DNAhealthControl

Schizophrenia Sensor

Max Mustermann
DEMO_LOTH

novogenia

COVER LETTER

Dear Ms. Mustermann,

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

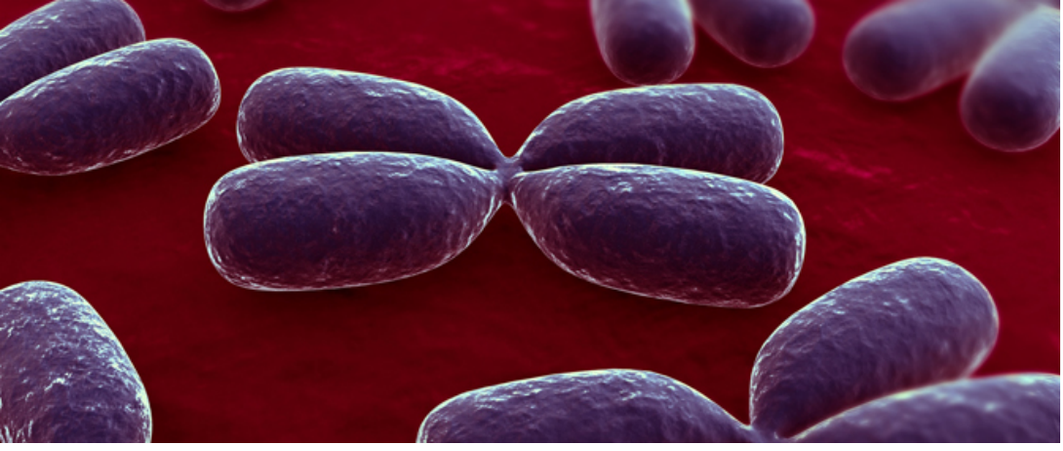
René Rohrmanstorfer, MSc.
Laboratory Manager

Schizophrenia Sensor

Personal analysis results for:
Max Mustermann | Date of birth: 01/01/1990

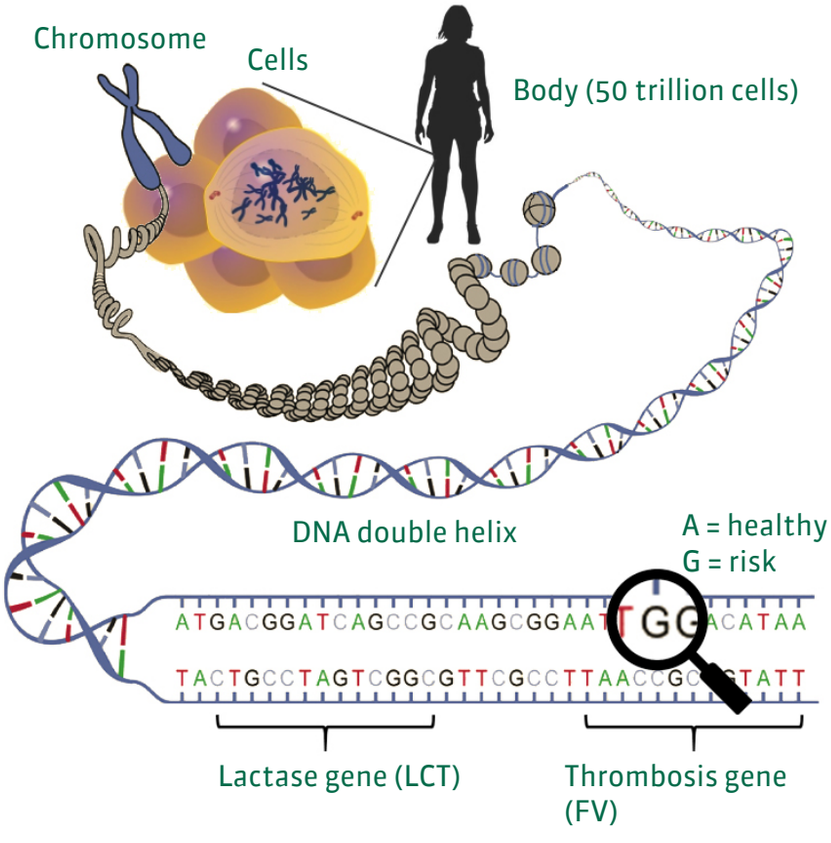
Order number:
DEMO_LOTH

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.

