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CARDIOVASCULAR GENETICS

FROM GENES TO HEALTHIER HEARTS

PREDICT
PREVENT
PERSONALIZE
PROTECT

GENES

RISK

PREVENTION

PRECISION

LONGER
HEALTHIER LIVES



GENETIC
RISK ASSESSMENT

EARLIER
DETECTION

PERSONALIZED
CARE

HEALTHIER
GENERATIONS

YOUR HEART'S FUTURE STARTS IN YOUR DNA

CARDIOVASCULAR GENETICS

— Common Variant (rs) Cardiovascular Predisposition Report

PATIENT · SAMPLE · LABORATORY

PATIENT	**John Doe**	REPORT NO	**CVG-JD-2026-001**
SAMPLE NO	**GSA-2026-09-JD001**	SAMPLE TYPE	**Buccal swab**
GENOTYPING PLATFORM	**Illumina Global Screening Array v4 — sample template**	GENOME BUILD	**GRCh38**
REPORT DATE	**18 Sep 2026**	REPORT TYPE	**Common variant (rs) genetics only**
REPORT STATUS	**Sample patient — all genotypes are fictional**		

SCOPE OF THE REPORT

This document reports selected common genetic variants associated in the literature with cardiovascular traits. It does not include clinical history, family history, laboratory measurements, clinical risk scores, diagnosis, treatment recommendations or monogenic disease screening results. Each result describes a population-level genetic association, not an individual disease prediction.



About / *This Report*

Cardiovascular traits reflect the combined contribution of many genetic variants and non-genetic factors. The selected rs variants in this report relate to lipoprotein(a), LDL cholesterol, triglyceride metabolism, coronary artery disease predisposition, glucose metabolism, vascular biology and the electrical properties of the heart.

The report uses only two result fields for each variant:

– **What your gene does:** the relevant biological role of the gene or region.
– **What your result means:** an evidence-based interpretation of the reported genotype.

Grades describe the relative strength and reproducibility of the association in this panel. They do not indicate disease severity, certainty, clinical urgency or cardiovascular fitness.

GRADING SCALE

GRADE	MEANING IN THIS REPORT
★★★★★	**Strong genetic association with a directly measurable cardiovascular trait**
★★★★☆	**Well-replicated common variant association of established biological importance**
★★★☆☆	**Replicated association with moderate individual effect**
★★☆☆☆	**Limited individual predictive value; genetic background information**

Genetic Profile / *Overview*

COMMON VARIANT PROFILE BY DOMAIN

GENETIC DOMAIN	JOHN DOE'S COMMON VARIANT PROFILE	GRADE
Lipoprotein(a) predisposition	**One allele linked to high Lp(a) at LPA rs10455872; common genotype at rs3798220**	★★★★★
LDL-C / apoB pathway	**No PCSK9 p.Arg46Leu LDL-lowering allele; common LDLR/APOB context; pattern consistent with APOE ε3/ε4**	★★★★☆
Triglyceride metabolism	**APOA5 rs662799 TT; one minor allele at LPL rs328 linked to a lower triglyceride tendency**	★★★★☆
Coronary disease genetics	**Heterozygous 9p21 predisposition locus pattern and a low-effect MIA3 marker**	★★★★☆
Glucose / diabetes predisposition	**One TCF7L2 risk allele and additional low-effect metabolic loci**	★★★☆☆
Blood pressure genetics	**Candidate vascular regulation loci; low individual predictive value**	★★☆☆☆
Rhythm trait genetics	**Heterozygous 4q25/PITX2 atrial fibrillation predisposition context; non-diagnostic sodium channel markers**	★★★☆☆

LPA Genetics / *Predisposition*

Lipoprotein(a), or Lp(a), is an LDL-like particle carrying apolipoprotein(a). Its blood level is largely determined by variation in the LPA gene region.

VARIANTS IN THIS SECTION

RS	GENE / REGION	GENOTYPE	GRADE
rs10455872	**LPA**	AG	♥♥♥♥♥
rs3798220	**LPA**	CC	♥♥♥♥♡

rs10455872 — LPA

Genotype: **AG** ♥♥♥♥♥

WHAT DOES YOUR GENE DO?

LPA encodes apolipoprotein(a), the defining component of Lp(a) particles, and largely determines the blood Lp(a) level.

WHAT DOES YOUR RESULT MEAN?

