ProMedicDNA

Diabetes 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

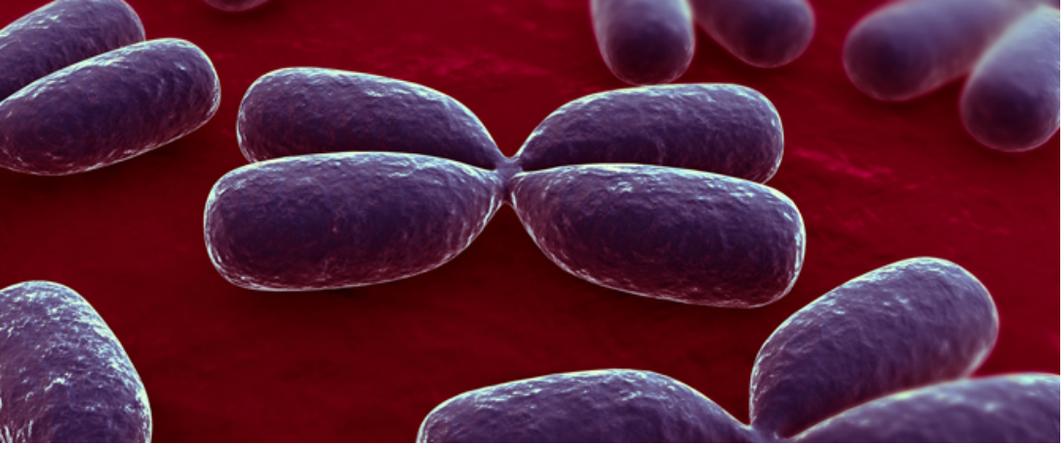
Florian Schneebauer, MSc.
Laboratory Manager

Diabetes Sensor

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

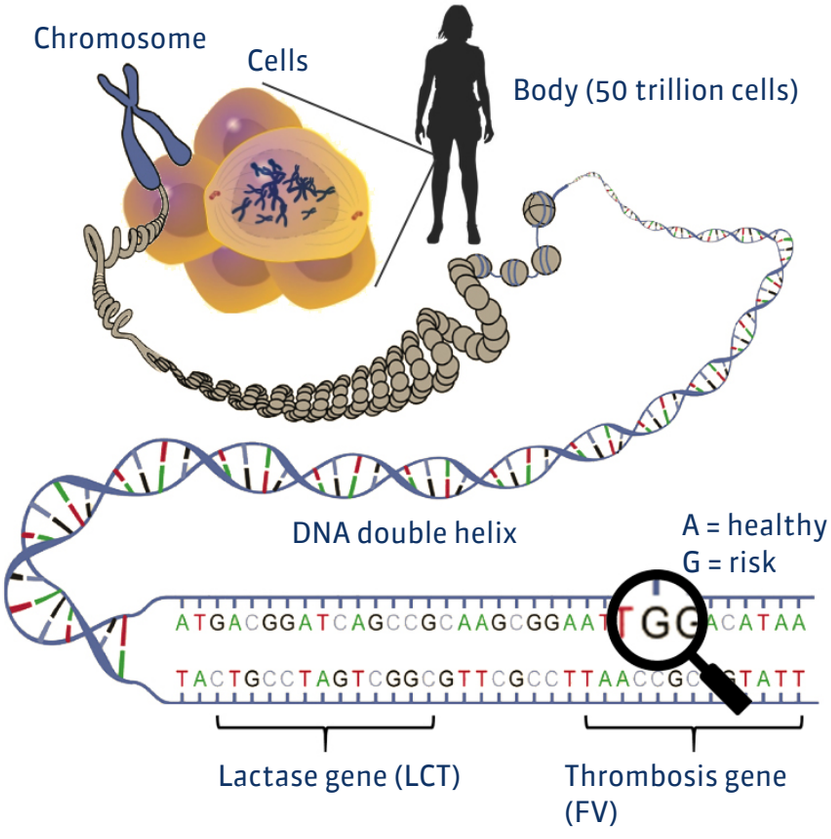
Order number:
DEMO_DS

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.

