ProMedicDNA

Premium Sensor PLUS

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

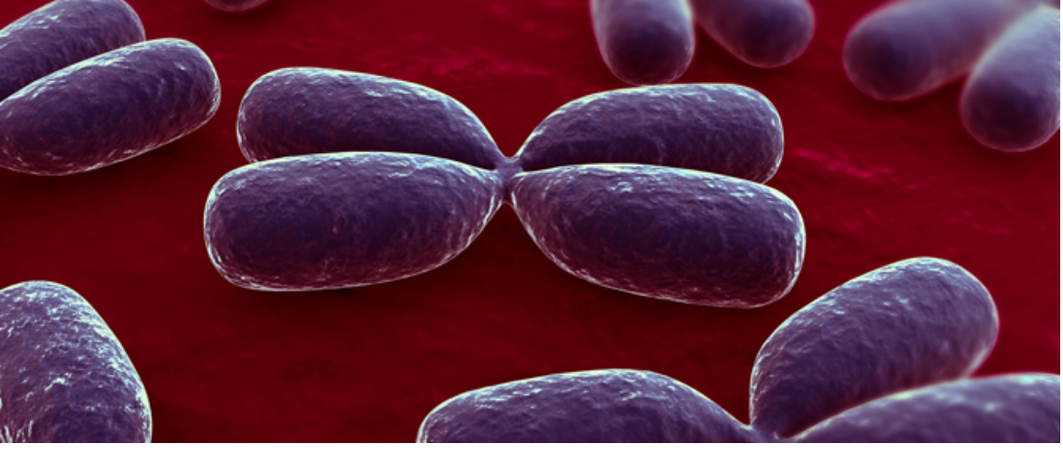
Florian Schneebauer, MSc.
Laboratory Manager

Premium Sensor PLUS

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

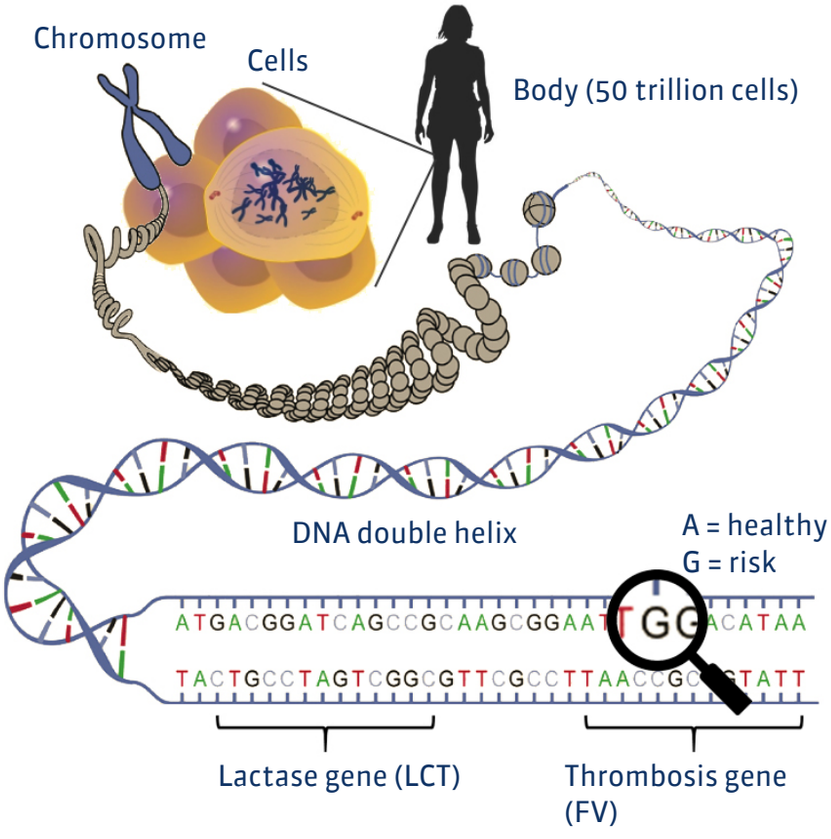
Order number:
DEMO_DS

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.

