins, enzymes, and other substances present in the gut or gut lining, and thus travel through the bloodstream to reach more distant parts of the body such as the brain.3 Research has also shown that AH can then detach from the red blood cells or proteins it traveled with through the bloodstream, thus enabling AH to damage cells far from the site of its intestinal production by Candida.3

For those suffering from the yeast syndrome, the ingestion of beer, wine, and liqueurs provides a double-barreled dose of AH. Not only is the alcohol in these beverages turned into AH, but the malt and grain in beer and the sugar in wine and liqueurs provide excellent fuel for Candida to produce the energy it needs to live.2 More AH is the inevitable by-

product of the yeast's sugar fermentation.

When oil, gasoline, diesel fuel, and natural gas are burned, ending up in the air, AH is produced.4 Thus, another major route of entry into the body for AH is through inhaling air laden with vehicle and factory exhaust. People who spend hours commuting in dense freeway traffic, professional drivers such as truck and taxi drivers in urban areas, and even those who live or work in heavily trafficked areas or near freeways or major streets are especially at risk for inhaling small but significant chronic levels of AH.

AH is also produced through the burning of tobacco.7 Thus, heavy cigarette smokers are also at risk of inhaling AH through the inhaled smoke. And while the amounts of AH inhaled through auto exhaust and cigarette smoke may be small compared to that from alcohol, research shows that low-dose chronic AH exposure may still be sufficient to gradually damage proteins, enzymes and other cellular structures in the brain and other organs.21

How Acetaldehyde Damages the Brain
There are many ways that acetaldehyde (AH) can gradually damage brain structure and function through chronic, low-dose AH exposure. The following are some of them.

Acetaldehyde alters red blood cell structure. It has been known since 1941 that AH easily combines with red blood cell membrane proteins to convert the red blood cells into a "time- release capsule" for AH, releasing the AH in the body far from the site where it attached to the red blood cell.3 As this happens, however, the membrane covering the red blood cell becomes stiffer.21 Yet in order to travel through the capillaries, which are the smallest blood vessels and which feed the trillions of individual cells, the red blood cell must be able to fold or deform. The average red blood cell diameter is 7 microns; yet a typical capillary is only 2 microns in diameter. Red blood cells stiffened through chronic AH exposure will have difficulty deforming sufficiently to pass through capillaries. Consequently, red blood cell-carried oxygen to many cells is reduced.3 (Our brains require 20% of all the oxygen we breathe!) In addition, the work of K.K. Tsuboi and colleagues has shown that AH forms stable combinations with hemoglobin in red blood cells. This reduces the ability of red blood cells to accept, hold, and transport oxygen through the bloodstream, which is their primary function.5

Acetaldehyde decreases the ability of the protein tubulin to assemble into microtubules.6 Microtubules are long, thin, tube-like structures that serve several functions in the brain cell. They help provide structural support to the nerve cell, somewhat like girders in a bridge or a building, keeping the nerve cell and the dendrites semi-rigid. Dendrites are the feathery-looking extensions from the main body of the nerve cell which connect nerve cells to each other, with some neurons connecting through dendrites to as many as 100,000 other neurons. Microtubules also serve to transport nutrients and biochemical raw materials manufactured in the cell body to the dendrites. When this raw material transport is compromised, the dendrites will gradually atrophy and die off. Two classic examples of brain pathology involving degeneration of the dendrites in humans are chronic alcoholic brain damage and Alzheimer's disease.

Acetaldehyde induces a deficiency of vitamin B1. Thiamin, or Vitamin B1, is so critical to brain and nerve function it is often called the "nerve vitamin." AH has a very strong tendency to combine with B1, as the work of Herbert Sprince, M.D. (discussed below) has shown.7 Unfortunately, in detoxifying AH through combination with it, B1 is destroyed. Moderately severe BI deficiency in humans leads to a group of symptoms called Wernicke-Korsakoff syndrome.9 This syndrome is characterized by mental confusion, poor memory, poor neuromuscular coordination, and visual disturbances. Its primary accepted cause is chronic alcoholism. B1 is also necessary for the production of ATP bioenergy in all body cells including the brain, and the brain must produce and use 20% of the body's energy total, even while asleep. Vitamin B1 is also essential for production of acetylcholine. Acetylcholine is one of the brain's major neurotransmitters, facilitating optimal memory, mental focus and concentration, and learning. Alzheimer's disease represents a rather extreme case of memory loss and impaired concentration due to destruction of acetylcholine-using brain cells. In a classic experiment reported in 1942, R.R. Willams and colleages found that even mild B1 deficiency in humans continued over a long period of time (the experiment ran six months) products symptoms including apathy, confusion, emotional instability, irritability, depression, feelings of impending doom, fatgue, insomnia, and headaches 8 all symptoms of less-than-optimal brain function.

