Nutrition Genotype Report

Client Name: Sample Client

HUNGER & SATIETY

GENETIC DATA

GENE	GENO TYPE
ADIPOQ (1)	GG
ADIPOQ (2)	GG
FTO	TT
MC4R	TT
ANKK1	GG
COMT	GG
DRD2	AG
NMB	GG
FTO	TT
HTR2C	CC
GSHR(1)	TC
GSHR(2)	GC
POMC	..
LEP	n/a
LEPR(2)	AG
LEPR(1)	AG
MTHFR 1298	GG

Appetite is a combination of hunger response and satiety. Many scientific studies have been undertaken to identify genomic variations that contribute to these aspects of eating. What we have discovered is that it is a complex interaction between many systems:

- Brain neurotransmitters (dopamine & serotonin)
- Intestinal peptides
- Signals from fat cells
- Appetite hormones

All components of this network require coordination in central sensing mechanisms of the brain in order to create your response.

INTERPRETATION

LOW	TYPICAL
HUNGER	SATIETY

RECOMMENDATIONS

INSULIN RESISTANCE

GENETIC DATA

GENE	GENO TYPE
FABP2	GG
GCKR	GG
LIPC	TC
PPARD	AA
IRS1	
VDR FOK1	AA
CRY2	AC
FADS1	TC
PROX1	TC
ADCY5	AG
MTNR1B	CG
SLC30A8	TC
TCF7L2	TC
G6PC2	TC
MADD	AA
ADRA2A	GG
GLIS3	AA

HIGH

RISK

Insulin resistance is a state where the body requires greater and greater amounts of insulin in order to drive down blood sugar levels. It is usually associated with diabetes or the pre-diabetic state.

Studies have demonstrated that some individuals actually possess a greater predisposition towards insulin resistance and this predisposition can be predicted based on genetic variations. Individuals that carry greater risk tend to have higher, though frequently normal, fasting blood sugar levels and insulin levels. These higher fasting blood glucose levels also promote accelerated rates of aging in the body.

Individuals with a greater propensity towards insulin resistance often report greater difficulty losing weight than others that follow similar diets despite aggressive adherence to the diet.

Insulin Resistance Score (IRS) 63%

INTERPRETATION:

RECOMMENDATIONS

DAIRY

GENETIC DATA

GENE	GENO TYPE
MCM6(1)	AA
MCM6(2)	n/a
APOA2	AG

TYPICAL

SENSITIVITY

TYPICAL

DAIRY FAT

SENSITIVITY

Dairy genes relate to the processing of the sugar and the fat in dairy products.

Lactose is a sugar found in milk. Some individuals have deficiencies in the enzyme or lack the enzyme lactase that is required to fully digest the sugar. The actual gene involved is LCT but the MCM6 is a regulator of the LCT expression. Variants of the MCM6 gene only indicate a PROPENSITY toward lactose intolerance.

Certain genotypes will also express a greater propensity toward weight gain and obesity when consuming high fat dairy.

INTERPRETATION:

RECOMMENDATIONS

GLUTEN

GENETIC DATA

GENE	GENO TYPE
HLA-DQA1	TC
HLA-DQA2	AA
HLA-DRA	TG
HLA-DQB1	TT

LOW

RISK

Gluten is a mixture of proteins found in wheat and related grains. It is also found in many food preparations because it provides elasticity and chewiness to many prepared foods. There is a difference between allergy and sensitivity, these genes relate to potential for developing allergy. See the section relating to grain sensitivity for more information. These genetics related to gluten are based on predisposition and are only suggestive of susceptibility to developing reactions to gluten in foods. This is not a diagnostic test.

INTERPRETATION:

RECOMMENDATIONS

GRAIN SENSITIVITY

GENETIC DATA

GENE	GENO TYPE
GAD1(1)	AG
GAD1(2)	TC
GAD1(3)	GG
GAD1(4)	GG
GAD1(5)	AC

MODERATE

RISK

HIGH GLUTAMIC ACID SOURCES

-Wheat and Grains
-Soy
-Dairy
-Eggs
-Chicken & Turkey
-Seeds
-MSG

Glutamic acid decarboxylase is an enzyme responsible for the conversion of glutamate into GABA. GAD1 is only present in the brain and helps us to convert the excitatory neurotransmitter, glutamate, into the inhibitory neurotransmitter GABA.

The GAD1 genes relate to the handling of glutamic acid containing foods and the potential for creation of an imbalance between excitatory and inhibitory neurotransmitters in the brain. Certain variations can lead to decreased activity of this enzyme and a tendency toward higher glutamate and lower GABA levels in the brain. This can lead to anxiety, agitation, and difficulty sleeping.

Many grains are high in glutamic acid and frequently people will misinterpret a negative response to grains as a negative response to gluten.

When these variations are significant and the symptoms are expressed, it is important to reduce exposure to glutamic acid and make sure that B6 levels remain healthy since it is required for the enzyme to work optimally

INTERPRETATION:

RECOMMENDATIONS

SWEETS & SNACKING

GENETIC DATA

GENE	GENO TYPE
FTO(1)	TT
LEPR	AG
MC4R	TT
FTO(3)	TC
FGF21	AG
ANKK1	GG
COMT	GG
DRD2	AG
SLCA2	GG
SLC2A2	n/a
TAS1R2	GG
TAS1R3	..
TAS2R38	GG
MTHFR 1298	GG

INCREASED

SWEET PERCEPTION

TYPICAL

SNACKING DRIVE

LOW

ADDICTION RISK

Many people perceive that snacking behaviors and the inability to stop eating sweets are willpower based. While this may be true at times, much of the drive toward snacking and sweets is coded in our DNA. The snacking gene variations that we analyze have been applied in clinical practice for several years and there is an extremely high correlation between genetic variations and client reported snacking behaviors. The same holds true for sweets, there are genes that code for perception of sweet taste where each person can have a different perception of sweetness based on their gene variations.

There are also genes that code for the way our brains respond when we taste something sweet.

INTERPRETATION:

RECOMMENDATIONS

CARBOHYDRATES

GENETIC DATA

GENE	GENO TYPE
KCDT10	GG
MMAB	GC
PLIN1	TC
UCP1	TT
TCF7L2(1)	TC
TCF7L2(2)	TG
TCF7L2(3)	CC
TCF7L2(4)	CC
CEBPA	AG
ABCG4	AA
VLDLR	GG
IGF1R	GG
LPIN(2)	TT
AGER	CC
FTO(4)	CC
GIPR	TC

TYPICAL

OPTIMAL INTAKE

HIGH

OPTIMAL FIBER

Carbohydrates are frequently praised or villainized in dietary recommendations, but the one aspect that we have identified in the genomic data is that there is no right answer that fits every person.

Carbohydrates are a very individualized component of the diet and using the current scientific literature and our experience with genomics in clinical practice, the relevant and highest impact genes have been identified.

This is especially relevant when it comes to ideal body composition as some people will do better on lower carbohydrate intake while others tend to burn fat in the flame of a carbohydrate.

Be mindful of the fact that much of this response can be modified through epigenetics. Review your past experience and your food preferences with your coach.

INTERPRETATION:

RECOMMENDATIONS:

TOTAL FATS

GENETIC DATA

GENE	GENO TYPE
APOE (1)	TC
APOE (2)	TC
APOE (3)	..
PPARG	CG
FABP2	GG
APOA2	AG
APOB	AG
ADIPOQ	GG
TFAP2B	AA
FTO	TT
TNF	GG
LIPC	AG

MODERATE

OPTIMAL INTAKE

Primary fats of the human diet:

- Saturated fats (SFA)
- Monounsaturated fats (MUFA)
- Polyunsaturated fats (PUFA)

Depending on the source of the advice, you will hear about which ones are good for you and which ones are bad. The problem with this advice is two-fold; first, fats are a macronutrient that our bodies require for optimal health so there is no strict classification of good and bad. Second, there are significant individual differences in how each person responds to the different types of fat.

When using genetic variations to provide guidance on fat intake, it is important to understand that many of the studies used did not differentiate the types of fat. This section provides guidelines for planning the ideal percentage of calories from fat in your daily diet.

INTERPRETATION:

RECOMMENDATIONS:

SATURATED FATS

GENETIC DATA

GENE	GENO TYPE
APOE (1)	TC
APOE (2)	TC
APOE (3)	..
PPARG	CG
APOA2	AG
APOB	AG

LOW

OPTIMAL INTAKE

Dietary Sources of Saturated Fat:

- Pork (bacon, sausage)
- Red meats
- Cheeses
- Potato chips/fries
- Butter
- Coconut oil
- Chocolate

Saturated fats (SFA) represent one of the most debated aspects of human nutrition today. Various studies go back and forth regarding whether it is healthy or not healthy. The Atkins and Paleo movements have brought saturated fat into the forefront of discussions.

Bottom line is that saturated fats are needed for healthy human function. Saturated fat makes up 50% of the membrane fats in every cell of our body and is essential for healthy immune function. Our brain is 60% fat and is predominantly saturated fat and cholesterol. Despite this, there can be something to getting too much of a good thing.

Each individual carries genetic variations that can change the way they respond to saturated fats from a health and wellness standpoint. The algorithm used in this profile is based on leading scientific studies into genome wide associations as well as from our extensive experience in applying this in clinical practice.

Even with moderate intake recommendations it is best for most individuals to keep saturated fat intake to less than 10% of total calories.

INTERPRETATION:

RECOMMENDATIONS:

POLYUNSATURATED FATS

GENETIC DATA

GENE	GENO TYPE
APOA5	AA
BDNF	CC
TNF	GG
FADS1	TG
ELOVL2	AG
PTGS2	AA
COX-2	TT
IL-1B	AG

MODERATE

OMEGA-6 INTAKE

MODERATE

OMEGA-3 NEED

Polyunsaturated fatty acids (PUFA) have a role in many physiological processes, including energy production, modulation of inflammation, and maintenance of cell membrane integrity. Polyunsaturated fats (PUFAs) include the omega-6 and omega-3s, essential for life and there are health benefits to consuming both in the appropriate ratios.

Research has been focused on omega-6/omega-3 ratios and there is a clear benefit to keeping this ratio at 4:1 or less. While this is the beneficial zone, most people consume these fats in a 10:1 ratio. Many in the industrialized world are reaching levels as high as 25:1. These large ratios in favor of omega-6 are unhealthy and lead to significant inflammation and increased risk for detrimental health effects.

Several GWAS studies have looked at the genetic variations that impact serum levels of PUFAs in the population. Certain variations correlate with rate limiting enzyme activity in the conversion to beneficial forms while others can predict weight loss response to percentages of PUFAs in the diet.

INTERPRETATION:

RECOMMENDATIONS:

MONOUNSATURATED FATS

GENETIC DATA

GENE	GENO TYPE
ADIPOQ(1)	GG
ADIPOQ(2)	GG
APOA5	AA
BDNF	CC
TNF	GG
FAAH	AC
LPL	TC
IL-1B	AG

MODERATE

MUFA INTAKE

There are currently no strict recommendations on MUFA intake but suggestions range from 12-25% of total calories.

Monounsaturated fatty acids (MUFA) have a long list of studies in the scientific literature supporting the health benefits. Reported health benefits include; decreased inflammation, decreased cancer rates, decreased heart disease, and weight loss.

MUFA is suspected to be the major health benefit of the Mediterranean diet where some traditionally consume as much as 40% of their total calories from olive oil, a major source of MUFA.

MUFA are mainly omega-9 fatty acids but also includes the omega-7 fatty acids. The main sources of MUFA in our diets include; oils, nuts, meats, salmon, and avocado.

MUFA SOURCES:

- Olive oil
- Macadamia nut oil
- Avocado Almonds
- Macadamia nuts
- Beef
- Salmon
- Pumpkin seeds
- Chicken

INTERPRETATION:

RECOMMENDATIONS:

PROTEIN

GENETIC DATA

GENE	GENO TYPE
FTO(1)	TT
FTO(2)	TA
LPIN1	AA
BDNF-AS	AA
TFAP2B	AA

LOW

OPTIMAL INTAKE

Consider the biologic value of proteins. The biologic value is a measure of the proportion of absorbed protein from a food which becomes incorporated into the proteins of the body.

Protein is an important macronutrient that provides the amino acid building blocks for structures, enzymes, antibodies, and hormones. There are 20 amino acids that the body uses to create millions of different proteins and of those, ten are considered essential, meaning that we are not able to make them and we must consume them in our diets.

There are many GWAS that look at how our mix of macronutrients can affect our gene expression to create a specific response. Most of these studies have focused on body composition. This means that we can look at certain genetic variations that correlate with an outcome of changing the way certain genes are expressed that relate to obesity, fat storage, and body composition.

Some people will respond better to a diet with a higher percentage of calories from protein, while other do better with a lower percentage. This is a complex network of gene interactions and there are ways to epigenetically shift the expressions of these genes to achieve desired outcomes.

INTERPRETATION:

RECOMMENDATIONS

PLANT STEROLS

GENETIC DATA

GENE	GENO TYPE
ABCG8 (1)	TG
ABCG8 (2)	TC
ABCG8 (3)	TC
CETP	AA
ABCG5(1)	GG
ABCG5(2)	GG

LOW

PLANT STEROL RISK

LOW

PLANT STEROL BENEFIT

Plant sterols have been reported to lower LDL and triglycerides.

Plant sterols is the term for phytosterols and phytostanols, regardless of biological source. These are cholesterol-like molecules found in all plant foods, with the highest concentrations occurring in vegetable oils. They are absorbed only in trace amounts in normal circumstances, but some individuals possess the genetics to absorb greater amounts. Plant sterols work by inhibiting the absorption of intestinal cholesterol basically through competition for receptors and uptake. This also happens if they get absorbed into our blood stream. This can increase cardiovascular risk.

