COMPLETE HEALTH & WELLNESS GENETIC ASSESSMENT

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Catherine Kruger
Sample ID: 117765937398
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Anantlife is pleased to provide you with your complete health and wellness assessment based on your DNA. Our laboratory has utilized cutting edge procedures for genetic testing to carry out comprehensive analysis of your DNA which we isolated from your saliva sample. We have looked at your genetic profile to identify how your genes can influence your health and lifestyle. The assessment is based on the most current evidence-based scientific research published in peer reviewed journals as well as extensive bioinformatic analysis.

Following a review of your genetic profile, your healthcare practitioner can come up with a personalized plan based on your DNA to help maintain/improve your health.

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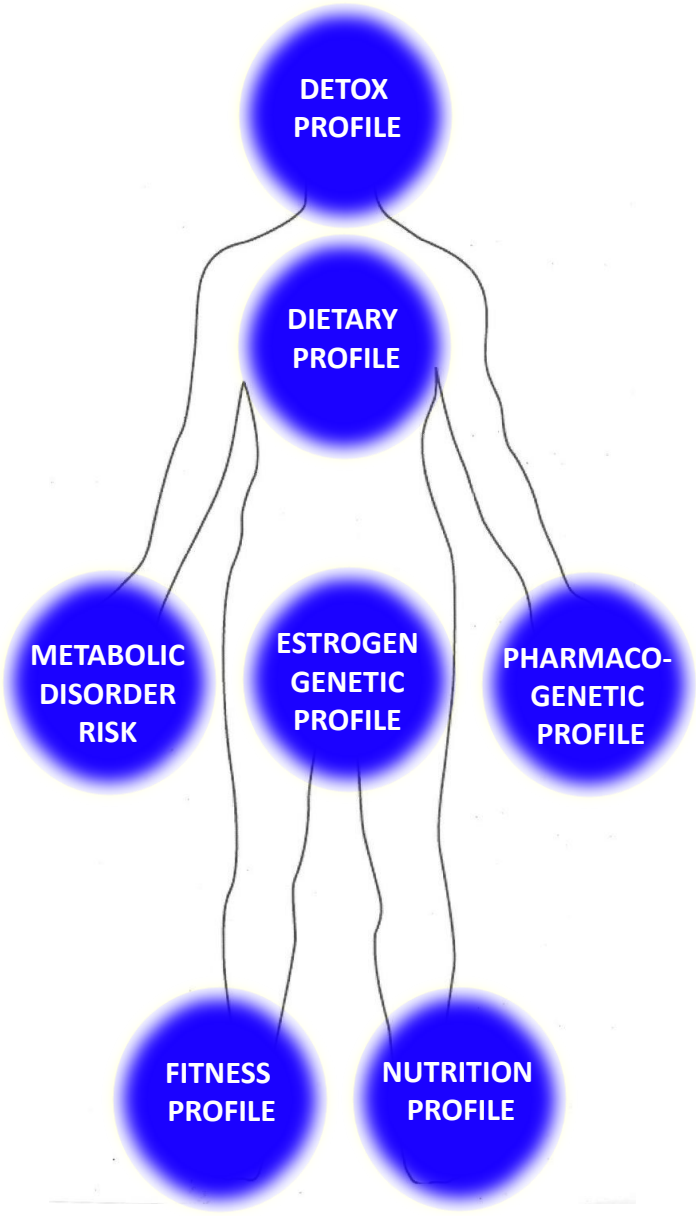
HOW DOES THE TEST WORK?

The completion of the human genome initiative has provided life science with a blueprint including goals of basic research and opportunities to translate research to improvements in human health. The human genome consists of about 25,000 genes and virtually all can exist in different forms. The variations in our genes make us unique from one another. Genetic variation plays a role in determining our health, metabolism, detoxification, nutritional needs, exercise/fitness physiology, dietary differences and predisposition to diseases. Understanding your genetic profile and its implications will provide you and your health practitioner with the tools needed to make the best choices to improve or sustain your health.



How does your genetic makeup impact your Nutritional Needs?

Vitamins and minerals are required as essential co-reactants or substrates in many pathways; deficiencies have the potential to cause metabolic failures. However, everyone has different needs when it comes to vitamins and minerals which is largely due to differences in DNA. For instance an individual may have a higher need of a particular vitamin because their genes may be not as efficient at metabolizing it. Thus these individuals may require a higher intake of the particular vitamin to ensure optimal levels. We have carried out an extensive assessment of your DNA to identify your predisposition to developing deficiencies to 14 essential vitamins and minerals. If you are identified to be at a risk, then increasing intake of those nutrients may help to prevent development of nutritional deficiency.

We have carried out extensive analysis of the following genes to identify your genetic predisposition to developing deficiencies for various vitamins and minerals:
BMCO1, NBPF3, FUT2, PEMT, GSTT1, CYP2R1, GC, F5, MTHFR, SLC17A1, HFE, TMPRSS6, TFR2, TF, MUC1, SHROOM3, TRPM6, DCDC5, ATP2B1, DMGDH, CA1, PPCDC, LINC01420

GENETIC PROFILE - NUTRIENTS

Biological function of Vitamins and Minerals

Vitamins

Vit A — Vision, neurological function, skin, immune system & reproductive function.

