DNAhealthControl

ADHD 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

ADHD 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.

ADHD Sensor

Risk assessment, prevention and better treatment

Attention deficit-hyperactivity disorder

Children typically develop the symptoms before the age of seven and there are three different subtypes of the disorder, one of which predominantly causes inattentiveness, one which causes hyperactive impulses and one which consists of a combination of the two [Reference: 2]. ADHD usually affects school aged children and results in restlessness, impulsiveness and lack of focus which has a significant negative impact on their ability to learn. It is the most commonly diagnosed psychiatric disorder in children [3, 4], affecting around 5% of children globally and it is estimated that around 5% of American adults live and cope with ADHD [Reference: 1].

Signs and symptoms

The symptoms of ADHD are difficult to identify, as it is sometimes difficult to draw the line between normal levels of inattention and hyperactivity and clinically significant levels pointing to the presence of ADHD.

The US National Institute of mental health defines the symptoms of the various types follows:

The predominantly inattentive type [5]:

- Be easily distracted, miss details, forget things, and frequently switch from one activity to another
- Have difficulty maintaining focus on one task
- Become bored with a task after only a few minutes, unless doing something enjoyable
- Not seem to listen when spoken to
- Daydream, become easily confused, and move slowly
- Have difficulty processing information as quickly and accurately as others
- Struggle to follow instructions.
- Have difficulty focusing attention on organizing and completing a task or learning something new or trouble completing or turning in homework assignments, often losing things (e.g., pencils, toys, assignments) needed to complete tasks or activities

The predominantly hyperactive impulsive type [6]:

- Fidget and squirm in their seats
- Talk nonstop
- Dash around, touching or playing with anything and everything in sight
- Have trouble sitting still during dinner, school, and story time
- Be constantly in motion
- Have difficulty doing quiet tasks or activities
- and also these manifestations primarily of impulsivity:[26]
- Be very impatient
- Blurt out inappropriate comments, show their emotions without restraint, and act without regard for consequences
- Have difficulty waiting for things they want or waiting their turns in games

The genetics

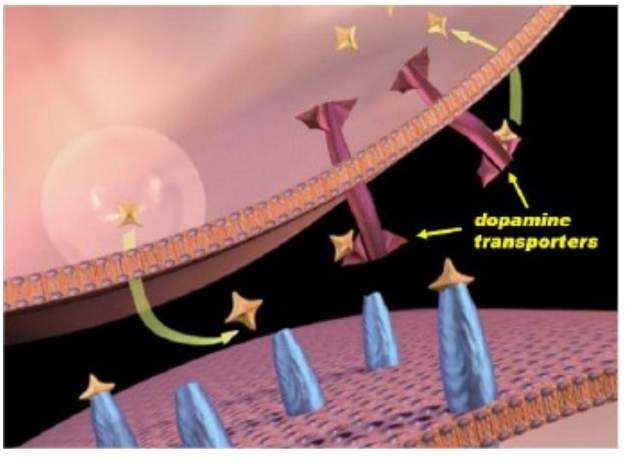
ADHD is a disorder with a strong genetic impact, as around 75% of all cases are suspected to be at least partly caused by genetic variants [6]. Certain drugs used to treat ADHD have been developed and have shown great success, but our genes play an important part of how these drugs are metabolised and broken down by our body. It is commonly known, that drugs only work in around 40% of the population as desired. Some people show no effect of the drug at all, or suffer from serious side-effects, which make adverse drug reactions the fifth most common cause of death in the developed world [7-9].

Through advanced genetic analysis it is now possible to determine the genetic predisposition of developing ADHD in children, as well as choosing the most suitable drugs for the best possible treatment success.

ADHD gene 1 (Dopamine transp. SLC6A3, Dat1)

When a nerve impulse moves from one brain cell to the next, the first brain cell releases a chemical (dopamine) into the space between the two different cells (the synapses). The second brain cell recognizes this chemical and creates a nerve impulse that it then passes on to the next cell in the same chemical releasing way. This way an impulse can jump from one cell to the next, thereby passing through the brain and controlling various aspects of our mind and body.

After one cell has released the chemical for the other cell, it needs to absorb the chemical again in order to be ready for the next impulse. This is exactly what the ADHD gene 1 does. It absorbs the chemical and moves it back to the cell for the next nerve impulse [10]. Genetic variants can modify the effectiveness, by which the ADHD gene can perform this function. Any reduction in function can lead to a number of different neurological roblems such as ADHD [11-16, 34-42].



ADHD gene 2 (COMT Val158Met)

One of the chemicals that brain cells release to signal to the surrounding brain cells to transmit a brain impulse is called dopamine. After dopamine has been released and taken up again to be

reused,it eventually needs to be broken down and removed from the cells. The ADHD gene 2 (COMT) facilitates this breakdown and removal of dopamine. One very common genetic variant in this gene can cause this function of dopamine removal to be around 4 times more efficient [18], thereby leading to much lower dopamine levels than the other genetic variant, that breaks down dopamine significantly slower [19].

ADHD gene 3 (OPRM1)

The opoid receptor, mu1 (ADHD gene 3) produces a receptor in certain brain cells that is known to modify the risk of addiction to alcohol, nicotine or illegal drugs [27]. People carrying certain genetic variants are at an increased risk of dependence on these substances and should take preventive measures to minimize contact to such substances.

