DEMO DEMO

Name: DEMO DEMO
Date of Birth: 04-19-1965
Biological Sex: Male
Age: 60
Height:
Weight:
Fasting:

Telephone: 000-000-0000
Street Address:
Email:

FINAL REPORT

Accession ID: 2821919475

Provider Information

Practice Name: DEMO CLIENT, MD
Provider Name: DEMO CLIENT, MD
Phlebotomist: 0

Telephone: 000-000-0000
Address: 3521 Leonard Ct, Santa Clara, CA 95054

Report Information

Current Result | Previous Result | In Control | Moderate | Risk

Specimen Information

Sample Type	Collection Time	Received Time	Report	Final Report Date
EDTA	2025-09-02 00:00 (PDT)	2025-09-02 12:04 (PDT)	Cardio Genetics - P2	2025-09-03 06:27 (PDT)

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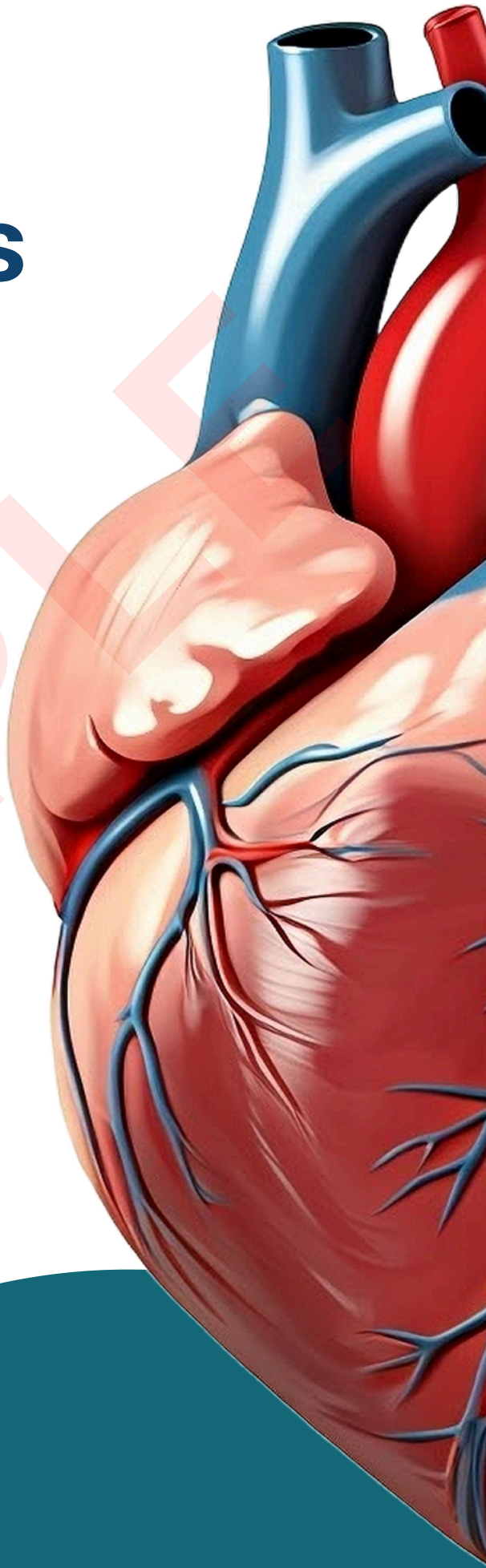
VibrantWellness
3521 Leonard Ct, Santa Clara, CA 95054
1-866-364-0963 | support@vibrant-america.com | www.vibrant-wellness.com

VibrantWellness

Cardio Genetics

Your Cardiac Health Report

The Cardio Genetics panel assesses genetic predispositions associated with your cardiac health. It uses real-time PCR to evaluate SNPs associated with different markers such as LDL, ApoB, Lp(a) as well as systemic conditions like hypertension, atherosclerosis, foam cell formation etc. DNA is extracted and purified from blood samples and a genotyping assay is performed to detect specific allele targets to determine the risk associations for the individual tested.



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Cardio Genetics

INTRODUCTION

Vibrant Wellness is pleased to present to you, Cardio Genetics testing, to help you make healthy lifestyle choices in consultation with your healthcare providers and dietitians. It is intended to be used as a tool to encourage a general state of health and well-being.
Cardio Genetics is a genetic test which detects and interprets variants known to be associated with increased predispositions to various heart conditions and metabolic responses to certain associated pharmacological agents. Its intended use is to help reduce the risk of certain heart conditions by making healthy lifestyle choices.

Methodology:

The Vibrant Cardio Genetics panel uses real-time PCR methodology. DNA is extracted and purified from blood samples and a SNP (single nucleotide polymorphism) genotyping assay is performed using real-time PCR to detect the specific allele targets of each assay performed. Insertion/deletion polymorphism using real-time PCR is used for detecting rs464994 target.

Interpretation of Report:

The Cardio Genetics report starts with a summary page which contains the half-life score of various categories and markers. The genetic variants on the report are organized as multiple charts with wheels under different subheadings for associated markers. The summary page lists the set of analytes with risk associated variants. Following this section is the complete list of the genetic markers measured in the panel. Elevated risk associated variants are indicated with red, partially elevated risk associated variants are indicated with yellow and alleles with no risk are indicated with green. All contents provided in the report are purely for informational purposes only and should not be considered medical advice. Any changes based on the information provided should be made in consultation with the clinical provider.

The Vibrant Wellness platform provides tools for you to track and analyze your general wellness profile. Testing for the Cardio Genetics panel is performed by Vibrant Genomics, a CLIA certified lab CLIA#: 05D2098445. Vibrant Wellness provides and makes available this report and any related services pursuant to the Terms of Use Agreement (the "Terms") on its website at www.vibrant-wellness.com. By accessing, browsing, or otherwise using the report or website or any services, you acknowledge that you have read, understood, and agree to be bound by these terms. By accessing or using this report, you acknowledge that you have read and understood the Risks and Limitations – Genetics section and agree to consider its contents when interpreting your results. If you do not agree to these terms, you shall not access, browse, or use the report or website. The statements in this report have not been evaluated by the Food and Drug Administration and are only meant to be lifestyle choices for potential risk mitigation. Please consult your healthcare provider for medication, treatment, diet, exercise, or lifestyle management as appropriate. This product is not intended to diagnose, treat, or cure any disease or condition.

Please note:

Consider all supplements in relation to medical history and symptoms. Not all recommended supplements are appropriate in all individual cases. It is important that you discuss any modifications to your diet, exercise, and nutritional supplementation with your healthcare provider before making any changes. Pediatric ranges have not been established for these tests. Pediatric ranges have not been established for this test. It is important that you discuss any modifications to your diet, exercise, and nutritional supplementation with your healthcare provider before making any changes.

SAMPLE

Cardio Genetics - Summary

Questionnaire Data

DEMOGRAPHICS

Date of Birth	2000-01-01	Biological sex	Male		
Height	70 inches	Weight	160 lbs	Ethnicity	Asian
Please specify:			Chinese		

CARDIAC HEALTH SYMPTOMS

Chest pain or tightness	No symptoms	Shortness of breath	No symptoms	Fatigue or weakness	Mild
Dizziness or fainting	No symptoms	Elevated systemic blood pressure	No symptoms	Lightheadedness or unusual fatigue during or after exercise	No symptoms
Rapid or irregular heartbeat	No symptoms	Palpitations	No symptoms		

SYSTEMIC SYMPTOMS

Hyperpigmented skin patches	No symptoms	Sudden weight fluctuations	Mild	Persistent feeling of hunger or lack of Satiety	No symptoms
Swelling in the hands, feet, or face	No symptoms	Altered urinary patterns	No symptoms	Brain fog	Mild
Yellowing of the skin or eyes	No symptoms	Right upper abdominal discomfort or pain	No symptoms	Frequent infections	No symptoms

MEDICAL BACKGROUND

FAMILY HISTORY

Cardiovascular disease	No	High blood pressure	Yes	High cholesterol	No
Atherosclerosis	No	Peripheral arterial disease	No	Diabetes	Yes
Insulin resistance	No	Kidney disease	No	Liver disease	No
Other, please specify:					

MEDICAL HISTORY

Cardiovascular disease	No	High blood pressure	No	High cholesterol	No
Atherosclerosis	No	Peripheral arterial disease	No	Diabetes	No
Insulin resistance	No	Kidney disease	No	Liver disease	No
Other, please specify:					

Have you had your blood pressure checked recently?	Yes	If yes, then please specify:	Systolic: 110, Diastolic: 75
Have you had any heart-related surgeries or procedures?	No	If yes, then please specify:	
Are you currently on any medications?	No	If yes, then please list:	
Are you currently on any supplements?	Yes	If yes, then please list:	Takes multivitamins and omega-3

SAMPLE

Questionnaire Data

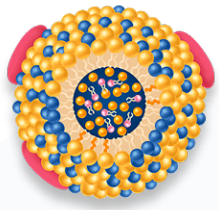
DIETARY PATTERNS	
Diet high in processed foods and sugars	Sometimes
Balanced diet	Often
High-fat diet	Rarely
LIFESTYLE	
On average, how often do you engage in physical activity	Moderately active
On average, how often do you engage in different types of physical activity	Three to four per week
What types of physical activity do you engage in	Aerobic exercise, strength training
How many hours of sleep do you get on average per night	Seven to nine hours
How would you rate your stress levels	Moderate
How often do you engage in any stress-reducing techniques	Occasionally
During the past 6 months, on average, about how many alcoholic drinks did you have per month	One to three per month
Do you smoke or use tobacco products	No
Have you recently experienced significant stress or mental health issues	No

SAMPLE

Cardio Genetics - Summary

Genetics Summary

Lipoprotein



This figure depicts the structure of a lipoprotein, the molecular carrier that transports cholesterol and triglycerides through the bloodstream. The balance and types of lipoproteins are crucial indicators of cardiovascular health. Genetic variations influencing apolipoproteins, lipid metabolism enzymes, or lipid transport pathways can alter lipid profiles, contributing to dyslipidemia (abnormal blood lipids) and atherosclerotic cardiovascular disease (ASCVD) risk.

Chylomicron/Triglycerides (Half life: 5 - 15 mins)

Lipoproteins carrying dietary fats; triglycerides are their main lipid component.

Genes increasing risk: ANGPTL3, ANGPTL4, LPL, GPIHBP1, APOA5, SCD1

LDL (Half life: 2 - 4 days)

Transports cholesterol to tissues; contains APOB-100.

Genes increasing risk: LDLR, LDLRAP1, ANGPTL4, APOB

HDL (Half life: 3 - 5 days)

Removes cholesterol from tissues to the liver; contains APOA1, APOA2.

Genes increasing risk: APOA1, SCARB1, CETP, APOA2

Lp(a) (Half life: 3 - 4 days)

LDL-like particle with APOB-100 and apo(a), prothrombotic.

Genes increasing risk: LPA, APOB

oxLDL (Half life: ~ 2 - 4 days)

LDL modified by oxidative stress, highly atherogenic.

Genes increasing risk: APOB, PON1

Macrophages (Half life: Weeks to months)

Immune cells that engulf oxLDL, forming foam cells in plaques.

Genes increasing risk: MPO

Platelets (Clotting Risk) (Half life: 7 - 10 days)

Blood cells that form clots; hyperactive in atherosclerosis.

Genes increasing risk: Prothrombin

Foam Cells (Half life: Weeks to months)

Macrophages filled with oxLDL, key in plaque formation.

