Reading Your Genetic Profile….Without Losing Your Mind!

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The Center for Bio-Individualized Medicine™

Finding Answers Through Genetics and Integrative Medicine

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Introduction

- The purpose of this presentation is to give the reader a general idea of what the polymorphisms (AKA: single nucleotide polymorphisms [snp]) mean and how you may determine if they are expressing in you.
- The presence of snps does NOT mean that there is a problem in the indicated pathway!
- The absence of a snp does not mean that that the pathway is working normally!
- The snps may or may not be expressing in you….this is why it is advisable to consult with a health care provider who understands snps to help put it all together.

Legal Stuff

- This is a informational lecture….the information may or may not pertain to your condition.
- I cannot give specific recommendations for treatment…treatment should be properly done on an INDIVIDUAL basis in consultation with your health care provider.
- There are numerous interpretations of the snps…this informational lecture is from my own personal research, knowledge and experience. There may be other, differing, interpretations.

Permissions

- **The Study we will look at is one of my patients utilizing the raw data from 23andme.com and the app at MTHFRsupport.com.**
- **He has given me his kind permission to share his findings with you. His identifying information has been deleted**
- **Research for the snps was done utilizing www.snpedia.com or http://www.genecards.org unless otherwise indicated**
- **BTW….see what can happen if you treat the snps incorrectly!!!!**
- **LET'S GO!!!!!**

What are these genes and snps we are looking at?

- A gene encodes an enzyme.
- Enzymes run metabolic processes in the body
- A snp *MIGHT* indicate that the enzyme encoded by the gene in question may not be working at its usual efficacy.

A normal gene (-/-) (green) means that the enzyme is working at usual efficiency.

Heterozygous (-/+) snps (yellow) indicates that the enzyme is working at 60% efficiency.

Homozygous (+/+) snps (red) indicates that the enzyme is working at 10-20% efficiency

Think of it this way

- Think of the biochemical pathways as highways that are able to process a certain level of "traffic" to produce their stated result (detoxifying, creating glutathione, metabolizing excitatory neurotransmitters, etc.)
- Normal expression (-/-) (green) is like an 8 lane highway.
- Heterozygous (-/+) (yellow) is like a 4 lane highway
- Homozygous (+/+) (red) is like a 2 lane highway

- All is well if the traffic is light (like when you were a baby)
- Pushing 12 lanes of traffic through an 8 or 4 lane highway will slow down processes
- Pushing 12 lanes of traffic through a 2 lane highway may "crash" the pathway.
- Increased traffic comes from chronic infection, viral loads, food allergies, leaky gut syndrome, immune upregulation, autoimmune diseases, fungal (yeast), parasites, and any combination thereof.

Principles in Interpreting the SNPS

- Interpreting each snp, one by one, is courting confusion at best and mental illness at worst.
- Each snp is but ***PART*** of a biochemical pathway designed to create a certain result.
- It's best to view the snps from the "30,000 foot" point of view.
- Therefore….we will look at the snps in groups

One last thing before we dive in:

Gene & Variation	rsID #	Risk Allele	Your Alleles & Results		Notes
CYP1A1*2C A4889G	rs1048943	C	CT	+/-	
CYP1A1*4 C2453A	rs1799814	T	GG	-/-	

The SNP, if present

CYP1A1 is the Gene

The rs# is how you can research the gene on your own

The "risk allele" determines if your gene is a snp.

Gene & Variation	rsID #	Risk Allele	Your Alleles & Results		Notes
CYP1A1*2C A4889G	rs1048943	C	CT	+/-	
CYP1A1*4 C2453A	rs1799814	T	GG	-/-	
CYP1A2 C164A	rs762551	C	AC	+/-	
CYP1B1 L432V	rs1056836	C	GG	-/-	
CYP1B1 N453S	rs1800440	C	CC	+/+	
CYP1B1 R48G	rs10012	C	GG	-/-	
CYP2A6*2 A1799T	rs1801272	T	AA	-/-	
CYP2C19*17	rs12248560	T	CT	+/-	
CYP2C9*2 C430T	rs1799853	T	CC	-/-	
CYP2C9*3 A1075C	rs1057910	C	AA	-/-	
CYP2D6 S486T	rs1135840	G	GG	+/+	
CYP2D6 T100C	rs1065852	A	GG	-/-	
CYP2D6 T2850C	rs16947	A	AA	+/+	
CYP2E1*1B G9896C	rs2070676	G	CC	-/-	
CYP2E1*4 A4768G	rs6413419	A	GG	-/-	
CYP3A4*1B	rs2740574	C	TT	-/-	

Estrogen Dominance

dextromethorphan, beta-blockers, antiarrhythmics, and antidepressants.

can result in the ultrafast metabolizer phenotype

CYP3A4*3 M445T	rs4986910	G	AA	-/-
GPX3	rs8177412	C	CT	+/-
GSTM1	rs12068997	T	CC	-/-
GSTM1	rs4147565	A	GG	-/-
GSTM1	rs4147567	G	AA	-/-
GSTM1	rs4147568	A	TT	-/-
GSTM1	rs1056806	T	CC	-/-
GSTM1	rs12562055	A	TT	-/-
GSTM1	rs2239892	G	AA	-/-
GSTP I105V	rs1695	G	GG	+/+
GSTP1 A114V	rs1138272	T	CC	-/-
NAT1 A560G(?) (R187Q)	rs4986782	A	GG	-/-
NAT2 A803G (K268R)	rs1208	G	GG	+/+
NAT2 C190T (R64W)	rs1805158	T	CC	-/-
NAT2 G590A (R197Q)	rs1799930	A	GG	-/-
NAT2 G857A (G286E)	rs1799931	A	GG	-/-
NAT2 T341C (I114T)	rs1801280	C	CC	+/+