John Doe carries one G allele. In population studies this allele has been associated with higher Lp(a) levels and increased coronary artery disease predisposition. It is one of the most consistently reported common genetic signals for Lp(a)-related cardiovascular risk.

rs3798220 — LPA

Genotype: **CC** ♥♥♥♥♡

WHAT DOES YOUR GENE DO?

This LPA variant lies in the same genomic region that regulates apo(a) structure and blood Lp(a) levels.

WHAT DOES YOUR RESULT MEAN?

John Doe has the common CC genotype and does not carry the minor allele linked to higher Lp(a) in many cohorts. This result does not cancel the rs10455872 finding, because multiple SNPs and structural variation in the LPA region contribute to Lp(a) biology.

LDL Receptor / *and ApoB Pathway*

This section covers genes involved in hepatic clearance of LDL particles, apolipoprotein-B particle structure and remnant lipoprotein processing. The listed variants are common markers; this is not a familial hypercholesterolemia test.

VARIANTS IN THIS SECTION

RS	GENE	GENOTYPE	GRADE
rs11591147	**PCSK9**	GG	
rs2228671	**LDLR**	CC	
rs693	**APOB**	AG	
rs429358	**APOE**	TC	
rs7412	**APOE**	CC	

rs11591147 — PCSK9 (p.Arg46Leu)

Genotype: GG

WHAT DOES YOUR GENE DO?

PCSK9 controls the recycling of LDL receptors in the liver. Lower PCSK9 activity leaves more receptors on the liver cell surface and facilitates LDL particle clearance.

WHAT DOES YOUR RESULT MEAN?

John Doe is homozygous GG and does not carry the T allele encoding the naturally LDL-lowering p.Arg46Leu variant. In large cohorts the T allele has been associated with lower LDL-C and lower coronary heart disease risk. GG is the reference/common genotype here; it is not evidence of a pathogenic PCSK9 variant.

rs2228671 — LDLR

Genotype: CC

WHAT DOES YOUR GENE DO?

LDLR encodes the cell surface receptor responsible for receptor-mediated clearance of LDL particles from plasma, mainly by the liver.

WHAT DOES YOUR RESULT MEAN?

John Doe carries CC, the common genotype at this LDL-C-associated locus. Common variation in or near LDLR may contribute to small average differences in LDL-C; however, this genotype can neither show nor exclude a pathogenic LDLR variant. Pathogenic LDLR variants are the main molecular cause of familial hypercholesterolemia.

rs693 — APOB

Genotype: AG

WHAT DOES YOUR GENE DO?

APOB produces apolipoprotein B, the essential structural protein of LDL, IDL, VLDL and other atherogenic particles.

WHAT DOES YOUR RESULT MEAN?

John Doe carries one copy of each allele at this common APOB lipid marker. This locus has been associated with small population-level shifts in total cholesterol and LDL-C. This genotype is not equivalent to a rare pathogenic APOB variant causing familial defective apoB.

rs429358 — APOE Genotype: TC

WHAT DOES YOUR GENE DO?

APOE encodes apolipoprotein E, which enables the uptake and clearance of remnant lipoproteins via liver receptors.

WHAT DOES YOUR RESULT MEAN?

Together with rs7412, this result contributes to determining the APOE haplotype. The TC result at rs429358 is consistent with an ε4-associated allele under the stated allele orientation. It should be interpreted with rs7412, not alone.

rs7412 — APOE Genotype: CC

WHAT DOES YOUR GENE DO?

Together with rs429358, this APOE coding region contributes to the ε2/ε3/ε4 haplotype structure that affects remnant particle metabolism.

WHAT DOES YOUR RESULT MEAN?

Assessed together with rs429358 TC, the genotype pattern is consistent with the APOE ε3/ε4 haplotype, provided haplotype phase and allele orientation are confirmed. ε4-associated haplotypes are on average linked to less favorable LDL cholesterol handling than ε3/ε3; effect size varies considerably between individuals and populations.

Triglyceride Metabolism / *Pathway*

Genes in this group are involved in the production and breakdown of triglyceride-rich lipoproteins and in hepatic metabolic regulation.

VARIANTS IN THIS SECTION

RS	GENE	GENOTYPE	GRADE
rs662799	**APOA5**	TT	
rs328	**LPL**	CG	
rs3135506	**APOA5**	GG	
rs1260326	**GCKR**	CT	

rs662799 — APOA5 Genotype: TT

WHAT DOES YOUR GENE DO?

