ProMedicDNA

Depression 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

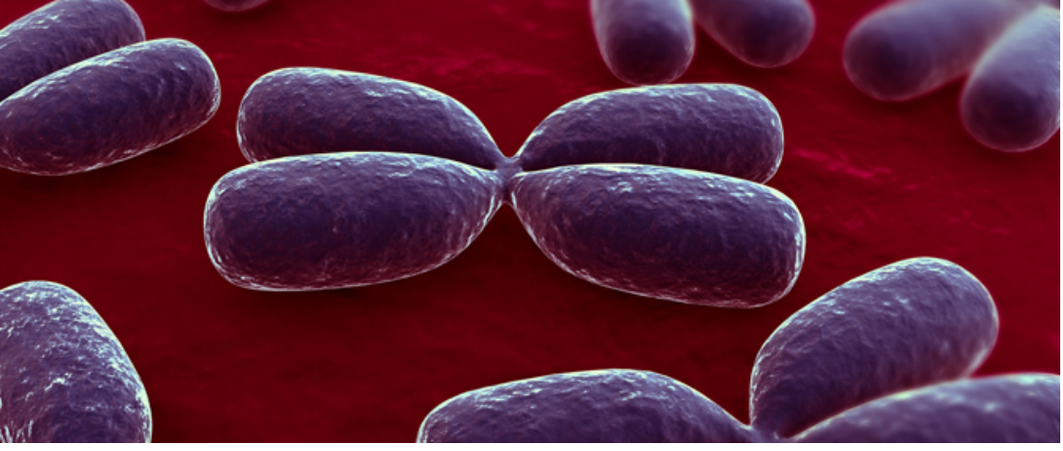
Florian Schneebauer, MSc.
Laboratory Manager

Depression Sensor

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

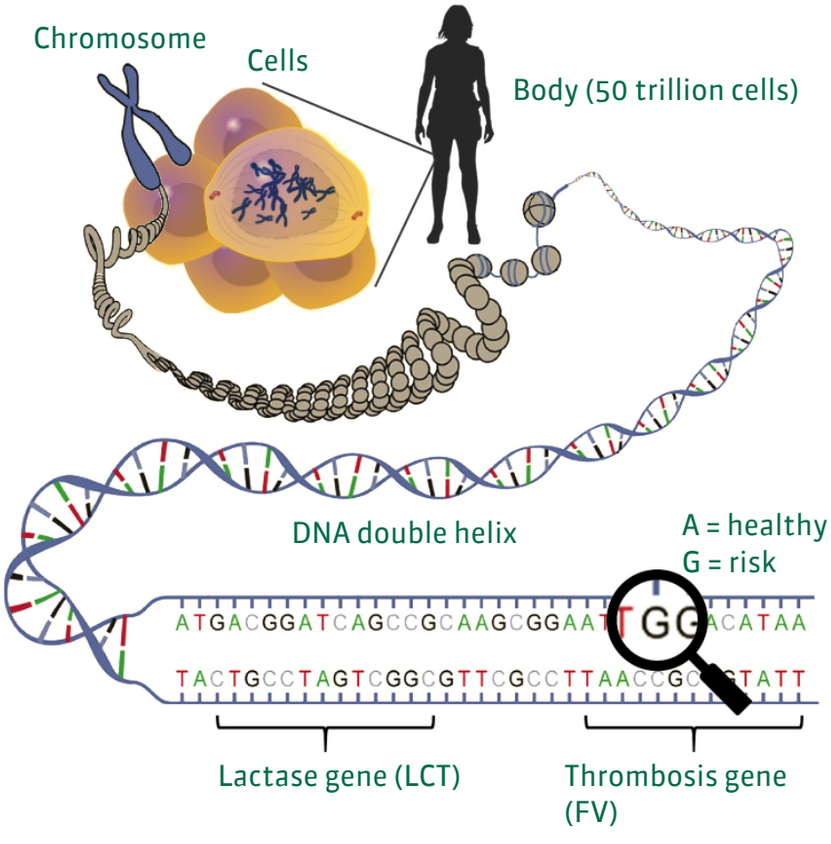
Order number:
DEMO_DS

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.