Below is a range of possible nutrients levels that may offer protection to those suffering from chronic AH toxicity. (divide into 2-3 doses, take with meals)

Nutrient Amount/Day
Coenzyme-A 2000-3000 mg
Thiamin(B1) 50-500 mg
Niacin or Niacinamide (B3)* 50-500 mg
Pantothenic Acid (B5) (Pantethine) 25-200 mg
Pyridoxine (B6) 25-150 mg
N-Acetyl-Cysteine (NAC) 500-2000 mg
Ascorbate(C) 500-3000 mg
Zinc (Monomethionine, Ascorbate or Citrate) 15-30 mg
Gamma Linolenic Acid(GLA)** 120-480 mg
Lipoic Acid (Thioctic Acid) 50-200 mg
Silymarin(Milk Thisle Extract, 70-80%) 200-600 mg
Molybdenum 300-500 mcg

* Those with know or suspected liver disease or gout should use this only their physician's permission. Also those who find the "hot Flush" action of niacin too unpleasant should use niacinamide form of B3.
** From Borage Seed Oil or Evening Primrose Seed Oil.

Acetaldehyde induces deficiencies of niacin and NAD. Niacin (Vitamin B3) is present in the human body primarily in its coenzyme form, NAD.1 NAD is involved in the majority of steps in which sugar and fat are burned for energy in all cells.10 NAD is normally the most plentiful vitamin coenzyme in the human brain. NAD is important as a catalyst in the production of many key, brain neurotransmitters, such as serotonin. Neurotransmitters are the biochemicals that allow nerve cells to communicate with each other. NAD is also the coenzyme that activates alcohol dehydrogenase and aldehyde dehydrogenase, the enzymes that break down alcohol and AH.11 Zinc is also required along with NAD to activate these two enzymes.12

Since the need for NAD in all cells is great, yet the supply is limited, NAD is normally recycled continually during cellular energy production. Yet, when NAD helps detoxify AH, this recycling of NAD is blocked, and an altered form of NAD called "NADH" accumulates, impairing cellular biochemistry in many ways.1, 21 Thus, chronic AH exposure may produce a mild, functional, niacin/NAD deficiency, even in a person consuming a so-called "balanced diet" which meets RDA levels of niacin intake.

Extreme niacin deficiency produces the classic nutritional disease Pellegra with dramatic symptoms, both physical and mental. Since niacin is needed in large amounts for optimal brain function, a mild niacin deficiency tends to produce mostly psychological symptoms. These symptoms may include feeling fearful, apprehensive, suspicious, and worrying excessively with a gloomy, downcast, angry and depressed outlook. Headaches, insomnia, depression, agitation, and inability to concentrate may also occur.13 This profile certainly applies to many chronic alcoholics and Candida patients, who obviously suffer from long-term, mild AH exposure.

Acetaldehyde reduces Acetyl Coenzyme A and impairs cellular energy production. Pantothenic Acid (Vitamin B5) is one of the most critical vitamins for normal brain function. The active form of B5 is Coenzyme A. Coenzyme A in turn is combined with acetate in all cells to form Acetyl Coenzyme A. Acetyl Coenzyme A is perhaps the most pivotal single biochemical in all cellular biochemistry; both sugar and fat must be transformed into Acetyl Coenzyme A to power the Krebs' cycle which produces 90% of all the energy used by every cell in the body, including brain cells.11 Unfortunately, for Acetyl Coenzyme A, however, AH has a strong affinity to combine with Acetyl Coenzyme A. The work of biochemist H.P. Ammon has shown that AH suppresses the activity of Acetyl Coenzyme A in a dose-dependent fashion. He has also demonstrated that the energy-producing activity of cells falls in parallel with the declining levels of Acetyl Coenzyme A as the concentration of AH increases.1 The brain use. 20% of all body energy for normal function. Acetyl Coenzyme A is also necessary for the production of acetylcholine, the memory, learning and concentration neurotransmitter. 14

Acetaldehyde induces a deficiency of Pyridoxal-5-Phosphate (P5P). P5P is the major coenzyme necessary to form virtually all major brain neurotransmitters.10 It is involved in all transamination reactions, whereby cells may convert many different amino acids into each other to satisfy their ever-shifting amino acid needs.10 P5P is necessary to convert essential fatty acids into their final use forms, as well as to turn linoleic acid into the key, nerve cell-regulating biochemical, Prostaglandin E1.15 P5P helps regulate magnesium entry into cells,16 and the level of excitability of

nerve cells is strongly dependent upon their magnesium level. P5P is also necessary to convert vitamin B3, niacin/niacinamide, into the active coenzyme form, NAD.17 Unfortunately for P5P (and we humans who are so dependent on it), AH is known to strongly combine with the protein portion of P5P enzymes in a way that displaces the P5P portion of the molecule. This subjects P5P to an increased rate of destruction and results in abnormally low blood and tissue levels of this coenzyme.1,18

