ProMedicDNA

Cardiovascular Sensor

Jane Doe
DEMO_DS

ProGenom
www.progenom.com

COVER LETTER

Dear Ms. Doe,

Your sample for the analysis arrived on in the laboratory and was evaluated according to the highest laboratory quality standards. The results were evaluated and released by two independent geneticists and molecular biologists. After obtaining the results, your personal report was compiled. We hereby convey the results to you in the format of your choice.

We would like to thank you for your trust and hope that you are satisfied with our service. We are always open to questions and suggestions. Please do not hesitate to contact us. We value your feedback. This is the only way we can continuously improve our services.

We hope the analysis meets your expectations.

Kind regards,

Dr. Daniel Wallerstorfer BSc.
Laboratory Director

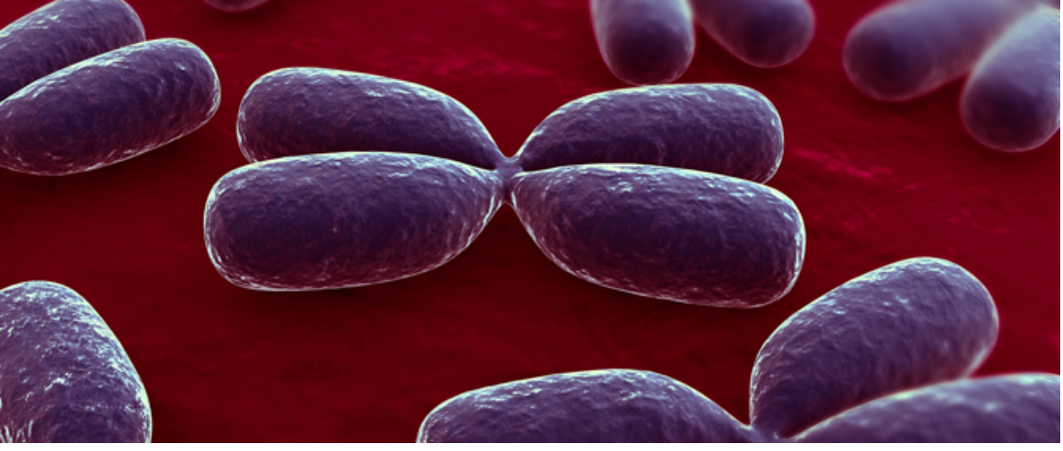
Florian Schneebauer, MSc.
Laboratory Manager

Cardiovascular Sensor

Personal analysis results for:
Jane Doe | Date of birth: 01/01/1990

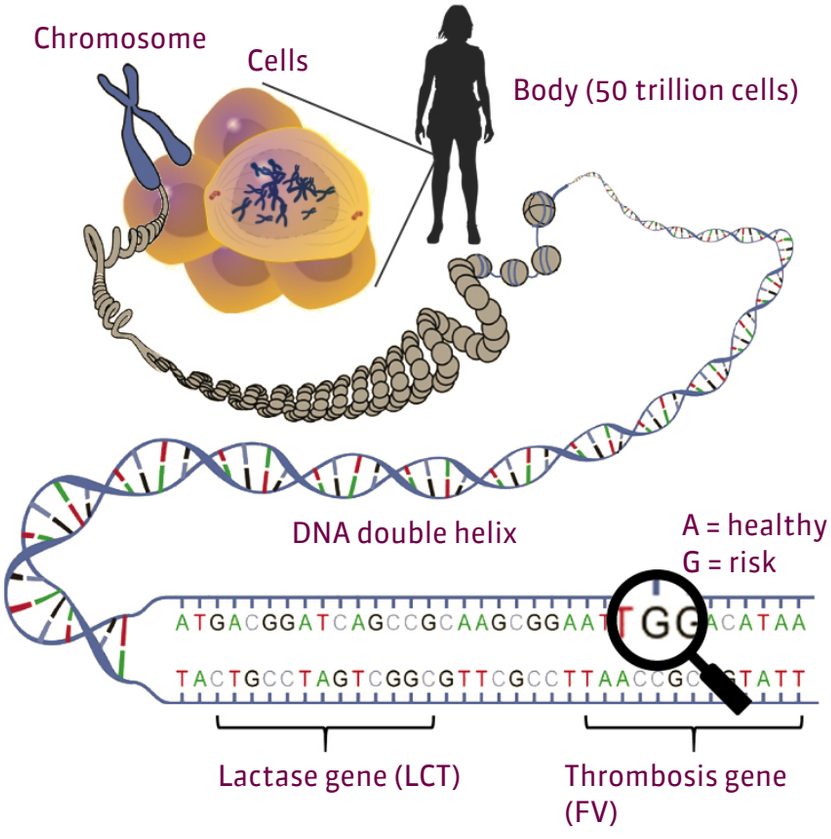
Order number:
DEMO_DS

This report contains personal medical information that is highly confidential. Data protection must be ensured.



How genes influence our health

The human body consists of about 50 trillion individual cells. Most of these cells have a nucleus, which contains 46 chromosomes. A chromosome consists of a very closely wound thread, the DNA "double helix."



DNA, the genetic code, is the blueprint of the human body. This genetic code consists of approximately 3.1 billion molecules, which are each represented by a letter. About 1% of this code makes up the genes. Each gene is an instruction for the body, usually with a single function. For example, some genes tell the body how to colour the iris and differences in these genes produce different eye colors. Every function of the body is controlled by one or more genes, including the way we break down food or medication.

Our genes are not completely error-free. The genes of each person are altered slightly by environmental effects. Most of these changes have no effect but a small number have a harmful effect. An even tinier number can produce a beneficial effect. Parents pass these changes, including defects, to their children. Thus most of our genetic defects are inherited from our parents.

In addition, our genes evolved to help us live in a completely different world, and some of our genetic traits can interact with our modern environment to create negative effects on the body. For example, the genetic predisposition to store dietary fat quickly and lose it slowly is beneficial for people who go through times when food is scarce: they have a better chance of surviving because their bodies use fat efficiently and store it for later. However, in the modern world, this trait is harmful because it programs the body to gain weight quickly and lose weight

slowly. Genes increase our risk of heart attacks, trigger asthma and allergies, cause lactose intolerance, and many other disorders.

Genetic traits can affect our health. While some genetic defects cause disease in all cases, most genetic traits just increase our risk of developing a disease. For example, a person may have genes that increase their risk for diabetes. However, not everyone at risk for diabetes actually develops the disease. Furthermore, even people with a high risk of diabetes can lower their risk with the right diet and exercise plan. Other genetic traits only cause illness when they are triggered by a specific environmental feature. For example, lactose intolerance is a genetic condition that causes a person who drinks milk to have digestive issues. A lactose-intolerant person who never drinks milk will not have any symptoms.

Thanks to the latest technologies, it is now possible to test specific genes to determine if you have genetic traits that are linked to various diseases. Based on the results of the analysis, we can develop a prevention program that significantly reduces your personal disease risk and helps you stay healthy.