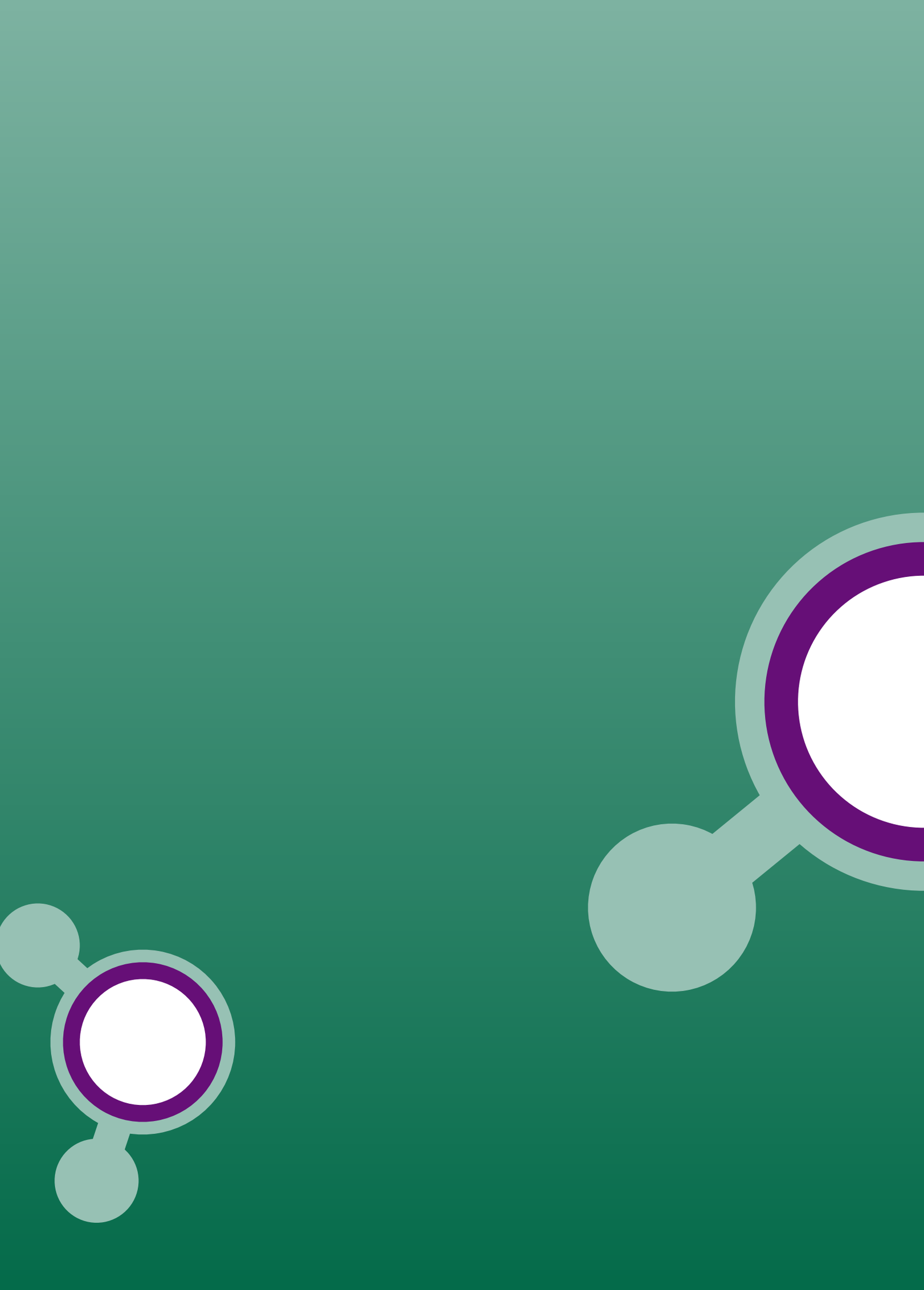
External influences can affect individual genes and disrupt their function, but the majority of our defective genes are inherited from our parents. Each embryo receives half of its genes from the father and half from the mother, resulting in a new human being with some characteristics of each parent.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

Depression Sensor



PHARMACO GENETICS
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CARDIOVASCULAR SYSTEM
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OPHTHALMOLOGY
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ODONTOLOGY
Not ordered

OTHERS
Not ordered

SCIENCE

ADDITIONAL INFORMATION

Depression Sensor

Effective prevention, risk assessment and treatment of depression

Depression (major depressive disorder)

Depression is one of the most common mental illnesses affecting around 7% of adults. It affects how people feel, think, sleep, eat, work and handle social activities and relationships. Depression comes in many different forms and may vary widely from patient to patient. Episodes can last from a few days to several years, can be triggered after pregnancy, can include psychosis and delusions or be seasonally triggered.

A number of genetic variations have been identified that increase the likelihood that a person will develop major depressive disorder. The disease is believed to be caused by a combination of genetic, environmental and psychological factors.

