

THE AFIB REPORT
Your Premier Information Resource for Lone Atrial Fibrillation
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VIRTUAL LAF CONFERENCE

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SUBJECT: Mitral Valve Prolapse, Magnesium & LAF

In the previous research on Cardiac Fibrosis, a paper on magnesium deficiency and how it relates to mitral valve prolapse (MVP) was referenced. I mentioned to Hans that the paper noted some interesting associations, such as the incidence of MVP and candidiasis, and other symptoms common among afibbers, indicated from conversations here on the BB. He suggested I post this topic for examination in the CR.

It would be interesting if everyone would acknowledge if they have had a MVP diagnosis by responding to this post. ...also indicate if they have any of the symptoms or conditions of MVPS even if they do not have MVP.

I'm interested because I have my diagnosis based on echogram. Mine is very insignificant, could easily be missed even on the electronic survey, and is definitely not detectable by auscultation. Nevertheless, after reading more, I note that I definitely have other symptoms that go along with a syndrome called MVPS which I'll cover below. In a separate post, I'll list the symptoms I have that match what's indicated.

Here's the article and an excerpt. If you want to examine this topic, reading the article is a must.

I've also included other references at the end which may be of interest to anyone who determines from the common symptoms that they have something to consider. As I said, without an echogram...I wouldn't have known, but I have so many symptoms that fall in the MVPS category and this explains a lot of the problems I've had/have over the years....along with the AF, of course.

Apparently, many MVP patients remain asymptomatic until arrhythmia surfaces. (I lost the reference for this – surfing with too many open windows. Sorry.)

This is another Chicken-and- Egg thing. Does AF cause MVP or the reverse?

I didn't spend a huge amount of time finding studies that show absolutes, but the common symptoms certainly point to some association....and the very fact that I had/have so many of the common or overlap symptoms, indicates to me it is important to acknowledge, since people could be treated for one condition when they really have the other.

And it also appears that magnesium deficiency, once again, plays an important role in this condition.

Read on.....

Magnesium Deficiency in the Pathogenesis of Mitral Valve Prolapse

Leo D. Galland, Sidney M. Baker, Robert K McLellan
Gesell Institute of Human Development, New Haven, Conn., USA
<http://www.mdheal.org/magnesi.htm>

The MVP Syndrome: Dysautonomia

Symptoms most commonly encountered in patients with MVP are chest pain, dyspnea, fatigue, dizziness, syncope, palpitations and anxiety [62, 96].

A cardiac origin for these symptoms has not been established and Wooley [96] and Boudoulas et al. [8] propose that they are neuroendocrine in origin. This group found higher urinary catecholamine excretion, higher blood glucose and shorter systolic time intervals in patients than in controls [8]. Elevated plasma catecholamine levels were reported by a Canadian team [79]. Gaffney et al. [41] measured cardiovascular responses to a series of maneuvers in 35 women and concluded that those with MVP had decreased parasympathetic, increased adrenergic and normal P-adrenergic tone.

On the other hand, orthostatic hypotension, a common finding among MVP patients with dizziness, appears to be due to excessive adrenergic tone [85]. Resting bradycardia, a sign of parasympathetic overactivity, is common in MVP [68] even among patients with elevated catecholamine levels [79].

After analyzing cardiovascular and respiratory responses to postural change and Valsalva maneuver, Coghlan et al. [16] concluded that different patterns of dysautonomia may occur in MVP; cholinergic hyperactivity is as frequent as adrenergic, especially in patients with fatigue and poor concentration.

MVP occurs in 15-50% of patients with panic disorder [51, 64, 76, 92], a condition of adrenergic hyperfunction [14, 77], and in 40% of patients with hyperthyroidism [13], another hyperadrenergic state associated with autoimmunity.

The absence of MVP in juvenile hyperthyroidism [11] and the finding of MVP in 41 % of patients with autoimmune thyroiditis [74] suggest that adrenergic hyperactivity is not a cause of prolapse. It is more likely that dysautonomia, autoimmune phenomena and MVP are all manifestations of the same disturbance.

Galland [42] found a 46% prevalence of MVP in patients being treated for chronic infection with *Candida albicans*, - these patients all had symptoms of hypersensitivity to *Candida*, as well as chronic infection. Recently, investigators at The Omega Institute in New Orleans, La., reported an unusually high frequency (96%) of MVP in women with infertility due to pelvic fibroadhesive disease [4]

From a nursing continuing education site comes this information:
<http://www.continuingeducation.com/nursing/mitral/mitral.pdf>

In most individuals, MVP is benign and causes few symptoms, if any. Those with more significant abnormalities are at greater risk for serious complications such as bacterial endocarditis (when regurgitation is present), arrhythmia and sudden death.