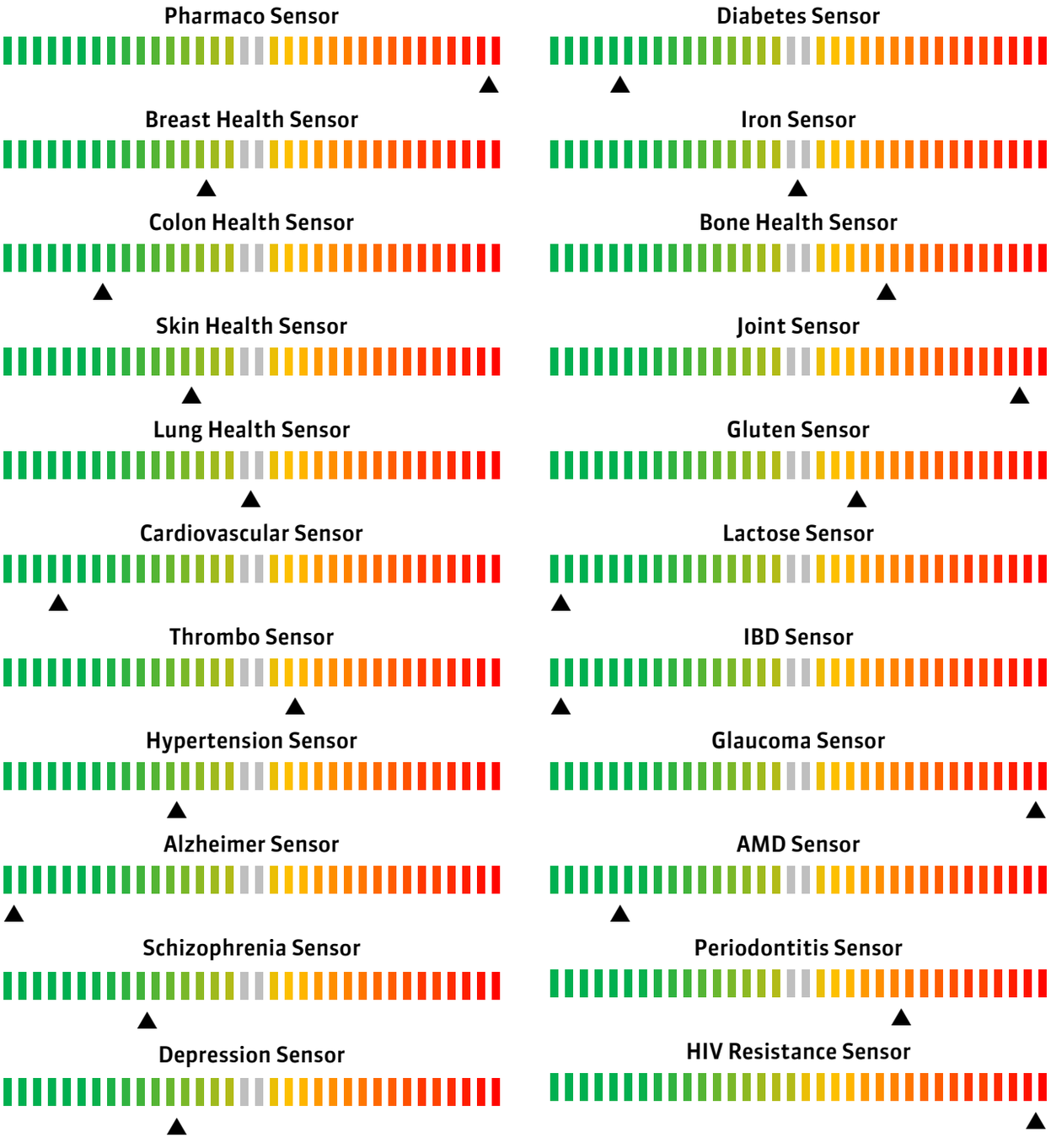
Experts estimate that every person carries about 2,000 genetic defects, which may affect their health, and in some cases, cause illnesses. A variety of factors can cause changes in our genes (also called mutations). In a few cases, these mutations can benefit us. However, the vast majority either have 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.

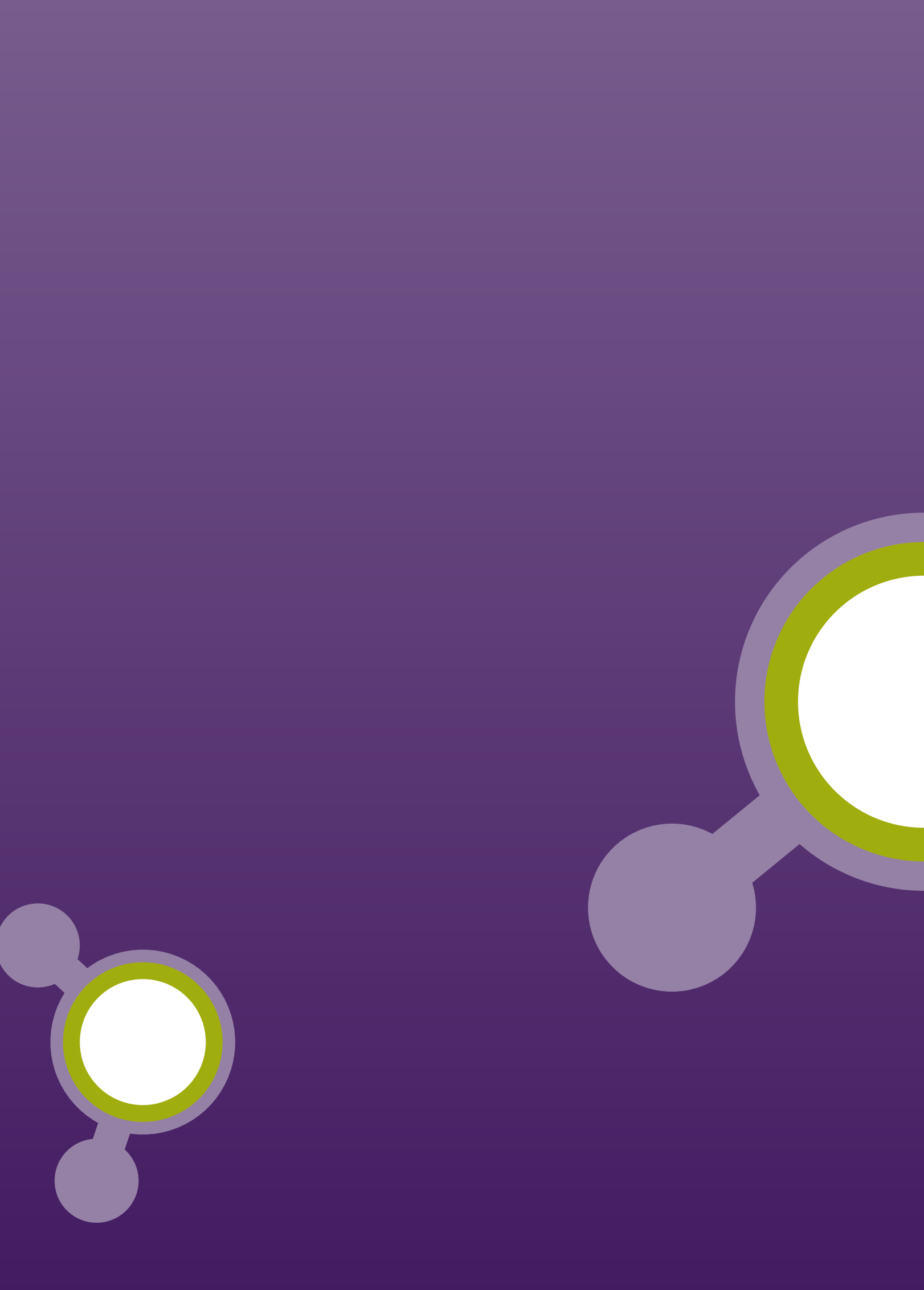
External influences can affect individual genes and disrupt their function, but the majority of our defective genes are inherited from our parents. Each embryo receives half of its genes from the father and half from the mother, resulting in a new human being with some characteristics of each parent.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