Vit B6 — Brain development & function, Neurotransmitter synthesis.

Vit B12 — Proper red blood cell formation, neurological function, and DNA synthesis.

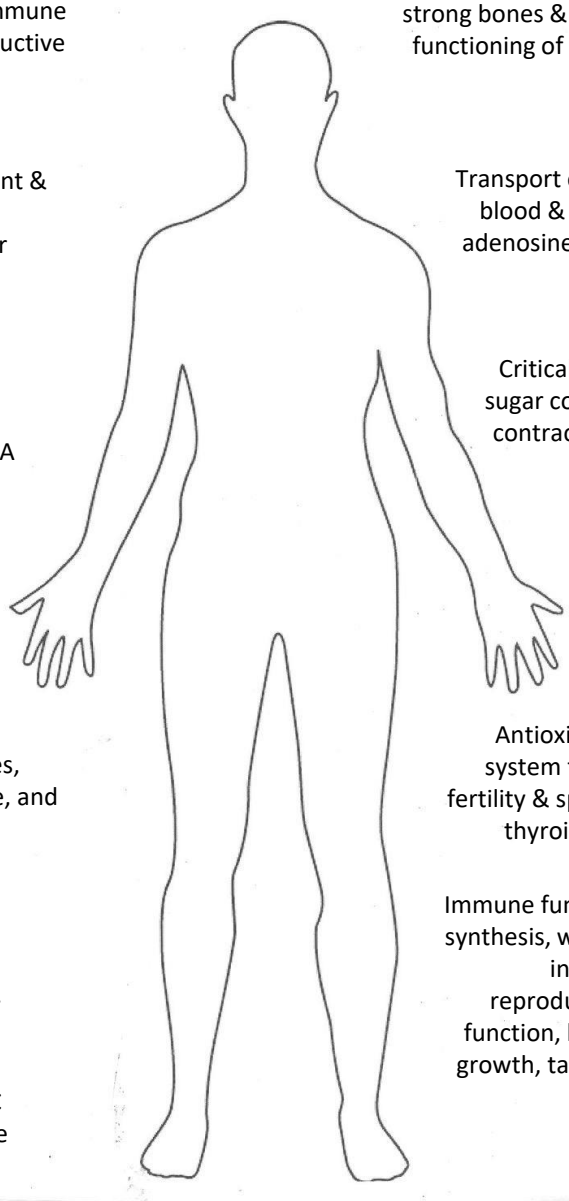
Choline — Liver, muscle and brain function

Vit C — Collagen in bones, cartilage, muscle, and blood vessels.

Vit D — Normal blood levels of calcium and phosphorus.

Vit E — Antioxidant, RBC production & use of vitamin K.

Folate — Brain function, mental & emotional health



Minerals

Calcium — Development & maintenance of strong bones & teeth, proper functioning of heart, nerves, muscles

Iron — Transport of oxygen rich blood & production of adenosine triphosphate (ATP)

Magnesium — Critical role in blood sugar control, muscle contractions & heart rhythm.

Selenium — Antioxidant, immune system function, male fertility & sperm health & thyroid metabolism.

Zinc — Immune function, protein synthesis, wound healing, insulin function, reproduction, thyroid function, blood clotting, growth, taste, vision and smell.

GENETIC PROFILE - NUTRIENTS

YOUR GENETIC PROFILE:

VITAMIN	GENE	YOUR GENOTYPE	ENZYME ACTIVITY	YOUR RISK OF/WITH DEFICIENCY
VITAMIN A	BMCO1 – enzyme that converts beta carotene into active Vitamin A	GG	Reduced	ELEVATED
VITAMIN B6	NBPF3 is associated with the synthesis of NBPF3, a hormone found to be associated with the clearance of vitamin B6 from the body	TT	Normal	NORMAL
VITAMIN B12	FUT2 plays a role in the cellular transport of B12	GG	Reduced	ELEVATED
CHOLINE	PEMT is needed for synthesis of phosphatidylcholine	GG	Normal	NORMAL
VITAMIN C	GSTT1 is needed for utilization of Vitamin C by body	INS	Normal	NORMAL
VITAMIN D	CYP2R1 plays a role in activation of Vitamin D GC plays a role in cellular transportation of Vitamin D	GA GG	Reduced	ELEVATED
VITAMIN E	F5 encodes for coagulation factor V and mutations can increase risk of Venous thromboembolism (VTE), which can be reduced by increasing vitamin E intake.	CT	Reduced	ELEVATED
FOLATE	MTHFR converts folate into an active form utilized by body	CC	Normal	NORMAL

GENETIC PROFILE - NUTRIENTS

YOUR GENETIC PROFILE:

MINERAL	GENE	YOUR GENOTYPE	ENZYME ACTIVITY	YOUR RISK OF/WITH DEFICIENCY
CALCIUM	GC plays a role in Vitamin D transportation and adequate Vitamin D needed for Calcium absorption	TG CA	Reduced	ELEVATED
IRON OVERLOAD	SLC17A1- certain variants correlate with iron overload HFE regulates Hepcidin, which regulates iron absorption and release	CC GG	Normal	NORMAL
LOW IRON	TMPRSS6 helps regulate iron balance TFR2 helps iron enter into cells TF plays a role in iron transfer to body	GA CA AA	Reduced	ELEVATED
MAGNESIUM	MUC1 encodes a protein which is part of mucus layer changes in which can impact nutrient absorption including magnesium SHROOM3 regulates cell shape in certain tissues and associated with Magnesium deficiency TRPM6 plays a role in epithelial magnesium transport and in the active magnesium absorption in the gut and kidney. DCDC5 associates with magnesium deficiency ATP2B1 encodes enzyme for ion transport	AA TT CC GG AA	Normal	NORMAL
SELENIUM	DMGDH shows strong association with serum selenium levels	AA	Normal	NORMAL
ZINC	CA1 is a zinc metalloenzyme PPCDC shows association with zinc deficiency LINC01420 shows association with zinc deficiency	GG TT CC	N/A	NORMAL

How does your genetic makeup impact Fat Consumption?

Fats provide us with a concentrated form of energy. They supply essential fatty acids the body itself cannot produce, help the body store energy, insulate tissues, and absorb fat-soluble vitamins and hormones. There are two main types of dietary fats: saturated and unsaturated. Saturated fats are primarily found in animal-derived foods such as fatty meats, cheese, butter and whole milk dairy.

A diet high in saturated fat has long been associated with health conditions such as diabetes, cardiovascular disease and obesity. Unsaturated fats, such as those found in olive oil, almonds and grape seed oil, may help to decrease the risk of diabetes, cardiovascular disease and obesity. Monounsaturated fats such as olive oil, almonds and avocados have been associated with reduced risk for heart disease. Monounsaturated fats can help reduce “bad” (LDL) cholesterol levels and may also help increase “good” (HDL) cholesterol. Omega-3 fatty acids are a type of unsaturated fat, often referred to as essential fatty acids because our bodies need them to function normally. We don’t naturally manufacture these within our body so it’s very important we take in enough omega-3 as part of our diet. There are many health benefits associated with Omega-3 fatty acids, including amongst others, lowering blood fats, helping reduce rheumatoid arthritis and having an effective anti -inflammatory affect.

We have carried out extensive analysis of the following genes to identify your genetic predisposition to various fat associated tendencies:
TCF7L2, FTO, PPARg2, NOS3, CD36

GENETIC PROFILE – FAT CONSUMPTION

YOUR GENETIC PROFILE:

FAT ASSOCIATED TENDENCIES	GENE	YOUR GENOTYPE	YOUR RISK OF/WITH DEFICIENCY
Weightloss with dietary fat reduction	TCF7L2 plays a role in differentiation of fat cells	TT	ELEVATED PROPENSITY FOR WEIGHTLOSS
Weightloss with increasing unsaturated fat consumption	PPARγ2 activation improves the action of insulin and its lipid metabolism, hence genetic variants in PPARγ2 can impact weight management.	TT	NO ELEVATED PROPENSITY
Weightloss with increasing monounsaturated fat consumption	TMPRSS6 helps regulate iron balance TFR2 helps iron enter into cells TF plays a role in iron transfer to body	CC	NO ELEVATED PROPENSITY
Elevated triglycerides risk with low omega-3 consumption	NOS-3 interacts with omega-3 fatty acids to impact how the body processes triglycerides	GG	NO ELEVATED PROPENSITY
Ability to taste fat	CD36 encodes CD36 translocase protein which shows strong association with ability to taste fat.	GG	ELEVATED PROPENSITY TO TASTE FAT

How does your genetic makeup impact your Diet?

Everyone has a different response to different foods and this can be attributed to the difference in our DNA profile. Our DNA plays a role in determining our dietary preferences, our nutritional needs, food intolerance, as well as the impact of diet on our health whereby certain individuals may be more prone to developing health conditions such as blood pressure with consumption of certain foods than others. We have carried out an extensive assessment of your DNA to identify how different foods are metabolized by your body along with your genetic predisposition to food intolerance.

We have carried out extensive analysis of the following genes to identify your genetic predisposition to various fat associated tendencies:
CYP1A2, ACE, GLUT2, HLA, MCM6, TCF7L2, FTO, AMY1 and MC4R

GENETIC PROFILE - DIET

YOUR GENETIC PROFILE:

DIET & FOOD INTOLERANCE	GENE	YOUR GENOTYPE	YOUR PROFILE
Caffeine associated blood pressure	CYP1A2 encodes for an enzyme that is metabolizing caffeine. The rate of at which caffeine is broken down determines how much caffeine remains bioavailable in the blood stream, which has an impact on heart health.	GA	ELEVATED PROPENSITY
Sodium associated blood pressure	ACE plays a role in regulating blood pressure response in response to sodium intake.	GA	ELEVATED PROPENSITY
Sugar Preference	GLUT2 is involved in glucose transport across cellular membranes and its variants dictate sugar preference.	TT	ELEVATED PROPENSITY
Gluten intolerance	HLA – DQ2 and DQ8 versions are associated with gluten intolerance	rs2187668 (GG)	LOW PROPENSITY
Lactose intolerance	MCM6 is part of the MCM complex which acts as an enhancer to promote production of lactase (an enzyme that breakdowns lactose)	CC	ELEVATED PROPENSITY