Generally, the amount of plant sterols taken in through dietary sources are tolerable but excess amounts are a potentially harmful. Supplement sources can come in a variety of forms; sterols, stanols, phytosterols, beta-sitosterol, campesterol and stigmasterol.

INTERPRETATION:

RECOMMENDATIONS:

METABOLISM

GENETIC DATA

GENE	GENO TYPE
GCKR	TC
LEPR	n/a
PPARGC1A	TT
MC4R	TT
UCP2	CC
FTO(4)	TT
UCP2(2)	GG
FTO(6)	TT

TYPICAL

ESTIMATED RMR

In this report, we look at genetic variations and how they tend to affect resting metabolic rate (RMR). RMR is a complex combination of genetics and environment and the genetics can be modified through epigentic influences.

The basal metabolic rate calculators (BMR) are rough estimates and should only be used as guides. In fact, the weight variable in the equation adds even more variability since it is most accurate when using the fat free mass (FFM), and FFM can be very different even for individuals that weigh the same in total body weight.

The BMR calculators are reported in some studies to be as much as 700 kcal off even when using FFM. BMR does not take into account the number of calories burned in daily activity, only resting.

INTERPRETATION:

RECOMMENDATIONS

MACRONUTRIENT WORKSHEET

CLIENT DEMOGRAPHIC:

Height: inches

Weight: lbs

Age:

Gender:

MACRONUTRIENT DAILY GOALS:

Carbohydrates	0	grams/day
Protein	0	grams/day
Fats	0	grams/day

BASAL METABOLIC RATE:

(estimated)

Calories/Day

Probability Based on Genetic Data

CARBS: 45%

FATS: 30%

PROTEIN: 25%

Recommended calorie mix is based on a combination of genetics and your lifestyle evaluation

Recommended Calorie Mix

CARBS:%

FATS:%

PROTEIN:%

TOTAL DAILY CALORIE GOAL: CALORIES/DAY

RECOMMENDATIONS

CENTER FOR HUMAN POTENTIAL
APEIRON

Genomic Supplementation Report

Client: Sample Client

VITAMIN B12

GENE	GENO TYPE
FUT2 (1)	GG
FUT2 (2)	AA
FUT(3)	AA
MTR	GG
VDR taq	AA
COMT	GG
TCN1	AG
MTRR A66G	GG
MTHFR 677T	GG

Since your body can't make vitamin B12, you should get it either from supplements or food sources. Foods that contain vitamin B12 are all animal products or have been fortified with B12. See end of report for foods high in B12.

TYPICAL SUPPLEMENTATION NEED

It's estimated that 40 percent of American's don't get enough vitamin B12. Vitamin B12 is absorbed through the stomach lining typically in the form of animal-based foods.

Benefits of Vitamin B12

B12 deficiency can contribute to fatigue and brain fog.

Benefits to increasing your vitamin B12 intake, include

- Increased energy – Because your body needs B12 to convert carbohydrates into glucose, it increases your overall energy and reduces fatigue.
- Improved brain function – Vitamin B12 helps make DNA and keep your nervous system healthy by reducing depression, stress levels, and reducing brain shrinkage.
- Healthy digestive system – B12 helps the gut and prevents heart disease by curbing cholesterol levels, protecting against stroke, and high blood pressure.

INTERPRETATION:

RECOMMENDATIONS

VITAMIN B6

GENE	GENO TYPE
NBPF3	TC
ALPL(1)	TC
ALPL(2)	AA

If you'd like to increase your vitamin B6 intake, you can do so naturally by adding certain foods to your diet. Foods that are notably high in B6 are listed at the end of this report. B6 is involved in over 100 metabolic processes.

TYPICAL SUPPLEMENTATION NEED

Vitamin B6, also called pyridoxine, helps your body turn food into energy, supports adrenal function, maintains a healthy nervous system, is important in metabolic processes, and supports the healthy development in babies' brains during pregnancy and breastfeeding.

Considered one of the most common vitamin deficiencies, vitamin B6 is consumed through diet or supplements.

Common symptoms of low vitamin B6 are irritability, depression, and anxiety. Some scientists believe that the levels for considering someone vitamin B6 deficient should be increased.

Benefits of Vitamin B6

- **Adrenal function** – Through regulating hormones, B6 helps you battle stress, stabilize your mood, and stay happy.
- **Metabolism** – Crucial to your hundreds of metabolic processes, B6 helps your body turn food into energy.
- **Healthy nervous system** – Vitamin B6 is often referred to as the happy vitamin because it helps make serotonin and norepinephrine, which impact your mood.
- **Digestive support** – B6 helps maintain healthy digestive processes.

INTERPRETATION:

RECOMMENDATIONS

VITAMIN A

GENE	GENO TYPE
BCMO1(1)	CC
BCMO1(2)	AA
BCMO1(3)	GG
BCMO1(4)	GG
BCMO1(5)	GG
BCMO1(6)	GG

Vitamin A is a nutrient that is easy to add to your diet with both supplements and food. Some of the best food sources are listed at the end of this report, add to your meals for a boost in skin, eye, and teeth health.

LOW

CONVERSION EFFICIENCY

Vitamin A is also called beta-carotene or retinoic acid. When it's found in food it's in the form of beta-carotene. In supplements, vitamin A can be in either form or both. Beta-carotene is the naturally occurring form that is converted by the body into retinoic acid, which is its usable version.

The gene BCMO1 is what the body relies to properly convert beta-carotene into its usable form. Your body relies on this conversion of vitamin A for healthy maintenance of the heart, kidneys, lungs, and eyes.

Benefits of Vitamin A

Improved immune system – Boosts the immune system and helps fight infection by increasing the lymphocytic responses against antigens. It also helps mucus membranes stay moist, which helps strengthen white blood cell activity

Healthy skin – With the ability to trap free radicals and toxins, vitamin A keeps your skin supple & healthy.

Increased tooth strength – Through forming a hard material just beneath the surface of your teeth called dentin, vitamin A keeps your teeth strong.

Eye health From moisture to macular health, it supports your eyes.

INTERPRETATION:

RECOMMENDATIONS

SELENIUM

GENE	GENO TYPE
GPx1	GG
SEPP1	TC
AGA	AC
BHMT	CC
MST1	GG
DMGDH	CC

While you can add selenium to your diet with a supplement, there are several foods that are naturally high in selenium. Some of these seleniumrich foods are listed at the end of this report.

INCREASED

SUPPLEMENTATION NEED

Selenium is an antioxidant, meaning it protects your body from harmful free radicals and oxidative stress. This delays cell damage and helps protect your body from oxidizing agents caused by many diseases and pollutants.

As an immunomodulator, Selenium is a more potent antioxidant than vitamins A, C or E. It's nutritionally essential for everyone, supports thyroid hormone metabolism, and protects against infections.

Benefits of Selenium

Thyroid support – Selenium is an important cofactor for three of the four thyroid hormone deiodinases, which activate and deactivate thyroid hormones and metabolites.

DNA repair – By neutralizing free radicals, selenium protects DNA, preventing serious damage.

Metal detoxification – Studies have shown that organic selenium supports the excretion of the harmful element mercury.

Reproductive health – Selenium is vital for both male and female reproductive health. In men, it enables sperm movement. In women, low selenium can negatively impact fertility and fetal development

INTERPRETATION

RECOMMENDATIONS

MAGNESIUM

GENE	GENO TYPE
CNNM2	CG
MUC1	CC
DCDC5	TT
Shroom3	AA
TRPM6(1)	TT
TRPM6(2)	CC

Magnesium is found in both plants and animalbased foods, making it easy to add to your diet. Foods that are rich in magnesium are listed at the end of this report.

There are a lot of delicious foods that contain magnesium so increasing your intake shouldn't be a problem. Though, if you think you are magnesium deficient you can also take it in supplement form.

INCREASED SUPPLEMENTATION NEED

Magnesium one of the seven macronutrients that is needed by your body in relatively high amounts. It's recommended that you consume at least 100 milligrams per day. Magnesium is vital to over 300 enzymatic reactions in your body including metabolism, transmission of nerve impulses, and synthesis of fatty acids and proteins. It impacts several bodily systems and can even affect your mood.

Too much magnesium can cause diarrhea and upset stomach.

Benefits of Magnesium

Bone strength – Magnesium helps assimilate calcium into your bones by activating vitamin D in your kidneys.

Healthy metabolism– Essential co-factor in many metabolic processes especially carbohydrate processing.

heart health – Magnesium is responsible for keeping your heart muscles healthy and strong. It also helps with the transmission of electrical signals throughout the body. Proper magnesium levels have shown to lower artery calcification, hypertension, and atherosclerosis (fatty buildup on artery walls).

Anxiety – Low magnesium levels have been shown to increase anxiety.

Gut – Magnesium works as a stool softener and can relieve constipation naturally.

INTERPRETATION:

RECOMMENDATIONS

CHOLINE

GENE	GENO TYPE
BHMT	GG
CHDH	TT
MTHFD1	AG
PEMT	TT
CHKA	CC

our liver produces choline, but not in sufficient quantities. Add choline to your diet through food or through supplements. Both vegetables and animal products contain choline. Not surprisingly, beef liver is the highest source of choline. Foods high in choline are listed at the end of this report.

Increasing your intake of these food or adding choline to your diet through a supplement can improve overall health.

TYPICAL SUPPLEMENTATION NEED

Your body can produce choline, although, it doesn't produce sufficient quantities of this essential nutrient to maintain optimal health and less than 10 percent of adults get enough choline in their diet.

Some risks of choline deficiency include muscle damage, anxiety, brain fog, and fatty liver.

People that are at risk for choline deficiency include:

- Pregnant women
- Choline depleted diets (plant based diets)
- People with genetic variations

**It's particularly important for pregnant women and babies to get enough choline to ensure healthy brain development.

Benefits of Choline

Cell structure – Your body relies on choline to make the fats that help maintain the structural integrity of all cell membranes.
Cell messaging – Choline assists with the production of compounds that act as cell messengers.
Fat transport and metabolism – Insufficient choline levels can cause fatty liver.
DNA synthesis – Choline, vitamin B12, and folate are three vital nutrients in DNA synthesis.
Nervous system health – Acetylcholine, a neurotransmitter involved in memory, muscle movement, and regulating heartbeat, is derived from choline.

INTERPRETATION:

RECOMMENDATIONS

VITAMIN C

GENE	GENO TYPE
SLC23A1(1)	AA
SLC23A1(2)	TC
SLC23A2	AA

Citrus foods, such as oranges, are known to have high vitamin C levels but there are many foods that have high concentrations of vitamin C. These are listed at the end of this report. By adding these fruits and vegetables to your diet or increasing your intake you can generally not require supplementation.

INCREASED

SUPPLEMENTATION NEED

Also known as ascorbic acid, vitamin C is a nutrient that plays several key roles in bodily functions. Vitamin C is a powerful antioxidant, trapping free radicals and preventing the harmful effects of toxins. It isn't produced by the body naturally and must be consumed in the form of fruits and vegetables.

People at risk for inadequate levels of vitamin C are those with particular lifestyle habits, genetics, or diets lacking in vitamin C.

Benefits of Vitamin C

Collagen synthesis – VVitamin C helps repair and regenerate tissues. Maintains healthy skin and connective tissue.

Protection against heart disease – Through increasing the body's level of glutathione, vitamin C protects the arteries.

Iron absorption – Assists iron absorption, vitamin C prevents anemia.

Cholesterol and triglyceride reduction – Vtamin C reduces the risk of heart attack and stroke.

Blunts oxidative stress – In diseases states, vitamin C has shown to help reduce cellular damage by free radicals.

INTERPRETATION:

RECOMMENDATIONS

VITAMIN E

GENE	GENO TYPE
CD36	CC
SCARB1	GG
ZPR1	CC
GSTP1	AA
TNF	AG
IL10	CC

Vitamin E is relatively easy to add to your diet in plant-based forms. Foods that are high in Vitamin E are listed at the end of this report.

INCREASED

SUPPLEMENTATION NEED

LOW

INFLAMMATION RISK

Vitamin E is a fat-soluble antioxidant that plays a vital role in many aspects of your health.

Vitamin E is a term that includes eight compounds in two subgroups (tocopherols and tocotrienols) that each vary in biological activity. Alpha-tocopherol is the only form of the eight that is readily absorbed and used by your body.

Your liver is primarily responsible for using the alpha-tocopherol form of vitamin E taken in through food, supplements, and by converting it from other vitamin E forms.

Benefits of Vitamin E

Helps store vitamins A, K, iron, and selenium – Vitamin E helps maintain sufficient levels of many essential nutrients.
Supports the formation of red blood cells – ed blood cells rely on vitamin E to strengthen their interior lining, which is another way it toughens the immune system.
Keeping skeletal, cardiac, and smooth muscles healthy – Vitamin E is important for both the structural functional and maintenance of these.
Prevent eye damage – Studies have suggested that relatively high vitamin E intake may reduce the risk of macular degeneration and cataracts in elderly individuals.

INTERPRETATION

RECOMMENDATIONS

VITAMIN D

GENE	GENO TYPE
GC	TG
CYP2R1	GG
CYP2R1(2)	AA
DHCR7	AC
VDR fok	AA
CYP27B1	TT
CYP24A1	AA
Klotho	TT
VDR bsm	CC
VDR taq	AA
VDR apal	CC

Vitamin D is not just a matter of getting it from food. Sunlight is needed to adequately convert Vitamin D to usable forms.. It is important to consume foods which are high in vitamin D, listed at the end of this report.

