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FINAL REPORT

Accession ID: 2183412850

Provider Information

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Report Information

● Current Result ● Previous Result ■ In Control ■ Moderate ■ Risk

Specimen Information

Sample Type	Collection Time	Received Time	Report	Final Report Date
EDTA	2025-10-22 09:45 (PST)	2025-10-23 11:04 (PST)	Cardio Genetics - P2	2025-11-08 02:36 (PST)



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TNP Test not performed

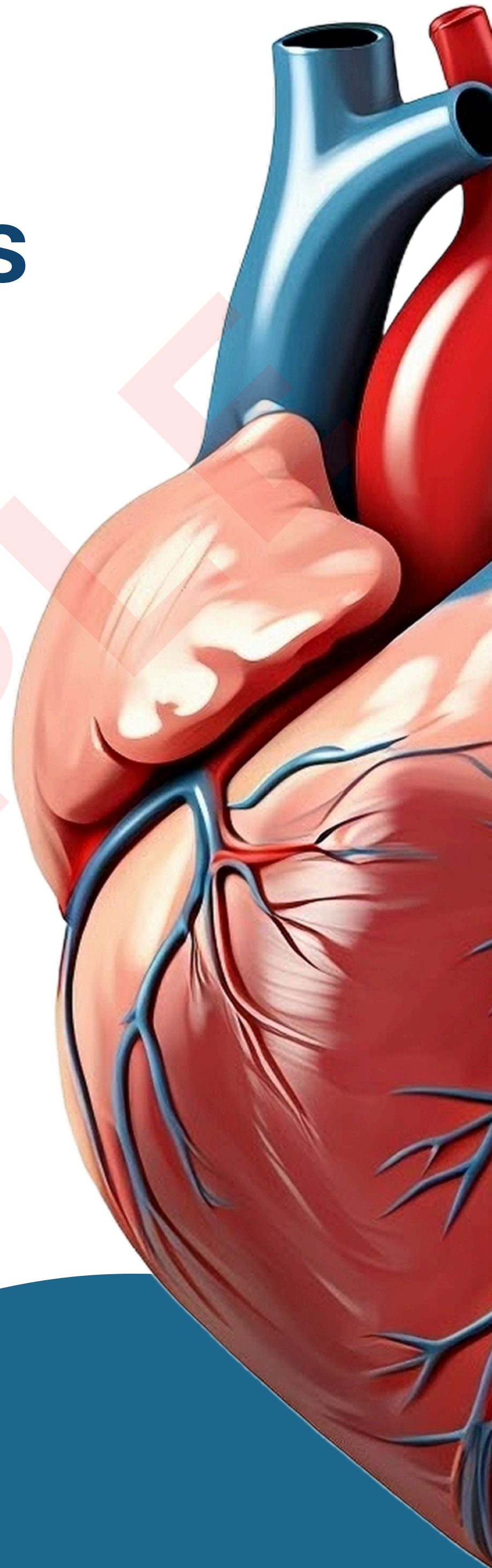
R&L Refer to risks and limitations at the end of report

Notes Refer to Lab notes at the end of the table

Cardio Genetics

Your Cardio 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.



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.

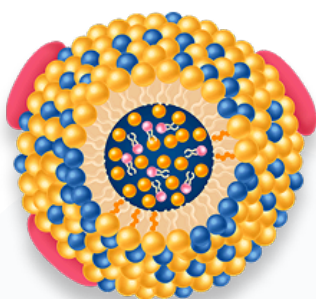
Risk Level	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
 SNP ID	C638T	MTHFR	Detox Capability	A A	Elevated	A G

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.

Genetics Summary

Lipoprotein



-  Free Cholesterol
-  Phospholipid
-  Triglyceride
-  Apolipoprotein
-  Cholesterol Esters

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: **TCF7L2, GCKR, ANGPTL3, ANGPTL4, LPL, APOA5**

LDL (Half life: 2 - 4 days)

Transports cholesterol to tissues; contains APOB-100.

Genes increasing risk: **MTP, SORT1, LDLR, LDLRAP1, PCSK9, ANGPTL4, APOB**

HDL (Half life: 3 - 5 days)

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

Genes increasing risk: **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, ILR-6, GPX1**

Macrophages (Half life: Weeks to months)

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

Genes increasing risk: **NOS2**

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

Blood cells that form clots; hyperactive in atherosclerosis.

Genes increasing risk: **PLG, Prothrombin**

Foam Cells (Half life: Weeks to months)

Macrophages filled with oxLDL, key in plaque formation.

Genes increasing risk: **ALDH2, LIPA**

Marker SNPs

Chylomicron/ Triglycerides	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs7903146	C>T	TCF7L2	Deposition of triglycerides	T/T	Elevated	C/C
<p>TCF7L2 gene encodes a protein that plays a role in the Wnt pathway (signaling pathway) and glucose homeostasis. The Wnt pathway is involved in the differentiation of adipocytes and the regulation of adipokines. As a result, the TCF7L2 gene affects adipogenesis (accumulation of fat-laden cells) and the formation and modification of triglycerides. A mutation may alter the function of the gene leading to the deposition of triglycerides. This can promote lipid storage and adipocyte hypertrophy (dysfunctional adipose tissue that is associated with increased cellular stress, decreased metabolic flexibility, and systemic diabetes). As a result, a mutation in the TCF7L2 gene may increase the levels of altered triglyceride. Individuals with TT genotypes have altered TCF7L2 gene and is associated with elevated triglyceride levels.</p>						
rs12255372	c.483+9017G>T	TCF7L2	Deposition of triglycerides	T/T	Elevated	G/G
<p>The TCF7L2 gene encodes a protein involved in the Wnt signaling pathway and glucose homeostasis. This pathway influences adipocyte differentiation and the regulation of adipokines, thereby affecting adipogenesis (the accumulation of fat cells) and triglyceride metabolism. Mutations in TCF7L2 can impair its function, leading to increased triglyceride deposition. This disruption promotes excessive lipid storage and adipocyte hypertrophy, which can contribute to altered triglyceride levels seen in familial combined hyperlipidemia. Individuals with TT genotypes have altered TCF7L2 gene and is associated with elevated triglyceride levels.</p>						
rs780094	A>G	GCKR	Elevated triglyceride levels	A/G	Elevated	G/G
<p>The GCKR gene encodes for a glucokinase regulatory protein which is involved in glucose homeostasis. Additionally, the GCKR gene is also known to play a role in regulating fatty acid and triglyceride synthesis from glucose or other substrates. A mutation in the gene upregulates lipogenesis (a process of conversion of fats) in the liver resulting in elevated levels of triglycerides. Individuals with AG genotype have altered GCKR gene and is associated with elevated triglyceride levels.</p>						
rs1748195	c.1801-668G>C	ANGPTL3	Altered serum triglycerides concentrations	C/C	Elevated	G/G
<p>The ANGPTL3 gene encodes Angiotensin-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 CC genotype have higher plasma triglyceride levels associated with the increased risk of hypertriglyceridemia.</p>						
rs2967605	C>T	ANGPTL4	Vascular inflammation and endothelial dysfunction	C/C	Elevated	T/T, C/T
<p>The ANGPTL4 gene encodes angiotensin-like 4, a protein involved in regulating lipid metabolism and vascular homeostasis. It inhibits lipoprotein lipase (LPL) activity, preventing triglyceride breakdown in tissues. Mutations in ANGPTL4, although they do not affect serum lipid traits, contribute to coronary artery disease (CAD) by influencing vascular inflammation and endothelial function. These mutations can promote plaque formation and destabilization, increasing CAD risk independently of lipid levels. Individuals with CC genotype have a higher risk of developing coronary artery disease (CAD).</p>						
rs328	c.1421C>G	LPL	High triglyceride levels	C/C	Elevated	G/G, C/G
<p>The LPL gene encodes lipoprotein lipase which degrades circulating triglycerides in the bloodstream. The LPL variants have been associated with lipoprotein lipase deficiency leading to increased triglyceride levels. Thus, a mutation in the LPL gene affects plasma triglycerides which can increase the risk of hypertriglyceridemia (elevated triglycerides). Individuals with CC genotype have higher plasma triglyceride levels associated with the increased risk of hypertriglyceridemia.</p>						

Marker SNPs

Chylomicron/ Triglycerides	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
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rs7007797	T>G	LPL	High triglyceride levels	T/T	Elevated	G/G, G/T
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The LPL gene encodes lipoprotein lipase which degrades circulating triglycerides in the bloodstream. The LPL variants have been associated with lipoprotein lipase deficiency leading to increased triglyceride levels. Thus, a mutation in the LPL gene affects plasma triglycerides which can increase the risk of hypertriglyceridemia (elevated triglycerides). Individuals with TT genotype have higher plasma triglyceride levels associated with the increased risk of hypertriglyceridemia.

rs264	c.776-172G>A	LPL	Impairs triglyceride metabolism	A/G	Elevated	G/G
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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).

rs3135506	c.56C > G	APOA5	Elevated plasma triglyceride levels	G/G	Elevated	C/C
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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 GG 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).

