geneticgenie Methylation and detox analysis from 23andMe results

Methylation Analysis Results

If you find any bugs, typos, or errors in this analysis please contact us via feedback.

There is some confusion over VDR Taq. Genetic Genie has set this result to match that of Dr. Amy Yasko's Nutrigenomic Testing. This was done on purpose so that Amy Yasko's book and other resources will match up with the results. Read more about this: Clearing up the VDR Taq Confusion

If you find this info helpful, please donate. Donations are needed to pay for the domain and server. I would like to say that anything extra will go to starving children in Africa, but in reality anything extra will probably be used for healthcare costs and overpriced vitamins.

Your Donation Amount:
10.00

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Gene & Variation	rsID	Alleles	Result
COMT V158M	rs4680	AG	+/-
COMT H62H	rs4633	CT	+/-
COMT P199P	rs769224	GG	-/-
VDR Bsm	rs1544410	CC	-/-
VDR Taq	rs731236	AA	+/+
VDR Fok-I	not found	n/a	n/a
MAO A R297R	rs6323	G	-
ACAT1-02	rs3741049	GG	-/-
MTHFR C677T	rs1801133	AG	+/-
MTHFR 03 P39P	rs2066470	AG	+/-
MTHFR A1298C	rs1801131	GT	+/-
MTR A2756G	rs1805087	AG	+/-
MTRR A66G	rs1801394	AA	-/-
MTRR H595Y	rs10380	CC	-/-
MTRR K350A	rs162036	AA	-/-
MTRR R415T	rs2287780	CC	-/-
MTRR S257T	not found	n/a	n/a
MTRR A664A	rs1802059	GG	-/-
BHMT-01	not found	n/a	n/a
BHMT-02	rs567754	CC	-/-
BHMT-04	rs617219	AA	-/-
BHMT-08	rs651852	CT	+/-
AHCY-01	rs819147	TT	-/-
AHCY-02	rs819134	AA	-/-
AHCY-19	rs819171	TT	-/-
CBS C699T	rs234706	GG	-/-

CBS A360A	rs1801181	GG	-/-
CBS N212N	rs2298758	GG	-/-
SUOX S370S	not found	n/a	n/a
NOS3 D298E	not found	n/a	n/a
SHMT1 C1420T	rs1979277	AG	+/-

Before getting started: Understanding the basics

We have two copies of most of the genes we are born with - one from our mother and one from our father. Genetic Genie uses the SNPs (Single Nucleotide Polymorphisms) generated from your unique DNA sequence to determine if one or both copies of your genes have a mutation at a specific location in a specific gene. If there are no mutations present, your result will be displayed as (-/-). If one gene is mutated, the result will read (+/-). If both copies have a mutation, the result is (+/+). Along with the (+/-) symbols, the colors on the table also denote the type of mutation for visual comprehension. The color red indicates a homozygous (+/+) mutation, the color yellow indicates a (+/-) heterozygous mutation and the color green (-/-) indicates that you don't carry the specific mutation.

The terms heterozygous and homozygous are used by geneticists to denote whether one or both copies of a gene are mutated. Heterozygous mutations (+/-) may differ from homozygous mutations (+/+) in associated disease risk since a person with a heterozygous mutation will often still have one fully functioning copy of the gene. It is also important to understand that having a gene with a SNP mutation does not mean that the gene is defective or nonfunctioning, only that it is working with an altered efficiency. Sometimes this means that it is working at a decreased level, but it could also mean that it is functioning at a higher than normal efficiency, or that the gene is lacking regulatory mechanisms normally involved in its expression.

Although mutations can occur at any time during our lifetime, it is most likely that we are born with these mutations and will have them throughout our life. These inherited mutations have been passed down to us from previous generations (our parents and grandparents) and may be passed to future generations (our children). This may provide an explanation as to why certain traits or diseases "run in the family".

Although we cannot change our genetic code, we can change how our genes are expressed. Research has revealed that our gene expression is not determined solely by hereditary factors, but it is also influenced by our diet, nutritional status, toxic load and environmental influences or stressors. This phenomenon has been termed "epigenetics". Researchers in the growing field of epigenetics have demonstrated that certain genes can be over- or under-expressed with certain disease processes. Researchers in this field hope that by understanding of how these genes are regulated and what is influencing them, we may be able to change their expression. Using epigenetic concepts along with a good understanding of the methylation cycle, researchers have begun to make recommendations to optimize genetic expression and help to restore health.