Acetaldehyde unfavorably influences prostaglandin metabolism. Delta-6-Desaturase is the enzyme that converts the common fatty acid linoleic acid into gamma linolenic acid, which is totally absent from any typical diet. Gamma linolenic acid in turn is the only raw material that can be converted into prostaglandin El. Prostaglandin El is a key regulatory biochemical for both nerve cells and the immune system. It also serves to regulate the production of the pro-inflammatory prostaglandin E2. Prostaglandin El prevents excessive production of prostaglandin E2 from the dietary fatty acid, arachidonic acid, which is plentiful in meat, poultry and dairy products. Researchers in prostaglandin biochemistry have discovered, however, that AH is a powerful deactivator of Delta-6-Desaturase.15 AH thus tends to suppress gamma linolenic acid production, which in turn suppresses prostaglandin El production. Low prostaglandin El production "takes the brakes off" production of prostaglandin E2 and a related compound, TXB2, increasing their levels far above normal. The published research of David Horrobin, M.D.,15 and psychiatrist Julian Lieb,l9 has shown high levels of prostaglandin E2 and TXB2, coupled with low levels of prostaglandin El,to be a major causal factor in some forms of depression.

Acetaldehyde promotes addiction to toxic substances. Perhaps one of the most surprising ways AH may alter normal brain function is due to its tendency to combine in the brain with two key neurotransmitters, dopamine and serotonin.20 When AH and dopamine combine, they form a condensation product called salsolinol. When AH combines with serotonin, another product called beta-carboline is formed. Salsolinol and beta-carboline are two of a group of inter-related and interconvertible compounds called tetrahydro-isoquinolines.20 The various tetrahydro- isoquinolines which both animal and human research have shown to occur at high levels in the brains, spinal fluids, and urine of chronic alcoholics are closely related in structure, function, and addictive power to opiates!20 Successfully detoxifying alcoholics have been shown to excrete especially high levels of these opiate-like chemicals in their urine.20 Thus, these AH-generated, opiate-like biochemicals may at least partly explain why alcoholics are so addicted to alcohol, cigarette smokers to cigarettes, and Candida-sufferers to sugar, since all three of these conditions promote chronic excessive body AH levels.20 And, like opiates, these tetrahydroisoquinoline biochemicals would tend to promote lethargy, mental cloudiness and fogginess, depression, apathy, inability to concentrate, etc. These, of course, are symptoms common to both alcoholism and Candidiasis, the two conditions which would tend to generate the highest chronic AH levels in the body.20

The difficulties discussed above that are caused by chronic AH toxicity should indicate to the reader that AH has a significant ability to compromise brain function. A partial summary of AH’s damaging effects on brain function includes the following:

Impaired memory
Decreased ability to concentrate ("brain fog")
Depression
Slowed reflexes
Lethargy and apathy
Heightened irritability
Decreased mental energy
Increased anxiety and panic reactions
Decreased sensory acuity Increased tendency to alcohol, sugar, and cigarette addiction
Decreased sex drive
Increased PMS and breast swelling/tenderness in women.

How Nutrition Can Help
Fortunately, applied nutrition science offers some protection against chronic AH toxicity, even when it is not possible to completely avoid the four main offenders that promote AH in our bodies alcohol, Candida, cigarettes, and heavy auto exhaust.

Herbert Sprince, M.D. and his colleagues published many articles in the 1970’s detailing the results of their

experiments which used various nutrients to protect rats from AH poisoning. Sprince fed a control group of rats an amount of AH sufficient to kill 90% of the control group in 72 hours. The experimental group of rats given the same amount of AH were also given various nutrients, either singly or in combination, that might detoxify the AH. After 72 hours, the death rate for rats given large oral doses of Vitamin C was only 27% (vs 90% in controls), 20% for rats given the sulfur amino acid L-cysteine, 10% for rats receiving Vitamin BI, and an amazing 0% for rats protected by N-acetyl cysteine or lipoic acid. A lower dose combination of C, B1 and either L-Cysteine or N-acetyl cysteine also gave near 0% death rates!7 But, the nutrient doses Sprince administered were rather gigantic compared to RDA levels of nutrients, being equivalent to multi-gram doses for humans. Fortunately, however, most people are not subjected to such high levels of AH, so lower doses of these nutrients would doubtless provide significant AH- detoxifying power when used on a long-term basis.