A healthy lifestyle will decrease your risk of many diseases whether or not you have specific information about your genetic traits. However, we provide you with additional information that may point out other changes to your lifestyle that are not part of the standard medical advice. There are many examples, but one of the traits we test for is a gene that increases your body's ability to absorb iron. If you have this trait, you must not take iron supplements as the iron would accumulate and cause a life-threatening disease called haemochromatosis.

Experts estimate that every person carries about 2,000 genetic defects, which may affect their health, and in some cases, cause illnesses. A variety of factors can cause changes in our genes (also called mutations). In a few cases, these mutations can benefit us. However, the vast majority either have no effect or have a negative impact on our health. The best-known cause of mutations is radioactivity. Radioactive rays and particles actually impact the DNA in our cells and physically alter our genes. They mostly go unnoticed or cause deadly diseases, such as cancer, or congenital abnormality in newborns. Mutations are also caused by substances in burned food. The substances enter the cells and damage our genes, which can lead to colon cancer, among other forms of cancer. UV radiation from the sun can also damage our genes and cause diseases, such as skin cancer.

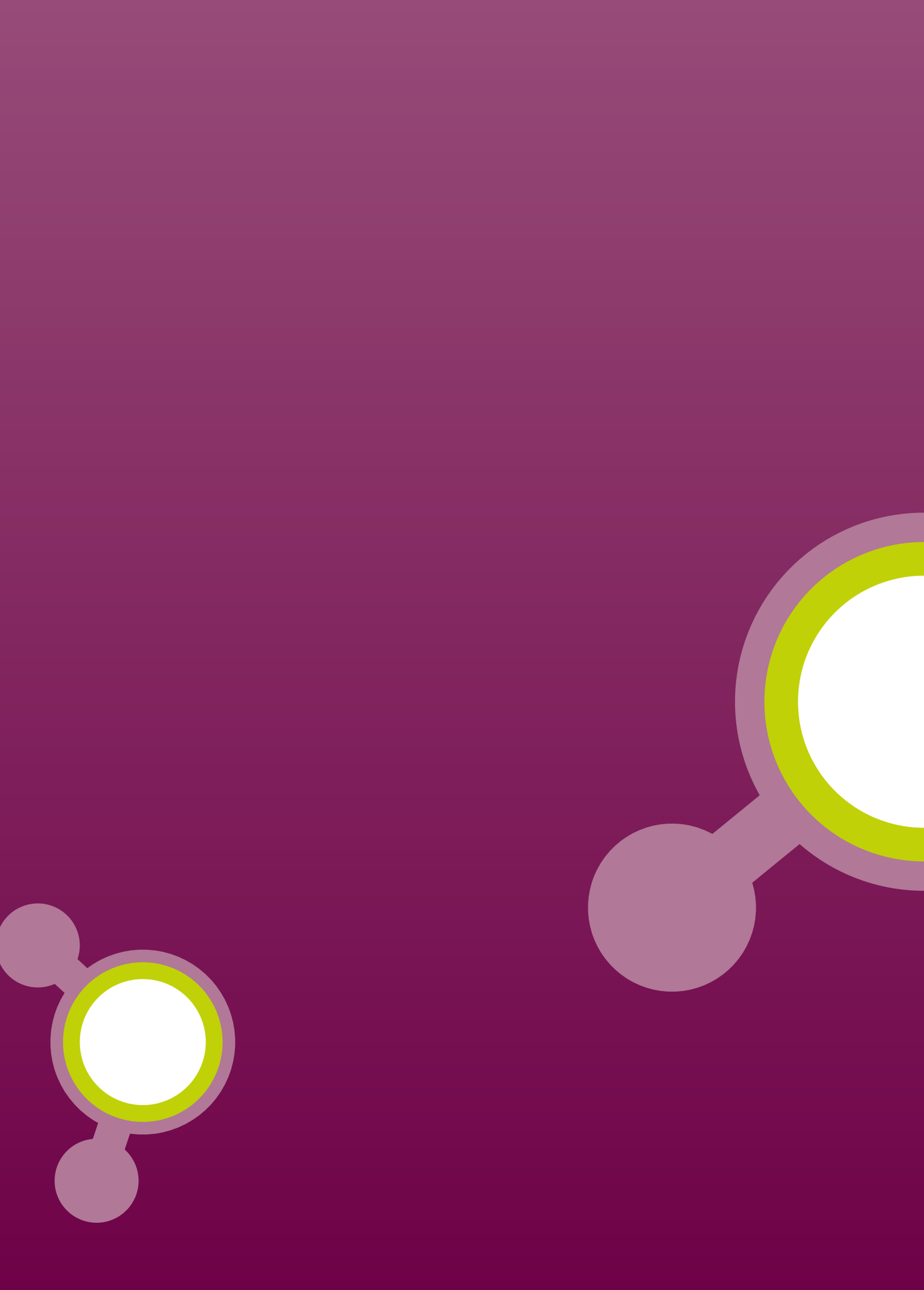
External influences can affect individual genes and disrupt their function, but the majority of our defective genes are inherited from our parents. Each embryo receives half of its genes from the father and half from the mother, resulting in a new human being with some characteristics of each parent. Whether a genetic defect is passed on, is determined randomly, and it may be that some of the children carry the defective gene and others do not.

Each person is the unique product of generations of accumulation and combination of different genetic traits. Some of those traits have negative effects on our health. With the latest technology, it is now finally possible to examine genes and determine personal health risks and strengths. In many cases, taking advantage of this knowledge, and following some precautionary measures, the diseases may be prevented. This is the next step in preventive medicine and a new generation of health care.

Action index

Discuss risks marked in orange or red with your doctor. All other results do not require any further attention assuming there are no current medical conditions.

Cardiovascular Sensor



PHARMACO GENETICS
Not ordered

ONCOLOGY
Not ordered

CARDIOVASCULAR SYSTEM

NEUROLOGY
Not ordered

METABOLISM
Not ordered

MOVEMENT
Not ordered

DIGESTION
Not ordered

OPHTHALMOLOGY
Not ordered

ODONTOLOGY
Not ordered

OTHERS
Not ordered

SCIENCE

ADDITIONAL INFORMATION

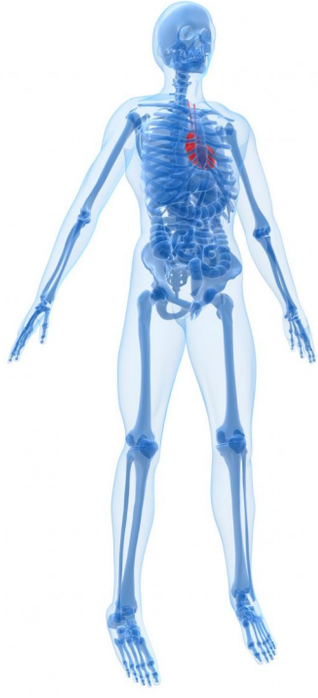
Cardiovascular Sensor

Prevention and treatment of lipid metabolism problems

Cardiovascular Disease

Cholesterol and triglycerides are vital fats our body uses to produce cell membranes, store energy, absorb fat-soluble vitamins and produce hormones. These fats are so essential, that our body even produces cholesterol itself. This cholesterol is then transported and made available for cell growth throughout the body via the bloodstream. In this way, our body produces about 70% of the cholesterol it requires itself. The other 30% comes from what we eat.