Approximately 60% of individuals with MVP never exhibit symptoms and are unaware of the condition. And the symptoms that occur also occur commonly in the population without MVP. Some symptoms related to the mitral valve malfunction include:

Irregular heartbeat, or palpitations, particularly when lying on the left side.

Mild, non-specific chest pain lasting from a few seconds to several hours, occurring predominantly at rest and occasionally during exertion.

Mild shortness of breath, orthopnea or paroxysmal nocturnal dyspnea.

Fatigue and weakness after slight exertion (sometimes diagnosed as chronic fatigue syndrome).

MVP Syndrome – some crossover exists between the symptoms of MVP and MVPS. 40% of patients with MV also have an imbalance of the autonomic nervous system called dysautonomia. When this system is out of balance, it can cause a number of symptoms as MVPS and while rarely dangerous, it causes many people a good deal of discomfort.

Symptoms include.
Fatigue – most common

A sudden feeling of anxiety, fear of impending death or panic attack and occurs with no apparent reason, even waking one from sound sleep.

Migraine headache, irritable bowel, flushing and or cold extremities, palpitations or pounding heartbeat, dizziness or fainting.

Individuals with MVPS also have a higher incidence of TMJ involvement, fibromyalgia, PMS fibrocystic disease, infertility, seasonal affective disorder, depression, altitude sickness, sea sickness and endometriosis.

From the book, *The Miracle of Magnesium* – parallel symptoms of interest.....

<http://www.ctds.info/mvp1.html>

Conditions Linked to Mitral Valve Prolapse and Conditions Linked to Magnesium Deficiency -both include:

Allergies

Anxiety disorders

Excess catecholamines

Fibromyalgia

Keratoconus

Migraines

Myopia

Depression

Also from the book - check this excerptMitral Valve Prolapse, What Causes It? (shortage of hyaluronic acid?)

<http://www.ctds.info/mvp1.html#hyaluronic>

From the book, *The Miracle of Magnesium* there is a section online for viewing

Other References:

Considerations on the pathogenesis of mitral valve prolapse.

Zeana C, Briciu M, Florescu A, Dumitrescu C.

Med Interne. 1985 Jul-Sep;23(3):165-70.

PMID: 4048797 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=4048797&dopt=Abstract

Mitral valve prolapse: causes, clinical manifestations, and management.

Devereux RB, Kramer-Fox R, Kligfield P.

Ann Intern Med. 1989 Aug 15;111(4):305-17.

New York Hospital-Cornell Medical Center, New York.

PMID: 2667419 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&dopt=Abstract&list_uids=89334187

Pathology and histochemistry of mitral valve prolapse]

Watanabe C, Sugiura M, Ohkawa S, Ito Y, Toku A, Maeda S, Kuboki K, Imai T. Department of Clinical Pathology, *Tokyo Metropolitan Institute of Gerontology. J Cardiol. 1993;23(1):69-77.*

PMID: 8164136 [PubMed - indexed for MEDLINE]

Therapeutic effect of a magnesium salt in patients suffering from mitral valvular prolapse and latent tetany.

Simoes Fernandes J, Pereira T, Carvalho J, Franca A, Andrade R, Nogueira Pereira J, Rodrigues JC, Laires MJ, Halpern MJ.

Magnesium. 1985;4(5-6):283-90.

PMID: 3914582 [PubMed - indexed for MEDLINE]

http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=3914582&dopt=Abstract

Mitral Valve Prolapse in Active Patients: Recognition, Treatment, and Exercise Recommendations

Elizabeth Joy, MD

THE PHYSICIAN AND SPORTSMEDICINE - VOL 24 - NO. 7 - JULY 96

http://www.physsportsmed.com/issues/1996/07_96/joy.htm

Jackie

I'm surprised (and also interested) that we're pursuing this subject (MVP) as I understand it is "an underlying heart condition" and so not associated with LAF, the primary focus of this site. However, I was diagnosed with AF and MVP with moderate regurgitation as of July 2001. My cardio said I might need valve repair in the future.

The MVP was revealed by ultrasound and the regurgitation was heard by a nurse when I was sotalol loading in the hospital.

My AF is virtually totally controlled by sotalol.

I have allergies, occasional visual disturbances related to migraine (thankfully w/o the headaches), and myopia.

Will

Will, I am glad Sotalol is controlling your afib. I took it for six years and it started losing its' effectiveness. I had to switch to flecainide once again in January. It is helping somewhat but nothing like it did when I was taking it before switching to Sotalol.

Glenn

Jackie,

Sorry I can't add much either.