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CARDIOVASCULAR SYSTEM
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SCIENCE

ADDITIONAL INFORMATION

Diabetes Sensor

Prevention and effective treatment of diabetes

Diabetes Type 2

Diabetes is a common metabolic disease in which the body loses the ability to properly regulate blood sugar. The ability to regulate blood sugar declines somewhat with age, and almost one in ten people in the industrialized world suffers from diabetes.

Sugar is the primary fuel for our cells. It is transported through the blood along with oxygen and other nutrients. High levels of blood sugar damage cells and low levels prevent them from functioning properly. Therefore, the body has a mechanism that precisely regulates blood sugar levels and keeps it in the right range. When you consume a large amount of sugar, that sugar enters the bloodstream. After a certain point, the body begins to filter the sugar from the blood and stores it. If you go without eating for a long time, your body releases sugar from the reserve into the bloodstream. In this way, the blood sugar level remains constant and ensures that all cells are supplied with just the right amount of fuel.

As we grow older, this regulatory process gradually becomes less exact. Certain risk factors -including lack of exercise, excessive weight and certain genetic traits -accelerate this gradual decline in precision. In some people, blood sugar rises to levels that trigger a variety of physical ailments, some of which can be life-threatening. In this case, the condition is called diabetes type 2 and requires medical treatment. Diabetes is often accompanied by a number of other ailments. High blood pressure, blood lipid disorders, neuropathy, blood vessel damage, kidney disease and even blindness are all common effects of untreated diabetes. In order to prevent these secondary conditions, a person with diabetes must maintain consistent and regular control of their blood sugar levels. A physician is usually able to perform a fasting blood glucose test or a glucose tolerance test to diagnose diabetes. In these tests, the patient drinks a large amount of sugary liquid and the doctor then measures the blood glucose levels at regular intervals. The results will show how effectively the body regulates blood sugar.

The treatment plan for diabetes depends on the level of blood sugar. In most cases, diet and exercise will keep diabetes in check. Sometimes oral medication will be prescribed and in rare cases, injections of insulin will become necessary.

Diabetes type 2 is a lifestyle disease that is especially prevalent in developed countries where large quantities of many processed foods are available. Obesity is the most important risk factor for diabetes. Certain genetic traits that play a role in regulating blood sugar also increase the risk of diabetes in some individuals.

By analysing relevant genes, your personal genetic risk of developing diabetes can be determined. Individuals with a high risk of diabetes can then follow specific preventative programs that will reduce their risk of developing the disease.

Genes associated with diabetes type 2

So far, scientists have identified several genetic traits that can increase the risk of developing type 2 diabetes. An analysis of all relevant traits allows us to determine your risk of diabetes, as well as some other genetic traits linked to this disease.The following genes influence blood sugar regulation and are associated with the risk of diabetes type 2.

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
TCF7L2	rs7903146	VS3C>T	C/C
HIGD1C	rs12304921	A>G	A/A
HHEX	rs1111875	A>G	G/A
IL6	rs1800795	G/C Pos. -174	G/C
IL10	rs1800872	C/A Pos. -592	C/A
PPARG	rs1801282	Pro12Ala	C/C
FTO	rs9939609	A/T	T/A
KCNJ11	rs5219	C>T	C/T
NOS1AP	rs10494366	T>G	T/T

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

Summary of effects

Several risk genes for the development of type 2 diabetes were analysed and many of the genetic traits that increase the risk of diabetes are fairly common. Thus, almost every person has, to some extent, a genetic risk. You may carry more traits than an average person and have a higher risk of developing diabetes; or you may carry fewer genes that increase the risk of diabetes and have a lower risk of developing diabetes. Here, you can see a summary of the influence that genetic variations have on your health:

- You do not have an elevated risk of type 2 diabetes
- Metformin is less likely to prevent you from developing diabetes type 2
- Although the drug glibenclamide is effective in reducing blood sugar, its breakdown is impaired.
- The drug tolbutamide is not very effective in reducing blood sugar.
- The drug glimepiride is not very effective in reducing blood sugar.
- If you develop type 2 diabetes, you are less likely to need insulin
- You do not have an elevated risk of gestational diabetes