LDL	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
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rs6752026	P145S G>A	APOB	Higher levels of LDL-cholesterol	G/G	Elevated	A/A, A/G
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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. Mutation may impair this process resulting in higher levels of LDL-cholesterol in the blood and an increased chance of developing heart disease. Individuals with GG genotype have high levels of LDL-cholesterol.

rs693	c.7545C>T	APOB	High levels of APOB, TG, TC and LDL-C, and lower levels of HDL-C	T/T	Elevated	C/C
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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.

Marker SNPs

LDL	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs515135	A>G	APOB	Elevated LDL cholesterol level	G/G	Elevated	A/A
<p>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.</p>						
rs1800591	-493G > T	MTP	Higher LDL and total cholesterol levels	G/T	Elevated	T/T
<p>MTP gene encodes for microsomal triglyceride transfer protein. It is a heterodimeric lipid transfer protein mainly present in the liver, intestinal, and muscle cells which transfers lipid to a nascent apolipoprotein B (apo B) molecule, making it an indispensable genetic component in the maintenance of body cholesterol balance via pathways of absorption, synthesis, transfer, or secretion. An alteration in the gene can affect its function leading to dysregulated lipid levels in the blood. As a result, a mutation in the gene can lead to altered low-density lipoprotein cholesterol (LDL-C) and triglyceride (TG) levels. Individuals with GT genotype have high levels of LDL-C and TC.</p>						
rs599839	G>A	SORT1	Elevated LDL cholesterol levels	A/G	Elevated	G/G
<p>SORT1 gene encodes for a receptor that plays a role in transporting proteins to either the cell surface or subcellular compartments such as lysosomes and endosomes. The gene has also been associated with lipid metabolism, for example, its association with lipoprotein lipase (the enzyme that degrades lipids). As a result, a variation in the gene can affect lipid levels. A mutation causes low expression of SORT1 thereby elevating LDL cholesterol levels. This increases the chances of developing coronary artery disease (CAD). Individuals with the AG genotype have elevated LDL levels and are associated with an increased risk of developing CAD.</p>						
rs72658855	c.90C>T	LDLR	Accumulation of LDL-cholesterol molecules	C/C	Elevated	T/T, C/T
<p>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.</p>						
rs12071264	c.533-22A>G	LDLRAP1	Increased levels of LDL-cholesterol	A/A	Elevated	G/G, A/G
<p>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.</p>						

Marker SNPs

LDL	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs562556	c.1420G>A	PCSK9	Increased LDL_C levels	A/A	Elevated	G/G, A/G
<p>The PCSK9 gene is mainly expressed as an amino acid glycoprotein. It is involved in lipid metabolism. PCSK9 regulates lipid metabolism mainly through the degradation of low-density lipoprotein (LDL) receptors. Intracellularly, PCSK9 binds to the LDL receptor to promote lysosomal degradation of the receptor, while extracellularly, PCSK9 binds to LDL receptors to prevent LDL receptors from recycling to the cell surface. Mutations in the gene can alter its function leading to increased LDL-C levels, which is a major risk factor for coronary artery disease (CAD). Individuals with AA genotype have higher LDL-C levels and are associated with a higher risk of CAD.</p>						
rs45613943	c.799+158T>C	PCSK9	Increased LDL-cholesterol levels	T/T	Elevated	C/C, C/T
<p>The PCSK9 gene is mainly expressed as an amino acid glycoprotein. It is involved in lipid metabolism. PCSK9 has effects on lipid metabolism mainly through the degradation of low-density lipoprotein (LDL) receptors. Intracellularly, PCSK9 binds to the LDL receptor to promote lysosomal degradation of the receptor, while extracellularly, PCSK9 binds to LDL receptors to prevent LDL receptors from recycling to the cell surface. The rare variant of this gene reduces transcription resulting in low levels of the protein which increases the turnover of LDLR thereby reducing LDL-C levels. Individuals with TT genotype have high levels of LDL-cholesterol.</p>						
rs4076317	C>G	ANGPTL4	Higher LDL and total cholesterol levels	C/G	Elevated	G/G
<p>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.</p>						
HDL	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs5888	c.1050T>C	SCARB1	Altered lipid metabolism	C/T	Elevated	C/C
<p>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 CT genotype may have altered lipid metabolism which may be associated with higher risk of diabetic dyslipidemia.</p>						
rs1532624	c.658+186C>A	CETP	Disrupted lipid transport	C/C	Elevated	A/A, A/C
<p>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 CC genotype may have increased CETP gene activity, leading to reduced HDL cholesterol levels, which are associated with a higher risk of atherosclerotic lesions and coronary artery disease (CAD).</p>						

Marker SNPs

HDL	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs247616	C>T	CETP	Disrupted lipid transport	C/C	Elevated	T/T
<p>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 CC genotype may have increased CETP gene activity associated with a higher risk of atherosclerotic lesions and coronary artery disease (CAD).</p>						
rs5082	APOA2 -265T>C	APOA2	Higher total cholesterol, triglyceride, Cholesterol/HDLc ratio and non-HDL cholesterol levels	T/T	Elevated	C/C
<p>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 TT genotype have altered gene function associated with the risk of diabetic dyslipidemia.</p>						
Lp(a)	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs3798221	G>T	LPA	Increased lipoprotein(a) levels	G/G	Elevated	T/T
<p>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 GG 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).</p>						
rs7770628	C>T	LPA	Increased lipoprotein(a) levels	C/C	Elevated	T/T
<p>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 CC 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).</p>						
rs6752026	P145S G>A	APOB	Higher levels of LDL-cholesterol	G/G	Elevated	A/A, A/G
<p>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. Mutation may impair this process resulting in higher levels of LDL-cholesterol in the blood and an increased chance of developing heart disease. Individuals with GG genotype have high levels of LDL-cholesterol.</p>						

Marker SNPs

Lp(a)	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
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rs693	c.7545C>T	APOB	High levels of APOB, TG, TC and LDL-C, and lower levels of HDL-C	T/T	Elevated	C/C
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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	A>G	APOB	Elevated LDL cholesterol level	G/G	Elevated	A/A
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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	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
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rs4845625	T>C	ILR-6	Triggered inflammatory responses	C/T	Elevated	C/C
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ILR-6 gene is involved in inflammation. Immune cells take up oxidized LDL (ox-LDL) through their cell surface scavenger receptors to form foam cells which secrete proinflammatory cytokines, such as IL-6. ILR-6 signaling initiates downstream inflammation cascades. The minor allele of this gene is associated with increased levels of C-reactive protein or CRP, LDL-C, and ApoB and high risk of coronary heart diseases or CHD. Thus, susceptible individuals with the mutant allele may have a high risk of developing CHD due to persistent inflammation. Individuals with CT genotype have high levels of CRP and a 4% increased risk of coronary heart disease (CHD).

rs1050450	198C > T	GPX1	Oxidative stress	C/T	Partially elevated	C/C
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The GPX1 gene encodes glutathione peroxidase-1 (GPx-1), an enzyme essential for reducing toxic molecules such as hydrogen peroxide and lipid peroxides to less harmful substances, thus aiding in antioxidant defense. Genetic variations in GPX1 can impair this function, increasing oxidative stress. In the context of atherosclerosis, GPx-1 deficiency exacerbates foam cell formation induced by oxidized low-density lipoproteins (oxLDL), which accelerates plaque development. This deficiency also leads to increased macrophage proliferation via the p44/42 MAPK (ERK1/2) signaling pathway. Polymorphism such as rs1050450 impaired GPx-1 activity reduces nitric oxide availability, promoting arterial hypertension and increasing the risk of cardiovascular diseases (CVD). Individuals with the CT genotype may have altered enzymatic activity leading to increased oxidative stress, thus, a higher risk of CVD.

Marker SNPs

Macrophages	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
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rs1800482	(-954G>C)	NOS2	Excessive Nitric oxide production	C/C	Elevated	G/G
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The NOS2 gene encodes inducible nitric oxide synthase (iNOS), an enzyme responsible for producing nitric oxide (NO), a key molecule in immune responses. In macrophages, iNOS is upregulated during inflammation, generating NO to combat pathogens. The rs1800482 polymorphism (G>C) in the NOS2 gene, located in the promoter region, increases iNOS activity, leading to excessive NO production. This overproduction of NO contributes to endothelial dysfunction by forming reactive nitrogen species, which damage blood vessels. Consequently, carriers of the C allele have a higher risk of essential arterial hypertension (EAH) due to vascular inflammation. Individuals with CC genotype exhibit excessive NO production, leading to endothelial dysfunction, which is associated with a higher risk of developing essential arterial hypertension (EAH).

rs3730017	c.661C>T	NOS2	Excessive Nitric oxide production	C/C	Elevated	T/T, C/T
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The NOS2 gene encodes inducible nitric oxide synthase (iNOS), responsible for producing nitric oxide (NO), a signaling molecule involved in immune responses and inflammation. In macrophages, iNOS generates NO to combat pathogens and regulate immune activity. The rs3730017 polymorphism in NOS2 leads to an Arg221Trp substitution, affecting enzyme activity and NO production. Carriers of the T allele exhibit lower NO levels, which may protect against endothelial dysfunction. Conversely, elevated NO in carriers of the C allele promotes inflammation and contributes to the development of essential arterial hypertension (EAH) by damaging vascular endothelial cells. Individuals with the CC genotype exhibit excessive NO production, leading to endothelial dysfunction, which is associated with a higher risk of developing essential arterial hypertension (EAH).