PHARMACO GENETICS

ONCOLOGY

CARDIOVASCULAR SYSTEM

NEUROLOGY

METABOLISM

MOVEMENT

DIGESTION

OPHTHALMOLOGY

ODONTOLOGY

OTHERS

SCIENCE

ADDITIONAL INFORMATION

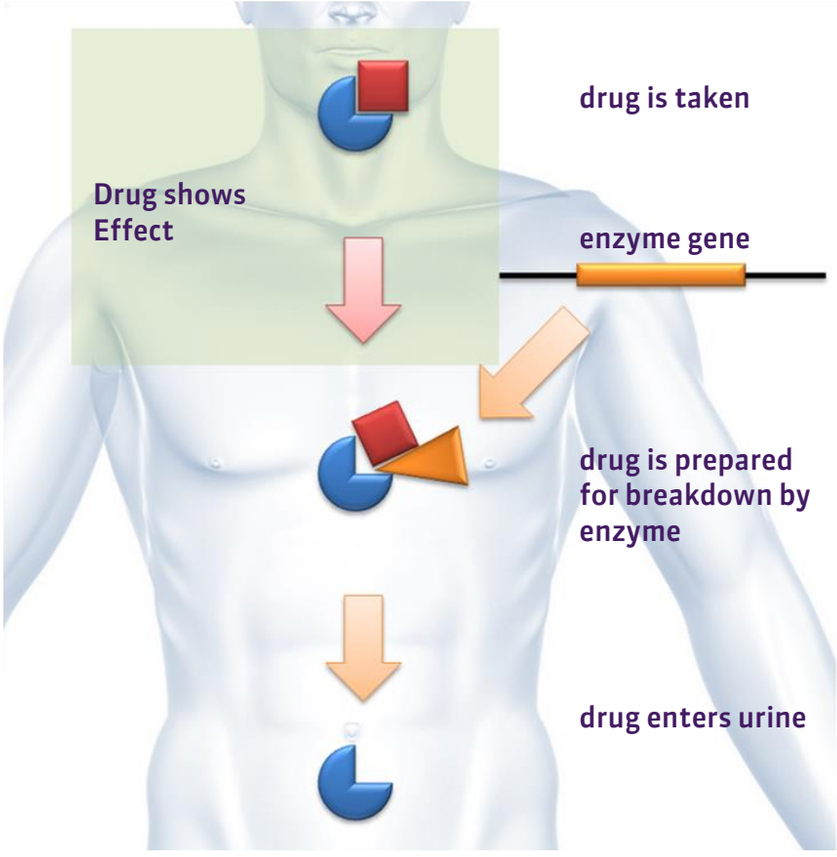
Pharmaco Sensor

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.

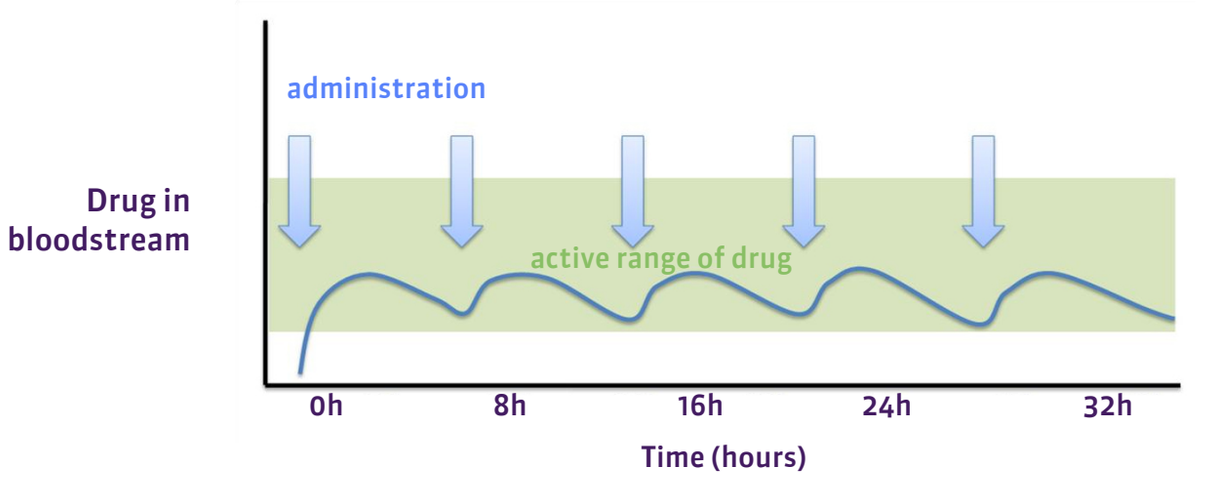
The drug pathways in the body



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.