Your genetic risk of diabetes type 2

requiring insulin with DMT2

Effectiveness of glimepiride

Glibenclamide as a blood sugar regulator

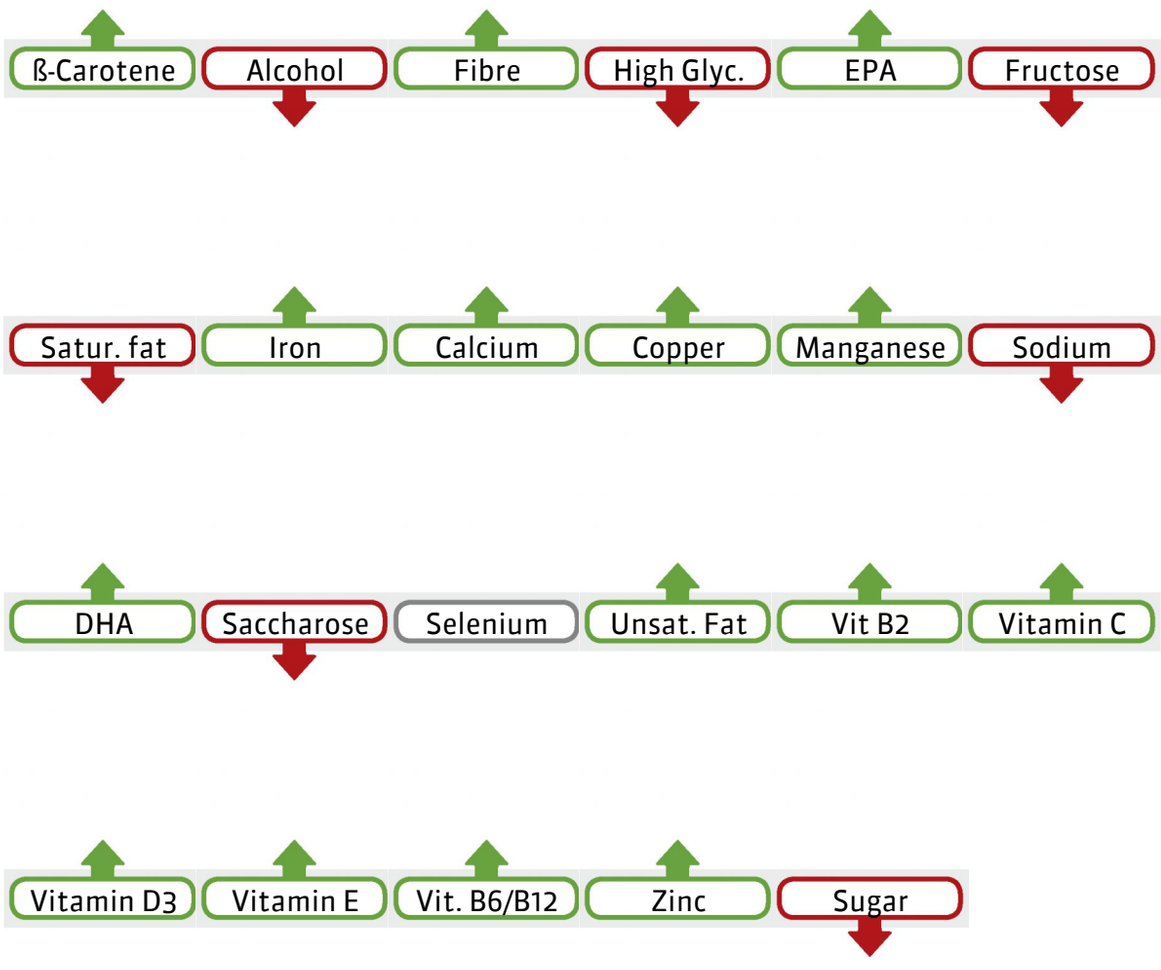
Effectiveness of metformin for prevention

Effectiveness of tolbutamide

Nutritional Genes - Metabolism

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.