Platelets(Clotting risk)	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
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rs4252120	c.1256+9T>C	PLG	Impaired fibrinolysis	T/T	Elevated	C/C
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The PLG gene encodes a protein called plasminogen, which is produced in the liver. Enzymes called plasminogen activators to convert plasminogen into the protein plasmin, which breaks down another protein called fibrin. Fibrin is the main protein involved in blood clots and is important for wound healing, creating the framework for normal tissue to grow back. Excess fibrin is broken down when no longer needed, and the new, more flexible normal tissue takes its place. The mutation in the gene may lead to plasminogen deficiency and blood clots due to impaired fibrinolysis. This may predispose to coronary artery disease (CAD). Individuals with the TT genotype who have decreased plasminogen activity increase the risk for coronary artery disease (CAD).

rs3136516	A19911G	Prothrombin	Excessive blood clot	A/G	Partially elevated	A/A
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The prothrombin gene (F2) encodes prothrombin, a precursor to thrombin, which is critical for blood clot formation. In the cardiovascular system, balanced prothrombin activity helps maintain normal blood flow and vessel integrity. The 19911 A>G mutation in the F2 gene can lead to elevated prothrombin levels, creating a hypercoagulable state. This mutation increases the risk of venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE), by promoting excessive clot formation. Individuals with this mutation face a higher risk of developing VTE, particularly under additional risk factors such as immobility or surgery. Individuals with the AG genotype exhibit excessive blood clot formation and are associated with a higher risk for venous thromboembolism (VTE).

Marker SNPs

Foam cell	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
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rs1412444	C>T	LIPA	Plaque deposition	C/T	Partially elevated	C/C
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The LIPA gene encodes lysosomal acid lipase (LAL), an enzyme crucial for hydrolyzing cholesteryl esters and triglycerides within lysosomes. This process is vital for lipid metabolism and the removal of lipids from cells. Mutations in the LIPA gene impair LAL activity, leading to the accumulation of these lipids within lysosomes. This accumulation can cause the formation of foam cells and contribute to the development of atherosclerosis. The resulting atherosclerotic plaques can narrow arteries, increasing the risk of hypertension and coronary artery disease (CAD). Individuals with the CT genotype may have a risk of developing CAD.

rs2246833	C>T	LIPA	Plaque deposition	C/T	Partially elevated	C/C
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The LIPA gene encodes lysosomal acid lipase (LAL), an enzyme crucial for hydrolyzing cholesteryl esters and triglycerides within lysosomes. This process is vital for lipid metabolism and the removal of lipids from cells. Mutations in the LIPA gene impair LAL activity, leading to the accumulation of these lipids within lysosomes. This accumulation can cause the formation of foam cells and contribute to the development of atherosclerosis. The resulting atherosclerotic plaques can narrow arteries, increasing the risk of hypertension and coronary artery disease (CAD). Individuals with the CT genotype may have a risk of developing CAD.

rs671	c.1510G>A	ALDH2	Increased oxidative stress	G/G	Elevated	A/A, A/G
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Aldehyde dehydrogenase 2 (ALDH2) is an enzyme encoded by the ALDH2 gene, located in the mitochondria, and is crucial for cellular antioxidant processes. It metabolizes aldehydes and reduces reactive oxygen species, thereby mitigating oxidative damage. Mutations in ALDH2, such as the rs671 polymorphism, can impair this function, leading to reduced antioxidant activity and increased oxidative stress. Specifically, ALDH2 deficiency impacts foam cell formation, a key step in atherosclerosis. In the presence of oxidized low-density lipoproteins (ox-LDL), ALDH2-deficient macrophages show reduced foam cell formation, suggesting that ALDH2 modulates the atherosclerotic process through its interaction with ox-LDL and related oxidative pathways. This impaired cellular protection due to ALDH2 dysfunction can also elevate the risk of hypertension. Individuals with GG genotype who have ALDH2 deficiency could be associated with a higher incidence of hypertension.

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: **ADR-B2, KCNK5**

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

Genes increasing risk: **PHACTR1, 9p21**

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

Genes increasing risk: **CYP11B2, ADD1, 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.

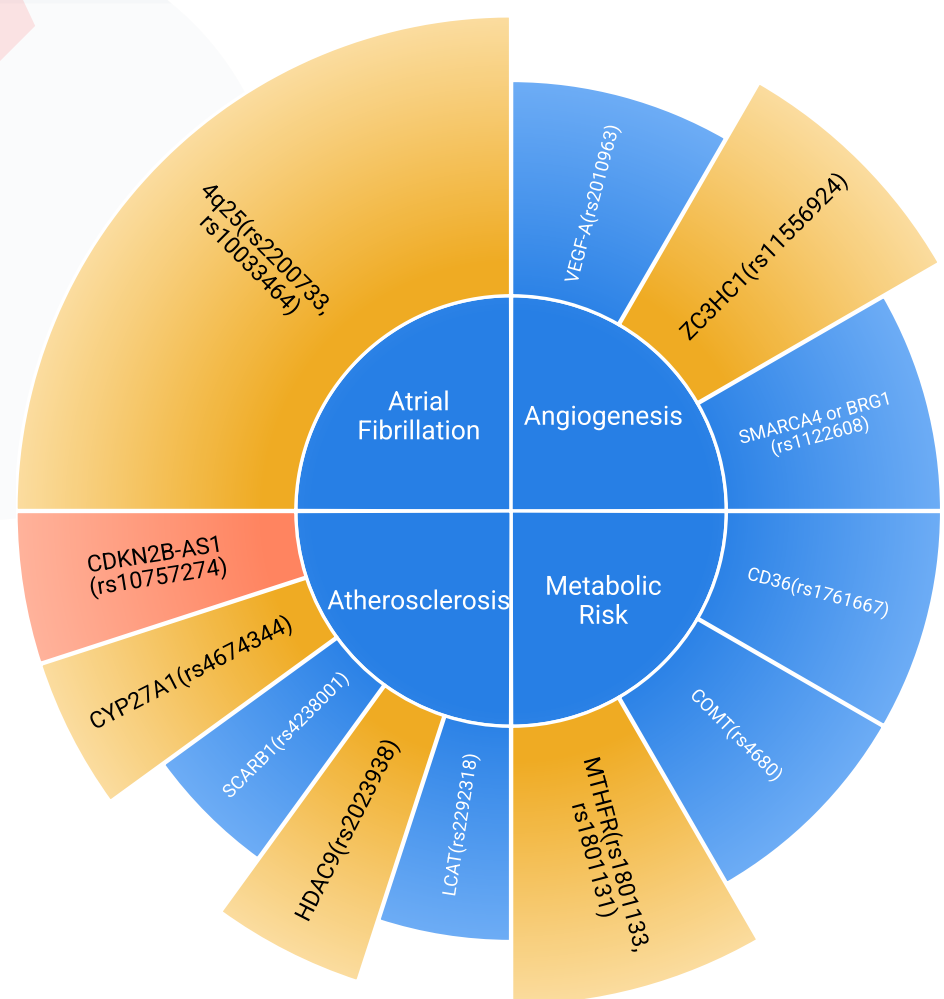
Genes increasing risk: **ZC3HC1**

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

Genes increasing risk: **MTHFR**

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

Genes increasing risk: **CDKN2B-AS1, CYP27A1, HDAC9**



Systemic SNPs

Vascular health	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs10947789	g.39207146T>C	KCNK5	Impaired vascular smooth muscle function	T/T	Elevated	C/C

The KCNK5 gene encodes a potassium channel protein which plays a crucial role in regulating potassium ion transport. Mutations in KCNK5 can disrupt its normal function, leading to altered membrane potential and excitability of vascular smooth muscle cells. Such disruptions may contribute to the development of coronary artery disease (CAD) by promoting vascular dysfunction, increased vascular resistance, and impaired blood flow. Additionally, the dysregulation of potassium channels can influence the inflammatory response and vascular remodeling, further exacerbating CAD risk. Individuals with the TT genotype have decreased gene activity increasing the risk for coronary artery disease (CAD).

rs1042714	c.79G>C	ADR-B2	Adverse cardiovascular events	G/G	Elevated	C/C
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The ADR-B2 gene encodes the beta-2 adrenergic receptor, a critical component in the regulation of cardiovascular function. This receptor mediates the effects of catecholamines such as adrenaline and noradrenaline, influencing heart rate, blood vessel dilation, and overall cardiovascular response to stress. Variants in the ADR-B2 gene can alter receptor function, affecting the efficiency of these physiological processes. Specific mutations, such as those leading to altered receptor signaling, can disrupt normal cardiovascular responses and contribute to the development of coronary artery disease (CAD). These mutations may impair the regulation of blood pressure and heart rate, potentially leading to adverse cardiovascular events and increased risk of CAD. Individuals with Glu27Glu exhibit impaired regulation of blood pressure and heart rate, potentially leading to adverse cardiovascular events and increased risk of CAD.

