



CVD StripAssays®

Identify the most relevant genetic variations to estimate the risk for Cardiovascular Diseases

Cardiovascular Diseases (CVD) are common, but in many cases can be avoided.

Atherosclerosis and venous thrombosis are the two major manifestations of CVD. Both are caused by complex interactions of environmental and genetic parameters.

An unhealthy lifestyle in combination with certain genetic variants can contribute to atherosclerosis. Relevant genes include those

involved in endothelial dysfunction, hyperlipidemia, hypertension, and inflammation.

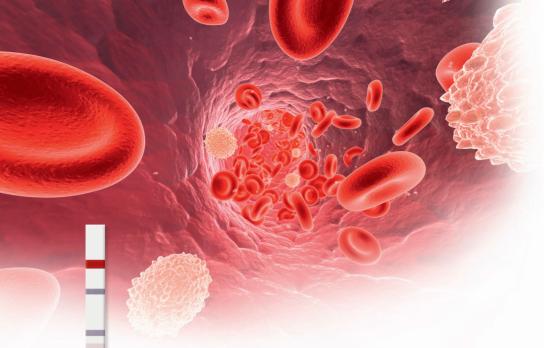
A combination of adverse influences (female hormone intake, immobilization, surgery or cancer) and variations in genes responsible for the coagulation system can also lead to thrombosis.

Testing for genetic variations and adequate prophylaxis contributes to lower CVD risks.

ViennaLab CVD StripAssays®

- Simple protocol for complex diagnostic questions
- Manual or automated processing
- No expensive lab equipment
- Ready-to-use reagents
- CE/IVD-labeled complete kits

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CVD StripAssays® detect genetic variants associated with Cardiovascular Diseases

Factor V (FV):

FV Leiden (1691G>A; R506Q):

represents one of the most important genetic risk factors for inherited thrombophilia; leads to activated protein C resistance; occurs in 20-50% of patients with VTE.

FV R2 haplotype (H1299R):

mild risk factor for thrombosis; increases CVD risk for carriers of FV Leiden.

Prothrombin (PTH; Factor II) 20210G>A:

carriers have about 3-fold elevated risk for cerebral and deep vein thrombosis; risk significantly increases in combination with FV Leiden; the A allele is associated with increased prothrombin levels.

5,10-Methylenetetrahydrofolate Reductase (MTHFR):

MTHFR 677C>T:

homozygosity predisposes to arterial and venous thrombosis in the presence of additional risk factors; the thermolabile variant (T allele) is associated with reduced enzyme activity and elevated plasma homocysteine levels in conjunction with folate deficiency.

MTHFR 1298A>C:

compound heterozygosity for 677C>T and 1298A>C is associated with reduced MTHFR enzyme activity.

Factor XIII (FXIII) V34L:

the L variant offers a protective effect against VTE.

Plasminogen Activator Inhibitor 1 (PAI-1, Serpin E1) 4G/5G:

The 4G allele is associated with higher PAI-1 transcription rates. It is considered to be a risk factor for VTE, MI and early pregnancy loss.

Endothelial Protein C Receptor (EPCR):

EPCR 4600A>G (A3 haplotype):

carriers of A3 are predisposed to VTE and fetal loss due to higher soluble EPCR plasma levels.

EPCR 4678G>C (A1 haplotype):

homozygous A1 exerts a protective effect in carriers of FV Leiden.

Apolipoprotein B (Apo B) R3500Q:

is a dominant but rare mutation, which is associated with severe hypercholesterolemia and elevated risk for atherosclerosis.

Apolipoprotein E (Apo E) E2/E3/E4:

the E4 allele is associated with increased susceptibility to early-onset MI, particularly in smokers; Apo E is an important predictor of the plasma lipid profile with E2 showing lowest and E4 showing highest LDL and total cholesterol levels.

Beta-Fibrinogen (FGB) -455G>A:

increases the risk for premature MI and ischemic stroke; confers elevated betafibrinogen plasma levels.