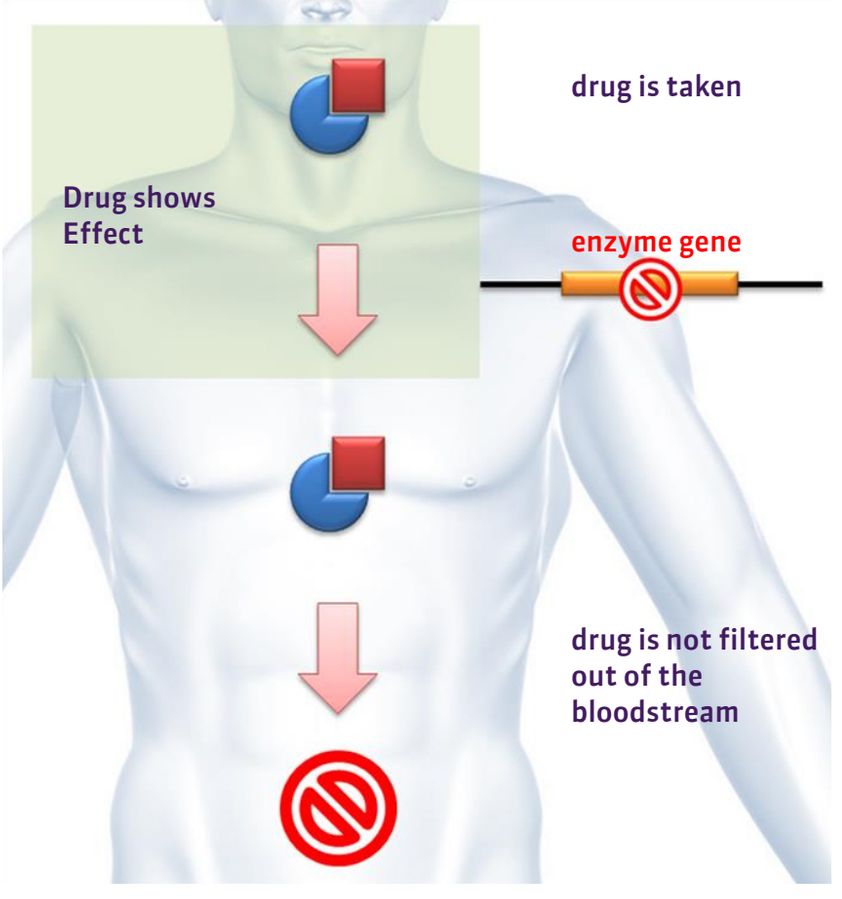
Long-term Drug Treatment

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

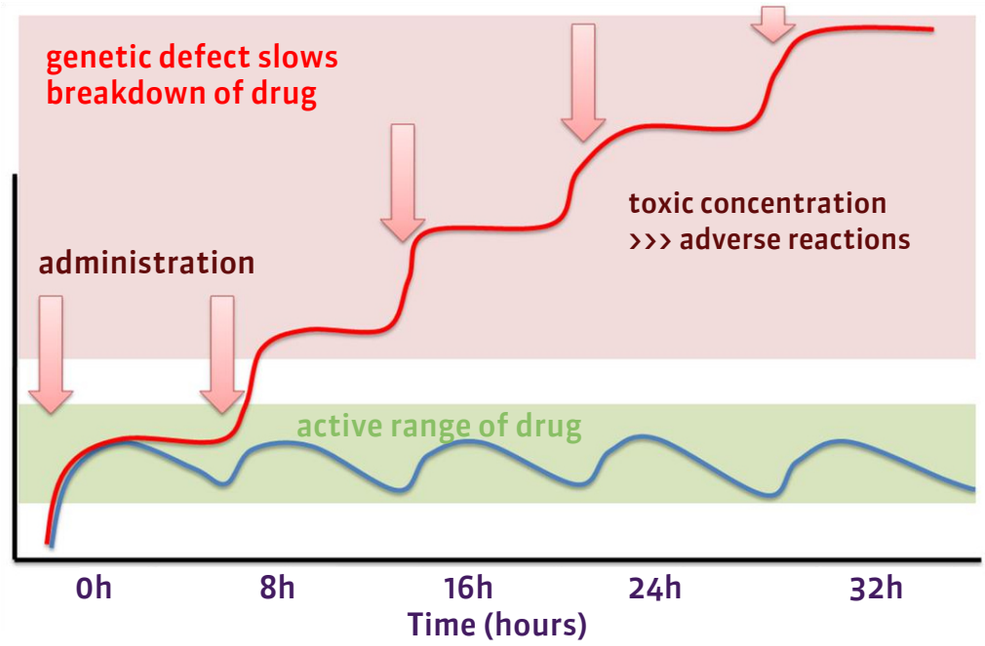


Unfortunately, many people carry a defect in one of the enzyme-producing genes that are crucial in this process.