Vascular remodeling	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs12526453	c.251-126053C>G	PHACTR1	Affected endothelial nitric oxide synthesis	C/C	Elevated	G/G

The PHACTR1 gene encodes a member of the phosphatase and actin regulator family of proteins. PHACTR1 is involved in various cellular processes including the modulation of protein phosphatase 1, an enzyme that regulates a wide array of physiological processes such as endothelial nitric oxide synthesis in endothelial cells. It also binds to actin for cytoskeletal reorganization. Polymorphisms in this gene are associated with susceptibility to myocardial infarction and coronary artery disease (CAD). However, the mechanism by which the gene is involved in the pathogenesis of heart diseases is still to be elucidated. A mutation in the PHACTR1 gene at the 6p24.1 locus can affect its function of endothelial nitric oxide synthesis in endothelial cells, leading to the risk of CAD. Individuals with CC genotype have altered gene function and they are associated with an increased risk of CAD.

rs10116277	G>T	9p21	Coronary heart disease	T/T	Elevated	G/G, G/T
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Extensive Genome-wide association studies (GWAS) have identified large number of common genetic variants leading to coronary heart disease (CHD). Of these variants, the chromosome 9p21 locus was the first to be discovered. The variant is known to have the largest individual effect and is seen to be the mostly widely replicated genetic risk factor for CHD. As a result, polymorphism associated with this locus significantly increases the risk of CHD in susceptible individuals. Despite various hypotheses, such as cardiac hypertrophy, vascular remodeling, and atherosclerosis susceptibility, the exact mechanism by which the 9p21 genetic locus increases CHD risk is not fully understood. Individuals with TT genotype have high risk of developing coronary heart disease.

Systemic SNPs

Vascular remodeling	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs4977574	A>G	9p21	Myocardial infarction	G/G	Elevated	A/A, A/G
<p>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 myocardial infarction (MI). 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 MI 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 MI in susceptible individuals. Individuals with GG genotype have high risk of developing myocardial infarction.</p>						
rs1333049	G>C	9p21	Coronary artery disease	C/G	Partially elevated	G/G
<p>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.</p>						
rs10757278	A>G	9p21	Coronary artery disease	A/G	Partially elevated	A/A
<p>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 AG genotype have significant risk of developing coronary artery disease.</p>						
rs2383207	A>G	9p21	Coronary heart disease	G/G	Elevated	A/A
<p>Extensive Genome-wide association studies (GWAS) have identified large number of common genetic variants leading to coronary heart disease (CHD). Of these variants, the chromosome 9p21 locus was the first to be discovered. The variant is known to have the largest individual effect and is seen to be the mostly widely replicated genetic risk factor for CHD. As a result, polymorphism associated with this locus significantly increases the risk of CHD in susceptible individuals. Despite various hypotheses, such as cardiac hypertrophy, vascular remodeling, and atherosclerosis susceptibility, the exact mechanism by which the 9p21 genetic locus increases CHD risk is not fully understood. Individuals with GG genotype have a high risk of developing coronary heart disease.</p>						

Systemic SNPs

Vascular remodeling	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs2383206	A>G	9p21	Myocardial infarction	G/G	Elevated	A/A
<p>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 myocardial infarction (MI). 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 MI 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 MI in susceptible individuals. Individuals with GG genotype have high risk of developing myocardial infarction.</p>						
Vascular tone	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs2271037	T>G	CORIN	Altered blood pressure	G/T	Partially elevated	G/G
<p>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 GT genotype have impaired gene function and are associated with an increased risk of hypertension.</p>						
rs3749585	T>C	CORIN	Altered blood pressure	C/T	Partially elevated	T/T
<p>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.</p>						
rs1126742	8590 T>C	CYP4A11	Elevated blood pressure	C/T	Partially elevated	T/T
<p>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 TC genotype exhibit a disrupted balance of 20-HETE, impairing blood pressure regulation and increasing the risk of essential hypertension.</p>						
rs1799998	CYP11B2-344C/T	CYP11B2	Altered blood pressure	T/T	Elevated	C/C
<p>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. Homozygous wild (abnormal) individuals have altered aldosterone levels and are associated with an elevated risk of hypertension.</p>						

Systemic SNPs

Vascular tone	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs4961	c.1378G>T	ADD1	Elevated blood pressure	G/T	Partially elevated	G/G
<p>Alpha-adducin is a protein that in humans is encoded by the ADD1 gene, found in many tissues. Adducin is thought to affect kidney function by modulating the capacity of the tubular epithelial cells to transport ions via modification of the assembly of the actin cytoskeleton. It is involved in intercellular contact, signal transduction, and sodium ion transport across the cell membrane. The mutation increases sensitivity to changes in sodium balance and increases blood pressure which can cause hypertension. Individuals with GT genotypes may have increased sensitivity to changes in sodium balance and a higher incidence of hypertension.</p>						
Atrial fibrillation	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs10033464	T>G	4q25	Atrial fibrillation	G/T	Partially elevated	G/G
<p>A locus of chromosome 4q25 was identified to be associated with atrial fibrillation. The polymorphism associated with this locus increases the risk of developing atrial fibrillation in susceptible individuals. Individuals with the GT genotype have significant risk of developing atrial fibrillation.</p>						
Angiogenesis	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs11556924	C>T	ZC3HC1	Endothelial dysfunction	C/T	Partially elevated	T/T
<p>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 CT genotype who have decreased gene activity have increased risk for coronary artery disease (CAD).</p>						
Metabolic Risk	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs1801131	A1298C; A>C	MTHFR	Active folate deficiency	A/C	Partially elevated	A/A
<p>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.</p>						
Atherosclerosis	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs2023938	A>G	HDAC9	Vascular inflammation	A/G	Partially elevated	A/A
<p>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).</p>						

Systemic SNPs

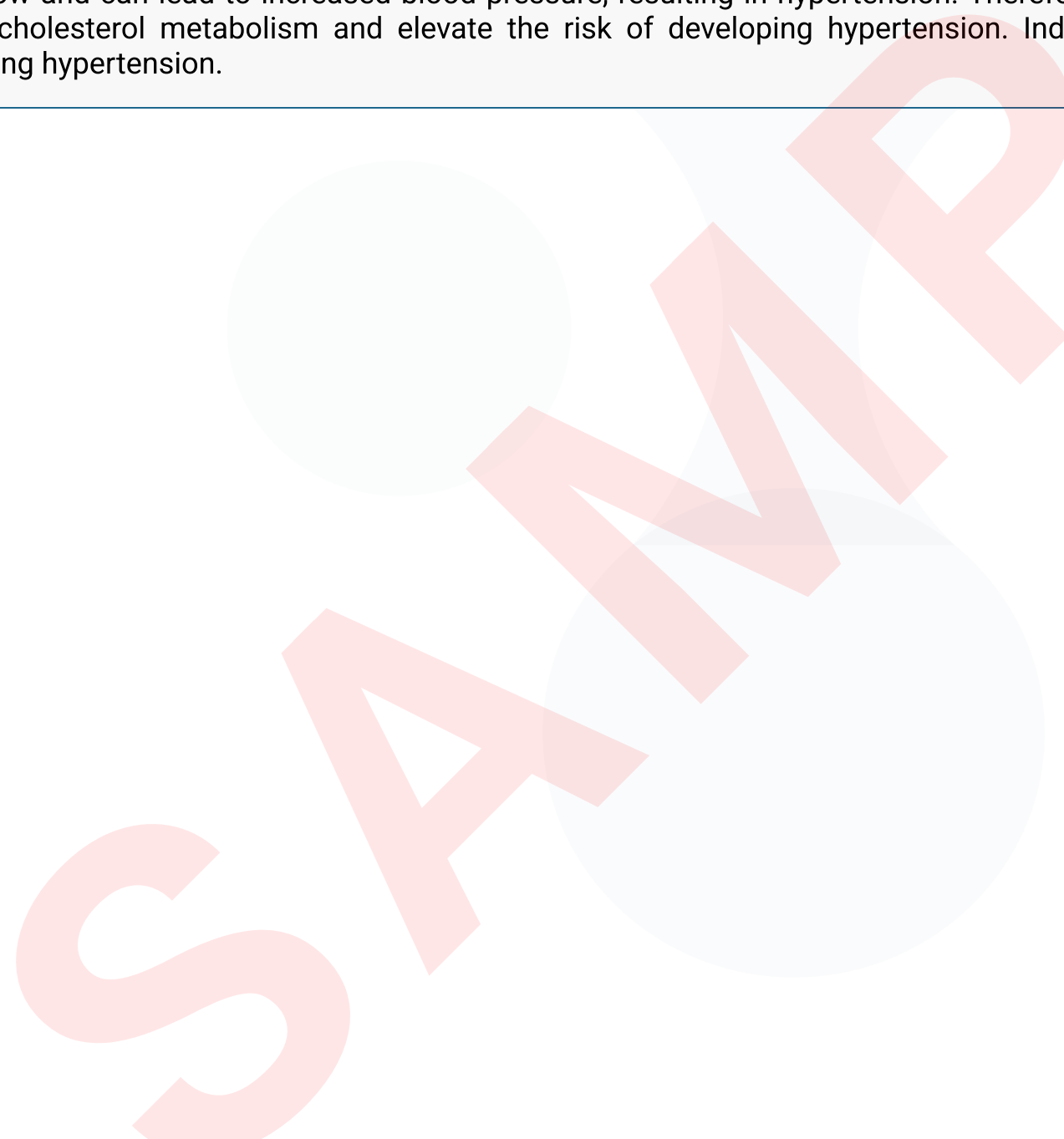
Atherosclerosis	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
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rs10757274	A>G	CDKN2B-AS1	Atherosclerotic plaque formation	G/G	Elevated	A/A
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CDKN2B-AS1 gene is a non-coding RNA that accelerates lipid uptake and intracellular lipid accumulation. CDKN2B-AS1 is expressed in blood vessels and coronary smooth muscle cells and is involved in preventing inflammatory response and promoting cholesterol efflux. Mutation in the gene leads to decreased gene activity and is associated with cardiovascular disease by mediating the response to inflammatory signaling. CDKN2B-AS1 promotes atherosclerotic plaque formation increasing the risks associated with coronary artery disease (CAD). Individuals with the GG genotype have decreased gene activity increasing the risk for coronary artery disease (CAD).