Glutathione peroxidase

Glutathione pathway

These snps predict the bio-availability of glutathione

Glutathione S-transferase… influences asthma risk

N-acetyltransferase (NAT) may influence the breakdown of aldehydes

http://www.news-medical.net/health/Acetylation-What-is-Acetylation.aspx

SOD2	rs2758331	A	AC	+/-	
SOD2	rs2855262	T	CT	+/-	
SOD2 A16V	rs4880	G	AG	+/-	
PON1 Q192R	rs662	C	TT	-/-	
TONGUE TIE / CLEFT PALATE					
Gene & Variation	**rsID #**	**Risk Allele**	**Your Alleles & Results**		**Notes**
CTH S403I	rs1021737	T	GT	+/-	
IRF6	rs987525	A	CC	-/-	
IRF6	rs861020	A	GG	-/-	
RARA	rs7217852	G	AG	+/-	
RARA	rs9904270	T	CC	-/-	
TBX22	rs41307258	A	T	-	
TBX22	rs28935177	T	A	-	
ALLERGY/MOLD					
Gene & Variation	**rsID #**	**Risk Allele**	**Your Alleles & Results**		**Notes**
HLA	rs7775228	C	TT	-/-	
HLA	rs2155219	T	GT	+/-	
IgE					

Super Oxide Dismutase…influences the conjugation of free radicals and is an important indications for mitochondrial dysfunction

Organophosphates (pesticides)

Dr. Ben has done a great amount of research in this area. He has developed some treatment ideas. Go to : http://www.seekinghealth.com/mthfr-tongue-tie-webinar.html

Mold **may** not your friend

Gene & Variation	rsID #	Risk Allele	Your Alleles & Results		Notes
FCER1A	rs2427837	A	AA	+/+	
IL-13 C1112T	rs1800925	T	CT	+/-	
DARC	rs2814778	C	TT	-/-	
IL13	rs1295685	A	AG	+/-	
CD14	rs2569191	C	CT	+/-	
SOCS-1 -820G>T	rs33977706	A	AC	+/-	
C3	rs366510	G	GG	+/+	
FCER1A / OR10J2P	rs2494262	A	AA	+/+	
FCER1A	rs2251746	C	CC	+/+	
RAD50	rs2040704	G	AG	+/-	
RAD50	rs2240032	T	CT	+/-	
IgG					
Gene & Variation	**rsID #**	**Risk Allele**	**Your Alleles & Results**		**Notes**
FCGR2A	rs1801274	A	AG	+/-	
GSTM3 V224I	rs7483	T	TT	+/+	
TNFRSF13B	rs4792800	G	AA	-/-	
IgA					

The IgE pathway ends up in Histamine release. OMG! This person will produce a ton of histamine! Why is this important?

IgG…..Possibly From food allergies…upregulation of immunoglobulins will cause havoc by creating INFLAMMATION.

Gene & Variation	rsID #	Risk Allele	Your Alleles & Results		Notes
TRAF1	rs3761847	G	AG	+/-	
IRF5	rs4728142	A	AG	+/-	
IGF1R	rs2229765	A	AG	+/-	
IFIH1 (HLA)	rs1990760	C	CC	+/+	
HLA	rs9271366	G	AA	-/-	
CFH	rs6677604	A	AA	+/+	
HLA-DQA2	rs9275224	A	AA	+/+	
MTCO3P1	rs9275596	C	CT	+/-	
PSMB8 / TAP1 / TAP2	rs9357155	A	GG	-/-	
HLA-DPB2 / COL11A2P	rs1883414	A	GG	-/-	

CLOTTING FACTORS

Gene & Variation	rsID #	Risk Allele	Your Alleles & Results		Notes
CETP	rs1800775	C	AC	+/-	
CYP4V2	rs13146272	C	AC	+/-	
GP6	rs1613662	G	AA	-/-	
ITGB3 T196C	rs5918	C	TT	-/-	
KNG I598T	rs2731672	T	CT	+/-	

IgA…..Possibly from food allergies…upregulation of immunoglobulins/antibodies will cause havoc in the body!

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NR1I2	rs1523127	C	AA	-/-	
SERPINC1	rs2227589	T	CC	-/-	
HRG	rs9898	T	CT	+/-	
F12	rs1801020	A	AG	+/-	
F11	rs2289252	T	CC	-/-	
F11	rs2036914	T	CT	+/-	
F10 113777509	rs3211719	G	AA	-/-	
F7 A353G	rs6046	A	GG	-/-	
F2 (Prothrombin 20210A)	I3002432	A	GG	-/-	
F3 94997288	rs1324214	A	GG	-/-	
F5 (Factor V Leiden)	rs6025	T	CC	-/-	
F9 G580A	rs6048	G	A	-	

METHYLATION

Gene & Variation	rsID #	Risk Allele	Your Alleles & Results		Notes
ACE Del16	rs4343	G	AG	+/-	
ADD1 G460W	rs4961	T	GT	+/-	
ACAT1-02	rs3741049	A	GG	-/-	
AGT M235T/C4072T	rs699	G	AA	-/-	