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

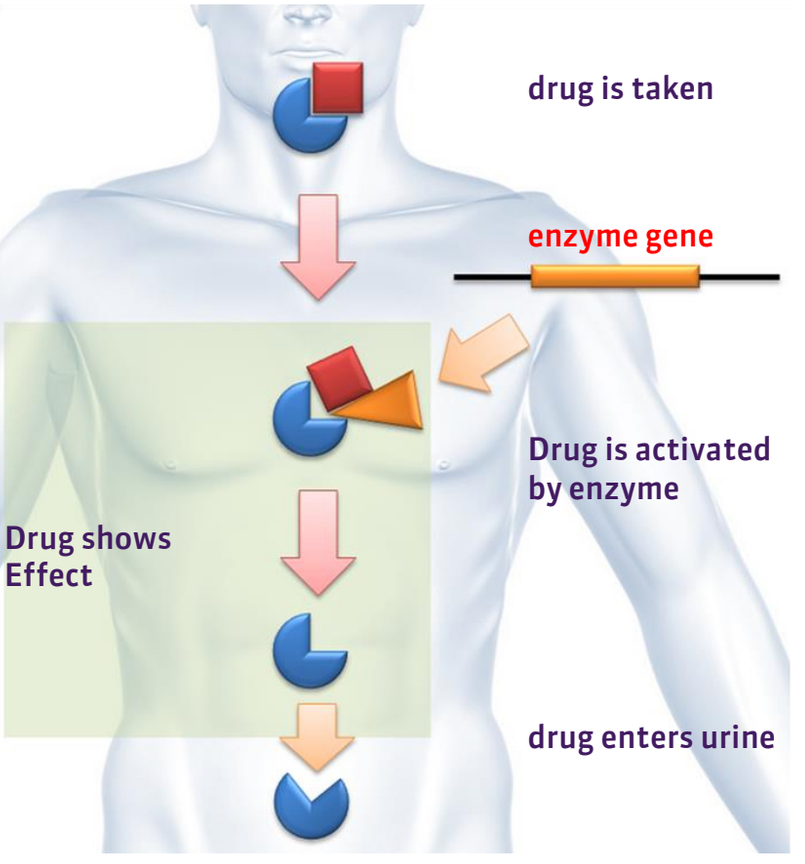
In the case of blood thinners, drug action is at the optimum level at the beginning of therapy but the drug concentration increases subsequently with every dose until it reaches the point of causing uncontrolled bleeding.

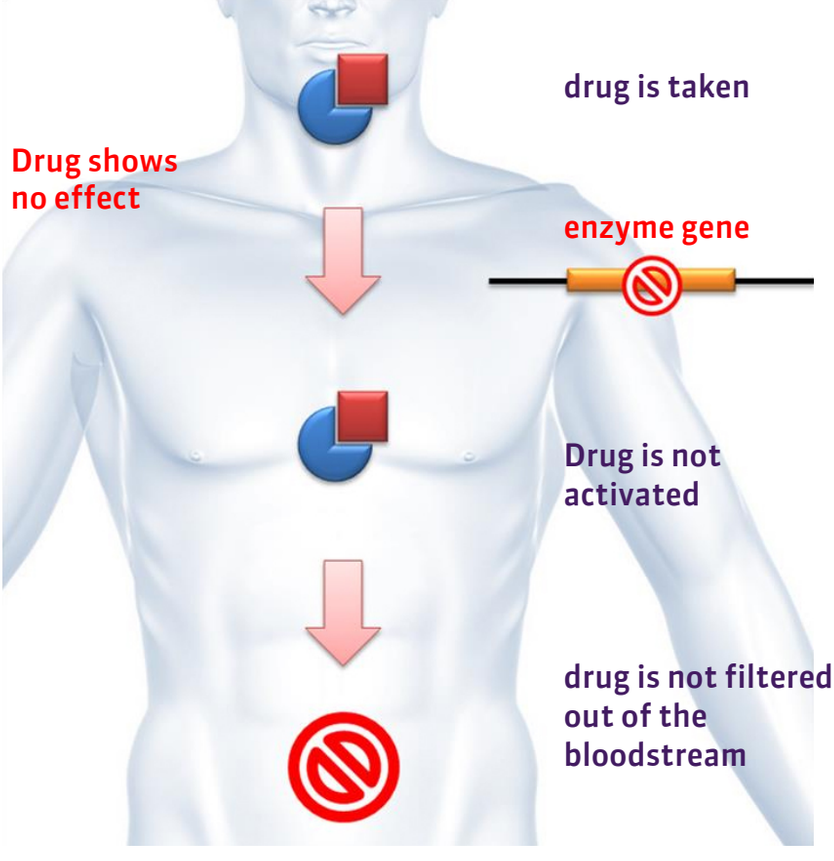


This means that the 20% of the population that carry a genetic defect need a significantly lower dose of warfarin because the usual dose could lead to serious adverse reactions.

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

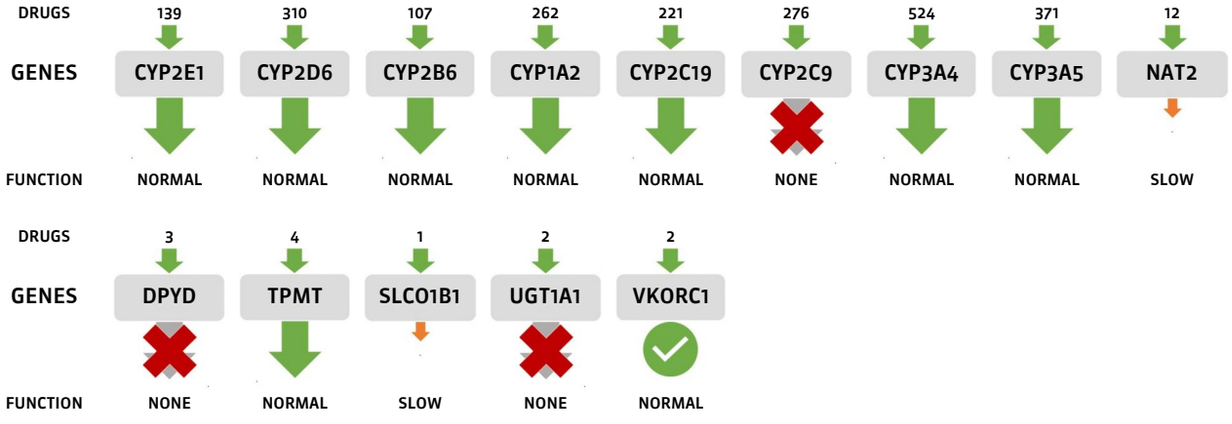
rs NCBI	POLYMORPH	GENOTYPE
rs887829	C>T	T/T
GENOTYPE	**METABOLIZER**	**ACTIVITY**
***80/*80**	**POOR**	**NONE**

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.