APOA5 supports the metabolism and clearance of triglyceride-rich lipoproteins; it is a key regulator of plasma triglyceride levels.

WHAT DOES YOUR RESULT MEAN?

John Doe is TT at this locus and does not carry the C allele linked to higher triglycerides in many populations. A large collaborative analysis associated each C allele with about 16% higher triglycerides and a moderate increase in coronary heart disease risk.

rs328 — LPL (Ser447Ter) Genotype: CG

WHAT DOES YOUR GENE DO?

LPL produces lipoprotein lipase, the enzyme that breaks down triglycerides in circulating chylomicron and VLDL particles.

WHAT DOES YOUR RESULT MEAN?

John Doe carries one copy of the minor allele at this marker. In population studies this allele has been associated with lower triglycerides and higher HDL-C, consistent with more efficient clearance of triglyceride-rich particles.

rs3135506 — APOA5 Genotype: GG

WHAT DOES YOUR GENE DO?

This coding APOA5 variant affects a protein involved in triglyceride-rich lipoprotein metabolism.

WHAT DOES YOUR RESULT MEAN?

John Doe has the common GG genotype. Other alleles at this locus have been associated with triglyceride differences, but reported effect sizes vary across study populations. This result is supporting information within the APOA5 pathway, not a separate, independent risk result.

rs1260326 — GCKR Genotype: CT

WHAT DOES YOUR GENE DO?

GCKR regulates glucokinase activity in liver cells and affects the balance between hepatic glucose metabolism and triglyceride synthesis.

WHAT DOES YOUR RESULT MEAN?

John Doe carries the heterozygous CT genotype, which genetic association studies have linked to small average differences in triglyceride- and glucose-related traits. Its effect is moderate and reflects general metabolic background rather than a specific disease mechanism.

Coronary Disease / *Genetic Background*

Coronary artery disease is a polygenic (multi-gene) trait. The variants below are representative common loci identified in genome-wide association studies; they do not form a validated coronary artery disease polygenic risk score.

VARIANTS IN THIS SECTION

RS	GENE / REGION	GENOTYPE	GRADE
rs10757274	**9p21 / CDKN2B-AS1 region**	AG	
rs1333049	**9p21 / CDKN2B-AS1 region**	GC	
rs4977574	**9p21 / CDKN2B-AS1 region**	AG	
rs17465637	**MIA3**	AG	

rs10757274 — 9p21 / CDKN2B-AS1 region

Genotype: AG

WHAT DOES YOUR GENE DO?

This non-coding 9p21 region lies near the CDKN2A, CDKN2B and CDKN2B-AS1 regulatory region, involved in vascular cell proliferation, cellular senescence and arterial wall biology.

WHAT DOES YOUR RESULT MEAN?

John Doe carries AG, i.e. one copy of the risk-associated allele. The 9p21 region is one of the most reproducible common genetic associations with coronary artery disease. Early replication studies reported a moderate relative increase in coronary disease predisposition in heterozygous carriers and, on average, a larger association in homozygous risk genotypes.

rs1333049 — 9p21 / CDKN2B-AS1 region

Genotype: GC

WHAT DOES YOUR GENE DO?

This marker lies in the same 9p21 regulatory interval as rs10757274 and tags the broader coronary artery disease-associated haplotype.

WHAT DOES YOUR RESULT MEAN?

John Doe has the heterozygous GC genotype at this locus. This result supports the presence of a 9p21 coronary predisposition haplotype signal; however, it should not be counted separately from rs10757274, as these markers may be correlated through linkage disequilibrium.

rs4977574 — 9p21 / CDKN2B-AS1 region

Genotype: AG

WHAT DOES YOUR GENE DO?

This variant is another common marker in the chromosome 9p21 coronary disease region.

WHAT DOES YOUR RESULT MEAN?

John Doe carries the AG genotype, which includes one allele associated with coronary disease in genome-wide association data. Its interpretation supports the same 9p21 regional pattern and does not create an additional, independent coronary risk category.

rs17465637 — MIA3

Genotype: AG

WHAT DOES YOUR GENE DO?

MIA3 encodes melanoma inhibitory activity family member 3, a protein involved in intracellular transport and extracellular matrix biology. This locus has been identified in coronary artery disease genetic association studies.

WHAT DOES YOUR RESULT MEAN?

John Doe is heterozygous AG. The variant contributes to a small but replicated common variant association with coronary artery disease predisposition; it is best interpreted as low-effect polygenic background.