GENETIC PROFILE - DIET

YOUR GENETIC PROFILE:

DIET & FOOD INTOLERANCE	GENE	YOUR GENOTYPE	YOUR PROFILE
Whole grain – diabetes risk	TCF7L2 shows strong association with diabetes with certain variants correlating with reducing risk upon increasing whole grain consumption	TT	ELEVATED PROPENSITY FOR DIABETES – MAY BE REDUCED WITH INCREASED WHOLE GRAIN CONSUMPTION
Protein diet – weight loss	FTO has strong association with BMI with certain variants associated with weightloss upon increasing protein consumption.	AA	ELEVATED PROPENSITY
Starch breakdown – Increased insulin resistance	AMY1 encodes amylase which breaks down starch. Reduced AMY1 activity results in increased insulin resistance.	AA	ELEVATED PROPENSITY
Snacking	MC4R is a melanocortin receptor and melanocortins regulate appetite.	CC	ELEVATED PROPENSITY

GENETIC PROFILE – ALCOHOL CONSUMPTION

How does your genetic makeup impact Alcohol Consumption?

Alcohol degradation in our body involves its conversion into acetaldehyde followed by conversion of acetaldehyde into acetic acid. The conversion of acetaldehyde to acetic acid is carried out by aldehyde dehydrogenase – 2 enzyme which is encoded by the ALDH2 gene. Individuals with lower activity of ALDH2 show "alcohol flushing". Such individuals upon consumption of even slight amounts of alcohol become red and they experience headaches, nausea and increased heart rate which is caused by accumulation of aldehyde. Individuals with lower activity of ALDH2 have also shown associated with a high risk of developing certain cancers.

We have carried out extensive analysis of the following gene(s) to assess your alcohol consumption profile:
ALDH2

	GENE	YOUR GENOTYPE	ENZYME ACTIVITY	YOUR SENSITIVITY TO ALCOHOL
ALCOHOL	ALDH2 plays a key role in conversion of aldehyde into acetic acid.	AA	Reduced	HIGHLY ELEVATED, AVOID ALCOHOL

How does your genetic makeup impact Fitness?

DNA sequence differences contribute to human variation in physical activity level, cardiorespiratory fitness in the untrained state, cardiovascular and metabolic response to acute exercise, and responsiveness to regular exercise. If we all exercise in the same way, not all of us will end up as athletes, regardless how hard we exercise.

This is to a great extent due to the differences in our genetic makeup. Everyone will see a response when they go on a diet and are physically active, but there is a difference in the strength of this response. Due to certain genetic variables, some people may require major lifestyle changes and another type of training than others. For instance, it appears that exercising is particularly important for those who are genetically predisposed to becoming overweight. Physical activity appears to reduce the effect of their genetic predisposition to obesity. Here we have provided an assessment of your DNA to identify your complete genetic profile for exercise to identify how your body reacts to different exercises and which ones are preferable for your genetic makeup.

We have carried out extensive analysis of the following genes to identify your genetic predisposition to various exercise associated tendencies: **LIPC, LPL, PPARD, INSIG2, ADRB3, NRF2, GSTP1, NFIA-AS2, EDN1, COL5A1 and COMT.**

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FITNESS PROFILE

EXERCISE BEHAVIOUR	GENE	YOUR GENOTYPE	YOUR PROFILE
Benefit from endurance training	LIPC encodes for hepatic lipase which promotes fat hydrolysis. LPL encodes lipoprotein lipase which breaks down fats to triglycerides. PPARD is a nuclear hormone receptor which controls number of peroxisomes (cellular organelles involved in fat breakdown) in cells.	CC CC AA	ELEVATED PROPENSITY
Fat gain with strength training	INSIG2 encodes proteins involved in blocking activity of transcription factors which bind sterols (steroids).	CC	ELEVATED PROPENSITY
Potential for endurance training	ADRB3 codes for the beta-3 adrenergic receptor, which plays a role in energy metabolism. NRF2 gene codes for the nuclear respiratory factor and is associated with VO2 max. GSTP1 encodes for a detoxification enzyme and is associated with VO2 max. NFIA-AS2 encodes for antisense RNA and shows association with VO2 max.	CC TT CA AG	NO ELEVATED PROPENSITY
Low cardiorespiratory fitness associated blood pressure	EDN1 encodes for a protein which in activated form is a potent vasoconstrictor and its receptors are targets in treating pulmonary arterial hypertension.	GG	ELEVATED PROPENSITY
Risk of exercise induced injury	COL5A1 provides instructions for making type 5 collagen. Collagen strengthens and supports muscles and tendons.	GG	NO ELEVATED PROPENSITY
Pain tolerating ability	COMT encodes an enzyme that plays a role in pain signaling.	AA	NO ELEVATED PROPENSITY

How does your genetic makeup impact your predisposition to metabolic disorders?

