ProMedicDNA

Prostate Health Sensor

John Doe
DEMO_DS

ProGenom
www.progenom.com

COVER LETTER

Dear Mr. 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

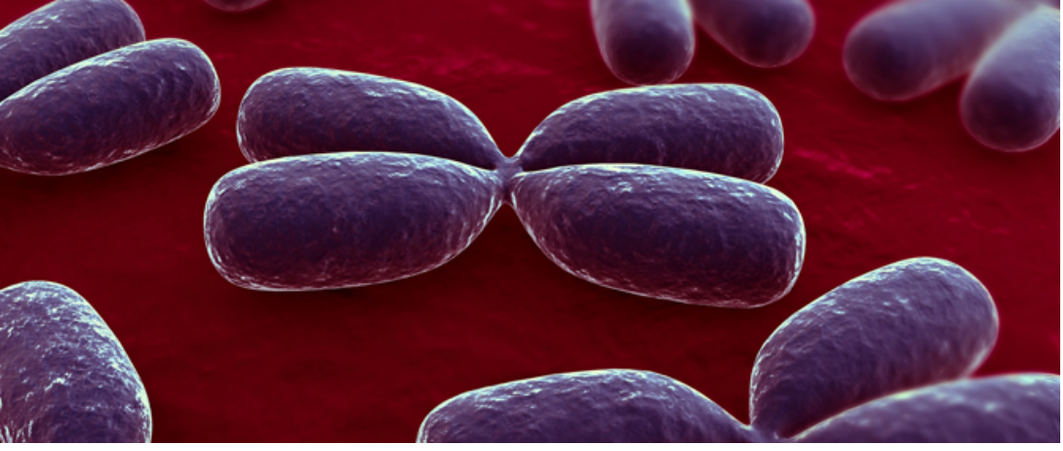
Florian Schneebauer, MSc.
Laboratory Manager

Prostate Health Sensor

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

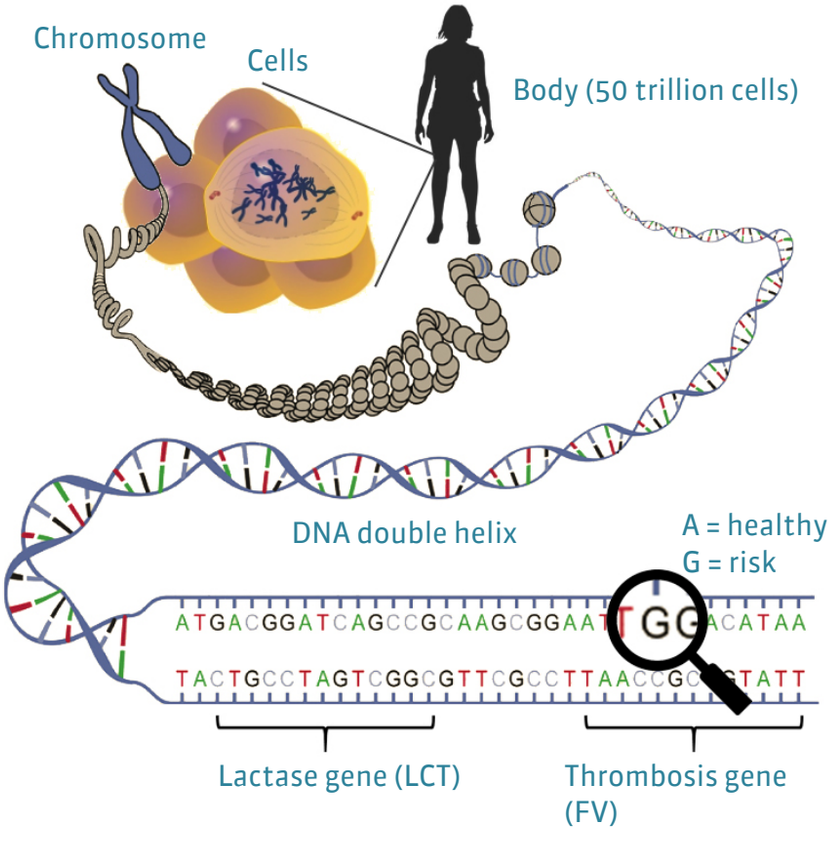
Order number:
DEMO_DS

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.