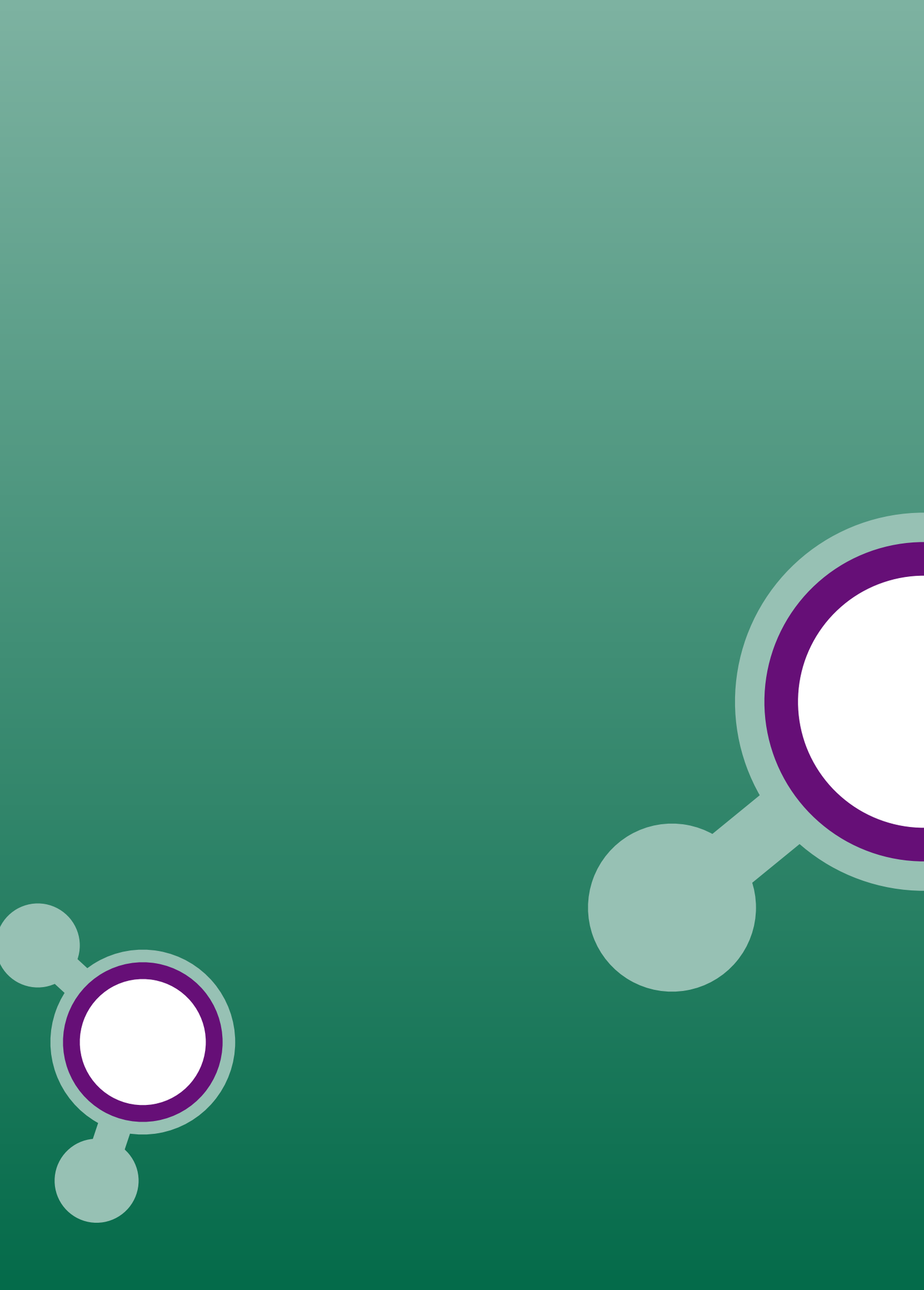
External influences can affect individual genes and disrupt their function, but the majority of our defective genes are inherited from our parents. Each embryo receives half of its genes from the father and half from the mother, resulting in a new human being with some characteristics of each parent.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

Schizophrenia Sensor



PHARMACO GENETICS
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ONCOLOGY
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CARDIOVASCULAR SYSTEM
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NEUROLOGY

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OTHERS
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SCIENCE

ADDITIONAL INFORMATION

Schizophrenia Sensor

Effective early detection and treatment of schizophrenia

Schizophrenia

Schizophrenia is a chronic and severe mental disorder that affects how a person thinks, feels and behaves. Patients sometimes appear to have lost touch with reality and often have difficulty in managing their everyday lives.