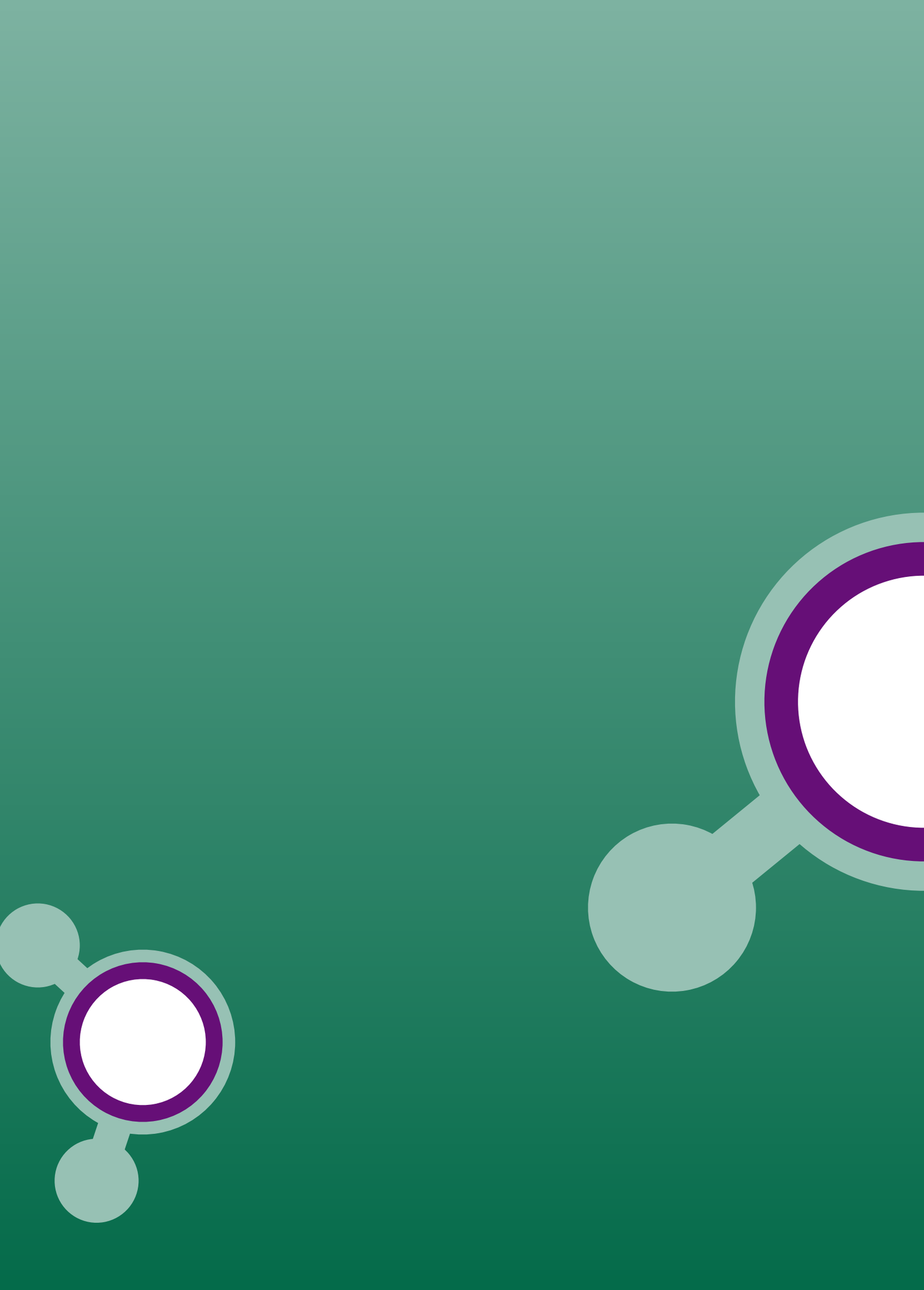
ADHD gene 4 (ADRA2A rs1800544)

The ADHD gene 4 is involved in the release of the chemicals (neurotransmitters) that enable one brain cell to signal to another brain cell and thereby pass on a nerve impulse. Certain genetic variants have different effects on the activity of this gene and scientific research has shown, that certain genetic types responded very well to Methylphenidate (eg. Ritalin) as a possible treatment option for ADHD [28].

ADHD gene 5 (CYP2D6 – 5 common variants)

ADHD gene 5 creates a liver enzyme that is responsible for the breakdown and elimination of more than 25% of currently prescribed drugs from the body. If this gene is defective, the drugs (including amphetamines, Atomexetine and Methoxyamphetamine) may be eliminated from the body at a slower rate and cause severe side effects if taken over long periods. Drugs that are metabolised and broken down by this enzyme should be taken at significantly lower dosages if no alternative drugs can be used in order to prevent side-effects [33].

There are 5 common genetic defects in this gene, all of which disrupt the function and cause the relevant ADHD medication levels in the blood to be abnormally high. Should any of these genetic defects be present (which is the case in more than 10% of the population), the dosages of amphetamines, Atomexetine and Methoxyamphetamine should be significantly reduced [31, 33].



RESULT

This chapter will give you information on your laboratory results

Genetic Risk

Genetics play a role in the development of ADHD in approximately 75% of all cases. This strong genetic component makes people with unfortunate genetic variants more likely to develop the disorder and helps with diagnosing suspected cases of ADHD.

Still, 25% of all ADHD cases do not carry genetic variants that predispose them to the disorder [6] so this section should be seen as providing possible confirmation for suspected cases that already show a number of symptoms. It should not be seen as a definite diagnosis of children without any symptoms of ADHD.

The relevant genetic variants in the ADHD genes 1 and 2

GENE	POLYMORPH	GENOTYPE
DAT1 - rs27072	T>C	C/C
DAT1 - 40 bp repeat	VNTR	5R
COMT1 - rs4680	G>A	A/G

A disease causing genetic variant has been identified. The risk of ADHD is therefore significantly increased [11-16].

NORMAL RISK HIGH RISK

In addition, one genetic variant (COMT) causes the symptoms to typically be more severe than in carriers of other genetic variants [17].

MILD SEVERE

Medical consequence

If the patient also exhibits typical symptoms leading to a suspected diagnosis of ADHD, this analysis further confirms the diagnosis [11-17].

Other Risks

The combination of ADHD and certain genetic variants can also increase the risk of other psychological disorders or symptoms.

By analysing the relevant genes, we can predict the risk of other complications from arising and enabling you and your clinician to counteract appropriately.

The relevant genetic variants in the ADHD genes 2 and 3

GENE	POLYMORPH	GENOTYPE
COMT - rs4680	G>A	A/G
OPRM1 - rs1799971	A>G	A/A

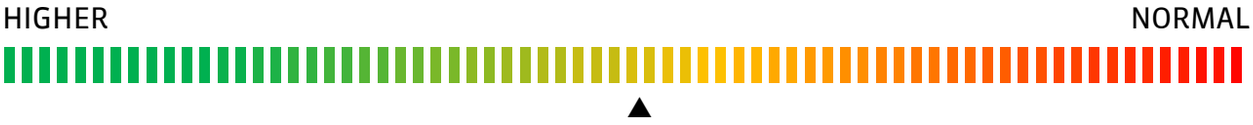
The genetic variant in the COMT gene predisposes the patient to the hyperactive impulsive type of ADHD instead of the inattentive type [20].

INATTENTIVE TYPE — HYPERACTIVE TYPE

Research has also shown that adolescent use of the illegal drug cannabis in combination with this genetic variant leads to an increased risk of schizophrenia and psychoses in later life [21].

NORMAL RISK — HIGHER (11x)

People carrying this genetic variant typically have a higher feeling of reward and also experience pleasant events approximately twice as pleasant as other genetic types do [22].