Prevention

Your genetic analysis shows that you have a reduced risk of diabetes type 2. However, even individuals with a low risk may develop the disease, therefore we want to give you some general guidelines for a healthy lifestyle.

We recommend an annual checkup- including a glucose tolerance test- for people 45 years and older. This test measures how your body reacts to sugar. Your blood sugar should also be tested regularly so that diabetes is detected earlier and treated properly.

Complications associated with diabetes, such as high blood pressure and problems with blood lipids, can be prevented or treated if diabetes is detected early enough.

It is also advisable to follow a low-calorie, high-fibre diet in order to maintain your blood sugar levels. Frequent exercise (at least 30 minutes daily, 5 days a week) is healthy for everyone and also reduces the risk of developing diabetes. Physical activity speeds up your metabolism and causes your body to use more blood sugar for fuel thereby lowering your blood sugar level to a safe range.

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
Acarbose	✓	✓	✓
Atorvastatin	✓	↑	↑
Chlorpropamide	✓	↓	↓
Fluvastatin	✓	✓	✓
Gliclazide	✓	✕	✕
Gliquidone	✓	✓	✓
Insulin Glargine	✓	✓	✓
Liraglutide	✓	✓	✓
Metformin	↓	✓	✓
Pioglitazone	✓	↓	↓
Saxagliptin	✓	✓	✓
Tolbutamide	↓	✕	✕

	Effect	Breakdown	Dose
Acetohexamide	✓	✓	✓
Carbutamide	✓	✓	✓
Dapagliflozin	✓	✓	✓
Glibenclamide	✓	↓	↓
Glimepiride	↓	✕	✕
Glisoxepide	✓	✓	✓
Insulin Lispro	✓	✓	✓
Lovastatin	✓	↑	↑
Nateglinide	✓	↓	↓
Repaglinide	✓	↑	↑
Sitagliptin	✓	✓	✓
Troglitazone	✓	↓	↓

	Effect	Breakdown	Dose
Alogliptin	✓	✓	✓
Cerivastatin	✓	↑	↑
Exenatide	✓	✓	✓
Glibornuride	✓	✕	✕
Glipizide	✓	✕	✕
Insulin Aspart	✓	✓	✓
Linagliptin	✓	✓	✓
Metahexamide	✓	✓	✓
Phenformin	✓	✓	✓
Rosiglitazone	✓	✕	✕
Tolazamide	✓	✓	✓
Vildagliptin	✓	✓	✓

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

This chapter shows the science behind the test.

Diabetes Sensor

TCF7L2 - Transcription factor 7-like 2 (T-cell specific, HMG-box) (rs7903146)

TCF7L2 (transcription factor 7-like 2) is a transcription factor which affects many different genes. The polymorphism rs7903146 is considered the most important genetic risk factor for type 2 diabetes.

RES	Genotype	POP	Possible results
X	C/C	61%	No increased risk of type 2 diabetes mellitus
	C/T	32%	Increased risk of type 2 diabetes mellitus (OR: 1.65) In case of diabetes, insulin substitution treatment is necessary sooner
	T/T	7%	Increased risk of type 2 diabetes mellitus (OR: 2.77) In case of diabetes, insulin substitution is typically required sooner

References

Lyssenko et al. Mechanisms by which common variants in the TCF7L2 gene increase risk of type 2 diabetes. J Clin Invest. Aug 1, 2007, 117(8): 2155–2163.

Cauchi et al. TCF7L2 genetic defect and type 2 diabetes. Curr Diab Rep. 2008 Apr,8(2):149-55.

Bodhini et al. The rs12255372(G/T) and rs7903146(C/T) polymorphisms of the TCF7L2 gene are associated with type 2 diabetes mellitus in Asian Indians. Metabolism. 2007 Sep,56(9):1174-8.

Hivert MF et al. Updated genetic score based on 34 confirmed type 2 diabetes Loci is associated with diabetes incidence and regression to normoglycemia in the diabetes prevention program. Diabetes. 2011,60(4):1340-8.

