ProMedicDNA

# Thrombo 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

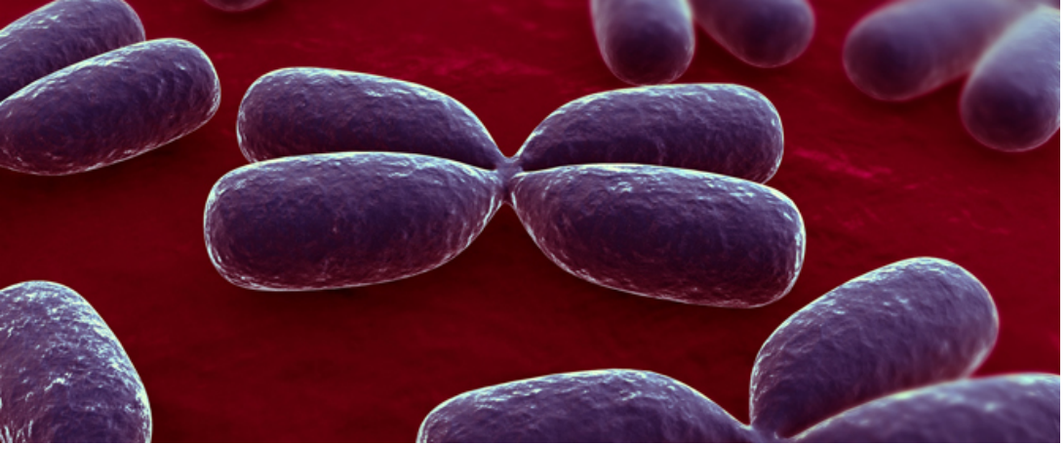
Florian Schneebauer, MSc.
Laboratory Manager

# Thrombo Sensor

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

Order number:
**DEMO_DS**

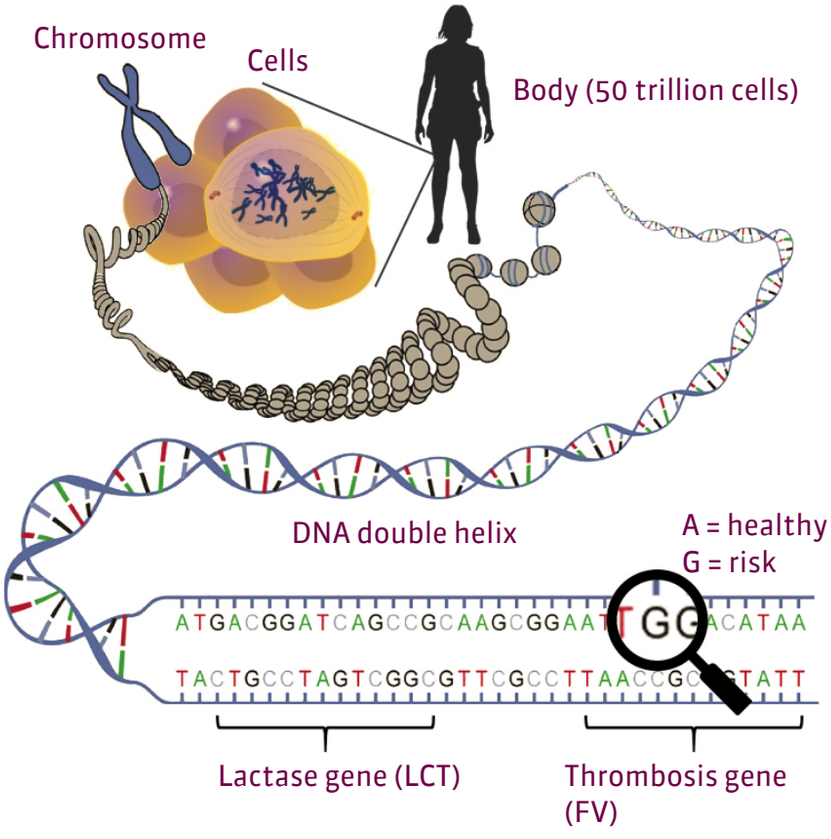
**This report contains personal medical information that is highly confidential. Data protection must be ensured.**