Genes increasing risk: *None*

SAMPLE

Cardio Genetics - Summary

Marker SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Chylomicron/ Triglycerides	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1748195	ANGPTL3	Altered serum triglycerides concentrations	⊕⊖ C/G	Elevated	G/G

The ANGPTL3 gene encodes Angiopoietin-like protein 3 which is a secretory protein regulating plasma lipid levels via affecting lipoprotein lipase- and endothelial lipase-mediated hydrolysis of triglycerides and phospholipids. Mutation in the gene can alter serum lipid concentrations. Polymorphisms in the ANGPTL3 is seen to affect plasma triglycerides which can give rise to the risk of hypertriglyceridemia (elevated triglycerides). Thus, a mutation in this gene can increase the levels of triglycerides in the plasma and the risk of hypertriglyceridemia. Individuals with CG genotype have higher plasma triglyceride levels associated with the increased risk of hypertriglyceridemia.

rs264	LPL	Impairs triglyceride metabolism	⊕⊖ A/G	Elevated	G/G

LPL, which encodes lipoprotein lipase plays a role in triglyceride metabolism in different tissues and represents a well-known multilocus for coronary artery disease (CAD). This mutation decreases the gene activity and impairs triglyceride metabolism leading to CAD. Individuals with AG genotype have high levels of triglycerides and high risk for coronary artery disease (CAD).

rs320	LPL	Altered lipoprotein metabolism	⊕⊖ G/T	Elevated	T/T

The LPL gene encodes for lipoprotein lipase (LPL) which is a key enzyme in lipoprotein metabolism. The enzyme hydrolyzes triglycerides from very-low-density lipoproteins and separates lipoproteins from the circulation. Genetic variants of the LPL gene have been associated with plasma lipoprotein levels. Mutations in the gene can lead to alterations in lipoprotein metabolism by affecting the LPL gene's expression which is one of the main etiologic mechanisms of diabetic dyslipidemia. Individuals with TG genotype have altered LPL gene expression that increases the risk of diabetic dyslipidemia.

rs72691625	GPIHBP1	Increased triglyceride levels	⊕⊕ A/A	Elevated	G/G

The GPIHBP1 gene encodes glycosylphosphatidylinositol-anchored high-density lipoprotein (HDL) binding protein, which is crucial for lipid metabolism and the transport of triglyceride-rich lipoproteins. GPIHBP1 plays a key role in facilitating the uptake of triglycerides by endothelial cells and the subsequent breakdown of these triglycerides. Mutations in the GPIHBP1 gene can disrupt this process, leading to impaired clearance of triglyceride-rich lipoproteins from the bloodstream. As a result, this disruption causes elevated triglyceride levels, a condition known as hypertriglyceridemia. Elevated triglycerides are associated with an increased risk of cardiovascular diseases, including coronary artery disease and atherosclerosis. Individuals with AA genotype exhibit impaired clearance of triglyceride-rich lipoproteins from the bloodstream, leading to elevated triglyceride levels and resulting in hypertriglyceridemia.

rs2075291	APOA5	Increased plasma triglyceride levels	⊕⊕ A/A	Elevated	G/G, C/C

The APOA5 gene encodes a protein that regulates plasma triglyceride levels by activating lipoprotein lipase, a key enzyme in triglyceride breakdown. Mutations in APOA5 lead to reduced gene expression, impairing its function and resulting in elevated plasma triglyceride levels. Excess cholesterol in the blood accumulates in the arterial walls, causing narrowing of the arteries and reducing blood flow to the heart muscle. This blockage can lead to coronary artery disease (CAD). Therefore, mutations in APOA5 can increase the risk of developing CAD by contributing to hypertriglyceridemia. Individuals with TT genotype have elevated triglyceride levels associated with an increased risk of developing CAD.

SAMPLE

Cardio Genetics - Summary

Marker SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Chylomicron/ Triglycerides	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs3135506	APOA5	Elevated plasma triglyceride levels	⊕⊖C/G	Partially elevated	C/C

The APOA5 gene encodes apolipoprotein A-V, a key regulator of plasma triglyceride levels, which are a major risk factor for coronary artery disease (CAD). APOA5 facilitates the breakdown of triglyceride-rich lipoproteins by activating lipoprotein lipase, a crucial enzyme in triglyceride metabolism. Mutations in the APOA5 gene can lead to decreased expression, impairing its normal function and causing elevated plasma triglyceride levels. High triglyceride levels contribute to the formation of small, dense low-density lipoprotein (LDL) particles, which are more likely to infiltrate arterial walls, leading to the development of atherosclerotic plaques. This process exacerbates arterial narrowing and significantly increases the risk of CAD. Individuals with the CG genotype exhibit reduced APOA5 gene expression and impaired triglyceride metabolism. This leads to elevated triglyceride levels, which in turn increases the risk of coronary artery disease (CAD).

rs508384	SCD1	Higher plasma apoB-48 levels	⊕⊖A/C	Partially elevated	C/C

The SCD1 gene encodes the stearoyl-CoA desaturase-1 enzyme, which is involved in lipogenesis and fuel metabolism, specifically catalyzing the conversion of saturated fatty acids into monounsaturated fatty acids. In the heart, SCD1 is crucial for maintaining lipid homeostasis, which is essential for normal cardiac function. Mutations in the SCD1 gene, including polymorphisms like rs508384, are associated with an increased risk of cardiovascular disease (CVD). These mutations can impair the enzyme's function, leading to elevated plasma levels of apolipoprotein B-48 (apoB-48), an independent risk factor for CVD. Elevated apoB-48 levels are linked to an adverse lipid profile, insulin resistance, and increased oxidative stress, all of which contribute to the development of cardiovascular disease. Individuals with the CA genotype exhibit elevated plasma apoB-48 levels associated with increased systolic blood pressure, small-dense LDL frequency, triglycerides, and oxidatively modified LDL.

rs2167444	SCD1	Higher plasma apoB-48 levels	⊕⊖A/T	Partially elevated	T/T

The SCD1 gene encodes the stearoyl-CoA desaturase-1 enzyme, which is involved in lipogenesis and fuel metabolism, specifically catalyzing the conversion of saturated fatty acids into monounsaturated fatty acids. In the heart, SCD1 is crucial for maintaining lipid homeostasis, essential for normal cardiac function. Mutations in the SCD1 gene, including polymorphisms like rs2167444, are associated with an increased risk of cardiovascular disease (CVD). These mutations can impair the enzyme's function, leading to elevated plasma levels of apolipoprotein B-48 (apoB-48), an independent risk factor for CVD. Elevated apoB-48 levels are linked to an adverse lipid profile, insulin resistance, and increased oxidative stress, all of which contribute to the development of cardiovascular disease. High plasma apoB-48 levels were associated with increased systolic blood pressure, small-dense LDL frequency, triglycerides, and oxidatively modified LDL. Individuals with the TA genotype exhibit elevated plasma apoB-48 levels associated with increased systolic blood pressure, small-dense LDL frequency, triglycerides, and oxidatively modified LDL.

LDL	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs693	APOB	High levels of APOB, TG, TC and LDL-C, and lower levels of HDL-C	⊕⊕T/T	Elevated	C/C

APOB gene encodes apolipoprotein that carries fats and fat-like substances (such as cholesterol) in the blood. It allows LDLs to attach to specific receptors on the surface of cells, particularly in the liver. Once attached, the receptors transport LDLs into the cell, where they are broken down to release cholesterol. The cholesterol is then used by the cell, stored, or removed from the body. Mutations in this gene results in the synthesis of dysfunctional protein resulting in the buildup of LDL in the bloodstream thereby increasing the risk of heart disease and stroke. Susceptible individuals with this polymorphism have a higher risk of ischemic stroke. Individuals with TT genotype exhibit high levels of APOB, TG, TC, and LDL-C, and lower levels of HDL-C.

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Cardio Genetics - Summary

Marker SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

LDL	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs515135	APOB	Elevated LDL cholesterol level	⊕⊕ G/G	Elevated	A/A

The APOB gene encodes apolipoprotein B, a protein essential for the formation of lipoproteins that transport fats and cholesterol in the blood. Apolipoprotein B allows low-density lipoproteins (LDLs) to bind to specific receptors on cell surfaces, including those in the liver, facilitating the uptake and breakdown of LDLs. Mutations in the APOB gene can disrupt this binding process, preventing effective LDL removal from the blood. As a result, LDL accumulates, leading to high LDL cholesterol levels. Excess cholesterol deposits in arterial walls, particularly in coronary arteries, increase the risk of heart attack. Individuals with GG genotype have increased risk of elevated LDL cholesterol and coronary artery disease.

rs72658855	LDLR	Accumulation of LDL-cholesterol molecules	⊕⊕ C/C	Elevated	T/T, C/T

The LDLR gene encodes proteins that form receptors for LDL on the cell membranes. LDL binds to LDL receptors and moves into the cell via endocytosis. LDL receptors are required to remove LDL from the bloodstream and digest them in the lysosomes. Mutations in the LDLR gene decrease the number of LDL receptors on cell membrane resulting in the accumulation of LDL-cholesterol molecules in the blood. Individuals with CC genotype have altered gene function resulting in high levels of LDL-cholesterol.

rs12071264	LDLRAP1	Increased levels of LDL-cholesterol	⊕⊕ A/A	Elevated	G/G, A/G

LDLRAP1 gene encodes a protein that helps remove low-density lipoproteins (LDLs) from the bloodstream. The LDLRAP1 protein interacts with a protein called, the LDL receptor. This receptor attaches (binds) to LDL. The receptor is present on the outer surface of cells, where it picks up LDLs circulating in the bloodstream. The LDLRAP1 protein plays a critical role in moving this receptor, together with its attached LDL, from the cell surface to the interior of the cell. LDLRAP1 protein is particularly important in the liver, which is the organ responsible for clearing most excess cholesterol (LDL) from the body. A mutation in the LDLRAP1 gene may alter its function leading to increased levels of LDL-cholesterol in the blood. Individuals with AA genotype have altered gene function resulting in high levels of LDL-cholesterol.

rs4076317	ANGPTL4	Higher LDL and total cholesterol levels	⊕⊖ C/G	Elevated	G/G

The ANGPTL4 gene encodes Angiopoietin-like protein 4, which plays a crucial role in lipid metabolism by inhibiting lipoprotein lipase (LPL). In the heart, ANGPTL4 influences lipid regulation, directly affecting cardiovascular health. Mutations in ANGPTL4 disrupt this regulatory function, leading to impaired lipid metabolism and elevated levels of low-density lipoprotein cholesterol (LDL-C) and total cholesterol. These increased lipid levels contribute to a higher risk of coronary artery disease. Thus, mutations in ANGPTL4 are linked to altered lipid profiles and increased cardiovascular risk. Individuals with CG genotype have higher TC and LDL-C levels and an increased risk of coronary artery disease risk.

HDL	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs5888	SCARB1	Altered lipid metabolism	⊕⊕ T/T	Elevated	C/C

The SCARB1 gene encodes the scavenger receptor class B member 1 (SR-B1), a plasma membrane receptor facilitating cholesterol transfer to and from high-density lipoprotein (HDL). This protein plays a critical role in maintaining lipid homeostasis. Mutations in SCARB1 can impair cholesterol uptake and efflux, leading to dysregulated lipid metabolism without necessarily altering overall lipid levels. These mutations may act as early markers of diabetic dyslipidemia, signaling an imbalance in lipid handling that contributes to metabolic dysfunction associated with diabetes. Early detection of such mutations could aid in managing diabetic complications. Individuals with TT genotype may have altered lipid metabolism which may be associated with higher risk of diabetic dyslipidemia.