John Cleary, M.D. has published papers summarizing many doctors' and researchers' successful use of niacin (Vitamin B3) and zinc in alcohol and AH detoxification.1 Since the enzymes that break down alcohol and AH are both B311 and zinc-activated,12 this provides an obvious rationale for their protective use in chronic alcohol/AH toxicity situations. Finally, because chronic high tissue levels of AH impair the normal process of recycling the active form of B3 (NAD) for continual re-use,1 it is obvious why normal dietary levels of B3 might be insufficient to provide optimal brain B3 levels in chronic AH toxicity situations.

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http://www.coenzyme-a.com/effects_of_smoking_alcohol_airpollution_article.html

Here's a study that speaks of molybdenum causing copper excretion, but it seems to be in higher doses.
http://www.epa.gov/iris/subst/0425.htm

So there must be a balance. Right now, I'm only taking a small amt. of copper, and 1000mcg. of Mo. for a week, then I'll back off to 500mcg. I'll retest later and see where these levels are in about 3 mths.

Richard

Richard

Have you read the link I posted above. I think it has your answers about sulphur and molybdenum.

By supplementing molybdenum and histidine (needed in the molybdenum-histidine containing enzymes, sulfite oxidase and cysteine dioxygenase, that oxidize sulfur), along with iron, and the B-complex (preferably in coenzyme form), glucosamine/chondroitin sulfate (stimulates synthesis of the GAGs we studied about above, and is mildly anti-inflammatory without inhibiting the synthesis of Prostaglandins, and more effective when taken together), minerals in sulfate form, such as iron sulfate, and Epsom salts (magnesium sulfate-taken orally it is a good laxative for those that need it), one may supply both the minerals and the sulfate needed to detoxify phenols and other metabolites. When glucosamine gives up its sulfate, it supplies glutamine. Chondroitin is comprised of N-acetyl-D-galactosamine and D-glucuronate. Collagen Type II™ may be even better for it supplies at least 50 other types of sulfate such as heparan, keratan, and dermatan sulfate.

In addition, take an Epsom salts bath (two cups or more in a tub of hot water). It may be best not to use soap as there may be chemical reactions that could be adverse. Soak it up through the skin for 20 minutes, and don't rinse off-and don't worry if the child drinks some of the water. This bath has been shown to increase sulfur content of the blood up to four times.

Fran

Fran,

I'll re-read. The first time I read it I was interrupted frequently and the last time I did, I was very tired. Time to get down to concentrating, which is difficult sometimes. BTW, I started my regimen slowly. I didn't administer the aminos separately as your article indicated, as some are combined, but instead of 3x's a day, I started with once. No problems so far.

Richard

Richard said

I'm now searching for how molybdenum helps with glutamate toxicity, but am not finding anything.

I don’t know if this will help.... but people who are sensitive to free glutamate cannot take suphites either (used in dried fruit, fresh orange juice etc). The symptoms are the same.... so must be connected.

http://www.pdrhealth.com/drug_info/nmdrugprofiles/nutsupdrugs/mol_0332.shtml

"The deleterious effects of molybdenum deficiency were primarily due to the accumulation of sulfite coming from the catabolism of L-cysteine. Sulfite is toxic to the nervous system and molybdenum is necessary for its metabolism to a nontoxic form."

This paper is a LONG discussion on the phenol-sulphotransferase issue

http://www.newtreatments.org/fromweb/sulfur.html

I think perhaps the answer may lie here but I have yet to read it in its entirety.

"PST (phenol-sulfotransferase) is a Phase II enzyme that detoxifies leftover hormones and a wide variety of toxic molecules, such as phenols and amines that are produced in the body (and even in the gut by bacteria, yeast, and other fungi) as well as food dyes and chemicals. These reactions include the breakdown of bilirubin and biliverdin, which are the breakdown products of hemoglobin. There are many varieties of phenols.....

Similar sulfate deficiencies have been reported in people with migraine, rheumatoid arthritis, jaundice, and other allergic conditions all of which are anecdotally reported as common in the families of people with autism....

The PST enzymes are inhibited or overloaded by chocolate, bananas, orange juice, vanillin, and food colorants such as tartrazine. Removal of these from the diet and supplementation of sulfates may well relieve all these symptoms. The lack of sulfation could well be due to the largely carbohydrate diet of most of these children. It is likely a combination of all these things. In any case, toxic compounds of these aforementioned chemicals can build to dangerous levels.

