Sample Report

Client Report

Venus deMilo

Opus23 Explorer

Opus23 Explorer™ is a fully functional version of the well-regarded and widely used Opus23 Pro™ genomic exploration software designed and programmed by Dr. Peter D'Adamo and distributed under license to Diagnostic Solutions Lab (DSL) by Datapunk Bioinformatics LLC for use in the interpretation of genomic raw data produced by the DSL 'Opus' genomic microarray chip.

Opus23 Explorer scans over 20 peer-reviewed, evidence-based scientific databases and cross-references their information with the results of your raw data. This report summarizes the findings from your genomic data that have been curated by your clinical team into a human-understandable format. However, before we begin, let's introduce a few genetic concepts to set the stage and advance your understanding a bit.

REPORT FOCUS

ANDROGEN GENOMICS

Welcome to your owner's manual

Opus23 Explorer is a very sophisticated computer program that looks for very simple things: variations in the code of DNA (the A, T, C, and G of the genetic alphabet) that can exist between people. Not all of our DNA varies from person to person, but about 9% of it can. The variations are called 'snips' (SNPs) which stands for single nucleotide polymorphism.

Although SNPs are the 'letters' of individuality, genes are in fact the words and vocabulary. After all, it is the genes that have to do the work, coding for the construction for a myriad of enzymes and proteins. Because gene function is central to any sort of biochemical prediction, Opus 23 Pro groups all the SNP outcomes under their parent gene, and presents its results as a reflection of their combined influence on the effectiveness of that gene. Although SNPs are pretty much unchangeable, our genes can be influenced (for better or worse) by lifestyle, diet, emotions and nutritional supplementation.

The DNA in our bodies is a double-stranded molecule, meaning that for every location that we might find a SNP there exists two letters, one for each strand. Taken together, these two letters comprise the **genotype** for that location. Over the years, much research has been done to examine whether a particular SNP variation (or mutation) can be shown to result in an effect on our health. For example, let's look at two different people, John and Jane. At location 12345678 on chromosome #1 most people, as does John, have the 'AA' genotype. It has been noticed that 15% of the population have one 'G' (genotype 'AG') while 5% of the population have genotype 'GG'. Separate studies show that people with at least one 'G' genotype have an increased risk of eczema. Jane's genotype at this location is 'GA' so she may have this susceptibility. As you might have noticed, genotypes come in two types: two identical letters ('GG', 'AA') known as *homozygous* and one of each letter ('GA' or 'AG') known as *heterozygous*.

Because the presence of a 'G' at this SNP location is associated with a condition, for this SNP 'G' is known as the *risk nucleotide* or *risk allele*. Most of the time, having the risk allele negatively impacts the function of its parent gene, but sometimes the mutations can convey a benefit or advantage.

Something like 99.6% of the human genome is identical in all people. This is true of everyone, regardless of race or heritage. However, it is at the SNP location that variation does take place. SNPs only make up a tiny portion of the genome (0.4%) but because the genome is so enormous, this equals over 12 million locations. It's the differences at these SNP locations that make each of us unique. If your genotype at SNP rs17822931 is TT, then you probably have dry earwax. If you have any other genotype at this SNP, then you have wet earwax.

By the way, you're **CT** for the rs17822931 SNP.

This owner's manual was produced by your clinician who, using the Opus23 Explorer software, has curated what, in the great sea of data that Opus23 Explorer provides, they believe is most important to your health care. It would be untrue (and unkind) to pretend that much of the material in this report is easy to understand. Although the editors of Opus23 Explorer try to provide explanations in layperson terminology when and where possible, things can get quite technical. Don't panic! Make note of your questions and remeber to discuss these with your clinician next opportunity. Also, use online resources such as Google and Wikipedia as research tools.

Genetics can be complicated to the layperson. Sometimes a word is used to describe a gene function that you might not recognize. If *Opus23 Explorer* thinks that you might need some help with a technical term, 'Mr. Smart Owl' will try to explain it to you.

Now, a few caveats

Depending on how your health professional has decided to structure this report, you might find the information that follows to be intimidating or even potentially disturbing. For example, nobody enjoys hearing that they may have an increased risk for a disease or health complication. While Opus23 Explorer cannot guarantee that all of its findings will be of a positive nature, it's important to understand what this information can and cannot do. Let's discuss a few facts that you should keep in mind.