# 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.

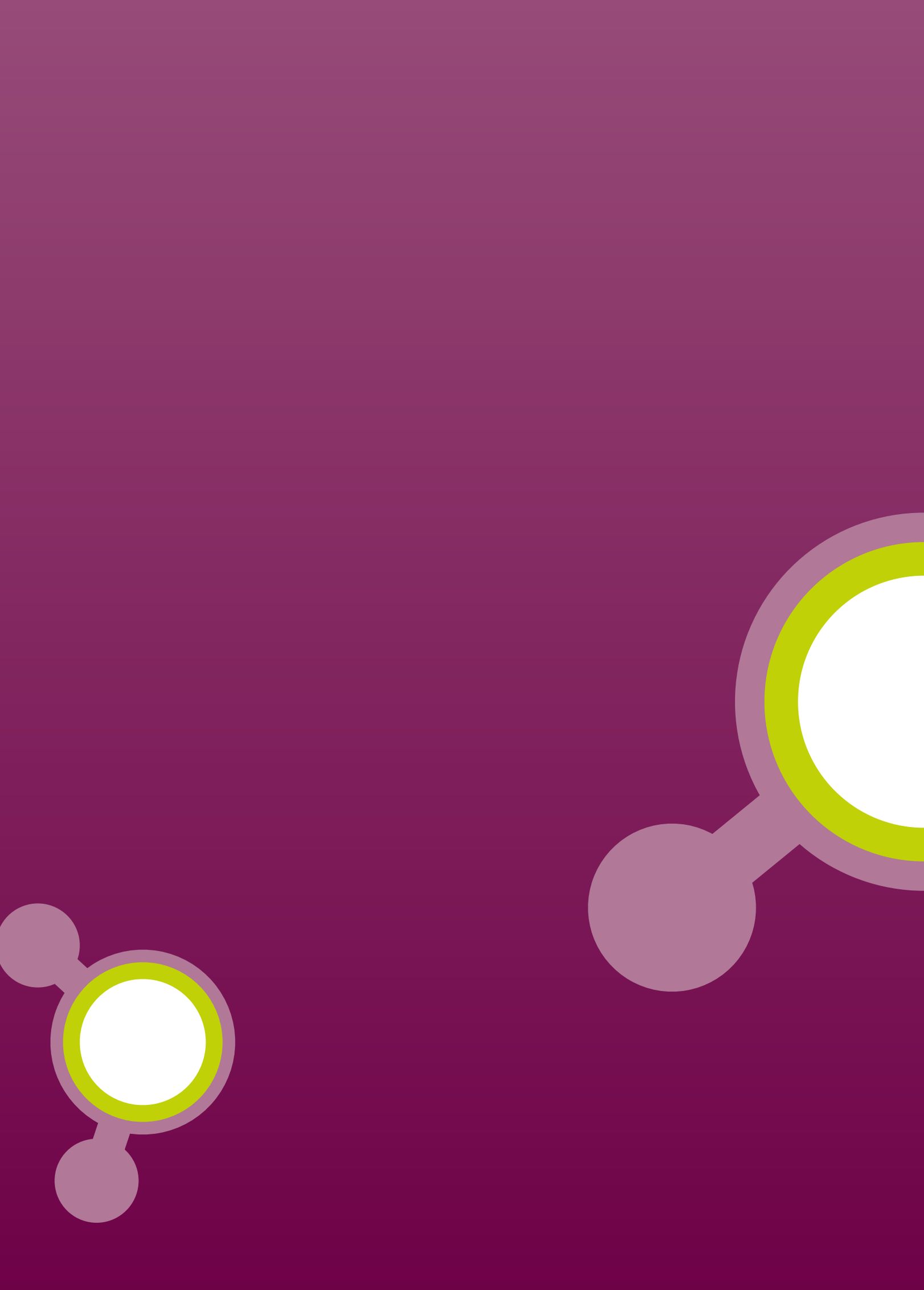
External influences can affect individual genes and disrupt their function, but the majority of our defective genes are inherited from our parents. Each embryo receives half of its genes from the father and half from the mother, resulting in a new human being with some characteristics of each parent. 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.