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PHARMACO GENETICS
Not ordered

ONCOLOGY

CARDIOVASCULAR SYSTEM
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METABOLISM
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SCIENCE

ADDITIONAL INFORMATION

Prostate Health Sensor

Effective prevention and treatment of prostate cancer

Prostate

The prostate is a sex gland. It lies below the bladder, near the beginning of the urethra and is about the size and shape of a chestnut. It consists of several glands that produce a secretion that is discharged during ejaculation into the urethra, where it is mixed with the sperm. Thus, it plays an important role in reproduction. Unfortunately, the prostate is also associated with a number of diseases, some of which can be serious or even fatal. As a result, every man should undergo an annual examination of the prostate after the age of 45.

Benign prostatic hyperplasia (BPH) is a non-cancerous enlargement of the prostate gland caused by the abnormal proliferation of certain cells. The disease is very common and usually develops with age. The risk of an enlarged prostate depends on genetic and lifestyle factors. About 10-20% of men between ages 50 and 59 have this condition but this increases to 25-35% of men between ages 60 and 69. In contrast to prostate cancer, the symptoms of prostate enlargement develop very rapidly. Typical symptoms are: pain during urination, frequent urination, difficult and long-lasting urination accompanied by abdominal pressure. If the bladder is significantly obstructed, urine may accumulate in the kidney, which can cause a life-threatening condition. An enlarged prostate can be treated either with medication depending on the extent or reduced surgically using a laser beam. However, the best option remains prevention. Since benign enlargement of the prostate is closely related to a combination of genes and lifestyle, modifications in lifestyle can greatly reduce the risk of disease for genetically predisposed people.

Prostate cancer is one of the most common cancers for men. About 15% of men will be diagnosed with prostate cancer at some point in life. In most cases, prostate cancer does not produce symptoms until it has progressed, and so it is usually detected only at a later stage through symptoms such as urinary symptoms, bone pain, weight loss and anemia. The main complaints arising refer to urination disorders: delayed onset, a weak jet/drip or the interruption of the urinary stream during urination. Residual urine often remains in the bladder. Other frequently observed symptoms include: increased or predominantly nocturnal urination, frequent urination in small quantities and difficulty or pain when urinating. Pressure damage to nerves in the sacral area may cause erection problems. Visible blood in the urine or sperm is rare but significant. A prostate tumor does not cause discomfort so
symptoms usually appear only after the tumor has spread to nearby lymph nodes or into the bones. At that point, the most common symptoms are pain in the spine and pelvis. In most cases, bone metastases are the predominant disease and they are also the most common cause of death from prostate cancer. The advanced stage is often accompanied by anaemia and weight loss. Prostate cancer can only be treated successfully before it has spread, so early detection is crucial for effective treatment. That is why men over the age of 45 should be tested annually for prostate cancer. Treatment options include surgery with complete removal of the prostate, radiation therapy, hormone therapy and, in some cases, chemotherapy.

If the cancer is treated early, before spreading to other tissues, the 5-year survival rate is approximately 90%. After the cancer spreads, the 5-year survival rate is only around 35%, which is why early detection is so important. The lifetime risk for the diagnosis of prostate cancer is about 11%. It is estimated that approximately 20% of patients with prostate cancer die. About half of the cases of prostate cancer are caused by genetic variations. It is now possible to test these genes to determine the personal risk prior to the occurrence of prostate cancer. If the risk is significantly increased, a preventive program can greatly reduce the risk of developing of the disease. Additionally, you can detect potential diseases at an early stage through a more intensive monitoring program and allow for timely treatment. More serious and unpleasant consequences can be prevented in most cases.

Relevant genes for prostate

Several genetic variations have been identified, which taken individually slightly increase or decrease the risk of prostate cancer. Taken together, they have a significant impact on the risk probability. The analysis of relevant genetic variations came to the following conclusion:

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
TCF2	rs4430796	G>A	A/A
LOC124685	rs1859962	T>G	T/T
8q24 region2	rs16901979	C>A	C/C
8q24 region3	rs6983267	T>G	G/G
8q24 region1	rs1447295	C>A	C/C
VDR	rs2107301	C>T	C/C
8q24	rs4242382	G>A	G/G
8q24	rs7837688	T>G	G/G
8q24	rs2011077	A>G	G/A
RNASEL	rs627928	G>T	G/T

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

Summary of effects

Here you can see a summary of the impact your genetic variations have on your health:

- Your risk of developing prostate cancer is lower than that of the general population.