Advances in genetic technology have made the process of discovering new SNPs very easy. However the process of linking a SNP to particular trait or illness requires epidemiologic studies that are far more expensive and labor intensive. Thus there is a large gap between the SNPs we know and what in fact we know about them. Opus 23 Pro is constantly updated with new information and your health care provider can very easily update your data to include any new information as it arrives. Opus23 Pro strives to provide the most accurate possible data interpretation. As part of this mission, we constantly monitor and refine our data analysis algorithms. When an improvement is identified, the new algorithm becomes available immediately on creation. In that event, a corrected report will be available to your health care provider. Such re-analysis of patient data may lead to reclassification of your results.

Opus23 Explorer can only supply correlations and relationships

Opus23 Explorer can only compare your genetic data with published data linking your results to the outcomes in the research. It can't diagnose disease. Nor should it. However, it can point the way to areas of possible further clinical interest, and perhaps guide both you and your health care professional in the process of developing a more evidence-based approach to prevention. The etiology (cause) of many diseases is multifactorial; that is, disease can occur as a result of various factors, including both inherited and acquired genetic variants, diet, lifestyle choices and age.

Opus23 Explorer results are as good as the starting data

The interpretations given by Opus23 Explorer are the result of evaluated inherited genetic variants in data uploaded to our server, and interpretations are only as accurate as the data received from the genomic test. It is possible that inaccuracies in the genomic test results could lead to false interpretations. It is also possible that variants in genes and genetic regions not tested in the DNA sequencing test may contribute to an individual’s risk for disease. Therefore, a negative result in a gene where no pathogenic variants are detected does not eliminate the individual’s disease risk.

Genetic findings can only report the starting point

Your genome is similar to the blueprint for a house that is yet to be built. If the builder follows the architect's instructions exactly, the house will match the blueprint perfectly. However, all throughout the construction process alterations will most certainly be made: For example, if the new owners are running short on funds, perhaps the original plans for an expensive slate roof may have to be altered to a less expensive, though still-functional, asphalt version. It's the same with genomics, although variations in your gene data may reflect an increased or decreased risk of a health issue, many of these risks may have been altered by environmental factors (such as your pre-existing lifestyle and health habits) acting epigenetically to control the expression of these genes. If you've carefully watched your diet over time and kept your weight at a healthy level, a finding that you are at risk for obesity might do nothing more than encourage you to continue what you are already doing.

Genetic findings can only reflect probabilities

Very few gene mutations result in a direct, absolutely certain, health consequence. Most of the time, they instead reflect a change to your odds of developing a particular health condition. This is defined as the 'risk' for a certain event. This is usually expressed as an 'odds ratio' (OR). Understanding the meaning of an OR for a particular risk is a key to minimizing stress when encountering dire results. For example, being told you are 110% more likely to get struck by lightning (OR=1.1) is much less distressing when you realize that:

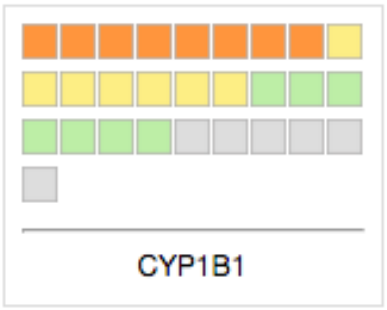
- This is a very small difference from normal
- Very few people get struck by lightning regardless

When it comes to a particular disease or syndrome, most SNPs have rather small ORs. This does not mean that they are unworthy of attention, but rather that the findings must be interpreted as part of an integrated whole, including: other SNP results that also support the conclusion; lifestyle factors; family history, and environmental exposures. Further, a positive test result does not guarantee an occurrence of disease since the SNP variants in most genes are not 100% penetrant (even genes with several risk SNPs will very likely function to some degree). Rather, pathogenic variants may predispose a person to a higher or lower risk of disease. The results of genomic testing must be interpreted in the context of your clinical history. Genetic counseling is recommended for the individual and for other at-risk family members.