HIGD1C - HIG1 hypoxia inducible domain family, member 1C (rs12304921)

A comprehensive study associated the polymorphism rs12304921 on HIGD1C gene with an increased risk of type 2 diabetes.

RES	Genotype	POP	Possible results
X	A/A	56%	No increased risk of diabetes mellitus type 2.
	G/A	36%	Increased risk of type 2 diabetes mellitus (OR: 2.5)
	G/G	8%	Increased risk of type 2 diabetes mellitus (OR: 1.94)

References

The Wellcome Trust Case Control Consortium. 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.

Ryoo H et al. Heterogeneity of genetic associations of CDKAL1 and HHEX with susceptibility of type 2 diabetes mellitus by gender. Eur J Hum Genet. 2011 Jun,19(6):672-5.

Prasad RB et al. Genetics of type 2 diabetes-pitfalls and possibilities. Genes (Basel). 2015 Mar 12,6(1):87-123.

HHEX - Hematopoietically expressed homeobox (rs1111875)

The HHEX gene encodes a transcription factor involved in many developmental processes. A genome-wide study has shown that carriers of the G-allele have an increased risk of type 2 diabetes.

RES	Genotype	POP	Possible results
	A/A	34%	No increased risk of diabetes mellitus type 2.
X	G/A	40%	Increased risk of type 2 diabetes mellitus (OR: 1.21)
	G/G	26%	Increased risk of type 2 diabetes mellitus (OR: 1.44)

References

van Vliet-Ostaptchouk et al. HHEX gene polymorphisms are associated with type 2 diabetes in the Dutch Breda cohort. Eur J Hum Genet. 2008 May,16(5):652-6

Omori et al. Association of CDKAL1, IGF2BP2, CDKN2A/B, HHEX, SLC30A8, and KCNJ11 with susceptibility to type 2 diabetes in a Japanese population. Diabetes. 2008 Mar,57(3):791-5. Epub 2007 Dec 27.

Furukawa et al. Polymorphisms in the IDE-KIF11-HHEX gene locus are reproducibly associated with type 2 diabetes in a Japanese population. J Clin Endocrinol Metab. 2008 Jan,93(1):310-4.

Hivert MF et al. Updated genetic score based on 34 confirmed type 2 diabetes Loci is associated with diabetes incidence and regression to normoglycemia in the diabetes prevention program. Diabetes. 2011,60(4):1340-8.

IL-6 - interleukin 6 (rs1800795)

Interleukin-6 is (IL-6) one of the pro-inflammatory cytokines and it is an essential part of the immune response to inflammatory processes. The polymorphism rs1800795, located in the promoter region of the gene, alters the expression of the cytokine. Carriers of the C- allele produce less IL-6.

RES	Genotype	POP	Possible results
	C/C	5%	Protection against type 2 diabetes mellitus (OR: 0.91)
X	G/C	19%	Protection against type 2 diabetes mellitus (OR: 0.91)
	G/G	77%	Increased risk of type 2 diabetes mellitus (OR: 1.51)

References

Huth et al. IL6 gene promoter polymorphisms and type 2 diabetes: joint analysis of individual participants' data from 21 studies. Diabetes. 2006 Oct,55(10):2915-21.

Illig et al. Significant association of the interleukin-6 gene polymorphisms C-174G and A-598G with type 2 diabetes. J Clin Endocrinol Metab. 2004 Oct,89(10):5053-8.

Fishman et al. The effect of novel polymorphisms in the interleukin-6 (IL-6) gene on IL-6 transcription and plasma IL-6 levels, and an association with systemic-onset juvenile chronic arthritis. J Clin Invest. 1998 Oct 1,102(7):1369-76.

IL10 - Interleukin 10 (rs1800872)

Interleukin-10 (IL-10) is one of the anti-inflammatory cytokines and has numerous functions in the immune system. The polymorphism is associated with an increased risk of type 2 diabetes and increased resistance to insulin.