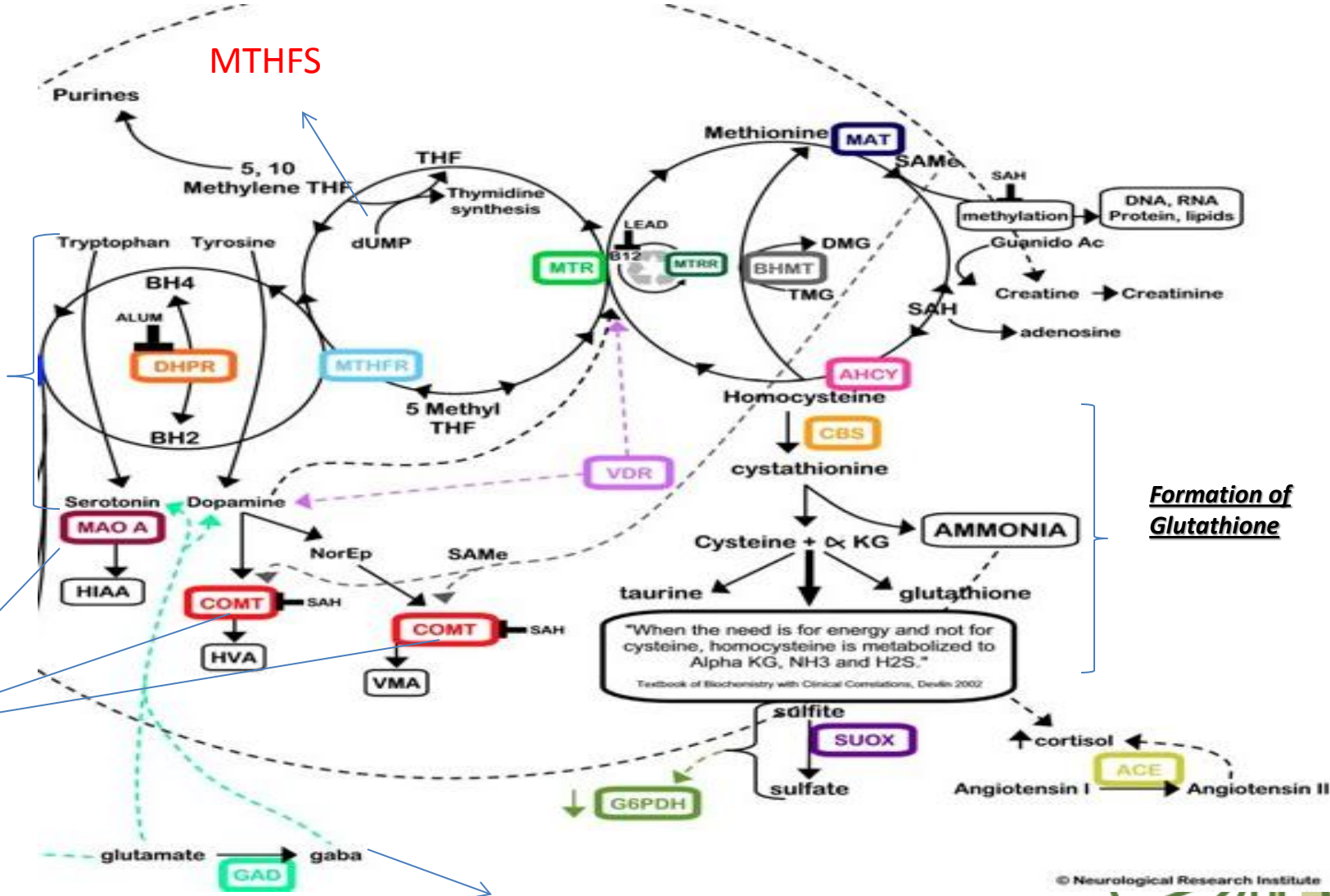
Look for hyper-coagulability clinically. If you clot is less than 1 min…this can mean a problem…why???

Hypertension (HTN)
Alzheimers

Aductin 1, also HTN

acetyl-CoA C-acetyltransferase

HTN, pre-eclampsia



Creation of neurotransmitters

Formation of Glutathione

NT breakdown

Glutamate transformed to GABA

AHCY-01	rs819147	C	TT	-/-
AHCY-02	rs819134	G	AA	-/-
AHCY-19	rs819171	C	TT	-/-
BHMT	rs16876512	T	CC	-/-
BHMT	rs6875201	G	AA	-/-
BHMT-02	rs567754	T	CT	+/-
BHMT-04	rs617219	C	AC	+/-
BHMT-08	rs651852	T	TT	+/+
BHMT R239Q	rs3733890	A	GG	-/-
CBS A13637G	rs2851391	T	CC	-/-
CBS A360A	rs1801181	A	AG	+/-
CBS C19150T	rs4920037	A	GG	-/-
CBS C699T	rs234706	A	AG	+/-
CBS N212N	rs2298758	A	GG	-/-
COMT	rs6269	G	AA	-/-
COMT -61 P199P	rs769224	A	GG	-/-
COMT H62H	rs4633	T	CT	+/-

Catalyzes the hydrolysis of AdoHcy to adenosine and homocysteine. … is believed also to play a critical role in the regulation of biologic methylations. Dr. Amy Yasko

Changes TMG into DMG providing a methyl group. Inner sadness…. BHMT gene is central to the 'short cut' through the methylation cycle, again helping to convert homocysteine to methionine. The activity of this gene product can be affected by stress, by cortisol levels and may play a role in ADD/ADHD by affecting norepinephrine levels. (Dr. Amy Yasko)

Important in the Trans sulfation pathway that produces glutathione. Difficulty with sulfur foods, sensitivity to sulfur meds or vitamins like NAC

Metabolizes catecholamines (dopamine, epi, nor epi). Snps may mean anxiety, anger, etc.

COMT V158M	rs4680	A	AG	+/-
DAO	rs2070586	A	GG	-/-
DAO	rs2111902	G	TT	-/-
DAO	rs3741775	C	AA	-/-
DHFR	rs1643649	C	TT	-/-
FOLR1	rs2071010	A	GG	-/-
FOLR2	rs651933	A	AA	+/+
FOLR3	rs7925545	G	AA	-/-
FOLR3	rs7926875	A	CC	-/-
FUT2	rs492602	G	GG	+/+
FUT2	rs601338	A	AA	+/+
FUT2	rs602662	A	AA	+/+
G6PD	rs1050828	T	C	-
G6PD	rs1050829	C	T	-
GAD1	rs3749034	A	AA	+/+
GAD1	rs2241165	C	CC	+/+
GAD1	rs769407	C	GG	-/-

Diamine Oxidase: Important for the metabolism of Histamine (extracellular histamine)

Folate receptors can be blocked by folic acid. ***Folic Acid*** binds preferentially to the FOLR receptors leaving the active reduced folates in the blood---NOT ABLE TO GET INTO THE CELLS!---really BAD!