INCREASED SUPPLEMENTATION NEED

YES

SUNLIGHT BENEFIT

NO

TESTING CAUTION

Vitamin D is a fat-soluble nutrient that is not readily found in many foods. An estimated 70 percent of the population is thought to be vitamin D deficient. This is concerning because it's a nutrient that's responsible for regulating over 1000 genes in the human genome.

From a genetic standpoint, people tend to vary in their ability to process vitamin D. This means that there's a difference in the baseline amount needed to maintain healthy vitamin D levels from person to person.

Benefits of Vitamin D

Bone health – Through increasing calcium and phosphorus absorption, vitamin D strengthens bones.
Prevention of diabetes – Studies have shown that vitamin D can decrease your risk of getting both Type 1 and Type 2 diabetes.
Heart health – Studies have shown vitamin D deficiency as a risk factor for congestive heart failure and heart attacks.
Mood regulator – Vitamin D is thought to reduce or prevent depression.
Muscle growth – Vitamin D has been shown to aid in muscle growth and retention in both adults and the elderly.

INTERPRETATION

ECOMMENDATIONS

VITAMIN K

GENE	GENO TYPE
APOE(1)	TC
APOE(2)	TC

TYPICAL SUPPLEMENTATION NEED

Vitamin K includes a family of compounds, including vitamin K1 and vitamin K2. Also known as phylloquinone, vitamin K1 is found in plants, mostly leafy green vegetables. Vitamin K2 or menaquinones, is usually of bacterial origin and can be found in some animal-based and fermented foods but is mostly converted by the large intestine or liver from vitamin K1.

Many people get an adequate amount of vitamin K through their diet. It's also present in most multivitamin supplements. Genetics can be an active player in this process and can be predictive of absorption and conversion.

Benefits of Vitamin K

Bone health – In a study in the Netherlands, vitamin K2 was three times more effective in raising osteocalcin than K1, which is important to bone metabolism.
Blood clotting – Vitamin K is essential to blood clotting. In fact, in studies of severe vitamin K deficiency, clotting was almost impossible.
Supporting the efficacy of vitamin D – Vitamin K improves the impact of vitamin D when they are taken in combination.

INTERPRETATION

RECOMMENDATIONS

FOLATE

GENE	GENO TYPE
MTHFR 677T	GG
MTHFR 1298C	GG
DHFR(1)	TC
FOLR1	GG
DHFR(2)	ID
SLC19A1	TT

TYPICAL SUPPLEMENTATION NEED

Note that there is a difference between folate and folic acid when you look at supplements or fortified foods. Folic acid is a synthetic form that requires the body to convert to usable form - folate. Certain genetic variants can create a risk of potential adverse outcomes with excess folic acid.

Folate or vitamin B9 is a water-soluble nutrient that is available in many foods and typically found in multivitamin supplements. Getting enough folate is important to cognitive function, cardiovascular disease, cancer, birth defects, and depression.

While it's difficult to get too much folate from food, it's possible to take too much folic acid in the form of supplements or fortified foods.

Crucial parts of your body – your brain, heart, and all the way down to your DNA – rely on sufficient folate levels for optimal health.

Benefits of Folate

DNA synthesis and repair – Functioning as a coenzyme, folate helps with the synthesis of DNA and RNA and the metabolism of amino acids.

Tissue growth – Folate's role in synthesis makes it essential to tissue and cell growth.

Cardiovascular health – Studies have shown that folate encourages normal cholesterol levels.

Neurological health – Most observational studies show that higher folate levels correlate with low Alzheimer's disease and dementia.

INTERPRETATION

RECOMMENDATIONS

THIAMINE

GENE	GENO TYPE
SLC19A3	CC

INCREASED

SUPPLEMENTATION NEED

Thiamine deficiency can cause difficulty digesting carbohydrates. In severe deficiencies this allows a substance called pyruvic acid to build up in the bloodstream, causing a loss of mental alertness, difficulty breathing, and potentially heart damage.

Thiamine is a vitamin that is also referenced as vitamin B1. Thiamine is essential to many body functions, including nervous system integrity, muscle function, digestion, and carbohydrate metabolism. Very little thiamine is stored in the body and depletion can occur quickly when not supplied through diet or supplementation. It is sometimes called an "anti-stress" vitamin because it can strengthen the immune system and improve the body's ability to withstand stressful conditions.

Benefits of Thiamine

- **Energy production** - B1 is responsible for converting sugar into energy. The vitamin acts as a co-enzyme in oxidizing sugar to produce energy for the smooth functioning of the body organs, especially the heart, brain, lungs, and kidneys.
- **Improves brain function** - It ensures smooth functioning of the brain and helps improve memory and concentration. Vitamin B1 helps relieve stress and also helps strengthen the nerves.

INTERPRETATION

RECOMMENDATIONS

COPPER

GENE	GENO TYPE
SELENB1	TT
ATP7B	GG

TYPICAL SUPPLEMENTATION NEED

Copper is a micronutrient that is involved in many processes in the body and can easily become deficient due to the lack of intake in even a healthy diet. The body cannot produce copper on its own so it requires intake in the diet or through supplementation. It is also a micronutrient that has a narrow range of safety - very common to be deficient and easy to become toxic if over-supplemented. Most whole food sources are very low in copper.

Benefits of Copper

Cognitive function - Too little or too much copper can have a negative impact on the brain. Ideal levels promote growth and development of brain pathways.

Thyroid health - An important cofactor in promoting optimal thyroid levels and a healthy thyroid is important in keeping adequate absorption of copper from the gut.

Long-term health - Copper is a requirement for ongoing maintenance & repair DNA.

Bone - Copper is important in maintenance and repair of bone and cartilage. Deficiencies can lead to low bone density.

Blood - Lack of copper can also lead to anemia.

Skin & hair - Copper is involved in the production of melanin, a pigment responsible for skin and hair color.

INTERPRETATION

RECOMMENDATIONS

ZINC

GENE	GENO TYPE
CA1	GG
PPCDC(1)	TT
PPCDC(2)	TT

CAUTION

SUPPLEMENTATION NEED

Zinc is considered an essential trace element and is involved in assisting at least 100 different enzymes in the human body. It is also a common micronutrient deficiency. Deficiencies can lead to many health issues and optimal levels are essential to thrive. Plant based sources of zinc generally provide significantly lower bioavailability than animal based food sources.

Benefits of Zinc

Immunity -Zinc is an essential nutrient for our immune system..

Cognitive function - Essential for optimal communication between neurons in the brain.

Common cold - Zinc has been shown to lessen the severity and duration of the common cold.

Wound healing - From decreasing inflammation and reducing bacterial growth to helping maintain skin integrity.

Taste & smell - The taste and smell are reliant on zinc for proper function and has been shown to heighten these senses.

Weight loss - Deficiencies can lead to overeating. It works through the appetite hormone ghrelin to decrease the urge to overeat.

INTERPRETATION

RECOMMENDATIONS

IRON

GENE	GENO TYPE
TMPRSS6	AG
TF	AA
TFR2	AA
HFE(1)	CG
HFE(2)	GG

TYPICAL SUPPLEMENTATION NEED

TYPICAL RISK FOR EXCESS

Iron is essential mineral and is the world's most common nutritional deficiency disease. Deficiency is most prevalent among children and women of childbearing age. Almost 10% of women in developed countries are iron deficient.

Fatigue, insomnia, hair loss, and ice crunching are common signs of deficiency. Inadequate intake of vitamin C can can contribute to iron malabsorption. Plant based iron is not incorporated as well as heme-iron from animal sources.

Benefits of Iron

Hemoglobin production - Dietary iron is a critical component in the formation of hemoglobin and oxygen transport.
Oxygen transport - A form of hemoglobin found in muscle cells is myoglobin. Myoglobin carries oxygen from hemoglobin and diffuses it throughout muscle cells.
Muscle function - Iron is critical for oxidative metabolism in the brain and it is a co-factor in the synthesis of neurotransmitters. Insufficient iron in the diet is associated with decreased brain iron and with changes in behavior and cognitive functioning.

INTERPRETATION

RECOMMENDATIONS

NITRIC OXIDE

GENE	GENO TYPE
NOS1(1)	TC
NOS1(2)	GG
NOS2	AG
NOS3(1)	AG
NOS3(2)	TC
NOS3(3)	TG

HIGH

SUPPLEMENTATION NEED

Nitrates have had a negative connotation over the years due to the suspicion of causing health problems such as stomach cancer. This was due to their association with the nitrosamines that could be found in cured and smoked meats as well as fermented foods. In many countries the amount of nitrates in processed meats has been substantially reduced and they add vitamin C which reduces the chances of nitrosamine formation during high heat cooking.

Our bodies are capable of producing nitric oxide but some genetic variants can alter that ability. Nitrates are the natural forms of nitric oxide that the body can utilize.

Kale and spinach will generally have substantially more nitrate content than a hot dog or bacon.

80% of the nitrates in our diet come from vegetables. Interestingly, organic vegetables have less nitrates than conventionally farmed food due to reduced use of nitrogen based fertilizers.

Benefits of Nitric Oxide

Cardiovascular – Nitric oxide, endogenously produced or from nitrates can lower blood pressure and dilate blood vessels.

Exercise – Nitrates have been shown to decrease oxygen requirements of muscles during exercise and lead to greater time to fatigue.

Brain – Nitric oxide is a potentent antioxidant in the brain and it serves a function as a neurotransmitter.

Immunity – Nitric oxide is used by our immune cells to kill invading bacteria.

INTERPRETATION

RECOMMENDATIONS

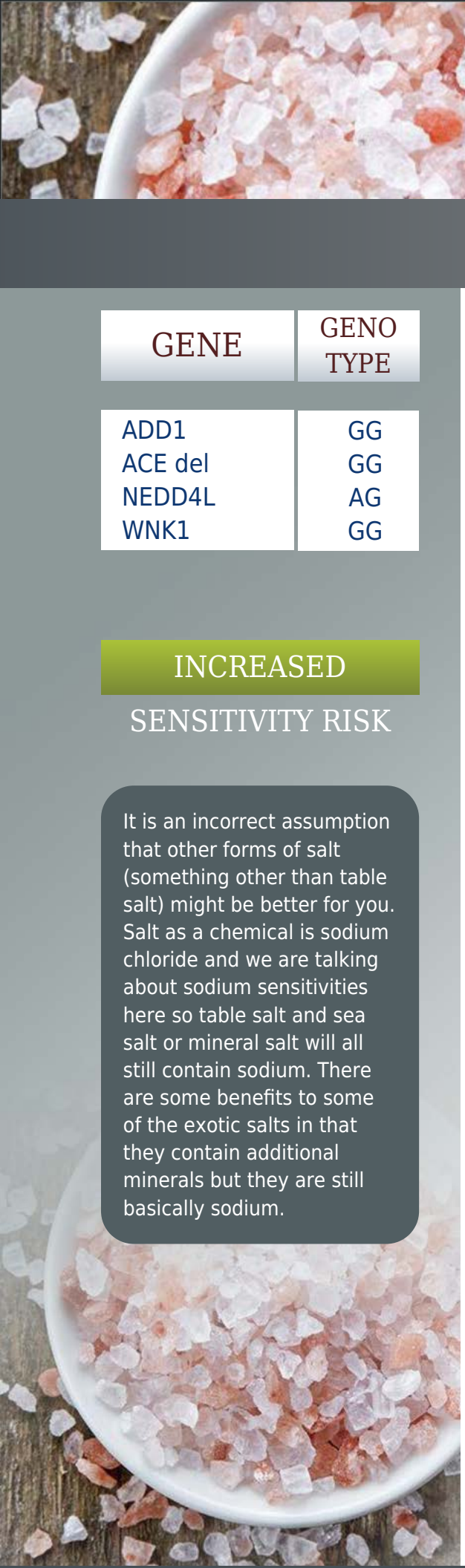
SODIUM

GENE	GENO TYPE
ADD1	GG
ACE del	GG
NEDD4L	AG
WNK1	GG

INCREASED

SENSITIVITY RISK

It is an incorrect assumption that other forms of salt (something other than table salt) might be better for you. Salt as a chemical is sodium chloride and we are talking about sodium sensitivities here so table salt and sea salt or mineral salt will all still contain sodium. There are some benefits to some of the exotic salts in that they contain additional minerals but they are still basically sodium.



Sodium or salt is another nutrient that has had its share of bad press. Sodium balance is one of the most exquisitely monitored systems in the body. Because sodium is so important to the maintenance of health, it is finely tuned to a very narrow and precise level. Taking excess sodium for most people will not result in health problems, assuming the body is functioning well.

One aspect to keep in mind is the genetics we possess may impart an increased risk for taking excess sodium in the diet. There are genetic variations that can result in an alteration in the processing pathways which can create potential adverse reactions to too much salt in the diet.

Types of Sodium:

Table salt: sodium chloride and iodine

Sea salt: slightly lower sodium plus calcium, potassium, iron oxide (pink color)

Himalayan salt: slightly lower sodium plus calcium, potassium, iron oxide (pink color)

Celtic salt: slightly lower sodium and trace minerals

INTERPRETATION

RECOMMENDATIONS

CAFFEINE

GENE	GENO TYPE
CYP1A2(1)	AA
CYP1A2(2)	TC
AHR	TC
ADORA2A	..
ADA	..

HIGH

METABOLISM

LOW

ANXIETY RISK

Metabolism – How well do we metabolize caffeine? The half-life of caffeine is 5.7 hours.

Anxiety – How does your brain respond to caffeine? Brain wave patterns can have variable responses to caffeine depending on your individual genetics.

Sleep – Caffeine can assist some people with shaking off a nights sleep and clearing up the brain fog; while others will experience sleeplessness from even small doses of caffeine.