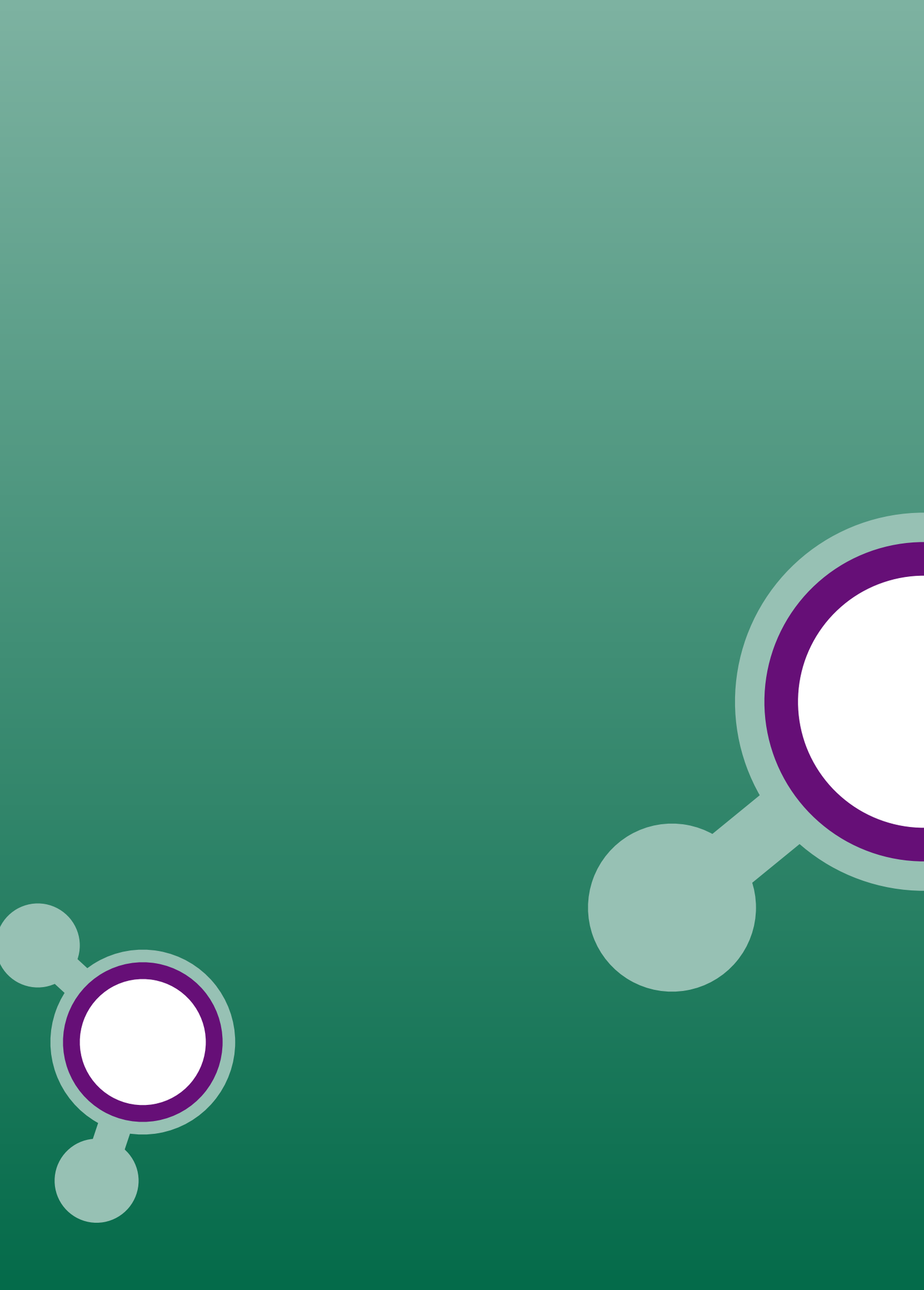


Unfortunately the genetic variant in the COMT gene interacts with ADHD and makes severe antisocial and/or aggressive behavior 3 times as likely [23-26].

The genetic variant in the OPRM1 gene does not increase the risk of substance addiction [27].

Medical consequence

As the hyperactive and impulsive type of ADHD is more likely (as opposed to the inattentive type), this can support a suspected diagnosis as certain hyperactivity traits are more likely to arise and can be correctly recognized.
The higher risk of aggressive and antisocial behavior is due to reduced social understanding and can be counteracted with a good upringing. Studies have shown that this link between this genetic variant and antisocial behavior is significantly more prominent in families with lower socio economic status.



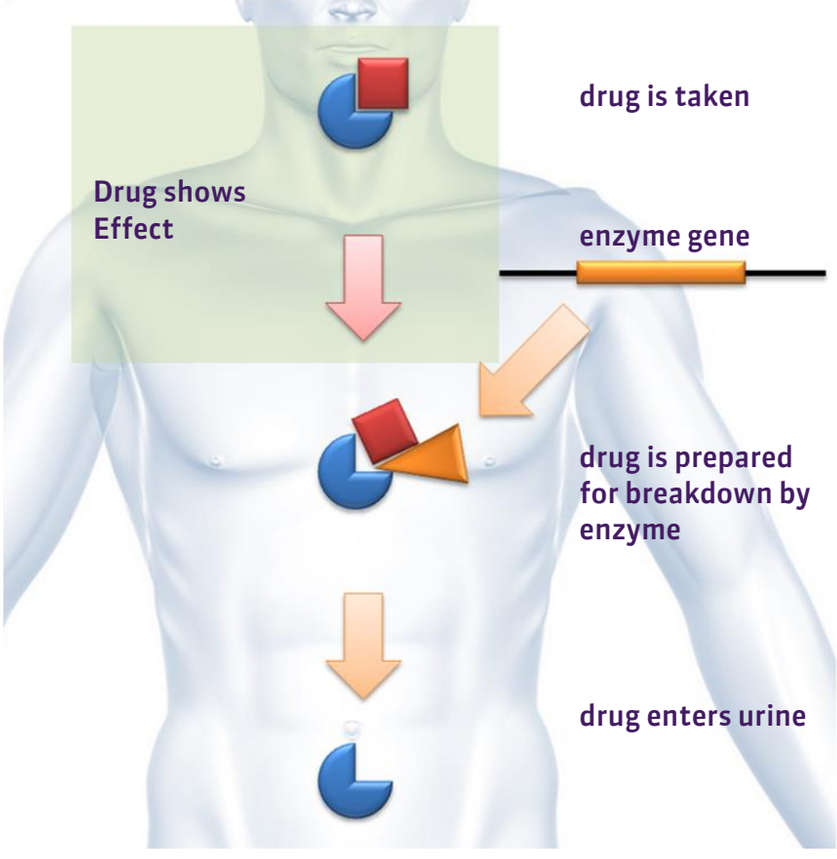
PHARMACOGENETICS

Avoiding side effects from medication and improving the outcome

How drugs work in the human body

Every person reacts differently to drugs/medications. Some people benefit significantly from a particular medication, while others experience adverse effects with symptoms that can range from mild to fatal. According to estimates, approximately 7% of patients suffer from severe adverse reactions and about 0.4% suffer fatal consequences. Adverse reactions to drugs are the fifth most frequent cause of death in the developed world. In most cases, these reactions are determined by inherited genetic variations or certain drug interactions.