Blood sugar and cholesterol are the two important metabolic factors which play a role in development of several chronic diseases. High blood cholesterol is a condition in which your blood has too much cholesterol. The higher your blood cholesterol level, the greater your risk of coronary heart disease (CHD) and heart attack. Low-density lipoproteins (LDL). LDL cholesterol sometimes is called "bad" cholesterol. This is because it carries cholesterol to tissues, including your heart arteries. A high LDL cholesterol level raises your risk of CHD. High-density lipoproteins (HDL). HDL cholesterol sometimes is called "good" cholesterol. This is because it helps remove cholesterol from your arteries. A low HDL cholesterol level raises your risk of CHD. Triglycerides are a type of fat found in the blood. Some studies suggest that a high level of triglycerides in the blood may raise the risk of CHD. Genetics plays a strong role in determining your predisposition to high LDL, high triglycerides as well as low HDL levels.

Over time, a high blood sugar level can lead to increased plaque buildup in your arteries. Having diabetes doubles your risk of CHD. Prediabetes is a condition in which your blood sugar level is higher than normal, but not as high as it is in diabetes. If you have prediabetes and don't take steps to manage it, you'll likely develop type 2 diabetes within 10 years. With modest weight loss and moderate physical activity, people who have prediabetes may be able to delay or prevent type 2 diabetes. They also may be able to lower their risk of CHD and heart attack. Using your individual DNA we have identified your genetic profile for a propensity to have elevated blood sugar, triglycerides, LDL and decreased HDL.

We have carried out extensive analysis of the following genes to identify your genetic predisposition to metabolic factors: **ADCY5, ADRA2A, CRY2, FADS1, G6PC2, GCK, GCKR, GLIS3, MADD, MTNR1B, PROX1, SLC2A2, TCF7L2, ANGPTL3, APOB, FADS1, LPL, MLXIPL, NCAN, PLTP, TRIB1, XKR6, ZNF259, ABCA1, ANGPTL4, CETP, FADS1, GALNT2, HNF4A, KCTD10, LCAT, LIPC, LIPG, PLTP,TTC39B, ABCG8, CELSR2, HMGCR, HNF1A, LDLR, MAFB, NCAN and PCSK9.**

GENETIC PROFILE – METABOLIC DISORDERS

YOUR GENETIC PROFILE:

METABOLIC ISSUE	GENE	YOUR GENOTYPE	YOUR PROFILE
Blood sugar	ADCY5 ADRA2A CRY2 FADS1 G6PC2 GCK GCKR GLIS3 MADD MTNR1B PROX1 SLC2A2 TCF7L2	AA GG AC TT GG AG GA AC AT CC CC TT CC	NO ELEVATED PROPENSITY
High triglycerides	ANGPTL3 APOB FADS1 GCKR LPL MLXIPL NCAN PLTP TRIB1 XKR6 ZNF259	AC AA TT CT AA TT CC CT AA AA CG	ELEVATED PROPENSITY

GENETIC PROFILE – METABOLIC DISORDERS

YOUR GENETIC PROFILE:

EXERCISE BEHAVIOUR	GENE	YOUR GENOTYPE	YOUR PROFILE
Decreased HDL Cholesterol	ABCA1 ANGPTL4 CETP FADS1 GALNT2 HNF4A KCTD10 LCAT LIPC LIPG LPL PLTP TTC39B ZNF259	GG GG CC TT AG CC CC AG CC TT AA CT AA CG	ELEVATED PROPENSITY
Elevated LDL Cholesterol	ABCG8 APOB CELSR2 HMGCR HNF1A LDLR MAFB NCAN PCSK9	CT GA GG CT AC GG CT TT TT	ELEVATED PROPENSITY

How does your genetic makeup impact Detoxification?

Our liver plays a vital role in detoxification by converting fat soluble toxins into water soluble forms which can be eliminated through urine. This protective ability of the liver stems from the expression of a wide variety of xenobiotic biotransforming enzymes whose common underlying feature is their ability to catalyse the oxidation, reduction and hydrolysis (Phase I) and/or conjugation (Phase II) of functional groups on drug and chemical molecules. The broad substrate specificity, isoenzyme multiplicity and inducibility of many of these enzyme systems make them particularly well adapted to handling the vast array of different chemical structures in the environment to which we are exposed daily. However, some chemicals may also be converted to more toxic metabolites by certain of these enzymes, implying that genetic variation in detoxification pathway genes plays a very important role in predisposition for toxicity.

The “Phase I” cytochrome P450 superfamily of enzymes (CYP450) is generally the first defense employed by the body to biotransform xenobiotics, steroid hormones, and pharmaceuticals. The activity of these enzymes use oxygen to modify toxins by forming a reactive site. However, this can also lead to generation of radicals which can contribute to oxidative damage. Genetic variation in CYP450 enzymes impact enzyme activity and expression which impacts how the individual responds to various toxins.