There is another nice article on MVP and Mg that I couldn't find amongst those you listed at

<http://www.mgwater.com/prev1808.shtml>

PC

Jackie:

On my last few echos, the tech did note that I have very mild MVP, my EP did not pick this up from listening to my heart. He is a very cautious EP and has given me a script for an antibiotic to take whenever I have any dental work done.

My grandmother had what they used to call "leakage of the heart", that was many years ago and they didn't know how to treat it, I remember she became very weak and was bedridden and within a few months passed away.

As you know I have thyroid disease, and many thyroid patients seem to also have MVP. I have some of the symptoms you cite, but they can be attributed to my thyroid, stress and afib, difficult to know.

Liz

That was an excellent summary by Jackie. I have had MVP for many years, and mostly suffered extra beats off and on with occasional vague chest pains. I also had an episode of parox. AF every 5 or 6 years. The worst problem was when after a stressful episode I developed mitral valve prolapse syndrome. The symptoms were mostly PVCs and PACs numbering approximately 5000 a day (Holter monitor), along with weakness, chest pain, anxiety, and frequent episodes of AF of relatively short duration. Most of the time, I felt as if I drank 50 cups of coffee. I'm sorry I didn't get a catecholamine level. Fortunately, around that time I ran into an article from Warsaw, Poland from the Amer. J. Cardiology, March 15, '97, on the use of magnesium in individuals with heavily symptomatic MVP. I also noted around that time that if I took a shot of Maalox or Mylanta, I felt much better. I then started on Mag-Ox 400 twice a day along with a beta blocker, and I've been fairly comfortable since with AF so far once or twice a year for about 3 - 4 hours. I consider myself fairly lucky so far and hopefully my regime will continue to keep me comfortable. As far as the MVP, I take antibiotics when I have dental work. The only other consistent finding that I've had, and I still have is extra beats after salty meals.

Bernie

Jackie,

Even though I wasn't diagnosed with MVP, I find your post very interesting. A post I presented to you a while back, pertaining to taking MSM, which depleted molybdenum stores, also mentioned hyaluronic acid. I didn't pay much attention to what that was at the time, however. The focus on Mg is extremely important, but the focus on sulfur containing aminos should also be at the forefront, and I still believe this is the missing link. And link, is the key word, because sulfur links the chains of aminos together, and is Mg dependent. It methylates, holds tissues together, and detoxifies the body, and we are not getting enough of it, because of the constant demands of our environment. I can't stress this enough. If I had to summarize all I've learned in the last year, and tell someone what to do, it would be this. Avoid MSG, take a multi with additional Mg, and take methionine or N-acetyl cysteine, or both, with a wholesome diet. That would be at the top of my list, but of course other nutrients are important; these would just be what I find the most important.

Here's what the link I mentioned says, and there is a diagram of the pathway of methionine:

Chondroitin sulfate, glucosamine sulfate, N-acetyl glucosamine, hyaluronic acid, and mucopolysaccharides are all building blocks of cartilage, and they are all dependent upon sulfur groups for complete synthesis of healthy cartilage tissue. These sulfur groups are provided by a correctly functioning homocysteine pathway.

Additionally, resources such as Jonathan Wright, MD point out that MSM can create a molybdenum deficiency because exogenous, (outside) sources of sulfur will drain molybdenum resources because the molybdenum is essential for sulfur metabolism. On the contrary, NATURAL provision of sulfur makes what the body needs, and does not create an excess pool which then must drain molybdenum to be metabolized.

<http://www.nutriwest.com/articles/homovmsm.htm>

When I get back, I'll do some additional researching and see what I find on hyaluronic acid. Thank you, Jackie.

Richard

I'm going to comment on a reference that I used in the originating post for this thread.... the one that I thought was a book excerpt. It turns out it is a summary article on the web site listed in the reference. When I attempted to learn of the author's credentials, I was told I was not permitted to quote her or anything from her site...but since I follow the fair use law for educational purposes and I am not copying and selling this information.... I'm going ahead with this article....just as if you had gone there and read it yourself - on line. So don't "turn me in."

But to start - I'd like to mention my symptoms as they relate to the MVP and magnesium deficiency article that started this whole topic in the first place.

In my case, with the virtually undetectable MVP, I was still given a diagnosis of LAF with no underlying heart disease or structural defect. This was confirmed by two cardiologists.

So what does that say? MVP is not a underlying factor? or if it is as insignificant as mine, it is ignored in the diagnosis description?

Additional matching symptoms I've had/have include:

Food allergies – former
Fibromyalgia - ongoing/ recurrent
Fatigue - recurrent
Myopia- present for 35 years; corrected naturally
TMJ - former
Magnesium deficiency – former
Excess catecholamines
Candida overgrowth – former
Hypothyroid - former
Orthostatic hypotension
Parasympathetic overactivity
Sea sickness (ongoing- actually motion sickness)
Endometriosis – former (actually fibroids)
PMS, fibrocystic breast disease - former

My original question remains the same: does the presence of MVP have any influence of the occurrence of AF? Or does the overuse of the valvular apparatus because of long standing AF, create the MVP tendency.....