The following symptoms are common for depression and should be investigated by a specialist if they occur collectively and for an extended time:

- persistent feelings of sadness, anxiety or emptiness
- hopelessness or pessimism
- irritability
- feelings of guilt, worthlessness or helplessness
- loss of interest in hobbies
- fatigue
- slow movement or speech
- restlessness
- mental impairment in concentration, memory or decision-making
- difficulty falling asleep
- weight gain
- pains, headaches or digestive problems without obvious cause
- thoughts of death or suicide

Genes relevant to depression

Several genes and polymorphisms associated with a risk of developing depression 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
BDNF	rs10835210	C>A	A/C
FKBP5	rs1360780	C>T	C/C
FKBP5	rs9470080	C>T	C/C
FKBP5	rs4713916	G>A	G/G
FKBP5	rs9296158	G>A	G/G
MTHFR	rs1801133	C>T	C/C
NR3C1	rs6198	A>G	A/A

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 depression is not higher than the population average

Risk of developing depression

LOW AVERAGE RISK HIGHER

Effects for sufferers only:

- No increased risk of developing chronic depression
- No increased risk of suicide in depression
- Normal response to antidepressants

Risk of developing chronic depression

NORMAL ELEVATED

Suicide risk in depression

NORMAL ELEVATED

Response to antidepressants

NORMAL BETTER

Prevention

Since people without any genetic predisposition can also develop depression, it is generally recommended to adhere to the following lifestyle recommendations to reduce the probability of developing the disease:

- EXERCISE: regular exercise improves and stabilizes your mood
- GOALS: set realistic goals in your private life and career
- SOCIAL CONTACT: be socially active and interact with people that are close to you and present a positive influence.
- AVOID ISOLATION: avoid isolation and maintain an active social life
- TIMING: if you're feeling low, postpone important decisions such as relationships, moving or career until you feel better.

Should you ever experience symptoms of depression, do not hesitate to contact a specialist for a proper diagnosis and treatment.

Treatment

Treatment is vitally important for people suffering from depression and can greatly improve the patient's quality of life. There are a number of treatments but not every treatment is equally effective for every individual. As someone who is suffering from depression, you should be under specialist medical supervision who can choose the right treatment for you.

Medication

There are a number of antidepressants that may be used to treat depression. Unfortunately, it usually takes 2 to 4 weeks for an antidepressant to show its effect. Side effects sometimes include symptoms such as sleep, appetite and concentration problems, which sometimes negatively impacts patient compliance. Should you experience any adverse effects from the medication you're taking, discuss this with your specialist, however, give the medication time before judging if the drug helps you or not.

Today we know more about how drugs are metabolized and how certain genetic variations can influence the side effects of medication. A genetic test can help to choose the right antidepressants in the correct dosage.

Psychotherapy

Psychological counseling and talk therapy can help people with depression. Talk to your specialist about the potential benefits of psychotherapy to better deal with symptoms.

Electroconvulsive therapy

This type of treatment uses electrical currents to stimulate the brain. The therapy is done under anaesthesia with a muscle relaxant and is painless for the patient. A number of studies

have shown it to be effective in cases where acute treatment was required or where other treatment methods have not shown to be effective. Should you wish to explore this therapy, talk to your specialist about the potential benefits of this treatment.

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	✓	✓	✓
Amoxapine	✓	✓	✓
Buspirone	✓	↑	↑
Clobazam	✓	↑	↑
Clozapine	✓	✓	✓
Desvenlafaxine	✓	✓	✓
Duloxetine	✓	✓	✓
Fluvoxamine	✓	✓	✓
Imipramine	✓	✓	✓
Levetiracetam	✓	✓	✓
Mianserin	✓	✓	✓
Moclobemide	✓	✓	✓
Olanzapine	✓	✓	✓
Perphenazine	✓	✓	✓
Protriptyline	✓	✓	✓
Remoxipride	✓	✓	✓

	Effect	Breakdown	Dose
Alprazolam	✓	↑	↑
Aripiprazole	✓	↑	✓
Chlorpromazine	✓	✓	✓
Clomipramine	↑	✓	✓
Cyclobenzaprine	✓	✓	✓
Diazepam	✓	↑	↑
Escitalopram	✓	↑	✓
Haloperidol	✓	↑	✓
Isocarboxazid	✓	✓	✓
Lithium	✓	✓	✓
Minaprine	✓	✓	✓
Nefazodone	✓	↑	↑
Paliperidone	✓	✓	✓
Phenelzine	✓	↑	↑
Quetiapine	✓	↑	↑
Risperidone	✓	✓	✓