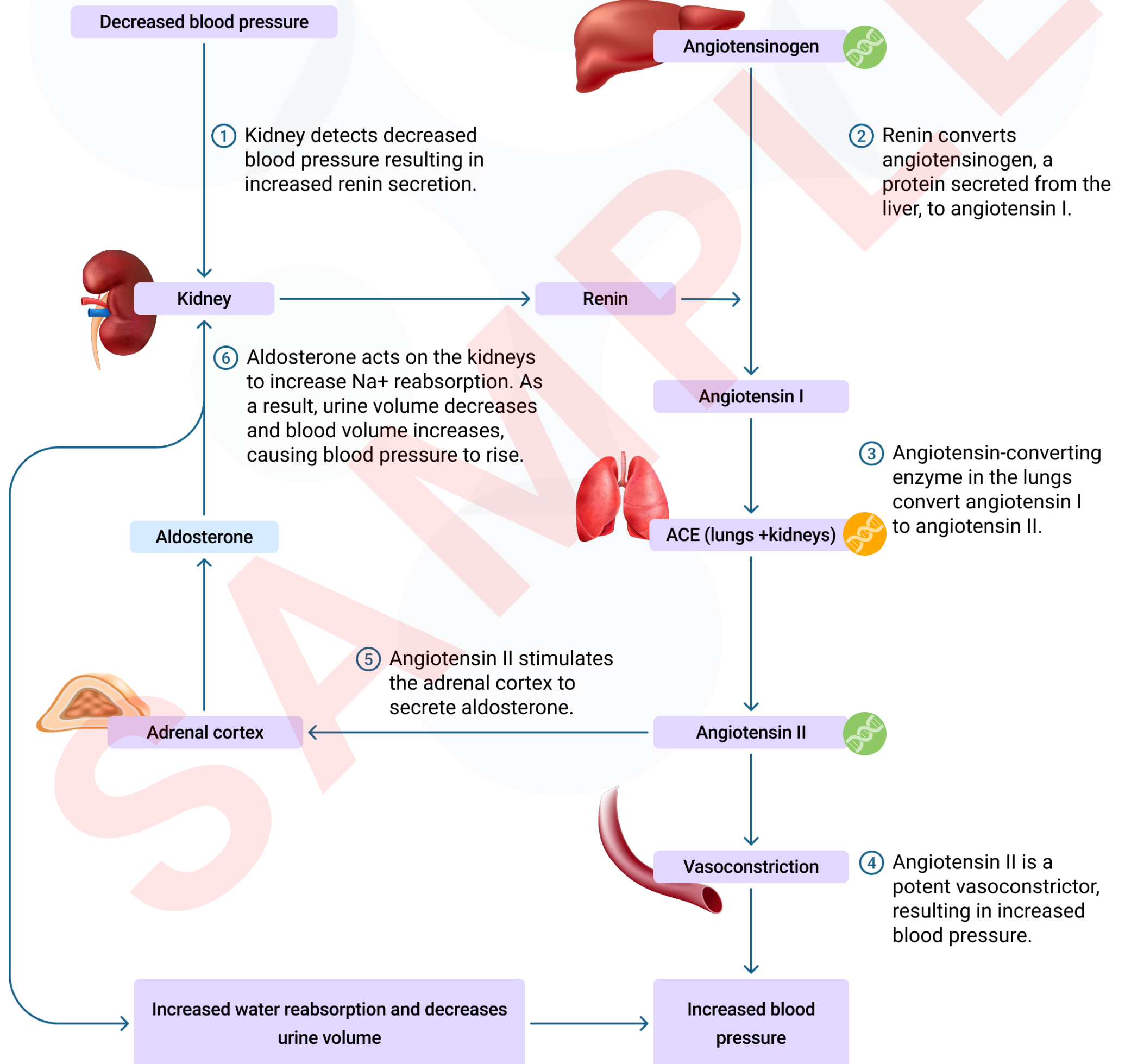
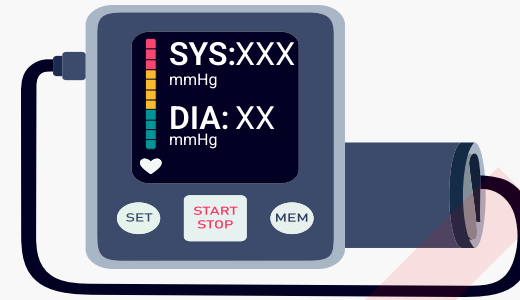
rs4674344	c.256-4425A>T	CYP27A1	Plaque deposition	A/T	Partially elevated	A/A
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The CYP27A1 gene encodes a mitochondrial enzyme involved in cholesterol metabolism, expressed in various tissues. In the liver, CYP27A1 catalyzes the conversion of cholesterol 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.



Hypertension Risk Genetics

Renin-Angiotensin-Aldosterone System



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.

Hypertension







Angiotensin Converting Enzyme	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs4646994	I/D	ACE	Altered blood pressure	I/D	Partially elevated	I/I

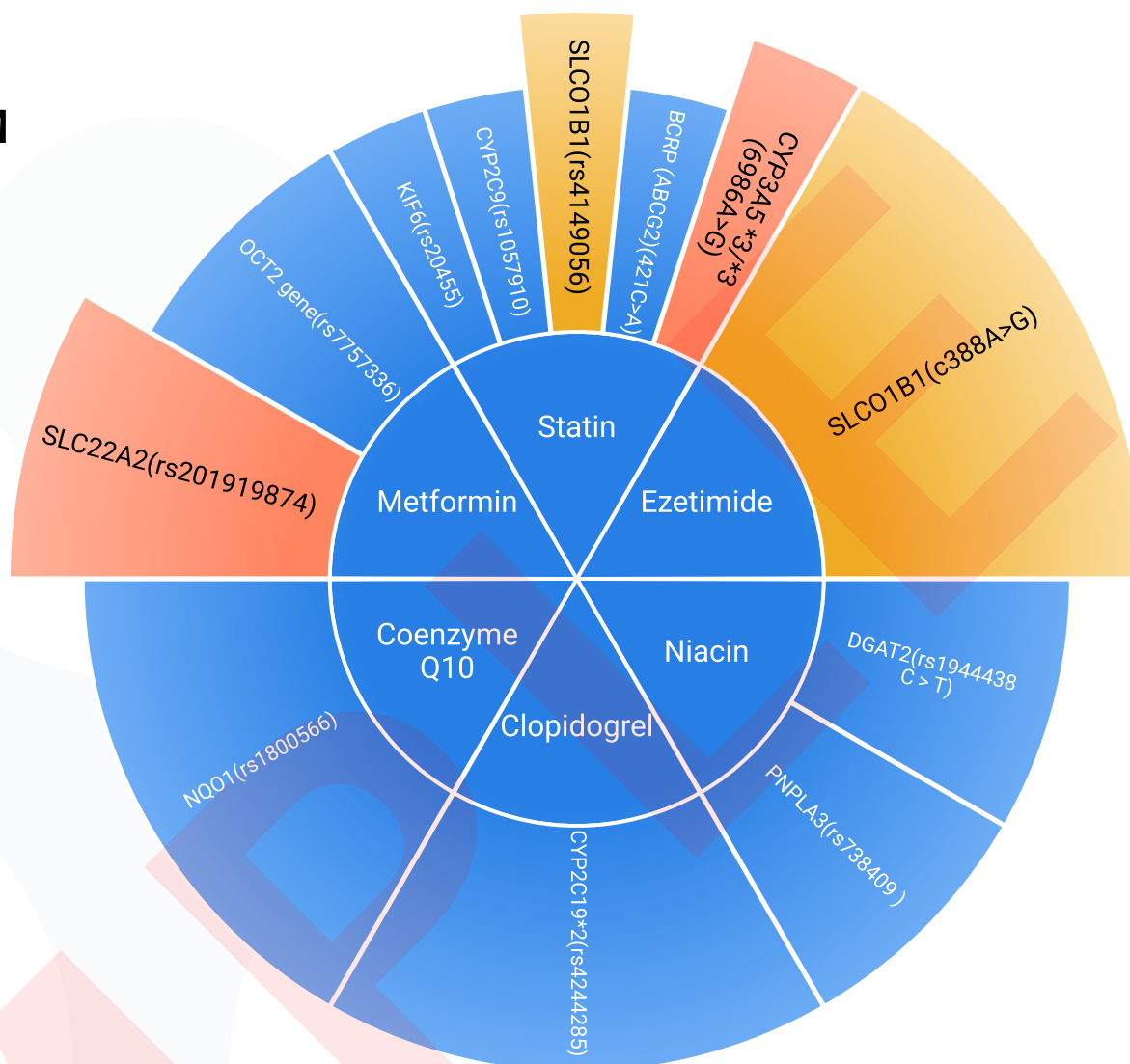
The ACE gene encodes an enzyme involved in blood pressure regulation and electrolyte balance. It catalyzes the conversion of angiotensin I into a physiologically active peptide angiotensin II. Angiotensin II is a potent vasopressor and aldosterone-stimulating peptide that controls blood pressure and 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.