**Thrombo Sensor**



## PHARMACO GENETICS
*Not ordered*

## ONCOLOGY
*Not ordered*

## CARDIOVASCULAR SYSTEM

## NEUROLOGY
*Not ordered*

## METABOLISM
*Not ordered*

## MOVEMENT
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## DIGESTION
*Not ordered*

## OPHTHALMOLOGY
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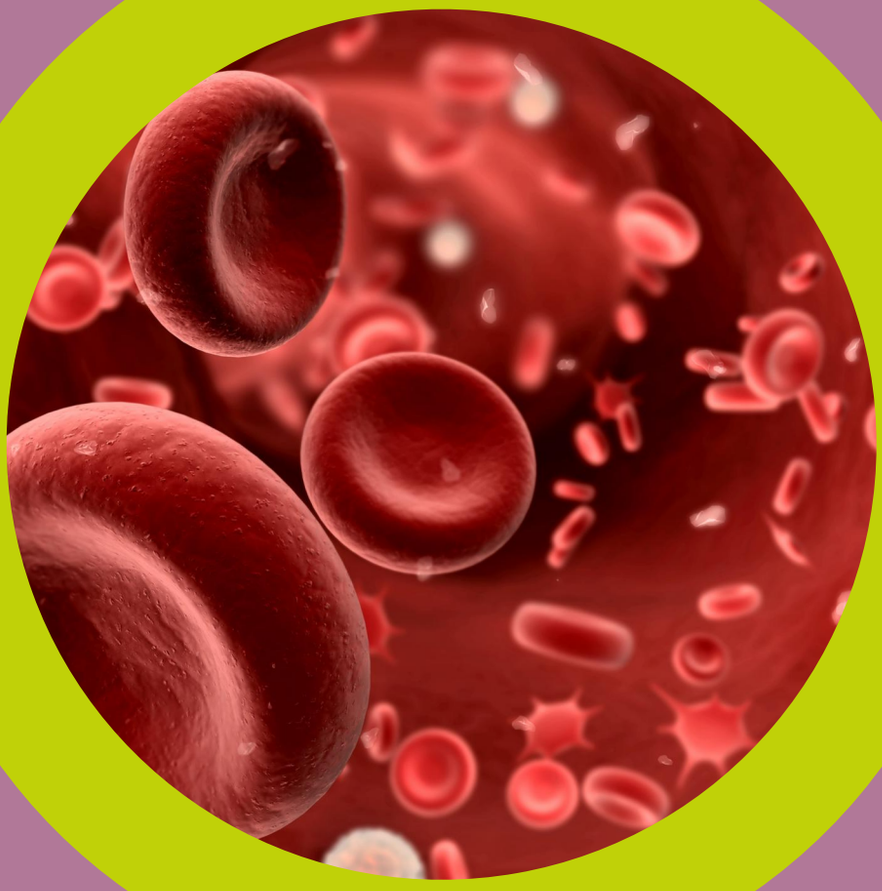
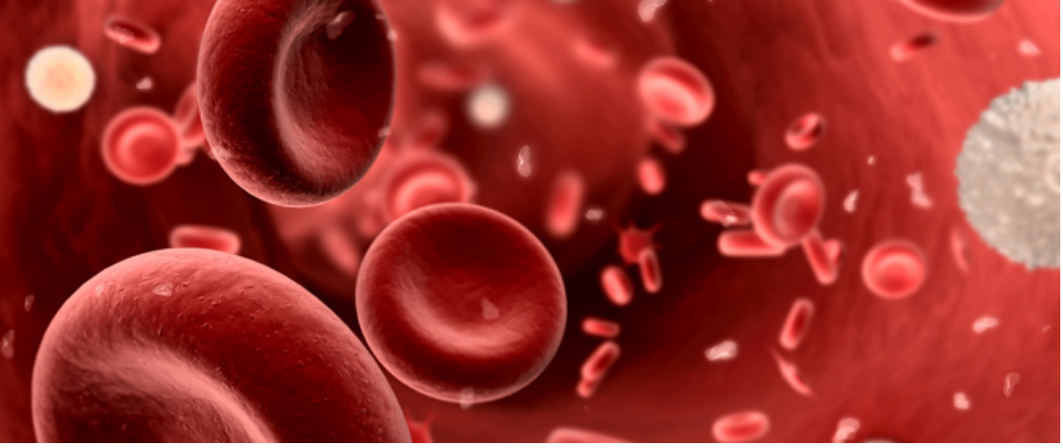
## SCIENCE

## ADDITIONAL INFORMATION



# Thrombo Sensor

**Effectively prevent thrombosis**





# Thrombosis

**Thrombosis is a disease where blood clots form in the bloodstream. These clots can obstruct certain blood vessels and reduce blood flow to important organs, like the heart or to areas in the brain. This may lead to damage or even the death of the affected tissue. When blood supply to part of the brain is impaired, a stroke may occur. If blood supply to the heart is affected, it can cause a heart attack. The most common form of thrombosis is decreased blood circulation in the legs caused by a blood clot. The risk in this case is if the clots dissolve they can move along the bloodstream and restrict the blood flow to other important organs ,like the brain, heart or the lungs.**

Genetic screening tests for thrombosis are unfortunately rarely performed. Since there are no obvious symptoms until after the event, most people do not know that they are genetically predisposed to thrombosis.

Therefore, a genetic predisposition is usually not detected until after the occurrence of thrombosis; this, however, may have fatal consequences. Genetic screening tests are still performed too infrequently even though they increase awareness of the risk so that the necessary preemptive measures can be taken. In some cases, it may even allow the individual to avoid the disease altogether. Several genes prevent the formation of blood clots in the veins. If one of these genes is defective, it cannot perform its task and the risk of forming a blood clot increases significantly. Everyone has two genes of each type but about 1 in 20 people carries a defect in at least one gene, thus being a carrier with an approximate 8- fold higher risk of thrombosis than the general population. About 1 in 200 people carries an error in both genes of a genotype and has a 80 -fold higher thrombosis risk. Having defective genes does not necessarily mean the patient will suffer from thrombosis because only a fraction of those affected will develop the disease. Other factors also contribute strongly, such as excessive weight, bed rest and inactivity, prolonged air travel, contraceptive pills, pregnancy, etc.

This is why genetic testing is so important; if you know your genetic health risk, you can take precautionary measures and, in most cases, even prevent the occurrence of thrombosis.