Caffeine is one of the most researched substances in the history of science. Good or bad? The answer may actually reside in your genetics. The interactions of caffeine in our body is a complex process and requires a full systems look to see if it is truly good or not so good for each individual.

Benefits of Caffeine

Energy – Caffeine can improve daily energy by interfering with a substance called adenosine.
Fat burning – Caffeine is one of only a handful of natural substances that has been proven to improve fat loss.
Physical performance – Caffeine is a true performance enhancing substance.
Reduced risk of neurodegenerative diseases - Coffee itself has been linked to reduced risk of cognitive decline.

INTERPRETATION

RECOMMENDATIONS

INTERPRETIVE NOTES:

VITAMIN FOOD SOURCES:

Vitamin B12:

- Liver • Salmon • Milk • Yogurt • Tuna • Mackerel • Sardines • Red meat • Raw cheese • Eggs

Vitamin B6:

- Brewer's yeast • Bananas • Milk • Cheese • Eggs • Fish • Sunflower seeds • Carrots • Spinach • Peas • Legumes • Potatoes

Vitamin A:

- Fish liver oil • Cream • Egg yolk • Beef liver • Cheddar cheese • Butter • Sweet potato • Carrots • Broccoli • Mango • Spinach • Pumpkin • Apricot • Peach • Papaya • Collard greens

Selenium:

- Brazil nuts • Tuna • Halibut • Beef liver • Turkey • Sardines • Sunflower seeds • Pork • Mushrooms

Magnesium:

- Dark leafy greens • Sesame seeds • Brazil nuts • Mackerel • White beans • Quinoa • Avocados • Yogurt • Bananas • Dark chocolate

Choline:

- Beef liver • Eggs • Chicken breast • Cauliflower • Broccoli • Mushrooms • Soybeans • Dark leafy greens • Shellfish • Asparagus • Brussel sprouts • Bok choy • Cod

Vitamin C:

- Bell peppers • Guava • Dark leafy greens – especially turnip greens • Kiwi • Broccoli • Strawberries • Tomatoes • Peas • Papaya • And of course, citrus fruits – oranges, grapefruits, lemons

Vitamin E:

- Almonds • Sunflower seeds • Swiss chard • Pine nuts • Broccoli • Mustard greens • Avocado • Spinach • Turnip greens • Kale • Plant oils • Hazelnuts

Vitamin D:

- Fatty fish – Tuna, mackerel, salmon • Beef liver • Cheese • Egg yolks • Cod liver oil • Fortified drinks – Milk and sometimes orange juice • Fortified foods – Cereals and grains

Vitamin K:

- Spinach • Kale • Turnip greens • Collards • Swiss chard • Mustard greens • Parsley • Romaine • Brussel sprouts • Broccoli • Cauliflower • Cabbage

Folate:

- Beef liver • Spinach • Broccoli • Bananas • Strawberries • Oranges • Beans • Avocado • Tomatoes • Beets • Celery • Asparagus • Legumes • Yeast • Cereal • Mushrooms • Fish • Eggs

Nitric Oxide:

- Spinach • Kale • Beets • Carrots • Legumes • Celery • Eggplant • Ham • Bacon • Pastrami • Salami • Hot dogs • Sausages

Thiamine:

- Beef • Brewer's yeast • Legumes (beans, lentils) • Milk • Nuts • Oats • Oranges • Pork • Rice • Seeds • Wheat • Whole-grain cereals • Yeast • In industrialized countries, food made with white rice or white flour is often enriched with thiamine.

Iron:

- Legumes • Lentils • Soy beans • Whole grains • Green leafy vegetables • Cereals • Bread • Spinach • Turnip • Fish • Eggs • Meat (especially high in red meats) • Sprouts • Broccoli • Dry fruits

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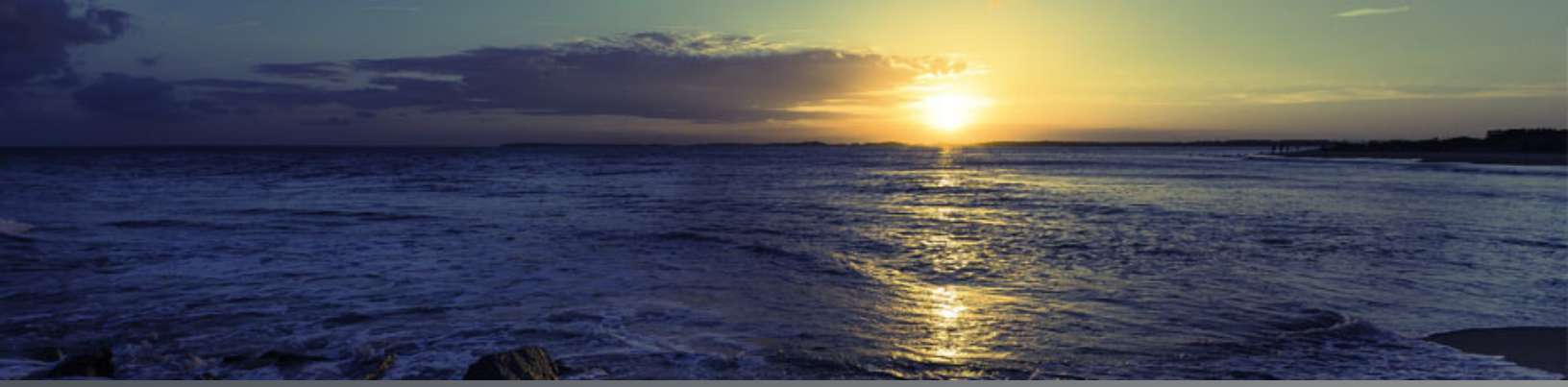
Sleep Genotype Report

Client Name: Sample Client



CIRCADIAN PROPENSITY

GENETIC DATA

GENE	GENO TYPE
PER2(1)	n/a
PER2(2)	CC
PER3(1)	AG
PER3(2)	CC
AANAT	..
CSNK1D	n/a
GNB3	
ARNTL	CC

MORNING

YOUR CHRONOTYPE

Humans are a diurnal species. We are active during the day and sleep at night. Many us feel more awake, alert and capable of our best work effort in the morning. However, there are those at the opposite end of the spectrum who prefer to stay up late and sleep well into the daytime hours. These individuals find themselves most alert in the evening.

The circadian rhythm is a cycle that signals our bodies when to sleep, rise and eat. Individual circadian variations are governed by the internal circadian clock network. This internal biological clock network resides in the brain and regulates the timing of functions such as appetite, hormone release, and metabolism. Our perception of this clock is the basic sleep-wake process.

Recent research has revealed that the circadian clock is not as basic as we once suspected. While we have tendencies toward a basic rhythm, what determines our desire to wake early or stay up late, is influenced by the same system that regulates the cycling of many bodily functions. Forcing the body to fit into a sleep-wake cycle that does not match our genetics, can lead to circadian dyssynchronization

INTERPRETATION:

RECOMMENDATIONS

SLEEP ONSET

GENETIC DATA

GENE	GENO TYPE
NPSR1	AT
CLOCK(3)	AA
PER3(1)	AG
PER3(2)	CC
AANAT	..
CACNA1C(1)	TT
CACNA1C(2)	AA
COMT	GG

TYPICAL PROPENSITY

Normal sleep latency, the time from lying down to the first stages of sleep, is approximately 15-20 minutes. Falling asleep faster indicates a degree of sleep deprivation. Often, individuals with a genetic propensity for later sleep, try to force themselves into a different chronotype (circadian rhythm) which can result in significant sleep onset delays

Individual genetics play a large role in the prediction of longer or shorter periods of sleep onset. Despite possessing a propensity for delayed sleep onset, identifying the genetic components that are most impactful to the process allows a much more directed and personalized approach to optimal sleep interventions.

Normal time to onset of sleep is about 15-20 minutes. Onset of sleep outside of this range usually indicates either a genetic or lifestyle component

- 0-5 minutes = severe sleep deprivation
- 6-15 minutes = moderate sleep deprivation
- 15-20 minutes = normal
- >20 minutes = probable genetic or environmental

INTERPRETATION:

RECOMMENDATIONS:

SLEEP DURATION

GENETIC DATA

GENE	GENO TYPE
NPSR1	AT
CLOCK(1)	AG
CLOCK(2)	AA
PER2(1)	n/a
GNB3	CC
ADA	..
ABCC9	n/a
GRIA3(1)	AC
ABCC9	n/a
CLOCK(4)	AG
DEC2	GG
COMT	GG

TYPICAL PROPENSITY

When we don't get the sleep we need, we experience surges of stress hormones which disrupt our cognition and ability to regulate emotions. 90% of adults require 7 - 9 hours of sleep a night.. Lost sleep reduces brain power and productivity, diminishes concentration and impairs memory. It lowers creativity, reduces the ability to communicate, impairs motor skills and increases stress and anxiety

Studies have demonstrated that just two hours of sleep deprivation (5 - 6 hours of sleep) results in a vigilance level equivalent to the consumption of two alcoholic drinks. Interestingly, while there is a detrimental decline in vigilance our perceived level of vigilance will be normal.

Many factors contribute to how long we sleep. Assessing your genetic sleep variations and establishing effective sleep strategies are essential steps in the process of sleep optimization.

Sleep requirements:
Teens 9-10 hours
Adults 7-9 hours

INTERPRETATION:

RECOMMENDATIONS:

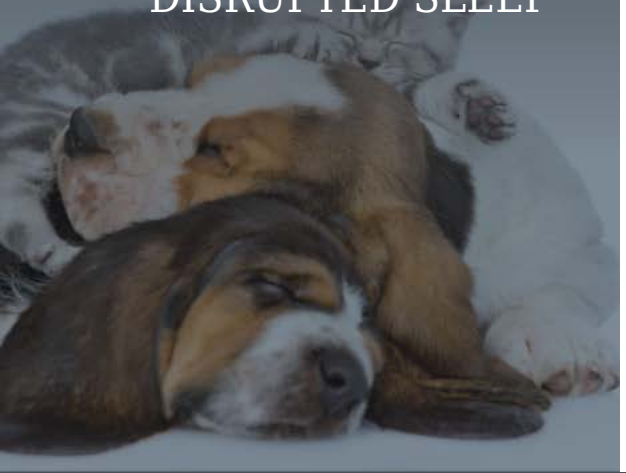
DISRUPTION OF SLEEP

GENETIC DATA

GENE	GENO TYPE
TNFa	GG
BDNF	CC
MTNRB	CC
PPP2R4	AG
ADORA2A	..
GRIA3(1)	AC
COMT	GG
ARNT	GC
FABP7	CC
GNB3	CC

LOW

PROPENSITY FOR DISRUPTED SLEEP



Sleep Matters. It is the single most important thing you can do to improve performance in life. Sleep quality is determined by a complex network of interacting physiological processes which are strongly influenced by lifestyle. The quality of our sleep is influenced by the amount of deep sleep, the number and duration of waking episodes and the number of REM episodes. When our lifestyle is not in sync with our chronotype, sleep quality can be significantly impacted.

Genetic predispositions can evaluate variables such as melatonin production and response, excitatory versus inhibitory neurotransmitters and responses to our environment - light, caffeine, etc. Once a genetic variable is identified, a directed approach can be taken to improve the underlying issue. It is important to first get sleep patterns matching before addressing sleep quality.

INTERPRETATION

RECOMMENDATIONS

NARCOLEPSY RISK

GENETIC DATA

GENE	GENO TYPE
HLA-DRB1	AG
TRCA	TT
P2RY11	AA

TYPICAL

RISK

Narcolepsy is a long-term neurological disorder that involves a decreased ability to regulate sleep-wake cycles. It has been estimated that 1 in 2000 people are affected by narcolepsy.

There are two types of narcolepsy:

Type 1

Irrepressible need to sleep or daytime lapses into sleep occurring for ≥3 months.

Cataplexy (paralysis of motor control)

Cataplexy is absent

Spinal fluid hypocretin-1 concentration ≤110 pg/mL.

Type 2

Daily periods of irrepressible need to sleep occurring for ≥3 months

A mean sleep latency of ≤8 minutes and ≥2 sleep-onset REM periods.

Cataplexy is absent

Spinal fluid hypocretin-1 concentration >110 pg/mL

INTERPRETATION:

RECOMMENDATIONS:

RESTLESS LEG

GENETIC DATA

GENE	GENO TYPE
MEIS1	TT
BTBD9(1)	TT
BTBD9(2)	AA
MAP2K5(1)	AG
MAP2K5(2)	AG
PTPRD	GG

TYPICAL

PROBABILITY

Excessive sleep movement is a condition that can disrupt not only your sleep but also the sleep of your bedmate. This is sometimes referred to as restless leg syndrome (RLS) and describes a condition that causes a strong urge to move one's legs. It is characterized by an overwhelming need to move the legs and symptoms tend to be worse at night. Spontaneous movements are triggered by rest, relaxation, or sleep.

Potential causes include iron deficiency, genetic predisposition, brain neurotransmitter imbalances and increased brain glutamate. There are no specific tests to confirm this condition.

Carrying genetic predispositions does not suggest that it is present, this is solely based on probability. There will be many people that have the symptoms without a genetic predisposition as well. The genetic variations can provide a guide to designing interventions that will have a higher probability of mitigating the symptoms.