The amount of cholesterol in your bloodstream goes up after every meal. To control this, your body has strict regulatory processes that maintain your blood cholesterol at normal levels. If there is too much cholesterol in your bloodstream, your body transports HDL to your liver, which filters it from your blood, lowering your cholesterol level. However, the liver will release LDL cholesterol into your bloodstream, which increases your cholesterol level. High cholesterol levels may cause arteriosclerosis, therefore a low cholesterol level keeps you healthy.



Due to this, HDL cholesterol—the kind transported to the liver—is called "good" cholesterol, whereas LDL cholesterol—which the liver releases into the bloodstream—is called "bad" cholesterol.

That is why it is important for your health to maintain a high HDL and a low LDL cholesterol level.

A number of genes are responsible for regulating cholesterol and triglyceride levels, or for increasing the risk of cardiovascular disease. If you carry an adverse trait in one or more of these genes, you should pay special attention to your fat intake and metabolism. Since diet is the most significant influence on your body's lipid metabolism, it is important to follow a diet tailored to your genes.

Arrhythmia

Long QT syndrome is a life-threatening disease that can lead to sudden cardiac death in people with otherwise perfect health.

The heartbeat is triggered by a recurring electrical pulse which propagates through the heart. The time to initiate a heartbeat up to the point at which the cells are ready for the next heartbeat, is called the QT interval. If this interval is particularly long, it increases the risk of symptoms such as paroxysmal tachycardia, arrhythmia, vertigo or loss of consciousness. In severe cases, such episodes end in cardiac arrest due to ventricular fibrillation. However, most people with this condition have no symptoms until a life-threatening condition develops. The symptoms usually occur during physical exertion or stressful situations. A resting ECG (a measurement of the heart rate in the resting state) and a gene analysis help to better identify the risk.

A long QT interval is usually not noticeable: more than half of the patients with long QT syndrome experience no symptoms. When symptoms do occur, they are caused by potentially life-threatening heart rhythm disorders that are signs of serious disease. Palpitations may be sustained (more than 30 seconds) or intermittent, and sometimes remain unnoticed depending on the following: the duration and pulse rate, body position and the general constitution, dizziness, loss of consciousness or even cardiac arrest. Thus, they may lead to sudden cardiac death. Since tachycardia occurs suddenly and usually during exercise or in stressful situations, the symptoms are often unexpected, and observed because they affect our general state of well-being.

People with this genetic risk should take steps to minimize symptoms. The steps include medical heart rate monitoring in high-risk situations such as: cardiovascular disease, diabetes, morbidly excessive weight, age over 55 years, extreme physical activity, and taking certain medications. If a prolonged QT interval is diagnosed in these situations, medical treatment may be necessary. Thanks to genetic analysis, you can find out if you are in a risk group and take the necessary precautions. Serious consequences, such as sudden death, can usually be prevented.

Relevant genes for cardiovascular disease

The scientific community has linked several genes and polymorphisms to a risk of various cardiovascular diseases. An analysis of these polymorphisms allows us to determine your genetic risk for these diseases, as well as some other genetic traits linked to this disease.

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
CDH13	rs8055236	G>T	T/G
CHDS8	rs1333049	G>C	G/G
APOA5	rs662799	-1131T>C	A/A
PON1	rs662	Q192R	A/A
PON1	rs854560	L55M	A/A
APOB	rs5742904	R3500Q	G/G
SREBF2	rs2228314	Gly595Ala	C/C
NOS3	Ins/Del Int. 4	Ins/Del Intron 4	Ins/Ins
NOS3	rs2070744	-786 T/C	T/T
NOS3	rs1799983	Glu298Asp	G/T
APOA1	rs670	-75G > A	G/G
MTRR	rs1801394	Ile22Met	G/G
MMP3	rs3025058	5A/6A	T/del
GJA4	rs1764391	Pro319Ser	T/T
ITGB3	rs5918	Leu33Pro	T/T
CETP	rs708272	Taq1(B1>B2)	C/T
MTHFR	rs1801133	C>T	C/C
APOE	rs429358	T>C	T/T
APOE	rs7412	T>C	T/C
ApoE type	combination	E2/E3/E4	E2/E3
NOS1AP	rs16847548	T>C	T/T
NOS1AP	rs12567209	G>A	G/G
NOS1AP	rs10494366	T>G	T/T
CYP1A2	rs762551	C/A Pos. -163	A/A

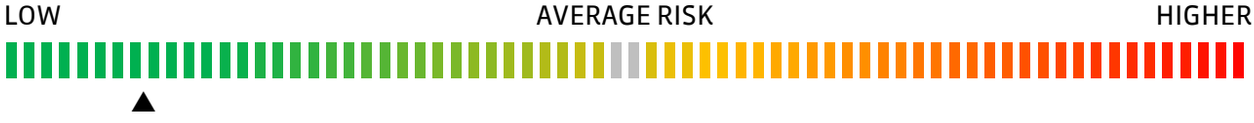
LEGEND: rsNCBI = description of examined genetic variation, POLYMORPHISM = form of the genetic variation, GENOTYPE = personal analysis result

Summary of effects

This analysis examines numerous genes that contribute to risks for several different cardiovascular conditions. Many of these risk variations are common, and almost everyone has some genes that increase the risk of cardiovascular disease. If you have an unusually small number of genes that increase risk, your genes may actually reduce your risk of developing cardiovascular disease. Here you can see a summary of the influence your genetic variations have on your health:

- You do not have an elevated risk of coronary heart disease
- No predisposition to elevated LDL cholesterol levels
- You have a slight predisposition for high triglyceride levels
- Vitamin B2 does not lower your homocysteine levels
- Omega-3 fatty acids reduce your HDL cholesterol levels
- Aspirin can be effective in preventing arterial thrombosis
- Predisposition for slightly lowered HDL cholesterol values
- No predisposition to increased QT-interval duration

Your risk of coronary heart disease, atherosclerosis and heart attack



Your LDL cholesterol levels are likely to be

NORMAL INCREASED

Your homocysteine levels are likely to be

NORMAL INCREASED

Your HDL cholesterol levels are likely to be

NORMAL LOW

Effect of vitamin B2 on homocysteine

REDUCTION NO

The effect of omega-3 on HDL cholesterol

IMPROVEMENT DETERIORATION

Effect of aspirin on arterial thrombosis (blood clots)