Type 2 Diabetes / *Predisposition Context*

The markers below are associated with blood glucose regulation, insulin secretion or type 2 diabetes predisposition. They are common variants and do not establish a metabolic diagnosis.

VARIANTS IN THIS SECTION

RS	GENE	GENOTYPE	GRADE
rs7903146	**TCF7L2**	CT	♥♥♥♥♡
rs12255372	**TCF7L2**	GT	♥♥♥♡♡
rs1801282	**PPARG**	CG	♥♥♥♡♡
rs7756992	**CDKAL1**	AG	♥♥♡♡♡

rs7903146 — TCF7L2 Genotype: CT ♥♥♥♥♡

WHAT DOES YOUR GENE DO?

TCF7L2 regulates transcriptional pathways involved in pancreatic beta cell function, incretin signaling and insulin secretion.

WHAT DOES YOUR RESULT MEAN?

John Doe carries CT, i.e. one copy of the allele associated with increased type 2 diabetes predisposition in large genome-wide studies. This is one of the most consistently replicated common diabetes loci; however, it is a probabilistic association, not a diagnostic result.

rs12255372 — TCF7L2 Genotype: GT ♥♥♥♡♡

WHAT DOES YOUR GENE DO?

This TCF7L2 marker lies in the same gene region affecting blood glucose regulation and beta cell function.

WHAT DOES YOUR RESULT MEAN?

John Doe carries the GT genotype, associated with moderate type 2 diabetes predisposition in population studies. As this marker may be correlated with rs7903146, it should be considered an accompanying signal from the same pathway, not an independent finding.

rs1801282 — PPARG (Pro12Ala) Genotype: CG ♥♥♥♡♡

WHAT DOES YOUR GENE DO?

PPARG regulates fat cell differentiation, fat storage and insulin sensitivity pathways.

WHAT DOES YOUR RESULT MEAN?

John Doe is heterozygous CG at the Pro12Ala site. The variant has been widely studied for insulin sensitivity and type 2 diabetes predisposition; however, the direction and size of the effect may differ across cohorts and exposure conditions. This genotype represents a moderate metabolic genetic context.

rs7756992 — CDKAL1 Genotype: AG ♥♥♡♡♡

WHAT DOES YOUR GENE DO?

CDKAL1 is associated with pancreatic beta cell function and insulin secretion biology.

WHAT DOES YOUR RESULT MEAN?

John Doe carries AG at this common type 2 diabetes predisposition locus. The individual effect size is small; therefore this marker is included in the report as low-weight polygenic metabolic context.

Vascular Regulation / *Context*

Blood pressure and endothelial function are largely polygenic. These variants are included to provide biological context and have limited individual predictive value.

VARIANTS IN THIS SECTION

RS	GENE	GENOTYPE	GRADE
rs699	**AGT**	AG	♥♥♡♡♡
rs5186	**AGTR1**	AC	♥♥♡♡♡
rs1799998	**CYP11B2**	TC	♥♥♡♡♡
rs1799983	**NOS3**	GT	♥♥♡♡♡

rs699 — AGT Genotype: AG

♥♥♡♡♡

WHAT DOES YOUR GENE DO?

AGT encodes angiotensinogen, the precursor of angiotensin peptides in the renin–angiotensin–aldosterone system.

WHAT DOES YOUR RESULT MEAN?

John Doe is heterozygous AG at this candidate blood pressure locus. Associations with blood pressure traits have been reported; however, the marker's individual predictive value is low and effect estimates vary between populations.

rs5186 — AGTR1 Genotype: AC

♥♥♡♡♡

WHAT DOES YOUR GENE DO?

AGTR1 encodes the angiotensin II type 1 receptor, which transmits vasoconstrictive and sodium-retaining signals.

WHAT DOES YOUR RESULT MEAN?

John Doe has the AC genotype at this candidate vascular regulation marker. Current associations are not specific enough to infer an individual blood pressure phenotype or drug response profile.

rs1799998 — CYP11B2 Genotype: TC

♥♥♡♡♡

WHAT DOES YOUR GENE DO?

CYP11B2 encodes aldosterone synthase, the final enzyme in aldosterone production.

WHAT DOES YOUR RESULT MEAN?

John Doe carries TC at this promoter region marker. It has been studied in relation to blood pressure regulation; however, it does not identify primary aldosteronism, hypertension or mineralocorticoid excess.

rs1799983 — NOS3 Genotype: GT

♥♥♡♡♡

WHAT DOES YOUR GENE DO?