After the Phase I carried out by CYP450 enzymes, the next step, “Phase II”, is conjugation of hydrophilic (water soluble) groups to the reactive site of the toxin (formed in phase I) to make it easily excreted by urine. Similar to the CYP450 enzymes, genetic polymorphisms can have profound influence on the function of these conjugating enzymes with potential implication in the development of several forms of cancer. The enzymes include glucuronyl transferases, sulfotransferases, glutathione transferases, amino acid transferases, N-acetyl transferases and N- and O-methyltransferases. Increased Phase I activity in absence of increased phase II activity can lead to the formation of toxic intermediates. Similarly, decreased Phase I clearance will cause toxic accumulation in the body.

We have carried out extensive analysis of the following genes to identify your genetic profile for detoxification: **CYP1A1, CYP1B1, CYP2A6, CYP2C9, CYP2C19, CYP2D6, CYP3A4, COMT, NAT1, NAT2, GSTM1, GSTP1, SOD1 and SOD2.**

GENETIC PROFILE – DETOXIFICATION

YOUR GENETIC PROFILE:

PHASE I GENE	GENE FUNCTION	YOUR GENOTYPE	ENZYME ACTIVITY	IMPACT
CYP1A1	Role in metabolizing procarcinogens, hormones, and pharmaceuticals. It is well-known for its role in the carcinogenic bioactivation of polycyclic aromatic hydrocarbons (PAHs), heterocyclic aromatic amines/amides, polychlorinated biphenyls, and other environmental toxins. Low CYP1A2 activity, for example, has been linked to higher risk of testicular cancer	CC	Increased	Lower levels of estradiol
CYP1B1	Role in the 4-hydroxylation of estrogen. Additionally, this enzyme is involved in the activation of common environmental toxins such as polycyclic aromatic hydrocarbons (e.g., products from cigarette smoke, car exhaust, and charbroiled foods), polychlorinated biphenyls (e.g., PCBs), and aflatoxin B1.	G	Increased	May increase risk of hepatocellular carcinoma, multiple myeloma, lung cancer and endometrial cancer, but lowers the risk of ovarian cancer and prostate cancer

GENETIC PROFILE – DETOXIFICATION

YOUR GENETIC PROFILE:

PHASE I GENE	GENE FUNCTION	YOUR GENOTYPE	ENZYME ACTIVITY	IMPACT
CYP2A6	Role in metabolizing nicotine along with retinoic acid, testosterone and progesterone.	A	Reduced	Reduced risk of cigarette dependence
CYP2C9	Responsible for the clearance of up to 15-20% of clinical drugs, including antidiabetics, antiepileptics, antihypertensive drugs & anticoagulants.	G	Normal	
CYP2C19	Detox enzyme responsible for clearing approximately 10% of commonly used clinical drugs, including antidepressants.	T	Increased	Depression Risk
CYP2D6	Responsible for the clearance of 20% of clinical drugs, including opioids, antitumor drugs, antidepressants, and antipsychotics. In addition, this enzyme also metabolizes dopamine and serotonin.	T	Intermediate activity	
CYP3A4	Responsible for degrading the majority of drugs and cancer-causing agents, to protect cells and the body from toxins	T	Decreased	Improved asthma control, for cholesterol control require less medication.

GENETIC PROFILE – DETOXIFICATION

YOUR GENETIC PROFILE:

PHASE 2 FUNCTION	GENE FUNCTION	YOUR GENOTYPE	ENZYME ACTIVITY	IMPACT
METHYLATION	COMT is a methyltransferase involved in estrogen detoxification as well as catecholamines.	A	Reduced	May increase risk of psychiatric disorders & impairment of estrogen metabolism
ACETYLATION	NAT 1 metabolizes p-aminobenzoic acid (PABA) and p-aminosalicylic acid (PAS). PABA used to be commonly found in sunscreens, and PAS is used as an antibiotic for tuberculosis. It also breaks down components of cigarette smoke and heterocyclic aromatic amines.	GG	Normal	
ACETYLATION	NAT 2 catalyzes the acetylation of a couple of types of carcinogens (aromatic and heterocyclic amines) which include tobacco smoke, well cooked meat, and exhaust fumes.	CC	Increased	May increase risk of colorectal cancer.
GLUTATHIONE CONJUGATION	GSTM1 functions in the detoxification of electrophilic compounds, including carcinogens, therapeutic drugs, environmental toxins and products of oxidative stress, by conjugation with glutathione.	AA	Reduced	May increase risk of certain cancers & diabetes.
GLUTATHIONE CONJUGATION	GSTP1 reducing the activity of toxins and facilitates their elimination. Also helps prevent neurodegeneration.	GG	Reduced	May increase risk of breast cancer.

GENETIC PROFILE – DETOXIFICATION

YOUR GENETIC PROFILE:

PHASE I GENE	GENE FUNCTION	YOUR GENOTYPE	ENZYME ACTIVITY	IMPACT
OXIDATIVE PROTECTION	SOD enzymes transforms superoxide free radical (major cause of oxidative stress in cells) into oxygen or hydrogen peroxide. SOD1 is located in the cellular fluid.	TT	Decreased	May increase risk of kidney problem in diabetics & heart disease complications.
OXIDATIVE PROTECTION	SOD2 is a SOD enzyme found in the mitochondria of the cell.	GG	Decreased	May increase risk of forgetfulness, a lack of mental clarity, confusion, and an inability to focus

MODULATING DETOXIFICATION PATHWAYS

Our dietary intake has a strong association with the detoxification pathways of our body and several studies have identified induction or inhibition of the detoxification pathway genes with changes in diet and environment. Following is a brief summary of what causes induction or inhibition of the detoxification pathway genes. The following are not recommendations but a summary of recent findings which can help your clinical provider to come up with clinical guidelines based on your genetic makeup to maximize the effects of food and reduce the impact of toxins.