PROTECTION NO EFFECT

Your triglyceride levels are likely to be

NORMAL INCREASED

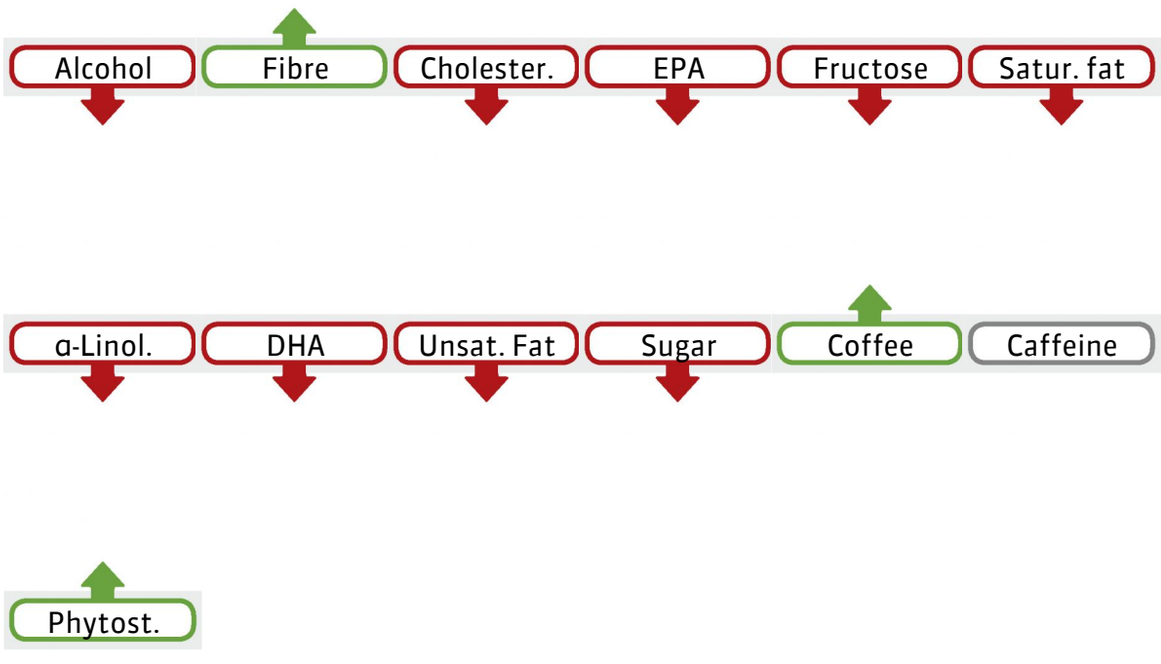
Risk of sudden cardiac death (QT)

NORMAL INCREASED

Nutritional Genes - Heart

Your nutrition is very important. Based on your genes and their associated strengths and weaknesses you should increase or decrease certain foods and nutrients. These recommendations are calculated based on your genetic profile.

Your personalized recommendations based on this section:

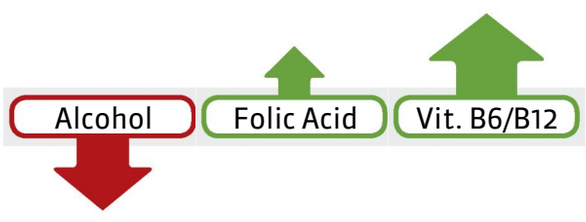


Legend: GREEN ARROWS > this nutrient or substance is classed as healthy for your genetic profile. Try to increase the intake of this substance. RED ARROWS > this substance is classed as unhealthy for your genetic profile. Try to reduce your intake of the substance. NO ARROWS > There is no effect of the nutrient on the genetics of this section. PLEASE NOTE! This interpretation only considers your genetic profile of this section.

Nutritional Genes - Blood

Your nutrition is very important. Based on your genes and their associated strengths and weaknesses you should increase or decrease certain foods and nutrients. These recommendations are calculated based on your genetic profile.

Your personalized recommendations based on this section:



Legend: GREEN ARROWS > this nutrient or substance is classed as healthy for your genetic profile. Try to increase the intake of this substance. RED ARROWS > this substance is classed as unhealthy for your genetic profile. Try to reduce your intake of the substance. NO ARROWS > There is no effect of the nutrient on the genetics of this section. PLEASE NOTE! This interpretation only considers your genetic profile of this section.

Nutritional Genes - Vitamin B2

Your nutrition is very important. Based on your genes and their associated strengths and weaknesses you should increase or decrease certain foods and nutrients. These recommendations are calculated based on your genetic profile.

Your personalized recommendations based on this section:

Vit B2

Legend: GREEN ARROWS > this nutrient or substance is classed as healthy for your genetic profile. Try to increase the intake of this substance. RED ARROWS > this substance is classed as unhealthy for your genetic profile. Try to reduce your intake of the substance. NO ARROWS > There is no effect of the nutrient on the genetics of this section. PLEASE NOTE! This interpretation only considers your genetic profile of this section.

Prevention

Based on the genes tested, you have no increased genetic risk for metabolic disorders and your risk is the same as the general population. However, even though you do not have genetic predisposition to these diseases, you can still get sick, especially if you follow an unhealthy lifestyle that leads to high levels of fat in the blood. We recommend you have your cholesterol and triglycerides checked every five years after age 20. If these values increase over time, you should take steps to reduce the risk of atherosclerosis. The specific steps will depend on the test results, so you should follow your doctor's advice.

Preventive measures

- Do sports or regular exercise. The best exercises are endurance sports (walking, Nordic walking, cycling, swimming, weight training, etc.), and watch your weight. Ideally, you should do at least 30 minutes of exercise, 5 days a week.
- Smoking greatly increases your risk for vascular disease along with its many other negative health effects. Quitting smoking is one of the most important ways to improve your health.
- In general, eat low-fat meals (fish, poultry and lean meats are recommended, but fatty meats like sausages, bacon and fat cheese should be reduced).
- You should also eat only low-fat dairy products, e.g. low-fat milk, low-fat cheese and low-fat yogurt.
- Eat as little animal products as possible and use mainly vegetable oils.
- Eat fruits and vegetables several times daily.

Omega-3-fatty acids

- Omega-3 fatty acids are commonly recommended for high cholesterol; however, because of your APoA1 gene, omega-3 fatty acids could raise your cholesterol levels. Phytosterols can be used as an alternative supplement.

Coffee

- Although coffee contains healthy ingredients, the caffeine contained in the coffee (if not metabolized by CYP1A2 gene) can increase the risk of cardiovascular diseases. Your CYP1A2 gene works normally and therefore moderate coffee consumption (2-5 cups a day) is healthy for your cardiovascular system.

Medical treatment is recommended when dietary changes and exercise do not lower cholesterol and triglycerides to a normal level. There are multiple options: statins, bile acid binders, fibrates, niacin (vitamin B3), and cholesterol synthesis and absorption inhibitors. Your doctor will decide which drug is suitable for you. It is particularly important to take action to prevent heart disease because treatment after symptoms develop can only slow down the progress of the disease.