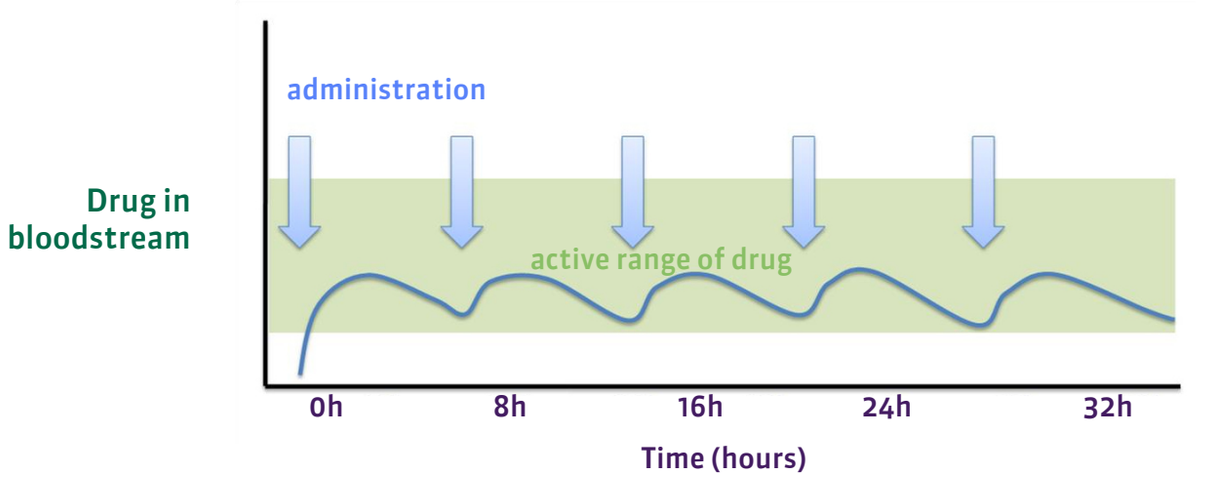
The drug pathways in the body



When a drug is administered- whether orally, intravenously or via any other route- it first enters the bloodstream. The blood transports the drug to the target organ where it will elicit the required response. However, the drug is recognised as foreign by certain enzymes which proceed to break it down and remove it from the bloodstream. This causes most drugs to lose their effect. The deactivated drug is then filtered out of the bloodstream with the help of the kidneys and finally excreted in the urine.

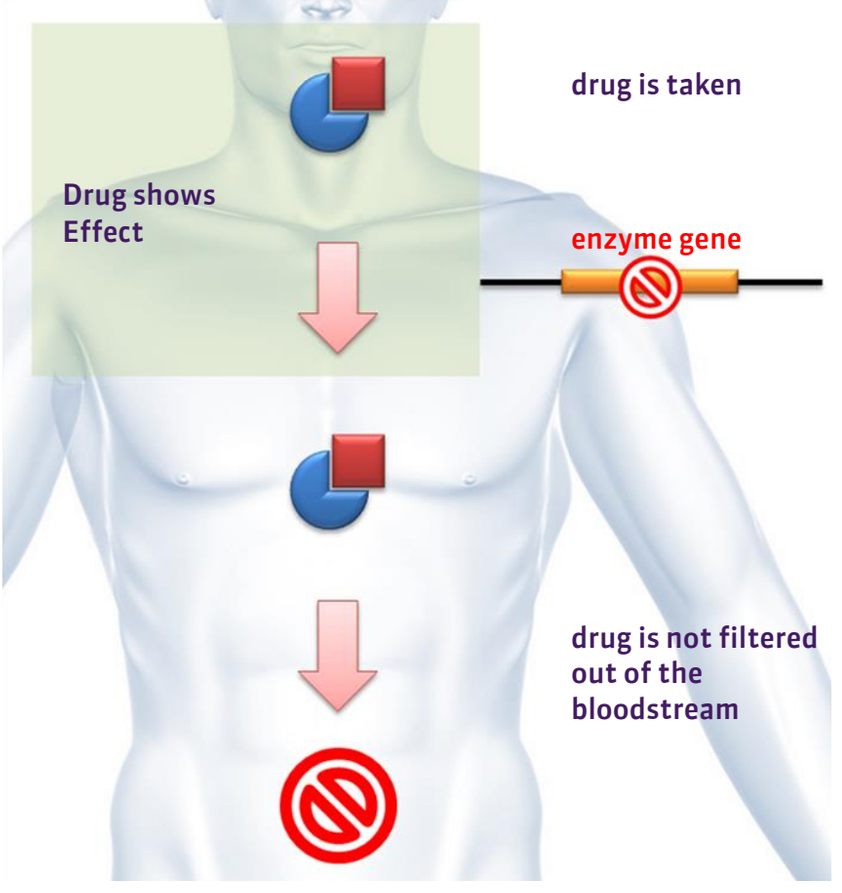
Long-term Drug Treatment

Due to the fact that many drugs work over an extended period, they need to be taken at regular intervals to ensure that the concentration of the drug in the bloodstream is maintained in the correct range.



This is how the drug always remains at the right concentration and shows its intended effect.

Genetic defects inhibit the breakdown of the drug

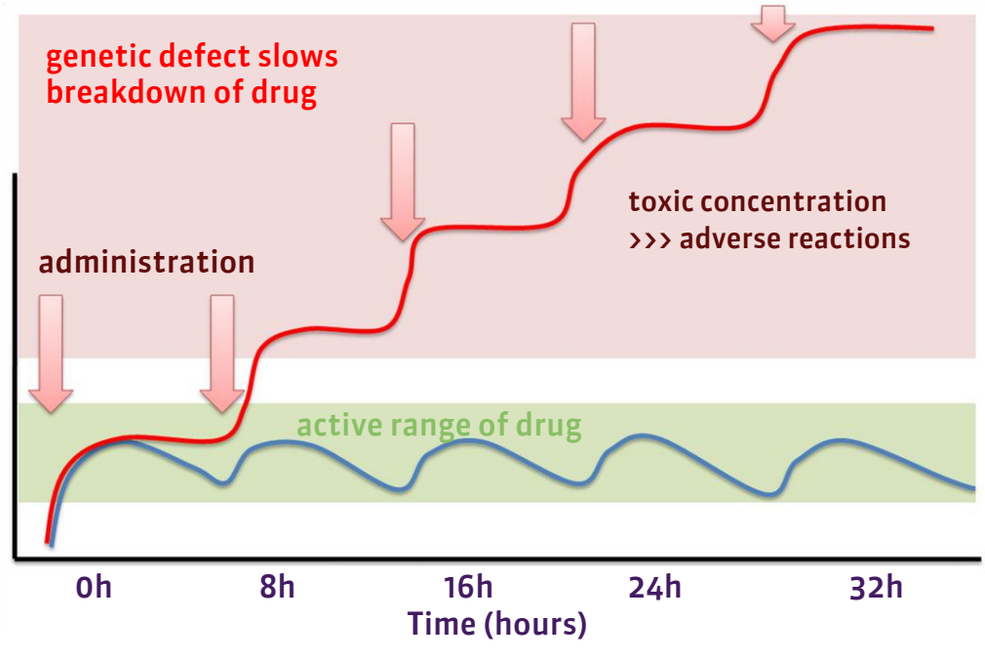


Unfortunately, many people carry a defect in one of the enzyme-producing genes that are crucial in this process.