And now, the usual indemnification statement:

Understanding the report

Each gene is depicted as a grid showing the result of its SNPs:



- The sum of the significant SNPs in the gene that indicate a higher (homozygous) risk are the orange squares
- The sum of the significant SNPs in the gene that indicate a lower (heterozygous) risk are the yellow squares
- The sum of the significant SNPs that are working just fine (no problem polymorphisms) risk are the gray squares
- You might even find that for some genes you may have a polymorphism that conveys some benefit. These are the green squares

SNP outcomes in GENE relevant to Venus deMilo:

SNP	RISK	TYPE	YOU	OUTCOME	MAGNITUDE	KEYWORDS
rs17367504	C	B	AC	-+		HYPERTENSION, ORTHOSTATIC HYPERTENSION, RESPONSE TO BETA BLOCKERS
rs1999594	A	R	AA	++		FOLATE TRANSPORTER, LOW SERUM FOLATE, HIGH HOMOCYSTEINE
rs1801131	G	R	GT	+-		NEUROTRANSMITTER SYNTHESIS

Multi SNP macros

Macros (algorithms) are perhaps the most significant and flexible aspect of your Opus 23 data. They are usually the easiest result for the non-medical person to understand, because their conclusions are usually simplified statements in everyday language.

Many correlations between SNPs and various traits exist as 'haplotypes,' clusters of SNPs, often on different genes, that must be evaluated as 'true' or 'false' based on their total outcome values. Some algorithms may identify risks for certain problems, while others identify special strengths or benefits you might possess. It's helpful to think of an Opus 23 algorithm as a tiny flowchart, that depending on which way the result branches, generates a 'true or false' result.

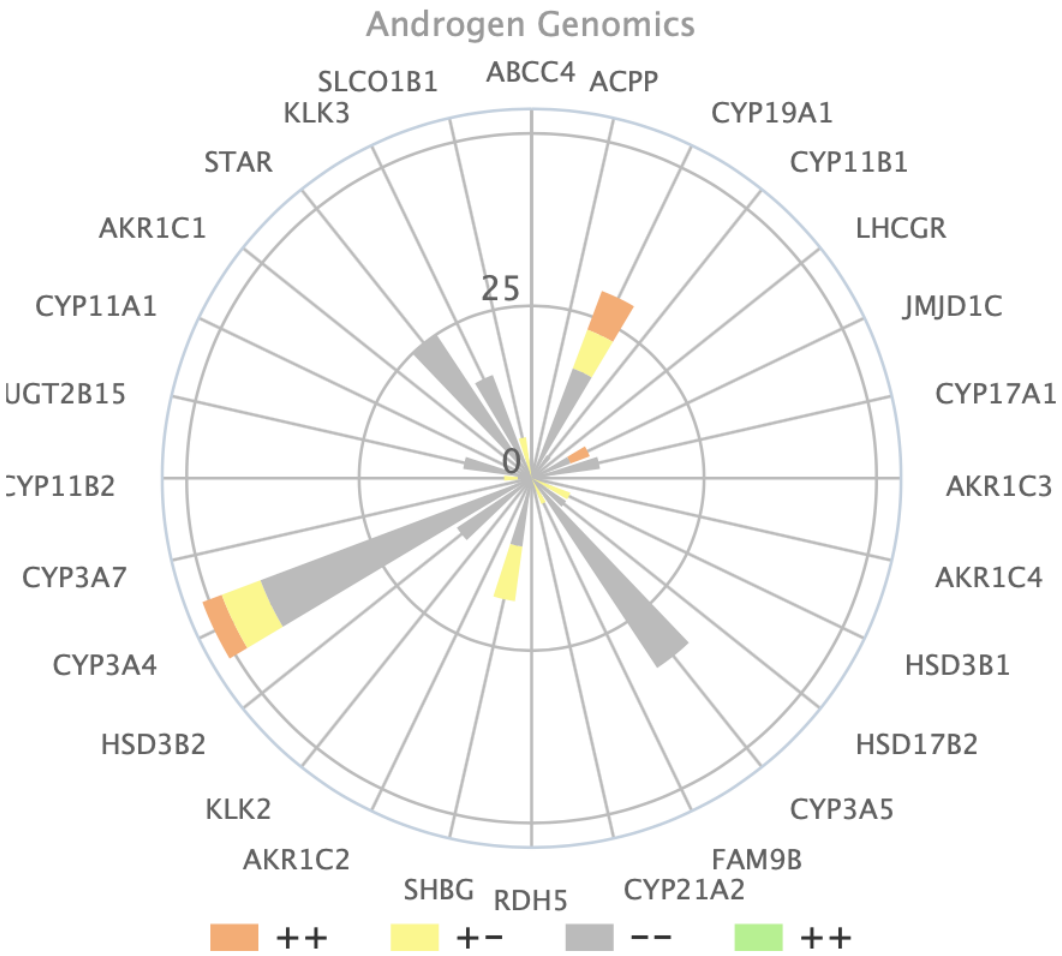
For example, a simple macro to determine if you should get out of bed might be:

- If you hear the alarm clock, open your eyes.
- If it's dark outside, go back to bed.
- If it's light outside, check the time.
- If it's earlier than 7AM, go back to bed.
- If it's later than 7AM, get up, check calendar
- If it's Saturday, go back to bed.

As can be seen, there are a lot of ways you can go back to bed with this algorithm! And this is also true as well for the Opus 23 Pro algorithms: In order for an algorithm to be true, it must fufill all of several conditions. *If even one condition fails, the whole algorithm will be false.*

Each macro algorithm is displayed in its own box, and contain information about the genes and SNPs used in its creation. The title of the algorithm is generally its conclusion. Typically, your report contains only true algoriths, although your clinical team may choose to include false algorithms as well, especially if it would be helpful to make you aware of something you're likely to not be prone to. Thus:

- An algorithm that returns a **true** will have a 'check' icon in the bottom left-hand box. The conclusions of these algorithms **pertain** to you based on your genomic data results.
- An algorithm that returns a **false** will have a 'cross' icon in the bottom left-hand box. The conclusions of these algorithms **do not pertain** to you based on your genomic data, other than perhaps the added knowledge that this is one less thing in life to worry about.



ANDROGEN GENOMICS

Androgen Genomics

An androgen is any natural or synthetic steroid hormone that regulates the development and maintenance of male characteristics in vertebrates by binding to androgen receptors. This includes the embryological development of the primary male sex organs, and the development of male secondary sex characteristics at puberty. Androgens are synthesized in the testes, the ovaries, and the adrenal glands. Androgens increase in both boys and girls during puberty. The major androgen in males is testosterone. Dihydrotestosterone (DHT) and androstenedione are of equal importance in male development. DHT in utero causes differentiation of penis, scrotum and prostate. In adulthood, DHT contributes to balding, prostate growth, and sebaceous gland activity. Although androgens are commonly thought of only as male sex hormones, females also have them, but at lower levels: they function in libido and sexual arousal. Also, androgens are the precursors to estrogens in both men and women.

CYP19A1

cytochrome P450, family 19, subfamily A, polypeptide 1

CYP19A1 (also known as 'aromatase') catalyzes the last steps of in the synthesis of estrogen. Polymorphisms in this gene can result in either increased or decreased aromatase activity. Aromatase activates many reactions involved in the production of steroids. In particular, aromatase is responsible for the aromatization of androgens (testosterone, DHEA, DHT) into estrogens. The aromatase enzyme can be found in many tissues including gonads, brain, adipose tissue, placenta, blood vessels, skin, and bone, as well as in tissue of endometriosis, uterine fibroids, breast cancer, and endometrial cancer. It is an important factor in sexual development.

CYP450 family member plays a much greater role in the regulation of oxidative stress due to its redox effects. When upregulated it is a major contributor of ROS and major consumer of reducing agent NADPH, important in glutathione recycling. CYP450 family also play a major role in cell danger signaling and cell turnover as it interacts electrically with the apoptosis mechanisms controlled by mitochondria.

SNP outcomes in CYP19A1 relevant to Venus deMilo:

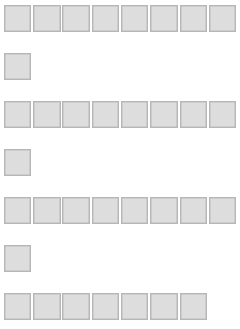
SNP	RISK	TYPE	YOU	OUTCOME	MAGNITUDE	KEYWORDS
rs10046	A	R	**AA**	**++**		ENDOMETRIOSIS, HARM AVOIDANCE PERSONALITY TRAIT, PSA LEVELS, BREAST CANCER, HYPERTENSION IN MALES, RECURRENT MISCARRIAGE, SPERM CONCENTRATION
rs4775936	C	R	**TT**	**--**		REDUCED PROSTATE CANCER SURVIVAL, ENDOMETRIAL CANCER, LOWER BMD
rs2470152	A	R	**AG**	**+-**		HIGHER ESTROGEN LEVELS AND BMD IN MEN, PROSTATE CANCER SURVIVAL, HETEROZYGOUS INHIBITS AROMATASE AND CAUSES HYPERANDROGENISM IN PCOS, FRACTURE RISK
rs10459592	T	R	**GG**	**--**		PROSTATE CANCER, LOWER RISK OF HEPATOCELLULAR CARCINOMA, REDUCED AROMATASE ACTIVITY