In one of my references, there is a summary article on MVP and connections to magnesium deficiency as well as hyaluronic acid deficiency. The author of this summary, Sandra Simmons, brings up some interesting connections and one might speculate from her research that an underlying cause of MVP could be from these deficiencies. I originally thought it was from book excerpts, but it is apparently a compilation of the author's research.

Ms. Simmons posits, does magnesium and hyaluronic acid deficiency predispose to MVP?

·Heart valves in MVP show hyaluronic acid abnormalities. The heart valves probably become defective when the body does not have sufficient magnesium to produce hyaluronic acid. With degraded hyaluronic acid, the valve stretches out and functions suboptimally.

·Magnesium supplementation often aids in the symptoms of mitral valve prolapse. It's probably because more magnesium makes it easier for the body to make hyaluronic acid which makes for better functioning heart valves.

Magnesium Deficiency in the Pathogenesis of Mitral Valve Prolapse - "Most features of the MVP syndrome can be attributed to direct physiological effects of magnesium deficiency or to secondary effects produced by blockade of EFA desaturation. These include valvular collagen dissolution, ventricular hyperkinesis, cardiac arrhythmias, occasional thromboembolic phenomena. autonomic dysregulation and association with LT, pelvic fibrosis, autoimmune disease, anxiety disorders, allergy and chronic candidiasis." (from the Leo Galland paper)

A paper in the journal, Magnesium, noted that magnesium deficiency hinders the mechanism by which fibroblasts degrade defective collagen, increases circulating catecholamines, predisposes to cardiac arrhythmias, thromboembolic phenomena and dysregulation of the immune and autonomic nervous systems. The paper authors noted that magnesium therapy provides relief of MVP symptoms.

·Up to 75% of the people with fibromyalgia have the disorder. Seventy-five percent of the people with fibromyalgia have

mitral valve prolapse. Mitral valve prolapse is thought to occur in about 10% of the population in general, so this statistically is quite significant. Mitral valve prolapse is another disorder also closely linked to Mg deficiency. They also have hyaluronic acid abnormalities.

·Some researchers think mitral valve prolapse syndrome is a mild form of a connective tissue disorder.

·A lack of hyaluronic acid would provide a logical explanation why people with connective tissue disorders not only have many symptoms linked to defective connective tissue, such as lax joints and stretched out heart valves, but also why they tend to have a high rate of bacterial and viral infections. It would also explain why symptoms of MVP often appear or increase after a viral illness. If a person's hyaluronic acid is less than robust, perhaps this make it easier for bacteria to break through their protective hyaluronic acid barrier.

·MVP can occur as a result of rheumatic fever.

Mitral Valve Prolapse: Is Magnesium a Cure?

Many people with mitral valve prolapse respond to magnesium treatment, but some do not. However, this doesn't mean magnesium isn't a factor in MVP, it just means there may be more than one factor involved in the disorder.

Hyaluronic acid depends upon other nutrients, too, like zinc, and there are other factors that degrade hyaluronic acid besides hyaluronidase, such as cigarette smoke. Since there are multiple causes of hyaluronic acid abnormalities, in turn it would be logical to expect there to be multiple causes of mitral valve prolapse.

Many medical web sites state that MVP is a normal variation in the population because it is so common. I'm not so sure this is a valid conclusion. Being common does not mean the same thing as being normal, or even desirable. Thirty percent of the adult U.S. population is obese, however no one is saying that obesity is "normal". There are many conditions such as breast cancer, heart attacks and osteoporosis that are even more common than mitral valve prolapse, but that doesn't mean that any of these conditions are normal, let alone desirable.

Summary

If we put all of the clues together about mitral valve prolapse, then there are some highly logical ways in which the data fit together. In the chart in my next section, Mitral Valve Prolapse Syndrome: Symptoms and Diet Treatments, I've assembled information gathered from a variety of different studies on PubMed and put them all together into a logical scenario. It is probably a highly simplified version compared to reality, however, it does provide logical explanations for many of the features we know about MVP.

If the root cause of mitral valve prolapse was in fact degraded hyaluronic acid and/or an excess of hyaluronidase, an enzyme that breaks down hyaluronic acid, then everything we know about the disorder would make sense.

It would explain why mitral valve prolapse is commonly, but not always, linked to magnesium deficiencies, why MVP occurs as a result of rheumatic fever and why people with disorders like Down syndrome, Marfan syndrome and/or fibromyalgia often have both mitral valve prolapse and hyaluronic acid abnormalities, too. I

don't think there is anything we know about mitral valve prolapse and its associations that doesn't fit in with this model, thus providing a pretty good clue that is on the right track, and that hyaluronic acid is indeed most likely a significant factor in mitral valve prolapse.

