



# DETOX GENOMICS

CLIENT NAME:



# PHASE 1 DETOXIFICATION

## GENETIC DATA

GENE	GENO TYPE
CYP1A1	GG
CYP1B1(1)	AA
CYP1B1(2)	TA
CYP2A6	GA
CYP2A6(2)	GG
CYP2C9	TT
CYP2C19	AT
CYP2D6	AG
CYP2D6(2)	AA
CYP2D6(3)	TT
CYP2E1(1)	AG
CYP2E1(2)	TT
CYP3A4	GG

ESTROGENS

MEDICATIONS

ANESTHETICS

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.

ACETAMINOPHEN

NSAIDS

INTERPRETIVE NOTES

# METHYLATION

## GENETIC DATA

GENE	GENO TYPE
COMT	GG
MTHFR 677T	AA
MTHFR 1298C	TA
DHFR	GA
SLC19A1	GG
CBS	TT

### OVERMETHYLATION

### 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.

### INTERPRETIVE NOTES



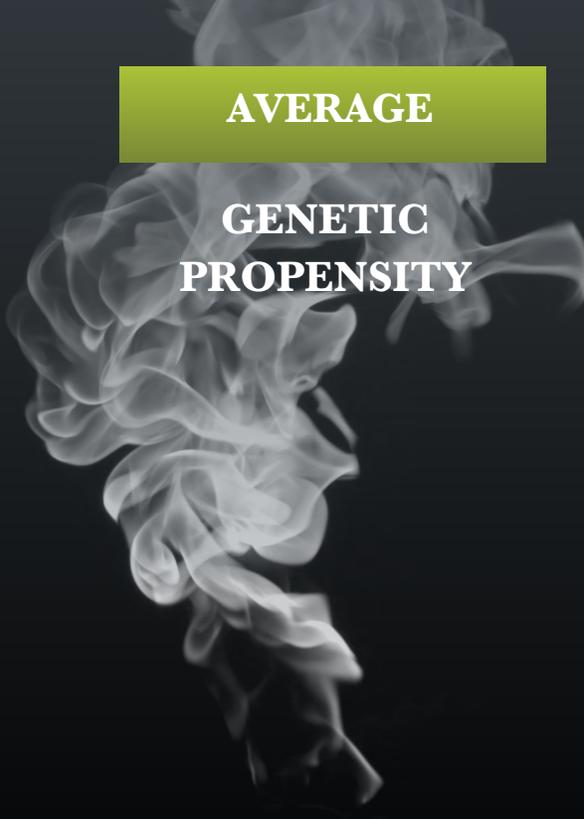
# ACETYLATION

## GENETIC DATA

GENE	GENO TYPE
NAT1(1)	GG
NAT1(2)	AA
NAT2(1)	TA
NAT2(2)	GA
NAT2(3)	GG

**AVERAGE**

**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 non-prescription 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.

**INTERPRETIVE NOTES**

# GLUTATHIONE SYSTEM

## GENETIC DATA

GENE	GENO TYPE
GPx1(1)	GG
GPx1(2)	AA
GPx1(3)	TA
GSTP1(1)	AG
GSTP1(2)	AT
GSTT1	AA
GSTM1(1)	GG
GSTM1(2)	GG
GCLC	AA
CAT	TA
CAT(2)	AG
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,

- Detoxifies
  - Heavy metals
  - Drugs
  - Environmental toxins
  - Pollutants
- Boosts immune function
- Enhances mitochondrial function
- Repairs our DNA

There are few available isolated forms of glutathione that can be absorbed and so it is important to look at providing the right precursors for the body to be able to optimize production.

## INTERPRETIVE NOTES



# MITOCHONDRIA

## GENETIC DATA

GENE	GENO TYPE
SOD2	GG
UCP2	TA
UCP4	GA
SIRT1	GG
SIRT5	TT
COX6B1	AT
ATP5C1(1)	AG
ATP5C1(2)	TA
NDUFS2	GG
NRF2(1)	TT
NRF2(2)	AT
NQO1	TT