NOS3 encodes endothelial nitric oxide synthase, which produces nitric oxide for vascular signalling and endothelial regulation.

WHAT DOES YOUR RESULT MEAN?

John Doe carries GT at this common coding marker. Associations with endothelial and vascular traits have been reported, but the effect is small and insufficient to infer endothelial dysfunction or cardiovascular disease status.

Common Rhythm Traits / *Context*

The common variants below have been associated with cardiac electrical traits or atrial fibrillation predisposition. They do not diagnose inherited arrhythmia syndromes.

VARIANTS IN THIS SECTION

RS	GENE / REGION	GENOTYPE	GRADE
rs2200733	**4q25**	CT	♥♥♥♡♡
rs6800541	**SCN10A / SCN5A region**	CC	♥♥♡♡♡
rs1805124	**SCN5A**	CC	♥♥♡♡♡

rs2200733 — 4q25 (near PITX2)

Genotype: CT ♥♥♥♡♡

WHAT DOES YOUR GENE DO?

This region lies near PITX2, a transcription factor involved in heart development and left atrial electrophysiology.

WHAT DOES YOUR RESULT MEAN?

John Doe carries CT, i.e. one copy of the allele associated with atrial fibrillation predisposition in genome-wide association studies. The association is replicable, but it does not diagnose atrial fibrillation in an individual or predict when it may occur.

rs6800541 — SCN10A / SCN5A region

Genotype: CC ♥♥♡♡♡

WHAT DOES YOUR GENE DO?

The SCN10A/SCN5A genomic region affects cardiac sodium channel regulation and has been associated with ECG conduction traits.

WHAT DOES YOUR RESULT MEAN?

John Doe has the common CC genotype at this marker. The result does not indicate a conduction disorder, ECG phenotype or inherited sodium channel disease.

rs1805124 — SCN5A

Genotype: CC ♥♥♡♡♡

WHAT DOES YOUR GENE DO?

SCN5A encodes the alpha subunit of the main cardiac sodium channel, central to electrical conduction in heart muscle.

WHAT DOES YOUR RESULT MEAN?

John Doe carries CC at this common coding polymorphism. This common result is not equivalent to a pathogenic SCN5A variant and cannot be interpreted as evidence of Brugada syndrome, long QT syndrome or inherited arrhythmia.

References / *Evidence Base*

01 **ClinVar. LPA rs10455872 and coronary artery disease evidence record**
https://www.ncbi.nlm.nih.gov/clinvar/RCV001251438/

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03 **Chora JR, et al. The Clinical Genome Resource Familial Hypercholesterolemia Variant Curation Expert Panel consensus guidelines for LDLR variant classification. Genetics in Medicine**
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04 **Talmud PJ, et al. Triglyceride-mediated pathways and coronary disease: collaborative analysis of 101 studies. Lancet. 2010**
https://cris.maastrichtuniversity.nl/en/publications/triglyceride-mediated-pathways-and-coronary-disease-collaborati…

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https://www.ahajournals.org/doi/10.1161/CIRCGENETICS.109.913426

06 **Wang F, et al. Genome-wide association study of coronary artery disease. International Journal of Molecular Sciences. 2010**
https://pmc.ncbi.nlm.nih.gov/articles/PMC2958466/

07 **Schunkert H, et al. Clinical utility and implementation of polygenic risk scores for predicting cardiovascular disease: ESC clinical consensus statement. European Heart Journal. 2025**
https://pubmed.ncbi.nlm.nih.gov/39906985/

Limitations / *Scope*

01 This report covers common variants only; it is not a clinical history, laboratory, phenotyping, diagnostic or treatment document.

02 No SNP in this report diagnoses or rules out coronary artery disease, hypercholesterolaemia, hypertension, diabetes, cardiomyopathy, inherited arrhythmia syndrome or aortopathy.

03 The listed markers do not form a validated cardiovascular polygenic risk score. A clinical polygenic risk score requires a named score, defined weights, genome build matching, validated imputation/quality control and ancestry-aware calibration.

04 Correlated markers from the same region, including 9p21 and the two TCF7L2 variants, are not independent risk findings.

05 Genetic effect estimates may vary by ancestry, sex, age, environment and gene-gene interaction.

06 SNP-array testing does not assess many rare pathogenic variants, copy number variants or other genomic changes linked to monogenic cardiovascular disease.