Fucosyltransferase 2 your ability to "hold onto" B12

Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency is a hereditary condition in which red blood cells break down when the body is exposed to certain drugs or the stress of infection.

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GAD1	rs2058725	C	CT	+/-
GAD1	rs3791851	C	TT	-/-
GAD1	rs3791850	A	AG	+/-
GAD1	rs12185692	A	CC	-/-
GAD1	rs3791878	T	GG	-/-
GAD1	rs10432420	A	AA	+/+
GAD1	rs3828275	T	CC	-/-
GAD1	rs701492	T	CC	-/-
GAD1	rs769395	G	AA	-/-
GAD2	rs1805398	T	GG	-/-
GAMT	rs17851582	A	AG	+/-
GAMT	rs55776826	T	CT	+/-
GIF (TCN3)	rs558660	A	GG	-/-
MAO A R297R	rs6323	T	G	-
MAT1A	rs72558181	T	CC	-/-
MTHFD1 C105T	rs1076991	C	TT	-/-
MTHFD1 G1958A	rs2236225	A	AG	+/-

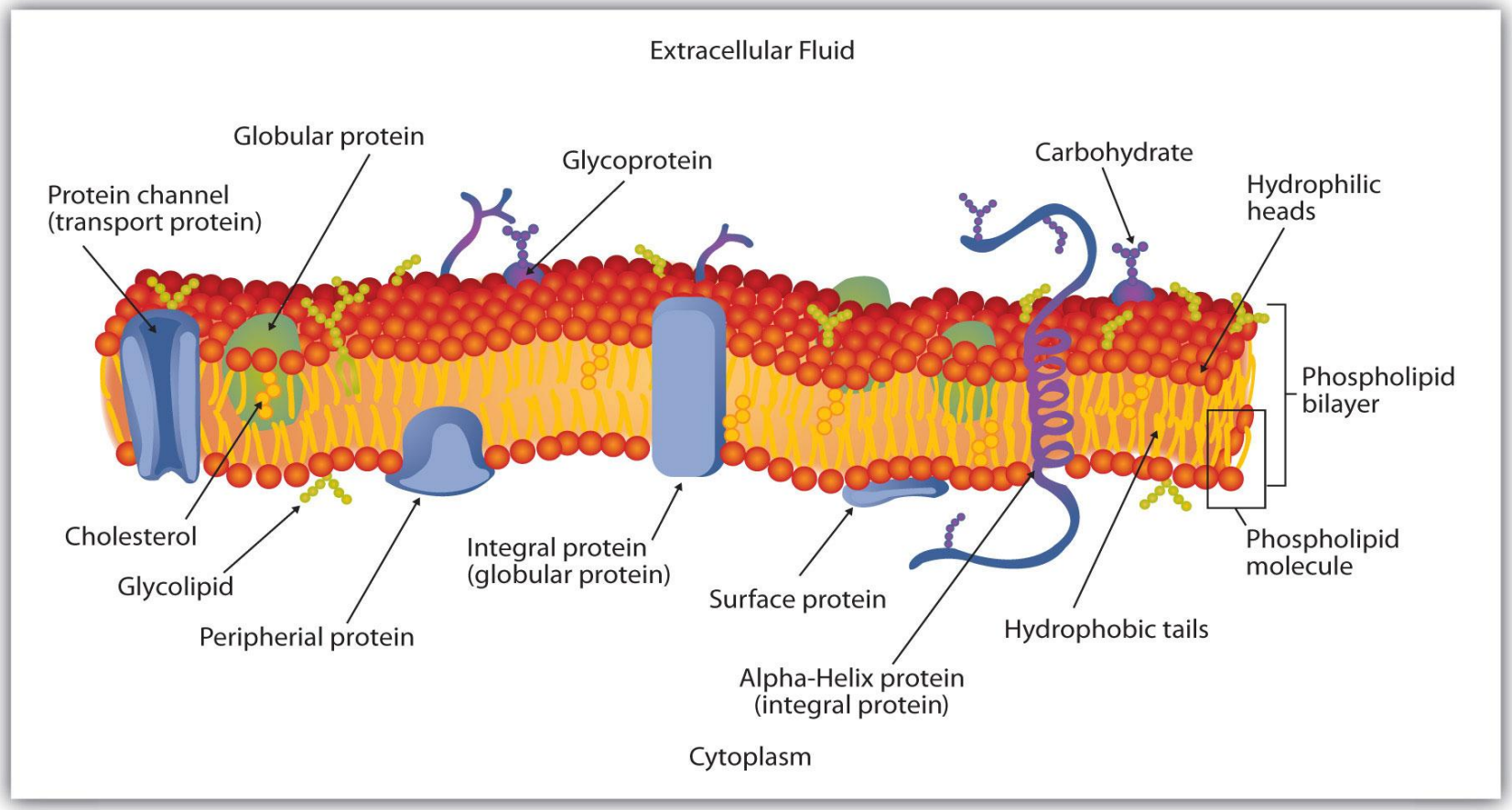
Glutamic Acid Decarboxylase genes and anxiety disorders, major depression, and neuroticism. I call them the "General Anxiety Disorder" genes

Guanidinoacetate N-Methyltransferase.. Defects in this gene have been implicated in neurologic syndromes and muscular hypotonia, probably due to **creatine deficiency** and accumulation of guanidinoacetate in the brain of affected individuals.