They found that the glucosaminoglycans (gags) in the gut were very under sulfated, and that this causes a thickening of the basement membrane of the gut. IGF (insulin-like growth factor) is important for cell growth. IGF-1 (which is reduced in zinc deficiency) increases the incorporation of sulfate in glucosaminoglycans. .....

Unfortunately, a lack of sulfated gags in the kidneys will allow loss of these sulfates. There is often found low plasma sulfate and high urine sulfate and high urinary thiosulfate as if the kidneys are not able to retain (recycle) sulfate. This needed retention requires the work of a transporter that has been found in "in vitro" studies to be blocked almost completely by mercury and by excess chromium (but not as thoroughly). One study found urinary sulfite to be elevated due to a lack of molybdenum in 36%. Supplementing moly showed improvements in clinical symptoms. Sugar increases the amounts of calcium, oxalate, uric acid, and glucosaminoglycans being wasted in the urine.....

A deficiency of cysteine, or a failure to metabolized it to sulfate, will produce multiple chemical sensitivities and food allergies. Being a major part of the powerful antioxidants alpha lipoic acid and glutathione, a deficiency of cysteine, or a failure to metabolize it into these antioxidants, would greatly affect the liver's ability to detoxify, and would lead to destruction throughout the body by free radicals This would also allow buildup of the heavy metals lead, cadmium, mercury, and aluminum. Supplementation of vitamin B2, B6, B12, folic acid, magnesium, and TMG may normalize metabolism of methionine into cysteine, but vitamin C is needed to prevent cysteine (which contributes its sulfur more readily) from converting to cystine, its oxidized form.....

Those with inadequate protein in the diet, or with poor assimilation, resulting in a deficiency of histidine and other nutrients, form poorly sulfated GAGS robbing the cells of ability to resist infection (that describes 100% of these children). Additionally, it produces dysbiosis (flora imbalance) in the gut. Those with chronic infection shed and replace GAGs so quickly that inadequate sulfate is available even with adequate protein intake.

I think perhaps the whole discussion can shed light.

I also think the conundrum about Cu inhibiting Mo or not - is down to balance. From the reading I have done very high Cu levels will create a shortfall in Mo. However, at the right levels the one works with the other...

Fran

Fran,

Sometimes it's uncanny how much we think on the same path, at the same time. We were posting on the BB at the same time about sulfites, and again today, as my post took me awhile to think about and type, with interruptions. I had seen that link, and I think I put it here, but I can't remember for sure. I found it very interesting, and have read it twice,

and need to print it out and study. I definitely think there's an answer in this information. As I posted last, I believe sulfur has something to do with helping minerals, in particular the minerals that help with detoxification. I'm off to search for a correlation.

Thank you, Fran,
Richard

Richard

Sometimes I skim the posts, take what seems relevant and then go off searching. Unless links are stressed with titbits of info I often ignore. I could have saved myself a lot of time .....

I wonder if research into plants has the same bearing on humans. I found this very interesting. Sulphur does seem to be the common denominator

Institute of Plant Nutrition and Soil Science
Sulphur Research in Europe - Abstract

GLUTATHIONE METABOLISM IN PLANTS IN RELATION TO STRESS TOLERANCE

http://www.pb.fal.de/en/index.htm?page=/en/COST/sia10.htm

Fran

Fran,

This excerpt from your above post:

Our morphological, biochemical and cytochemical observations revealed an important protective effect of these safeners against cadmium toxicity in maize roots, caused by an overproduction of GSH, which leads to a greater amount of phytochelatins (PCs). PCs, with primary structure (g-glu-cys)n-gly, are synthesized directly from GSH by a dipeptidyl transpeptidase. Their most important role is to enable the sequestration of heavy metals, and to carry them inside the vacuole, thus keeping non essential metals below their toxicity thresholds [9].

Very interesting. As you may read on the BB, and I'd like it here in the archives, as well, is that I went out of rhythm by eating Jimmy Dean sausage with MSG today. That is all I ate, along with eggs, so it has to be the MSG. Now in reading your post, and thinking, it occurred to me that glutamate (which I was to the low side in serum aminos) couples with glycine and cysteine to form Glutathione. What if one doesn't have enough cysteine to accomplish this, leaving glutamic acid in an overabundance. Heaven knows we could all use more Glutathione. Maybe sulfur even plays a part here, too. In thinking further, though, eggs are suppose to have a good amount of sulphur, but the key word "suppose" may mean even the eggs don't either, or it doesn't factor in.