RES	Genotype	POP	Possible results
	C/C	22%	No increased risk for type 2 diabetes mellitus No increased insulin resistance
X	C/A	44%	No increased risk for type 2 diabetes mellitus No increased insulin resistance
	A/A	35%	Increased risk of type 2 diabetes mellitus (OR: 1.63) Increased insulin resistance (OR: 1.99)

References

Bai et al. Association between interleukin 10 gene polymorphisms and risk of type 2 diabetes mellitus in a Chinese population. J Int Med Res. 2014 Apr 23.

Scarpelli et al. Variants of the interleukin-10 promoter gene are associated with obesity and insulin resistance but not type 2 diabetes in caucasian italian subjects. Diabetes. 2006 May,55(5):1529-33.

Tarabay M et al. African vs. Caucasian and Asian difference for the association of interleukin-10 promotor polymorphisms with type 2 diabetes mellitus (a meta-analysis study). Meta Gene. 2016 Mar 4,9:10-7.

Saxena M et al. An interleukin-10 gene promoter polymorphism (-592A/C) associated with type 2 diabetes: a North Indian study. Biochem Genet. 2012 Aug,50(7-8):549-59.

PPARG - Peroxisome proliferator-activated receptor gamma (rs1801282)

Peroxisome proliferator-activated receptor gamma (PPAR-γ or PPARG), also known as the glitazone receptor or NR1C3 (nuclear receptor subfamily 1, group C, member 3) is a type II nuclear receptor that, in humans, is encoded by the PPARG gene. PPARG regulates fatty acid storage and glucose metabolism. The genes activated by PPARG stimulate lipid uptake and adipogenesis by fat cells. PPARG knockout mice fail to generate adipose tissue when fed a high-fat diet.

RES	Genotype	POP	Possible results
	G/G	1%	No increased risk of diabetes mellitus type 2.
	G/C	13%	Increased risk of type 2 diabetes mellitus (OR: 1.19)
X	C/C	87%	Increased risk of type 2 diabetes mellitus (OR: 1:38)

References

Gouda et al. The association between the peroxisome proliferator-activated receptor-gamma2 (PPARG2) Pro12Ala gene variant and type 2 diabetes mellitus: a HuGE review and meta-analysis. Am J Epidemiol. 2010 Mar 15,171(6):645-55.

Altshuler et al. The common PPARgamma Pro12Ala polymorphism is associated with decreased risk of type 2 diabetes. Nat Genet. 2000 Sep,26(1):76-80.

Deeb et al. A Pro12Ala substitution in PPARgamma2 associated with decreased receptor activity, lower body mass index and improved insulin sensitivity. Nat Genet. 1998 Nov,20(3):284-7.

Hivert MF et al. Updated genetic score based on 34 confirmed type 2 diabetes Loci is associated with diabetes incidence and regression to normoglycemia in the diabetes prevention program. Diabetes. 2011,60(4):1340-8.

FTO - Fat mass and obesity associated (rs9939609)

Fat mass and excessive weight-associated protein also known as alpha-ketoglutarate-dependent dioxygenase FTO is an enzyme that, in humans, is encoded by the FTO gene located on chromosome 16. The amino acid sequence of the transcribed FTO protein shows high similarity with the enzyme AlkB, which oxidatively demethylates DNA. Recombinant FTO protein was first discovered to catalyze demethylation of 3-methylthymine in single-stranded DNA, and 3-methyluridine in single-stranded RNA, with low efficiency. The nucleoside N6-methyladenosine, an abundant modification in RNA, was then found to be a major substrate of FTO. The FTO gene expression was also found to be significantly up-regulated in the hypothalamus of rats after food deprivation and strongly correlated negatively with the expression of orexigenic galanin-like peptide which is involved in the stimulation of food intake.

RES	Genotype	POP	Possible results
	T/T	46%	No increased risk of diabetes mellitus type 2.
X	T/A	41%	Increased risk of type 2 diabetes mellitus (OR: 1.34)
	A/A	14%	Increased risk of type 2 diabetes mellitus (OR: 1.68)

References

Frayling et al. A Common Variant in the FTO Gene Is Associated with Body Mass Index and Predisposes to Childhood and Adult Obesity. Science. May 11, 2007, 316(5826): 889–894.

Wellcome Trust Case Control Consortium. 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.