New concepts:

- The *gene* is the fundamental physical and functional unit of heredity. A gene is an ordered sequence of nucleotides located in a particular position on a particular chromosome that encodes a specific product (i.e., a protein).
- *Apoptosis* is the process of programmed cell death that may occur in multicellular organisms. In contrast to traumatic cell death from cellular injury, apoptosis is a highly regulated and controlled process that confers advantages during an organism's lifecycle.
- *Mitochondria* are a cell constitutent (organelle) found in large numbers in most cells, in which the biochemical processes of respiration and energy production occur.
- An *androgen* is any natural or synthetic compound, usually a steroid hormone, that stimulates or controls the development and maintenance of male characteristics.
- *Oxidative stress* reflects an imbalance between the levels of reactive oxygen species and the body's ability to readily detoxify the reactive intermediates or to repair the resulting damage.
- To *Catalyze* is to cause or accelerate (a reaction) by acting as a catalyst.

CYP3A5

cytochrome P450, family 3, subfamily A, polypeptide 5



This gene encodes a member of the cytochrome P450 superfamily of enzymes. The cytochrome P450 proteins are phase I detoxification enzymes which catalyze many reactions involved in drug metabolism and synthesis of cholesterol, steroids and other lipids. CYP3A5 metabolizes drugs as well as the steroid hormones testosterone and progesterone. CYP3A5 expression is induced by glucocorticoids and some pharmacological agents. It oxidizes a variety of structurally unrelated compounds, including steroids, fatty acids, and xenobiotics. The human CYP3A subfamily, CYP3A4, CYP3A5, CYP3A7 and CYP3A43, is one of the most versatile of the biotransformation systems that facilitate the elimination of drugs (37% of the 200 most frequently prescribed drugs in the U.S. are eliminated via these mechanisms).

SNP outcomes in CYP3A5 relevant to Venus deMilo:

SNP	RISK	TYPE	YOU	OUTCOME	MAGNITUDE	KEYWORDS
No significant SNP mutations to report						

New concepts:

- A *xenobiotic* is a chemical compound foreign to the body. Xenobiotics include drugs, and environmental compounds such as pollutants that are not produced by the body. In the environment, xenobiotics include synthetic pesticides, herbicides, and industrial pollutants that would not be found in nature.
- *Proteins* are large molecules composed of one or more chains of amino acids. Proteins are required for the structure, function, and regulation of the bodys cells, tissues, and organs, and each protein has unique functions. Examples are hormones, enzymes, and antibodies.

FAM9B

family with sequence similarity 9, member B

This gene is a member of a gene family which arose through duplication on the X chromosome. The encoded protein may be localized to the nucleus as the protein contains several nuclear localization signals, and has similarity to a synaptonemal complex protein.
SNP outcomes in FAM9B relevant to Venus deMilo:

SNP	RISK	TYPE	YOU	OUTCOME	MAGNITUDE	KEYWORDS
rs5934505	T	R	**T**	**+-**		LOW SERUM TESTOSTERONE, LATE-ONSET HYPOGONADISM, AND CALCULATED FREE TESTOSTERONE

New concepts:

- The *nucleus* is the central part of most cells that contains genetic material and is enclosed in a membrane

SHBG

sex hormone-binding globulin

SHBG transports androgens (such as testosterone) and estrogens in the blood. Polymorphisms in this gene have been associated with polycystic ovary syndrome and type 2 diabetes mellitus. SHBG is produced mostly by the liver and is released into the bloodstream. SHBG levels are decreased by androgens, administration of anabolic steroids,[20] polycystic ovary syndrome, hypothyroidism, obesity, Cushing's syndrome, and acromegaly. Low SHBG levels increase the probability of Type 2 Diabetes. SHBG levels increase with estrogenic states (oral contraceptives), pregnancy, hyperthyroidism, cirrhosis, anorexia nervosa, and certain drugs. Long-term calorie restriction of more than 50 percent increases SHBG, while lowering free and total testosterone and estradiol.