End of excerpts from the online article at this web site:

<http://www.ctds.info/mvp1.html#hyaluronic>

Jackie

My original question remains the same: does the presence of MVP have any influence of the occurrence of AF? Or does the overuse of the valvular apparatus because of long standing AF, create the MVP tendency...

Jackie,

If the atria are not sustaining a contraction long enough for the ventricle to complete its contraction then the MV is not going to be supported by the blood pressure in the atria against the blood pressure in the ventricle. The pressure difference across the MV will be much larger and this could lead to MVP over time. If this is the case then MVP is an electrophysiological problem like afib. MVPS and afib Syndrome I think are one and the same and related to the electrophysiological situation in the atria.

Just my view of the world

Angus

Jackie wrote:

It would be interesting if everyone would acknowledge if they have had a MVP diagnosis by responding to this post.

I can't add much other than to say MVP is not a problem for me (I've had 4 echo scans since my first AF in 1998)

--

James D

I used to follow a very active MVP bulletin board. Ninety percent women-- many at "wits end" over their MVPS symptoms. No one ever had AF, but many had other aggravating arrhythmias including PACs, shortness of breath, dizziness, fatigue, and depression.

So far, only a few people have reported here with trace MVP, except for my own moderate MVP. I'm interested in the question, "Does one cause the other, and which one?"

Will

I have had MVP with chronic AFIB for the last 18 months. My cardiologist has referred me to CCF and Dr. Natale for ablation consultation with a repair to the MV later. I am on Sotalol, digoxin, and warfarin. My heart rate had been under control with just the Sotalol. Recently it had started to go up and digoxin was added. Cleveland Clinic had answered a forum question and said that they could repair the valve and take care of the AFIB with a MAZE procedure at the same time, or do an ablation and then the valve repair later. My cardiologist told me that it is very hard to know which one came first. He has been taking his time with me to see if the heart would start to heal as we managed the AFIB. It is much better now, but the fact remains that a valve repair will be needed.

Woody

I was diagnosed with MVP with very mild regurgitation two years ago. It was found on a stress echocardiogram done as part of the workup for newly diagnosed atrial fibrillation. My obstetrician noted a murmur during my third pregnancy, many years ago, but it was not considered significant and no other MD has mentioned it over the ensuing years. My current internist can sometimes hear it, sometimes not. I believe it may depend somewhat on how well hydrated I am at the time. I have some of the MVPS symptoms such as migraines (no longer since being on a beta blocker), palpitations, sharp chest pain, and, most annoying of all, the waves of anxiety, panic and fear that come about without warning, as you noted, even awakening one from sleep. Magnesium supplements seem to help, and I believe calcium seems to make things worse for me although I'm still trying to sort this out. My cardiologist credits the MVP and regurg as the reason for the AF. However, I'm 68 years old so age can also factor in, I suppose. The AF occurs for me, so far, only 2-3 times a year, lasting only 8 hours, so I'm quite fortunate compared to many others who post here. Starting when I was around 6 years of age until probably the age of 50, I would have episodes of fainting which were associated with arising from a prone to the upright position or with what I now realize was probably a hypoglycemic state. At the time, no MD was ever able to provide a reason for the syncopal episodes. I'm guessing that MVP or MVPS most likely was responsible.

Laura

Hi Jackie -

Just noticed this topic here.

I was diagnosed with MVP years before I developed afib. I believe the first studies on MVPS were done at the Univ of Alabama. I definitely had MVPS-dysautonomia, anxiety being my main symptoms. After I was diagnosed with AF, I asked if MVP could have anything to do with it. Some cardiologists said no, while others said yes. Those that believed MVP did have something to do with AF said the "flaps" of the mitral valve were elongated and would contact the side of the atrium while beating possibly interfering with electrical signals which might contribute to AF.

On another note, the last two times I had an echo(the last one at the CCF) MVP wasn't found-everything appeared very normal. Approximately 6 months before I went to CCF I was examined by Dr. Wharton at MUSC. He immediately told me that I had MVP by just listening to my heart-the same thing was also told to me by Dr. Vasey (who specializes in MVP) when jhe "listened" to my heart many years ago. Dr. Vasey also confirmed his findings by an echo. This was at least 15-17 years ago. So.....I don't know what to think. Personally, I believe MVP is "over diagnosed" and most patients take antibiotics needlessly before routine dental treatments. It seems almost that more people have MVP than those that do not.