INTERPRETATION:

RECOMMENDATIONS

RECOMMENDATIONS

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DETOX GENOMICS

Client: Sample Client

PHASE 1 DETOXIFICATION

GENETIC DATA

GENE	GENO TYPE
CYP1A1	AA
CYP1B1(1)	CG
CYP2A6(2)	AC
CYP2C9	TC
CYP2C19	AA
CYP2D6	CC
CYP2D6(3)	AG
CYP2D6(4)	AG
CYP2E1(1)	GG
CYP2E1(2)	CC
CYP3A4	TT

ESTROGENS

MEDICATIONS

ANESTHETICS

ACETAMINOPHEN

NSAIDS

Phase I detoxification is handled by a set of enzymes referred to as the Cytochrome P450's. There are around 18 different families of these enzymes and their production and function is controlled by over 50 genes. It is important to understand that despite the genetic controls, we do have the ability to alter the expressions of these genes in both positive and negative ways.

Cytochrome P450 enzymes are located predominantly in the liver but they are also found in other tissues such as the small intestine and even the brain. These enzymes are responsible for taking toxins through the first phase of detoxification. This first phase can convert toxins into benign forms but it also has the potential of creating an even more toxic product. Therefore, it is important to pay attention to both phase 1 and phase 2 detoxification pathways.

Toxins include; environmental toxins, medications, supplements, and even ones that are produced by our own metabolism and physiology.

INTERPRETATION:

RECOMMENDATIONS:

METHYLATION

GENETIC DATA

GENE	GENO TYPE
COMT	GG
MTHFR 677T	GG
MTHFR 1298C	GG
DHFR(1)	TC
DHFR(2)	ID
FOLR1	GG
SLC19A1	TT
CBS	AG

TYPICAL GENETIC PROPENSITY

Methylation is one of the most important processes in the body and it plays a significant role in detoxification. In fact, methylation is involved in over 200 enzymatic reactions in the body and these reactions are occurring over a billion times per second within our cells.

Methylation is involved in a host of chronic disease situations including; heart disease, diabetes, cancer, multiple sclerosis, autism, and other neurologic conditions. Much of these risks are related to the role that methylation plays in detoxification.

There are also many cofactors that we should address when optimizing this system. B12, B6 and folate are all important to consider when deal with variants of genes involved in the methylation process. Supplementation is important but there are also lifestyle factors that will impact methylation including; not smoking, reduction of alcohol, exercise, and stress optimization.

Genetics can play a major role in the function of this system.

INTERPRETATION:

RECOMMENDATIONS:

ACETYLATION

GENETIC DATA

GENE	GENO TYPE
NAT1(1)	CC
NAT1(2)	GG
NAT2(1)	TT
NAT2(2)	GG
NAT2(3)	GG

TYPICAL

GENETIC PROPENSITY

Acetylation is one of the many major phase II detoxification pathways. The acetylation pathway involves adding acetyl molecules to toxins to facilitate elimination from the body. The two primary enzymes involved are N-acetyltransferase 1 and 2 (NAT1 and NAT2)

Most detoxification is performed in the liver but NAT is in many organ systems of the body including; intestinal tract, lungs, and kidneys where it makes up a line of defense against many environmental toxins, metabolic byproducts, and various prescription as well as nonprescription medications.

It works to help detoxify:

- Histamines
- Tobacco smoke
- Exhaust fumes
- Medications

Genetics variations play a role in the function of NAT and certain variations can classify individuals into slow, intermediate, or rapid metabolizers. Diminished function of these NAT enzymes can lead to organ toxicities and potential cancer risks.

INTERPRETATION

RECOMMENDATIONS

GLUTATHIONE SYSTEM

GENETIC DATA

GENE	GENO TYPE
GPx1(1)	GG
GPx1(2)	CC
GPx1(3)	GG
GSTP1(1)	AA
GSTP1(2)	CC
GSTT1	TT
GSTM1(1)	CC
GSTM1(2)	AA
GCLC	TC
CAT	TC
CAT(2)	TC
TXN	AA

SUPPORT NEEDED

The glutathione system is the master regulator of detoxification and free radical manager of the human system. While many people focus on taking antioxidants and free radical scavenging supplements, this may not be the best option for optimizing the body. Focusing on the glutathione system will allow the body to manage these in a more efficient manner.

In addition to free radical scavenging, glutathione,

- Boosts immune function
- Enhances mitochondrial function
- Repairs DNA
- Detoxifies: Heavy metals, medications, environmental toxins, and pollutants

INTERPRETATIVE NOTES:

INTERPRETATIVE NOTES:

MITOCHONDRIA

GENETIC DATA

GENE	GENO TYPE
SOD2	GG
SOD(2)	AA
UCP2	CC
UCP4	CC
SIRT1	TT
SIRT5	TC
COX6B1	CC
ATP5C1(1)	GG
ATP5C1(2)	TT
NDUFS2	AA
NRF2(1)	GG
NRF2(2)	GG
NQO1	GG

STRONG

The mitochondria are classically viewed as the batteries of our cells. They produce the energy required for cell function, typically in the form of ATP. Mitochondria also have their own set of DNA and mitochondrial DNA is only passed to subsequent generations from the mother.

Many chronic health conditions are either directly or indirectly related to the function of our mitochondria and health experts have increasingly focused on maximizing mitochondrial health to optimize the human system.

Because of energy production, the mitochondria produce free radicals. Free radicals are thought of as damaging molecules. They can create oxidative stress which leads to chronic disease and poor health. This is not the whole truth, however, as some free radicals are essential and beneficial. Therefore, it is important to maintain balance and homeostasis in the body free radical system.

INTERPRETATION:

INTERPRETATION:

MOLD SENSITIVITY

GENETIC DATA

GENE	GENO TYPE
HLA-DRA	TT
HLA-DRA(2)	AG
HLA-DRA(3)	AA

INCREASED

GENETIC RISK

It is estimated that nearly 25% of the population carry genetics that predispose them to mold sensitivity. Persons carrying certain variants tend to not make the antibodies necessary to rid the body of the mold toxins. qhffs can lead to chronic inflammation and overall diminished health.

Mold is classified as a biotoxin and there are two factors that we need to consider when determining risk of chronic infection:

- Level and duration of exposure
- Genetics of the detoxification system

When it comes to mold, consider that according to the Environmental Protection Agency (EPA) Building Assessment Survey and Evaluation (BASE) study, 45% of U.S. buildings have current/ongoing water damage and 85% have past water damage. This is ripe breeding ground for toxic mold.

Symptoms can include:

- Chronic headache
- Fatigue/malaise
- Dizziness
- Memory problems/Brain fog
- Muscle aches
- Cough
- Shortness of breath

INTERPRETATION

RECOMMENDATIONS:

LYME DISEASE

GENETIC DATA

GENE	GENO TYPE
TLR1	AC
ACSL1	AG
GAD1(1)	AG
GAD1(2)	TC
GAD1(3)	GG
PON1	n/a
GSTP1	CC
SOD2(2)	AA

INCREASED

GENETIC RISK

Lyme is a bacterial disease caused by a bacterium called Borrelia and is transmitted by ticks. Acute infection is well documented and early treatment is important. Chronic Lyme disease is a bit more complicated and can be difficult to diagnose due to a low incidence of detecting the infectious organism after initial treatment. Antibody titers are used but only confirm past infection.

The diagnosis of chronic Lyme disease is based primarily on symptoms. These symptoms can include;

- Chronic fatigue
- Headache
- Muscle and joint aches
- Memory loss /other cognitive impairments
- Numbness/tingling
- Gastrointestinal symptoms

Chronic Lyme disease is becoming a significant issue and several genetic variants have been identified that predispose individuals to more significant health issues. The genetic variants look at immune response as well as glutamate production since this is a significant contributor to the symptoms associated with chronic Lyme.

INTERPRETATION:

RECOMMENDATIONS:

ENVIROTOXINS & POLLUTION

GENETIC DATA

GENE	GENO TYPE
CAT(2)	TC
NQO1	GG
IL6	GC
UGT2B15	AC
LOX	CC

INCREASED SUPPORT NEED

Partial List of Toxins:
Agricultural chemicals
Organotoxins
BPA's
Phthalates
Airborne pollutants
Cigarette smoke

The consequences of living in an industrialized world is the exposure to new toxins that are created by society. Many of these "EPI-toxins" can significantly alter gene expression if they are not adequately detoxified by our body defense systems. These toxins can cause chronic disruptions of metabolic and endocrine processes and can even lead to disruptions of genetic expressions in our children and subsequent generations.

It is impossible to avoid exposure to these chemicals so it is essential to make sure that defense system is optimized to deal with the exposure. The CDC reported on over 300 chemicals that it monitors as part of its biomonitoring system that began back in 1999 and the list grows every year.

INTERPRETATION:

RECOMMENDATIONS:

HISTAMINE SENSITIVITY

GENETIC DATA

GENE	GENO TYPE
AOC1(1)	TC
AOC1(2)	CC
AOC1(3)	GG
HNMT	AA
HNMT(2)	n/a
MAO-B(2)	TC
MTHFR 677T	GG
MTHFR 1298C	GG

INCREASED

GENETIC RISK

Histamine release is a natural part of our defense system. It is responsible for sneezing, itching, hives, rashes, stuffy nose, etc. These all sound unpleasant but they have a purpose. This system, like every system in the body, requires balance or homeostasis. This means that it is good to have the response but we also need to be able to control it and turn it off when it is no longer needed or unnecessary.

Histamines can also come from our environment. Many foods contain histamines and even our own gut bacteria can produce histamines. Histamines are a sort of neurotransmitter that the body uses to signal that it is under attack so histamines, not produced by our own defense system, are obviously not a benefit.

This balancing system that is designed to turn off histamines can vary in each individual and much of this can be predicted by genetics. In this section, we look at genetic variants that can predispose to histamine intolerance.

INTERPRETATION:

RECOMMENDATIONS:

MERCURY

GENETIC DATA

GENE	GENO TYPE
GSTM1	AA
GSTT1	TT
GSTP1(1)	AA
GSTP1(2)	CC
MT4	AG
GCLM	GG
GPx1	GG
GPx1(2)	CC
GPx1(3)	GG
SEPP1	TC
ABCC2(1)	AG
ABCC2(2)	TC
ABCC2(3)	GG
ATP7B	TC
BDNF	CC

OVERALL PROCESSING

LOW

INORGANIC MERCURY

HIGH

ORGANIC MERCURY

VERY LOW

Mercury's effect on health is a hot topic in the wellness community that has prompted substantial debate. It is important to truly understand four specific aspects in order to make educated decisions on reducing and mitigating the risks. The four areas to consider are:

- Absorption
- Distribution
- Metabolism
- Excretion

There are two main forms of mercury; inorganic and organic. Inorganic mercury exposure comes from inhaled environmental pollutants, food, dental amalgams, vaccinations, and even supplements. Yes, supplements, especially Ayurvedic herbs as they have been found to have significant heavy metal contamination.

Organic mercury is the form that we are principally exposed to through fish consumption.

These two different types of mercury are processed by common detoxification systems and there are pathways that are specific for the type of mercury. We assess some of the common genetic variants that contribute to these processes and evaluate genetic variants that contribute to the processing and elimination of both types.

INTERPRETATION:

MERCURY HEALTH IMPACT

GENETIC DATA

GENE	GENO TYPE
APOe(1)	TC
APOe(2)	TC
BDNF	CC
COMT	GG
CPOX4	TT
CPOX5	CC
PON1	TC
PGR	CC
TF	AA
MMP2	TC

OVERALL HEALTH IMPACT

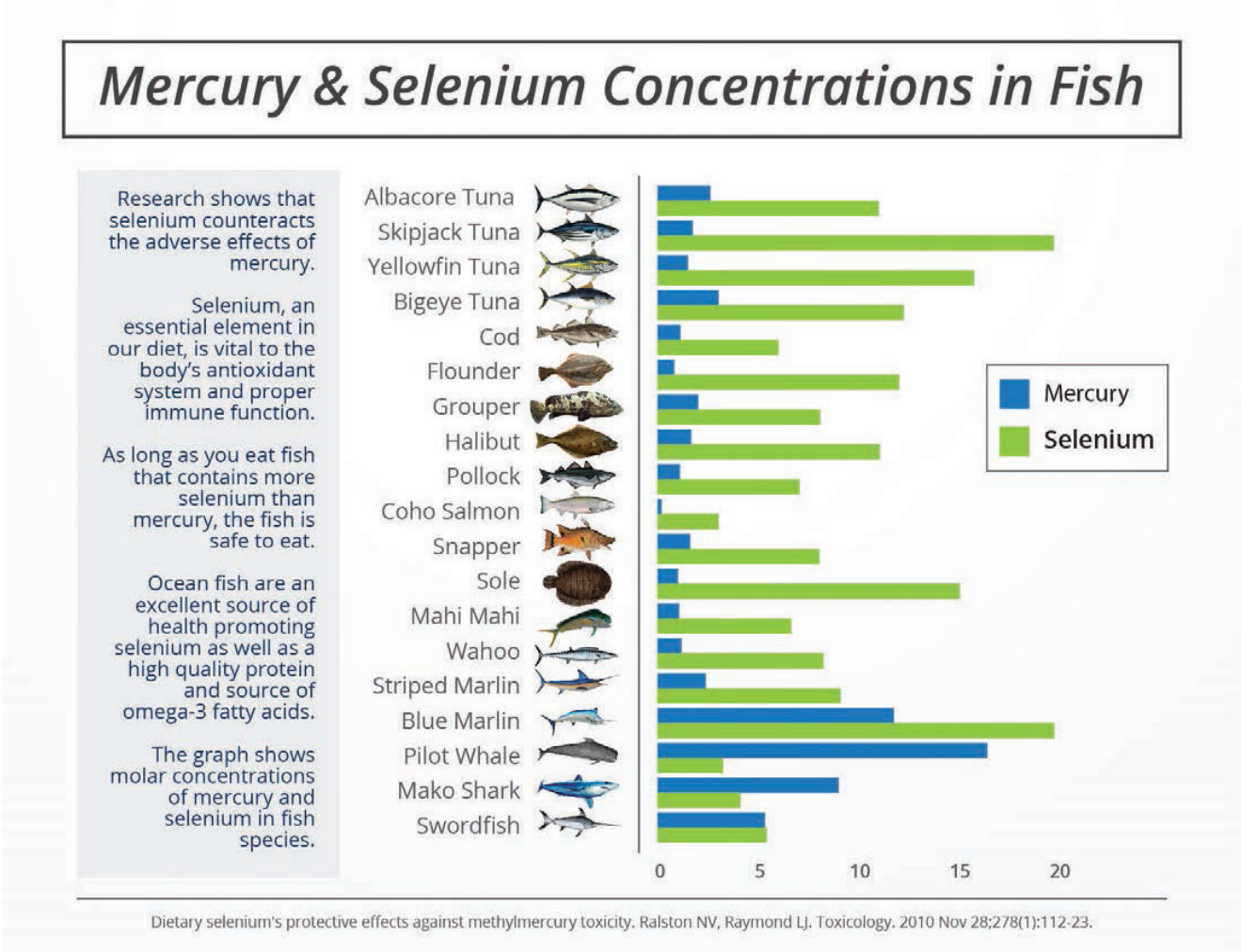
LOW

Although all forms of mercury have adverse effects on human health at high doses, the evidence that exposure to very low levels of exposure may potentially lead to significant consequences for humans is still open to interpretation. Mercury's health effects impact each individual differently due to variations in the distribution, metabolism, and elimination of this highly reactive heavy metal. Mercury can affect many body systems including the brain & nervous system, heart, and kidneys. There are impacts on reproductive and endocrine function. It can induce or exacerbate autoimmune disease and neurologic risks in the perinatal and early childhood period can lead to cognitive and behavioral changes. Certain gene variants can predispose an individual to significant impact from even low levels of mercury exposure. Studies have demonstrated that this may be related to our genetics and we assess gene variants to get a picture of how mercury ingestion affects overall health.