**SUPPORT NEEDED**

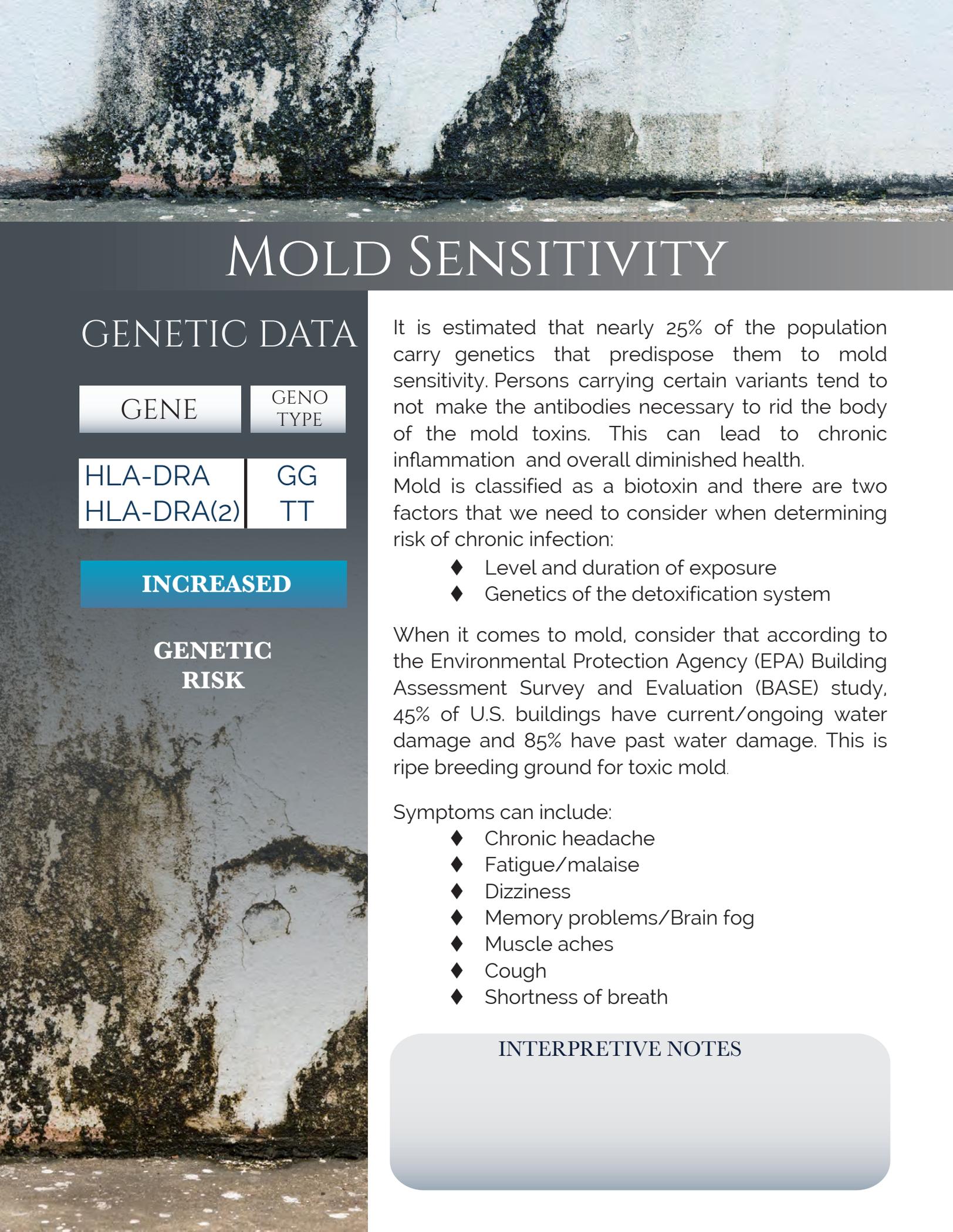
The mitochondria are classically viewed as the batteries of our cells. They produce the energy required for cell function, typically in the form of ATP. Mitochondria also have their own set of DNA and mitochondrial DNA is only passed to subsequent generations from the mother.

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.

### INTERPRETIVE NOTES

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# MOLD SENSITIVITY

## GENETIC DATA

GENE	GENO TYPE
HLA-DRA	GG
HLA-DRA(2)	TT

**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. This 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

**INTERPRETIVE NOTES**



# LYME DISEASE

## GENETIC DATA

GENE	GENO TYPE
TLR1	GG
ACSL1	AA
GAD1(1)	TA
GAD1(2)	GG
GAD1(3)	AG
PON1	TA
GSTP1	GG
SOD2(2)	AG

**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 frequently used but only confirm past infection.

The diagnosis of chronic Lyme disease is based primarily on symptoms and lack of other diagnose to explain them. 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 associated with this infection. 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. Identifying specific genetic variants can help guide interventions that provide better outcomes.



# ENVIROTOXINS & POLLUTION

## GENETIC DATA

GENE	GENO TYPE
CAT(2)	GG
NQO1	AA
IL6	TA
PTGS2	GA
UGT2B15	GG
LOX	TT

**TYPICAL**

**SUPPORT  
NEED**

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. It is a much bigger problem than most people realize. 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.

A partial list of these toxins:

- ⬢ Agricultural chemicals
- ⬢ Organotoxins
- ⬢ BPA's
- ⬢ Phthalates
- ⬢ Airborne pollutants
- ⬢ Cigarette smoke

**INTERPRETIVE NOTES**



# HISTAMINE SENSITIVITY

## GENETIC DATA

GENE	GENO TYPE
AOC1(1)	GG
AOC1(2)	AA
AOC1(3)	TA
HNMT	GA
HNMT(2)	GG
MAO-B(2)	GG
MTHFR 677T	AA
MTHFR 1298C	TA

**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.

**INTERPRETIVE NOTES**