Jim

I was diagnosed 21 yrs ago (at age 40) with MVP after experiencing PAC's for the first time. I was told it was very minor and to forget about it. I did. Over the years since then, I had less than a handful of days with PACs that I was aware of until my first AF episode two yrs ago. In 2 echocardiograms since then, my MVP was again described as very minor with no regurgitation, and not being responsible for my AF. Besides the PAC's and general anxiety, there have been no other symptoms (I have hypoglycemia if that's a symptom I'm forgetting). Twenty years ago I was told to medicate before dental work, but in the last several years both my doctor and dentist agreed that for such minor MVP, medication is not needed.

Lorraine

One more comment. Folks with pesticide exposures are also deficient in magnesium and treated with IV magnesium. Pesticide exposure also causes endocrine disruption so thyroid issues and MVP may be related to pesticides too.

Lynn

Hello all,

I will tell you what I know about MVP but the impact of my comments make me feel like a broken record. After my pesticide exposure, I too was diagnosed with MVP at Hopkins. Dr. Zeim, a Maryland doctor cited in the Pall article who specializes in the effects of pesticide exposures. She believes that many cases of MVP may be caused by pesticide exposure. In any event, after sufficient hydration and our moving to a hopefully pesticide free environment the prolapse has disappeared. In Maryland, I read a bit about MVP and was struck by the familial link- the probability of siblings having MVP was very high. Yet no one else in my family (not my brother or parents or cousins by the dozens or my aunts) had MVP. This suggested to me that my extraordinary exposure to pesticides might have been the cause.

Lynn

Lynn

Mention pesticides as much as you want. We between us are wanting/needing to get to the bottom of AF - and I believe that it is more than possible that OP's may be the bottom factor at play here. I only know how to go about avoiding AF through avoidance of those chemicals which play on the NMDA receptor. I often feel like a broken record on the hazards of free glutamate, yet if it helps one person then we have done good.

We should not feel bad about shouting about what we believe just because the mainstream does not accept it - yet. I often think in a hundred years people will read their history books and think to themselves how backward we were in exposing ourselves to all these poisons - just as we do today when we read about the Elizabethans putting lead on their faces and for the many centuries that followed where it was used liberally in just about everything. I forget who it was that finally got those with the power to backtrack and admit it was killing people. But we have the same need today - to make people wake up and take notice.

Fran

Lynn, you keep reminding us of pesticides. Fran, you keep drumming on us about MSG/free glutamate. PC and Jackie, you keep telling us the effects of deficiencies in Mg. Me, I'll keep reminding everyone that they are short in the sulfur aminos. That is my platform. But among other issues, are digestive enzymes, hydrating, to keep aldosterone at bay, and eating a good diet.

It takes awareness, and without all of you, I would be completely ignorant!!!!

My heartfelt thanks,

Richard

Dear Fran, Richard and others,

Please note that pesticide exposure does affect glutathione levels too. Hydration is also essential to clear the pesticides from the body. Like Fran, I suspect that OP exposure is at the root of many cases of afib and other arrhythmias. Check out Pall's article for the MSG link and also for the methylation information.

Again, for those of you suffering with MVP- mine appeared after pesticide exposure and then disappeared when I moved to a cleaner environment. Dr. Zeim indicates that this is a usual event following pesticide exposure. One other comment. Why are there so many more cases of afib now than 40 years ago? The same is true for Parkinson's and the increase motivated a search for environmental factors for Parkinson's. I just heard a very interesting lecture by a UCSD medical school faculty member in which he claimed that the incidence of Parkinson's was 4 times higher if one had monthly pesticide treatments in the home than if one did not. Why are some so susceptible and others not? Check out Clem Furlong's work (U of Washington in Seattle) on this. By the by, I did pay the \$500 to the U of Washington and was told that I have the Q-Q polymorphism which makes me very susceptible to OP damage.

Lynn

Lynn,

Do you believe the polymorphism came before or after the pesticide exposure? My guess would be, after. The pesticides are made to kill pests, so why wouldn't one believe that irreversible effects to our own systems would be incurred by higher exposures. It would be along the same line, as various drugs causing irreversible damage, especially when combined with another.

Richard

Richard, I think that polymorphism is a term having to do with one's genetic makeup, not with anything that happens to us after birth.

Peggy M

Peggy,

I guess I'm confused, but it won't be the first time. I thought a genetic defect was presented at birth, but a polymorphism was a change that could occur after birth, such as the word says, morph, or to change. Could anyone else clarify for me, as well? Thanks Peggy.

Richard

Peggy, Richard,

Perhaps this helps? I'll be looking to you guys for translation/clarification...