The "Warrior Gene", like COMT

Methionine Adenosyltransferase I… go to this link to learn more: http://www.genecards.org/cgi-bin/carddisp.pl?gene=MAT1A

The Cell Wall, A Phospholipid Bi-Layer



MTHFD1L	rs11754661	A	GG	-/-
MTHFD1L	rs17349743	C	TT	-/-
MTHFD1L	rs6922269	A	AG	+/-
MTHFD1L	rs803422	A	AG	+/-
MTHFR 03 P39P	rs2066470	A	GG	-/-
MTHFR A1298C	rs1801131	G	TT	-/-
MTHFR A1572G	rs17367504	G	AA	-/-
MTHFR C677T	rs1801133	A	GG	-/-
MTHFR G1793A (R594Q)	rs2274976	T	CC	-/-
MTHFR	rs12121543	A	CC	-/-
MTHFR	rs13306560	T	CC	-/-
MTHFR	rs13306561	G	AA	-/-
MTHFR	rs1476413	T	CT	+/-
MTHFR	rs17037390	A	GG	-/-
MTHFR	rs17037396	T	CC	-/-
MTHFR	rs3737964	T	CT	+/-
MTHFR	rs4846048	G	AG	+/-

Difficulty in storing phospholipid choline necessary for cell wall repair

MTHFR – reduced capacity to produce methylfolate. End consequences may lead to reduced levels of BH4 and SAMe. Downstream effects from reduced BH4 and SAMe levels are numerous as MTHFR is the regulator of methylation and biopterin (neurotransmitter) formation. The variant of MTHFR doesn't matter but some variants reduce enzymatic kinetics more potently than others – such as C677T vs A1298C. Combinations of MTHFR snps may indicate more restriction in the pathways.

MTHFR	rs4846049	T	GT	+/-
MTHFS	rs6495446	C	CC	+/+
MTR A2756G	rs1805087	G	AA	-/-
MTRR A66G	rs1801394	G	AG	+/-
MTRR H595Y	rs10380	T	CC	-/-
MTRR K350A	rs162036	G	AA	-/-
MTRR R415T	rs2287780	T	CC	-/-
MTRR-11 A664A	rs1802059	A	AG	+/-
MTRR	rs10520873	C	TT	-/-
MTRR	rs1532268	T	CT	+/-
MTRR	rs162049	G	AA	-/-
MTRR	rs3776467	G	AA	-/-
MTRR	rs9332	A	GG	-/-
NOS1	rs3782206	T	CT	+/-
NOS2	rs2297518	A	AG	+/-
NOS2	rs2274894	T	GT	+/-
NOS2	rs2248814	A	AG	+/-

Conversion to Methionine

Conversation to Methyl B12

The ability to break down Nitrous Oxide (free radicals). Possible association with mitochondrial dysfunction

NOS3	rs1800783	A	AA	+/+
NOS3	rs1800779	G	AG	+/-
NOS3	rs3918188	A	CC	-/-
NOS3 G10T	rs7830	T	GT	+/-
NOS3 T786C	rs2070744	C	CT	+/-
PEMT	rs4244593	T	GT	+/-
PEMT	rs4646406	A	TT	-/-
PEMT	rs7946	C	CC	+/+
SHMT1 C1420T	rs1979277	A	GG	-/-
SHMT1	rs9909104	C	CT	+/-
SHMT2	rs12319666	T	GG	-/-
SHMT2	rs34095989	A	AG	+/-
SLC19A1	rs1888530	T	CT	+/-
SLC19A1	rs3788200	A	AG	+/-
TCN1	rs526934	G	AG	+/-
TCN2 C766G	rs1801198	G	CG	+/-
TYMS	rs502396	C	TT	-/-

"…that gene variation of NOS1 and NOS2 was associated with longevity. In addition NOS1 rs1879417 was also found to be associated with a lower cognitive performance, while NOS2 rs2297518 polymorphism showed to be associated with physical performance. Moreover, SNPs in the NOS1 and NOS3 genes were respectively associated with the presence of depression symptoms and disability, two of the main factors affecting quality of life in older individuals" Biogerontology. 2013 Apr;14(2):177-86. doi: 10.1007/s10522-013-9421-z. Epub 2013 Apr 10.

This gene encodes an enzyme which converts phosphatidylethanolamine to phosphatidylcholine by sequential methylation in the liver.