SAMPLE

Cardio Genetics - Summary

Marker SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

HDL	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs247616	CETP	Disrupted lipid transport	⊕⊖C/T	Elevated	T/T

The CETP gene encodes cholesteryl ester transfer protein, which facilitates the exchange of cholesteryl esters and triglycerides between high-density lipoproteins (HDL) and low-density lipoproteins (LDL). This process is essential for maintaining lipid balance in the bloodstream. Mutations in CETP can impair its function, leading to ineffective lipid exchange. This dysfunction does not necessarily result in elevated HDL levels but disrupts normal lipid transport, promoting the accumulation of atherogenic LDL particles and contributing to plaque formation in arteries. Consequently, impaired CETP function is associated with an increased risk of coronary artery disease (CAD). Individuals with CT genotype may have increased CETP gene activity associated with a higher risk of atherosclerotic lesions and coronary artery disease (CAD).

rs708272	CETP	Altered lipid metabolism	⊕⊖A/G	Elevated	G/G

The CETP gene encodes cholesteryl ester transfer protein, which facilitates the exchange of cholesteryl esters and triglycerides between high-density lipoproteins (HDL) and low-density lipoproteins (LDL). This process is essential for maintaining lipid balance in the bloodstream. Mutations in CETP can impair its function, leading to ineffective lipid exchange. This dysfunction does not necessarily result in elevated HDL levels but disrupts normal lipid transport, promoting the accumulation of atherogenic LDL particles and contributing to plaque formation in arteries. Consequently, impaired CETP function is associated with an increased risk of coronary artery disease (CAD). Individuals with GA genotype may have altered lipid metabolism associated with high risk of diabetic dyslipidemia.

rs632153	APOA1	Elevated TC, LDLC, TG and VLDL levels	⊕⊕T/T	Elevated	G/G

The APOA1 gene encodes apolipoprotein A-I, the primary protein component of high-density lipoprotein (HDL) in plasma. It plays a critical role in regulating lipoprotein metabolism and maintaining cholesterol homeostasis by facilitating cholesterol efflux and HDL formation. Mutations in the APOA1 gene can disrupt this process, leading to imbalances in lipid concentrations. Such alterations are associated with elevated plasma triglycerides and altered low-density lipoprotein (LDL) levels, contributing to dyslipidemia. These genetic variations can impact total cholesterol (TC), LDL cholesterol (LDLC), triglycerides (TG), and very-low-density lipoprotein cholesterol (VLDL) levels, increasing the risk of cardiovascular diseases. Individuals with TT genotype have elevated TC, LDLC, TG and VLDL levels leading to the increased risk of dyslipidemia.

rs5082	APOA2	Higher total cholesterol, triglyceride, Cholesterol/HDLc ratio and non-HDL cholesterol levels	⊕⊖C/T	Partially elevated	C/C

The APOA2 gene encodes apolipoprotein A-II, the second most abundant protein in high-density lipoproteins (HDL-C), and plays a role in lipid metabolism. While its exact function remains unclear, APOA2 is implicated in modulating lipid levels. Mutations in the APOA2 gene are associated with diabetic dyslipidemia, characterized by lower HDL levels and elevated total cholesterol, triglycerides, cholesterol/HDL-C ratio, and non-HDL cholesterol levels. These alterations contribute to an increased risk of cardiovascular complications, as the lipid imbalances promote atherogenic changes and metabolic disturbances. Individuals with CT genotype have altered gene function associated with the risk of diabetic dyslipidemia.

Lp(a)	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs10455872	LPA	Increased lipoprotein(a) levels	⊕⊖A/G	Elevated	A/A

The LPA gene encodes apolipoprotein(a), a key component of the lipoprotein(a) [Lp(a)] particle, which is structurally similar to low-density lipoprotein (LDL) and is involved in cholesterol transport. Elevated levels of Lp(a) are linked to an increased risk of coronary artery disease, carotid atherosclerosis, and stroke. Variants in the LPA gene can lead to higher levels of Lp(a), contributing to elevated LDL cholesterol levels, disrupting lipid metabolism, and increasing the risk of coronary artery disease. Individuals with AG genotype have higher levels of Lp(a), which contributes to elevated LDL cholesterol, disrupts lipid metabolism, and increases the risk of coronary artery disease (CAD).

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Cardio Genetics - Summary

Marker SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Lp(a)	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs3798221	LPA	Increased lipoprotein(a) levels	⊕⊖ G/T	Elevated	T/T

The LPA gene encodes apolipoprotein(a), a key component of lipoprotein(a) [Lp(a)], which is structurally similar to low-density lipoprotein (LDL) and plays a role in cholesterol transport. Elevated Lp(a) levels are associated with an increased risk of coronary artery disease, calcific aortic valve disease (CAVD), and stroke. Variants in the LPA gene can lead to higher Lp(a) levels, increasing the risk of these conditions by promoting lipid deposition and calcification in blood vessels and heart valves. Individuals with GT genotype have higher levels of Lp(a) and altered lipid metabolism, which increases the risk of coronary artery disease (CAD) and calcific aortic valve disease (CAVD).

rs693	APOB	High levels of APOB, TG, TC and LDL-C, and lower levels of HDL-C	⊕⊕ T/T	Elevated	C/C

APOB gene encodes apolipoprotein that carries fats and fat-like substances (such as cholesterol) in the blood. It allows LDLs to attach to specific receptors on the surface of cells, particularly in the liver. Once attached, the receptors transport LDLs into the cell, where they are broken down to release cholesterol. The cholesterol is then used by the cell, stored, or removed from the body. Mutations in this gene results in the synthesis of dysfunctional protein resulting in the buildup of LDL in the bloodstream thereby increasing the risk of heart disease and stroke. Susceptible individuals with this polymorphism have a higher risk of ischemic stroke. Individuals with TT genotype exhibit high levels of APOB, TG, TC, and LDL-C, and lower levels of HDL-C.

rs515135	APOB	Elevated LDL cholesterol level	⊕⊕ G/G	Elevated	A/A

The APOB gene encodes apolipoprotein B, a protein essential for the formation of lipoproteins that transport fats and cholesterol in the blood. Apolipoprotein B allows low-density lipoproteins (LDLs) to bind to specific receptors on cell surfaces, including those in the liver, facilitating the uptake and breakdown of LDLs. Mutations in the APOB gene can disrupt this binding process, preventing effective LDL removal from the blood. As a result, LDL accumulates, leading to high LDL cholesterol levels. Excess cholesterol deposits in arterial walls, particularly in coronary arteries, increase the risk of heart attack. Individuals with GG genotype have increased risk of elevated LDL cholesterol and coronary artery disease.

oxLDL	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs662	PON1	High LDL oxidation	⊕⊕ G/G	Elevated	A/A

The PON1 gene encodes the enzyme paraoxonase 1, which is primarily associated with high-density lipoprotein (HDL) particles in the bloodstream. This enzyme plays a critical role in cardiac health by preventing the oxidation of low-density lipoprotein (LDL), thereby reducing oxidative stress and protecting against atherosclerosis, the buildup of fatty deposits in the arteries. PON1 helps maintain the balance between HDL and LDL, essential for cardiovascular health. Reduced levels of PON1 compromise its antioxidant function, leading to increased LDL oxidation. This promotes atherosclerosis, elevating the risk of coronary artery disease (CAD). Individuals with GG genotype may have reduced PON1 levels and are associated with an increased risk of CAD.

SAMPLE

Cardio Genetics - Summary

Marker SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Macrophages	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs2107545	MPO	Oxidative stress	⊕⊕ C/C	Elevated	T/T

The MPO gene encodes myeloperoxidase (MPO), an enzyme produced by activated leukocytes like neutrophils and monocytes, which contributes to oxidative stress. MPO catalyzes the formation of hypochlorous acid from hydrogen peroxide and chloride ions, generating reactive species that can oxidize low-density lipoprotein (LDL) into oxidized LDL (oxLDL). OxLDL is a key player in inflammation and atherosclerosis. Increased MPO levels are associated with elevated oxLDL and have been linked to the progression of Type 2 diabetes mellitus (T2DM) and coronary artery stenosis. Variants of the MPO gene are correlated with higher plasma MPO levels, which are associated with an increased risk of carotid plaque and atherosclerosis. Consequently, individuals with certain MPO gene variants have a heightened risk of T2DM, carotid plaque formation, and atherosclerosis. Individuals with the CC genotype may have high oxidative stress that increases the risk of T2DM. They also have a high risk of carotid plaque formation and atherosclerosis susceptibility.

Platelets(Clotting risk)	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1799963	Prothrombin	Higher levels of plasma prothrombin	⊕⊖ A/G	Partially elevated	G/G

The Factor II (F2) gene provides instructions for producing prothrombin, also known as coagulation factor II, a central regulator of the coagulation cascade. Its active form, thrombin, functions in both procoagulant and anticoagulant roles. Mutations in the F2 gene can lead to elevated plasma prothrombin levels, promoting excessive thrombin generation. This increase heightens the risk of venous thrombosis by facilitating abnormal blood clot formation. Individuals with AA genotype have an higher level of plasma prothrombin leading to increased risk for developing venous thrombosis.

SAMPLE

Genetics Summary

ENDOTHELIAL DYSFUNCTION

Nitric Oxide Synthesis
Regulates vessel dilation and endothelial function; disruption increases hypertension risk.

Genes increasing risk: *None*

Vascular Health
Encompasses vessel integrity and smooth muscle function.

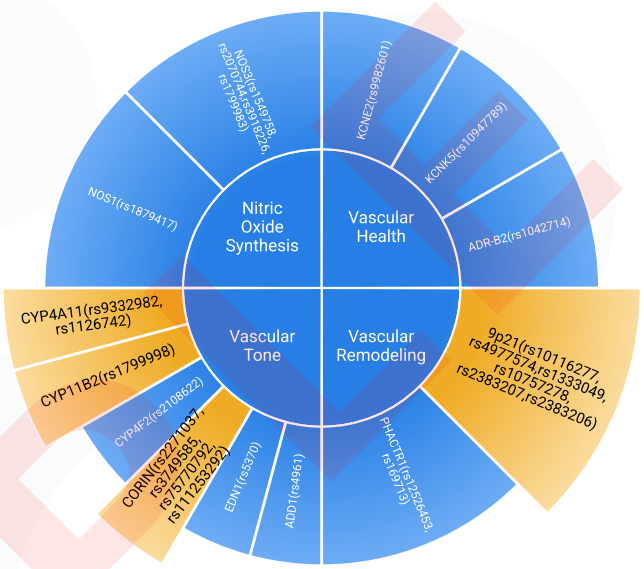
Genes increasing risk: *None*

Vascular Remodeling
Structural changes in vessels due to genetic effects on growth and stiffness.

Genes increasing risk: 9p21

Vascular Tone
Controls vessel contraction/relaxation via smooth muscle genes.

Genes increasing risk: CYP11B2,CYP4A11,CORIN



ATRIAL FIBRILLATION & OTHER

Atrial Fibrillation
Genetic variants affect heart rhythm and atrial structure, raising AFib risk.

Genes increasing risk: 4q25

Angiogenesis
Genetic control of new blood vessel growth; impacts healing and ischemia.