Still thinking and searching,
Richard

Fran,

Part of your interesting posting above caught my eye in particular:

""PST (phenol-sulfotransferase) is a Phase II enzyme that detoxifies leftover hormones and a wide variety of toxic molecules, such as phenols and amines that are produced in the body (and even in the gut by bacteria, yeast, and other fungi) as well as food dyes and chemicals. These reactions include the breakdown of bilirubin and biliverdin, which are the breakdown products of hemoglobin. There are many varieties of phenols.....

Similar sulfate deficiencies have been reported in people with migraine, rheumatoid arthritis, jaundice, and other allergic conditions all of which are anecdotally reported as common in the families of people with autism....

I was jaundiced as a baby and have high (above top of range - into Gilbert's Syndrome territory) blood bilirubin (and urate) as an adult, and also displayed some signs of mild autism as a child (obsessive behaviour - errrr... I still have THAT as an adult!). I also am plagued - on and off albeit mildly in the main - by candida. This discussion about sulphur is most interesting.

Gotta go, later,

Mike F.

Hello All,

In light of the fact that I had a breakthrough of flutter, that I feel was caused by a direct addition of MSG to the sausage that I ate, it made me step back and think a bit differently. This is what I see:

I'm low in the sulfur containing aminos, which are important antioxidants. I'm low in molybdenum, which is important for breaking down acetaldehydes and sulfites, with the latter being important for turning sulfites to useable sulfates. I was low to normal in glycine, a bit to the low side in glutamate, but pushing the high side of glutamine, which can be converted back to glutamate.

I've been eating a pretty high diet of proteins, with a lot of salads and vegetables. The few cheats that I've had, such as an occasional home baked cookie, haven't caused any problems, but the cheats of sausage and bean dip and chips have. At first I thought the bean's carbs were the problem, but now know it has more to do with the addition of MSG in its various guises. As you'll notice in the study below, when protein levels are upped, research indicated that free glutamate went down. Their hypothesis is, that glutamate attaches to cysteine to form glutathione. This made me think further. When we're eating anything of a carbohydrate fashion that has added MSG, or anything with free glutamate, and we haven't supplied its counterparts, glycine and cysteine, then it has a free run to cause neurotoxicity in an unnatural way. Because the bond between glutamate and salt is a weak bond, we need to supply the nutrients that create the strong bond; glutamate, glycine and sulfur, with the last two being the only added nutrients. As you all well know by now, my stance is, that we are definitely lacking sulfur, and you all know why. In my case, I will avoid MSG, but I am going to continue on with my regimen of vitamins, which include glycine (Mg. glycinate) and cysteine (alpa lipoic acid, reduced glutathione, taurine, and some cysteine). If I start having adverse reactions, then I'll up my intake of sulfur further and see what happens. If that doesn't work then I guess I'll eliminate the supplements.

So this is what I propose to anyone out there interested. If you decide to go to the movie theater and have popcorn, or go out to the restaurant and have something that could have free glutamate, take your Mg. glycinate and N-acetyl cysteine with your meal or snack, and see what results you get, but in the meantime, take it anyway, as a precautionary measure. I'm not a doctor, so I must add, that you need to do this under the care of your doctor.

I've also thought about why PC has more AF in HI, while Hans did not. They're certainly both relaxed. Could the mere difference be molybedum. This could be a stretch and its all a thinking process, but could PC be low in Mo and Hans be OK. Hawaii is known to have more sulfur, because of the volcanic rock, but is this kind of sulfur processed through the sulfite oxidase enzyme to a more usable sulfur. While Hans is swimming in the ocean, he's absorbing and processing the much needed minerals and sulfur, which left him in NSR, whereas PC is swimming in the same water, and his body isn't processing it the same. It may not have anything to do with this. They're just thoughts.

Here's the study and a few excerpts:

It seems relevant, in relation to the effect of chronic protein intake on plasma levels as noted above, that the rates of de novo synthesis of glutamate and glutamine change with the dietary protein level. As summarized in Table 5 , Matthews and Campbell (1992) reported a significant decline in both glutamate and glutamine de novo synthesis rates when dietary protein intake was increased from a level sufficient to meet requirements [0.8 g protein/(kg · d)] to a high intake

of 2.2 g protein/(kg · d). This finding presumably helps to explain the fall in plasma glutamate/glutamine levels with high protein feeding. However, the site(s) at which this response occurs (possibly muscle; Nurjhan et al. 1995) and the biochemical mechanisms involved remain to be defined. Furthermore, it will be important to understand how peripheral and visceral tissues/organs interact to achieve the new steady-state changes in the plasma amino acid concentrations.