Drug compatibility

DRUGS	139	310	107	262	221	276	524	371	12
GENES	CYP2E1	CYP2D6	CYP2B6	CYP1A2	CYP2C19	CYP2C9	CYP3A4	CYP3A5	NAT2
DEGRADATION	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NONE	NORMAL	NORMAL	SLOW

DRUGS	1	2
GENES	SLCO1B1	VKORC1
FUNCTION	SLOW	NORMAL

Effect on relevant medication

	Effect	Breakdown	Dose
Abciximab	✓	✓	✓
Alprenolol	✓	✓	✓
Amlodipine	✓	↑	↑
Atorvastatin	✓	↑	↑
Bendroflumethiazide	✓	✓	✓
Bisoprolol	✓	↑	↑
Captopril	✓	✓	✓
Cerivastatin	✓	↑	↑
Cilnidipine	✓	✓	✓
Clopidogrel	✓	↑	✓
Debrisoquine	✓	✓	✓
Dipyridamole	✓	✓	✓
Dorzolamide	✓	✓	✓
Encainide	✓	✓	✓
Eprosartan	✓	✓	✓

	Effect	Breakdown	Dose
Acebutolol	✓	✓	✓
Amiloride	✓	✓	✓
Anagrelide	✓	✓	✓
Barnidipine	✓	✓	✓
Benidipine	✓	✓	✓
Bumetanide	✓	✓	✓
Carvedilol	✓	✗	✗
Chlortalidone	✓	✓	✓
Cilostazol	✓	↑	↑
Colestipol	✓	✓	✓
Digoxin	✓	✓	✓
Disopyramide	✓	↑	↑
Dronedarone	✓	✓	✓
Enoxaparin	✓	✓	✓
Eptifibatide	✓	✓	✓

	Effect	Breakdown	Dose
Acetylsalicylic Acid	✓	✗	✗
Amiodarone	✓	↓	↓
Atenolol	✓	✓	✓
Benazepril	✓	✓	✓
Betaxolol	✓	✓	✓
Candesartan	✓	↓	↓
Celiprolol	✓	✓	✓
Cilazapril	✓	✓	✓
Clevidipine	✓	✓	✓
Cyclopenthiazide	✓	✓	✓
Diltiazem	✓	↑	↑
Dofetilide	✓	↑	↑
Enalapril	✓	✓	✓
Eplerenone	✓	↑	↑
Esmolol	✓	✓	✓

	Effect	Breakdown	Dose
Ezetimibe	✓	✓	✓
Fenofibrate	✓	✓	✓
Fondaparinux	✓	✓	✓
Gemfibrozil	✓	↑	↑
Ibutilide	✓	✓	✓
Isosorbide Mononitrate	✓	✓	✓
Lacidipine	✓	↑	↑
Lisinopril	✓	✓	✓
Manidipine	✓	✓	✓
Metoprolol	✓	✓	✓
Nadolol	✓	✓	✓
Nifedipine	✓	↑	↑
Nisoldipine	✓	↑	↑
Perhexiline	✓	✓	✓
Pitavastatin	✓	✓	✓
Procainamide	✓	✓	✓
Quinapril	✓	✓	✓
Ranolazine	✓	✓	✓
Sotalol	✓	✓	✓
Telmisartan	✓	✓	✓
Ticagrelor	✓	✓	✓
Tocainide	✓	✓	✓
Triamterene	✓	✓	✓
Vernakalant	✓	✓	✓

	Effect	Breakdown	Dose
Felodipine	✓	↑	↑
Flecainide	✓	✓	✓
Fosinopril	✓	✓	✓
Hydralazine	✓	✓	✓
Indapamide	✓	✓	✓
Isradipine	✓	↑	↑
Lercanidipine	✓	↑	↑
Losartan	✗	↓	✗
Methazolamide	✓	✓	✓
Mexiletine	✓	✓	✓
Nebivolol	✓	✓	✓
Nilvadipine	✓	✓	✓
Nitrendipine	✓	↑	↑
Perindopril	✓	✓	✓
Prasugrel	✓	✓	✓
Propafenone	✓	✓	✓
Quinidine	✓	↑	↑
Rosuvastatin	✓	✓	✓
Sparteine	✓	✓	✓
Theobromine	✓	✓	✓
Timolol	✓	✓	✓
Torasemide	✓	✗	✗
Valsartan	✓	✗	✗
Warfarin	✓	✗	✗

	Effect	Breakdown	Dose
Fendiline	✓	✓	✓
Fluvastatin	✓	✓	✓
Gallopamil	✓	✓	✓
Hydrochlorothiazide	✓	✓	✓
Irbesartan	✓	✗	✗
Labetalol	✓	✓	✓
Lidocain	✓	✓	✓
Lovastatin	✓	↑	↑
Metolazone	✓	✓	✓
Moexipril	✓	✓	✓
Nicardipine	✓	↑	↑
Nimodipine	✓	↑	↑
Penbutolol	✓	✓	✓
Pindolol	✓	✓	✓
Pravastatin	✓	✓	✓
Propranolol	✓	✓	✓
Ramipril	✓	✓	✓
Simvastatin	✓	↑	✗
Spironolactone	✓	✓	✓
Theophylline	✓	✓	✓
Tirofiban	✓	✓	✓
Trandolapril	✓	✓	✓
Verapamil	✓	↑	↑
Xipamide	✓	✓	✓

Please note: The right choice and dose of medication is always the responsibility of the doctor. Never make your own decision on whether to stop taking a medication or changing its dose!

Legend:

Effect: Normal. Degredation: Normal. Recommendation: Normal dosage.

Effect: Normal. Degradation: Slower. Recommendation: Reduce the dosage.

Effect: Normal. Degradation: None. Recommendation: Alternative drug.

Effect: Lower. Degradation: Normal. Recommendation: Normal dosage.

Effect: Lower. Breakdown: Lower. Recommendation: Reduce the dosage.

Effect: Stronger. Degradation: Stronger. Recommendation: Normal dosage.

PHARMACO GENETICS
Not ordered

ONCOLOGY
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OTHERS
Not ordered

SCIENCE

ADDITIONAL INFORMATION

SCIENCE

This chapter shows the science behind the test.

Cardiovascular Sensor

CDH13 - Cadherin 13 (rs8055236)

The CDH13 gene encodes a protein of the cadherin superfamily. The protein is localized on the cell membrane, and it is expressed, inter alia, in the heart, aortic wall, neurons and in the spinal cord. The polymorphism rs8055236 is associated with an increased risk of heart diseases.

RES	Genotype	POP	Possible results
	T/T	11%	No increased risk of disease
X	T/G	31%	Increased risk of coronary heart disease (OR: 1.91)
	G/G	59%	Increased risk of coronary heart disease (OR: 2.23)

References

The Wellcome Trust Case Control Consortium. Genome-wide association study of 14,000 cases of seven common diseases and 3,000 shared controls.

Linnea M. Baudhuin. Genetics of coronary artery disease: focus on genome-wide association studies. Am J Transl Res. 2009, 1(3): 221–234.

Yan Y et al. Evaluation of population impact of candidate polymorphisms for coronary heart disease in the Framingham Heart Study Offspring Cohort. BMC Proc. 2009 Dec 15,3 Suppl 7:S118.

CHDS8 - Coronary heart disease, susceptibility to, 8 (rs1333049)

The polymorphism rs1333049 on gene CHDS8 (Coronary heart disease, susceptibility to, 8) has been repeatedly associated with an increased risk of heart diseases.

RES	Genotype	POP	Possible results
X	G/G	36%	No increased risk of disease
	G/C	45%	Increased risk of coronary heart disease (OR: 1.47)
	C/C	19%	Increased risk of coronary heart disease (OR: 1.9)

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Helgadottir A. et al. The same sequence variant on 9p21 associates with myocardial infarction, abdominal aortic aneurysm and intracranial aneurysm. Nat Genet. 2008 Feb,40(2):217-24.