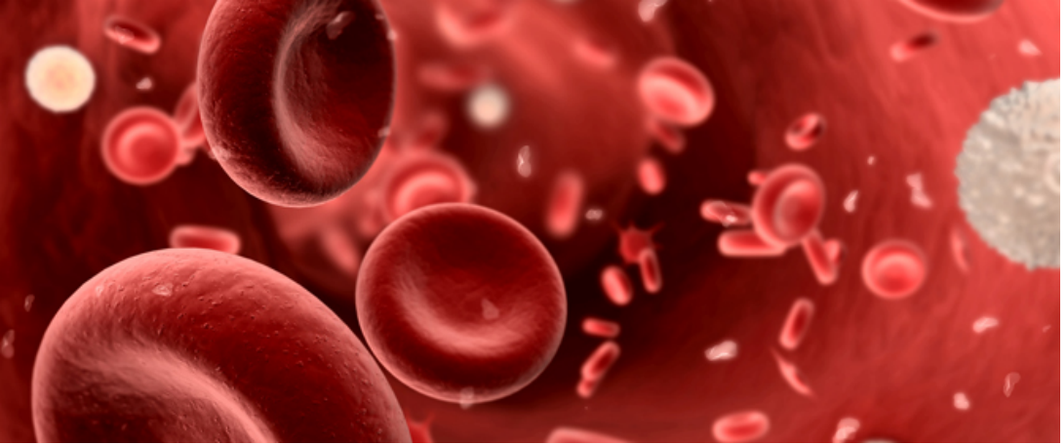
# Risk of thrombosis during pregnancy

**Studies have shown that the risk of thrombosis during pregnancy is between 4 to 10 times higher than the risk in non-pregnant women. This risk increases further in the months after delivery by between 10- 20 times. What is striking is that most cases occur in young mothers (15-19 years). Approximately 1 in 20 women is genetically predisposed to thrombosis even without pregnancy, and has an approximately 8-80-fold higher risk of thrombosis than the general population. If a genetically predisposed woman is pregnant, the combination of these two risk factors leads to a dangerous combination of genetic defect and risk situation, increasing the risk of thrombosis by 60-fold and leading to life-threatening conditions.**

It is estimated that thrombosis is responsible for about one third of deaths in pregnancy, and between 30-60% of women who develop thrombosis are genetically predisposed. Therefore, it is already widely medically accepted that genetically predisposed women should take medication to prevent thrombosis during pregnancy.
Unfortunately, very few women know of their genetic risk of thrombosis and so a significant percentage of these women remain untreated and unprotected. The treatment consists of daily administration of low molecular weight heparin, a drug that prevents the formation of blood clots in the veins. Medication will be carefully chosen so that it does not enter the fetal bloodstream. Heparin can be given to a pregnant woman without harming her fetus, under medical supervision. When used consistently, these drugs ensure that thrombosis is prevented both before and after childbirth.

## Risk of thrombosis and the use of hormonal compounds:

Hormone-containing medications are used by many women, either in the form of contraceptives or as treatment for complications caused by reduced hormone production after menopause. Although hormones have numerous benefits, they increase the risk of thrombosis. This risk is very small, unless there are additional risk factors, such as a genetic predisposition. A genetic predisposition for thrombosis significantly increases the risk; in combination with hormonal preparations the risk increases approximately 15 times, and in some cases even to more than 80 times. For women with a genetic predisposition to thrombosis the use of hormone preparations is therefore strongly discouraged, since it can lead to life-threatening conditions.





# Relevant genes for thrombosis

Three genetic variations have been identified that can significantly increase the risk of thrombosis. An analysis of these three polymorphisms will determine your risk of developing a thrombosis which can then be reduced with specific preventive measures. The following genes have an impact on your risk of thrombosis:

**Genetic traits**

| SYMBOL | rs NCBI | POLYMORPH | GENOTYPE |
|---|---|---|---|
| Factor-V | rs6025 | G>A | G/G |
| Factor-II | rs1799963 | G>A | G/G |
| PAI1 | rs1799889 | G>del | del/del |
| MTHFR | rs1801133 | C>T | C/C |
| ITGB3 | rs5918 | T>C | T/T |

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

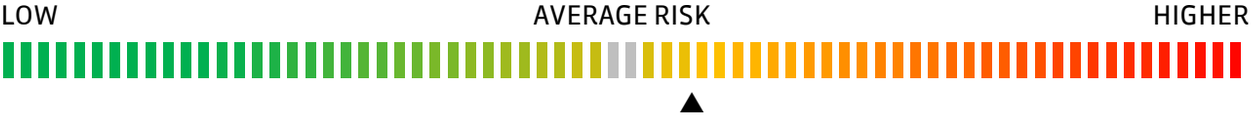
# Summary of effects

Certain genes are responsible for preventing blood clot formation in the blood vessels. Variations in these genes may interfere with this process, and therefore increase your risk of blood clot formation and subsequently, thrombosis. Below is a summary of the impact that genetic variations may have on your health:

- Your risk of developing venous thrombosis is not increased
- Therefore, hormonal preparations do not increase your risk of thrombosis.
- Your risk of developing arterial thrombosis is approximately 1.84 -times increased
- Aspirin can be effective in preventing arterial thrombosis