On the basis of the work of Windmueller (1982) , Windmueller Spaeth (1974 and 1975) and Neame and Wiseman (1958) , it was clear to Munro in 1979 that the intestinal tissues were responsible for a significant metabolism of dietary glutamate and glutamine. Since then, elegant stable isotope studies have extended our understanding of the quantitative handling by the intestine of dietary glutamate and glutamine, with a confirmation that little or no dietary glutamate enters either the systemic (Matthews et al. 1993) or the portal blood supply; it is significantly oxidized in the splanchnic region (Battezzati et al. 1995) and probably in the intestinal mucosa (Reeds et al. 1996), where it serves as a significant energy yielding substrate. Additionally, a significant fate of glutamate is glutathione synthesis (Reeds et al. 1997). These findings are discussed in detail elsewhere in these proceedings but they are highlighted here as exciting examples of our understanding about the quantitative aspects of the physiology of glutamate metabolism. A net effect of the extensive intestinal metabolism of glutamate is the achievement of relatively stable plasma concentrations of glutamate and glutamine throughout the fasting and fed periods of the 24-h day (Fig. 6) (glutamate data not shown).
http://www.nutrition.org/cgi/content/full/130/4/892S#SEC4

I need some input and stimulation. Thank you.
Richard

Richard

It's fantastic to have someone else researching WHY about free glutamate. I too can get away with carb cheats- even the odd sugar treat/cheat. But on no occasion have I got away with an free glutamate cheat. As much as I used to love good old steak and kidney pie made with beer...

According to this site what you need to detoxify is B6, Magnesium, niacin (B3), N acetyl cysteine, B2, pyruvate, vitamin C and zinc.

http://sportscarecenter.com/scc/id17.html

"The glutamate portion of monosodium glutamate is a stimulant that people react to in varying degrees. A simple way to test our body's ability to digest and eliminate this toxin is to place a tiny dab of glutamate on the tip of the tongue and see how long it takes for the taste to go away. This product is available free of charge at the Sports Care Center. Ideally the taste will dissipate in 2-5 minutes. In an overly sensitive person the taste may last as long as two days. Yes, that means a child may be "wired" for as long as two days on a single dose.

The vitamins necessary to break down and eliminate these toxins are B6, Magnesium, niacin (B3), N acetyl cysteine, B2, pyruvate, vitamin C and zinc. Many of these vitamins are very commonly found deficiencies in both children and adults. Properly guided supplementation of these nutrients would help one to reduce the negative effects of the MSG neurotoxin. However, avoidance wherever possible is the best prevention."

I thought it was interesting about the test but know I won't dare try it.

As to the theory between Hans and PC - I could buy that. I just know that free glutamate (I try not to refer to it as MSG as people think they avoid that when in reality...??) is implicated in us all. The other mineral and vitamin deficiencies we individually have may just make the difference between adrenal and vagal.. I'm sure a connection was tenuously made in the Conference Room at one point. I will go back and check.

I still believe avoidance is the best measure as it is not natural for the body to consume the levels we do today. Sorry it was not very stimulating.

Fran

Fran,

I found it stimulating, especially about the taste test and other vitamins that I didn't mention. I agree and will definitely avoid anything that knowingly has free glutamate in it, but I might try a test at some point, and see what your listed regimen does to counteract the effects. I at least have the crutch of flec. to fall back on. You don't.

I feel like everyone is getting an ablation, and is going to abandon us, and even though I'm happy for them and hope it works well, I will miss them if they go. I just wish they could of held out a bit longer, and tried some other combinations of nutrients, esp. upping or taking sulfur with the glycinate they took. Sulfur is haunting me. Tryptophan haunted me, and I was low. On the other hand, molybdenum never entered my mind.

Thank you, Fran.

Richard

OK, it's all starting to fall together. You stimulated the heck out of me Fran, by picking up on the idebenone in the Joe South article posted on the BB, under "Gutamate Again". This is where sulfur ties in and idebenone may be our new friend, along with Glutathione.

Idebenone attenuates neuronal degeneration induced by intrastriatal injection of excitotoxins.

Miyamoto M, Coyle JT.