RECOMMENDATIONS:

MERCURY AND FISH

Fish consumption is one of the more robustly discussed topics on the internet as well as in the scientific community as it represents the greatest source of exposure to organic mercury, referred to as methyl-mercury. This form of mercury in fish is about 95% absorbed and has a half-life in the body of approximately 70-80 days. This information by itself may prompt people to avoid fish yet it is only part of the story. The truth is that fish is very healthy in the diet, It is full of very healthy omega-3 fats and most fish contain a high molar ratio of selenium to mercury. This molar ratio is much more important than the absolute mercury content because selenium binds mercury and essentially makes it inert (nonreactive). In fact, recent evidence is suggesting that one of the major contributors of symptoms relating to mercury may actually be due to the mercury depleting our own body's stores of selenium and the amino acid selenocysteine. Consuming fish with a higher level of selenium than mercury is safe and healthy. Listed below are the average molar ratios for many of the common fish.



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Athletic Performance Genomics

Client: Sample Client

About This Report

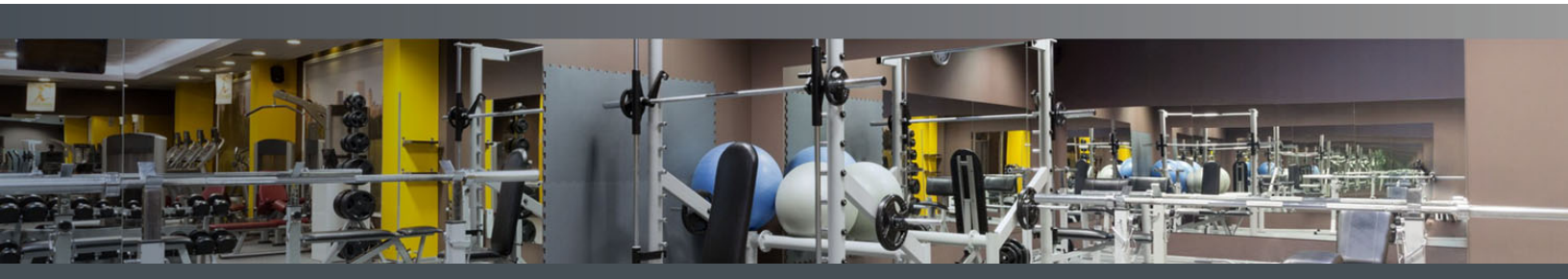
Strong evidence exists for the role that genetics play in athletic performance. Olympic and elite level athletes carry clear genetic markers that create a propensity for remarkable performance. It is important to point out however, that there are also many Olympic level and elite athletes that possess genetics that are not indicators of elite performance. This seemingly disparate occurrence can be attributed to several factors. First, athletic traits are generally expressed through a combination of genes as opposed to one specific variation in a single base pair out of 3 billion. Second, epigenetics provides the ability to alter the expressions of genes; we can upregulate or down-regulate expression through our environment and lifestyle.

The ACTN3 gene variant is one example; a certain variant of this gene will result reduced or no actinin-3 in muscle fibers. Elite sprinters mostly have the ACTN3 variant that codes for its presence in muscle. When it is missing, up-regulation is not possible, however, with appropriate training, even those missing the protein, can up-regulate production of a similar protein called actinin-2 through the gene ACTN2. Despite not having the gene for sprinting, there are still elite level sprinters and power athletes that have the variant without actinin-3.

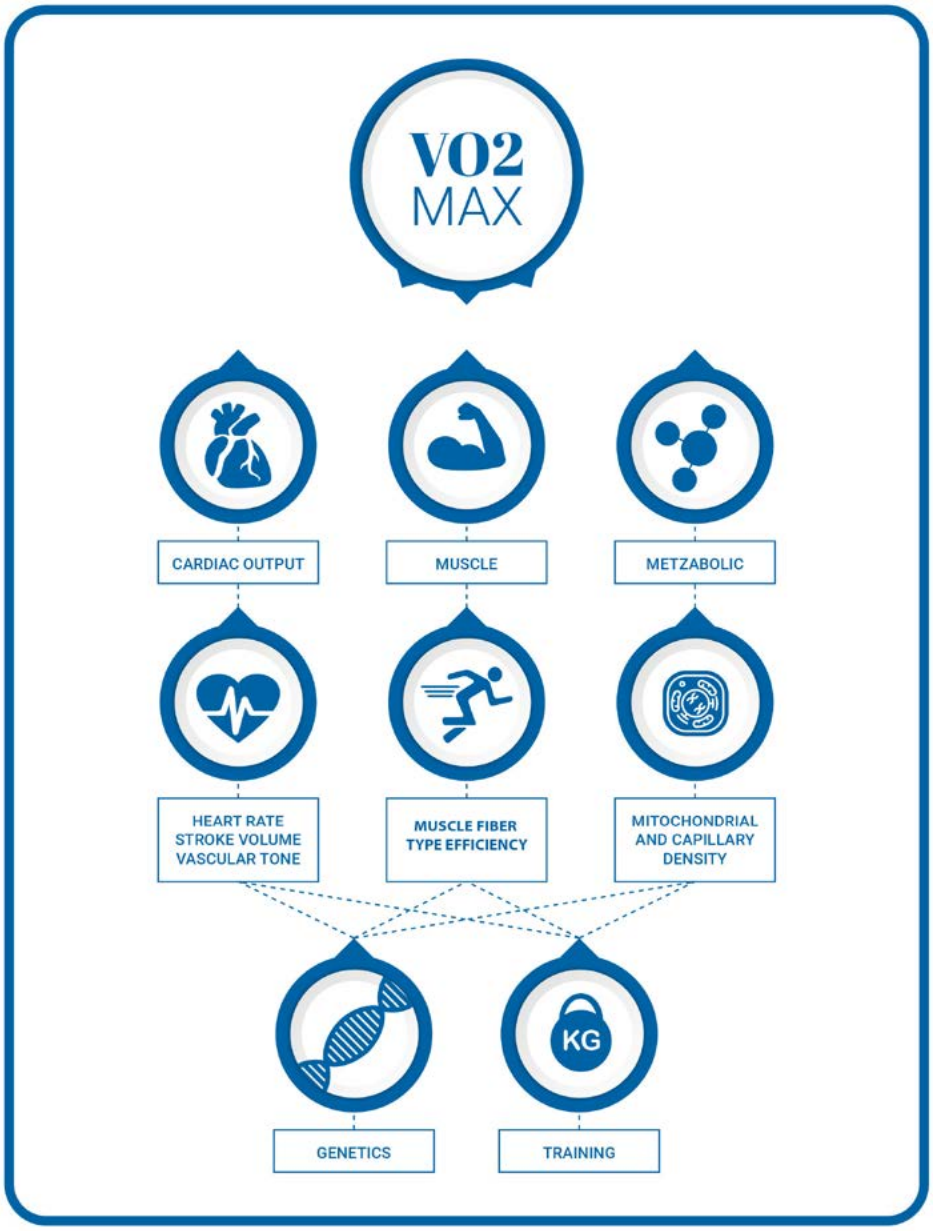
Finally, the alteration of specific genes also occurs when we take strategic action through the use of nutrigenomics (nutrition and supplementation).

Keep in mind that genetics is about propensities or probabilities, not absolutes. Having ideal genes for elite performance will not make you an elite athlete and not having optimal performance genetics does not make it out of reach.

This translation guide is not designed to provide direct to consumer guidance, but as a way to open a discussion with an Apeiron certified epigenetic human perfromance coach who understands the art and science of genetics, epigenetics, and the interactive component of using the personal genetic blueprint to guide success in alignment with your specific goals.



Understanding VO2 max



VO2 max = the maximum amount of oxygen a person can use during intense exercise.

Most genetic analysis is based on comparing athletes that have elite performance capabilities to non-elite athletes. These are mainly correlation studies and many of the gene variants that we consider are based on these correlations.

After looking at overall propensities for VO2 max, this report breaks down individual pieces of the system that contribute to VO2 max and the specific gene variants that contribute to the components. This helps to bio-individualize recommendations for improvement.

VO2 MAX OVERALL

GENETIC DATA

GENERAL

GENE	GENO TYPE
RGS18	GG
BTAF1	TC
TSHR	TT
GRIN3A	GG
KCNH8	TT
C9	GG
ZIC4	TT
CAMTA1	TT
BIRC7	..
NDN	CC
TTC6	TT
APOE (1)	TC
APOE (2)	TC
HFE(1)	CG
HFE(2)	GG
PPARA	CG
VEGFa(1)	CG
HBB	TT
CHRM2	AG
EPAS1	GG

VO2 max is a common measure of athletic performance and can be used to guide training programs for optimal outcomes. Elite athletes consider optimal VO2 max an essential area to optimize. The is a strong genetic component to VO2 max that can predict 25-50% of the observed variability. This variability falls into multiple physiologic categories that comprise VO2 max.

VO2 max is the highest rate of oxygen consumption that an individual can obtain during maximal exertion. VO2 max is calculated by the Fick equation (cardiac output x arterial O2 – cardiac output x mixed venous O2). So, VO2 max takes into account the following physiologic parameters:

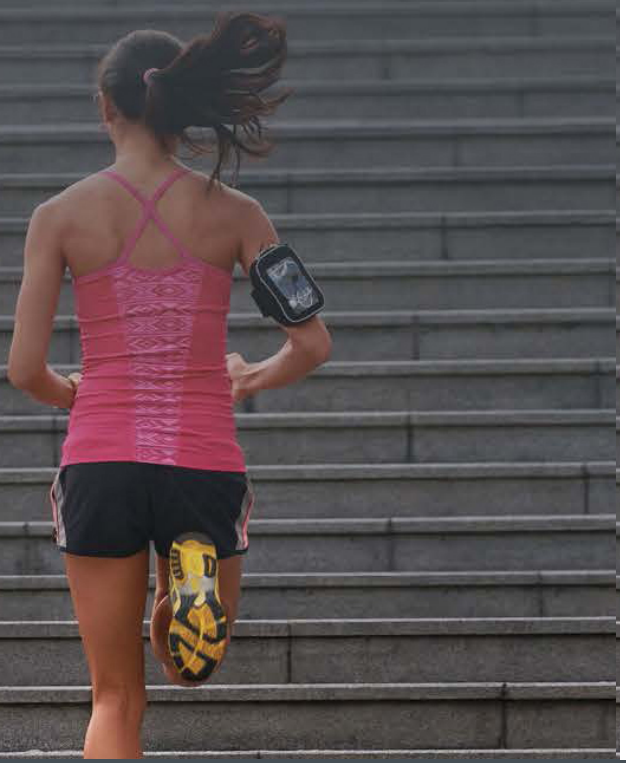
- Cardiac:
  - Stroke volume (contractility)
  - Heart rate
- Vascular tone
- Muscle fiber type
- Metabolic machinery of the cell

Overall VO2 Max Propensity

VO2 MAX CARDIOVASCULAR

CARDIOVASCULAR

GENE	GENO TYPE
ADRB1	..
ADRB2(1)	AG
ADRB2(2)	GC
NFIA-AS2	GG
EDN1	GG
DBX1	TT
HIF1A	AG
CREB1	AG
KIF5B	CG
NPY	TT
BDKRB2	TC



One of the three main components of VO2 max is cardiovascular function . In this section, genes involved in creating optimized cardiac output are analyzed. These include genes that are involved in inotropic effect (modifying force or speed of contracting muscles) and chronotropic effect (changes in heart rate or rhythm). Additional aspects include genes involved in blood flow to the heart, the response to sympathetic nervous system stimulation, blood capillary density, and utilization & transport of oxygen to the cardiac mu s culature.
By identifying gene variants and their function, it may be possible to address specific interventions to optimize the cardiac aspect s of VO2 max.