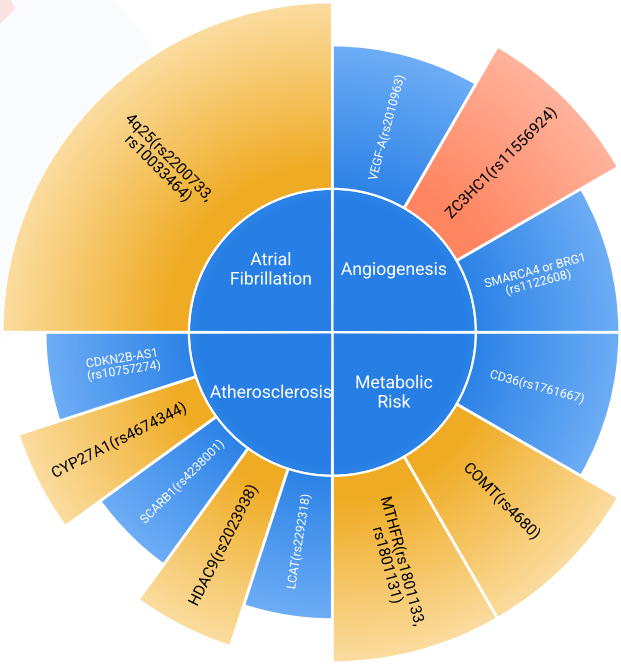
Genes increasing risk: ZC3HC1

Metabolic Risk
Involves genes linked to insulin resistance, lipid metabolism, and obesity.

Genes increasing risk: MTHFR,COMT

Atherosclerosis
Genetic factors influence plaque formation, cholesterol handling, and inflammation.

Genes increasing risk: CYP27A1,HDAC9



SAMPLE

Cardio Genetics - Summary

Systemic SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Vascular remodeling	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1333049	9p21	Coronary artery disease	⊕⊖ C/G	Partially elevated	G/G

Extensive genome-wide association studies (GWAS) have identified a large number of common genetic variants leading to cardiovascular diseases (CVD). Of these variants, the chromosome 9p21 locus was the first to be discovered. This variant is known to have the largest individual effect and is considered the most widely replicated genetic risk factor for CVD. Recent GWAS have shown associations between 9p21 and coronary artery disease (CAD). The nearest genes to this locus are CDKN2B and CDKN2A. As these genes are key regulators of the cell cycle, it is hypothesized that they might play a role in the pathogenesis of CAD via reduced re-growth of arterial intimal cells which is implicated in the development of atherosclerosis. As a result, a polymorphism associated with this locus increases the risk of CAD in susceptible individuals. Individuals with CG genotypes may have a risk of developing coronary artery disease.

Vascular tone	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs3749585	CORIN	Altered blood pressure	⊕⊖ C/T	Partially elevated	T/T

The CORIN gene encodes for a member of the type II transmembrane serine protease class of the trypsin superfamily. It controls blood pressure. The encoded protein converts pro-atrial natriuretic peptide (peptide that regulates blood pressure) to biologically active atrial natriuretic peptide, a cardiac hormone that regulates blood volume and pressure. Although the actual mechanism is unclear, mutations in the CORIN gene can affect its function and give rise to the risk of hypertension. Individuals with TC genotype have impaired gene function and are associated with an increased risk of hypertension.

rs9332982	CYP4A11	Elevated blood pressure	⊕⊖ C/T	Partially elevated	C/C

The CYP4A11 gene produces an enzyme that plays a key role in regulating blood pressure in the kidneys by creating a substance called 20-HETE. This substance can either constrict blood vessels, raise blood pressure, or promote the excretion of excess salt, lowering blood pressure. However, mutations in the CYP4A11 gene can disrupt the balance of 20-HETE, impairing blood pressure regulation and increasing the risk of hypertension. Individuals with CT genotype exhibit a disrupted balance of 20-HETE, impairing blood pressure regulation and increasing the risk of hypertension.

rs1799998	CYP11B2	Altered blood pressure	⊕⊖ C/T	Partially elevated	C/C

The CYP11B2 gene encodes an enzyme called aldosterone synthase, which is located in the adrenal glands on top of the kidneys. Aldosterone synthase is part of the cytochrome P450 family of enzymes and is responsible for producing aldosterone, a hormone that helps control blood pressure by regulating salt and fluid levels in the body. Mutations in the CYP11B2 gene can affect the production of aldosterone, increasing the risk of hypertension. Heterozygous (partially abnormal) individuals may have altered aldosterone levels and are associated with an elevated risk of hypertension.

Atrial fibrillation	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs2200733	4q25	Atrial fibrillation	⊕⊖ C/T	Partially elevated	C/C

Extensive genome-wide association studies (GWAS) have identified several genetic variants that increase the risk of atrial fibrillation. Among these variants, the most arrhythmogenic are those located on chromosome 4q25. Several studies have hypothesized that the homeodomain transcription factor Pitx2 (regulatory protein), located near the 4q25 variants, acts as a molecular link between these risk variants and atrial fibrillation. Although the exact mechanism remains to be elucidated, variation in the 4q25 locus increases the risk of developing atrial fibrillation in susceptible individuals. Individuals with the CT genotype may have a risk of developing atrial fibrillation.

SAMPLE

Cardio Genetics - Summary

Systemic SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Angiogenesis	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs11556924	ZC3HC1	Endothelial dysfunction	⊕⊕ C/C	Elevated	T/T

Nuclear-interacting partner of ALK (NIPA), also known as zinc finger C3HC-type protein 1 (ZC3HC1), is a protein that in humans is encoded by the ZC3HC1 gene. ZC3HC1 gene mediates angiogenesis which plays an important role in the regulation of endothelial integrity (inside of blood vessels and lymphatic vessels) and inflammation. The mutation may alter the stability and functional properties of ZC3HC1 protein causing endothelial dysfunction and, in the long run, may lead to coronary artery disease (CAD). It causes increased expression with lower rates of cell growth supporting the role of cell proliferation in atherosclerosis and its clinical consequences. Individuals with the CC genotype who have decreased gene activity have increased risk for coronary artery disease (CAD).

Metabolic Risk	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1801133	MTHFR	Active folate deficiency	⊕⊖ C/T	Partially elevated	C/C

The MTHFR gene produces an enzyme that converts folate into its active form, 5-methyltetrahydrofolate, essential for DNA methylation and gene regulation. Mutations reduce enzyme activity, lowering folate levels and impairing methylation, which can elevate homocysteine and trigger IL-6-driven inflammation. Individuals with CT genotype are associated with impaired methylation.

rs1801131	MTHFR	Active folate deficiency	⊕⊖ A/C	Partially elevated	A/A

The MTHFR gene produces an enzyme that converts folate into its active form, 5-methyltetrahydrofolate, essential for DNA methylation and gene regulation. Mutations reduce enzyme activity, lowering folate levels and impairing methylation, which can elevate homocysteine and trigger IL-6-driven inflammation. Individuals with AC genotype are associated with impaired methylation.

rs4680	COMT	Optimum COMT gene activity	⊕⊖ A/G	Partially elevated	G/G

The COMT gene provides instructions for making an enzyme called catechol-O-methyltransferase. This enzyme is crucial for the degradation of catecholamine neurotransmitters and the systemic elimination of catechol estrogens. It also plays a role in DNA repair and the modulation of estrogen-induced carcinogenesis. By facilitating the detoxification and elimination of catechol estrogens and regulating catecholamine levels, COMT helps maintain cardiovascular and renal function. A common functional polymorphism, rs4680 (Val158Met), alters enzyme activity which has been linked to impaired methylation, altered vascular tone, and elevated oxidative stress. Thepolymorphism is associated with a significantly increased risk of coronary artery disease (CAD), possibly through interactions with homocysteine levels, estrogen metabolism, and lifestyle factors like alcohol consumption. Individuals with GA genotypes may have impaired COMT function and a increased risk to coronary artery disease (CAD).

Atherosclerosis	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs2023938	HDAC9	Vascular inflammation	⊕⊖ A/G	Partially elevated	A/A

Histone deacetylase 9 is an enzyme that in humans is encoded by the HDAC9 gene. HDAC9 plays an essential role in diverse physiological processes including cardiac muscle development, bone formation, adipocyte differentiation, and innate immunity. Mutations in HDAC9 can disrupt its normal function, leading to altered gene expression patterns that contribute to the pathogenesis of coronary artery disease (CAD). Specifically, dysfunctional HDAC9 may promote vascular inflammation and smooth muscle cell proliferation, both of which are critical in the development of atherosclerosis. Additionally, the impaired regulation of genes involved in lipid metabolism and endothelial function can further exacerbate CAD risk Individuals with AG genotype have increased gene activity increasing the risk for coronary artery disease (CAD).

SAMPLE

Cardio Genetics - Summary

Systemic SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Atherosclerosis	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs4674344	CYP27A1	Plaque deposition	⊕⊖A/T	Partially elevated	A/A

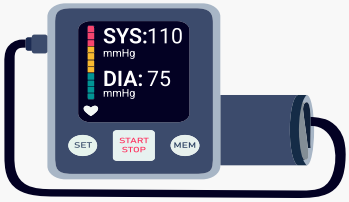
The CYP27A1 gene encodes a mitochondrial enzyme involved in cholesterol metabolism, expressed in various tissues. In the liver, CYP27A1 catalyzes the conversion of cholesterol to 27-hydroxycholesterol (27-HC), a key intermediate in bile acid synthesis. Elevated levels of 27-HC can promote atherosclerotic plaque formation, contributing to the narrowing of arteries. This arterial narrowing impairs blood flow and can lead to increased blood pressure, resulting in hypertension. Therefore, mutations in the CYP27A1 gene can disrupt normal cholesterol metabolism and elevate the risk of developing hypertension. Individuals with AT genotype may have a risk of developing hypertension.