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MTHFR Mutations

First we'll look at a few of your MTHFR mutations. According to research, these mutations are important and can be implicated in various disease states.

You have 3 heterozygous (yellow) mutation(s). These are generally not as bad as red homozygous mutation, but they may still worth paying attention to. They include:

- MTHFR C677T
- MTHFR 03 P39P
- MTHFR A1298C

Now let's move on to discuss what these MTHFR mutation(s) mean.

MTHFR C677T

One function of MTHFR (Methylenetetrahydrofolate reductase) is to help convert homocysteine to methionine. A MTHFR C677T mutation means that the MTHFR enzyme may have trouble performing its task leading to high levels of homocysteine. According to Dr. Ben Lynch, impaired function of the enzyme can cause or contribute to conditions such as Autism, Chronic Fatigue Syndrome, Fibromyalgia, Miscarriages, IBS, many birth defects, Multiple Sclerosis, Alzheimer's, Bipolar Disorder, blood clots, Stroke, Chemical Sensitivity, and many other conditions.

MTHFR C677T can also lead to high homocysteine. You can ask your doctor to test for homocysteine levels. If you have high levels of homocysteine, it may be related to your MTHFR C677T mutation. But even if one has a (+/+) or (+/-) mutation, it does not necessarily mean that they will have high homocysteine levels.

As S-adenosylhomocysteine (SAH) accumulates, the COMT enzyme may become impaired. Inhibiting COMT can increase dopamine levels for those with COMT V158M (-/-), but for those with COMT V158M (+/+), the high level of SAH can lead to behavior problems and mood swings according to Dr. Amy Yasko.

Nutritional Support of MTHFR C677T

Supplementing with Folate (preferably as L-Methylfolate) can help alleviate the effects of MTHFR C677T as well as lower one's homocysteine levels. There are a lot of different types of folate on the market, and I recommend reading this article by Dr. Ben Lynch about folate. It might be a good idea to avoid synthetic folic acid and folic acid fortified foods such as cereals. Also, lowering other doses of forms of folate or folinic acid may be important as it can compete with L-methylfolate.

To avoid adverse effects, one can start with very low doses of folate and work to higher doses. Side effects can occur as a detoxification effect as this pathway becomes unblocked. In the case of extreme adverse effects, time-released niacin and/or potassium may be able to stop the side effects.

MTHFR 03 P39P

There is currently not enough research or data to draw conclusions from this SNP.

MTHFR A1298C

MTHFR A1298C is involved in converting 5-methylfolate (5MTHF) to tetrahydrofolate (THF). Unlike MTHFR C677T, the A1298C mutation does not lead to elevated homocysteine levels. This reaction helps generate BH4. BH4 is important in the detoxification of ammonia. The gene is compromised about 70% in MTHFR A1298C (+/+) individuals, and about 30% in people with a heterozygous (+/-) mutations.

BH4 acts as a rate limiting factor for the production of neurotransmitters and catecholamines including serotonin, melatonin, dopamine, norepinephrine, and epinephrine. A MTHFR A1298C + status may cause a decrease in any of these neurotransmitters or catecholamines. It's also a cofactor in the production of nitric oxide. If your BH4 cycle is not working effectively, you may experience mental/emotional and/or physical symptoms. Mercury, lead, and aluminum may act as a drain on BH4.

Adressing MTHFR A1298C

L-methylfolate supplementation may be implicated. One should start with low doses of L-methylfolate, and in the case of adverse reaction time-released niacin and/or potassium may help.

Metal detoxification (especially aluminum) can help address dysfunctions associated with MTHFR A1298C and BH4 deficiency, and can help many other biochemical abnormalities as well. Aluminum toxicity can hinder one's ability to fight infection, so addressing the gut and treating chronic bacterial infection may be important. Since the A1298C mutation can lead to excess ammonia, one can address these elevated levels with things like charcoal/magnesium flushes, Yucca Root, and L-Ornithine. Keeping ammonia low helps preserve BH4 levels.

Low doses of BH4 may be helpful after one's methylation cycle is fully supported.