Nutrition Genotype Report

Client Name: Frank Zappa

REPORT SUMMARY

PHENOTYPE	RESULT
HUNGER	
SATIETY	
INSULIN RESISTANCE	
GLUTEN RISK	
DAIRY SENSITIVITY	
GRAIN SENSITIVITY	
SWEET PERCEPTION	
SNACKING DRIVE	
SWEET ADDICTION	

PHENOTYPE	RESULT
CARBOHYDRATES	
FIBER	
TOTAL FATS	
SATURATED FATS	
PUFA	
OMEGA-3	
MUFA	
PROTEIN	
METABOLISM	

HUNGER & SATIETY

GENETIC DATA

GENE	GENO TYPE
ADIPOQ (1)	AG
ADIPOQ (2)	GG
FTO	TT
MC4R	CT
ANKK1	GG
COMT	GG
DRD2	AA
NMB	GG
FTO	TT
HTR2C	CC
GHSR(1)	CT
GHSR(2)	GG
POMC	GG

LOW

HUNGER

TYPICAL

SATIETY

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.

Identifying which of these components can potentially be contributing to a non-desirable outcome can provide the guidance for the individual to create an optimal outcome.

INTERPRETATIVE NOTES:

Good genetics for appetite signaling and processing. You should interpret hunger and satiety signals accurately. Low risk for obesity.

INSULIN RESISTANCE

GENETIC DATA

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

MODERATE

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) | 44%

INTERPRETATIVE NOTES:

Overall low risk of genetics playing into insulin resistance so a healthy low simple carbohydrate diet should translate into healthy fasting blood sugar levels.

DAIRY

GENETIC DATA

GENE	GENO TYPE
MCM6(1)	GG
MCM6(2)	CC
APOA2	AA

INCREASED

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.

GLUTEN

GENETIC DATA

GENE	GENO TYPE
HLA-DQA1	CC
HLA-DQA2	GG
HLA-DRA	TT
HLA-DQB1	CT

MODERATE

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.

INTERPRETATIVE NOTES:

High probability of lactose intolerance. Genetic risk for developing celiac disease, be sure to maintain healthy gut and minimize gluten.

GRAIN SENSITIVITY

GENETIC DATA

GENE	GENO TYPE
GAD1(1)	AG
GAD1(2)	CT
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

INTERPRETATIVE NOTES:

Increased risk of brain fog and busy brain with glutamic acid intake. This usually manifests as difficulty shutting off the brain at night with dinners high in glutamic acid foods.

SWEETS & SNACKING

GENETIC DATA

GENE	GENO TYPE
FTO(1)	TT
LEPR	AA
MC4R	CT
FTO(3)	TT
FGF21	GG
SLCA2	GG
SLC2A2	CC
TAS1R2	GT
TAS1R3	CC
TAS2R38	AG

TYPICAL

SWEET PERCEPTION

LOW

SNACKING DRIVE

TYPICAL

ADDICTION RISK

Many people perceive that snacking behaviors and the inability to stop eating sweets once we start, 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.

INTERPRETATIVE NOTES:

Typical perception of sweet taste in foods. Unlikely to be a snacker.

CARBOHYDRATES

GENETIC DATA

GENE	GENO TYPE
KCDT10	CG
MMAB	CG
PLIN1	CC
UCP1	TT
TCF7L2(1)	CT
TCF7L2(2)	GT
TCF7L2(3)	CT
CEBPA	GG
ABCG4	AA
VLDLR	GG
IGF1R	AG
LPIN(2)	TT

LOW

OPTIMAL INTAKE

MODERATE

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.

Your genes suggest a propensity toward abdominal fat gain with higher complex carbohydrate intake.

INTERPRETATIVE NOTES:

Your genetics indicate a probable best health response with a slightly lower intake of complex carbohydrates. Best to keep total grams 200 or less. This is for starches and vegetables. Simple carbs should always be low.

TOTAL FATS

GENETIC DATA

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

MODERATE

OPTIMAL INTAKE

The primary fats of the human diet are:

- 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.

Subsequent sections will break down the types of fat that your physiology will tend to respond best to based on your genetic variations and current scientific studies in genome wide association.