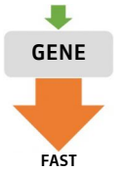


Legend

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
Abacavir	Abacavir is not recommended. High risk of hypersensitivity (~6% of patients) due to the presence of at least one HLA-B*57:01 allele.
Acenocoumarol	Check INR more frequently.
Amitriptyline	There is no dose recommendation for this drug.
Aripiprazole	There is no dose recommendation for this drug.
Atazanavir	Consider an alternative agent particularly where jaundice is of concern to the patient.
Atomoxetine	There is no dose recommendation for this drug.
Azathioprine	There is no dose recommendation for this drug.
Capecitabine	Select alternative drug. Tegafur is not a suitable alternative because this drug is also metabolized by DPD.
Citalopram	There is no dose recommendation for this drug.
Clomipramine	There is no dose recommendation for this drug.
Clopidogrel	There is no dose recommendation for this drug.
Codeine	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.
Esomeprazole	There is no dose recommendation for this drug.
Flecainide	There is no dose recommendation for this drug.
Fluorouracil	Select alternative drug. Tegafur is not a suitable alternative because this drug is also metabolized by DPD.
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.
Irinotecan	Reduce initial dose by 30% for patients receiving more than 250 mg/m2. Increase dose in response to neutrophil count.
Lansoprazole	There is no dose recommendation for this drug.
Mercaptopurine	There is no dose recommendation for this drug.
Metoprolol	There is no dose recommendation for this drug.
Nortriptyline	There is no dose recommendation for this drug.
Ondansetron	There is no dose recommendation for this drug.
Oxycodone	There is no dose recommendation for this drug.
Pantoprazole	There is no dose recommendation for this drug.
Paroxetine	There is no dose recommendation for this drug.
Phenprocoumon	Check INR more frequently.
Phenytoin	Use standard loading dose and reduce maintenance dose by 50%. Evaluate response and serum concentration after 7-10 days. Be alert to ADEs (e.g. ataxia, nystagmus, dysarthria, sedation).
Propafenone	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.

Simvastatin	Prescribe a lower dose or consider an alternative statin (e.g. pravastatin or rosuvastatin); consider routine CK surveillance.
Tacrolimus	Increase starting dose 1.5-2 times recommended starting dose. Total starting dose should not exceed 0.3mg/kg/day. Use therapeutic drug monitoring to guide dose adjustments.
Tamoxifen	Avoid moderate and strong CYP2D6 inhibitors. Initiate therapy with recommended standard of care dosing.
Tegafur	Select alternative drug. Fluorouracil or capecitabine are not suitable alternatives because both are also metabolized by DPD.
Thioguanine	There is no dose recommendation for this drug.
Tramadol	There is no dose recommendation for this drug.
Tropisetron	There is no dose recommendation for this drug.
Venlafaxine	There is no dose recommendation for this drug.
Voriconazole	There is no dose recommendation for this drug.
Warfarin	Use www.warfarindosing.org to calculate exact warfarin dosing recommendation.
Zuclopenthixol	There is no dose recommendation for this drug.

Source: https://www.pharmgkb.org/page/citingPharmgkb

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
[32P]Natriumphosphat	✓	✓	✓
Abarelix	✓	✓	✓
Acadesine	✓	✓	✓
Acebutolol	✓	✓	✓
Acefylline Piperazine	✓	✓	✓
Acepromazine	✓	✓	✓
Acetohexamide	✓	✓	✓
Acetoxolone	✓	✓	✓
Acetylcysteine	✓	✓	✓
Acetylglycinamide Chloral Hydrate	✓	✓	✓
Acipimox	✓	✓	✓
Acriflavinium Chloride	✓	✓	✓
Adefovir Dipivoxil	✓	✓	✓
Adinazolam	✓	✓	✓
Afatinib	✓	✓	✓
Ajmaline	✓	✓	✓
Albendazole	↑	↑	↓
Alcuronium	✓	✓	✓
Alemtuzumab	✓	✓	✓
Alfentanil	✓	↑	↑
Alginic Acid	✓	✓	✓

	Effect	Breakdown	Dose
4-dimethylaminophenol	✓	✓	✓
Abciximab	✓	✓	✓
Acamprosate	✓	✓	✓
Aceclidine	✓	✓	✓
Acemetacin	✓	✓	✓
Acetarsol	✓	✓	✓
Acetohydroxamic Acid	✓	✓	✓
Acetylcarnitine	✓	✓	✓
Acetyldigitoxin	✓	✓	✓
Acetylleucine	✓	✓	✓
Acitretin	✓	✓	✓
Acrivastine	✓	✓	✓
Ademetionine	✓	✓	✓
Adrafinil	✓	✓	✓
Afelimomab	✓	✓	✓
Alanyl Glutamine	✓	✓	✓
Alclofenac	✓	✓	✓
Aldesulfone Sodium	✓	✓	✓
Alendronic Acid	✓	✓	✓
Alfuzosin	✓	↑	↑
Alimemazine	✓	✓	✓

	Effect	Breakdown	Dose
Abacavir	✓	✓	✗
Abiraterone	✓	✓	✓
Acarbose	✓	✓	✓
Aceclofenac	✓	✗	✗
Acenocoumarol	✓	↓	✓
Acetazolamide	✓	✓	✓
Acetophenazine	✓	✓	✓
Acetylcholin	✓	✓	✓
Acetyldigoxin	✓	✓	✓
Acetylsalicylic Acid	✓	✗	✗
Aclarubicin	✓	✓	✓
Adalimumab	✓	✓	✓
Adenosine	✓	✓	✓
Adrenalone	✓	✓	✓
Agomelatine	✓	✓	✓
Alaproclate	✓	✓	✓
Alclometasone	✓	✓	✓
Aldosterone	✓	✓	✓
Alfaxalone	✓	✓	✓
Algeldrate	✓	✓	✓
Aliskiren	✓	↑	↑