What is your risk of prostate cancer?

LOW AVERAGE RISK HIGHER

When are medical checkups recommended?

FROM 45 YEARS EARLIER

Prevention

You do not have an elevated genetic risk for this disease. In fact, your genetic profile actually gives you a below-average chance of developing this disease. You should follow only the general rules of a healthy life and submit to an annual prostate examination after the age of 45 years. In this way, the age-related (non-genetic) forms of prostate diseases can be recognized and treated in time.

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

Effect on relevant medication

	Effect	Breakdown	Dose
Abiraterone	✓	✓	✓
Aprepitant	✓	↑	↑
Cabazitaxel	✓	↑	↑
Degarelix	✓	✓	✓
Domperidone	✓	✓	✓
Erlotinib	✓	↑	↑
Flutamide	✓	↑	↑
Halothane	✓	✓	✓
Imatinib	↑	↑	↓
Lidocain	✓	✓	✓
Metoclopramide	✓	✓	✓
Oxycodone	✓	↑	✓

	Effect	Breakdown	Dose
Alfentanil	✓	↑	↑
Bicalutamide	✓	↑	↑
Codeine	✓	✓	✓
Docetaxel	✓	↑	↑
Doxorubicin	✓	↑	↑
Etoposide	✓	↑	↑
Gefitinib	✓	↑	↑
Hydrocodone	✓	✓	✓
Isoflurane	✓	✓	✓
Methadone	✓	↑	↑
Mitoxantrone	✓	✓	✓
Paclitaxel	✓	✓	✓

	Effect	Breakdown	Dose
Anastrozole	✓	✓	✓
Buprenorphine	✓	↑	↑
Cyclophosphamide	✓	↑	↑
Dolasetron	✓	✓	✓
Enflurane	✓	✓	✓
Fentanyl	✓	↑	↑
Goserelin	✓	✓	✓
Ifosfamide	↑	↑	↓
Levacetylmethadol	✓	↑	↑
Methoxyflurane	✓	✓	✓
Nilutamide	✓	✓	✓
Paracetamol	✓	✓	✓

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.

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SCIENCE

ADDITIONAL INFORMATION

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This chapter shows the science behind the test.

Prostate Health Sensor

TCF2 - Transcription factor 2 (rs4430796)

The transcription factor 2 (TCF-2 or HNF1B) forms a heterodimer with TCF1, and activates or inhibits the expression of different target genes. The polymorphism rs4430796 is associated with an increased risk of prostate cancer.

RES	Genotype	POP	Possible results
X	A/A	34%	Increased risk of prostate cancer (OR: 1.4)
	A/G	44%	No increased risk of prostate cancer
	G/G	23%	No increased risk of prostate cancer

References

Zheng et al. Cumulative association of five genetic variants with prostate cancer. N Engl J Med. 2008 Feb 28,358(9):910-9.

Levin et al. Chromosome 17q12 variants contribute to risk of early-onset prostate cancer. Cancer Res. 2008 Aug 15,68(16):6492-5.

Gudmundsson et al. Two variants on chromosome 17 confer prostate cancer risk, and the one in TCF2 protects against type 2 diabetes. Nat Genet. 2007 Aug,39(8):977-83.

LOC124685 - Myosin, light chain 6, alkali, smooth muscle and non-muscle pseudogene (rs1859962)

A genome-wide association study has shown that the polymorphism rs1859962 on chromosome 17q24.3 is associated with an increased risk of prostate cancer.

RES	Genotype	POP	Possible results
X	T/T	34%	No increased risk of prostate cancer
	T/G	47%	No increased risk of prostate cancer
	G/G	19%	Increased risk of prostate cancer (OR: 1.28)

References

Sun et al. Cumulative effect of five genetic variants on prostate cancer risk in multiple study populations. Prostate. 2008 Sep 1,68(12):1257-62.