Association of folate receptor (folr1, folr2, folr3) and reduced folate carrier

May reduce the transport of cobalamin reducing B12 levels

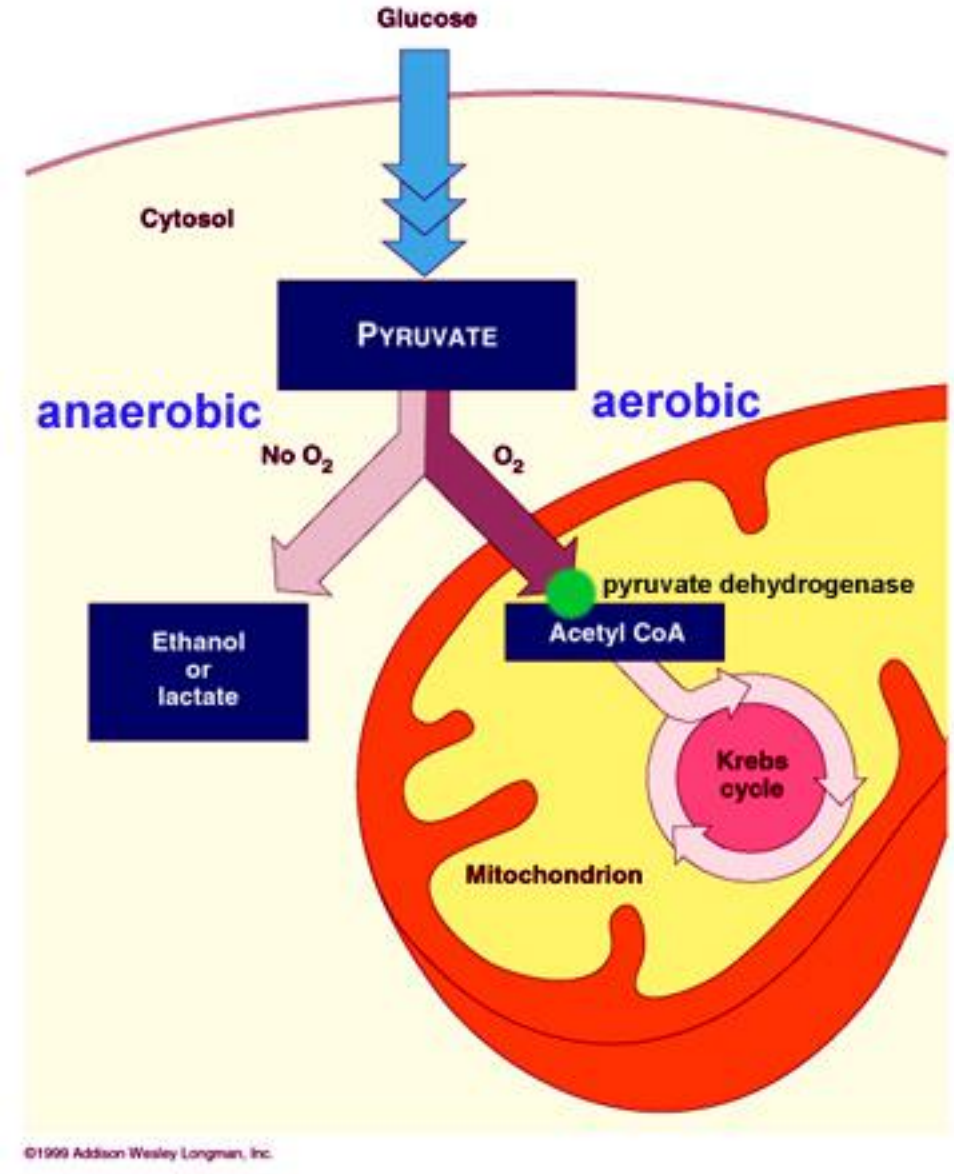
VDR Bsm	rs1544410	T	CT	+/-	
CELIAC DISEASE/GLUTEN INTOLERANCE					
Gene & Variation	**rsID #**	**Risk Allele**	**Your Alleles & Results**		**Notes**
HLA	rs2858331	G	AA	-/-	
HLA DQA1	rs2187668	T	CT	+/-	
THYROID					
Gene & Variation	**rsID #**	**Risk Allele**	**Your Alleles & Results**		**Notes**
CTLA4	rs231775	G	AG	+/-	
FOXE1	rs1867277	A	GG	-/-	
FOXE1	rs7043516	C	AA	-/-	
FOXE1	rs10984009	A	GG	-/-	
EYE HEALTH					
Gene & Variation	**rsID #**	**Risk Allele**	**Your Alleles & Results**		**Notes**
BCMO1	rs4889294	C	CT	+/-	
BCMO1 R267S	rs12934922	T	AT	+/-	
BCMO1 A379V	rs7501331	T	CC	-/-	
MITOCHONDRIAL FUNCTION					
Gene & Variation	**rsID #**	**Risk Allele**	**Your Alleles & Results**		**Notes**
ATP5g3	rs185584	G	AA	-/-	

VDR (vitamin D (1,25- dihydroxyvitamin D3) receptor) is a protein-coding gene. Diseases associated with VDR include *osteoporosis*, and *vitamin d-dependent rickets type ii*,