Hertel et al. Genetic analysis of recently identified type 2 diabetes loci in 1,638 unselected patients with type 2 diabetes and 1,858 control participants from a Norwegian population-based cohort (the HUNT study). Diabetologia. 2008 Jun,51(6):971-7.

KCNJ11 - Potassium inwardly-rectifying channel, subfamily J, member 11 (rs5219)

The KCNJ11 gene (potassium inwardly rectifying-channel, subfamily J, member 11) is encoding the Kir2.6 protein, a subunit of the ATP-sensitive potassium channels. These channels are located in the cell membrane, and can use the hormone insulin to regulate the glucose concentration in blood. A defect can lead to increased glucose levels, and thus an increased risk of diabetes.

RES	Genotype	POP	Possible results
	C/C	56%	No increased risk of diabetes mellitus type 2. The drug Metformin is effective
X	C/T	35%	Increased risk of type 2 diabetes mellitus (OR: 1.23) The drug Metformin is less effective than normal
	T/T	9%	Increased risk of type 2 diabetes mellitus (OR: 1.65) The drug Metformin is less effective than normal

References

Florez et al. Type 2 Diabetes–Associated Missense Polymorphisms KCNJ11 E23K and ABCC8 A1369S Influence Progression to Diabetes and Response to Interventions in the Diabetes Prevention Program. Diabetes. Feb 2007, 56(2): 531–536.

Zhou et al. The E23K variation in the KCNJ11 gene is associated with type 2 diabetes in Chinese and East Asian population. J Hum Genet. 2009 Jul,54(7):433-5.

Omori et al. Association of CDKAL1, IGF2BP2, CDKN2A/B, HHEX, SLC30A8, and KCNJ11 with susceptibility to type 2 diabetes in a Japanese population. Diabetes. 2008 Mar,57(3):791-5. Epub 2007 Dec 27.

Florez et al. Haplotype structure and genotype-phenotype correlations of the sulfonylurea receptor and the islet ATP-sensitive potassium channel gene region. Diabetes. 2004 May,53(5):1360-8.

Hivert MF et al. Updated genetic score based on 34 confirmed type 2 diabetes Loci is associated with diabetes incidence and regression to normoglycemia in the diabetes prevention program. Diabetes. 2011,60(4):1340-8.

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. This NOS1AP polymorphism decreases the glucose-reducing effect of different drugs and is associated with an increased mortality rate.

RES	Genotype	POP	Possible results
X	T/T	20%	The drug Glibenclamide is effective The drug Tolbutamide is less effective/mortality rate is increased when using this drug The drug Glimepiride less effective/mortality rate is increased when using this drug
	G/T	43%	The drug Glibenclamide is less effective/mortality rate is increased when using this drug The drug Tolbutamide is effective The drug Glimepiride is effective
	G/G	37%	The drug Glibenclamide is less effective/mortality rate is increased when using this drug The drug Tolbutamide is effective The drug Glimepiride is effective

References

Tomás M et al. Polymorphisms in the NOS1AP gene modulate QT interval duration and risk of arrhythmias in the long QT syndrome. JACC. 2010 Jun 15,55(24):2745-52.

Treuer AV et al. NOS1AP modulates intracellular Ca(2+) in cardiac myocytes and is up-regulated in dystrophic cardiomyopathy. Int J Physiol Pathophysiol Pharmacol. 2014 Mar 13,6(1):37-46. eCollection 2014.

Becker et al. Common variation in the NOS1AP gene is associated with reduced glucose-lowering effect and with increased mortality in users of sulfonylurea. Pharmacogenet Genomics. 2008 Jul,18(7):591-7.

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

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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

qualityaustria
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 17:13:19

Product codes
M3DIA

Current version
V538

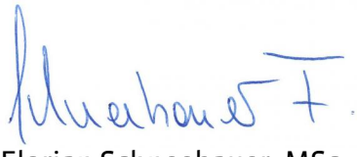
Ordering company
ProGenom GmbH
Riedstrasse 1
6343 Rotkreuz
SWITZERLAND

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

Diabetes Sensor

Jane Doe
DEMO_DS

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