GENE	WHAT INDUCES THE GENE?	WHAT INHIBITS THE GENE?
CYP1A1	Cruciferous vegetables, Resveratrol, Black Rasperry, Blueberry, Ellagic acid, Black soybean, Black tea and Turmeric	Green tea, Sulforaphane found in broccoli, Lycopene and Naringenin
CYP1B1	Diesel Exhaust Particles, UV and Biotin.	St. John's wort, Apigenin, Ginseng, Lycopene, Chrysoeriol, Naringenin and Quercetin
CYP2A6	Quercetin, Chicory root and Broccoli	Starfruit, Cinnamon and Celery
CYP2C9	Resveratrol and Myricetin	Apigenin, Starfruit, Licorice, Caffeic acid and Quercetin
CYP2C19	Kale	Propolis, Caffeic acid, Quercetin, Ginger and Capsaicin
CYP2D6	Resveratrol, Garden Cress and Kale	Starfruit juice, Aloe vera, Goldenseal, Fennel, Garden cress, Curcumin, Asafetida, Berberine, Quercetin, Caffeic acid, Gallic acid and Ellagic acid
CYP3A4	Curcumin	Grapefruit, Starfruit, Aloe vera, Kale, Garden cress, Golden seal, Fennel, Raspberry leaf, Quercetin, Berberine, Black pepper, Sesame seeds and Ginseng
COMT	Magnesium may have some impact	Green tea and High sugar diet
NAT1 and NAT2	Androgens	Caffeic acid, Esculetin, Quercetin, Kaempferol, Genistein, Scopuletin, Coumarin and Garlic

MODULATING DETOXIFICATION PATHWAYS

GENE	WHAT INDUCES THE GENE?	WHAT INHIBITS THE GENE?
GSTM1 and GSTP1	Gingko biloba, Cruciferous vegetables, Grapefruit, Limonene, Allium vegetables, Resveratrol, Fish oil, Black soy bean, Purple sweet potato, Curcumin, Green tea, Rooibos tea, Honeybush tea, Ellagic acid, Rosemary, Ghee, Genistein, Butyrate, Vitamin B6, Magnesium, Selenium, Curcumin, Milk thistle, Folic acid and Alpha-lipoic acid.	Phenols, quinones, dopamine and derivatives of Vitamin C.
SOD1 and SOD2	Fish oil, Zinc, Copper, Manganese, Phosphorus, Curcumin, Lutein, Acetylcholine, Resveratrol, Honey, Ellagic acid, Fennel, Carnitine, Lycopene, Malic acid, Dandelion, Histidine and Glycine	Uric acid, Nicotinamide Riboside, Folate, Garlic, Bilberry Anthocyanins, Inositol, L Plantarum, Ursolic acid, Myricetin and Spirulina

How does your genetic makeup impact Estrogen Metabolism?

Estrogen serve several critical roles in human body, however the high bio-activity of the molecule makes it a potent carcinogen. Increased exposure to estrogen has been linked to high rates of breast cancer development along with aggressiveness of breast cancer. As such, estrogen metabolism by the detoxification pathway is the key to removal of estrogen. Estradiol is converted into 2-OHE1 by CYP1A1 which then subsequently is metabolized into 2-Methoxy-E2 for elimination by COMT. However when COMT activity is reduced, excessive activity of CYP1B1 and CYP3A4 can covert 2-OHE1 to cancerous forms of estrogen (4-OHE-1, 16-OHE-1). Nevertheless, GSTM1 and GSTP1 are capable of converting 4-OHE-1 into non-cancerous glutathione-conjugates.

We have carried out extensive analysis of the following gene(s) to assess your alcohol consumption profile:

CYP1A1, CYP1B1, CYP3A4, COMT, GSTM1, GSTP1

GENETIC PROFILE – ESTROGEN METABOLISM

YOUR GENETIC PROFILE:

PHASE I GENE	YOUR GENOTYPE	ENZYME ACTIVITY	IMPACT
CYP1A1	CC	Increased	Faster conversion of estrogen to 2-OHE-1
CYP1B1	G	Increased	Faster conversion of estrogen into cancerous 4-OHE-1
CYP3A4	T	Decreased	Reduced conversion of estrogen into cancerous 16-OHE-1
COMT	A	Decreased	Reduced conversion of estrogen into excretable forms (2-Methoxy-E2)
GSTM1	AA	Decreased	Reduced conversion of toxic estrogen metabolites into non-toxic forms
GSTP1	GG	Decreased	Reduced conversion of toxic estrogen metabolites into non-toxic forms

OUR ADVISORY BOARD:

ApoE Wolfram C.M. Dempke MD, PhD, MBA
Dr. Dempke is a board-certified physician in internal medicine as well as haematology and oncology. At the same time, Dr. Dempke is a full professor at Cambridge University (UK) and Munich University Medical School (Germany) and has authored and co-authored more than 150 publications including 5 text books. Dr. Dempke also holds a MBA in healthcare and marketing and has held senior leadership roles with several global pharmaceutical leaders including Bristol-Myers Squibb, MerckSerono, Astrazeneca and Kyowa Kirin.