The drug still enters the blood circulation and has its effect, but the specific enzymes do not break it down and the drug remains in the body for a significantly longer time. This is only a minor problem after a single dose, but when a person takes warfarin three times a day, for example, the level of warfarin in the blood gradually increases until it causes toxic side effects.

The complications of regular administration of a drug when there is a genetic defect

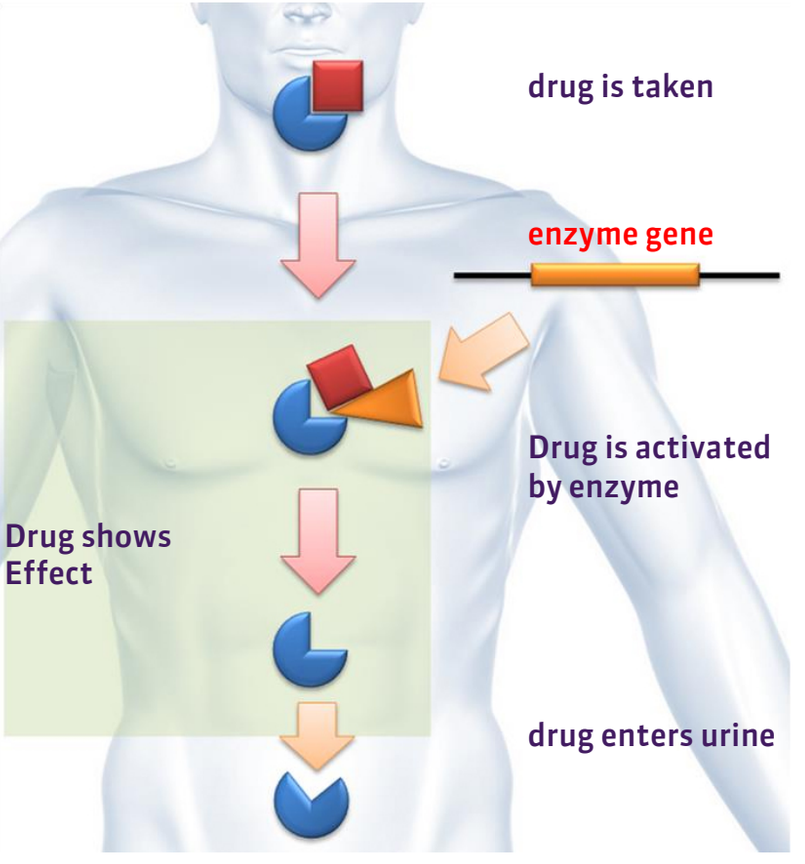
In the case of blood thinners, drug action is at the optimum level at the beginning of therapy but the drug concentration increases subsequently with every dose until it reaches the point of causing uncontrolled bleeding.

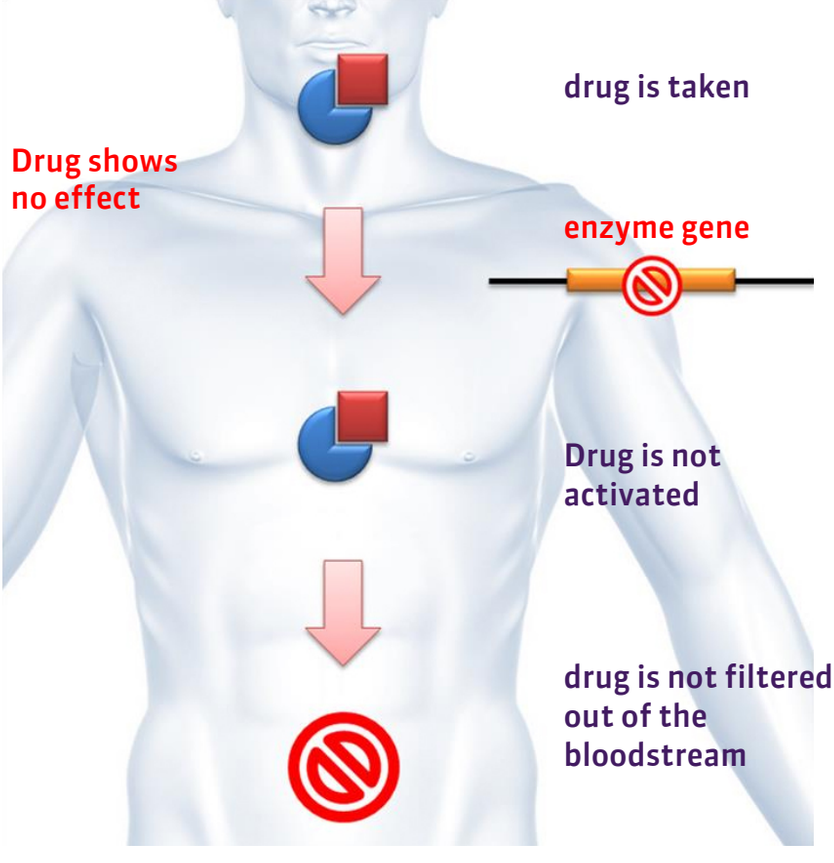


This means that the 20% of the population that carry a genetic defect need a significantly lower dose of warfarin because the usual dose could lead to serious adverse reactions.

Prodrugs: the precursors of active drugs

Some drugs are taken in an inactive form and are only activated by the enzymes of the body.These are called prodrugs. Examples for this kind of drugs include the cancer-prevention drug tamoxifen and the painkiller codeine.
A prodrug enters the bloodstream in its inactive form. Enzymes in the blood transform it into its active form, and then it takes effect. For example, the painkiller codeine (prodrug) is transformed into morphine (active form), which then relieves pain.





In some people, the enzyme that converts a specific prodrug into active drug does not function, so that the drug never has an effect on the body, other than potential side effects.
In the case of codeine, there is no pain relief after administration and an alternative drug needs to be chosen.
In case of tamoxifen, a drug that prevents breast cancer, the drug's inefficacy will only be discovered if cancer develops.