The disease has a strong genetic basis that affects both the risk of a person developing the disease as well as the likely age of onset. Genetic analyses allow us to identify a person's risk, the likely age of onset, and the effectiveness and potential side effects of common medications used to treat the disease.

Typical symptoms of the disease appear in three classes:

- "positive symptoms": include hallucinations, delusions and movement disorders.
- "negative symptoms": include flat emotions and intonation, reduced feelings of pleasure, difficulty sustaining activities and reduced speaking.
- "cognitive symptoms": include a decreased ability to understand complex concepts and make rational decisions, difficulty in focusing and an inability to use newly acquired information shortly after learning it.

Genes relevant to schizophrenia:

Several genes and polymorphisms associated with a risk of developing schizophrenia have already been scientifically identified. An analysis of these polymorphisms reveals the disease risk as well as other genetic characteristics relevant to this disease.

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
BDNF	rs6265	G>A	G/G
MTHFR	rs1801131	A>C	A/C
COMT	rs4680	G>A	A/G
MTHFR	rs1801133	C>T	C/C

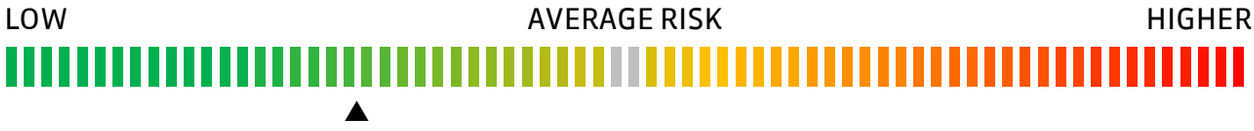
LEGEND: SYMBOL = Name of investigated genetic variation, rsNCBI = description of investigated genetic variation, GENOTYPE = result.

Summary of effects

Here is a summary of the impact genetic variations have on your health:

- Your risk of developing schizophrenia is not higher than the population average
- Should you develop schizophrenia, the average age of onset according to your genetic profile is: 30 Years

Risk of developing schizophrenia

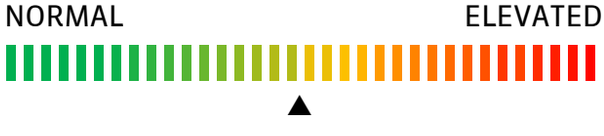


Average age of onset

Effects for sufferers only:

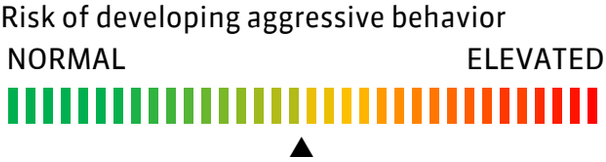
- Increased risk of developing so-called negative or minus symptoms (e.g. reduced emotional capacity for experience, indifference, anhedonia, lack of motivation, attention disorders)
- Increased risk of developing compulsive behavior
- Increased risk of developing aggressive behavior
- Weakness in solving complex tasks (so-called executive functions such as planning of tasks, problem-solving, action control, control over motivation and emotions)
- No increased risk of developing more severe symptoms

Risk of developing negative symptoms

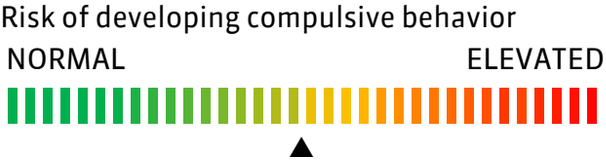


Solving of complex tasks

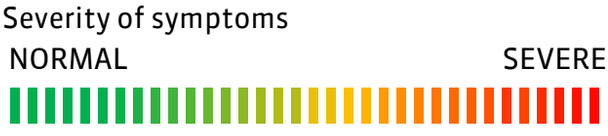
Risk of developing aggressive behavior



Risk of developing compulsive behavior



Severity of symptoms



Early detection

Diagnosing the first signs of the disease early on is important to make sure you receive adequate treatment in time. These are some of the symptoms you should look out for. Should you develop one or several of the symptoms, talk to your doctor to reach an accurate diagnosis:

- HALLUCINATIONS: seeing or hearing something that is not there
- PARANOIA: a constant feeling of being watched
- BEHAVIOUR: usual way of speaking or writing
- POSTURE: odd body posture
- INDIFFERENCE: to usually very important situations
- PERFORMANCE: deteriorates (academic/work)
- PERSONALITY: sudden or gradual change in personality
- WITHDRAWAL: from social situations
- BEHAVIOUR: irrational, angry or fearful responses to loved ones
- SLEEP: inability to sleep
- CONCENTRATION: difficult to concentrate
- RELIGION: extreme engagement in religion or occultism

Should you develop any of these symptoms, talk to your doctor so that he/she can diagnose the cause of the symptoms correctly.

Treatment

Treatment of schizophrenia can greatly increase a person's ability to function in society and improve the quality of life. The primary treatment for schizophrenia is medication, but it also includes rehabilitation programs, self-help groups, therapy and counseling.

Medication

Anti-psychotic medication can control the symptoms by reducing biochemical imbalances in the brain and thereby decreasing the likelihood of relapse.

Today we know a lot about the impact of the personal genetic profile on the effectiveness and side effects of potential medications used for schizophrenia. Your doctor can use the results of your genetic test to choose the right type and dosage of medication for optimal treatment.

Therapy and Counselling

Psychological counselling and different forms of talk therapy can help the patient better understand and manage the complications 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

Effect on relevant medication

	Effect	Breakdown	Dose
Agomelatine	✓	✓	✓
Asenapine	✓	✓	✓
Citalopram	✓	↑	✓
Clozapine	✓	✓	✓
Diazepam	✓	↑	↑
Duloxetine	✓	✓	✓
Fluphenazine	✓	✓	✓
Iloperidone	✓	✓	✓
Minaprine	✓	✓	✓
Nefazodone	✓	↑	↑
Paliperidone	✓	✓	✓
Pimozide	✓	↑	↑
Reboxetine	✓	↑	↑
Sertindole	✓	↑	↑
Thioridazine	✓	✓	✓
Trimipramine	✓	✓	✓
Ziprasidone	✓	↑	↑

	Effect	Breakdown	Dose
Amitriptyline	✓	✓	✓
Buspirone	✓	↑	↑
Clobazam	✓	↑	↑
Cyclobenzaprine	✓	✓	✓
Doxepin	✓	✓	✓
Escitalopram	✓	↑	✓
Fluvoxamine	✓	✓	✓
Imipramine	✓	✓	✓
Mirtazapine	✓	✓	✓
Nortriptyline	✓	✓	✓
Paroxetine	✓	✓	✓
Protriptyline	✓	✓	✓
Remoxipride	✓	✓	✓
Sertraline	✓	✓	✓
Trazodone	✓	↑	↑
Valproic Acid	✓	↓	↓
Zotepine	✓	✓	✓

	Effect	Breakdown	Dose
Aripiprazole	✓	↑	✓
Chlorpromazine	✓	✓	✓
Clomipramine	↑	✓	✓
Desipramine	✓	✓	✓
Droperidol	✓	↑	↑
Fluoxetine	✓	✕	✕
Haloperidol	✓	↑	✓
Mianserin	✓	✓	✓
Moclobemide	✓	✓	✓
Olanzapine	✓	✓	✓
Perphenazine	✓	✓	✓
Quetiapine	✓	↑	↑
Risperidone	✓	✓	✓
Sulpiride	✓	✓	✓
Trifluoperazine	✓	✓	✓
Venlafaxine	✓	✓	✓
Zuclopenthixol	✓	✓	✓

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.