Risk of venous thrombosis

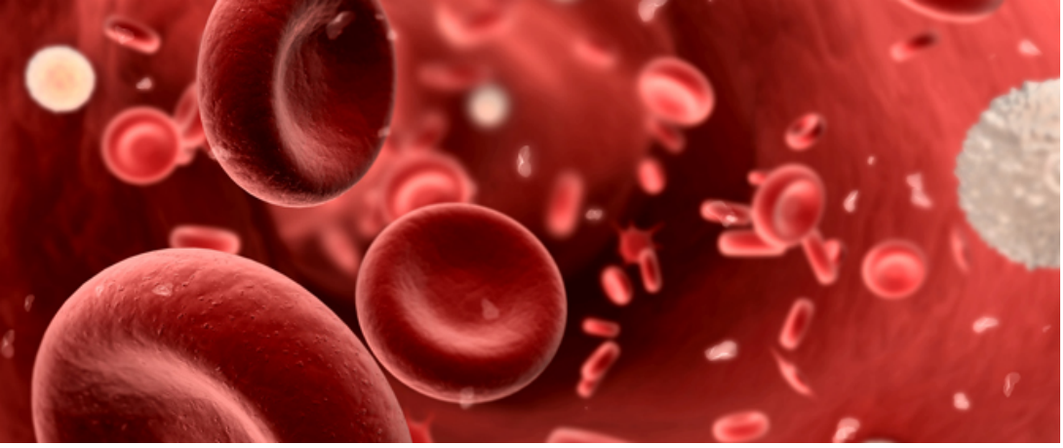

Risk of arterial thrombosis




Risk of gestational thrombosis


Is your risk significantly increased by hormone preparations?


Effect of aspirin on arterial thrombosis (blood clots)





# Prevention

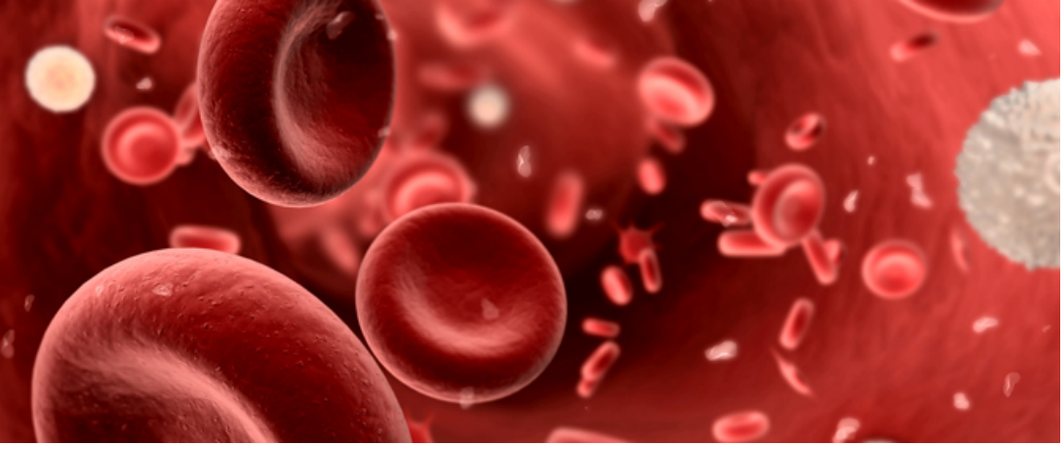
**Based on your genetic profile, you have an increased risk of thrombosis. Therefore, it is highly recommended that you take some precautionary measures to reduce your risk. A genetic predisposition to thrombosis only increases your risk of developing a clot but it does not mean that you will definitely experience thrombosis. Complications occur only when a blood clot forms in the blood vessels, which then impairs blood supply to certain parts of the body. The preemptive measures focus on preventing this. You have a significantly higher risk of developing thrombosis and other factors may increase the risk even further.**

## Special high-risk situations in which precautionary measures should be considered:

- Bed rest and inactivity, eg. when wearing a plaster cast. In this case, heparin injections are recommended after surgery (especially after surgery in the abdomen, hip or knee surgery).
- Symptoms can frequently occur during pregnancy. However, this increased risk of thrombosis should not be an obstacle to pregnancy, but it will require closer medical supervision and possibly blood-thinning drugs, such as heparin. Heparin does not enter breast milk, so women can take it even while nursing.
- Cancers or diseases that are associated with the loss of fluid (for example, diarrhoea). Varicose veins in the legs. Heart diseases such as heart failure or after a heart attack.

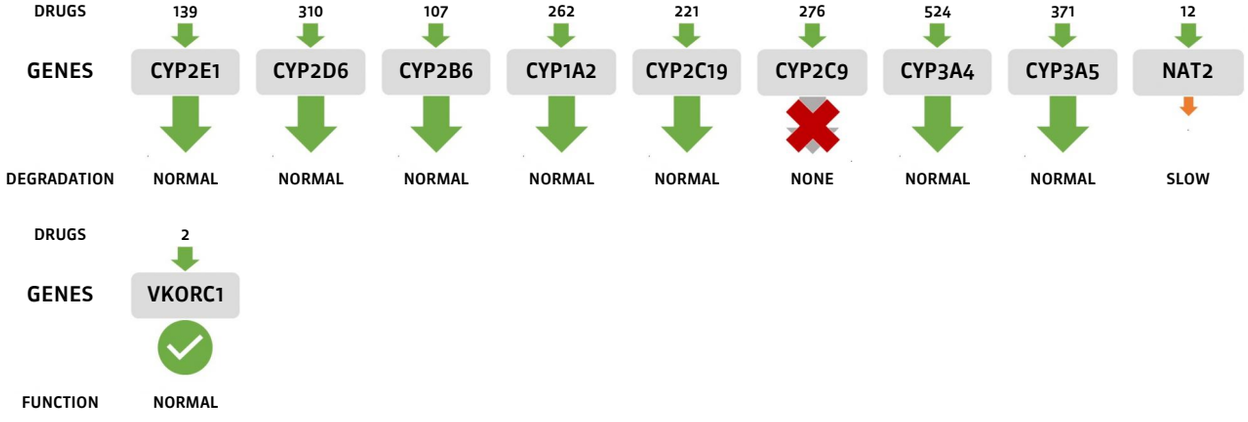
## Certain medications can cause additional complications. Discuss the medication you are taking and your genetic risks with your doctor. The following medications may be unsuitable for those affected:

- The "skin pill"- hormonal therapy with ethinylestradiol and cyproterone
- Drugs for the treatment of breast cancer– tamoxifen
- Vaccine for the HPV virus
- Certain blood pressure medications
- Contraceptive pill - ethinylestradiol and drospirenone
- Contraceptive hormone ring - etonogestrel and ethinylestradiol
- Sedative - Thalidomide drug for the treatment of anemia
- Doping drugs – Erythropoietin
- Cortisone
- Menopausal preparations





# Drug compatibility




## Effect on relevant medication

| | Effect | Breakdown | Dose |
|---|---|---|---|
| Acenocoumarol | ✓ | ↓ | ✓ |
| Clopidogrel | ✓ | ↑ | ✓ |
| Fondaparinux | ✓ | ✓ | ✓ |
| Ticagrelor | ✓ | ✓ | ✓ |
| Warfarin | ✓ | ✗ | ✗ |

| | Effect | Breakdown | Dose |
|---|---|---|---|
| Acetylsalicylic Acid | ✓ | ✗ | ✗ |
| Desirudin | ✓ | ✓ | ✓ |
| Prasugrel | ✓ | ✓ | ✓ |
| Ticlopidine | ✓ | ↑ | ↑ |

| | Effect | Breakdown | Dose |
|---|---|---|---|
| Bemiparin | ✓ | ✓ | ✓ |
| Enoxaparin | ✓ | ✓ | ✓ |
| Reteplase | ✓ | ✓ | ✓ |
| Urokinase | ✓ | ✓ | ✓ |

*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*

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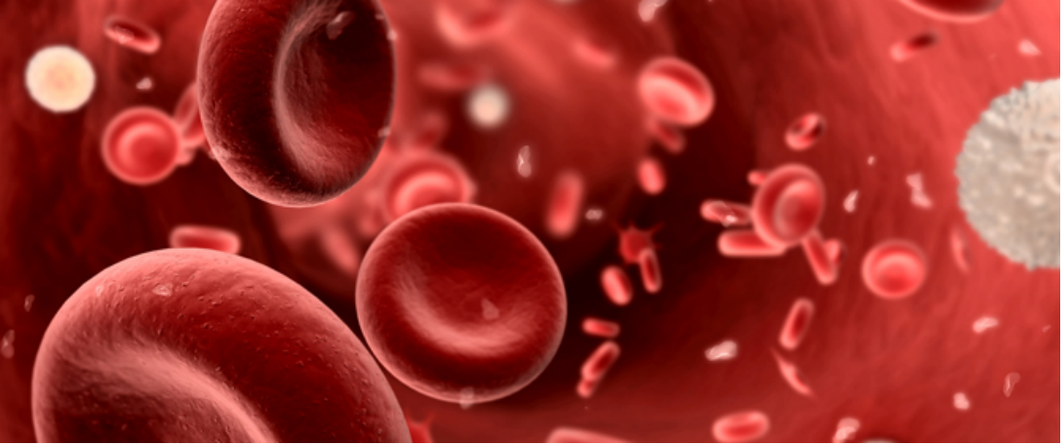
## SCIENCE

## ADDITIONAL INFORMATION

# SCIENCE

**This chapter shows the science behind the test.**





# Thrombo Sensor

## Factor-V - Coagulation factor V (proaccelerin, labile factor) (rs6025)

The so-called factor V Leiden mutation is a genetically transmitted clotting defect, associated with an increased risk of thrombosis. This defect inhibits the degradation of factor V and the protein retains its coagulant effect.

| RES | Genotype | POP | Possible results |
|---|---|---|---|
| | A/A | 1% | Increased risk of thrombosis (venous) (OR: 80!) |
| | A/G | 1% | Increased risk of thrombosis (venous) (OR: 7) |
| X | G/G | 98% | No increased risk of thrombosis (venous) |

### References


Juul et al. Factor V Leiden and the risk for venous thromboembolism in the adult Danish population. Ann Intern Med. 2004 Mar 2,140(5):330-7.

Brenner et al. Venous Thromboembolism Associated With Double Heterozygosity for R506Q Mutation of Factor V and for T298M Mutation of Protein C in a Large Family of a Previously Described Homozygous Protein C -Deficient Newborn With Massive Thrombosis: Blood. 1996 Aug 1,88(3):877-80.

Zee et al. An Evaluation of Candidate Genes of Inflammation and Thrombosis in Relation to the Risk of Venous Thromboembolism: Circulation. Feb 2009, 2(1): 57–62.

Rosendaal et al. High risk of thrombosis in patients homozygous for factor V Leiden (activated protein C resistance). Br J Haematol. 2002 Mar,116(4):851-4.

Kamphuisen et al. Thrombophilia screening: a matter of debate. Neth J Med. 2004,62:180-187.