Genetics Summary

DRUG AND SUPPLEMENT METABOLISM

-  **Metformin**
Genes increasing risk: **SLC22A2**
-  **Statin**
Genes increasing risk: **CYP3A5 *3/*3, SLC01B1**
-  **Ezetimide**
Genes increasing risk: **SLC01B1**
-  **Niacin**
Genes increasing risk: *None*
-  **Clopidogrel**
Genes increasing risk: *None*
-  **Coenzyme Q10**
Genes increasing risk: *None*



DRUG AND SUPPLEMENT METABOLISM

-  **Caffeine**
Genes increasing risk: **CYP1A2**
-  **Aspirin**
Genes increasing risk: **LPA**
-  **Angiotension II R B**
Genes increasing risk: **AGTR1**
-  **Ace Inhibitor**
Genes increasing risk: **BDKRB2**



Drug Metabolism

Metformin	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs201919874	G>A	SLC22A2	Increased metformin concentration	G/G	Elevated	A/A, A/G

The OCT2 gene, or SLC22A2, 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 GG genotype have impaired metformin elimination, contributing to elevated plasma metformin concentrations. Consult with healthcare professionals for personalized advice.

Statin	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
6986A>G	c.219-237A>T	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.

rs4149056	T521C	SLC01B1	Increased plasma statin concentration	C/T	Partially elevated	T/T
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The SLC01B1 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 SLC01B1 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 SLC01B1 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.

Drug Metabolism

Ezetimide	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
c388A > G	c388A > G	OATP1B1*1b (SLC01B1)	Increased ezetimibe concentration	G/G	Elevated	A/A

OATP1B1, or organic anion transporting polypeptide 1B1, is a hepatic transporter responsible for facilitating the uptake of various substances, including drugs, into liver cells. The primary function of OATP1B1 in the context of Ezetimibe metabolism is to facilitate the hepatic uptake of the drug. Ezetimibe, an inhibitor of the Niemann-Pick C1-like 1 protein involved in intestinal cholesterol uptake, undergoes extensive enterohepatic circulation. This process is highly dependent on hepatic uptake transporters, among which OATP1B1 plays a crucial role. The mutation OATP1B1b, represented by the SLC01B1 c.388A > G alteration and resulting in the p.Asn130Asp substitution, leads to notable changes in enzyme activity. Cells expressing OATP1B1b exhibit reduced uptake of Ezetimibe compared to non-mutated variants. In vivo studies further confirm this observation, revealing a gene-dose-dependent decrease in the area under the curve of Ezetimibe in individuals with the OATP1B1b protein. This diminished activity of OATP1B1b contributes to an increase in Ezetimibe concentration in the body. Individuals with the GG genotype have diminished activity of OATP1B1b resulting in a slower breakdown of ezetimibe drugs in the body. This leads to an increase in ezetimibe concentration in the body. Consult with healthcare professionals for personalized advice.

Caffeine	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs762551	AA	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	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs3798220	T>C	LPA	Aspirin non-responsiveness	T/T	Elevated	C/C, C/T

The LPA gene encodes lipoprotein(a) [Lp(a)], which is composed of low-density lipoprotein (LDL) and apolipoprotein(a). Elevated levels of Lp(a) are a significant risk factor for coronary artery disease (CAD) because they promote atherosclerosis and arterial plaque buildup. Mutations in the LPA gene, especially those affecting the Kringle IV repeats in apolipoprotein(a), can lead to higher Lp(a) levels, which increases the risk of vascular inflammation and thrombosis. Aspirin reduces this risk by inhibiting platelet aggregation, which helps mitigate the CAD risk associated with high Lp(a) levels. Individuals with the TT genotype show no added benefit from aspirin for thrombotic risk reduction. Consult with healthcare professionals for personalized advice.

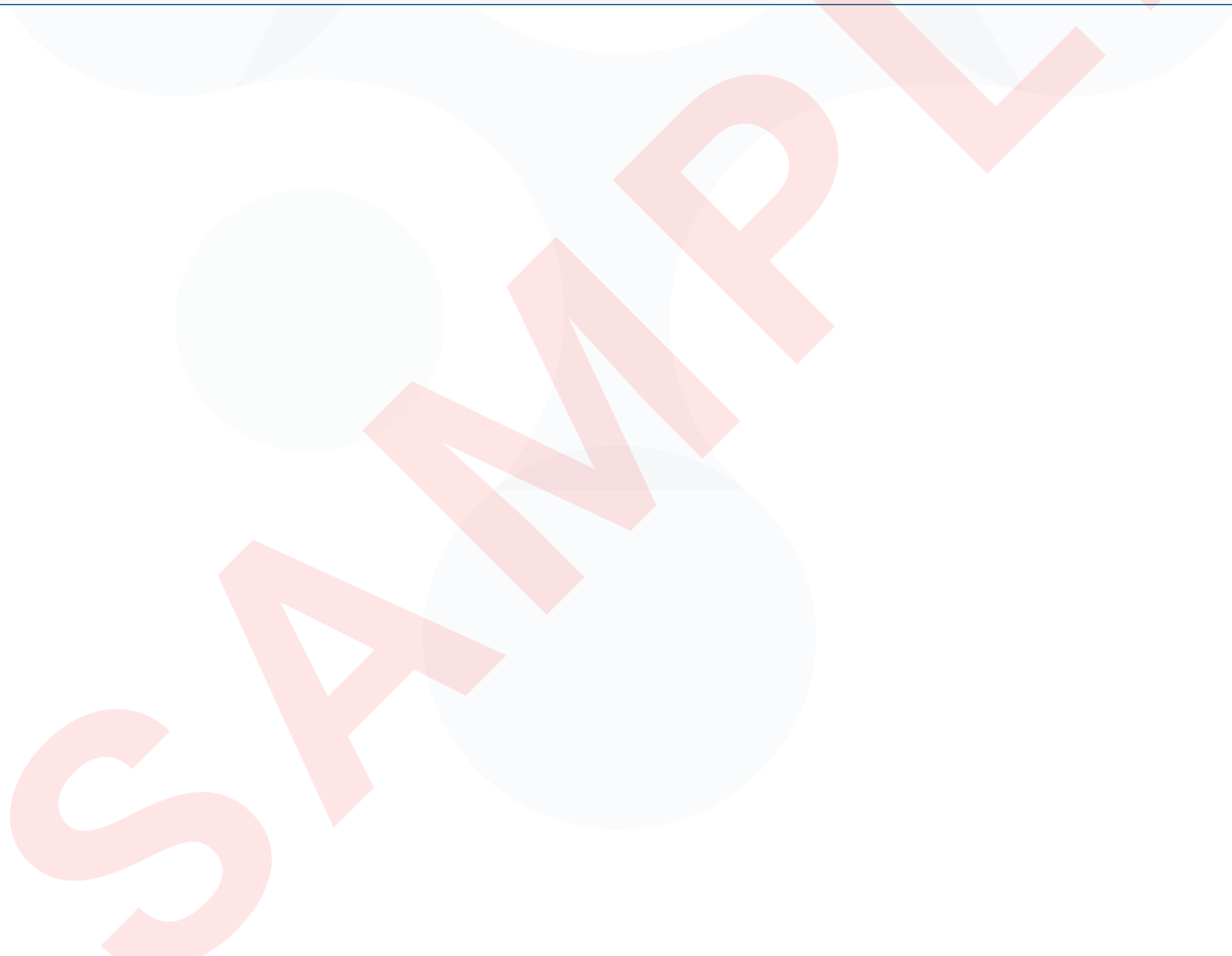
Angiotensin II R B	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
A1166C	AGTR1 +1166A/C	AGTR1	Impaired RAAS system	A/C	Partially elevated	A/A

The AGTR1 gene encodes angiotensin II receptor type 1, a key component in the signal transduction of the renin-angiotensin system (RAS). The RAS plays a fundamental role in regulating blood pressure. Therefore, the AGTR1 gene is crucial for blood pressure control, and polymorphisms in this gene are implicated in the pathogenesis of hypertension. Variants of AGTR1 can directly impact the RAS system, and their effects may be influenced by dietary potassium intake. Individuals with the AC genotype may have altered gene function and are associated with an increased risk of hypertension. Susceptible individuals are advised to consume a Dietary Approaches to Stop Hypertension (DASH) diet low in sodium. Regular exercise is recommended for health.