INTERPRETATION

RECOMMENDATIONS

VO2 MAX METABOLIC

METABOLIC

GENE	GENO TYPE
DEPTOR	AA
MIPEP	GG
ACSL1	GG
NRF1(1)	TC
NRF1(2)	AG
HIF1A	AG
PPARGC1A	TT
CKMM	TT
UCP3	GG
KIF5B	CG
AMPD1	GG
GABPB1	AA

MUSCLE

TTN	TC
DAAM1	TT
AMPD1	GG
AGTR2(2)	AA

Metabolic and muscular components of VO2 max are grouped together in this section because there is crossover in the genes involved with each.
VO2 max is t he body's ability to uptake oxygen, deliver it efficiently to the cells, transport it effectively into the muscle and then rely on the muscles metabolic machinery to use it in an optimized fashion.
Oxygen is a key component of the electron transport chain where macronutrients are converted into energy for contraction . Without ad e quate and efficient supply of oxygen, the conversion of nutrients into usable energy currency can be compromised.

Metabolic

Muscle

INTERPRETATION

RECOMMENDATIONS

MUSCLE FATIGABILITY

GENETIC DATA

GENE	GENO TYPE
NAT2	AA
HNF4A	AG
AMPD1	GG
COL5A1(1)	TC
IL15Ra(1)	TT
AGTR2	CC
AGTR2(2)	AA
TTN	TC
ACE	TC

In athletic performance, an important parameter to consider is muscle fatigue. Muscle fatigue is defined as the decline in a muscles ability to generate force. Genetics play an important role in guiding optimized performance and certain forms of training can be implemented to enhance gene expression.

- Muscle fiber types
- Metabolic function
- Oxygen delivery
- Lactate clearance

Epigenetics - Adaptive training

Genetics provide probabilities or predispositions that can guide interventions and the epigenetics can be modified by specific training protocols or supplementation.

Fatigability

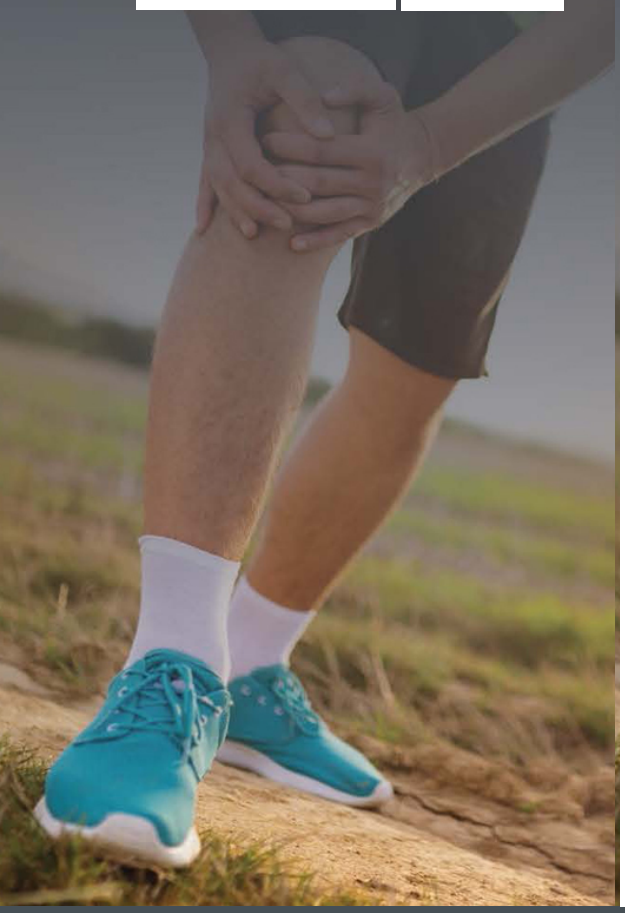
INTERPRETATION

RECOMMENDATIONS

RECOVERY

GENETIC DATA

GENE	GENO TYPE
CRP(1)	CC
TNF	GG
SOD2	GG
IL1B	AG
IL6	GC
NAT2	AA
CKMM	TT
COL5A1(1)	TC
CHRM2	AG



Routine intense exercise is not a natural process, in fact, fitness training only became part of human life in the past century. To early hominoids, exercise was part of survival. Hunting required brief bursts of intense activity followed by long periods of rest, agriculturalists generally had prolonged low intensity without the bouts of intense exertion.

How much rest do we require between intense workouts?

The answer is not simple as the re is significant variability within populations and genetics play a key role. The body requires a certain amount of time to repair the damage incurred by intense workouts and understanding your genetic propensities can guide strategic planning to achieve the highest impact from your routine.

Probable beneficial rest interval after intense exertion:

INTERPRETATION

RECOMMENDATIONS

STRENGTH/HYPERTROPHY

GENETIC DATA

GENE	GENO TYPE
ACTN3	TC
ACE del	GG
AGTR2	CC
AGTR2(2)	AA
SHBG	TC
MSTN	TT
RETN(1)	CG
IGFBP3	AA
IGF1	TT
CNTF	GG
DIO1	CC
IGF2(2)	CC
IGF2(3)	TT
IL6	GC
CCL2(1)	AA
CCL2(2)	TA
CCR2	TC
IL15Ra(1)	TT
IL15Ra(2)	AA
IL15Ra(3)	AA
ACVR1B	AG
RETN(2)	AG
IL6R	CC

Endomorph, mesomorph, or ectomorph; we are all aware that we possess certain genetic predispositions toward a specific body type. This genetic predisposition resides in the genetic variations that deal do with muscle strength and hypertrophy (muscle size).

It has been estimated across multiple genomic studies that >50% of muscle strength and muscle mass is attributable to heritable genetics.

We can investigate genetic variants that specifically code for hypertrophy and ones that code for strength development. There is no doubt that genetics will gift us with a specific proportion of muscle fiber types but through lifestyle approaches we have an ability to create different varying body type outcomes.

Strength

Hypertrophy

INTERPRETATION

RECOMMENDATIONS

POWER/SPRINT

GENETIC DATA

POWER VS ENDURANCE

GENE	GENO TYPE
AGTR2	CC
AGTR2(2)	AA
ACE del	GG
ACTN3	TC
DMD	AG
AGT	AG
VDR	CC
TTN	TC
ACVR1B	AG
NAT2	AA
NOS3(3)	TC

An individual's ability to produce short burst explosive power can be strongly influenced by their genetic code. Looking at the genetics for muscle type, energy production, metabolic capacity, and neuromuscular response, can provide insight into your potential. Response to training is another important marker.

Knowing the code will also provide guidance for the techniques to improve response. Genetics will provide the clues to the hardware but the epigenetics provides us with the ability to modify expression.

Power/Sprint

INTERPRETATION

RECOMMENDATIONS

ENDURANCE

GENETIC DATA

ENDURANCE

GENE	GENO TYPE
AGTR2	CC
AGTR2(2)	AA
ACE del	GG
ACTN3	TC
BDKRB2	TC
GNB3	CC
ADRB2(1)	AG
ADRB2(2)	GC
ADRB2(3)	CC
PPARGC1A	TT
PPARD(1)	TT
PPARD(2)	AA
ADRA2A	GG
EPHX1	TC
LTBP4	GG
SCGB1A1	AG
UCP2	GG
VEGFA(1)	CG

E ndurance genetics take into consideration muscle type, metabolic capacity, efficiency of use of specific nutrients for fuel, energy turn-over, and cardiac response to all contribute to optimal performance. Knowing which areas are strong and which could benefit from training is important in planning training routines

Some athletes will be balanced in both power and endurance but elite athletes are usually shifted to one end of the spectrum

Endurance

INTERPRETATION

RECOMMENDATIONS

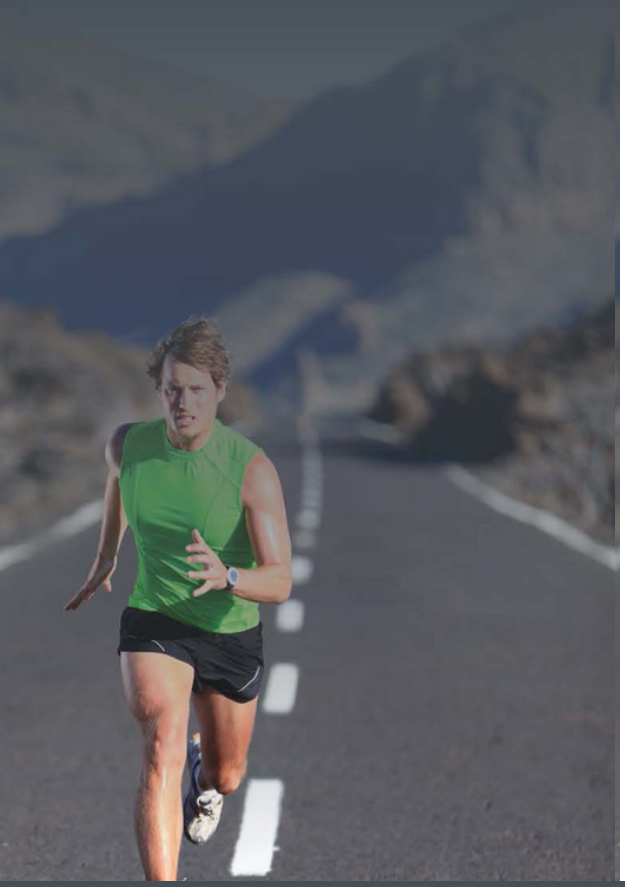
POWER VS. ENDURANCE

Genetic Propensity

Are you predisposed to better performance in sprint and power athletics or are you designed more for distance and endurance? Most people fall somewhere in the middle, referred to as a mixed athlete.

We look at wide ranging genetic factors that contribute to these various outcomes but possessing a specific predisposition does not relegate you to staying within that spectrum.

Knowing your genetics can guide your training to suggest a focus on enhancing your strengths or training less than optimal propensities.



INTERPRETATION

RECOMMENDATIONS

SOFT TISSUE

GENETIC DATA

GENE	GENO TYPE
COL1A1	CC
COL3A1	GG
COL12A1	AG
COL5A1 (1)	TC
COL5A1 (2)	AC
GDF5	AA
MMP3(1)	TC
MMP3(2)	..
FAM46A	TT
CILP	AA
COL11A1	GG
CRP(2)	GG
CRP(1)	CC
IL-6	GG
ADAM12	TC
ESRRB1	TT
FGFR1	TT
SASH1	CC
SAP30BP	AG
COL6A4P1	CC
IL1RN	GG
IL1A	AG
CCDC111	TC
FGF10	TT
HIF1A(2)	GG
KDR(1)	TA
KDR(2)	AA

Genetic predispositions toward connective tissue injuries is an important consideration whether you are an athlete or a weekend warrior. These genes look at specific tissue types and based on population studies can provide valuable information regarding the potential for injuries. Knowing your genetic predispositions can guide risk mitigation.

Tendon and ligament injury risk

Cartilage risk

Lumbar disc injury risk

Rotator cuff injury risk

INTERPRETATION

RECOMMENDATIONS

CENTER FOR HUMAN POTENTIAL

APEIRON

HORMONE REPORT

Client: Sample Client

THYROID

GENETIC DATA

GENE	GENO TYPE
PDE8B(1)	AG
PDE8B(2)	TC
PDE8B(3)	TT
PDE8B(4)	AG
THRA	CC
DIO1(1)	CC
DIO1(2)	AA
DIO2(1)	TC
DIO2(2)	TC
SLCO1B1	TC
SLC16A2	TC

HIGH

TSH PROPENSITY

AVERAGE

D1 ACTIVITY

LOW

D2 ACTIVITY

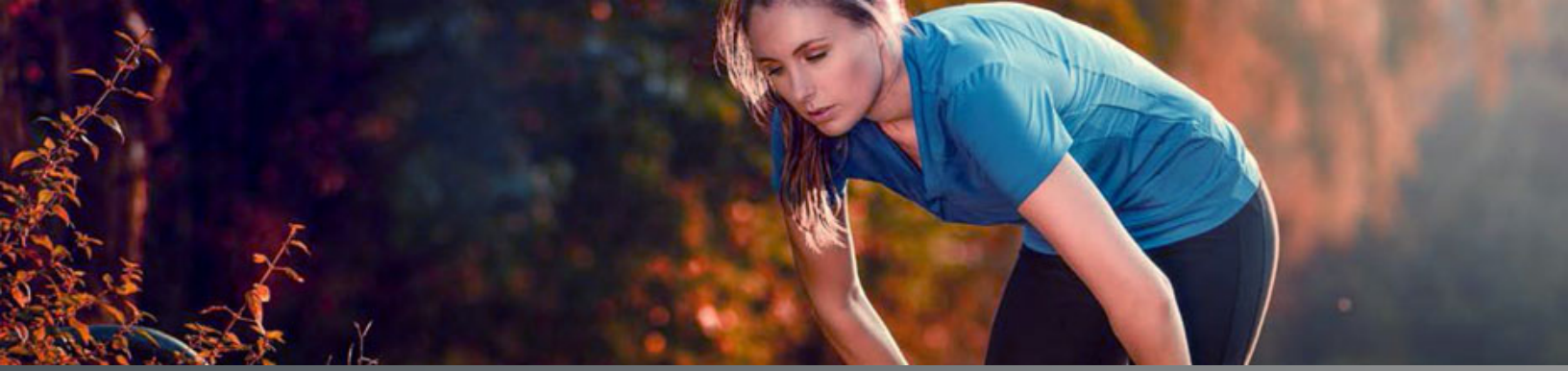
The thyroid gland is one of the most important endocrine organs in the body. It is responsible for production of hormones that control metabolism. The glands involvement in metabolism affects a range of body functions;

- Body weight
- Cognitive function
- Body temperature
- Menstrual cycles
- Muscle performance
- Cholesterol

The production and utilization if thyroid hormone is highly complex and optimizing outcomes requires a deep knowledge of the interactions of lifestyle, environment, genetics, and epigenetics. Understanding the genetic polymorphisms involved can lead to much more precise interventions that can lead to optimizing the function to achieve greater potential. Genetic propensities can be used to better understand proper function of the gland, ranging from receptor sensitivity, conversions to active forms, brain responses, and supplements to improve function.