	Effect	Breakdown	Dose
Amitriptyline	✓	✓	✓
Bupropion	✓	✓	✓
Citalopram	✓	↑	✓
Clonazepam	✓	↑	↑
Desipramine	✓	✓	✓
Doxepin	✓	✓	✓
Fluoxetine	✓	✗	✗
Iloperidone	✓	✓	✓
Lamotrigine	✓	✓	✓
Maprotiline	✓	✓	✓
Mirtazapine	✓	✓	✓
Nortriptyline	✓	✓	✓
Paroxetine	✓	✓	✓
Pimozide	✓	↑	↑
Reboxetine	✓	↑	↑
Selegiline	✓	✗	✗

	Effect	Breakdown	Dose
Sertraline	✓	✓	✓
Tranylcypromine	✓	✓	✓
Valproic Acid	✓	↓	↓
Ziprasidone	✓	↑	↑

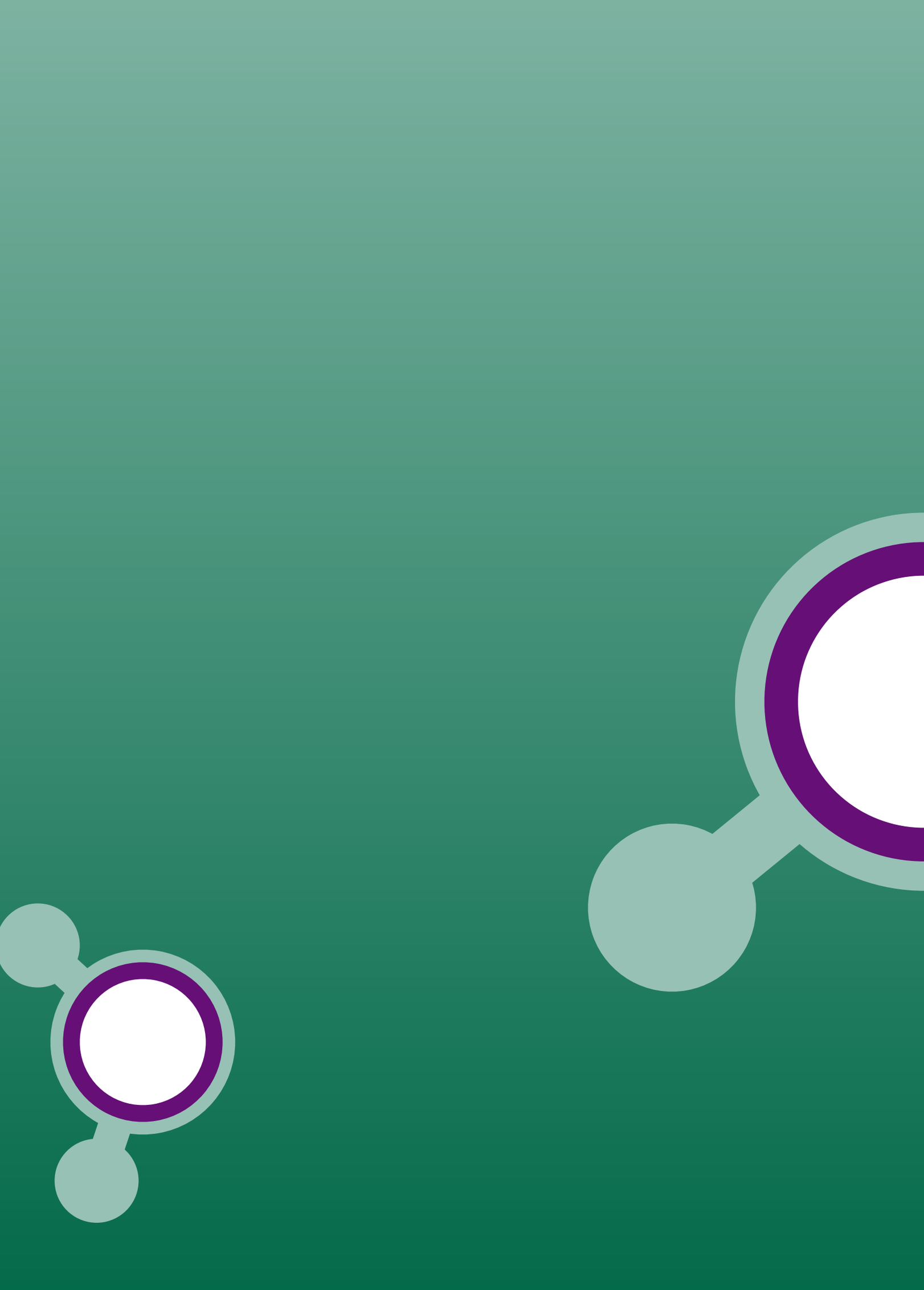
	Effect	Breakdown	Dose
Thioridazine	✓	✓	✓
Trazodone	✓	↑	↑
Venlafaxine	✓	✓	✓
Zuclopenthixol	✓	✓	✓

	Effect	Breakdown	Dose
Topiramate	✓	✓	✓
Trimipramine	✓	✓	✓
Vilazodone	✓	✓	✓

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

SCIENCE

This chapter shows the science behind the test.