Ridker et al. Ethnic distribution of factor V Leiden in 4047 men and women. Implications for venous thromboembolism screening, Jama 277 (1997) 1305-1307.


## Factor-II - Coagulation factor II (thrombin) (rs1799963)

The prothrombin mutation (factor II mutation) is a blood coagulant disorder. The risk of venous thrombosis is significantly increased by the polymorphism rs1799963, which allows the creation of too much clotting factor, prothrombin, in the blood.

| RES | Genotype | POP | Possible results |
|---|---|---|---|
| | A/A | 1% | Increased risk of thrombosis (venous) (OR: 25) |
| | A/G | 1% | Increased risk of thrombosis (venous) (OR: 5) |
| X | G/G | 98% | No increased risk of thrombosis (venous) |

### References


Zee et al. An Evaluation of Candidate Genes of Inflammation and Thrombosis in Relation to the Risk of Venous Thromboembolism: The Women's Genome Health Study. Circ Cardiovasc Genet. Feb 2009, 2(1): 57–62.

Rosendaal et al. Hormonal replacement therapy, prothrombotic mutations and the risk of venous thrombosis. Br J Haematol. 2002 Mar,116(4):851-4.

Ye et al. Seven haemostatic gene polymorphisms in coronary disease: meta-analysis of 66,155 cases and 91,307 controls. Lancet. 2006 Feb 25,367(9511):651-8.

## PAI1 - Phosphoribosylanthranilate isomerase (rs1799889)

Plasminogen activator inhibitor-1 (PAI-1) is a glycoprotein belonging to the group of serine protease inhibitors. It inhibits the fibrinolytic activity, by inactivating tPA and urokinase. A defect in the PAI-1 gene leads to increased transcription and a higher concentration PAI-1. This condition is associated with an increased risk of thrombosis.

| RES | Genotype | POP | Possible results |
|---|---|---|---|
| X | Del/Del | 24% | Increased risk of thrombosis (arterial) (OR: 1.84) |
| | Del/G | 48% | Increased risk of thrombosis (arterial) (OR: 1.83) |
| | G/G | 28% | No increased risk of thrombosis (arterial) |

### References


Tsantes et al. Association between the plasminogen activator inhibitor-1 4G/5G polymorphism and venous thrombosis. A meta-analysis. Thromb Haemost.

Fernandes et al. 4G/5G polymorphism modulates PAI-1 circulating levels in obese women. Mol Cell Biochem. 2012 May,364(1-2):299-301.

Gardemann et al. The 4G4G genotype of the plasminogen activator inhibitor 4G/5G gene polymorphism is associated with coronary atherosclerosis in patients at high risk for this disease. Thromb Haemost. 1999 Sep,82(3):1121-6.

Rosendaal et al. Hormonal replacement therapy, prothrombotic mutations and the risk of venous thrombosis. Br J Haematol. 2002 Mar,116(4):851-4.

Ye et al. Seven haemostatic gene polymorphisms in coronary disease: meta-analysis of 66,155 cases and 91,307 controls. Lancet. 2006 Feb 25,367(9511):651-8.


## 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 |
|---|---|---|---|
| X | C/C | 59% | No increased risk of thrombosis (venous) |
| | C/T | 33% | No increased risk of thrombosis (venous) |
| | T/T | 8% | Increased risk of thrombosis (venous) (OR: 3) |

### References


M.G. Andreassi et al. Factor V Leiden, prothrombin G20210A substitution and hormone therapy: indications for molecular screening, Clin Chem Lab Med 44 (2006) 514-521.

I. Fermo et al. Prevalence of moderate hyperhomocysteinemia in patients with early-onset venous and arterial occlusive disease, Annals of internal medicine 123 (1995) 747-753.

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.

## 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% | Aspirin protects against arterial thrombosis |
| | T/C | 15% | Aspirin does not provide any protection from thrombosis |
| | C/C | 1% | Aspirin does not provide any protection from thrombosis |