SAMPLE

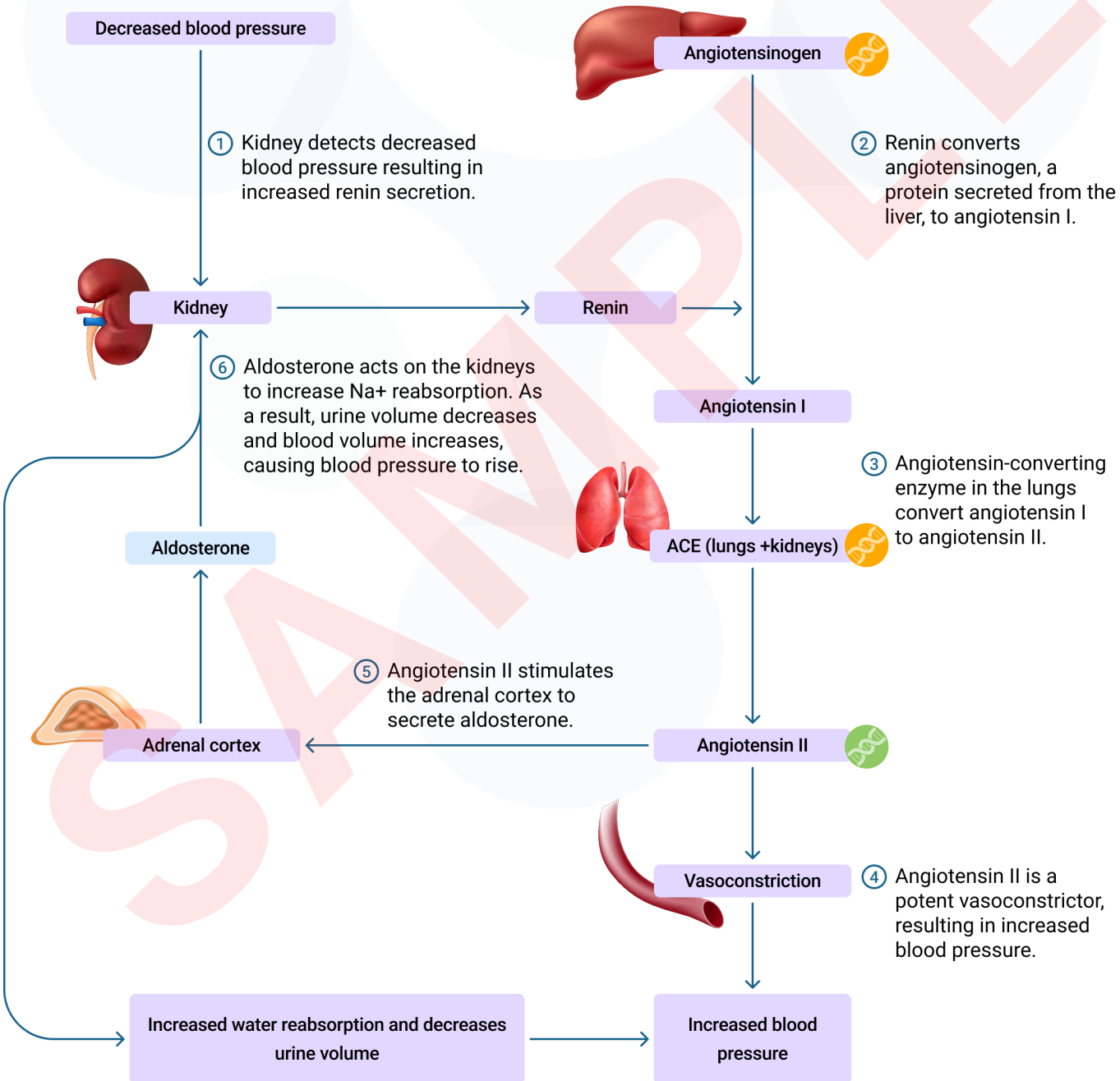
Cardio Genetics - Summary

Hypertension Risk Genetics

Renin-Angiotensin-Aldosterone System



Blood Pressure	
Systolic: 110	Diastolic: 75



This figure illustrates the Renin–Angiotensin–Aldosterone System (RAAS), a key hormonal pathway that maintains blood pressure and fluid balance. Genetic variations affecting components of the RAAS (such as ACE, CYP, or AGTR1 genes) can influence an individual's tendency toward hypertension (high blood pressure) and their responsiveness to medications targeting this pathway.

SAMPLE

Cardio Genetics - Summary

Hypertension

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Angiotensin Converting Enzyme	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs4646994	ACE	Altered blood pressure	⊕⊖ I/D	Partially elevated	I/I

The ACE gene encodes an enzyme involved in blood pressure regulation and electrolyte balance. It catalyzes the conversion of angiotensin I into a physiologically active peptide angiotensin II. Angiotensin II is a potent vasopressor and aldosterone-stimulating peptide that controls blood pressure and fluid-electrolyte balance. The ACE gene polymorphism results in increased ACE activity and altered blood pressure which might be associated with the atherosclerotic process and consequently the increased risk of coronary artery disease (CAD). Individuals with the ID genotype may have increased ACE and are associated with an elevated risk of CAD.

Angiotensinogen	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs5051	AGT	Higher angiotensin II levels	⊕⊖ A/G	Partially elevated	G/G

The AGT gene provides instructions for making a protein called angiotensinogen. This protein is part of the renin-angiotensin system, which regulates blood pressure and the balance of fluids and salts in the body. The angiotensin-converting enzyme (ACE) converts Angiotensin I to angiotensin II. Angiotensin II causes blood vessels to narrow (constrict), which results in increased blood pressure. A mutation in the AGT gene alters its function and may elevate blood pressure through increased production of Angiotensin II resulting in hypertension, endothelial dysfunction, and vascular inflammation, contributing to the development of coronary artery disease (CAD) by accelerating atherosclerosis. Individuals with the GA genotype have higher angiotensin II levels resulting in coronary artery disease (CAD) risk.

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Cardio Genetics - Summary

Genetics Summary

DRUG AND SUPPLEMENT METABOLISM

Metformin
Genes increasing risk: OCT2 gene

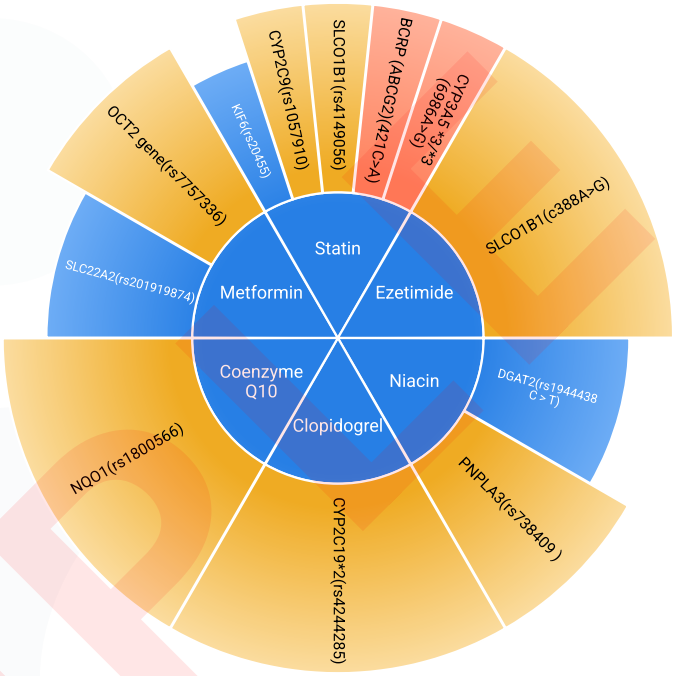
Statin
Genes increasing risk: CYP3A5 *3/*3,BCRP (ABCG2), SLCO1B1,CYP2C9,CYP2C9

Ezetimide
Genes increasing risk: SLCO1B1

Niacin
Genes increasing risk: PNPLA3

Clopidogrel
Genes increasing risk: CYP2C19*2

Coenzyme Q10
Genes increasing risk: NQO1



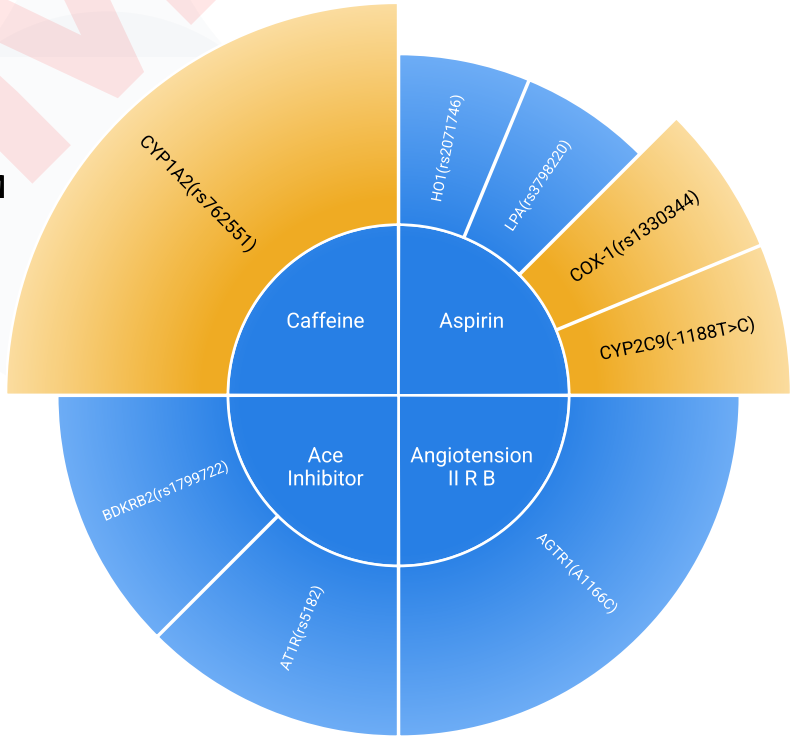
DRUG AND SUPPLEMENT METABOLISM

Caffeine
Genes increasing risk: CYP1A2

Aspirin
Genes increasing risk: COX-1,CYP2C9

Angiotension II R B
Genes increasing risk: *None*

Ace Inhibitor
Genes increasing risk: *None*



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Cardio Genetics - Summary

Drug Metabolism

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Metformin	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs7757336	OCT2 gene	Increased metformin concentration	⊕⊖ A/G	Partially elevated	A/A

The OCT2 gene, or organic cation transporter 2, encodes a membrane transporter protein that belongs to the solute carrier family and is primarily expressed in the kidney. The function of the OCT2 gene is to facilitate the uptake of organic cations, including metformin, from the blood into renal tubular cells. In metformin metabolism, OCT2 plays a crucial role in the renal elimination of the drug. The transporter actively transports metformin from the bloodstream into renal cells, allowing for subsequent excretion into the urine. The rs7757336 mutation in the OCT2 gene is associated with alterations in enzyme activity, impacting metformin metabolism. Individuals carrying this allele exhibit increased metformin concentrations in the body. The mutation leads to decreased OCT2 enzyme activity, resulting in reduced uptake of metformin by renal tubular cells. Consequently, metformin elimination is impaired, contributing to elevated plasma concentrations of the drug. Individuals with the AG genotype have impaired metformin elimination, contributing to elevated plasma metformin concentrations. Consult with healthcare professionals for personalized advice.

Statin	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
6986A>G	CYP3A5 *3/*3	Increased statin concentration in blood	⊕⊕ CYP3A5*3/*3	Elevated	CYP3A5*1/*1

CYP3A5 is a gene that encodes the enzyme cytochrome P450 3A. This enzyme is predominantly expressed in the liver and small intestine. It is responsible for metabolizing a broad spectrum of drugs, particularly statins, which are commonly prescribed to lower cholesterol levels and reduce the risk of cardiovascular diseases. The CYP3A5 421C>A mutation involves a single nucleotide change in the gene sequence, leading to a modification in enzyme activity. Individuals with this mutation often demonstrate decreased CYP3A5 enzyme activity. In the context of statin metabolism, the reduced enzyme activity can result in a slower and less efficient breakdown of statin drugs such as simvastatin in the body, consequently increasing the concentration of simvastatin in the bloodstream. This altered drug metabolism may elevate the risk of statin-related side effects, as the body is exposed to higher concentrations of the drug for an extended period. Individuals with *3/*3 genotype have decreased CYP3A5 enzyme activity resulting in a slower breakdown of statin drugs in the body. This leads to increased statin concentration in the blood. Consult with healthcare professionals for personalized advice.

421C>A	BCRP (ABCG2)	Increased statin concentration in blood	⊕⊕ A/A	Elevated	C/C

The Breast Cancer Resistance Protein (BCRP), also known as ATP-binding cassette sub-family G member 2 (ABCG2), is a gene that encodes a membrane transporter protein found in various tissues, including the liver and intestine. This protein plays a crucial role in drug metabolism and elimination by transporting a variety of substrates, including statins, across cell membranes. Statin drugs are commonly prescribed for managing cholesterol levels by inhibiting the enzyme HMG-CoA reductase, a key player in cholesterol synthesis. BCRP, being expressed in the liver and intestine, contributes to the elimination of statins from these tissues, thereby influencing their systemic availability. Mutations in the BCRP gene can lead to alterations in the structure or function of the BCRP protein. In the context of statin such as simvastatin metabolism, certain mutations may result in decreased BCRP enzyme activity. This reduced activity impairs the efficient removal of simvastatin from liver and intestinal cells, leading to increased concentrations of simvastatin in the systemic circulation. The consequence of elevated simvastatin levels in the bloodstream may manifest as an increased risk of simvastatin-related adverse effects. Individuals with AA genotype have increased BCRP enzyme activity resulting in an optimum breakdown of statin drugs in the body. This leads to lower statin concentrations in the blood. Consult with healthcare professionals for personalized advice.