Depression

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
	A/A	7%	Increased risk of depression Increased risk of chronic depression Increased suicide risk in depression
	A/G	26%	Increased risk of depression Increased risk of chronic depression Increased suicide risk in depression
X	G/G	67%	No increased risk of depression No increased risk of chronic depression No increased suicide risk in depression

References

Verhagen M et al. Meta-analysis of the BDNF Val66Met polymorphism in major depressive disorder: effects of gender and ethnicity. Mol Psychiatry. 2010 Mar,15(3):260-71.

Pei Y et al. The brain-derived neurotrophic-factor (BDNF) val66met polymorphism is associated with geriatric depression: a meta-analysis. Am J Med Genet B Neuropsychiatr Genet. 2012 Jul,159B(5):560-6.

Lee Y et al. Association between the BDNF Val66Met Polymorphism and Chronicity of Depression. Psychiatry Investig. 2013 Mar,10(1):56-61.

Su N et al. The brain-derived neurotrophic factor is associated with alcohol dependence-related depression and antidepressant response.

Dooley LN et al. Val66Met BDNF polymorphism as a vulnerability factor for inflammation-associated depressive symptoms in women with breast cancer. J Affect Disord. 2016 Jun,197:43-50.

Kang HJ et al. BDNF val66met polymorphism and depressive disorders in patients with acute coronary syndrome. J Affect Disord. 2016 Apr,194:1-8.

Gujral S et al. The BDNF Val66Met polymorphism does not moderate the effect of self-reported physical activity on depressive symptoms in midlife. Psychiatry Res. 2014 Aug 15,218(1-2):93-7.

Hosang GM et al. Stressful life events and the brain-derived neurotrophic factor gene in bipolar disorder. J Affect Disord. 2010 Sep,125(1-3):345-9.

Sarchiapone M et al. Association of polymorphism (Val66Met) of brain-derived neurotrophic factor with suicide attempts in depressed patients. Neuropsychobiology. 2008,57(3):139-45.

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Zai CC et al. Investigation of the genetic interaction between BDNF and DRD3 genes in suicidical behaviour in psychiatric disorders. World J Biol Psychiatry. 2015 Apr,16(3):171-9.

Ratta-Apha W et al. Association study of BDNF with completed suicide in the Japanese population. Psychiatry Res. 2013 Oct 30,209(3):734-6.

Pregelj P et al. The association between brain-derived neurotrophic factor polymorphism (BDNF Val66Met) and suicide. J Affect Disord. 2011 Feb,128(3):287-90.

Schenkel LC et al. The BDNF Val66Met polymorphism is an independent risk factor for high lethality in suicide attempts of depressed patients. Prog Neuropsychopharmacol Biol Psychiatry. 2010 Aug 16,34(6):940-4.

Kim B et al. Brain-derived neurotrophic factor Val/Met polymorphism and bipolar disorder. Association of the Met allele with suicidal behavior of bipolar patients. Neuropsychobiology. 2008,58(2):97-103.

Sarchiapone M et al. Association of polymorphism (Val66Met) of brain-derived neurotrophic factor with suicide attempts in depressed patients. Neuropsychobiology. 2008,57(3):139-45.

Iga J et al. The Val66Met polymorphism of the brain-derived neurotrophic factor gene is associated with psychotic feature and suicidal behavior in Japanese major depressive patients. Am J Med Genet B Neuropsychiatr Genet. 2007 Dec 5,144B(8):1003-6.

BDNF - Brain derived neurotrophic factor (rs10835210)

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
	A/A	8%	Increased risk of depression
X	A/C	33%	Increased risk of depression
	C/C	59%	No increased risk of depression

References

Meng XF et al. Association between polymorphism of BDNF and internalizing disorders. Zhonghua Liu Xing Bing Xue Za Zhi. 2009 Dec,30(12):1265-8.

Meng X et al. Susceptibility genes, social environmental risk factors and their interactions in internalizing disorders among mainland Chinese undergraduates. J Affect Disord. 2011 Jul,132(1-2):254-9.