Pharmacogenetic genes

The following genes and polymorphisms have an impact on the breakdown and effect of various drugs. Your genetic analysis found the following:

CYP1A2

rs NCBI	POLYMORPH	GENOTYPE
rs2069514	-3860G>A	G/G
rs762551	-163C>A	C/C
GENOTYPE	**METABOLIZER**	**ACTIVITY**
***1/*1**	**EXTENSIVE**	**NORMAL**

CYP2B6

rs NCBI	POLYMORPH	GENOTYPE
rs28399499	983T>C	T/T
rs34223104	-82T>C	T/T
rs3745274	516G>T	G/G
GENOTYPE	**METABOLIZER**	**ACTIVITY**
***1/*1**	**EXTENSIVE**	**NORMAL**

CYP2C19

rs NCBI	POLYMORPH	GENOTYPE
rs4244285	681G>A	G/G
rs4986893	636G>A	G/G
rs28399504	1A>G	A/A
rs56337013	1297C>T	C/C
rs72552267	395G>A	G/G
rs72558186	19294T>A	T/T
rs41291556	358T>C	T/T
rs17884712	431G>A	G/G
rs12248560	-806C>T	C/C
rs6413438	19153C>T	C/C
GENOTYPE	**METABOLIZER**	**ACTIVITY**
***1/*1**	**EXTENSIVE**	**NORMAL**

CYP2C9

rs NCBI	POLYMORPH	GENOTYPE
rs1799853	430C>T	C/C
rs1057910	1075A>C	A/A
rs28371686	1080C>G	C/C
rs9332131	818delA	A/A
rs7900194	449G>A	G/G
rs7900194	449G>T	G/G
rs28371685	1003C>T	T/T
rs56165452	1076T>C	T/T
GENOTYPE	**METABOLIZER**	**ACTIVITY**
***11/*11**	**POOR**	**NONE**

CYP2D6

rs NCBI	POLYMORPH	GENOTYPE
Dup/Del	xN	x2
rs1080985	-1584C>G	C/C
rs1065852	100C>T	C/C
rs774671100	del>A	del/del
rs201377835	883G>C	C/C
rs28371706	1023C>T	C/C
rs5030655	1707delT	T/T
rs5030865	1758G>T	C/C
rs5030865	1758G>A	C/C
rs3892097	1846G>A	G/G
rs35742686	2549delA	A/A
rs5030656	2615_2617delAAG	T/T
rs16947	2850C>T	G/G
rs5030867	2935A>C	A/A
rs28371725	2988G>A	G/G
rs59421388	3183G>A	C/C
rs1135840	4180G>C	G/G
rs5030862	124G>A	C/C
GENOTYPE	**METABOLIZER**	**ACTIVITY**
***1/*1**	**EXTENSIVE**	**NORMAL**

CYP2E1

rs NCBI	POLYMORPH	GENOTYPE
rs72559710	1132G>A	G/G
GENOTYPE	**METABOLIZER**	**ACTIVITY**
***1/*1**	**EXTENSIVE**	**NORMAL**

CYP3A4

rs NCBI	POLYMORPH	GENOTYPE
rs2740574	A>G	A/A
rs55785340	A>G	A/A
rs4986910	T>C	T/T
rs55951658	T>C	T/T
rs55901263	G>C	G/G
rs4646438	del>A	del/del
rs4986908	C>G	C/C
rs67784355	G>A	G/G
rs4987161	T>C	T/T
rs28371759	T>C	T/T
rs67666821	del>T	del/del
rs35599367	C>T	C/C
GENOTYPE	**METABOLIZER**	**ACTIVITY**
***1/*1**	**EXTENSIVE**	**NORMAL**

CYP3A5

rs NCBI	POLYMORPH	GENOTYPE
rs776746	6986A>G	A/A
rs10264272	14690G>A	C/C
rs55817950	3699C>T	G/G
rs28383479	19386G>A	G/G
rs41303343	27131_27132insT	del/del
GENOTYPE	**METABOLIZER**	**ACTIVITY**
***1/*1**	**EXTENSIVE**	**NORMAL**

DPYD

rs NCBI	POLYMORPH	GENOTYPE
rs3918290	1905+1G>A	A/A
GENOTYPE	**METABOLIZER**	**ACTIVITY**
***2A/*2A**	**POOR**	**NONE**

NAT2

rs NCBI	POLYMORPH	GENOTYPE
rs1801279	G191A	G/G
rs1041983	C282T	C/C
rs1801280	T341C	T/C
rs1799929	C481T	C/T
rs1799930	G590A	G/G
rs1208	A803G	G/A
rs1799931	G857A	G/G
GENOTYPE	**METABOLIZER**	**ACTIVITY**
N/A	**INTERMEDIATE**	**SLOW**

TPMT

rs NCBI	POLYMORPH	GENOTYPE
rs1800460	G>A	G/G
rs1142345	A>G	A/A
rs1800462	G>C	G/G
GENOTYPE	**METABOLIZER**	**ACTIVITY**
***1/*1**	**EXTENSIVE**	**NORMAL**

SLCO1B1

rs NCBI	POLYMORPH	GENOTYPE
rs4149056	521T>C	C/T
rs2306283	388A>G	T/T
GENOTYPE	**METABOLIZER**	**ACTIVITY**
***1A/*5**	**INTERMEDIATE**	**SLOW**

VKORC1

rs NCBI	POLYMORPH	GENOTYPE
rs9923231	-1639G>A	C/C
GENOTYPE	**RISK**	
C/C	**NO**	

UGT1A1

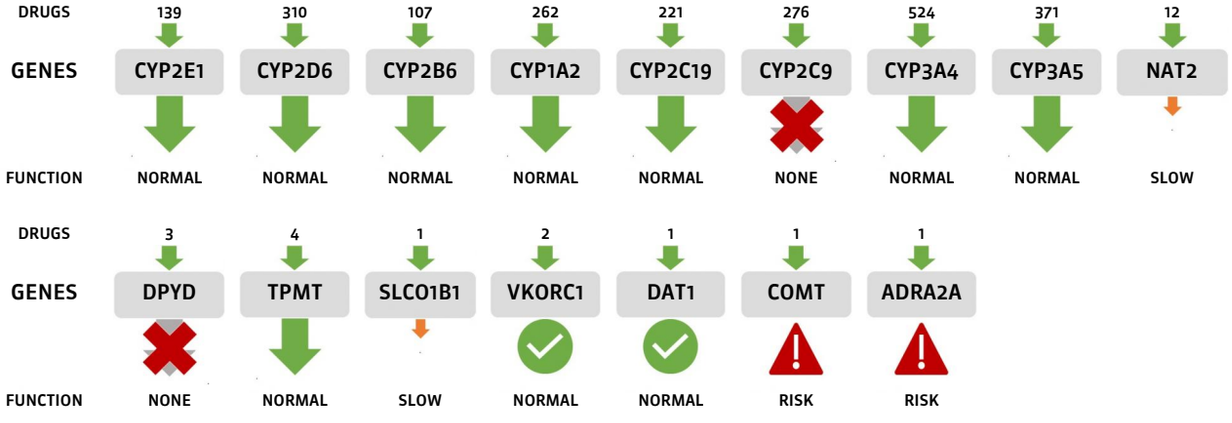
rs NCBI	POLYMORPH	GENOTYPE
rs887829	C>T	T/T
GENOTYPE	**METABOLIZER**	**ACTIVITY**
***80/*80**	**POOR**	**NONE**

LEGEND: rsNCBI = name of examined genetic variation, POLYMORPHISM = pattern of genetic variation, GENOTYPE = personal test result, METABOLIZER = personal metabolism profile, ACTIVITY = enzymatic activity

Please note: We examined a selection of the most common genetic variations affecting your drug metabolism. There are other variations, though only very rarely occurring, which we did not test thoroughly that may affect your drug metabolism, as well. Additionally you have to consider drug interactions, inhibitors, inductors, life style and existing medical conditions prior choosing a treatment or medication.