Drug Metabolism

ACE inhibitor	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs1799722	g.603C>T	BDKRB2	Enalapril non-responsive	C/C	Elevated	T/T, C/T

The BDKRB2 gene encodes the bradykinin receptor B2, a G-protein-coupled receptor responsible for mediating the effects of bradykinin, a peptide that promotes vasodilation, enhances nitric oxide production, and regulates vascular tone. Through these actions, it plays a critical role in maintaining normal blood pressure and vascular resistance. Mutations or polymorphisms in BDKRB2 can impair receptor function or expression, leading to reduced bradykinin signaling, increased vascular resistance, and a higher risk of developing hypertension. Because enalapril enhances bradykinin activity as part of its antihypertensive mechanism, variations in BDKRB2 can influence treatment outcomes. In general, individuals with functional receptor activity benefit from effective blood pressure reduction with enalapril, while impaired receptor function may limit the drug's full therapeutic effect. Individuals with the CC genotype typically exhibit a poor blood pressure-lowering response to enalapril, indicating reduced treatment efficacy. This may be due to diminished bradykinin B2 receptor-mediated vasodilation, resulting in higher vascular resistance and increased hypertension risk. Consult with healthcare professionals for personalized advice.



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

FOOD SOURCES	Fruits	Orange, Avocado, Banana, Strawberry, Apricot, Citrus Fruit, Tomato, Apple, Cantaloupe, Grapefruit
	Vegetables	Spinach, Broccoli, Brussels Sprout, Green Pea, Pea, Potato, Asparagus, Kale, Carrot, Leafy Green
	Dairy	Milk, Yogurt, Cheese, Butter, Cottage Cheese, Buttermilk, Soy Yogurt
	Fiber	Flaxseed, Nuts, Walnut, Lentil, Whole Grain, Beans, Legumes, Seed, Pea, Brown Rice
	Animal Protein	Salmon, Tuna, Egg, Mackerel, Sardine, Poultry, Chicken, Liver, Beef, Fish

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
	Ashwagandha	1000 mg/day	Flaxseed	250 mg/day	Resveratrol	500 mg/day
	Silymarin	420 mg/day	Aloe vera	300 mg/day	Anthocyanins	320 mg/day
	Citrus flavonoids	650 mg/day				

Suggestions

FOOD SOURCES

Fruits

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

Vegetables

Aloe Vera Leaf, Ashwagandha Root, Barberry, Brussels Sprout, Eggplant, Emblica Officinali, Garlic Bulb (allium Sativum), Goldenseal, Green Tea, Ocimum Sanctum

Fiber

Peanut, Almond, Brown Rice, Flaxseed, Milk Thistle Seed, 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

Suggestions

FOOD SOURCES

Vegetables

Onion, Turmeric

Fiber

Coffee Beans

Hormone Support

Hormone support compounds are designed to support optimal endocrine function and help maintain healthy hormone balance throughout various life stages and physiological changes. These agents work by providing hormone precursors or factors that support glandular function, modulating hormone metabolism, or enhancing cellular hormone sensitivity. Based on individual health assessments, this report provides recommendations for appropriate hormone-supporting supplements tailored to the specific endocrine imbalances identified. These recommendations serve as guidance and must be reviewed with a qualified healthcare provider to ensure proper selection, dosage, and monitoring protocols. Responsible use of hormone support supplements is essential to optimize endocrine function while preventing potential disruption of delicate hormonal feedback systems.



SUPPLEMENTS

Melatonin

0.5 mg/day

FOOD SOURCES

Fruits

Grape, Tart Cherry

Vegetables

Tomato

Fiber

Pistachio, Walnut

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Cardio Genetics - All Markers

Marker SNPs

Chylomicron/ Triglycerides	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs7903146	C>T	TCF7L2	Deposition of triglycerides	T/T	Elevated	C/C
rs12255372	c.483+9017G>T	TCF7L2	Deposition of triglycerides	T/T	Elevated	G/G
rs780094	A>G	GCKR	Elevated triglyceride levels	A/G	Elevated	G/G
rs1748195	c.1801-668G>C	ANGPTL3	Altered serum triglycerides concentrations	C/C	Elevated	G/G
rs2967605	C>T	ANGPTL4	Vascular inflammation and endothelial dysfunction	C/C	Elevated	T/T, C/T
rs328	c.1421C>G	LPL	High triglyceride levels	C/C	Elevated	G/G, C/G
rs7007797	T>G	LPL	High triglyceride levels	T/T	Elevated	G/G, G/T
rs264	c.776-172G>A	LPL	Impairs triglyceride metabolism	A/G	Elevated	G/G
rs320	T>G	LPL	Altered lipoprotein metabolism	T/T	Normal	T/T
rs72691625	g.-469G > A	GPIHBP1	Increased triglyceride levels	G/G	Normal	G/G
rs964184	G>C	ZNF259	Elevated triglyceride and LDL levels	C/C	Normal	C/C
rs2075291	c.553G > T	APOA5	Increased plasma triglyceride levels	G/G	Normal	G/G
rs662799	-1131T>C	APOA5	Elevated plasma triglyceride levels	T/T	Normal	T/T, C/T
rs3135506	c.56C > G	APOA5	Elevated plasma triglyceride levels	G/G	Elevated	C/C
rs5128	G>C	APOC3	High plasma APOC3, TG, TC, and LDL-C levels.	C/C	Normal	C/C
rs138326449	IVS2+1G>A	APOC3	Increased triglyceride levels	G/G	Normal	G/G
rs508384	g.100365004C>A	SCD1	Higher plasma apoB-48 levels	C/C	Normal	C/C
rs2167444	g.100364987T>A	SCD1	Higher plasma apoB-48 levels	T/T	Normal	T/T
LDL	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs6752026	P145S G>A	APOB	Higher levels of LDL-cholesterol	G/G	Elevated	A/A, A/G
rs693	c.7545C>T	APOB	High levels of APOB, TG, TC and LDL-C, and lower levels of HDL-C	T/T	Elevated	C/C
rs515135	A>G	APOB	Elevated LDL cholesterol level	G/G	Elevated	A/A

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Cardio Genetics - All Markers

Marker SNPs

LDL	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs429358	ε2/ε2, ε2/ε3	APOE	Amyloid plaque buildup	ε3/ε3	Normal	ε3/ε3, ε1/ε4, ε1/ε3, ε1/ε2
rs7412	ε2/ε2, ε2/ε3	APOE	Amyloid plaque buildup	ε3/ε3	Normal	ε3/ε3, ε1/ε4, ε1/ε3, ε1/ε2
rs1800591	-493G > T	MTP	Higher LDL and total cholesterol levels	G/T	Elevated	T/T
rs599839	G>A	SORT1	Elevated LDL cholesterol levels	A/G	Elevated	G/G
rs688	c.1773C>T	LDLR	Elevated LDL cholesterol levels	C/C	Normal	C/C
rs72658855	c.90C>T	LDLR	Accumulation of LDL-cholesterol molecules	C/C	Elevated	T/T, C/T
rs12071264	c.533-22A>G	LDLRAP1	Increased levels of LDL-cholesterol	A/A	Elevated	G/G, A/G
rs562556	c.1420G>A	PCSK9	Increased LDL_C levels	A/A	Elevated	G/G, A/G
rs45613943	c.799+158T>C	PCSK9	Increased LDL-cholesterol levels	T/T	Elevated	C/C, C/T
rs4076317	C>G	ANGPTL4	Higher LDL and total cholesterol levels	C/G	Elevated	G/G
HDL	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs17120425	c.1906G>A	SIDT2	Lower HDL levels	Val/Val	Normal	Val/Val
rs5888	c.1050T>C	SCARB1	Altered lipid metabolism	C/T	Elevated	C/C
rs1532624	c.658+186C>A	CETP	Disrupted lipid transport	C/C	Elevated	A/A, A/C
rs247616	C>T	CETP	Disrupted lipid transport	C/C	Elevated	T/T
rs708272	CETP +279 G>A	CETP	Altered lipid metabolism	G/G	Normal	G/G
rs632153	G>T	APOA1	Elevated TC, LDLC, TG and VLDL levels	G/G	Normal	G/G
rs670	G>A	APOA1	Elevated TC, LDLC, TG and VLDL levels	G/G	Normal	G/G
rs5082	APOA2 -265T>C	APOA2	Higher total cholesterol, triglyceride, Cholesterol/HDLc ratio and non-HDL cholesterol levels	T/T	Elevated	C/C
Lp(a)	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs10455872	A>G	LPA	Increased lipoprotein(a) levels	A/A	Normal	A/A
rs3798221	G>T	LPA	Increased lipoprotein(a) levels	G/G	Elevated	T/T

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Cardio Genetics - All Markers

Marker SNPs

Lp(a)	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs6415084	T>C	LPA	Increased lipoprotein(a) levels	C/C	Normal	C/C
rs7770628	C>T	LPA	Increased lipoprotein(a) levels	C/C	Elevated	T/T
rs6752026	P145S G>A	APOB	Higher levels of LDL-cholesterol	G/G	Elevated	A/A, A/G
rs693	c.7545C>T	APOB	High levels of APOB, TG, TC and LDL-C, and lower levels of HDL-C	T/T	Elevated	C/C
rs515135	A>G	APOB	Elevated LDL cholesterol level	G/G	Elevated	A/A
oxLDL	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs662	Q192R A>G	PON1	High LDL oxidation	A/A	Normal	A/A
rs4845625	T>C	ILR-6	Triggered inflammatory responses	C/T	Elevated	C/C
rs1050450	198C > T	GPX1	Oxidative stress	C/T	Partially elevated	C/C
Macrophages	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs2107545	T>C	MPO	Oxidative stress	T/T	Normal	T/T
rs1800482	(-954G>C)	NOS2	Excessive Nitric oxide production	C/C	Elevated	G/G
rs3730017	c.661C>T	NOS2	Excessive Nitric oxide production	C/C	Elevated	T/T, C/T
Platelets(Clotting risk)	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs4252120	c.1256+9T>C	PLG	Impaired fibrinolysis	T/T	Elevated	C/C
rs1799963	20210 G>A	Prothrombin	Higher levels of plasma prothrombin	G/G	Normal	G/G
rs3136516	A19911G	Prothrombin	Excessive blood clot	A/G	Partially elevated	A/A
rs6025	1691G>A	Factor V Leiden	Abnormal blood clots in blood vessels	G/G	Normal	G/G
Foam cell	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs1412444	C>T	LIPA	Plaque deposition	C/T	Partially elevated	C/C
rs2246833	C>T	LIPA	Plaque deposition	C/T	Partially elevated	C/C
rs671	c.1510G>A	ALDH2	Increased oxidative stress	G/G	Elevated	A/A, A/G