	Effect	Breakdown	Dose
Alitretinoin	✓	✓	✓
Allopurinol	✓	✓	✓
Almasilate	✓	✓	✓
Almotriptan	✓	↑	↑
Alprazolam	✓	↑	↑
Alsactide	✓	✓	✓
Aluminium Acetoacetate	✓	✓	✓
Aluminium Glycinate	✓	✓	✓
Aluminium Phosphate	✓	✓	✓
Amantadin	✓	✓	✓
Ambrisentan	✓	✓	✓
Amezinium Metilsulfate	✓	✓	✓
Amiloride	✓	✓	✓
Aminobutyric Acid	✓	✓	✓
Aminohippuric Acid	✓	✓	✓
Aminophenazone	✓	✓	✓
Amiodarone	✓	↓	↓
Amlexanox	✓	✓	✓
Amobarbital	✓	✓	✓
Amoxicillin	✓	✓	✓
Amprenavir	✓	↑	↑
Amsacrine	✓	✓	✓
Anakinra	✓	✓	✓
Anecortave	✓	✓	✓
Anidulafungin	✓	✓	✓
Antimony Pentasulfide	✓	✓	✓
Aprepitant	✓	↑	↑
Apronal	✓	✓	✓
Arbutamine	✓	✓	✓

	Effect	Breakdown	Dose
Alizapride	✓	✓	✓
Allylestrenol	✓	✓	✓
Alminoprofen	✓	✓	✓
Alogliptin	✓	✓	✓
Alprenolol	✓	✓	✓
Altretamine	✓	✓	✓
Aluminium Acetotartrate	✓	✓	✓
Aluminium Hydroxide	✓	✓	✓
Alverine	✓	✓	✓
Ambazone	✓	✓	✓
Ambroxol	✓	✓	✓
Amfepramone	✓	✓	✓
Amineptine	✓	✓	✓
Aminocaproic Acid	✓	✓	✓
Aminolevulinic Acid	✓	✓	✓
Aminophylline	✓	✓	✓
Amisulpride	✓	✓	✓
Amlodipine	✓	↑	↑
Amodiaquine	✓	✓	✓
Amphotericin B	✓	✓	✓
Amrinone	✓	✓	✓
Amyl Nitrite	✓	✓	✓
Anastrozole	✓	✓	✓
Anethole Trithione	✓	✓	✓
Anileridine	✓	✓	✓
Apomorphine	✓	✓	✓
Aprindine	✓	✓	✓
Aprotinin	✓	✓	✓
Argatroban	✓	↑	↑

	Effect	Breakdown	Dose
Allobarbital	✓	✓	✓
Almagate	✓	✓	✓
Almitrine	✓	✓	✓
Alosetron	✓	✓	✓
Alprostadil	✓	✓	✓
Alum	✓	✓	✓
Aluminium Clofibrate	✓	✓	✓
Aluminium Nicotinate	✓	✓	✓
Alvimopan	✓	✓	✓
Ambenonium	✓	✓	✓
Amcinonide	✓	✓	✓
Amifostine	✓	✓	✓
Amino(Diphenylhydantoin) Valeric Acid	✓	✓	✓
Aminogluthetimide	✓	✓	✓
Aminomethylbenzoic Acid	✓	✓	✓
Aminosalicylic Acid	✓	✓	✓
Amitriptyline	✓	✓	✓
Ammonium Chloride	✓	✓	✓
Amoxapine	✓	✓	✓
Ampicillin	✓	✓	✓
Amrubicin	✓	✓	✓
Anagrelide	✓	✓	✓
Androstanolone	✓	✓	✓
Angiotensinamide	✓	✓	✓
Aniracetam	✓	✓	✓
Apraclonidine	✓	✓	✓
Aprobarbital	✓	✓	✓
Arbekacin	✓	✓	✓
Arginine Glutamate	✓	✓	✓

	Effect	Breakdown	Dose
Arginine Hydrochloride	✓	✓	✓
Arsenic Trioxide	✓	✓	✓
Artemisinin	✓	✓	✓
Artesunate	✓	✓	✓
Asparaginase	✓	✓	✓
Atazanavir	✓	↑	✗
Atorvastatin	✓	↑	↑
Atracurium	✓	✓	✓
Aurothioglucose	✓	✓	✓
Azacitidine	✓	✓	✓
Azapropazone	✓	✓	✓
Azidamfenicol	✓	✓	✓
Azlocillin	✓	✓	✓
Baclofen	✓	✓	✓
Bamethan	✓	✓	✓
Barbital	✓	✓	✓
Beclamide	✓	✓	✓
Bekanamycin	✓	✓	✓
Benazepril	✓	✓	✓
Bendroflumethiazide	✓	✓	✓
Benorilate	✓	✓	✓
Benproperine	✓	✓	✓
Benzathine Phenoxymethylpenicillin	✓	✓	✓
Benzethonium	✓	✓	✓
Benznidazole	✓	✓	✓
Benzonatate	✓	✓	✓
Benzyl Benzoate	✓	✓	✓
Bephenium	✓	✓	✓
Bergapten	✓	✓	✓