INTERPRETATION

RECOMMENDATIONS



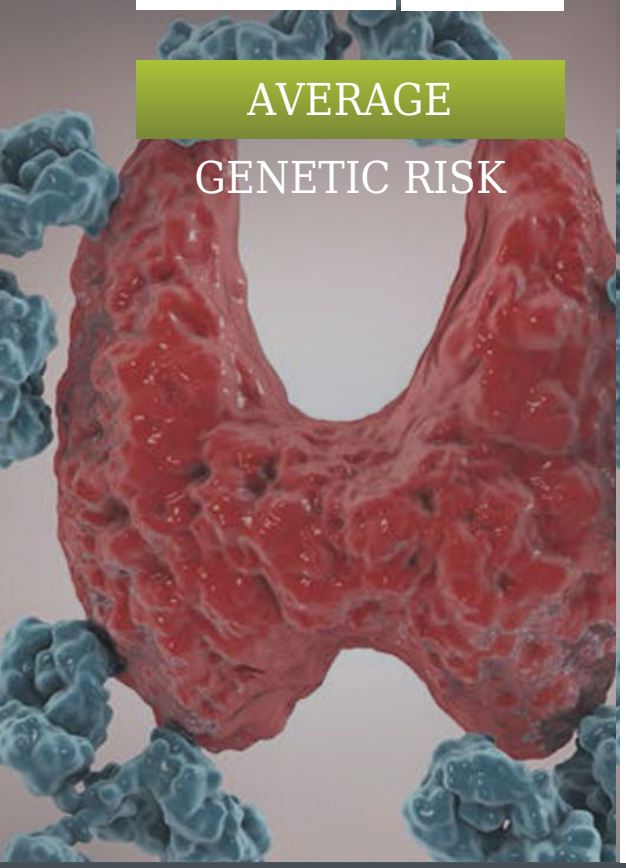
AUTOIMMUNE THYROID

GENETIC DATA

GENE	GENO TYPE
FOXE1(1)	AG
FOXE1(2)	AG
PTPN22	GG
DIO1(1)	CC
CTLA4(1)	AG
CTLA4(2)	AG
FCRL3	AA
IL23R	AC
TNFa	GG
IL6	GC

AVERAGE

GENETIC RISK



Autoimmune thyroid conditions are frequently over diagnosed. An underactive thyroid is not necessarily an autoimmune condition. Autoimmune hypothyroidism (or Hashimoto's thyroiditis) requires the presence of antibodies to certain cells in the thyroid gland and there is a genetic predisposition that people can carry. Autoimmune hyperthyroidism (or Graves' disease) is a condition where the autoimmune antibodies stimulate the receptor on the thyroid gland causing it to overproduce. The propensity for this condition is also passed along in our genes.

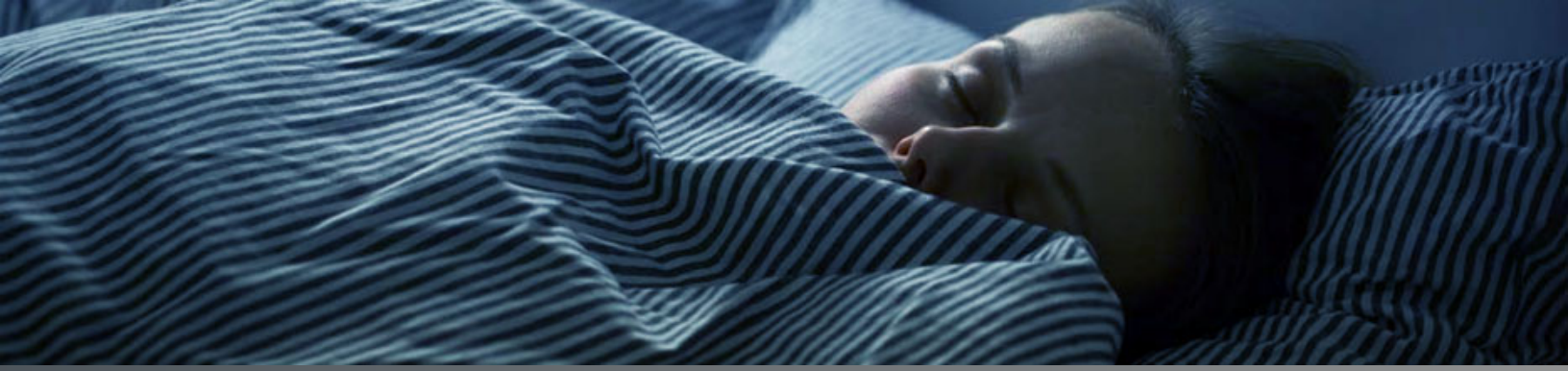
Symptoms of hyperthyroidism:

- Anxiety
- Insomnia
- Shaking hands
- Weight loss
- Sweating
- Hair loss

Symptoms of hypothyroidism:

- Weight gain
- Fatigue
- Dry skin
- Hair loss
- Cold hands/feet
- Constipation

RECOMMENDATIONS



MELATONIN

GENETIC DATA

GENE	GENO TYPE
MTNR1B(1)	TC
MTNR1B(2)	CG
MTNR1B(4)	CC
TPH2(1)	TG
AANAT	..
ADA	..
CYP1A2	AA

DECREASED

PRODUCTION

LOW

RESPONSE

RAPID

BREAKDOWN

Melatonin is a hormone that is secreted predominantly by the pineal gland in the brain and is also produced in smaller amounts in other organs. Production drops off dramatically as we age and this can have significant health impacts.

Functions of melatonin:

Sleep - melatonin is intimately involved with regulation of our circadian rhythm

Antioxidant - a powerful free radical scavengerit interacts with immune cells to help boost response to infectious organisms

Immune function - it interacts with immune cells to help boost response to infectious organisms

Anti-aging - plays a suspected role in longevity

Skin Pigmentation - present in melanocytes in the skin

Melatonin can easily be supplemented in conditions where production or response is diminished. Genetic variants can help to guide the need for supplementation by looking at variants that are involved in production, receptor numbers, receptor responses, and breakdown to inactive form.

INTERPRETATION

RECOMMENDATIONS

CORTISOL

GENETIC DATA

GENE	GENO TYPE
FKBP5(1)	CC
FKBP5(2)	AA
FKBP5(3)	GG
COMT	GG
HTR2C	GG
GSTP1	AA
NR3C1	CC
CRHR1	AA
CRHR2(1)	AC
CRHR2(2)	AG

AVERAGE

PRODUCTION PROPENSITY

AVERAGE

RESPONSE

Cortisol is frequently referred to as the “stress” hormone. The hormone itself is not the enemy and is essential to optimized human flourishing. Cortisol is essential to wake us up in the morning, it helps us to upregulate our physical response to acute stress while boosting performance in these situations, and very low cortisol levels impair decision making.

The brain is the ultimate controller for cortisol release, the adrenal gland is only the messenger and this is clearly demonstrated when we look at genetics. Studies are finding that most the genetic polymorphisms associated with cortisol levels are related to receptors that reside predominantly in the brain. These genetic variations are correlated with baseline and peak cortisol levels in the blood as well as rates of return to baseline levels. Many of these gene polymorphisms are highly vulnerable to life events that can epigenetically modify their expression. This can be good news in that it suggests that lifestyle factors may produce significant impacts on expressions despite carrying hard coded genetic predispositions.

INTERPRETATION

RECOMMENDATIONS

ESTROGEN

GENETIC DATA

GENE	GENO TYPE
ESR1(1)	GG
ESR1(2)	CC
CYP1A1	AA
CYP1B1	CG
CYP3A4	TT
COMT	GG
GSTP1	AA

AVERAGE

PRODUCTION PROPENSITY

AVERAGE

METABOLISM

Estrogen is a beneficial hormone in both males and females. It has many benefits beyond involvement in reproduction.

In males and females, estrogen is essential to bone health. Some studies have suggested that it is as important as Vitamin D in maintaining or stimulating bone growth. Decreased estrogen levels have been correlated with decreased memory and cognitive function.

In females, it is important for maintaining the health of the sex organs, sex drive, and sexual function. In males, lack of estrogen, even with normal testosterone, there can be issues with sex drive and erectile dysfunction so balance is essential. In genetic predispositions, we can assess receptor response to estrogen and look at the breakdown of healthy versus unhealthy detoxification. By identifying the genetic variants involved in the breakdown of estrogens, supplementation interventions that modify, or shift, the metabolism to more optimal and healthy outcomes.

INTERPRETATION

RECOMMENDATIONS

TESTOSTERONE

GENETIC DATA

GENE	GENO TYPE
JMJD1C	TT
FAM9B	TT
SHBG(1)	CC
SHBG(4)	TC
SHBG(3)	AG
PLCH2	TT
REEP3	TT
LHCGR	CC
APOe(1)	TC
APOe(2)	TC
CYP19A1(1)	CC
CYP19A1(2)	GG
CYP17A1	AA
SRD5A1	AA
SRD5A2	GG
HDAC4	AC
HDAC9	GG
TARDBP	AA
FOXA2	TT
MAOA	TT
MAOB	TC

AVERAGE

SHBG LEVELS

DECREASED

LH LEVELS

Testosterone is traditionally classified as the primary sex hormone; the reality is that it is the primary sexhormone in both males and females. It is a hormone of vitality and maintaining healthy balanced levels is essential to optimized wellbeing.

Potential benefits:

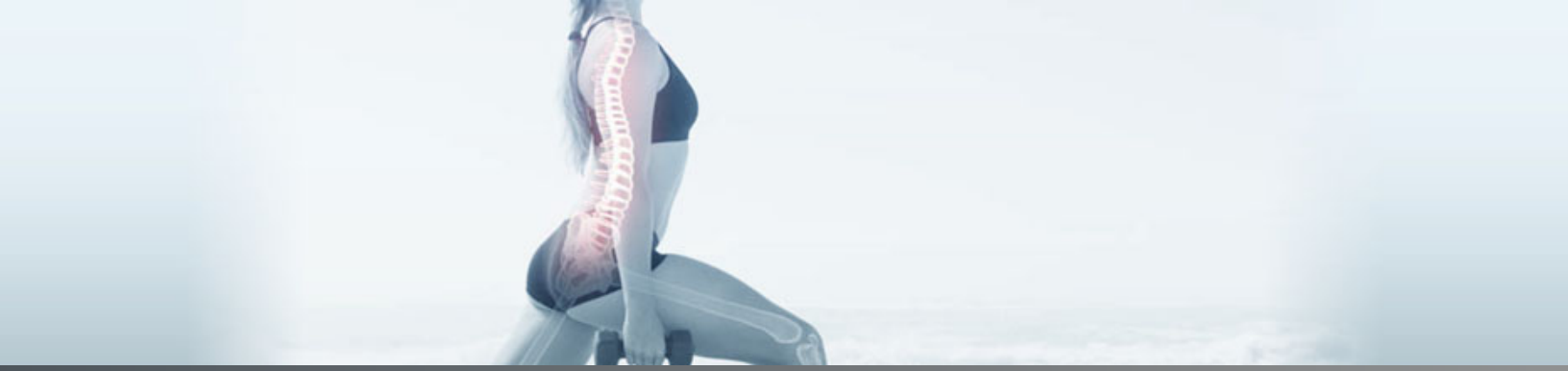
- Improved wellbeing
- Improved confidence and drive
- Improved sex drive
- Improved bone density
- Improved strength & muscle
- Decreased cognitive decline
- Decreased body fat
- Improved mood

To fully understand testosterone availability and benefits in the body, it is important to look at several factors that contribute to the outcome. These include; the amount of binding from SHBG (sex hormone binding globulin), response to LH (luteinizing hormone), and conversion to DHT (dihydrotestosterone), and conversion to estrogen to name a few.

AVERAGE	INCREASED
DHT LEVELS	ESTROGEN CONVERSION

INTERPRETATION

RECOMMENDATIONS



BONE DENSITY

GENETIC DATA

GENE	GENO TYPE
CYP19A1(1)	AG
CYP19A1(3)	n/a
CYP19A1(4)	CG
CYP19A1(5)	TC
LRP5(2)	TC
LRP5(3)	CC
FAM3C	TT
ARHGEF3	AG
IL1B	AG
IL6	GC
TNFa	GG
VDR taq	AA
VDR fok	AA
VDR bsm	CC
APOe(1)	TC
APOe(2)	TC

TYPICAL

GENETIC RISK

TYPICAL

VITAMIN K NEED

Osteoporosis and osteopenia and terms used to describe degrees of low bone density. This process is usually silent and is frequently only discovered after a major bone fracture. Therefore, regular screening and optimization are so important. Consider the statistics;

- > 50% of 50-59yo females have low bone density
- > 30% of 50-59yo males

Decreased bone density is becoming more common in the younger population where screening is showing low levels in males and females in their 20's and late teens. Lifestyle, genetics, and hormones are major contributors.

- Smoking
- Medications: thyroid, reflux PPI's, and prednisone
- Lack of weight bearing
- Low vitamin D levels

INTERPRETATION

RECOMMENDATIONS