Sunit Das MD, PhD
Dr. Das is a neurosurgeon and scientist at St Michael's Hospital and the Keenan Research Centre, and Assistant Professor in the Department of Surgery at the University of Toronto. Dr. Das studied English Literature at the University of Michigan and Philosophy at Harvard University before moving to Chicago for medical school at Northwestern University. He completed a PhD at the National Institutes of Health, where he studied the molecular processes that underly adult neurogenesis in the lab of Dr ZuHang Sheng. He returned to Chicago for his neurosurgical residency, during which time he also began work on cancer stem cells in primary brain tumours in the lab of Dr John Kessler. Dr Das's clinical practice and clinical research focus of the treatment of patients with tumours of the brain and spine. His laboratory at the Hospital for SickKids focuses on non-coding RNA and control of cell identity in glioblastoma and neural stem cells.

Mike Hart MD
Dr. Hart is the medical director and founder at Readytogo Clinic in London, Ontario.. Dr. Hart is a recognized speaker on the topic of cannabis and is considered an authority on medical cannabis. He has spoken at CME events throughout Ontario, multiple cannabis conferences and has been featured on a variety of cannabis websites. In March of 2017, Dr. Hart has seen first hand how the opioid epidemic is affecting our population and wanted to take action by finding a solution. Dr. Hart believes that cannabis is an excellent alternative to opioids and has seen excellent results in his practice

Abdel Halim, PharmD, MSc, PhD, DABCC, FACB
Dr. Halim is a PharmD, PhD in Clinical Biochemistry and Cancer Molecular Biology, and one of only four lab professionals in the USA who are triple board certified in Clinical Chemistry, Molecular Diagnostics and Toxicology, and a fellow of the National Academy of Clinical Biochemistry (NACB). Dr. Halim is the VP Translational Medicine, Biomarker and Diagnostics at Celldex Therapeutics, based in New Jersey, USA. In his current role, Dr. Halim is overseeing translational medicine including biomarker and target discovery, clinical biomarkers and diagnostics and their utilization in personalized medicine and companion diagnostics. Before Celldex, he held multiple leadership positions in the pharmaceutical and diagnostic industries.Dr. Halim oversaw development and validation of 700+ assays and their applications in drug development and patient managements and led several CDx programs. Dr. Halim has served on 15+ governmental and public expert panels and advisory groups covering different aspects in drug and diagnostic industries in the US, Canada and EU, and on 20+ committees to establish clinical laboratory and IVD guidelines to promote quality practices in the US and worldwide. Abdel has 65 peer-reviewed publications and about 90 presentations including approximately 30 invited and keynote speeches in national and international meetings.

Borhane Guezguez, PhD
Dr. Borhane Guezguez received his doctoral research training in Paris, France at the University Pierre and Marie Curie where he earned his PhD in Cell and Molecular Biology. He conducted his postdoctoral research under the supervision of Dr. Mick Bhatia at the McMaster Stem Cell and Cancer Research Institute where he studied the cellular and molecular mechanisms that regulate the function of stem cells and cancer cells in the hematopoietic system. His main interests are focused on understating genetic and epigenetic deregulations in hematologic malignancies and leukemias in the hopes to develop biomarkers for diagnostic and prognostic use, understanding of drug resistance and finally the identification of novel targets and development of next generation of anti-leukemia stem cell therapies

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DISCLAIMER: This report is for information purposes only and is not intended to be used as medical advice. The advice in this report is not intended to treat, diagnose or cure any medical condition or disease. It is intended for general health and wellness purposes only. The gene panel has not been cleared or approved by the United States Food and Drug Administration. If you have any questions, please ask your healthcare provider or contact us at info@anantlife.com. For Terms of Use and Privacy information please visit our website at www.anantlife.com. The testing laboratory has standard and effective procedures in place to protect against technical and operational problems. However, such problems may still occur. The testing laboratory receives samples collected by patients and physicians. Laboratory problems can occur that might lead to inability to obtain results. Examples include, but are not limited to, sample mislabeling, DNA contamination, uninterpretable results, and human and/or testing system errors. In such cases, the testing laboratory may need to request a new sample. As with all medical laboratory testing, there is a small chance that the laboratory could report inaccurate information. Tested individuals should not make any changes to any medical care without consulting a health care professional. The science behind the significance or interpretation of certain testing results continues to evolve. Although great strides have been made to advance the potential usefulness of genetic testing, there is still much to be discovered. Many of the conditions and drug responses that are tested are dependent on genetic factors as well as nongenetic factors such as age, personal health and family health history, diet, and ethnicity.