**References**

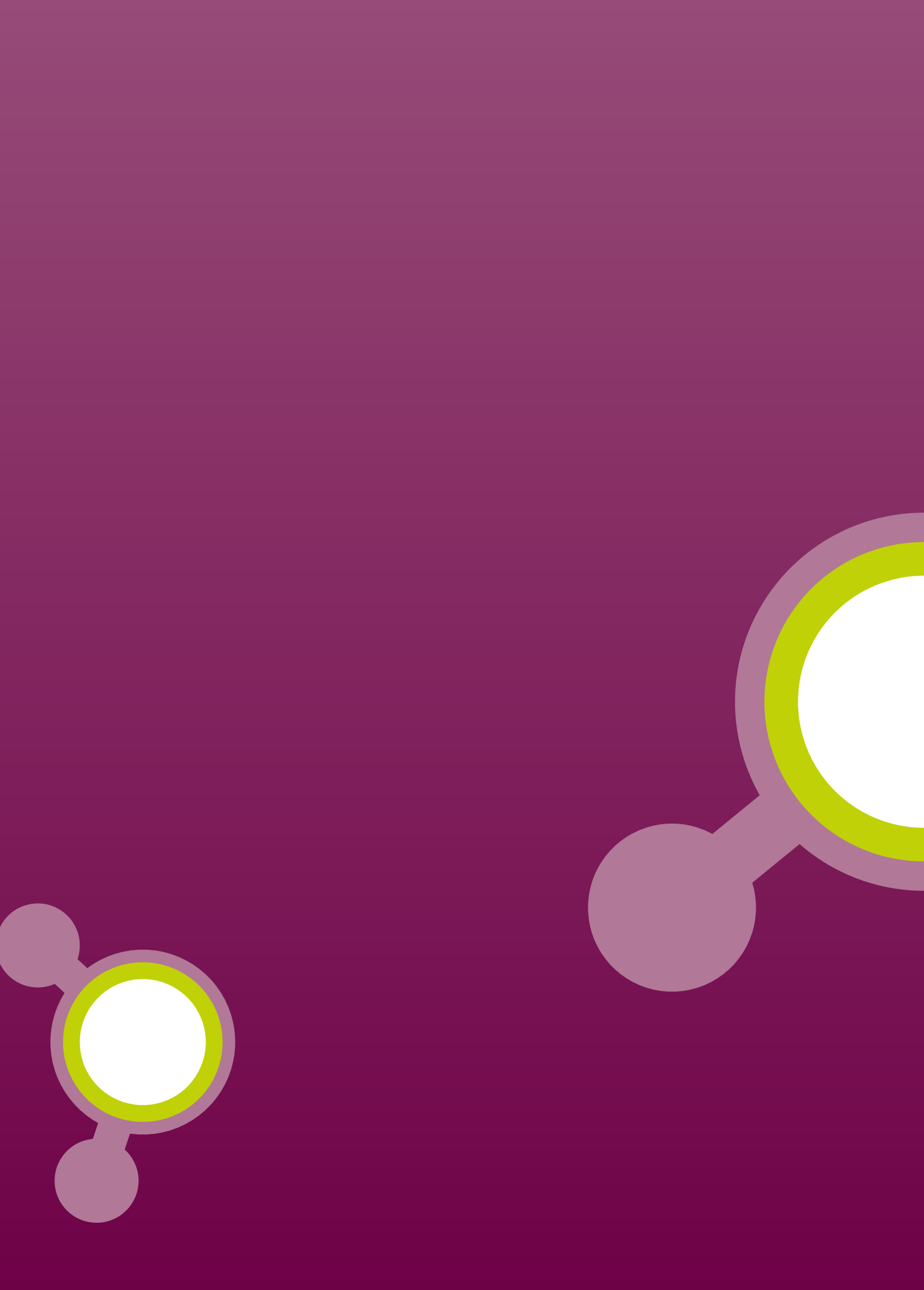

Undas et al. Pl(A2) polymorphism of beta(3) integrins is associated with enhanced thrombin generation and impaired antithrombotic action of aspirin at the site of microvascular injury. Circulation. 2001 Nov 27,104(22):2666-72.

Weiss et al. A polymorphism of a platelet glycoprotein receptor as an inherited risk factor for coronary thrombosis. N Engl J Med. 1996

Erdman V et al. OS 08-03 PHARMACOGENETIC MARKERS OF SURVIVAL. J Hypertens. 2016 Sep,34 Suppl 1 - ISH 2016 Abstract Book:e68.

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


*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.*



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*Not ordered*

## SCIENCE

## ADDITIONAL INFORMATION

# ADDITIONAL INFORMATION

In this chapter you will receive useful information




# Certifications

Our laboratory is one of the most modern and automated laboratories in Europe and has numerous certifications and quality assurance systems that meet, and even exceed, international standards. The various areas of business are certified separately to the highest standards.

## Laboratory diagnostics, manufacturing & sales

Quality management system in accordance with ISO 9001:2015

qualityaustria
SYSTEM CERTIFIED
ISO 9001:2015 No.14365/0

## Licensed for medical genetics

Approved by the Federal Ministry of Health, Austria

BUNDESMINISTERIUM FÜR GESUNDHEIT

## Cosmetic/genetic diagnostics and cosmetics manufacturing

Good manufacturing practice (GMP) in accordance with ISO 22716:2007

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SYSTEM CERTIFIED
EN ISO 22716:2007 No.00010/0

## Food supplement manufacturing

Management system for food safety in accordance with ISO 22000:2018

qualityaustria
SYSTEM CERTIFIED
ISO 22000:2018 No.00521/0



# Customer Service

## Questions or comments about our service?

Our customer service team is happy to help with any enquiries or problems. You can contact us in the following ways:

- Phone +41 (0) 41 525 100.1
- 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

**Report generated**
19/03/2021 17:44:09

**Product codes**
M1THR

**Current version**
V538

**Ordering company**
ProGenom GmbH
Riedstrasse 1
6343 Rotkreuz
SWITZERLAND

**Analyzing company**
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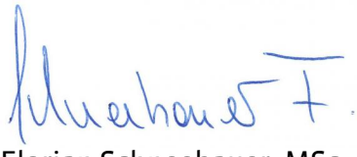

Florian Schneebauer, MSc.

**NOTES:**


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ProMedicDNA

# Thrombo Sensor

Jane Doe
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