FKBP5 - FK506 binding protein 5 (rs1360780)

FK506 binding protein 5 plays a role in immunoregulation and basic cellular processes involving protein folding and trafficking. Genetic studies have identified a role for FK506 in post-traumatic stress disorder, depression and anxiety.

RES	Genotype	POP	Possible results
X	C/C	45%	No increased risk of depression No increased suicide risk Normal response to antidepressants
	C/T	44%	Increased risk of depression Increased suicide risk Better response to antidepressants
	T/T	1%	Increased risk of depression Increased suicide risk Better response to antidepressants

References

Szczepankiewicz A et al. FKBP5 polymorphism is associated with major depression but not with bipolar disorder. J Affect Disord. 2014 Aug,164:33-7.

Menke A et al. Genetic variation in FKBP5 associated with the extent of stress hormone dysregulation in major depression. Genes Brain Behav. 2013 Apr,12(3):289-96.

Shinozaki G et al. Relationship between FKBP5 polymorphisms and depression symptoms among kidney transplant recipients. Depress Anxiety. 2011 Dec 21,28(12):1111-8.

Zimmermann P et al. Interaction of FKBP5 gene variants and adverse life events in predicting depression onset: results from a 10-year prospective community study. Am J Psychiatry. 2011 Oct,168(10):1107-16.

Tatro ET et al. Correlation of major depressive disorder symptoms with FKBP5 but not FKBP4 expression in human immunodeficiency virus-infected individuals. J Neurovirol. 2010 Oct,16(5):399-404.

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Kirchheiner J et al. Genetic variants in FKBP5 affecting response to antidepressant drug treatment. Pharmacogenomics. 2008 Jul,9(7):841-6.

Lekman M et al. The FKBP5-gene in depression and treatment response--an association study in the Sequenced Treatment Alternatives to Relieve Depression (STAR*D) Cohort. Biol Psychiatry. 2008 Jun 15,63(12):1103-10.

Fudalej S et al. Association between FKBP5 Functional Polymorphisms and Completed Suicide. Neuropsychobiology. 2015,72(2):126-31.

Minelli A et al. Role of allelic variants of FK506-binding protein 51 (FKBP5) gene in the development of anxiety disorders. Depress Anxiety. 2013 Dec,30(12):1170-6.

Roy A et al. Interaction of FKBP5, a Stress-Related Gene, with Childhood Trauma Increases the Risk for Attempting Suicide. Neuropsychopharmacology. 2010 Jul, 35(8): 1674–1683.

Binder EB et al. Polymorphisms in FKBP5 are associated with increased recurrence of depressive episodes and rapid response to antidepressant treatment. Nat Genet. 2004 Dec,36(12):1319-25. Epub 2004 Nov 21.

Willour VL et al. Family-based association of FKBP5 in bipolar disorder. Mol Psychiatry. 2009 Mar,14(3):261-8.

FKBP5 - FK506 binding protein 5 (rs9470080)

FK506 binding protein 5 plays a role in immunoregulation and basic cellular processes involving protein folding and trafficking. Genetic studies have identified a role for FK506 in post-traumatic stress disorder, depression and anxiety.

RES	Genotype	POP	Possible results
X	C/C	40%	No increased risk of depression
	C/T	46%	Increased risk of depression
	T/T	14%	Increased risk of depression

References

Szczepankiewicz A et al. FKBP5 polymorphism is associated with major depression but not with bipolar disorder. J Affect Disord. 2014 Aug,164:33-7.

Kang JI et al. FKBP5 polymorphisms as vulnerability to anxiety and depression in patients with advanced gastric cancer: a controlled and prospective study. Psychoneuroendocrinology. 2012 Sep,37(9):1569-76.

Shinozaki G et al. Relationship between FKBP5 polymorphisms and depression symptoms among kidney transplant recipients. Depress Anxiety. 2011 Dec 21,28(12):1111-8.

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Velders FP et al. Genetics of cortisol secretion and depressive symptoms: a candidate gene and genome wide association approach. Psychoneuroendocrinology. 2011 Aug,36(7):1053-61.

FKBP5 - FK506 binding protein 5 (rs4713916)

FK506 binding protein 5 plays a role in immunoregulation and basic cellular processes involving protein folding and trafficking. Genetic studies have identified a role for FK506 in post-traumatic stress disorder, depression and anxiety.