SNP outcomes in SHBG relevant to Venus deMilo:

SNP	RISK	TYPE	YOU	OUTCOME	MAGNITUDE	KEYWORDS
rs1799941	A	R	**AG**	**+-**		SEX HORMONES
rs6258	T	R	**CC**	**--**		TESTOSTERONE, ESTROGEN

CYP3A4

cytochrome P450, family 3, subfamily A, polypeptide 4

CYP3A4 is an important enzyme in the body, mainly found in the liver and in the intestine. Its purpose is to oxidize small foreign organic molecules (xenobiotics), such as toxins or drugs, so that they can be removed from the body. While many drugs are deactivated by CYP3A4, there are also some drugs which are *activated* by the enzyme. Some substances, such as grapefruit juice and some drugs, interfere with the action of CYP3A4. These substances will therefore either amplify or weaken the action of those drugs that are modified by CYP3A4. This enzyme is involved in the metabolism of approximately half the drugs in use today, including acetaminophen, codeine, cyclosporin A, diazepam and erythromycin. The enzyme also metabolizes some steroids and carcinogens. In 1998, various researchers showed that grapefruit juice, and grapefruit in general, is a potent inhibitor of CYP3A4, which can affect the metabolism of a variety of drugs, increasing their bioavailability. In addition to grapefruit, other fruits have similar effects. Noni (M. citrifolia), for example, is a dietary supplement typically consumed as a juice and also inhibits CYP3A4; pomegranate juice has this effect as well.

CYP40 family members compete for activity and process hormones and xenobiotics variably in patients. CYP450 family member plays a much greater role in the regulation of oxidative stress due to its redox effects. When upregulated it is a major contributor of ROS and major consumer of reducing agent NADPH, important in glutathione recycling. They also plays a major role in cell danger signaling and cell turnover as it interacts electrically with the apoptosis mechanisms controlled by mitochondria.

SNP outcomes in CYP3A4 relevant to Venus deMilo:

SNP	RISK	TYPE	YOU	OUTCOME	MAGNITUDE	KEYWORDS
rs28371759	G	R	**AA**	--		INCREASED ACTIVITY
rs4986910	G	R	**AA**	--		
rs55785340	G	R	**AA**	--		
rs3091339	C	R	**TT**	--		
rs2740574	C	R	**TT**	--		INCREASED ACTIVITY
rs55901263	C	R	**GG**	--		
rs2246709	G	R	**AA**	--		
rs4646437	A	R	**AG**	+-		DRUG METABOLISM

STAR

steroidogenic acute regulatory protein

The STAR gene provides instructions for making the steroidogenic acute regulatory protein. This enzyme plays a major role in regulating the production of steroid hormone when needed by increasing the rate of conversion of cholesterol into the hormone pregnenolone. This protein permits the splitting (cleavage) of cholesterol into pregnenolone by helping the transport of cholesterol into the mitochondria (the energy-producing part of the cell, which has two membranes) from the outer membrane of the mitochondria to the inner membrane. Variations in this gene are a cause of congenital lipoid adrenal hyperplasia (CLAH), also called lipoid CAH.

SNP outcomes in STAR relevant to Venus deMilo:

SNP	RISK	TYPE	YOU	OUTCOME	MAGNITUDE	KEYWORDS
rs137852690	A	R	**GG**	--	■■■■■	CHOLESTEROL MONOOXYGENASE DEFICIENCY
rs387907235	A	R	**GG**	--	■■■■■	CHOLESTEROL MONOOXYGENASE DEFICIENCY
rs104894085	A	R	**GG**	--	■■■■■	CHOLESTEROL MONOOXYGENASE DEFICIENCY
rs104894087	T	R	**CC**	--	■■■■■	CHOLESTEROL MONOOXYGENASE DEFICIENCY
rs104894090	A	R	**GG**	--	■■■■■	CHOLESTEROL MONOOXYGENASE DEFICIENCY

KLK3

kallikrein-related peptidase 3

KLK3 provides instructions for making the kallikrein-related peptidase 3 protein. It is present in the plasma of semen, and clinically is known as PSA, or prostate-specific antigen. PSA is present in small quantities in the blood of men with healthy prostates, but is often found at higher levels in men with prostate cancer or other prostate disorders such as prostatitis and benign prostatic hyperplasia (BPH).