From The On Line Medical Dictionary at <http://cancerweb.ncl.ac.uk/omd/>

POLYMORPHISM

1. <genetics> The regular and simultaneous occurrence in a single interbreeding population of two or more alleles of a gene, where the frequency of the rarer alleles is greater than can be explained by recurrent mutation alone (typically greater than 1%). The concept includes chromosome polymorphism.

HLA alleles of the major histocompatibility complex are very polymorphic.

2. <zoology> The differentiation of various parts of the units of colonial animals into different types of unit specialised for different purposes, for example as in the colonial hydroid Obelia.

(04 Jul 1999)

Erling

Hi Erling - nice to see your name "in lights" again.

Polymorphisms, as I understand it, are variations in genetic makeup - actual DNA alteration - which make a person particularly susceptible to a specific type of disease most often when exposed to certain often modifiable factors such as environment, diet and lifestyles.

The term SNPSs - pronounced "snips" - Single Nucleotide Polymorphisms were discussed in a Metagenics seminar I attended some time back. While SNPS don't cause disease, they are associated with almost every human disease. These are detectable through the newly developed genomic testing....although controversy is prevalent about how developed and accurate this testing really is. Great Smokies Diagnostic Labs are doing it and probably talk about it on their site. I do know it is very expensive.

A polymorphism could lie at the root of afib.

Jackie

Thank you Erling, Jackie, and Peggy,

So basically, if I'm understanding it correctly, a polymorphism is a genetic predisposition, whereas a genetic defect is something that would show up fairly early in life. But don't you think that an alteration could happen, by way of pesticides, destroying the process of mirroring the DNA, or is it RNA? Geez, another thing to know and understand. I mean, in the case of Lynn, how would they have known if the polymorphism existed before the pesticide exposure?

Anyway, how the heck are you, Erling? Hope all is well with you, and you're enjoying life. I know, that I for one, miss your presence here. You bring good energy.

Richard

Richard et al, if I understand correctly, all defects are polymorphisms, but not all polymorphisms are defects. Does that help?

Peggy M

Another quote:

We know that genes are polymorphic, which means there are differences in the structure and function of all genes among people. We also know that some genes code for more than one type of a specific protein, and one message may be translated under one set of circumstances and another message, under another set of circumstances.

This understanding implies that biochemically or functionally, we are pluripotential.

There are many forms of "you" encoded into your genes. The use who is trading this (book) right now is the result of the experiences of your life thus far bathing over your genes to produce the expression of who you are. Under a different set of experiences in life, a different "you" would be reading this book today. Your genes would be the same, but the way their message was expressed would be different.

Polymorphic: Occurring in several or many forms; appearing in different forms at different stages of development.

Pluripotential: Possessing the power to develop or act in any one of several possible ways.

Summary

1. Your genes don't change. Their expression does.
2. Specific characteristics may differ from individual to individual as a result of genetic polymorphism.
3. Each individual is biochemically unique, but family history is important in identifying general inheritance patterns.
4. Your genotype, when it is influenced by diet, lifestyle and environment, results in your phenotype.
5. Your overall dietary pattern and the vitamins, minerals, phytonutrients and accessory nutrients you consume modify gene expression.
6. Not all genetic characteristics can be changed. Some are constitutional and cannot be modified by diet and lifestyle.
7. Genetic characteristics are not "all or nothing." They can be seen in varying degrees of expression among

individuals.

Quoted from Genetic Nutritioneering
Author Jeffrey S. Bland PhD
Keats Publishing 1999
pp 21,22,67

Jackie

Jackie and Peggy,

I kind of know what your saying, but let's look at it in a simplistic way. The roach is milling around the house with no genetic defect, munching away at the crumbs on your counter. Boom, you spray him with organophosphates, and he dies. Was he predisposed to die from that pesticide? I don't think so. It altered something in his body, severely enough, to kill him.

On the other hand, the nutritioneering concept says we can change certain problems within, through targeted nutrition. This makes more sense to me, if the damage isn't severe enough. I know I can't change the color of my eyes, as that is my blueprint, but I do think things change, due to assaults of all kinds that we put on our bodies. Methylation and methionine are key in helping the body duplicate the DNA/RNA in our bodies, and with a shortage, I would think that something isn't getting duplicated, and if that happens enough, then whatever didn't get duplicated starts to fail. Is that a polymorphism?

I'm not being argumentative; I'm just trying to sort this out in my mind.

I have to re-read you post, Jackie.
Thinking out loud,

Richard

Richard, i think it would help you to get a genetics textbook - or a college biology textbook with a good chapter on genetics - read the genetics chapters, and do the exercises at the ends of the chapters. Better yet, find a programmed text on genetics and go thru it. Come to think of it, the latter is the better idea. You need some basic terms defined so you can make better sense out of the research you are reading. A lot of people are depending on you to make the best sense you can out of this stuff and distill it down for the rest of us. Do the best job you can.