Schizophrenia

COMT - Catechol-O-methyltransferase (rs4680)

The enzyme catechol-O-methyltransferase (COMT) can inactivate various substances (epinephrine, norepinephrine and dopamine) and initiate their breakdown. In addition, COMT may inhibit the effect of various drugs. The COMT rs4680 polymorphism is associated with psychological disorders, such as schizophrenia, eating disorders and alcoholism.

RES	Genotype	POP	Possible results
	G/G	41%	Higher risk of negative symptoms Higher risk of obsessive compulsive behaviour
X	A/G	44%	Higher risk of negative symptoms Higher risk of obsessive compulsive behaviour Higher risk of violent/aggressive behaviour Poorer executive function performance
	A/A	15%	Higher risk of violent/aggressive behaviour Poorer executive function performance

References

Wang Y et al. Analysis of association between the catechol-O-methyltransferase (COMT) gene and negative symptoms in chronic schizophrenia. Psychiatry Res. 2010 Sep 30,179(2):147-50.

Bhakta SG et al. The COMT Met158 allele and violence in schizophrenia: a meta-analysis. Schizophr Res. 2012 Sep,140(1-3):192-7.

Lock AA et al. Epistasis between COMT Val158Met and DRD3 Ser9Gly polymorphisms and cognitive function in schizophrenia: genetic influence on dopamine transmission. Rev Bras Psiquiatr. 2015 Jul-Sep,37(3):235-41.

Singh JP et al. A meta-analysis of the Val158Met COMT polymorphism and violent behavior in schizophrenia. PLoS One. 2012,7(8):e43423. doi: 10.1371/journal.pone.0043423. Epub 2012 Aug 14.

Bhakta SG et al. The COMT Met158 allele and violence in schizophrenia: a meta-analysis. Schizophr Res. 2012 Sep,140(1-3):192-7. doi: 10.1016/j.schres.2012.06.026. Epub 2012 Jul 10.

Tosato S et al. Effect of COMT genotype on aggressive behaviour in a community cohort of schizophrenic patients. Neurosci Lett. 2011 May 9,495(1):17-21.

Docherty AR et al. Anhedonia as a phenotype for the Val158Met COMT polymorphism in relatives of patients with schizophrenia. J Abnorm Psychol. 2008 Nov,117(4):788-98.

Kim YR et al. Catechol-O-methyltransferase Val158Met polymorphism in relation to aggressive schizophrenia in a Korean population. Eur Neuropsychopharmacol. 2008 Nov,18(11):820-5.

Zinkstok J et al. Catechol-O-methyltransferase gene and obsessive-compulsive symptoms in patients with recent-onset schizophrenia: preliminary results. Psychiatry Res. 2008 Jan 15,157(1-3):1-8. Epub 2007 Sep 12.

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
	C/T	33%	Increased risk of Schizophrenia Increased symptoms severity Poorer executive function performance
	T/T	8%	Increased risk of Schizophrenia Increased symptoms severity Poorer executive function performance

References

Roffman JL et al. MTHFR 677C -> T genotype disrupts prefrontal function in schizophrenia through an interaction with COMT 158Val -> Met. Proc Natl Acad Sci U S A. 2008 Nov 11,105(45):17573-8.

Roffman JL et al. Genetic variation throughout the folate metabolic pathway influences negative symptom severity in schizophrenia. Schizophr Bull. 2013 Mar,39(2):330-8.

Hei G et al. Association of serum folic acid and homocysteine levels and 5, 10-methylenetetrahydrofolate reductase gene polymorphism with schizophrenia. Zhonghua Yi Xue Za Zhi. 2014 Oct 14,94(37):2897-901.

El-Hadidy MA et al. MTHFR gene polymorphism and age of onset of schizophrenia and bipolar disorder. Biomed Res Int. 2014,2014:318483. doi: 10.1155/2014/318483. Epub 2014 Jul 3.

Hu CY et al. Methylenetetrahydrofolate reductase (MTHFR) polymorphism susceptibility to schizophrenia and bipolar disorder: an updated meta-analysis. J Neural Transm (Vienna). 2015 Feb,122(2):307-20.

Nishi A et al. Meta-analyses of blood homocysteine levels for gender and genetic association studies of the MTHFR C677T polymorphism in schizophrenia. Schizophr Bull. 2014 Sep,40(5):1154-63. doi: 10.1093/schbul/sbt154. Epub 2014 Feb 17.