RES	Genotype	POP	Possible results
X	G/G	61%	No increased risk of depression
	A/G	33%	Increased risk of depression Better response to antidepressants
	A/A	6%	Increased risk of depression Better response to antidepressants

References

Szczepankiewicz A et al. FKBP5 polymorphism is associated with major depression but not with bipolar disorder. J Affect Disord. 2014 Aug,164:33-7.

Zimmermann P et al. Interaction of FKBP5 gene variants and adverse life events in predicting depression onset: results from a 10-year prospective community study. Am J Psychiatry. 2011 Oct,168(10):1107-16.

Lekman M et al. The FKBP5-gene in depression and treatment response--an association study in the Sequenced Treatment Alternatives to Relieve Depression (STAR*D) Cohort. Biol Psychiatry. 2008 Jun 15,63(12):1103-10.

Collip D et al. FKBP5 as a possible moderator of the psychosis-inducing effects of childhood trauma. Br J Psychiatry. 2013 Apr,202(4):261-8.

Zou YF et al. Meta-analysis of FKBP5 gene polymorphisms association with treatment response in patients with mood disorders. Neurosci Lett. 2010 Oct 22,484(1):56-61.

FKBP5 - FK506 binding protein 5 (rs9296158)

FK506 binding protein 5 plays a role in immunoregulation and basic cellular processes involving protein folding and trafficking. Genetic studies have identified a role for FK506 in post-traumatic stress disorder, depression and anxiety.

RES	Genotype	POP	Possible results
X	G/G	41%	No increased risk of depression
	A/G	46%	Increased risk of depression
	A/A	13%	Increased risk of depression

References

Szczepankiewicz A et al. FKBP5 polymorphism is associated with major depression but not with bipolar disorder. J Affect Disord. 2014 Aug,164:33-7.

Kang JI et al. FKBP5 polymorphisms as vulnerability to anxiety and depression in patients with advanced gastric cancer: a controlled and prospective study. Psychoneuroendocrinology. 2012 Sep,37(9):1569-76.

Shinozaki G et al. Relationship between FKBP5 polymorphisms and depression symptoms among kidney transplant recipients. Depress Anxiety. 2011 Dec 21,28(12):1111-8.

Zimmermann P et al. Interaction of FKBP5 gene variants and adverse life events in predicting depression onset: results from a 10-year prospective community study. Am J Psychiatry. 2011 Oct,168(10):1107-16.

Kohrt BA et al. Cross-cultural gene- environment interactions in depression, post-traumatic stress disorder, and the cortisol awakening response: FKBP5 polymorphisms and childhood trauma in South Asia. Int Rev Psychiatry. 2015,27(3):180-96.

Collip D et al. FKBP5 as a possible moderator of the psychosis-inducing effects of childhood trauma. Br J Psychiatry. 2013 Apr,202(4):261-8.

Roy A et al. Interaction of FKBP5, a Stress-Related Gene, with Childhood Trauma Increases the Risk for Attempting Suicide. Neuropsychopharmacology. 2010 Jul, 35(8): 1674–1683.

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%	Increased risk of depression
	C/T	33%	Increased risk of depression
	T/T	8%	No increased risk of depression

References

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.

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.

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

NR3C1 - nuclear receptor subfamily 3 group C member 1 (rs6198)

The glucocorticoid receptor (GR or GCR) also known as NR3C1 (nuclear receptor subfamily 3, group C, member 1) is the receptor to which cortisol and other glucocorticoids bind. It can function both as a transcription factor that binds to glucocorticoid response elements in the promoters of glucocorticoid responsive genes to activate their transcription, and as a regulator of other transcription factors.

RES	Genotype	POP	Possible results
X	A/A	85%	No increased risk of depression
	A/G	13%	Increased risk of depression
	G/G	2%	Increased risk of depression

References

Kumsta R et al. Sex specific associations between common glucocorticoid receptor gene variants and hypothalamus-pituitary-adrenal axis responses to psychosocial stress. Biol Psychiatry. 2007 Oct 15,62(8):863-9. Epub 2007 Aug 23.

Szczepankiewicz A et al. Glucocorticoid receptor polymorphism is associated with major depression and predominance of depression in the course of bipolar disorder. J Affect Disord. 2011 Nov,134(1-3):138-44.