Human Platelet Antigen 1 (HPA1a/b; Gp Illa; integrin beta 3 L33P):

HPA1b is a risk factor for early-onset MI and stroke, particularly in smokers.

Angiotensin-Converting Enzyme (ACE) 287 bp insertion/deletion (I/D):

represents a risk factor for MI in elder patients and in smokers; the D allele is associated with elevated ACE activity and plasma levels.

MI: myocardial infarction, VTE: venous thromboembolism

Endothelial Nitric Oxide Synthase (eNOS; NOS3):

eNOS -786T>C:

the C allele causes a higher susceptibility to coronary heart disease.

eNOS 894G>T (E298D):

the T allele confers an increased risk for premature MI.

Lymphotoxin Alpha (LTA) 804C>A (T26N):

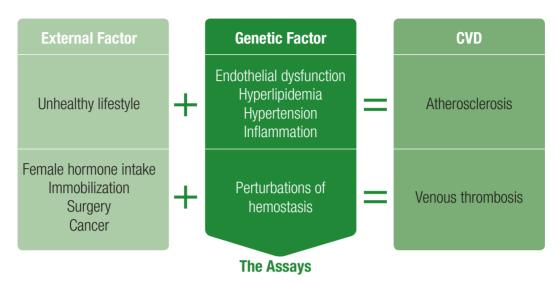
is in almost complete linkage with LTA 252A>G; both variants strongly act proinflamatory and are associated with coronary artery disease.

ViennaLab offers customers great flexibility to choose the optimal test between eight different CVD StripAssays[®]. Several genetic variants can be detected on a single teststrip.

Gene	Genetic Variant	FV StripAssay® 4-330	PTH StripAssay® 4-340	MTHFR StripAssay® 4-350	FV-PTH StripAssay® 4-290	FV-PTH-MTHFR StripAssay® 4-260	CVD StripAssay® T 4-360	CVD StripAssay® A 4-370	Apo E StripAssay® 4-280
FV	1691G>A (Leiden)	х			х	х	х		
	H1299R (R2)						х		
PTH	20210G>A		x		x	x	х		
MTHFR	677C>T			х		х	х		
	1298A>C						х		
FXIII	V34L						х		
PAI-1	4G / 5G						х		
EPCR	4600A>G (A3)						х		
	4678G>C (A1)						х		
АроВ	R3500Q							x	
ApoE	E2 / E3 / E4							x	х
FGB	-455G>A							х	
HPA1	a/b							x	
ACE	I/D							x	
eNOS	-786T>C							x	
	894G>T							х	
LTA	804C>A							x	



CVD StripAssays® identify the most important variations in genes that are relevant for atherosclerosis and venous thrombosis.



CVD StripAssays®

- are based on reverse-hybridization of biotinylated PCR products
- combine probes for variants and controls in a parallel array of allele-specific oligonucleotides
- work with immobilized oligos on a teststrip
- generate test results by enzymatic color reaction easily visible to the naked eye

The three steps of the StripAssays®

Step	Requirement
1. Amplification: Multiplex PCR. Simultaneous biotin-labeling	Thermocycler
2. Hybridization: Directly on the StripAssay® teststrips	Incubator
3. Identification: Labeled products detected by streptavidin-alkaline phosphatase	Naked eye or scanner & software

Order Information:

FV	StripAssay® 4-330 (20 tests/kit)	FV-PTH-MTHFR	StripAssay®	4-260 (20 tests/kit)
PTH	StripAssay® 4-340 (20 tests/kit)	CVD	StripAssay® T	4-360 (20 tests/kit)
MTHFR	StripAssay® 4-350 (20 tests/kit)	CVD	StripAssay® A	4-370 (20 tests/kit)
FV-PTH	StripAssay® 4-290 (20 tests/kit)	Apo E	StripAssay®	4-280 (20 tests/kit)

ViennaLab offers StripAssays® for a wide range of diagnostic applications. Visit www.viennalab.com



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