Summary of the relevant genes

Here, you can view your status of examined genes relevant to the breakdown and activation of various types of medication.

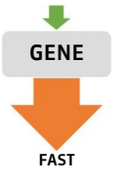


Legend

EXTENSIVE METABOLIZER

The breakdown and/or activation of drugs via this gene work normally.

(ULTRA-)RAPID METABOLIZER



The breakdown and/or activation of drugs via this gene is faster than usual.

INTERMEDIATE METABOLIZER

The breakdown and/or activation of drugs via this gene is slower than usual.

POOR METABOLIZER

The breakdown and/or activation of drugs via this gene is insufficient.

RISK ALLELE CARRIER

This genetic variation increases the risk of side effects of certain drugs.

NO RISK ALLELE CARRIER

This genetic variation does not increase the risk of side effects.

Evaluation of medications

Since the status of your medication-metabolizing genetics is now known, we can assess how the breakdown and activation of various drugs are impaired in your body. Based on this information, we've evaluated individual medications and active ingredients for you in 3 categories (effect, breakdown, dose). This information will help your doctor determine the correct selection and dosage for your medication.

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!

Here is an explanation of each symbol used in the results table:

Effect

Considering your genetic map, this medication has a normal effect. A dosage adjustment is not necessary from a genetic point of view.

Your body activates this medication too quickly (over 30% faster). This can lead to an overdose of the active ingredient. A lower dose is recommended from a genetic point of view.

Your body activates this medication too slowly (between 30%-70% of normal activation). This can lead to an under-dosing of the active ingredient. A higher dose will be necessary to achieve its optimal effect, but the breakdown speed must also be taken into account here.

Your body is unable to sufficiently activate the drug (less than 30% of normal activation). This may render the drug ineffective. An alternative to this medication is recommended from a genetic point of view.

Breakdown

Your body is able to break down this drug with sufficient speed. An adjustment of the dosage is not necessary based on genetics.

The medication is broken down by your body too quickly (more than 30% faster than normal). This may result in a drug concentration that is too low. Genetically speaking, a higher dose would be necessary to achieve the desired effect.

Your body is too slow in breaking down this medication (between 30%-70% of the normal breakdown rate). If you are taking this medication regularly, it may lead to a constantly increasing concentration of the drug in your body. A lower dose is recommended from a genetic point of view.

Your body is unable to sufficiently break down the drug (less than 30% of normal breakdown). If taken regularly, it can lead to a very high drug concentration in the body resulting in severe side effects. An alternative to this medication is recommended from a genetic point of view.

Dose

Neither the effect nor the breaking down of the medication is impaired. A dosage adjustment is not necessary from a genetic point of view.

Due to the faster breakdown, a dose increase of about 130%-200% is recommended from a genetic point of view. Start with the standard dose. In the absence of therapeutic success, a slow increase in dose under medical supervision is advised.

Due to a stronger effect or slower breakdown, a reduction of the dose to between 30% and 70% of the standard dose is recommended from a genetic point of view. It would be advisable to start with a small dose and only slowly increase the dose to the normal dose under medical supervision, if the therapeutic result is not reached.

Due to no effect or no breakdown, an alternative drug is recommended from a genetic point of view. If this is not possible, it is recommended to start with a small dose (3%- 70% of the standard dose) and slowly increase the dose to the normal dose under medical supervision, if the therapeutic result is not reached.

Effect on medication

The following list contains drug delivery guidelines that were published from organizations such as the CPIC (Clinical Pharmacogenetics Implementation Consortium), the Royal Dutch Association for the Advancement of Pharmacy (DPWG), the CPNDS (Canadian Pharmacogenomics Network for Drug Safety), and other professional societies. These results should always be considered by the treating physician.

Drug status	Recommendation for you
Amitriptylin	There is no dose recommendation for this drug.
Aripiprazole	There is no dose recommendation for this drug.
Atomoxetine	There is no dose recommendation for this drug.
Citalopram	There is no dose recommendation for this drug.
Clomipramine	There is no dose recommendation for this drug.
Desipramine	There is no dose recommendation for this drug.
Escitalopram	There is no dose recommendation for this drug.
Fluvoxamine	There is no dose recommendation for this drug.
Haloperidol	There is no dose recommendation for this drug.
Imipramine	There is no dose recommendation for this drug.
Nortriptyline	There is no dose recommendation for this drug.
Paroxetine	There is no dose recommendation for this drug.

Risperidone	There is no dose recommendation for this drug.
Sertraline	There is no dose recommendation for this drug.
Venlafaxine	There is no dose recommendation for this drug.
Zuclopenthixol	There is no dose recommendation for this drug.

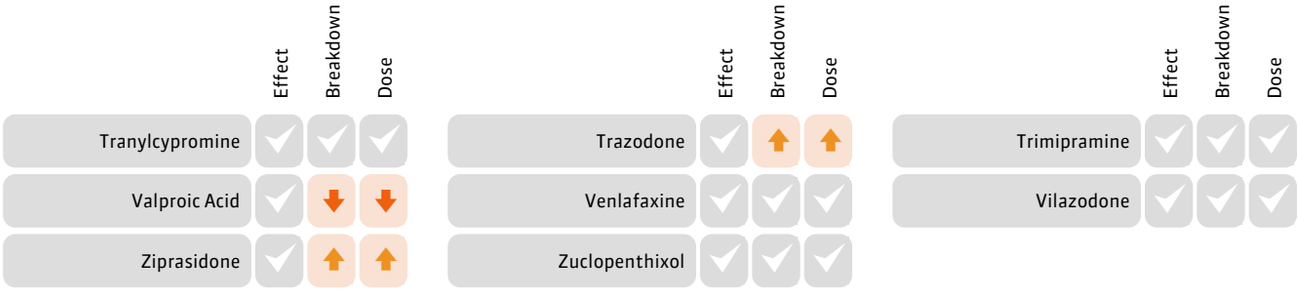
Effect on medication

The following list contains medications that have been evaluated by their degradation and activation pathways. This information will help your doctor to choose and dose your medication properly.