Zhang Y et al. Association of MTHFR C677T polymorphism with schizophrenia and its effect on episodic memory and gray matter density in patients. Behav Brain Res. 2013 Apr 15,243:146-52. doi: 10.1016/j.bbr.2012.12.061. Epub 2013 Jan 12.

Lajin B et al. Association between MTHFR C677T and A1298C, and MTRR A66G polymorphisms and susceptibility to schizophrenia in a Syrian study cohort. Asian J Psychiatr. 2012 Jun,5(2):144-9. doi: 10.1016/j.ajp.2012.03.002. Epub 2012 Apr 26.

Peerbooms OL et al. Meta-analysis of MTHFR gene variants in schizophrenia, bipolar disorder and unipolar depressive disorder: evidence for a common genetic vulnerability? Brain Behav Immun. 2011 Nov,25(8):1530-43. doi: 10.1016/j.bbi.2010.12.006. Epub 2010 Dec 24.

Muntjewerff JW et al. Effects of season of birth and a common MTHFR gene variant on the risk of schizophrenia. Eur Neuropsychopharmacol. 2011 Apr,21(4):300-5. doi: 10.1016/j.euroneuro.2010.10.001. Epub 2010 Nov 19.

Feng LG et al. Association of plasma homocysteine and methylenetetrahydrofolate reductase C677T gene variant with schizophrenia: A Chinese Han population-based case-control study. Psychiatry Res. 2009 Aug 15,168(3):205-8. doi: 10.1016/j.psychres.2008.05.009. Epub 2009 Jun 28.

Roffmann JL et al. Interactive effects of COMT Val108/158Met and MTHFR C677T on executive function in schizophrenia. Am J Med Genet B Neuropsychiatr Genet. 2008 Sep 5,147B(6):990-5.

Roffman JL et al. Contribution of methylenetetrahydrofolate reductase (MTHFR) polymorphisms to negative symptoms in schizophrenia. Biol Psychiatry. 2008 Jan 1,63(1):42-8. Epub 2007 Jun 1.

Roffman JL et al. Effects of the methylenetetrahydrofolate reductase (MTHFR) C677T polymorphism on executive function in schizophrenia. Schizophr Res. 2007 May,92(1-3):181-8. Epub 2007 Mar 6.

Gilbody S et al. Methylenetetrahydrofolate reductase (MTHFR) genetic polymorphisms and psychiatric disorders: a HuGE review. Am J Epidemiol. 2007 Jan 1,165(1):1-13. Epub 2006 Oct 30.

Lee YS et al. Serum homocysteine, folate level and methylenetetrahydrofolate reductase 677, 1298 gene polymorphism in Korean schizophrenic patients. Neuroreport. 2006 May 15,17(7):743-6.

Kempisty B et al. Association of 677C>T polymorphism of methylenetetrahydrofolate reductase (MTHFR) gene with bipolar disorder and schizophrenia. Neurosci Lett. 2006 Jun 12,400(3):267-71. Epub 2006 Mar 20.

Sazci A et al. Association of the C677T and A1298C polymorphisms of methylenetetrahydrofolate reductase gene with schizophrenia: association is significant in men but not in women. Prog Neuropsychopharmacol Biol Psychiatry. 2005 Sep,29(7):1113-23.

Muntjewerff JW et al. Hyperhomocysteinemia, methylenetetrahydrofolate reductase 677TT genotype, and the risk for schizophrenia: a Dutch population based case-control study. Am J Med Genet B Neuropsychiatr Genet. 2005 May 5,135B(1):69-72.

Sazci A et al. Methylenetetrahydrofolate reductase gene polymorphisms in patients with schizophrenia. Brain Res Mol Brain Res. 2003 Sep 10,117(1):104-7.

Joober R et al. Association between the methylenetetrahydrofolate reductase 677C-->T missense mutation and schizophrenia. Mol Psychiatry. 2000 May,5(3):323-6.

Arinami T et al. Methylenetetrahydrofolate reductase variant and schizophrenia/depression. Am J Med Genet. 1997 Sep 19,74(5):526-8.