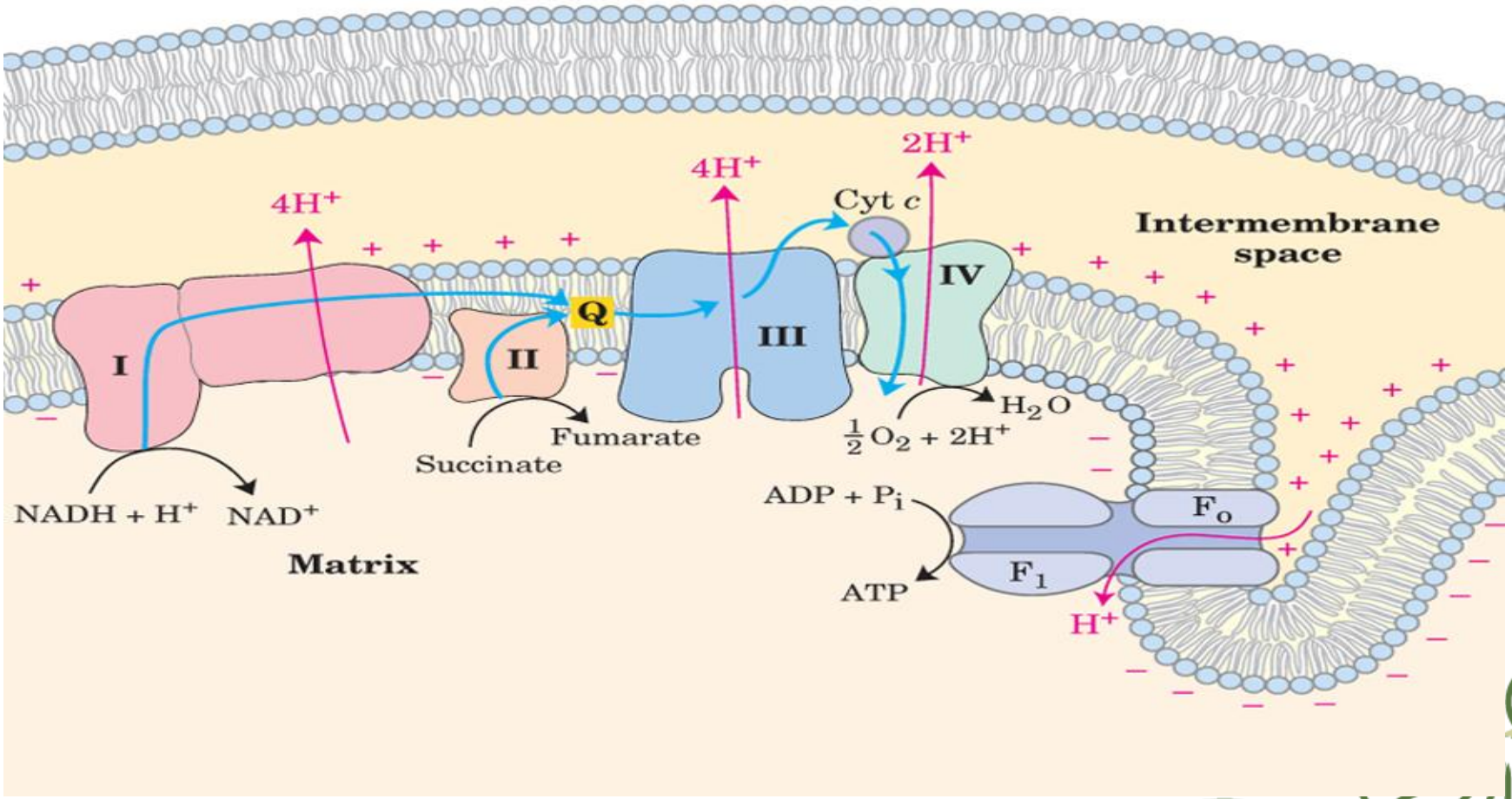
Possible Gluten Intolerence

Possible thyroid issues

Difficulty in creating Vitamin A from Beta Carotene



Electron Transport Chain



ATP5g3	rs36089250	C	TT	-/-
ATP5c1	rs2778475	A	GG	-/-
ATP5c1	rs1244414	T	CC	-/-
ATP5c1	rs1244422	T	CC	-/-
ATP5c1	rs12770829	T	CT	+/-
ATP5c1	rs4655	C	TT	-/-
COX5A	rs8042694	G	AG	+/-
COX6C	rs4626565	C	TT	-/-
COX6C	rs7844439	A	CC	-/-
COX6C	rs4510829	A	GG	-/-
COX6C	rs1135382	A	GG	-/-
COX6C	rs7828241	C	AA	-/-
COX6C	rs12544943	G	AA	-/-
COX6C	rs4518636	C	TT	-/-
NDUFS3	rs2233354	C	TT	-/-
NDUFS3	rs4147730	A	AG	+/-
NDUFS3	rs4147731	A	GG	-/-

Complex V- ATP Synthase

Complex III

Complex I

NDUFS7	rs2332496	A	AG	+/-
NDUFS7	rs7254913	G	AA	-/-
NDUFS7	rs1142530	T	TT	+/+
NDUFS7	rs7258846	T	TT	+/+
NDUFS7	rs11666067	A	AA	+/+
NDUFS7	rs2074895	A	AA	+/+
NDUFS7	rs809359	G	AA	-/-
NDUFS8	rs4147776	C	AA	-/-
NDUFS8	rs1122731	A	GG	-/-
NDUFS8	rs999571	A	GG	-/-
NDUFS8	rs2075626	C	TT	-/-
NDUFS8	rs3115546	G	TT	-/-
NDUFS8	rs1104739	C	AC	+/-
NDUFS8	rs1051806	T	CC	-/-
UQCRC2	rs6497563	C	TT	-/-
UQCRC2	rs4850	A	GG	-/-
UQCRC2	rs11648723	T	GG	-/-

Complex I

Oxidized glutathione can get stuck here blocking the electron transport chain

Complex IV

UQCRC2	rs12922362	A	CC	-/-	
UQCRC2	rs2965803	T	CC	-/-	
OTHER IMMUNE FACTORS					
Gene & Variation	**rsID #**	**Risk Allele**	**Your Alleles & Results**		**Notes**
4q27 Region	rs6822844	T	GG	-/-	
APOE	rs429358	C	TT	-/-	
ATG16L1	rs10210302	C	CT	+/-	
GSDMB	rs7216389	T	CT	+/-	
HLA-DRB1	rs660895	G	AA	-/-	
IL5	rs2069812	A	GG	-/-	
IL-13	rs20541	A	AG	+/-	
IL4R Q576R	rs1801275	G	AA	-/-	
MeFV A744S	I4000409	A	CC	-/-	
MeFV E148Q	rs3743930	G	CC	-/-	
MeFV F479L	I4000403	C	GG	-/-	
MeFV K695R	I4000407	C	TT	-/-	
MeFV M680I	rs28940580	G	CC	-/-	
MeFV M694I	rs28940578	T	CC	-/-	

Complex IV

The symptoms of familial Mediterranean fever are caused by the person's own inflammatory response; it is not an infectious disease. The condition is more common among Turks, Sephardic Jews, and people of Arab and Armenian ancestry.