Helgadottir A. et al. A common variant on chromosome 9p21 affects the risk of myocardial infarction. Science. 2007 Jun 8,316(5830):1491-3.

Karvanen J. et al. The impact of newly identified loci on coronary heart disease, stroke and total mortality in the MORGAM prospective cohorts. Genet Epidemiol. 2009 Apr,33(3):237-46.

Burton PR. et all. Genome-wide association study of 14,000 cases of seven common diseases and 3,000 shared controls. Nature. 2007 Jun 7,447(7145):661-78.

APOA5 - Apolipoprotein A-V (rs662799)

The protein encoded by this gene is an apolipoprotein and an important determinant of plasma triglyceride levels, a major risk factor for coronary artery disease. It is a component of several lipoprotein fractions including VLDL, HDL and chylomicrons. It is believed that apoA-V affects lipoprotein metabolism by interacting with LDL-R gene family receptors. Studies have shown that carriers of the G-allele experience low weight gain when eating a fatty diet.

RES	Genotype	POP	Possible results
X	A/A	71%	No increased risk of disease
	A/G	26%	Increased risk of coronary heart disease (OR: 1.98)/atherosclerosis/heart attack Predisposition to low HDL cholesterol (the good cholesterol) Predisposition to elevated triglyceride levels
	G/G	3%	Increased risk of coronary heart disease (OR: 1.98)/atherosclerosis/heart attack Predisposition to low HDL cholesterol (the good cholesterol) Predisposition to elevated triglyceride levels

References

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PON1 - Paraoxonase 1 (rs662)

Paraoxonase (PON1) is an antioxidant enzyme involved in radical elimination and lipometabolism. The polymorphisms rs854560 and rs662 lead to a reduced catalytic activity and an increased risk of cardiovascular diseases.

RES	Genotype	POP	Possible results
X	A/A	24%	No increased risk of disease
	G/A	43%	Increased risk of coronary heart disease/atherosclerosis/heart attack (OR: 2.3)
	G/G	33%	Increased risk of coronary heart disease/atherosclerosis/heart attack (OR: 3.2)

References

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PON1 - Paraoxonase 1 (rs854560)

Paraoxonase (PON1) is an antioxidant enzyme involved in radical elimination and lipometabolism. The polymorphisms rs854560 and rs662 lead to a reduced catalytic activity and an increased risk of cardiovascular diseases.

RES	Genotype	POP	Possible results
	A/A	68%	Increased risk of coronary heart disease/atherosclerosis/heart attack (OR: 2.25)
	A/T	27%	No increased risk of disease
X	T/T	5%	No increased risk of disease

References

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Oliveira SA et al. PON1 M/L55 mutation protects high-risk patients against coronary artery disease. Int J Cardiol. 2004 Mar,94(1):73-7.

APOB R3500Q - Apolipoprotein B (rs5742904)

Apolipoprotein B (ApoB) is the major protein component of the LDL proteins (low density lipoprotein), which are responsible for the transport of cholesterol in the blood. As such, ApoB regulates the LDL concentration in the individual. Rs5742904 polymorphism leads to an increased LDL cholesterol levels.

RES	Genotype	POP	Possible results
X	G/G	98%	No increased risk of disease
	A/G	1%	Significantly increased risk of coronary heart disease/atherosclerosis/heart attack Significantly increased risk of elevated LDL cholesterol levels (familial Hypercholesterolaemia)
	A/A	1%	Significantly increased risk of coronary heart disease/atherosclerosis/heart attack Significantly increased risk of elevated LDL cholesterol levels (familial Hypercholesterolaemia)

References

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Castillo et al. The apolipoprotein B R3500Q gene mutation in Spanish subjects with a clinical diagnosis of familial hypercholesterolemia. Atherosclerosis. 2002 Nov,165(1):127-35.

NOS3 - Nitric oxide synthase 3 (endothelial cell) (Ins/Del Int. 4)

NO-synthases (NOS) are oxidases which catalyze the reaction of arginine to citrulline and nitric oxide. NOS3 is an endothelial nitric oxide synthase, predominantly expressed in endothelial cells on the inside of the blood vessels, where it indirectly adjusts the blood pressure and the afterload of the heart. Several polymorphisms in the NOS3 gene are associated with an increased risk of cardiovascular diseases.

RES	Genotype	POP	Possible results
X	Ins/Ins	94%	No increased risk of disease
	Ins/Del	6%	No increased risk of disease
	Del/Del	0%	Increased risk of coronary heart disease/atherosclerosis/heart attack (OR: 1.34)

References

Casas et al. Endothelial nitric oxide synthase genotype and ischemic heart disease: meta-analysis of 26 studies involving 23028 subjects. Circulation. 2004 Mar 23,109(11):1359-65.

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Salimi S et al. Endothelial nitric oxide synthase gene intron4 VNTR polymorphism in patients with coronary artery disease in Iran. Indian J Med Res. 2006 Dec,124(6):683-8.

NOS3 - Nitric oxide synthase 3 (endothelial cell) (rs2070744)

RES	Genotype	POP	Possible results
X	T/T	60%	No increased risk of disease
	C/T	34%	No increased risk of disease
	C/C	7%	Increased risk of coronary heart disease/atherosclerosis/heart attack

References

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Lee CR et al. NOS3 polymorphisms, cigarette smoking, and cardiovascular disease risk: the Atherosclerosis Risk in Communities study. Pharmacogenet Genomics. 2006 Dec,16(12):891-9.

NOS3 - Nitric oxide synthase 3 (endothelial cell) (rs1799983)

RES	Genotype	POP	Possible results
	G/G	69%	No increased risk of disease
X	G/T	26%	Increased risk of coronary heart disease/atherosclerosis/heart attack (OR: 1.52)
	T/T	5%	Increased risk of coronary heart disease/atherosclerosis/heart attack (OR: 2.31)

References

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Colombo MG et al. Evidence for association of a common variant of the endothelial nitric oxide synthase gene (Glu298-->Asp polymorphism) to the presence, extent, and severity of coronary artery disease. Heart. 2002 Jun,87(6):525-8.

APOA1 - Apolipoprotein A-I (rs670)

Apolipoprotein A1 (ApoA1) is the major protein component of HDL (high density lipoprotein) particles in the blood. These are responsible for the transport of excess cholesterol to the liver, where it is further converted and eliminated. The polymorphism rs670 influences both the impact of polyunsaturated fatty acids on HDL cholesterol levels, as well as the risk of heart disease.

RES	Genotype	POP	Possible results
X	G/G	66%	No increased risk of disease Polyunsaturated fatty acids (such as omega-3) WORSEN the HDL cholesterol levels
	A/G	31%	Increased risk of coronary heart disease (OR: 1.47)/atherosclerosis/heart attack Polyunsaturated fatty acids (such as omega-3) improve the HDL cholesterol levels
	A/A	3%	Increased risk of coronary heart disease (OR: 1.9)/atherosclerosis/heart attack Polyunsaturated fatty acids (such as omega-3) improve the HDL cholesterol levels

References

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Juo SH et al. Mild association between the A/G polymorphism in the promoter of the apolipoprotein A-I gene and apolipoprotein A-I levels: a meta-analysis. Am J Med Genet. 1999 Jan 29,82(3):235-41.