MTHFR - Methylenetetrahydrofolate reductase (NAD(P)H) (rs1801131)

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 rs1801131 polymorphism leads to a reduced enzymatic activity of methylenetetrahydrofolate reductase, and thus to an increased homocysteine level.

RES	Genotype	POP	Possible results
	A/A	57%	No increased risk of Schizophrenia
X	A/C	35%	Increased risk of Schizophrenia
	C/C	8%	Increased risk of Schizophrenia

References

Hu CY et al. Methylenetetrahydrofolate reductase (MTHFR) polymorphism susceptibility to schizophrenia and bipolar disorder: an updated meta-analysis. J Neural Transm (Vienna). 2015 Feb,122(2):307-20.

Lajin B et al. Association between MTHFR C677T and A1298C, and MTRR A66G polymorphisms and susceptibility to schizophrenia in a Syrian study cohort. Asian J Psychiatr. 2012 Jun,5(2):144-9. doi: 10.1016/j.ajp.2012.03.002. Epub 2012 Apr 26.

Zintzaras, E., 2006. C677T and A1298C methylenetetrahydrofolate reductase gene polymorphisms in schizophrenia, bipolar disorder and depression: a metaanalysis of genetic association studies. Psychiatr. Genet. 16, 105–115.

Sazci A et al. Association of the C677T and A1298C polymorphisms of methylenetetrahydrofolate reductase gene with schizophrenia: association is significant in men but not in women. Prog Neuropsychopharmacol Biol Psychiatry. 2005 Sep,29(7):1113-23.

Gilbody, S., Lewis, S., Lightfoot, T., 2007. Methylenetetrahydrofolate reductase (MTHFR) genetic polymorphisms and psychiatric disorders: a HuGE review. Am. J. Epidemiol. 165, 1–13.

BDNF - Brain derived neurotrophic factor (rs6265)

The growth factor BDNF is a protein from the group of neurotrophins and is closely related to nerve growth factors. The protein acts on various neurons in the nervous system and is involved in the growth and protection of neurons and synapses. A deficiency or excess of BDNF is associated with, amongst others, various mental disorders.

RES	Genotype	POP	Possible results
X	G/G	67%	No increased risk of Schizophrenia Average age of onset
	A/G	26%	Increased risk of Schizophrenia Lower age of onset
	A/A	7%	Increased risk of Schizophrenia Lower age of onset

References

Zakharyan R et al. Functional variants of the genes involved in neurodevelopment and susceptibility to schizophrenia in an Armenian population. Hum Immunol. 2011 Sep,72(9):746-8.

Chao HM et al. BDNF Val66Met variant and age of onset in schizophrenia. Am J Med Genet B Neuropsychiatr Genet. 2008 Jun 5,147B(4):505-6.