MeFV M694V	I4000406	C	TT	-/-
MeFV P369S	rs11466023	A	GG	-/-
MeFV R761H	I4000410	T	CC	-/-
STAT4	rs10181656	G	CG	+/-
TNF -308	rs1800629	A	AG	+/-
TNF -238	rs361525	A	GG	-/-
TYR (MeFV) V726A	rs28940879	A	GG	-/-

SULFONOTRANSFERASE

Gene & Variation	rsID #	Risk Allele	Your Alleles & Results		Notes
SULT1A1	rs35728960	G	TT	-/-	
SULT1A1	rs1801030	C	TT	-/-	
SULT1A1	rs1042157	A	GG	-/-	
SULT1A1	rs36043491	T	CC	-/-	
SULT1A1	rs60749306	C	TT	-/-	
SULT1A1	rs9282862	C	TT	-/-	
SULT1A1	rs1042008	A	GG	-/-	
SULT1A1	rs2925627	C	TT	-/-	
SULT1A1	rs2925631	C	TT	-/-	

Cytosolic sulfotransferases are enzymes that catalyze the conjugation of sulfate groups to a variety of xenobiotic and endogenous substrates. A mutation in the SULT1A1 gene has been associated with decreased sulfotransferase activity. We studied 125 cancer patients and 100 healthy controls from Brazil matched by age and gender. The objective of this study was to assess the impact of the SULT1A1 polymorphism on sulfotransferase activity in a population of cancer patients. Both heterozygous and homozygous individuals for the mutant allele had significantly decreased sulfotransferase enzymatic activity. This decrease was more significant in cancer patients. The frequency of the SULT1A1(*)2 allele was increased in the myeloma group (odds ratio=0.53). These data suggest a functional role for the SULT1A1 gene polymorphism in cancer.(Cancer Genet Cytogenet. 2005 Jul 1;160(1):55-60.)

SULT1A1	rs3020800	G	AA	-/-
SULT1A1	rs4149385	T	CC	-/-
SULT1A1	rs60701883	A	CC	-/-
SULT1A1	rs4149381	G	TT	-/-
SULT1A1	rs8057055	A	CC	-/-
SULT1A1	rs6498090	A	GG	-/-
SULT1A1	rs7193599	C	AA	-/-
SULT1A1	rs7192559	T	CC	-/-
SULT1A3	rs1059667	A	TT	-/-
SULT2A1	rs296366	T	CT	+/-
SULT2A1	rs296365	C	CG	+/-
SULT2A1	rs11569679	T	CC	-/-
SULT2A1	rs4149452	T	CT	+/-
SULT2A1	rs8113396	G	AA	-/-
SULT2A1	rs2547242	C	TT	-/-
SULT2A1	rs2910393	T	CT	+/-
SULT2A1	rs4149449	T	CC	-/-

Sulfotransferase enzymes catalyze the sulfate conjugation of many hormones, neurotransmitters, drugs, and xenobiotic compounds.

Sulfotransferases (SULT2A1) aid in the metabolism of drugs and endogenous compounds by converting these substances into more hydrophilic water-soluble sulfate conjugates that can be easily excreted. This protein catalyzes the sulfation of steroids and bile acids in the liver and adrenal glands, and may have a role in the inherited adrenal androgen excess in women with polycystic ovary syndrome.

Tying It Together

By now, I hope I have conveyed the following:

1. The presence or absence of snps, in and of themselves, do not indicate the presence or absence of disease. SNPs are probabilities and need to be correlated with your entire clinical condition.
2. Treating only the snps with various available products designed for same without correlation with your clinical condition is inadvisable at best.
3. This correlation should be done by a trained and experienced health care provider. I have always gone by the saying, "A doctor who treats himself has a fool for a patient". The reverse is also true, IMHO.
4. Looking at the snps individually is usually bad. Looking at them as parts of pathways and considering the entire pathway is best!
5. Discovering what is "stressing" or "crashing" the pathways (remember the highway example) is the true way of healing an individual and, often, when you do this….the pathways fix themselves.
6. ***In choosing a health care practitioner, it is critically important to pick someone who:***

Thinks Like a Detective!

- A true “holistic” practitioner offers you the best of traditional and alternative medicine (integrative medicine)
- This practitioner is not beholden to a single protocol or single way of thinking
- Your detective will build a treatment plan based on your individual genetics and physiology and….most of all…will listen to you. It has been said by very old doctors, “if you listen, your patient will tell you what is wrong”.
- Hence, we have created BIO-INDIVIDUALIZED MEDICINE.

Bio-Individualized Medicine

- Bio-Individualized Medicine takes genetics and integrative medicine to a new level. By combining the knowledge of Neuro-Endo-Immunology, epigenetics/nutrigenomics, acquired (secondary) mitochondrial dysfunction and cell wall integrity, the practitioner trained and experienced in this arena has the capability of identifying and treating not only the root cause(s) of dysfunction but also attending to the "downstream" effect. That is, fixing whatever the primary causative agent did to the body as well as fixing the root cause(s). This MUST be done on an ***INDIVIDUAL*** basis (hence why I don't write a book). Each person is different with varied requirements. Practitioners of this paradigm are findings answers that have eluded others and are developing treatments that show promise in eradicating chronic illness and returning patients to normal function.
- Practitioners trained and experienced in this arena include myself, Shawn Bean, Cynthia Smith and, of course, Dr. Ben Lynch.

Alyssa's Story

Consults

Skype or phone consults are available. Also available is education and mentorship for health care practitioners in functional genetics and bio-individualized medicine.

Requests for consults for **Dr. Armine or Shawn Bean** can be obtained inside and outside the USA by:

E-mail: info@bio-individualmed.com

Phone: 610 449 9716

Fill in contact form at www.bio-individualmed.com

For **Cynthia Smith**, e-mail info@lifezonewellness.com or by calling ***(312) 451-6504***

Some practitioners may offer a complimentary 15 min "get acquainted" session to ascertain if they can help your condition. Contact the practitioner individually for information on this.

Q&A Time!

- Call in with your **(646) 595-2277**
- **Happy to answer any and all questions**
- **You can also e-mail questions to jess@drjessarmine.com, info@bio-individualmed.com or info@lifezonewellness.com**

SPECIAL TREAT!!!!

- Next Week, July 14th, 2014 at 8pm Eastern. We will have an EXPERT PANEL ON MTHFR, METHYLATION, SNPS AND BIO-INDIVIDUALIZED MEDICINE!!!!
- This will be OPEN MIC! You will be able to call in and ask your toughest questions!
- The panel will consist of, at least, Cynthia Smith, Shawn Bean and Yours Truly (Lil Ole Me ☺)