MTRR - 5-methyltetrahydrofolate-homocysteine methyltransferase reductase (rs1801394)

Methionine is a essential, sulfur containing proteinogenic amino acid. The synthesis of methionine is catalyzed by the methionine synthase enzyme, which in its turn requires homocysteine. The protein encoded by the MTRR gene (methionine synthase reductase) regenerates the inactive methionine synthase through methylation.

RES	Genotype	POP	Possible results
	A/A	43%	No increased risk of disease
	A/G	41%	Increased risk of coronary heart disease atherosclerosis/heart attack Predisposition to elevated homocysteine values
X	G/G	16%	Increased risk of coronary heart disease atherosclerosis/heart attack Predisposition to elevated homocysteine values

References

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Zeng W et al. A66G and C524T polymorphisms of the methionine synthase reductase gene are associated with congenital heart defects in the Chinese Han population. Genet Mol Res. 2011 Oct 25,10(4):2597-605.

Yu D et al. Association between methionine synthase reductase A66G polymorphism and the risk of congenital heart defects: evidence from eight case-control studies. Pediatr Cardiol. 2014 Oct,35(7):1091-8.

GJA4 - Gap junction protein, alpha 4, 37kDa (rs1764391)

The GJA4 gene (Gap junction alpha-4 protein) belongs to the connexin gene family. These transmembrane proteins are components of the intercellular channels (the so-called gap junctions), which link the adjacent cells with each other, and facilitate the exchange of ions and small molecules. Gap junctions are mainly found in the heart muscle, in epithelial cells and in the retina.

RES	Genotype	POP	Possible results
X	T/T	14%	No increased risk of disease
	C/T	39%	Increased risk of coronary heart disease (OR: 2.03)
	C/C	47%	Increased risk of coronary heart disease (OR: 2.03)

References

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Wen D et al. Association of Connexin37 C1019T with myocardial infarction and coronary artery disease: a meta-analysis. Exp Gerontol. 2014 Oct,58:203-7.

ITGB3 - Integrin beta 3 (platelet glycoprotein IIIa, antigen CD61) (rs5918)

The integrin beta 3 (ITGB3), or CD61, is a transmembrane protein involved in the signal transmission between cells and the extracellular matrix. It has been proven that carriers of the C-allele (rs5918) have an increased risk of cardiovascular diseases. In addition, the polymorphism influences the blood-thinning effect of the aspirin drug.

RES	Genotype	POP	Possible results
X	T/T	84%	No increased risk of disease Aspirin protects against arterial thrombosis
	T/C	15%	Increased risk of coronary heart disease (OR: 2.8) Aspirin does not provide protection from thrombosis
	C/C	1%	Increased risk of coronary heart disease (OR: 7.84) Aspirin does not provide any protection from thrombosis

References

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Goodman T et al. Pharmacogenetics of aspirin resistance: a comprehensive systematic review. Br J Clin Pharmacol. 2008 Aug,66(2):222-32.

CETP - Cholesteryl ester transfer protein, plasma (rs708272)

The cholesterol ester transfer protein (CETP) is a pore-forming protein involved in lipoprotein metabolism. It is mainly expressed in the liver, and ensures the transfer of cholesterol esters from HDL to LDL or VLDL, in exchange for triglycerides. The polymorphism rs708272 influences the regulation of HDL cholesterol levels.

RES	Genotype	POP	Possible results
	T/T	40%	No predisposition to bad HDL cholesterol values (the good cholesterol)
X	C/T	45%	Predisposition to bad HDL cholesterol values (the good cholesterol)
	C/C	15%	Predisposition to bad HDL cholesterol values (the good cholesterol)

References

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Agirbasli et al. Multi-locus candidate gene analyses of lipid levels in a pediatric Turkish cohort: lessons learned on LPL, CETP, LIPC, ABCA1, and SHBG. OMICS. 2013 Dec,17(12):636-45.

Wang et al. CETP gene polymorphisms and risk of coronary atherosclerosis in a Chinese population. Lipids Health Dis. 2013 Nov 27,12:176.

MTHFR - Methylenetetrahydrofolate reductase (NAD(P)H) (rs1801133)

The methylenetetrahydrofolate reductase (MTHFR) is involved in many metabolic pathways in the human body. In homocysteine metabolism, it is responsible for the degradation of homocysteine to methionine. The rs1801133 polymorphism leads to a reduced enzymatic activity of methylenetetrahydrofolate reductase, and thus to an increased homocysteine level.

RES	Genotype	POP	Possible results
	T/T	8%	Predisposition to elevated homocysteine values Vitamin B2 lowers the homocysteine levels
	C/T	33%	Predisposition to elevated homocysteine values Vitamin B2 does not lower the homocysteine levels
X	C/C	59%	No predisposition to elevated homocysteine values Vitamin B2 does not lower the homocysteine levels

References

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Ventura P et al. Hyperhomocysteinemia and MTHFR C677T polymorphism in patients with portal vein thrombosis complicating liver cirrhosis. Thromb Res. 2016 May,141:189-95.

Hustad et al. Riboflavin and Methylenetetrahydrofolate Reductase. Madame Curie Bioscience Database.

MMP3 - Matrix metallopeptidase 3 (stromelysin 1, progelatinase) (rs3025058)

The matrix metalloproteinase-3 (MMP3) or Stromelysin 1, is a zinc-dependent endopeptidase involved in the degradation of extracellular matrix components. It plays an important role in the remodeling of tissues, wound healing and inflammatory processes. The polymorphism (rs3025058) influences the risk of heart diseases.

RES	Genotype	POP	Possible results
	T/T	26%	No increased risk of disease
X	T/Del	49%	Increased risk of coronary heart disease (OR: 1.26)
	Del/Del	25%	Increased risk of coronary heart disease (OR: 1.59)

References

Abilleira et al. The role of genetic variants of matrix metalloproteinases in coronary and carotid atherosclerosis. J Med Genet. 2006 Dec,43(12):897-901. Epub 2006 Aug 11.

Zee et al. Genetic risk factors in recurrent venous thromboembolism: A multilocus, population-based, prospective approach. Clin Chim Acta. 2009 Apr,402(1-2):189-92.

Wang J et al. Polymorphisms of matrix metalloproteinases in myocardial infarction: a meta-analysis. Heart. 2011 Oct,97(19):1542-6. doi: 10.1136/heartjnl-2011-300342.

NOS1AP - Nitric oxide synthase 1 (neuronal) adaptor protein (rs16847548)

The nitric oxide synthase 1 adaptor protein (NOS1AP) is an adapter protein which binds the signal molecule nNOS (neuronal nitric oxide synthase) with other molecules, facilitating their interaction. NOS1AP polymorphisms are associated with a prolonged QT interval, and an increased risk of sudden cardiac death.

RES	Genotype	POP	Possible results
X	T/T	53%	No increased risk of sudden cardiac death
	T/C	38%	Increased risk of sudden cardiac death (OR: 1.3)
	C/C	9%	Increased risk of sudden cardiac death (OR: 2.6)

References

Arking et al. Multiple independent genetic factors at NOS1AP modulate the QT interval in a multi-ethnic population. PLoS One. 2009,4(1):e4333.

