APEIRON
CENTER FOR HUMAN POTENTIAL

Hormone Report

Client Name:

Thyroid

Genetic Data

General

Gene	Geno Type
TSHR(1)	GG
TSHR(4)	AA
PDE8B(1)	TA
PDE8B(2)	GA
PDE8B(3)	GG
PDE8B(4)	TT
CAPZB	AT
THRA	AG
DIO1(1)	AA
DIO1(2)	TT
DIO2(1)	AG
DIO2(2)	TT
SLCO1B1	GG

MODERATE

TSH RECEPTOR SENSITIVITY

MODERATE

D1 ACTIVITY

MODERATE

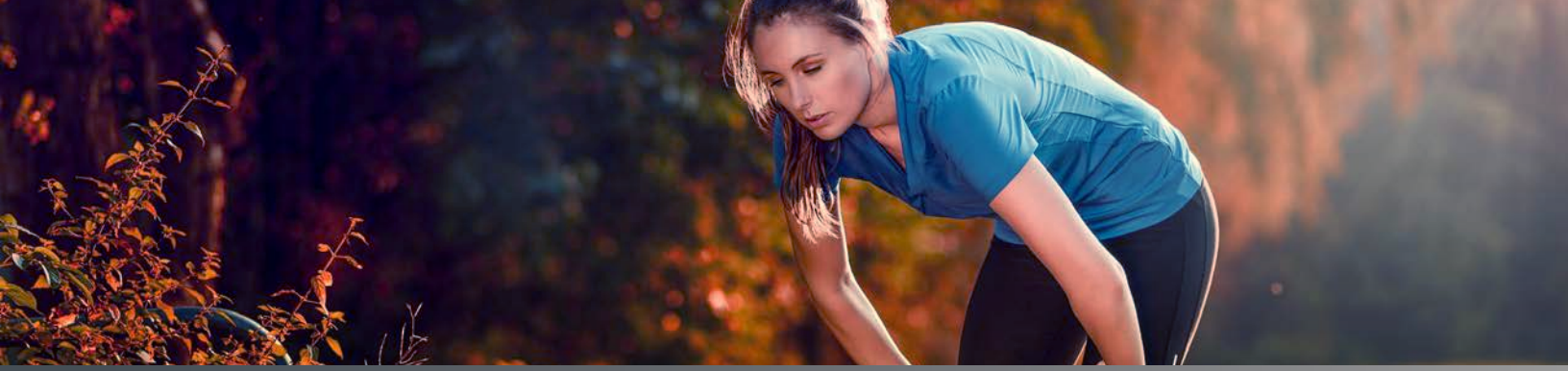
D2 ACTIVITY

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
- Body temperature
- Muscle performance
- Cognitive function
- Menstrual cycles
- Heart rate
- 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.

Interpretive Notes



Autoimmune Thyroid

Cardiovascular

Gene	Geno Type
TSHR(2)	GG
TSHR(3)	AA
FOXE1(1)	TA
FOXE1(2)	GA
PTPN22	GG
DIO1(1)	TT
CTLA4(1)	AT
CTLA4(2)	AG
FCRL3	AA
IL23R	TT
TNFa	AG
IL6	AT

LOW

PROPENSITY FOR HYPOTHYROIDISM

AVERAGE

PROPENSITY FOR HYPERTHYROIDISM

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

Interpretive Notes

Melatonin

Metabolic

Gene	Geno Type
MTNR1B(1)	GG
MTNR1B(2)	AA
MTNR1B(3)	TA
MTNR1B(4)	GA
TPH2(1)	GG
AANAT	TT
ADA	AT
CYP1A2	AG

AVERAGE

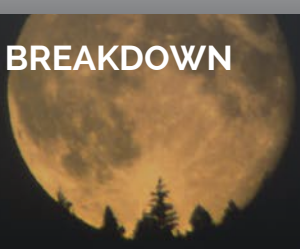
PRODUCTION

AVERAGE

RESPONSE

HIGH

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 were 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. Interestingly, melatonin and caffeine are broken down by the same enzyme.

Interpretive Notes

Cortisol

Genetic Data

Gene	Geno Type
FKBP5(1)	GG
FKBP5(2)	AA
FKBP5(3)	TA
COMT	AG
GSTP1	AT
NR3C1	AA
CRHR1	GG
CHRH2(1)	AA
CHRH2(2)	TA

HIGH

PRODUCTION PROPENSITY

AVERAGE

RECEPTOR SENSITIVITY

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.

Interpretive Notes

Estrogen

Genetic Data

Gene	Geno Type
ESR1(1)	GG
ESR1(2)	TA
ESR2	GA
CYP1A1	GG
CYP1B1	TT
CYP3A4	AT
COMT	AG
GSTP1	TA

AVERAGE

RECEPTOR RESPONSE

CAUTION

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.

Interpretive Notes

Testosterone

Genetic Data

Gene	Geno Type
SCARB1	GG
FAM9B	AA
SHBG(1)	TA
SHBG(2)	GA
SHBG(3)	GG
PLCH2	TT
REEP3	AT
LHCGR	GG
APOe(1)	AA
APOe(2)	TA
CYP19A1(1)	GA
CYP19A1(2)	GG
SRD5A1	TT
HSD3B1	AT
MAOA	TT
MAOB	AG

HIGH

SHBG LEVELS

AVERAGE

LH RESPONSE

Testosterone is traditionally classified as the primary sex hormone; the reality is that it is the primary sex hormone 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.

HIGH

DHT LEVELS

AVERAGE

ESTROGEN CONVERSION

Interpretive Notes

Osteoporosis

Genetic Data

Power vs Endurance

Gene	Geno Type
CYP19A1(1)	GG
CYP19A1(2)	AA
CYP19A1(3)	TA
CYP19A1(4)	GG
CYP19A1(5)	AA
LRP5(1)	TA
LRP5(2)	GA
LRP5(3)	GG
ARHGEF3	TT
IL1B	AT
IL6	GG
TNFa	AA
VDR taq	TA
VDR fok	GA
VDR bsm	GG
APOe(1)	TT
APOe(2)	AT

AVERAGE

GENETIC PROPENSITY

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 here.
Lifestyle risks include;

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

INCREASED VITAMIN K NEED

Interpretive Notes