SAMPLE

Cardio Genetics - Summary

Drug Metabolism

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Statin	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs4149056	SLCO1B1	Increased plasma statin concentration	⊕⊖C/T	Partially elevated	T/T

The SLCO1B1 gene encodes a protein known as organic anion transporting polypeptide 1B1 (OATP1B1), which plays a crucial role in the transportation of various substances, including drugs, across cell membranes in the liver. Specifically, OATP1B1 is responsible for the elimination and uptake of statin medications, such as fluvastatin, into hepatocytes, where these drugs exert their cholesterol-lowering effects. A noteworthy mutation in the SLCO1B1 gene is the T521C variant, which has been associated with alterations in fluvastatin metabolism. This genetic variation may impact the efficiency of OATP1B1 in transporting fluvastatin into liver cells, leading to changes in the drug's pharmacokinetics. Individuals carrying the SLCO1B1 T521C mutation may experience variations in the metabolism and clearance of fluvastatin, potentially influencing the drug's effectiveness and side effects. Reduced OATP1B1 uptake capacity at high fluvastatin concentrations may diminish the cholesterol-lowering effect, elevating plasma statin concentrations and the risk of muscle toxicity. Individuals with the TC genotype may experience reduced metabolism and clearance of statin, potentially increasing the statin concentration in the blood. Consult with healthcare professionals for personalized advice.

Niacin	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs738409	PNPLA3	Hindered niacin uptake	⊕⊖C/G	Partially elevated	C/C

The PNPLA3 gene, responsible for lipid metabolism, undergoes a mutation known as p.I148M, linked to increased fat accumulation in the liver. This specific mutation seems to affect niacin uptake, a form of Vitamin B3 known for reducing triglyceride synthesis in non-alcoholic fatty liver disease (NAFLD). In individuals with the PNPLA3 p.I148M mutation, there appears to be a diminished effectiveness of niacin in clearing fat. This suggests that the presence of this mutation hinders niacin uptake, potentially impacting its beneficial effects on lipid metabolism. Conversely, restoring the wild-type PNPLA3 protein in cells with the mutation improves niacin's efficacy, indicating the role of genetic factors in modulating niacin uptake and its impact on fat accumulation. Individuals with the CG genotype exhibit hindered niacin uptake, potentially impacting its beneficial effects on lipid metabolism. Consult with healthcare professionals for personalized advice.

Clopidogrel	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs4244285	CYP2C19*2	Clopidogrel resistance	⊕⊖A/G	Partially elevated	G/G

The CYP2C19*2 gene is a member of the cytochrome P450 superfamily, specifically the CYP2C subfamily. Its primary function lies in encoding an enzyme that plays a pivotal role in drug metabolism, particularly in the processing of various medications in the liver. In the context of clopidogrel resistance, the CYP2C192 gene assumes significance due to its involvement in the metabolism of clopidogrel, a widely used antiplatelet medication. Clopidogrel requires bioactivation in the liver to transform into its active form, which subsequently inhibits platelet aggregation. The CYP2C19 enzyme, encoded by the CYP2C192 gene, is crucial for this activation process. However, individuals carrying a mutation in the CYP2C192 gene exhibit altered enzymatic activity, leading to reduced or impaired conversion of clopidogrel into its active metabolite. This diminished bioactivation results in decreased efficacy of clopidogrel in inhibiting platelet aggregation, a phenomenon known as clopidogrel resistance. Consequently, individuals with the CYP2C192 mutation may experience suboptimal response to clopidogrel therapy, rendering the medication less effective in preventing platelet activation and potentially compromising its antiplatelet effects. Individuals with the GA genotype may exhibit altered enzymatic activity, leading to reduced or impaired conversion of clopidogrel into its active metabolite. This diminished bioactivation results in decreased efficacy of clopidogrel in inhibiting platelet aggregation leading to clopidogrel resistance Consult with healthcare professionals for personalized advice.

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Cardio Genetics - Summary

Drug Metabolism

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Coenzyme Q10	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1800566	NQO1	Hindered COQ10 uptake	⊕⊖P/S	Partially elevated	P/P

The NQO1 gene, responsible for encoding NAD(P)H:quinone oxidoreductase 1, plays a crucial role in the body's antioxidant defense system. NQO1 is instrumental in generating and maintaining the reduced state of ubiquinone in membrane systems and liposomes, contributing significantly to their antioxidant function. Among these ubiquinone, Coenzyme Q10 (CoQ10) stands out as a vital player, functioning in cellular energy production and providing antioxidant protection to the body. In the intricate landscape of cellular processes, NQO1 is key to the redox balance, particularly in preserving the reduced state of CoQ10. This partnership is pivotal for maintaining cellular health, as CoQ10's antioxidant properties are harnessed to counteract oxidative stress. Dysfunction in NQO1 could disrupt this delicate equilibrium, leading to potential alterations in CoQ10 metabolism. The NQO1P187S mutation, known as Pro187Ser SNP, has been under scrutiny for its potential influence on CoQ10 metabolism. Individuals with the P/S genotype may experience dysfunction in NQO1 activity leading to potential alterations in CoQ10 metabolism. This influences the dietary COQ10 status in the body. Consult with healthcare professionals for personalized advice.

Caffeine	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs762551	CYP1A2	Slow caffeine metabolizers	⊕⊖A/C	Partially elevated	A/A

The CYP1A2 gene encodes an enzyme that contributes to about 13% of liver cytochrome P450 activity and is crucial for metabolizing caffeine (1,3,7-trimethylxanthine) found in coffee. Individuals with normal CYP1A2 activity metabolize caffeine efficiently, which is linked to a lower risk of cardiovascular diseases (CVD) such as myocardial infarction and hypertension when consuming up to a cup of coffee per day. In contrast, genetic variants in CYP1A2 that result in lower enzyme activity lead to slower caffeine metabolism and are associated with an increased risk of CVD. Individuals with the AC genotype exhibit reduced enzyme activity, slower caffeine metabolism, and a higher risk of CVD. Consult with healthcare professionals for personalized advice.

Aspirin	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1330344	COX-1	Aspirin resistance	⊕⊖C/T	Partially elevated	T/T

The COX-1 (Cyclooxygenase-1) gene is a critical component in aspirin metabolism, playing a pivotal role in the synthesis of prostaglandins, which are essential for various physiological processes. Its primary function involves catalyzing the conversion of arachidonic acid into prostaglandin H2, a precursor to various prostanoids involved in inflammation and platelet aggregation. Within the context of aspirin metabolism, COX-1 is a target of aspirin's inhibitory action. Aspirin irreversibly acetylates a serine residue in the active site of COX-1, inhibiting its enzymatic activity. This inhibition disrupts the production of thromboxane A2, a potent platelet aggregator, thereby preventing excessive platelet activation and aggregation. The specific genetic variant of interest is COX-1 rs1330344 (-1676A>G). Mutations in this gene, particularly the G allele at position -1676, have been associated with aspirin resistance. Aspirin resistance refers to a reduced inhibitory effect of aspirin on platelet aggregation, leading to an increased risk of cardiovascular events. The mutation at rs1330344 may compromise the binding affinity of aspirin to COX-1, diminishing its inhibitory effect on the enzyme. Consequently, the normal balance of prostanoid production, particularly thromboxane A2, may be disrupted, allowing platelet aggregation to proceed despite aspirin treatment. This resistance to aspirin's antiplatelet effects poses a potential risk for adverse cardiovascular outcomes. Individuals with the TC genotype may exhibit reduced responsiveness to aspirin's antiplatelet effects, leading to aspirin resistance. Consult with healthcare professionals for personalized advice.

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Cardio Genetics - Summary

Drug Metabolism

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Aspirin	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
-1188T>C	CYP2C9	Slow metabolizers of aspirin	⊕⊖C/T	Partially elevated	T/T

The CYP2C9 gene encodes an enzyme belonging to the cytochrome P450 family, specifically CYP2C9. This enzyme plays a crucial role in drug metabolism, particularly in the liver, where it is involved in the processing of various medications, including nonsteroidal anti-inflammatory drugs (NSAIDs) such as aspirin. In aspirin metabolism, CYP2C9 is responsible for the conversion of aspirin to its active form. The enzyme facilitates the breakdown of aspirin into salicylic acid, which exerts its anti-inflammatory and antiplatelet effects. Any genetic mutation in the CYP2C9 gene can alter the efficiency of this metabolic process, influencing the body's response to aspirin. The -1188T>C mutation in the CYP2C9 gene has been associated with Aspirin-Intolerant Urticaria (AIU). This condition refers to hypersensitivity or intolerance to aspirin, characterized by skin reactions such as urticaria (hives) upon aspirin exposure. The mutation at position -1188 may affect the expression or activity of the CYP2C9 enzyme, leading to reduced metabolism of aspirin. As a consequence, individuals with this mutation may experience an altered and heightened response to aspirin, resulting in AIU. Individuals with the CT genotype are slow metabolizers of aspirin and are at a higher risk of developing AIU. Consult with healthcare professionals for personalized advice.

SAMPLE

Cardio Genetics - Summary

Suggestions

Nutrients

Nutrients are a diverse group of essential vitamins, minerals, and compounds designed to support fundamental cellular processes, energy production, and overall physiological function throughout the body. These agents work by serving as cofactors in enzymatic reactions, supporting cellular repair mechanisms, or providing building blocks for optimal metabolic function. Based on individual health assessments, this report provides recommendations for appropriate nutrient supplementation tailored to the specific deficiencies or requirements identified. These recommendations serve as guidance and must be reviewed with a qualified healthcare provider to ensure proper selection, dosage, and duration of supplementation. Responsible use of nutrients is essential to optimize absorption and utilization while preventing potential imbalances or toxicity.

SUPPLEMENTS

Vitamin C	500 mg/day	**Magnesium**	400 mg/day	**Ginger**	400 mg/ day
Vitamin D	600 IU/day	**Calcium**	1000 mg/day	**Vitamin B3**	500 mg/day
Zinc	25 mg/day	**Potassium**	99 mg/day	**Folate**	400 mcg/day
Seaweed	4 g/day				

FOOD SOURCES

Fruits
Banana, Orange, Avocado, Tomato, Apricot, Strawberry, Citrus Fruit, Papaya, Cantaloupe, Carrot

Vegetables
Spinach, Broccoli, Green Pea, Potato, Brussels Sprout, Carrot, Asparagus, Pea, Beets, Kale

Dairy
Milk, Yogurt, Cheese, Butter, Cottage Cheese, Buttermilk, Soy Yogurt

Fiber
Flaxseed, Walnut, Whole Grain, Lentil, Nuts, Beans, Brown Rice, Legumes, Pea, Sunflower Seed

Animal Protein
Egg, Salmon, Tuna, Chicken, Liver, Mackerel, Sardine, Beef, Pork, Heart

Botanicals

Botanicals are plant-derived compounds designed to support health and wellness through natural bioactive substances found in herbs, roots, leaves, and other plant materials. These agents work by providing phytochemicals that can modulate various physiological processes to promote optimal function, reduce inflammation, or support immune health. Based on individual health assessments, this report provides recommendations for appropriate botanical supplements tailored to the specific health concerns identified. These recommendations serve as guidance and must be reviewed with a qualified healthcare provider to ensure proper selection, dosage, and duration of use. Responsible use of botanicals is essential to optimize health benefits and minimize potential interactions or adverse effects.