Crotti et al.NOS1AP is a genetic modifier of the long-QT syndrome. Circulation. 2009 Oct 27,120(17):1657-63.

Kao et al. Genetic variations in nitric oxide synthase 1 adaptor protein are associated with sudden cardiac death in US white community-based populations. Circulation. 2009 Feb 24,119(7):940-51.

NOS1AP - Nitric oxide synthase 1 (neuronal) adaptor protein (rs12567209)

The nitric oxide synthase 1 adaptor protein (NOS1AP) is an adapter protein which binds the signal molecule nNOS (neuronal nitric oxide synthase) with other molecules, facilitating their interaction. NOS1AP polymorphisms are associated with a prolonged QT interval, and an increased risk of sudden cardiac death.

RES	Genotype	POP	Possible results
X	G/G	74%	No increased risk of sudden cardiac death
	A/G	23%	Protection against sudden cardiac death (OR: 0.51)
	A/A	3%	Increased risk of sudden cardiac death (OR: 1.31)

References

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NOS1AP - Nitric oxide synthase 1 (neuronal) adaptor protein (rs10494366)

The nitric oxide synthase 1 adaptor protein (NOS1AP) is an adapter protein which binds the signal molecule nNOS (neuronal nitric oxide synthase) with other molecules, facilitating their interaction. NOS1AP polymorphisms are associated with a prolonged QT interval, and an increased risk of sudden cardiac death.

RES	Genotype	POP	Possible results
	G/G	37%	Predisposition to increased QT-interval duration (+ 4 to 7,9 ms)
	G/T	43%	Predisposition to increased QT-interval duration (+ 1,7 to 4,6 ms)
X	T/T	20%	No predisposition to increased QT-interval duration

References

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Marjamaa et al. Common candidate gene variants are associated with QT interval duration in the general population. J Intern Med. 2009 Apr,265(4):448-58.

SREBF2 - Sterol regulatory element binding transcription factor 2 (rs2228314)

SREBF2 or SREBP2 (sterol regulatory element-binding protein 2) is a transcription factor involved in the regulation of cholesterol metabolism. The cholesterol concentration is kept in balance through the control of the transcriptional activity of various target genes.

RES	Genotype	POP	Possible results
	G/G	39%	Predisposition to elevated LDL cholesterol levels
	G/C	40%	No predisposition to elevated LDL cholesterol levels
X	C/C	20%	No predisposition to elevated LDL cholesterol levels

References

Fan et al. Expression of sterol regulatory element-binding transcription factor (SREBF) 2 and SREBF cleavage-activating protein (SCAP) in human atheroma and the association of their allelic variants with sudden cardiac death. Published online Dec 30, 2008.

Wang Y et al. Relationship of SREBP-2 rs2228314 G>C polymorphism with nonalcoholic fatty liver disease in a Han Chinese population. Genet Test Mol Biomarkers. 2014 Sep,18(9):653-7

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CYP1A2 - cytochrome P450, family 1, subfamily A, polypeptide 2 (rs762551)

The haeme protein cytochrome P450-1A2 (CYP1A2) belongs to the group of cytochrome P450 enzymes, and metabolizes various xenobiotic substances (including caffeine), medications and oestrogens. The polymorphism rs762551 is associated with the risk of breast cancer.

RES	Genotype	POP	Possible results
X	A/A	41%	Caffeine is broken down normally
	A/C	44%	Caffeine is broken down slowly
	C/C	15%	Caffeine is broken down slowly

References

Bågeman et al. Coffee consumption and CYP1A2*1F genotype modify age at breast cancer diagnosis and estrogen receptor status. Cancer Epidemiol Biomarkers Prev. 2008 Apr,17(4):895-901.

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APOE - apolipoprotein E (E2/E3/E4)

APoE (apolipoprotein E) metabolizes triglyceride-rich lipoprotein constituents, and plays a central role in the lipid metabolism. The ApoE gene is present in three common types, which are called allele E2, E3 and E4. The E4 allele is associated with an increased risk of heart disease and Alzheimer.

RES	Genotype	POP	Possible results
	E2/E2	1%	No increased risk of coronary heart disease/atherosclerosis/heart attack No predisposition to elevated LDL cholesterol levels Predisposition to elevated triglyceride levels
X	E2/E3	6%	No increased risk of coronary heart disease/atherosclerosis/heart attack No predisposition to elevated LDL cholesterol levels Predisposition to elevated triglyceride levels
	E3/E3	66%	No increased risk of coronary heart disease No predisposition to elevated LDL cholesterol levels No predisposition to elevated triglyceride levels
	E2/E4	2%	No increased risk of coronary heart disease No predisposition to elevated LDL cholesterol levels No predisposition to elevated triglyceride levels
	E3/E4	24%	Increased risk of coronary heart disease/atherosclerosis/heart attack Predisposition to elevated LDL cholesterol levels Predisposition to elevated triglyceride levels
	E4/E4	1%	Increased risk of coronary heart disease/atherosclerosis/heart attack Predisposition to elevated LDL cholesterol levels Predisposition to elevated triglyceride levels

References

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Dallongeville et al. Modulation of plasma triglyceride levels by apoE phenotype: a meta-analysis. J Lipid Res. 1992 Apr,33(4):447-54.

Breslow et al. Genetic Basis of Lipoprotein Disorders. Circulation. 1995 Jan 15,91(2):505-12.

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LEGEND: RES = your personal analysis result (marked with an X), GENOTYPE = different variations of the gene (called alleles),
POP = percent of the general population that have this genetic result,
POSSIBLE RESULTS = influence of the genetic variation.

PHARMACO GENETICS
Not ordered

ONCOLOGY
Not ordered

CARDIOVASCULAR SYSTEM

NEUROLOGY
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METABOLISM
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MOVEMENT
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DIGESTION
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SCIENCE

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ADDITIONAL INFORMATION

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- office.ch@progenom.com

Our team is looking forward to your call. Customer satisfaction is our first priority. If you are not fully satisfied with our service, please let us know. We will do our best to help find a satisfactory solution to your problem.

Contact | Impressum
ProGenom GmbH
Riedstrasse 1
6343 Rotkreuz
SWITZERLAND

Technical details

Order number
DEMO_DS

Date of birth
01/01/1990

Established analysis methods
qRT-PCR, DNA sequencing, fragment length analysis, CNV assay, GC-MS, Immunocap ISAC, Cytolisa

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Product codes
M1CAR

Current version
V538

Ordering company
ProGenom GmbH
Riedstrasse 1
6343 Rotkreuz
SWITZERLAND

Analyzing company
DNA Plus - Zentrum für Humangenetik
Georg Wrede Strasse 13
83395 Freilassing
Deutschland

Laboratory Director

Dr. Daniel Wallerstorfer Bsc.

Laboratory Manager

Florian Schneebauer, MSc.

NOTES:


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ProMedicDNA

Cardiovascular Sensor

Jane Doe
DEMO_DS

0000000000000

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