Genes in the KLK family provide instructions for making enzymes called kallikreins, which act as serine proteases. A protease is an enzyme that breaks down proteins. The protein to be broken down attaches (binds) to the serine protease at a region of the enzyme known as the active site. In serine proteases, this active site always contains the protein building block (amino acid) serine.

The serine proteases produced from genes in the kallikrein family help break down proteins called kininogens to produce smaller proteins known as kinins. Kinins are involved in many processes in the body, including inflammation, blood clotting, and blood pressure control. Changes in genes in this family can affect the risk of developing cardiovascular disease, cancer, and other conditions influenced by these processes.

SNP outcomes in KLK3 relevant to Venus deMilo:

SNP	RISK	TYPE	YOU	OUTCOME	MAGNITUDE	KEYWORDS
No significant SNP mutations to report						

New concepts:

- *Amino acid* are small molecules that are the components of proteins. There are 20 different kinds of amino acids in living things. Proteins are composed of different combinations of amino acids assembled in chain-like molecules.

SLCO1B1

solute carrier organic anion transporter family, member 1B1

This gene encodes a liver-specific member of the organic anion transporter family. The encoded protein is a transmembrane receptor that mediates the sodium-independent uptake of numerous endogenous compounds including bilirubin, 17-beta-glucuronosyl estradiol and leukotriene C4. This protein is also involved in the removal of drug compounds such as statins, bromosulfophthalein and rifampin from the blood into the hepatocytes. Polymorphisms in the gene encoding this protein are associated with impaired transporter function. [provided by RefSeq, Mar 2009]

References:

1. Voora D, Shah SH, Spasojevic I, Ali S, Reed CR, Salisbury BA, Ginsburg GS. The SLCO1B1*5 genetic variant is associated with statin-induced side effects. J Am Coll Cardiol. 2009 Oct 20;54(17): 1609-16. doi: 10.1016/j.jacc.2009.04.053.

SNP outcomes in SLCO1B1 relevant to Venus deMilo:

SNP	RISK	TYPE	YOU	OUTCOME	MAGNITUDE	KEYWORDS
rs4149056	C	R	**CT**	+-		SLCO1B1*5 STATIN INDUCED MYOPATHY, REDUCED STATIN AND METHOTREXATE METABOLISM, STATIN MYOPATHY

New concepts:

- A *receptor* is a molecule in a cell membrane, that responds specifically to a particular neurotransmitter, hormone, antigen, or other substance.

ANDROGEN GENOMICS

MULTI-SNP MACROS

Androgen Genomics macro algorithms returning as false:

- Significantly increased risk of male pattern baldness
- Severe risk of testicular germ cell tumors

NATURAL PRODUCTS

This section lists the top 25 natural products that may be worthy of attention as potentially valuable therapeutic agents:

RANK	AGENT	INDICATION VALUE
1.	Coleus Forskohlii	
2.	Selenium	
3.	Apis mellifica	
4.	Biochanin A	
5.	Hesperetin	
6.	Euonymus alatus	
7.	Galangin	
8.	Scutellaria barbata	
9.	Estradiol	
10.	Baicalein	
11.	Apigenin	
12.	Soy Protein	
13.	Naringenin	
14.	Chrysin	
15.	Silymarin	
16.	Glycyrrhetinic acid	
17.	Pinostilbene	
18.	Soybean Beans	
19.	Toxicant avoidance	
20.	Grapefruit	
21.	Glycyrrhizin	
22.	Rhamnus frangula	
23.	Salidroside	
24.	Chelidonium majus	
25.	Cholesterol	

DRUG INTERACTIONS

This section documents potential drug interactions or complications you may be genetically susceptible to.