INTERPRETATIVE NOTES:

Genetic privilege when it comes to fats. Still keep saturated fats less than 10% of total calories and take lots of monounsaturated fat & Omega-3 fats.

SATURATED FATS

GENETIC DATA

GENE	GENO TYPE
APOE (1)	TT
APOE (2)	CC
APOE (3)	AA
PPARG	CC
APOA2	AA
APOB	GG

MODERATE

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.

What we can learn from the new world of precision wellness is that there is not a single answer that can be applied across the board to the entire human race. 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.

HIGHLIGHTS

Your genes suggest that you may do fine with saturated fats but there are still health benefits from staying under 10% of total calories

These are only guidelines, please work with your coach to identify optimal intake levels.

POLYUNSATURATED FATS

GENETIC DATA

GENE	GENO TYPE
APOA5	AA
BDNF	CC
TNF	GG
FADS1	GT
ELOVL2	AA
PTGS2	AG
COX-2	n/a
IL-1B	GG

MODERATE

PUFA 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.

A significant amount of research has been focused on these 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.

HIGHLIGHTS

Your genomics suggest moderate intake of omega-3, focus on getting an omega-6 to omega-3 ratio below 4:1

Your genomics predict a higher probability of weight loss when keeping PUFA intake to 6-10% of total calories

MONOUNSATURATED FATS

GENETIC DATA

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

HIGH

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. There are few sources of MUFA that are pure so it is important to understand what other fatty acids are contained in the food, especially the omega-6 PUFA content, The main sources of MUFA in our diets include; oils, nuts, meats, salmon, and avocado.

MUFA should be a significant component of each persons diet and your coach can provide you with ways to optimize your intake.

DIETARY SOURCES

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

PROTEIN

GENETIC DATA

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

LOW

OPTIMAL INTAKE

Always pay attention to 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 to name a few. There are 20 amino acids that the body uses to create millions of different proteins and of those, nine 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. You coach can provide that guidance.

INTERPRETATIVE NOTES:

Overall healthy outcomes should do better with keeping protein intake to around 15-20% of total calories.

PLANT STEROLS

GENETIC DATA

GENE	GENO TYPE
ABCG8 (1)	TT
ABCG8 (2)	CC
ABCG8 (3)	TT
CETP	AA

LOW

PLANT STEROL RISK

LOW

PLANT STEROL BENEFIT

One of the several benefits from consumption of vegetables is related to the sterol content of these foods.

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. This is not a good trait necessarily. 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 potential with a heavily plant based diet. Supplementation of plant sterols will lead to higher levels and these products can come in a variety of forms; sterols, stanols, phytosterols, beta-sitosterol, campesterol and stigmasterol.

INTERPRETATIVE NOTES:

No genetic risk identified for problems with plant sterol intake.

METABOLISM

GENETIC DATA

GENE	GENO TYPE
GCKR	CT
LEPR	CG
PPARGC1A	CC
MC4R	CT
UCP2	CC
FTO(4)	TT
UCP2(2)	GG

TYPICAL

ESTIMATED RMR

When we analyze metabolism, we first have to clarify exactly what we are determining. 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.

The results of this report are best used as a guide for the coach to assess specific genetic variations that may have the best response to specific lifestyle or nutrigenomic interventions.

INTERPRETATIVE NOTES:

Typical to slightly above average basal metabolic rate. Strong on PPARGC1A which indicates good VO2 max response to training and benefit from cold thermogenesis.

MACRONUTRIENT WORKSHEET

CLIENT DEMOGRAPHIC:

Height: 70 inches

Weight: 177 lbs

Age: 44

Gender: male

MACRONUTRIENT DAILY GOALS:

Carbohydrates	210	grams/day
Protein	120	grams/day
Fats	105	grams/day

BASAL METABOLIC RATE:

(estimated)

1758.51 Calories/Day

Probability Based on Genetic Data

CARBS: 35%

FATS: 35%

PROTEIN: 30%

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

Recommended Calorie Mix

CARBS: 35%

FATS: 45%

PROTEIN: 20%

TOTAL DAILY CALORIE GOAL: 2100 CALORIES/DAY

RECOMMENDATIONS