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Cardio Genetics - All Markers

Systemic SNPs

Nitric Oxide synthesis	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs1879417	C > T	NOS1	Disrupted nitric oxide production	C/T	Normal	T/T, C/T
rs1549758	774 C>T	NOS3	Impaired eNOS function	C/C	Normal	C/C
rs2070744	NOS3 -786T/C	NOS3	Reduced NO levels	T/T	Normal	T/T
rs3918226	NOS3 -690 C>T	NOS3	Impaired eNOS function	C/C	Normal	C/C, C/T
rs1799983	894 G>T	NOS3	Reduced NO levels	G/G	Normal	G/G

Vascular health	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs10947789	g.39207146T>C	KCNK5	Impaired vascular smooth muscle function	T/T	Elevated	C/C
rs9982601	C>T	KCNE2	Impaired vascular function	C/C	Normal	C/C
rs1042714	c.79G>C	ADR-B2	Adverse cardiovascular events	G/G	Elevated	C/C

Vascular remodeling	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs12526453	c.251-126053C>G	PHACTR1	Affected endothelial nitric oxide synthesis	C/C	Elevated	G/G
rs169713	T>C	PHACTR1	Endothelial dysfunction	T/T	Normal	T/T
rs10116277	G>T	9p21	Coronary heart disease	T/T	Elevated	G/G, G/T
rs4977574	A>G	9p21	Myocardial infarction	G/G	Elevated	A/A, A/G
rs1333049	G>C	9p21	Coronary artery disease	C/G	Partially elevated	G/G
rs10757278	A>G	9p21	Coronary artery disease	A/G	Partially elevated	A/A
rs2383207	A>G	9p21	Coronary heart disease	G/G	Elevated	A/A
rs2383206	A>G	9p21	Myocardial infarction	G/G	Elevated	A/A

Vascular tone	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs2271037	T>G	CORIN	Altered blood pressure	G/T	Partially elevated	G/G
rs3749585	T>C	CORIN	Altered blood pressure	C/T	Partially elevated	T/T
rs75770792 (T555I)	c.1664C>T	CORIN	Reduced catalytic activity of Corin	C/C	Normal	C/C

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Cardio Genetics - All Markers

Systemic SNPs

Vascular tone	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs111253292 (Q568P)	c.1703A>C	CORIN	Reduced catalytic activity of corin	A/A	Normal	A/A
rs9332982	c.195+733C>T	CYP4A11	Elevated blood pressure	C/C	Normal	C/C
rs1126742	8590 T>C	CYP4A11	Elevated blood pressure	C/T	Partially elevated	T/T
rs2108622	c.1297G>A	CYP4F2	Impaired vascular function	C/C	Normal	C/C
rs1799998	CYP11B2-344C/T	CYP11B2	Altered blood pressure	T/T	Elevated	C/C
rs4961	c.1378G>T	ADD1	Elevated blood pressure	G/T	Partially elevated	G/G
rs5370	c.594G>T	EDN1	Increased vasoconstriction	G/G	Normal	G/G
Atrial fibrillation	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs2200733	C >T	4q25	Atrial fibrillation	C/C	Normal	C/C
rs10033464	T>G	4q25	Atrial fibrillation	G/T	Partially elevated	G/G
Angiogenesis	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs1122608	c.4521-5330G>T	SMARCA4 or BRG1	Impaired vascular smooth muscle function	G/G	Normal	G/G
rs11556924	C>T	ZC3HC1	Endothelial dysfunction	C/T	Partially elevated	T/T
rs2010963	c.945+241342C>G	VEGF-A	Arterial stiffness, Endothelial dysfunction and High blood pressure	G/G	Normal	G/G, C/G
Metabolic Risk	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs1801133	677C>T	MTHFR	Active folate deficiency	C/C	Normal	C/C
rs1801131	A1298C; A>C	MTHFR	Active folate deficiency	A/C	Partially elevated	A/A
rs4680	COMT-475G > A	COMT	Optimum COMT gene activity	G/G	Normal	G/G
rs1761667	c.-180+13244G>A	CD36	Increased blood pressure	A/G	Normal	A/A, A/G
rs10911021	CC	GLUL	Affected glutamine metabolism	C/C	Normal	C/C
Atherosclerosis	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs2023938	A>G	HDAC9	Vascular inflammation	A/G	Partially elevated	A/A

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Cardio Genetics - All Markers

Systemic SNPs

Atherosclerosis	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs10757274	A>G	CDKN2B-AS1	Atherosclerotic plaque formation	G/G	Elevated	A/A
rs4238001	C>T	SCARB1	Decreased cholesterol clearance	C/C	Normal	C/C
rs2292318	C>T	LCAT	Altered lipid metabolism	C/C	Normal	C/C
rs4674344	c.256-4425A>T	CYP27A1	Plaque deposition	A/T	Partially elevated	A/A

Hypertension

Angiotensin II receptor	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
A1166C	g.148742201A>C	AGTR1	Valsartan efficacy	A/C	Normal	C/C, A/C
Angiotensin Converting Enzyme	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs4343	c.2328G>A	ACE	Elevated blood pressure	A/G	Normal	A/A, A/G
rs4646994	I/D	ACE	Altered blood pressure	I/D	Partially elevated	I/I
Angiotensinogen	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs699	c.776T>C	AGT	Elevated blood pressure	T/T	Normal	T/T
rs4762	c.593C>T	AGT	Elevated blood pressure	C/C	Normal	C/C
rs5051	G>A	AGT	Higher angiotensin II levels	G/G	Normal	G/G

Drug Metabolism

Metformin	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs7757336	A>G	OCT2 gene	Increased metformin concentration	A/A	Normal	A/A
rs201919874	G>A	SLC22A2	Increased metformin concentration	G/G	Elevated	A/A, A/G
Statin	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
6986A>G	c.219-237A>T	CYP3A5 *3/*3	Increased statin concentration in blood	CYP3A5*3/*3	Elevated	CYP3A5*1/*1
421C>A	421C>A	BCRP (ABCG2)	Increased statin concentration in blood	C/C	Normal	C/C
rs4149056	T521C	SLCO1B1	Increased plasma statin concentration	C/T	Partially elevated	T/T

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Cardio Genetics - All Markers

Drug Metabolism

Statin	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs1057910	A>C	CYP2C9	Increased statin clearance	CYP2C9*1/*1	Normal	CYP2C9*1/*1
rs20455	Trp719Arg, c.2155T>C	KIF6	Effective statin treatment	Trp/Arg	Normal	Arg/Arg, Trp/Arg
Ezetimide	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
c388A > G	c388A > G	OATP1B1*1b (SLCO1B1)	Increased ezetimibe concentration	G/G	Elevated	A/A
Niacin	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs738409	c.444C>G	PNPLA3	Hindered niacin uptake	C/C	Normal	C/C
rs1944438 C > T	C>T	DGAT2	Hindered niacin uptake	C/C	Normal	C/C
Clopidogrel	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs4244285	681 G>A	CYP2C19*2	Clopidogrel resistance	G/G	Normal	G/G
Coenzyme Q10	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs1800566	Pro187Ser, c.559C>A	NQO1	Hindered COQ10 uptake	P/P	Normal	P/P
Caffeine	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs762551	AA	CYP1A2	Slow caffeine metabolizers	A/C	Partially elevated	A/A
Aspirin	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs2071746	413A>T	HO1	Aspirin resistance	A/A	Normal	A/A
rs1330344	-1676A>G	COX-1	Aspirin resistance	T/T	Normal	T/T
-1188T>C	-1188T>C	CYP2C9	Slow metabolizers of aspirin	T/T	Normal	T/T
rs3798220	T>C	LPA	Aspirin non-responsiveness	T/T	Elevated	C/C, C/T
Angiotension II R B	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
A1166C	AGTR1 +1166A/C	AGTR1	Impaired RAAS system	A/C	Partially elevated	A/A
ACE inhibitor	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs5182	c.573C>T	AT1R	Captopril resistance	C/T	Normal	C/C, C/T

Drug Metabolism

ACE inhibitor	Variant	Gene Name	Risk Association	Your Genotype	Your Risk	Non-risk Genotype
rs1799722	g.603C>T	BDKRB2	Enalapril non-responsive	C/C	Elevated	T/T, C/T



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.

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.