PeggyM

Peggy,

That's probably something I need to do. My strong point was never biology. I guess my only confusion here, is that I believe genes can be altered after birth, due to outside influences, and that is what I thought could have happened to Lynn. It was said she had a polymorphism, but how do they know that wasn't caused by the organophosphates?

Richard

Richard, i do not know much, but i have been an amateur geneticist since i learned to spell the word, and i can tell you this for sure: genes cannot be altered after birth, or after conception for that matter. What can be altered is the expression of those genes. Your genetic makeup says that you are to have 10 fingers, but if you have an accident in shop class in high school, you will be 9 fingered [or worse] thereafter. The blueprint specifications - the genotype - have not changed, but your phenotype has.

Lynn had an accident, not with a buzzsaw, but with some nasty pesticides. She too has been changed for the worse. She wasn't the first and won't be the last, because it is going to be a long time before organophosphates are banned. Even then they will still be with us, just as DDT is.

Don't forget the words of Will Rogers, a very funny man who has been dead a long time: it ain't what you don't know that will hurt you, its what you do know that ain't so.

Are you familiar with programmed texts? If not, any research librarian will explain. They are an easy way to acquire learning. Go find one, and brush up on basic biological terminology. Its useful stuff to know. In the here and now, we can't afford knowing stuff that ain't so. What we don't know can kill us.

PeggyM

Thank you, Peggy for that explanation. I knew you were a smart one. Could you explain this statement just a bit more, "What can be altered is the expression of those genes." Do you mean that the finger won't grow back? Is there anything else you could compare this statement to?

Thank you,
Richard

Richard, man, i don't mean any disrespect to you. You are invaluable to this forum and i expect you will be even more help to us in future. Sometimes i don't understand you at all, though, and this is one of the times. Have you truly never had contact with anyone whose arm, leg, finger, or toe has been amputated? Is there no veteran's hospital near you? With the present state of medical knowledge, amputation is forever. In some rare cases the severed part has been reattached, but no, it won't grow back. If that person could be cloned, the clone would have the original complement of limbs specified by the genetic blueprint, but of course the clone would not be the original person any more than identical twins are the same person. The genotype remains the same, but the phenotype has been changed by a physical event. The damage caused by pesticide exposure is a physical event too, though it occurred inside the body where it is not so readily visible.

Richard, don't just go by what i say. Go to the books and find out for yourself, it will not be any harder than what you are doing now with the research reports.

Peggy

Peggy,

I guess you misunderstood what I was driving at. Sometimes, I try to concentrate around my home, and it's difficult to do, with the commotion that goes on, and here lately it's been more than usual, so I lose my train of thought and don't make myself clear. I do know that limbs don't grow back, after being amputated. Anyway, I'll go study up on this, when I find time, so I get a clearer understanding.

Richard

I think that what Peggy means is that the blue print for what you are when you were born cannot be changed - eg the physical things like blond hair blue eyes (mind you that does not explain why hair goes from blond to brown or eyes from blue to green to hazel), two arms, two legs. However, the way the gene expresses itself can change. eg this statement - "In the most common, non-inherited forms of cancer, the genetic changes are acquired after birth". Things that will change the expression are things such as OP's.

From an online glossary Genetic polymorphism means - A difference in DNA sequence among individuals, groups, or populations, which may be the result of chance processes or may have been induced by external agents.

Here is an analogy that will help us all understand the difference better.

<http://www.sci-ed-ga.org/modules/dna/anals/genopheno.html>

Another analogy that works well to help students understand the relationship of genotype (genetic coding) to phenotype (expression of coding) is to compare a computer to a computer program or a Nintendo machine to a Nintendo game cartridge.

For years my son (neighbor, nephew etc.) had two Nintendo games that Uncle Fred had sent him for his birthday. There was only one "small" problem. He had a couple of cartridges, each with coded information for one game (DNA molecules coding for one gene) but was only able to play (express) them when they were in the environment of a machine (cell) that could use the code. If the game cartridge was placed in an incompatible machine (a Nintendo cartridge in a Sega machine) the game cannot be played.

Phenotype can be equated to the expression of the game that shows up on the screen. (If more than one game is found on one cartridge, each game represents a gene on a chromosome.) The game program represents the genotype. Other cartridges (chromosomes) contain other games (genes) which are played (expressed) when placed in the machine and accessed (turned on by cell).

Fran

Fran,

Thank you for the explanation you gave. That made more sense to me. There's so much to know, and just not enough hours in the day, to try and understand everything.

Richard

But don't you think that an alteration could happen, by way of pesticides,

I would go with that Richard. I think many things are capable of changing gene expression - and hence all (most) of the modern illnesses today.

Fran