SUPPLEMENTS

Berberine	1500 mg/day	**Beta-sitosterol**	3 g/day	**Hawthorn**	600 mg/day
Resveratrol	500 mg/day	**Flaxseed**	250 mg/day	**Cinnamon**	2 g/day
Aloe vera	300 mg/day	**Anthocyanins**	320 mg/day	**Citrus flavonoids**	650 mg/day
Garlic extract	300 mg/day				

SAMPLE

Suggestions

FOOD SOURCES

Fruits

Red Grape, Avocado, Banana, Berry, Blackberry, Blueberry, Cherry, Cranberry, Elderberry, Fermented Red Grape

Vegetables

Green Tea, Aloe Vera Leaf, Barberry, Brussels Sprout, Cinnamon Bark, Eggplant, Garlic Bulb (allium Sativum), Goldenseal, Oregon Grape, Purple Carrot

Fiber

Peanut, Almond, Brown Rice, Flaxseed, St. John's Wort, Sunflower Seeds. Wheat Germ

Prebiotics

Prebiotics are non-digestible fiber compounds designed to selectively nourish beneficial gut bacteria and promote healthy microbiome composition and function. These agents work by serving as food sources for beneficial microbes, stimulating their growth and metabolic activity, or creating an environment that supports optimal gut barrier function. Based on your health assessments, this report provides recommendations for appropriate prebiotic supplementation tailored to the specific requirements identified. These recommendations serve as guidance and must be reviewed with a qualified healthcare provider to ensure proper selection, dosage, and gradual introduction protocol. Responsible use of prebiotics is essential to optimize gut flora balance while minimizing potential digestive discomfort during microbiome adaptation.

SUPPLEMENTS

Glucomannan 2 g/day

FOOD SOURCES

Vegetables

Konjac Root (elephant Yam)

Antioxidants

Antioxidants are protective compounds designed to neutralize free radicals and reduce oxidative stress that can damage cells, proteins, and DNA throughout the body. These agents work by donating electrons to unstable molecules, supporting cellular repair mechanisms, or enhancing the body's natural antioxidant defense systems. Based on individual health assessments, this report provides recommendations for appropriate antioxidant supplementation tailored to the specific cellular protection needs identified. These recommendations serve as guidance and must be reviewed with a qualified healthcare provider to ensure proper selection, dosage, and duration of use. Responsible use of antioxidants is essential to optimize cellular protection while maintaining proper balance in natural oxidative processes.

SUPPLEMENTS

Curcumin 1500 mg/day **Quercetin** 500 mg/day **Coffee bean extract (CBE)** 400 mg/day

SAMPLE

Suggestions

FOOD SOURCES

Vegetables
Onion, Turmeric

Fiber
Coffee Beans

SAMPLE

Cardio Genetics

Marker SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Chylomicron/ Triglycerides	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs7903146	TCF7L2	Deposition of triglycerides	⊖⊖ C/C	Normal	C/C
rs12255372	TCF7L2	Deposition of triglycerides	⊖⊖ G/G	Normal	G/G
rs780094	GCKR	Elevated triglyceride levels	⊖⊖ G/G	Normal	G/G
rs1748195	ANGPTL3	Altered serum triglycerides concentrations	⊕⊖ C/G	Elevated	G/G
rs2967605	ANGPTL4	Vascular inflammation and endothelial dysfunction	⊕⊖ C/T	Normal	T/T, C/T
rs328	LPL	High triglyceride levels	⊕⊖ C/G	Normal	G/G, C/G
rs7007797	LPL	High triglyceride levels	⊕⊖ G/T	Normal	G/G, G/T
rs264	LPL	Impairs triglyceride metabolism	⊕⊖ A/G	Elevated	G/G
rs320	LPL	Altered lipoprotein metabolism	⊕⊖ G/T	Elevated	T/T
rs72691625	GPIHBP1	Increased triglyceride levels	⊕⊕ A/A	Elevated	G/G
rs964184	ZNF259	Elevated triglyceride and LDL levels	⊖⊖ C/C	Normal	C/C
rs2075291	APOA5	Increased plasma triglyceride levels	⊕⊕ A/A	Elevated	G/G, C/C
rs662799	APOA5	Elevated plasma triglyceride levels	⊖⊖ T/T	Normal	T/T, C/T
rs3135506	APOA5	Elevated plasma triglyceride levels	⊕⊖ C/G	Partially elevated	C/C
rs5128	APOC3	High plasma APOC3, TG, TC, and LDL-C levels.	⊖⊖ C/C	Normal	C/C
rs138326449	APOC3	Increased triglyceride levels	⊖⊖ G/G	Normal	G/G
rs508384	SCD1	Higher plasma apoB-48 levels	⊕⊖ A/C	Partially elevated	C/C
rs2167444	SCD1	Higher plasma apoB-48 levels	⊕⊖ A/T	Partially elevated	T/T
LDL	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs6752026	APOB	Higher levels of LDL-cholesterol	⊕⊖ A/G	Normal	A/A, A/G
rs693	APOB	High levels of APOB, TG, TC and LDL-C, and lower levels of HDL-C	⊕⊕ T/T	Elevated	C/C
rs515135	APOB	Elevated LDL cholesterol level	⊕⊕ G/G	Elevated	A/A
rs429358	APOE	Amyloid plaque buildup	⊖⊖ ε3/ε3	Normal	ε3/ε3, ε1/ε4, ε1/ε3, ε1/ε2

SAMPLE

Cardio Genetics

Marker SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

LDL	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs7412	APOE	Amyloid plaque buildup	⊖⊖ε3/ε3	Normal	ε3/ε3, ε1/ε4, ε1/ε3, ε1/ε2
rs1800591	MTP	Higher LDL and total cholesterol levels	⊖⊖T/T	Normal	T/T
rs599839	SORT1	Elevated LDL cholesterol levels	⊖⊖G/G	Normal	G/G
rs688	LDLR	Elevated LDL cholesterol levels	⊖⊖C/C	Normal	C/C
rs72658855	LDLR	Accumulation of LDL-cholesterol molecules	⊕⊕C/C	Elevated	T/T, C/T
rs12071264	LDLRAP1	Increased levels of LDL-cholesterol	⊕⊕A/A	Elevated	G/G, A/G
rs562556	PCSK9	Increased LDL_C levels	⊕⊖A/G	Normal	G/G, A/G
rs45613943	PCSK9	Increased LDL-cholesterol levels	⊕⊖C/T	Normal	C/C, C/T
rs4076317	ANGPTL4	Higher LDL and total cholesterol levels	⊕⊖C/G	Elevated	G/G

HDL	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs17120425	SIDT2	Lower HDL levels	⊖⊖Val/Val	Normal	Val/Val
rs5888	SCARB1	Altered lipid metabolism	⊕⊕T/T	Elevated	C/C
rs1532624	CETP	Disrupted lipid transport	⊖⊖A/A	Normal	A/A, A/C
rs247616	CETP	Disrupted lipid transport	⊕⊖C/T	Elevated	T/T
rs708272	CETP	Altered lipid metabolism	⊕⊖A/G	Elevated	G/G
rs632153	APOA1	Elevated TC, LDLC, TG and VLDL levels	⊕⊕T/T	Elevated	G/G
rs670	APOA1	Elevated TC, LDLC, TG and VLDL levels	⊖⊖G/G	Normal	G/G
rs5082	APOA2	Higher total cholesterol, triglyceride, Cholesterol/HDLc ratio and non-HDL cholesterol levels	⊕⊖C/T	Partially elevated	C/C

Lp(a)	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs10455872	LPA	Increased lipoprotein(a) levels	⊕⊖A/G	Elevated	A/A
rs3798221	LPA	Increased lipoprotein(a) levels	⊕⊖G/T	Elevated	T/T
rs6415084	LPA	Increased lipoprotein(a) levels	⊖⊖C/C	Normal	C/C
rs7770628	LPA	Increased lipoprotein(a) levels	⊖⊖T/T	Normal	T/T

SAMPLE

Cardio Genetics

Marker SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Lp(a)	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs6752026	APOB	Higher levels of LDL-cholesterol	⊕⊖ A/G	Normal	A/A, A/G
rs693	APOB	High levels of APOB, TG, TC and LDL-C, and lower levels of HDL-C	⊕⊕ T/T	Elevated	C/C
rs515135	APOB	Elevated LDL cholesterol level	⊕⊕ G/G	Elevated	A/A
oxLDL	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs662	PON1	High LDL oxidation	⊕⊕ G/G	Elevated	A/A
rs4845625	ILR-6	Triggered inflammatory responses	⊖⊖ C/C	Normal	C/C
rs1050450	GPX1	Oxidative stress	⊖⊖ C/C	Normal	C/C
Macrophages	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs2107545	MPO	Oxidative stress	⊕⊕ C/C	Elevated	T/T
rs1800482	NOS2	Excessive Nitric oxide production	⊖⊖ G/G	Normal	G/G
rs3730017	NOS2	Excessive Nitric oxide production	⊕⊖ C/T	Normal	T/T, C/T
Platelets(Clotting risk)	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs4252120	PLG	Impaired fibrinolysis	⊖⊖ C/C	Normal	C/C
rs1799963	Prothrombin	Higher levels of plasma prothrombin	⊕⊖ A/G	Partially elevated	G/G
rs3136516	Prothrombin	Excessive blood clot	⊖⊖ A/A	Normal	A/A
rs6025	Factor V Leiden	Abnormal blood clots in blood vessels	⊖⊖ G/G	Normal	G/G
Foam cell	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs1412444	LIPA	Plaque deposition	⊖⊖ C/C	Normal	C/C
rs2246833	LIPA	Plaque deposition	⊖⊖ C/C	Normal	C/C
rs671	ALDH2	Increased oxidative stress	⊖⊖ A/A	Normal	A/A, A/G

Systemic SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Nitric Oxide synthesis	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1879417	NOS1	Disrupted nitric oxide production	⊕⊖ C/T	Normal	T/T, C/T

SAMPLE

Cardio Genetics

Systemic SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Nitric Oxide synthesis	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1549758	NOS3	Impaired eNOS function	⊖⊖ C/C	Normal	C/C
rs2070744	NOS3	Reduced NO levels	⊖⊖ T/T	Normal	T/T
rs3918226	NOS3	Impaired eNOS function	⊕⊖ C/T	Normal	C/C, C/T
rs1799983	NOS3	Reduced NO levels	⊖⊖ G/G	Normal	G/G
Vascular health	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs10947789	KCNK5	Impaired vascular smooth muscle function	⊖⊖ C/C	Normal	C/C
rs9982601	KCNE2	Impaired vascular function	⊖⊖ C/C	Normal	C/C
rs1042714	ADR-B2	Adverse cardiovascular events	⊖⊖ C/C	Normal	C/C
Vascular remodeling	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs12526453	PHACTR1	Affected endothelial nitric oxide synthesis	⊖⊖ G/G	Normal	G/G
rs169713	PHACTR1	Endothelial dysfunction	⊖⊖ T/T	Normal	T/T
rs10116277	9p21	Coronary heart disease	⊕⊖ G/T	Normal	G/G, G/T
rs4977574	9p21	Myocardial infarction	⊖⊖ A/A	Normal	A/A, A/G
rs1333049	9p21	Coronary artery disease	⊕⊖ C/G	Partially elevated	G/G
rs10757278	9p21	Coronary artery disease	⊖⊖ A/A	Normal	A/A
rs2383207	9p21	Coronary heart disease	⊖⊖ A/A	Normal	A/A
rs2383206	9p21	Myocardial infarction	⊖⊖ A/A	Normal	A/A
Vascular tone	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs2271037	CORIN	Altered blood pressure	⊖⊖ G/G	Normal	G/G
rs3749585	CORIN	Altered blood pressure	⊕⊖ C/T	Partially elevated	T/T
rs75770792 (T555I)	CORIN	Reduced catalytic activity of Corin	⊖⊖ C/C	Normal	C/C
rs111253292 (Q568P)	CORIN	Reduced catalytic activity of corin	⊖⊖ A/A	Normal	A/A
rs9332982	CYP4A11	Elevated blood pressure	⊕⊖ C/T	Partially elevated	C/C