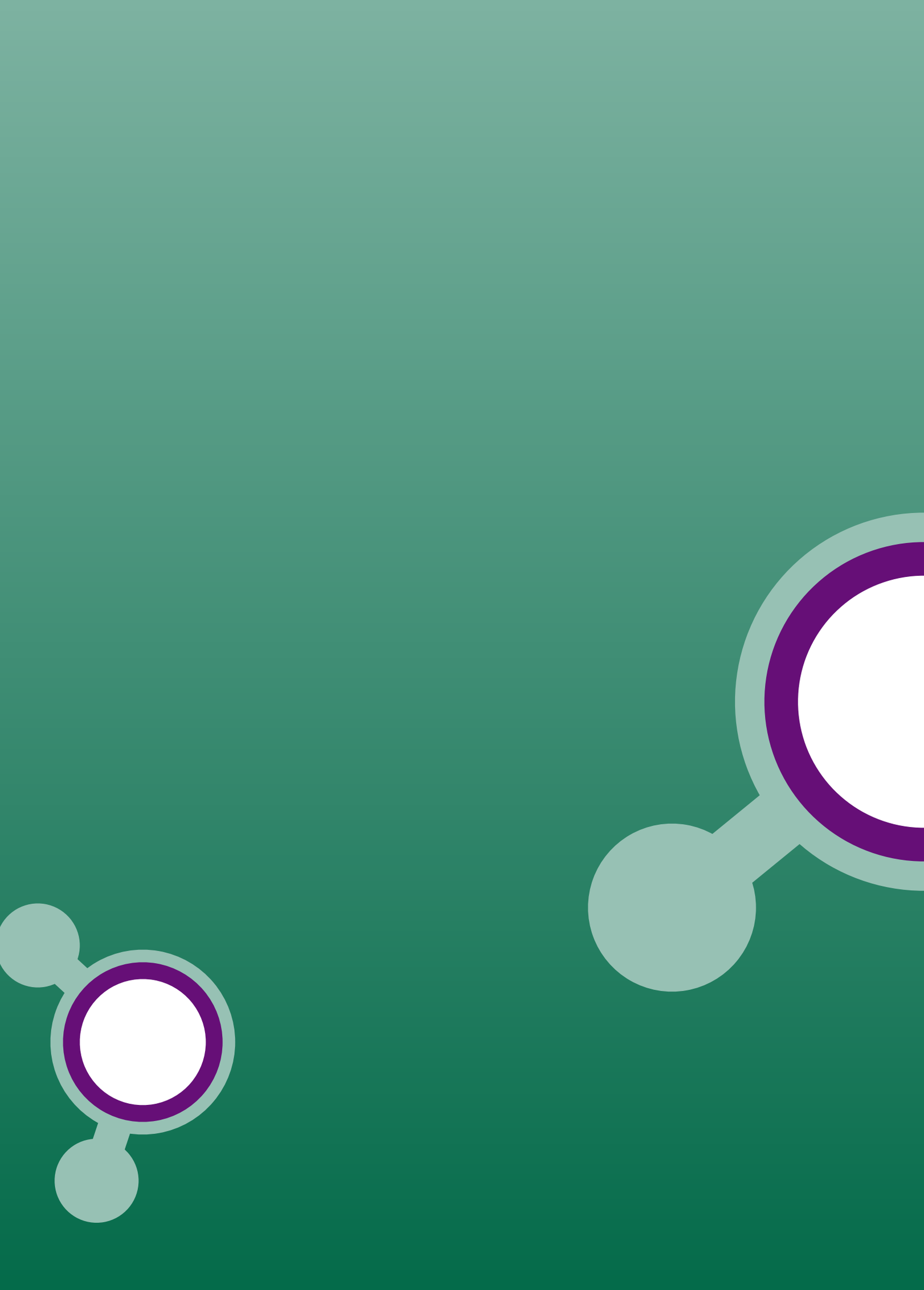
Kheirollahi M et al. Brain-Derived Neurotrophic Factor Gene Val66Met Polymorphism and Risk of Schizophrenia: A Meta-analysis of Case-Control Studies. Cell Mol Neurobiol. 2016 Jan,36(1):1-10.

Sun MM et al. BDNF Val66Met polymorphism and anxiety/depression symptoms in schizophrenia in a Chinese Han population. Psychiatr Genet. 2013 Jun,23(3):124-9.

Sun MM et al. Association study of brain-derived neurotrophic factor Val66Met polymorphism and clinical characteristics of first episode schizophrenia. Zhonghua Yi Xue Yi Chuan Xue Za Zhi. 2012 Apr,29(2):155-8.

Gratacòs M et al. Brain-derived neurotrophic factor Val66Met and psychiatric disorders: meta-analysis of case-control studies confirm association to substance-related disorders, eating disorders, and schizophrenia. Biol Psychiatry. 2007 Apr 1,61(7):911-22. Epub 2007 Jan 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.



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Not ordered

OTHERS
Not ordered

SCIENCE

ADDITIONAL INFORMATION

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In this chapter you will receive useful information

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:

- service@novogenia.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
Novogenia GmbH
Strass 19
5301 Eugendorf, Österreich

Technical details

Order number
DEMO_LOTH

Date of birth
01/01/1990

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

Report generated
22/05/2023 17:05:14

Product codes
M5SCH

Current version
V538

Ordering company
Novogenia GmbH
Strass 19
5301 Eugendorf, Österreich

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

Laboratory Director

Dr. Daniel Wallerstorfer Bsc.

Laboratory Manager

René Rohrmanstorfer, MSc.

NOTES:


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DNAhealthControl

Schizophrenia Sensor

Max Mustermann
DEMO_LOTH

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