DRUG	SNP	GENE	RISK ALLELE	YOUR GENOTYPE	SIDE EFFECT
Acitretin	rs7412	APOE	C	**CC**	Psoriasis
Amitriptyline	rs4244285	CYP2C19	A	**AG**	Those with the AA or AG genotype are poor metabolizers of amitriptyline
Azathioprine	rs1800460	TPMT	T	**CT**	Hepatotoxicity
Azathioprine	rs1142345	TPMT	C	**CT**	Hepatotoxicity
Azathioprine	rs1142345	TPMT	C	**CT**	Patients with CC or CT genotype have decreased inactivation of thiopurines and increased risk of toxicity
Carbamazepine	rs3909184	FLOT1	G	**GG**	Patients with the CG or GG genotype (in Asian patients) were at a higher risk of Steven-Johnson Syndrome compared to those with the CC genotype (non-carriers of HLA-b*1502)
Cisplatin	rs1695	GSTP1	A	**AG**	Tinnitus, hearing impairment, Raynaud syndrome
Clobazam	rs4244285	CYP2C19	G	**AG**	Clobazem is metabolized into N-desmethylclobazem (NCLB) mostly by CYP3A4. NCLB is primarily metabolized by 2C19. Those with one 2C19*2 allele mutation (1*/2*) are intermediate metabolizers of NCLB. Those with two (2*/2*) mutations will metabolize NCLB poorly in comparisone to extensive metabolizers (1*/1*). Levels of NCLB can be five times higher in poor metabolizers, and two times higher in intermediate metabolizers as compared to individuals who are extensive metabolizers. The safety and efficacy of clobazem may be affected by polymorphic expression of CYP2C19*2.
Cyclosporine	rs231775	CTLA4	A	**AG**	Gingival overgrowth, periodontal disease
Fluorouracil	rs1695	GSTP1	A	**AG**	Hematological toxicity, gastrointestinal toxicity
Gefitinib	rs2231142	ABCG2	T	**GT**	Diarrhea
Gefitinib	rs2231142	ABCG2	T	**GT**	In non-small lung cancer patients, those that are heterozygous (ABCG2 421C>A) have a higher risk of diarrhea.

Irinotecan	rs4149056	SLCO1B1	C	CT	Diarrhea, leucopenia, neutropenia
Isoniazid	rs6413419	CYP2E1	GG	GG	Hepatotoxicity
Mercaptopurine	rs1800460	TPMT	T	CT	Hepatotoxicity
Mercaptopurine	rs1142345	TPMT	C	CT	Hepatotoxicity
Venlafaxine	rs5030655	CYP2D6	I	II	Nausea, vomiting diarrhea
Almotriptan	rs5443	GNB3	T	CT	Better response to drug treatment
Citalopram	rs1954787	GRIK4	C	CC	Improved response to antidepressant medication
Clopidogrel	rs4244285	CYP2C19	A	AG	Patients are poor metabolizers of clopidogrel. More likely to experience poor cardiovascular outcomes.
Clopidogrel	rs4244285	CYP2C19	A	AG	Patients are poor metabolizers of clopidogrel. More likely to experience poor cardiovascular outcomes.
Codeine	rs5030655	CYP2D6	I	II	Poor drug metabolizer, lower dose requirements
Dextromethorphan	rs5030655	CYP2D6	II	II	Poor drug metabolizer, lower dose requirements
Eletriptan	rs5443	GNB3	T	CT	Better response to drug treatment
Frovatriptan	rs5443	GNB3	T	CT	Better response to drug treatment
Infliximab	rs1801274	FCGR3A	GG	GG	Better ACR20 response
Modafinil	rs4680	COMT	GG	GG	Those with the GG genotype respond better to drug therapy (improved vigor and well being). Those with the AA genotype do not respond well to drug therapy
Morphine	rs1799971	OPRM1	A	AA	Better response to pain relief drugs
Naratriptan	rs5443	GNB3	T	CT	Better response to drug treatment
Rizatriptan	rs5443	GNB3	T	CT	Better response to drug treatment
Rosuvastatin	rs2231142	ABCG2	T	GT	Greater response to drug therapy
Sildenafil	rs5443	GNB3	T	CT	Better response to drug treatment
Sumatriptan	rs5443	GNB3	T	CT	Better response to drug treatment
Trastuzumab	rs351855	FGFR4	G	AG	Reduced response to herceptin
					Poor drug metabolizer, lower dose requirements,

Venlafaxine	rs5030655	CYP2D6	I	**II**	nausea, vomiting and diarrhea
Venlafaxine	rs5030655	CYP2D6	I	**II**	Poor drug metabolizer, lower dose requirements, nausea, vomiting and diarrhea
Zolmitriptan	rs5443	GNB3	T	**CT**	Better response to drug treatment