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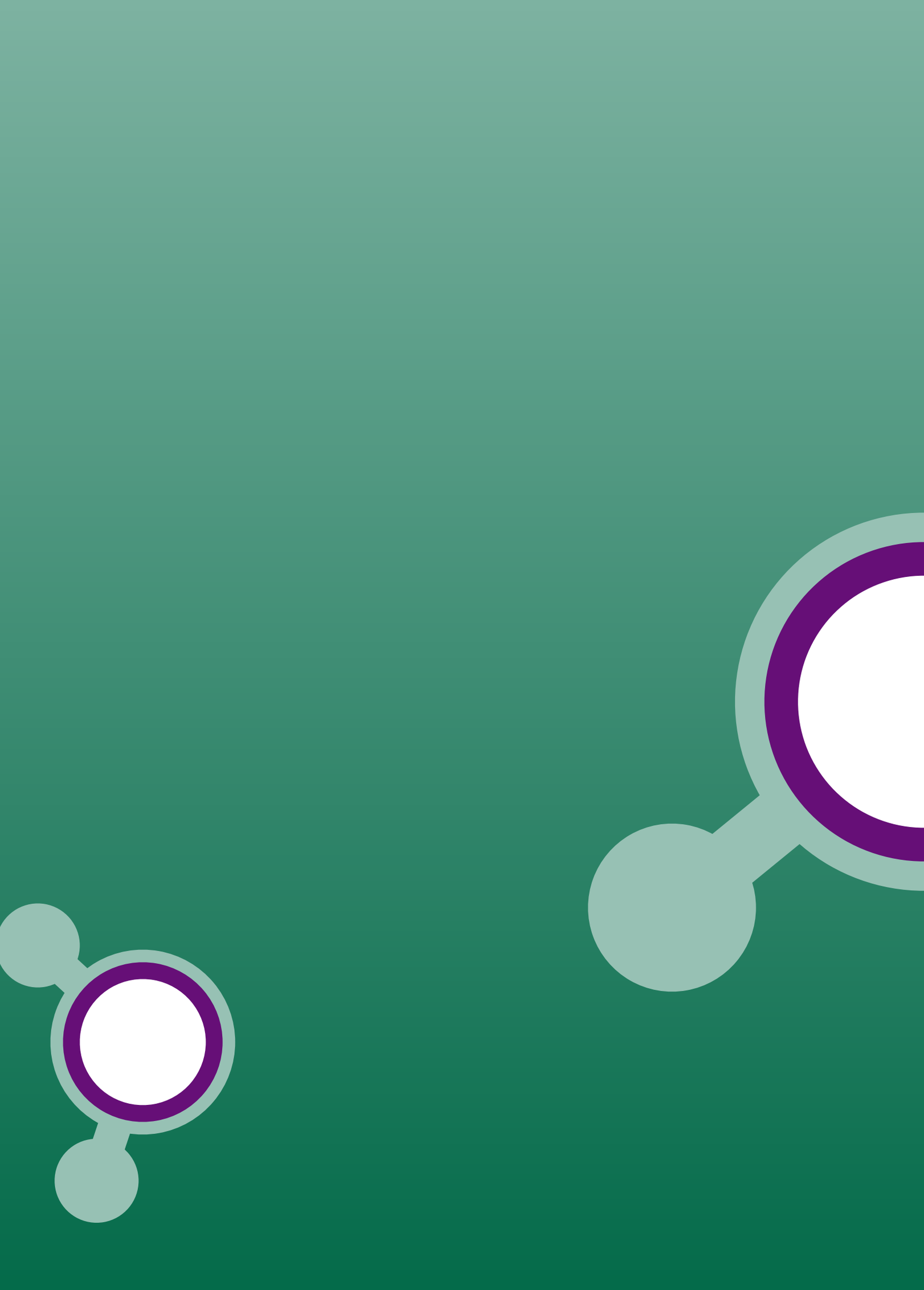
LEGEND: RES = your personal analysis result (marked with an X), GENOTYPE = different variations of the gene (called alleles),
POP = percent of the general population that have this genetic result,
POSSIBLE RESULTS = influence of the genetic variation.



PHARMACO GENETICS
Not ordered

ONCOLOGY
Not ordered

CARDIOVASCULAR SYSTEM
Not ordered

NEUROLOGY

METABOLISM
Not ordered

MOVEMENT
Not ordered

DIGESTION
Not ordered

OPHTHALMOLOGY
Not ordered

ODONTOLOGY
Not ordered

OTHERS
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

qualityaustria
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
ProGenom GmbH
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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 16:33:30

Product codes
M5DEP

Current version
V538

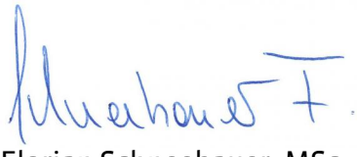
Ordering company
ProGenom GmbH
Riedstrasse 1
6343 Rotkreuz
SWITZERLAND

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

Depression Sensor

Jane Doe
DEMO_DS

0000000000000

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