Department of Psychiatry,
Johns Hopkins University School of Medicine,
Baltimore, Maryland 21205.
Exp Neurol 1990 Apr;108(1):38-45

ABSTRACT

Previous studies with the N18-RE-105 neuronal-like cell line and primary cortical cultures demonstrate that glutamate can produce a calcium-dependent, delayed form of neuronal degeneration that results from its competitive inhibition of cystine transport, which leads to cellular glutathione depletion and death by oxidative stress. Idebenone, a centrally active antioxidant used to treat multiinfarct dementia, protects cells from this form of glutamate-induced cytotoxicity in vitro. In the present study, we have examined the effects of systemic treatment with idebenone on the neurotoxic consequences of intrastriatal injection of kainic acid, quisqualic acid, or quinolinic acid, an NMDA receptor agonist, on neuronal degeneration. Striatal damage was assessed by quantitative neurochemistry with measurement of choline acetyltransferase activity and glutamate decarboxylase activity, by histochemical analysis for acetylcholinesterase and NADPH diaphorase staining and by behavioral assessment of circling produced by systemic apomorphine treatment 10 days after the unilateral lesion. The results indicate that treatment with idebenone provides significant protection against the neuronal degeneration induced by intrastriatal injection of kainic acid and quisqualic acid, but not the NMDA receptor agonist, quinolinic acid. The results suggest that oxidative stress may contribute to the proximate cause of neuronal degeneration induced by quisqualate and by kainate receptor agonists and that the mechanisms of neuronal degeneration caused by quisqualate/kainate receptor agonists differ from those associated with NMDA receptor agonists.
http://www.idebenone.org/Idebenone/idebenone-research-30.htm

Input?????

Richard

Richard and Fran,

I read all you post with great interest. A quick question, however, just popped into my head having just read Richard's latest abstract. The abstract refers to neuronal damage. Forgive any naivety here, but whilst it is beyond doubt that MSG affects the brain (and I guess by implication the ANS..... I'm sorta answering my own question to a degree here!), how does it DIRECTLY affect the heart? Fran, did you once post something whilst debating this point with PC which confirmed that the heart was indeed affected by MSG - i.e. directly (as if any ANS effects aren't enough....)??

Yours studiously (within time constraints) following the fruits of your hard work,

Mike F.

Mike

I have taken the liberty of cutting and pasting PC's most recent post on Idebenone to answer your question. He is much more eloquent than me and to the point. Square brackets mine.

"Glutamate is the neurotransmitter for vagal tone. Although the blood brain barrier is impermeable to glutamate, glutamate can diffuse into the hypothalamus and more importantly into the nucleus tractus solitarius (NTS) in the medulla oblongata. This latter structure is circumventricular and is easily accessed by glutamate from the cerebrospinal fluid.

[I just want to add here that hypoglycemia will actually allow the BBB to become permeable to free glutamate as well]

Please see figure 5 at
http://webteach.mccs.uky.edu/COM/DLOTW_cd/na_images_fr_2b.html
for an actual photo demonstrating how close the NTS is to the fourth ventricle.

The NTS is responsible for initiating signals to the nucleus ambiguus (NA) that control HR. This usually operates through stretch receptors in the lung (respiratory sinus arrhythmia), but dietary glutamate should, at least theoretically, be able to stimulate these same vagal motor nerves (NA) to the heart through the NTS.

[I believe that this is also the reason why many people get asthma and is due again to free glutamate - and a thought just struck me that it may be why some of us have problems breathing with AF]

Whenever I eat seaweed, e.g., sushi, my odds of triggering AF a few hours later are GREATLY increased. My PACs become markedly increased, my HRV increases, and HR decreases. This time frame fits very nicely with the time required for the glutamate to diffuse from the blood into the CSF and into the fourth ventricle and from there to the NTS. "

Fran

Richard

Like you I think everyone is choosing ablation and wonder if it is the long term treatment that we all hope for. My thoughts on the matter are, and I hope I am wrong here - but if like in my case - the problem is free glutamate, then surely if those with ablation continue without life style changes the problem will either 1) build up and come back again, 2) affect another part of their anatomy. I have often pondered over why ablation actually works for some and how if it was due to glutamate toxicity a few scars in the right place stop the AF. I came up with this conclusion. There are glutamate receptors all over the body. Breast, eyes, lungs, pancreas etc. The only study (I have found) that involves glutamate receptors in the heart are in a rat study article I posted in the CR under Managing Afib – Are we getting close?

http://jneurochem.highwire.org/cgi/content/full/75/6/2472

I have often wondered if there could be glutamate receptors in the pulmonary veins too. If this was the case - could ablation effectively be burning out these receptors so that it had no stimulation on the heart???? And without knowing it the hot spots where AF is noted is a receptor site??

"idebenone provides significant protection against the neuronal degeneration induced by intrastriatal injection of kainic acid and quisqualic acid, but not the NMDA receptor agonist, quinolinic acid."

Do you know what kainic acid, quisqualic acid and quinolinic acid are and what they are derived from?

Fran

FYI, the following site contains links to many references/studies on idebenone, such as its usefulness for treating Freidreich's ataxia and Alzheimer's disease, for improving cerebral mitochondrial oxidative metabolism, etc.

http://www.idebenone.org/

Nicole