SAMPLE

Cardio Genetics

Systemic SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Vascular tone	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1126742	CYP4A11	Elevated blood pressure	⊖⊖ T/T	Normal	T/T
rs2108622	CYP4F2	Impaired vascular function	⊖⊖ C/C	Normal	C/C
rs1799998	CYP11B2	Altered blood pressure	⊕⊖ C/T	Partially elevated	C/C
rs4961	ADD1	Elevated blood pressure	⊖⊖ G/G	Normal	G/G
rs5370	EDN1	Increased vasoconstriction	⊖⊖ G/G	Normal	G/G

Atrial fibrillation	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs2200733	4q25	Atrial fibrillation	⊕⊖ C/T	Partially elevated	C/C
rs10033464	4q25	Atrial fibrillation	⊖⊖ G/G	Normal	G/G

Angiogenesis	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1122608	SMARCA4 or BRG1	Impaired vascular smooth muscle function	⊖⊖ G/G	Normal	G/G
rs11556924	ZC3HC1	Endothelial dysfunction	⊕⊕ C/C	Elevated	T/T
rs2010963	VEGF-A	Arterial stiffness, Endothelial dysfunction and High blood pressure	⊖⊖ G/G	Normal	G/G, C/G

Metabolic Risk	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs1801133	MTHFR	Active folate deficiency	⊕⊖ C/T	Partially elevated	C/C
rs1801131	MTHFR	Active folate deficiency	⊕⊖ A/C	Partially elevated	A/A
rs4680	COMT	Optimum COMT gene activity	⊕⊖ A/G	Partially elevated	G/G
rs1761667	CD36	Increased blood pressure	⊖⊖ A/A	Normal	A/A, A/G
rs10911021	GLUL	Affected glutamine metabolism	⊖⊖ C/C	Normal	C/C

Atherosclerosis	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs2023938	HDAC9	Vascular inflammation	⊕⊖ A/G	Partially elevated	A/A
rs10757274	CDKN2B-AS1	Atherosclerotic plaque formation	⊖⊖ A/A	Normal	A/A
rs4238001	SCARB1	Decreased cholesterol clearance	⊖⊖ C/C	Normal	C/C
rs2292318	LCAT	Altered lipid metabolism	⊖⊖ C/C	Normal	C/C

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Cardio Genetics

Systemic SNPs

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Atherosclerosis	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs4674344	CYP27A1	Plaque deposition	⊕⊖ A/T	Partially elevated	A/A

Hypertension

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Angiotensin II receptor	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
A1166C	AGTR1	Valsartan efficacy	⊕⊖ A/C	Normal	C/C, A/C
Angiotensin Converting Enzyme	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs4343	ACE	Elevated blood pressure	⊕⊖ A/G	Normal	A/A, A/G
rs4646994	ACE	Altered blood pressure	⊕⊖ I/D	Partially elevated	I/I
Angiotensinogen	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs699	AGT	Elevated blood pressure	⊖⊖ T/T	Normal	T/T
rs4762	AGT	Elevated blood pressure	⊖⊖ C/C	Normal	C/C
rs5051	AGT	Higher angiotensin II levels	⊕⊖ A/G	Partially elevated	G/G

Drug Metabolism

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Metformin	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs7757336	OCT2 gene	Increased metformin concentration	⊕⊖ A/G	Partially elevated	A/A
rs201919874	SLC22A2	Increased metformin concentration	⊕⊖ A/G	Normal	A/A, A/G
Statin	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
6986A>G	CYP3A5 *3/*3	Increased statin concentration in blood	⊕⊕ CYP3A5*3/*3	Elevated	CYP3A5*1/*1
421C>A	BCRP (ABCG2)	Increased statin concentration in blood	⊕⊕ A/A	Elevated	C/C
rs4149056	SLCO1B1	Increased plasma statin concentration	⊕⊖ C/T	Partially elevated	T/T
rs1057910	CYP2C9	Increased statin clearance	⊖⊖ CYP2C9*1/*1	Normal	CYP2C9*1/*1
rs20455	KIF6	Effective statin treatment	⊕⊖ Trp/Arg	Normal	Arg/Arg, Trp/Arg
Ezetimide	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
c388A > G	OATP1B1*1b (SLCO1B1)	Increased ezetimibe concentration	⊖⊖ A/A	Normal	A/A

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Cardio Genetics

Drug Metabolism

⊕⊕ Homozygous Mutant ⊕⊖ Heterozygous ⊖⊖ Homozygous Wild

Niacin	Gene Name	Risk Association	Your Mutation	Your Risk	Reference
rs738409	PNPLA3	Hindered niacin uptake	⊕⊖C/G	Partially elevated	C/C
rs1944438 C > T	DGAT2	Hindered niacin uptake	⊖⊖C/C	Normal	C/C
Clopidogrel	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs4244285	CYP2C19*2	Clopidogrel resistance	⊕⊖A/G	Partially elevated	G/G
Coenzyme Q10	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs1800566	NQO1	Hindered COQ10 uptake	⊕⊖P/S	Partially elevated	P/P
Caffeine	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs762551	CYP1A2	Slow caffeine metabolizers	⊕⊖A/C	Partially elevated	A/A
Aspirin	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs2071746	HO1	Aspirin resistance	⊖⊖A/A	Normal	A/A
rs1330344	COX-1	Aspirin resistance	⊕⊖C/T	Partially elevated	T/T
-1188T>C	CYP2C9	Slow metabolizers of aspirin	⊕⊖C/T	Partially elevated	T/T
rs3798220	LPA	Aspirin non-responsiveness	⊕⊖C/T	Normal	C/C, C/T
Angiotension II R B	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
A1166C	AGTR1	Impaired RAAS system	⊖⊖A/A	Normal	A/A
ACE inhibitor	**Gene Name**	**Risk Association**	**Your Mutation**	**Your Risk**	**Reference**
rs5182	AT1R	Captopril resistance	⊕⊖C/T	Normal	C/C, C/T
rs1799722	BDKRB2	Enalapril non-responsive	⊕⊖C/T	Normal	T/T, C/T

SAMPLE

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Risk and Limitations

This test has been developed and its performance characteristics determined and validated by Vibrant Genomics LLC., a CAP and CLIA certified lab. These assays have not been cleared or approved by the U.S. Food and Drug Administration. Vibrant Wellness provides additional contextual information on these tests and provides the report in a more descriptive fashion.

The Vibrant Cardio Genetics panel does not demonstrate absolute positive and negative predictive values for any condition. Its clinical utility has not been fully established. Clinical history and current symptoms of the individual must be considered by the healthcare provider prior to any interventions. Test results should be used as one component of a healthcare provider's clinical assessment.

Cardio Genetics testing is performed at Vibrant Genomics, a CAP and CLIA certified laboratory. Vibrant Genomics has effective procedures in place to protect against technical and operational problems. However, such problems may still occur. Examples include failure to obtain the result for a specific test due to circumstances beyond Vibrant's control. Vibrant may re-test a sample to obtain these results but upon re-testing the results may still not be obtained. As with all medical laboratory testing, there is a small chance that the laboratory could report incorrect results. A tested individual may wish to pursue further testing to verify any results.

Genetic testing is helpful in analyzing the risk of various diseases. However, it is important to note that Genetic risk determinants are neither necessary nor sufficient for the development of diseases. Environmental and lifestyle risk factors could also affect the risk of disease development. Results from genetic analysis should always be interpreted along with clinical findings on the individual. Genetic testing evaluates only for the genotypes indicated; it does not test for other genetic abnormalities found elsewhere in the genome. Different genetic variants can be tested by different genetic labs to evaluate the risk for a particular disease, depending on what is tested, genetic risk may not be comparable between labs. It should be realized that there are possible sources of error like any lab testing which include sample misidentification, trace contamination of PCR reactions, technical errors and rare genetic variants that may interfere with analysis.

Some individuals may feel anxious about getting their genetic test health results. If the potential user feels very anxious, such user should speak to his or her doctor or other health care professional prior to collection of a sample for testing. Users should consult with their doctor or other health care professional if they have any questions or concerns about the results of their test or their current state of health. Users of the test are also encouraged to discuss their test results with a genetic counselor, board-certified clinical molecular geneticist, or equivalent health care professional.

The information in this report is intended for educational purposes only. While every attempt has been made to provide current and accurate information, neither the author nor the publisher can be held accountable for any errors or omissions. Tested individuals may find their experience is not consistent with Vibrant's selected peer reviewed scientific research findings of relative improvement for study groups. The science in this area is still developing and many personal health factors affect diet and health. Since subjects in the scientific studies referenced in this report may have had personal health and other factors different from those of tested individuals, results from these studies may not be representative of the results experienced by tested individuals. Further, some recommendations may or may not be attainable, depending on the tested individual's physical ability or other personal health factors. A limitation of this testing is that many of these scientific studies may have been performed in selected populations only. The interpretations and recommendations are done in the context of these studies, but the results may or may not be relevant to tested individuals of different or mixed ethnicities.

Vibrant Wellness makes no claims as to the diagnostic or therapeutic use of its tests or other informational materials. Vibrant Wellness reports and other information do not constitute medical advice and are not a substitute for professional medical advice. Please consult your healthcare practitioner for questions regarding test results, or before beginning any course of medication, supplementation, or dietary changes.

The supplement recommendations and dosage guidelines provided are intended for general informational purposes only and should not replace professional medical advice; final dosage decisions must be made in consultation with your healthcare provider. Vibrant disclaims any liability for adverse effects, outcomes, or consequences arising from the use of these suggestions.

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Risks and Limitations – Genetics

Genetic testing is helpful in analyzing risks to various diseases. However, it is essential to note that Genetic risk determinants are neither necessary nor sufficient for the development of diseases. Environmental and lifestyle risk factors could also affect the risk of disease development. Results from genetic analysis should always be interpreted along with clinical findings on the individual. It should be realized that there are possible sources of error like any lab testing which include sample misidentification, trace contamination of PCR reactions, technical errors and rare genetic variants that may interfere with the analysis.

Genetic testing evaluates only for the genotypes indicated; it does not test for other genetic abnormalities found elsewhere in the genome. Different genetic variants can be tested by different genetic labs to evaluate the risk for a particular disease, depending on what is tested, genetic risk may not be comparable between labs.

Some individuals may feel anxious about getting their genetic test health results. If the potential user feels very anxious, such user should speak to his or her doctor or other health care professional prior to collecting a sample for testing. Users should consult with their doctor or other health care professional if they have any questions or concerns about the results of their test or their current state of health.

Variant risk classification may not align with associated disease risk or may change ex: a benign variant may be reported as pathogenic. Misclassification may be due to updated research studies, allele dropouts or interpretation pitfalls. Variant risk classification may also not be relevant to the tested individual of different or mixed ethnicities in comparison to the study group(s) from literature. Vibrant conducts internal audits, post market surveillance and feedback from providers and customers on an ongoing basis to keep our reports updated with the most current findings. Users of the test are also encouraged to discuss their test results with a genetic counselor, board-certified clinical molecular geneticist, or equivalent health care professional prior to any interventions and diet/supplement/lifestyle changes.

Genetic SNP testing is performed using real time PCR systems. It is important to note that allele calling for a particular SNP is performed using the Autocall methodology of the instrument manufacturer. Failure or error in autocalling could occur and is usually associated with outlier wells or software issues relevant to making an allele call. As with all genetic SNP testing, there is a small chance that the laboratory could report these incorrect results.

Genetic testing is not intended to diagnose a disease, tell you anything about your current state of health, or be used to make medical decisions, including whether you should take a medication/supplement or how much of a medication/supplement you should take. It is intended to provide users with their genetic information and suggestions to inform lifestyle decisions and conversations with their doctor or other health care professionals.

SAMPLE