	Effect	Breakdown	Dose
Agomelatine	✓	✓	✓
Amoxapine	✓	✓	✓
Baclofen	✓	✓	✓
Carbamazepine	↑	↑	↓
Clobazam	✓	↑	↑
Clozapine	✓	✓	✓
Desvenlafaxine	✓	✓	✓
Dopamine	✓	✓	✓
Duloxetine	✓	✓	✓
Fluoxetine	✓	✗	✗
Haloperidol	✓	↑	✓
Isocarboxazid	✓	✓	✓
Maprotiline	✓	✓	✓
Minaprine	✓	✓	✓
Modafinil	✓	↑	↑
Nortriptyline	✓	✓	✓
Paroxetine	✓	✓	✓
Pimozide	✓	↑	↑
Protriptyline	✓	✓	✓
Remoxipride	✓	✓	✓
Sertraline	✓	✓	✓

	Effect	Breakdown	Dose
Amfetamine	✓	✓	✓
Aripiprazole	✓	↑	✓
Bupropion	✓	✓	✓
Chlorpromazine	✓	✓	✓
Clomipramine	↑	✓	✓
Cyclobenzaprine	✓	✓	✓
Dexmethylphenidate	✓	✓	✓
Doxazosin	✓	✓	✓
Epinephrine	✓	✓	✓
Fluvoxamine	✓	✓	✓
Iloperidone	✓	✓	✓
Levetiracetam	✓	✓	✓
Methylphenidate	✓	✓	✓
Mirtazapine	✓	✓	✓
Nefazodone	✓	↑	↑
Olanzapine	✓	✓	✓
Perphenazine	✓	✓	✓
Prazosin	✓	✓	✓
Quetiapine	✓	↑	↑
Risperidone	✓	✓	✓
Thioridazine	✓	✓	✓

	Effect	Breakdown	Dose
Amitriptyline	✓	✓	✓
Atomoxetine	✓	✓	✓
Buspirone	✓	↑	↑
Citalopram	✓	↑	✓
Clonidine	✓	✓	✓
Desipramine	✓	✓	✓
Diazepam	✓	↑	↑
Doxepin	✓	✓	✓
Escitalopram	✓	↑	✓
Guanfacine	✓	✓	✓
Imipramine	✓	✓	✓
Lisdexamfetamine	✓	✓	✓
Mianserin	✓	✓	✓
Moclobemide	✓	✓	✓
Norepinephrine	✓	✓	✓
Oxcarbazepine	✓	✓	✓
Phenelzine	✓	↑	↑
Propranolol	✓	✓	✓
Reboxetine	✓	↑	↑
Selegiline	✓	✗	✗
Topiramate	✓	✓	✓


Effect
Breakdown
Dose
Tranylcypromine
Valproic Acid
Ziprasidone
Trazodone
Venlafaxine
Zuclopenthixol
Trimipramine
Vilazodone

SCIENCE

This chapter shows the science behind the test.

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All our results are based on large scientifically validated replication studies to ensure the highest level of scientific validity. Here you can find a selection of scientific papers, this analysis was based on.

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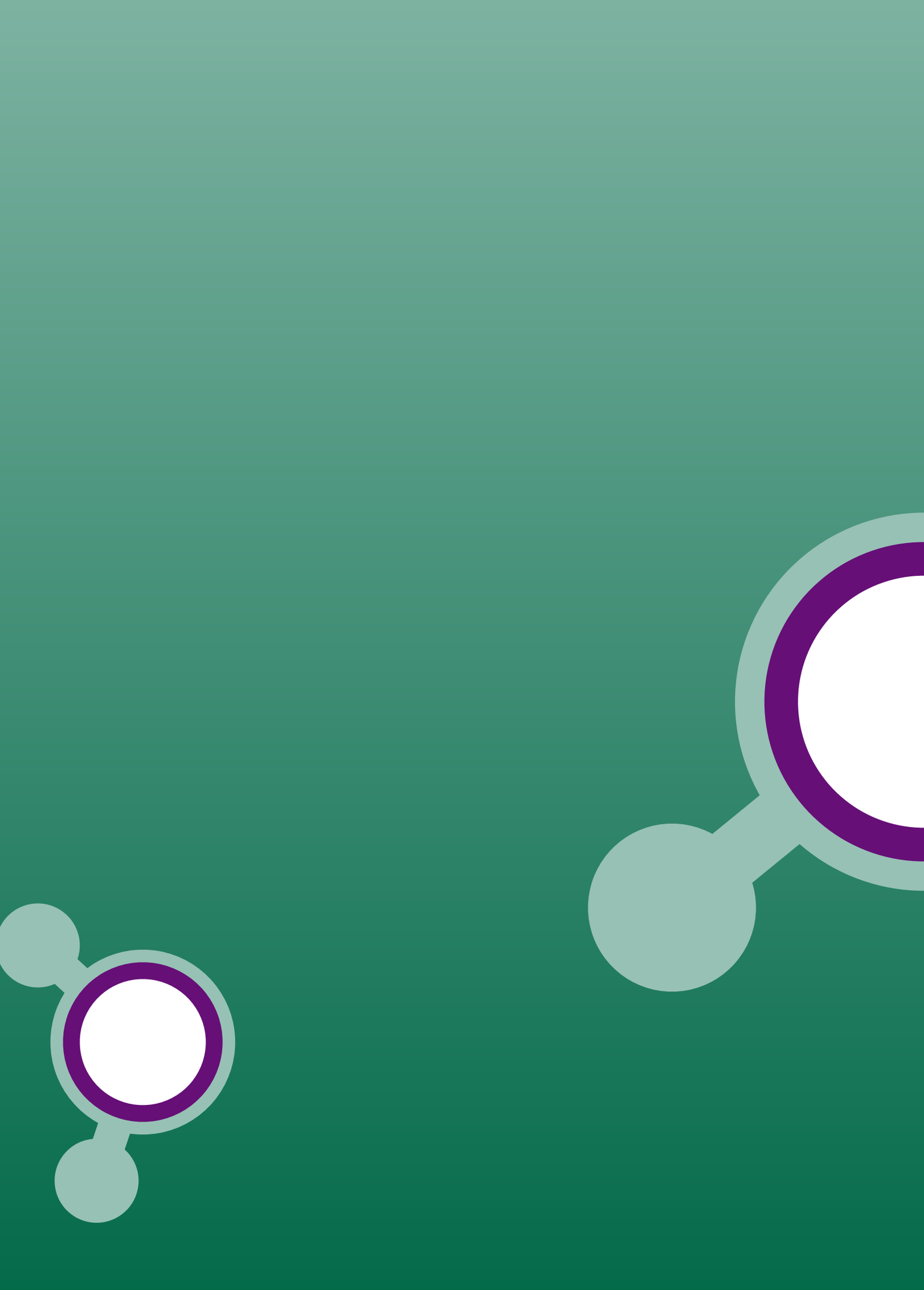

ADDITIONAL INFORMATION

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

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Product codes
M5ADH

Current version
V538

Ordering company
Novogenia GmbH
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5301 Eugendorf, Österreich

Analyzing company
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Deutschland

Laboratory Director

Dr. Daniel Wallerstorfer Bsc.

Laboratory Manager

René Rohrmanstorfer, MSc.

NOTES:


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ADHD Sensor

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