Levin et al. Chromosome 17q12 variants contribute to risk of early-onset prostate cancer. Cancer Res. 2008 Aug 15,68(16):6492-5.

Zheng et al. Cumulative association of five genetic variants with prostate cancer. N Engl J Med. 2008 Feb 28,358(9):910-9

8q24 region 2 (rs16901979)

Several studies have shown that different polymorphisms on 8q24 increase the risk of prostate cancer.

RES	Genotype	POP	Possible results
	A/A	8%	Increased risk of prostate cancer (OR: 1.53)
	A/C	26%	Increased risk of prostate cancer (OR: 1.53)
X	C/C	66%	No increased risk of prostate cancer

References

Zheng et al. Cumulative association of five genetic variants with prostate cancer. N Engl J Med. 2008 Feb 28,358(9):910-9

Cheng et al. 8q24 and prostate cancer: association with advanced disease and meta-analysis. Eur J Hum Genet. 2008 Apr,16(4):496-505.

Levin et al. Chromosome 17q12 variants contribute to risk of early-onset prostate cancer. Cancer Res. 2008 Aug 15,68(16):6492-5.

8q24 region 3 (rs6983267)

Several studies have shown that different polymorphisms on 8q24 increase the risk of prostate cancer.

RES	Genotype	POP	Possible results
	T/T	20%	No increased risk of prostate cancer
	T/G	39%	Increased risk of prostate cancer (OR: 1.25)
X	G/G	42%	Increased risk of prostate cancer (OR: 1.25)

References

Haiman et al. A common genetic risk factor for colorectal and prostate cancer. Nat Genet. 2007 Aug,39(8):954-6.

Yeager et al. Genome-wide association study of prostate cancer identifies a second risk locus at 8q24. Nat Genet. 2007 May,39(5):645-9. Epub 2007 Apr 1.

Zheng et al. Cumulative association of five genetic variants with prostate cancer. N Engl J Med. 2008 Feb 28,358(9):910-9.

Cheng et al. 8q24 and prostate cancer: association with advanced disease and meta-analysis. Eur J Hum Genet. 2008 Apr,16(4):496-505.

8q24 region 1 (rs1447295)

Several studies have shown that different polymorphisms on 8q24 increase the risk of prostate cancer.

RES	Genotype	POP	Possible results
	A/A	5%	Increased risk of prostate cancer (OR: 1.22)
	A/C	27%	Increased risk of prostate cancer (OR: 1.22)
X	C/C	69%	No increased risk of prostate cancer

References

Zheng et al. Association between two unlinked loci at 8q24 and prostate cancer risk among European Americans. J Natl Cancer Inst. 2007 Oct 17,99(20):1525-33. Epub 2007 Oct 9.

Amundadottir et al. A common variant associated with prostate cancer in European and African populations. Nat Genet. 2006 Jun,38(6):652-8. Epub 2006 May 7.

Freedman et al. Admixture mapping identifies 8q24 as a prostate cancer risk locus in African-American men. Proc Natl Acad Sci U S A. 2006 Sep 19,103(38):14068-73. Epub 2006 Aug 31.

VDR - vitamin D (1,25- dihydroxyvitamin D3) receptor (rs2107301)

The VDR gene encodes the vitamin D receptor, part of the steroid receptors family. It is a transcription factor that regulates the activity of specific target genes, and thus affects the metabolism. The rs2107301 polymorphism is associated with an increased risk of prostate cancer.

RES	Genotype	POP	Possible results
	T/T	49%	Increased risk of prostate cancer (OR: 2.47)
	T/C	35%	Increased risk of prostate cancer (OR: 1.11)
X	C/C	16%	No increased risk of prostate cancer

References

Schäfer et al. No association of vitamin D metabolism-related polymorphisms and melanoma risk as well as melanoma prognosis: a case-control study. Arch Dermatol Res. 2012 Jul,304(5):353-61.

Holt et al. Vitamin D pathway gene variants and prostate cancer risk. Cancer Epidemiol Biomarkers Prev. 2009 Jun,18(6):1929-33.

Holick et al. Comprehensive association analysis of the vitamin D pathway genes, VDR, CYP27B1, and CYP24A1, in prostate cancer. Cancer Epidemiol Biomarkers Prev. 2007 Oct,16(10):1990-9.

8q24 (rs4242382)

Several studies have shown that different polymorphisms on 8q24 increase the risk of prostate cancer.

RES	Genotype	POP	Possible results
	A/A	6%	Increased risk of prostate cancer (OR: 2.1)
	A/G	26%	Increased risk of prostate cancer (OR: 1.91)
X	G/G	68%	No increased risk of prostate cancer

References

Zheng et al. Association between two unlinked loci at 8q24 and prostate cancer risk among European Americans. J Natl Cancer Inst. 2007 Oct 17,99(20):1525-33. Epub 2007 Oct 9.

Zheng et al. Cumulative association of five genetic variants with prostate cancer. N Engl J Med. 2008 Feb 28,358(9):910-9.

Fitzgerald et al. Analysis of recently identified prostate cancer susceptibility loci in a population-based study: associations with family history and clinical features. Clin Cancer Res. 2009 May 1,15(9):3231-7.

8q24 (rs7837688)

Several studies have shown that different polymorphisms on 8q24 increase the risk of prostate cancer.

RES	Genotype	POP	Possible results
	T/T	2%	No increased risk of prostate cancer
	T/G	18%	Increased risk of prostate cancer (OR: 1.91)
X	G/G	81%	Increased risk of prostate cancer (OR: 1.67)

References

Zheng et al. Association between two unlinked loci at 8q24 and prostate cancer risk among European Americans. J Natl Cancer Inst. 2007 Oct 17,99(20):1525-33. Epub 2007 Oct 9.

Zheng et al. Cumulative association of five genetic variants with prostate cancer. N Engl J Med. 2008 Feb 28,358(9):910-9.

Lindstrom et al. Characterizing associations and SNP-environment interactions for GWAS-identified prostate cancer risk markers--results from BPC3. PLoS One. 2011 Feb 24,6(2):e17142.

8q24 (rs2011077)

Several studies have shown that different polymorphisms on 8q24 increase the risk of prostate cancer.

RES	Genotype	POP	Possible results
	A/A	62%	No increased risk of prostate cancer
X	A/G	30%	Increased risk of prostate cancer (OR: 2.4)
	G/G	8%	Increased risk of prostate cancer (OR: 6.2)

References

Ma et al. Polymorphisms of fibroblast growth factor receptor 4 have association with the development of prostate cancer and benign prostatic hyperplasia and the progression of prostate cancer in a Japanese population. Int J Cancer. 2008 Dec 1,123(11):2574-9.

RNASEL - Ribonuclease L (2',5'-oligoisoadenylate synthetase-dependent) (rs627928)

RNase L, encoded by the RNASEL gene, is a ribonuclease that metabolizes viral and cellular RNA. The rs627928 polymorphism is associated with an increased risk of prostate cancer.

RES	Genotype	POP	Possible results
	T/T	30%	Increased risk of prostate cancer (OR: 1.4)
X	T/G	44%	Increased risk of prostate cancer (OR: 1.24)
	G/G	26%	No increased risk of prostate cancer

References

Mi et al. An update analysis of two polymorphisms in encoding ribonuclease L gene and prostate cancer risk: involving 13,372 cases and 11,953 controls. Genes Nutr. 2011 Nov,6(4):397-402.

Breyer et al. Genetic variants and prostate cancer risk: candidate replication and exploration of viral restriction genes. Cancer Epidemiol Biomarkers Prev. 2009 Jul,18(7):2137-44.

Li et al. RNASEL gene polymorphisms and the risk of prostate cancer: a meta-analysis. Clin Cancer Res. 2006 Oct 1,12(19):5713-9.

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
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SCIENCE

ADDITIONAL INFORMATION

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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

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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
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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 19:21:52

Product codes
M7PRO

Current version
V538

Ordering company
ProGenom GmbH
Riedstrasse 1
6343 Rotkreuz
SWITZERLAND

Analyzing company
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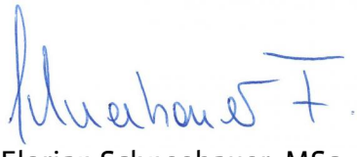
Laboratory Director

Dr. Daniel Wallerstorfer Bsc.

Laboratory Manager



Florian Schneebauer, MSc.

NOTES: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