	Effect	Breakdown	Dose
Argipressin	✓	✓	✓
Arsthinol	✓	✓	✓
Artemotil	✓	✓	✓
Articaine	✓	✓	✓
Aspoxicillin	✓	✓	✓
Atenolol	✓	✓	✓
Atosiban	✓	✓	✓
Atropine	✓	✓	✓
Aurotioprol	✓	✓	✓
Azanidazole	✓	✓	✓
Azatadine	✓	✓	✓
Azidocillin	✓	✓	✓
Aztreonam	✓	✓	✓
Balsalazide	✓	✓	✓
Bamifylline	✓	✓	✓
Barnidipine	✓	✓	✓
Beclometasone	✓	✓	✓
Bemegride	✓	✓	✓
Bencyclane	✓	✓	✓
Benfluorex	✓	✓	✓
Benoxaprofen	✓	✓	✓
Bentiromide	✓	✓	✓
Benzatropine	✓	✓	✓
Benzilone	✓	✓	✓
Benzocaine	✓	✓	✓
Benzoxonium Chloride	✓	✓	✓
Benzylpenicillin	✓	✓	✓
Bepridil	✓	✓	✓
Betahistine	✓	✓	✓

	Effect	Breakdown	Dose
Aripiprazole	✓	↑	✓
Artemether	✓	↑	↑
Artenimol	✓	✓	✓
Asenapine	✓	✓	✓
Astemizole	✓	↑	↑
Atomoxetine	✓	✓	✓
Atovaquone	✓	✓	✓
Auranofin	✓	✓	✓
Axitinib	✓	✓	✓
Azapetine	✓	✓	✓
Azathioprine	✓	✓	✓
Azithromycin	✓	↑	↑
Bacampicillin	✓	✓	✓
Bambuterol	✓	✓	✓
Barbexaclone	✓	✓	✓
Bazedoxifene	✓	✓	✓
Befunolol	✓	✓	✓
Bemiparin	✓	✓	✓
Bendamustine	✓	✓	✓
Benidipine	✓	✓	✓
Benperidol	✓	✓	✓
Benzathine Benzylpenicillin	✓	✓	✓
Benzbromarone	✓	✓	✓
Benziodarone	✓	✓	✓
Benzoctamine	✓	✓	✓
Benzydamine	✓	✓	✓
Benzylthiouracil	✓	✓	✓
Beraprost	✓	✓	✓
Betaine	✓	✓	✓

	Effect	Breakdown	Dose
Betaine Hydrochloride	✓	✓	✓
Betaxolol	✓	✓	✓
Bevacizumab	✓	✓	✓
Bexarotene	✓	✓	✓
Biapenem	✓	✓	✓
Bicalutamide	✓	↑	↑
Bilastine	✓	✓	✓
Biperiden	✓	✓	✓
Bismuth Subnitrate	✓	✓	✓
Bitolterol	✓	✓	✓
Boric Acid	✓	✓	✓
Bosentan	✓	↓	↓
Brimonidine	✓	✓	✓
Brodimoprim	✓	✓	✓
Bromfenac	✓	✓	✓
Bromisoval	✓	✓	✓
Bromperidol	✓	✓	✓
Broxyquinoline	✓	✓	✓
Bucladesine	✓	✓	✓
Budipine	✓	✓	✓
Buformin	✓	✓	✓
Bumetanide	✓	✓	✓
Bupivacaine	✓	✓	✓
Bupropion	✓	✓	✓
Busulfan	✓	↑	↑
Butanilicaine	✓	✓	✓
Butoconazole	✓	✓	✓
Butylscopolamine	✓	✓	✓
Cadralazine	✓	✓	✓

	Effect	Breakdown	Dose
Betamethason	✓	✓	✓
Betazole	✓	✓	✓
Bevantolol	✓	✓	✓
Bezafibrate	✓	✓	✓
Bibenzonium Bromide	✓	✓	✓
Bietaserpine	✓	✓	✓
Bimatoprost	✓	✓	✓
Bisacodyl	✓	✓	✓
Bisoprolol	✓	↑	↑
Bleomycin	✓	✓	✓
Bornaprine	✓	✓	✓
Bosutinib	✓	✓	✓
Brinzolamide	✓	↑	↑
Bromazepam	✓	↑	↑
Bromhexine	✓	✓	✓
Bromocriptine	✓	↑	↑
Brompheniramine	✓	✓	✓
Bucetin	✓	✓	✓
Buclizine	✓	✓	✓
Bufexamac	✓	✓	✓
Bufylline	✓	✓	✓
Bunaftine	✓	✓	✓
Bupranolol	✓	✓	✓
Buserelin	✓	✓	✓
Butalamine	✓	✓	✓
Butaperazine	✓	✓	✓
Butorphanol	✓	✓	✓
Cabazitaxel	✓	↑	↑
Cafedrine	✓	✓	✓

	Effect	Breakdown	Dose
Betanidine	✓	✓	✓
Bethanechol	✓	✓	✓
Bevonium	✓	✓	✓
Bezitramide	✓	✓	✓
Bibrocathol	✓	✓	✓
Bifemelane	✓	✓	✓
Bioallethrin	✓	✓	✓
Bismuth Subcitrate	✓	✓	✓
Bisoxatin	✓	✓	✓
Bopindolol	✓	✓	✓
Bortezomib	✓	↑	↑
Bretylium Tosilate	✓	✓	✓
Brivudine	✓	✓	✓
Bromazine	✓	✓	✓
Bromides	✓	✓	✓
Bromopride	✓	✓	✓
Brotizolam	✓	✓	✓
Bucillamine	✓	✓	✓
Budesonide	✓	✓	✓
Buflomedil	✓	✓	✓
Bumadizone	✓	✓	✓
Buphenine	✓	✓	✓
Buprenorphine	✓	↑	↑
Buspirone	✓	↑	↑
Butamirate	✓	✓	✓
Butobarbital	✓	✓	✓
Butriptyline	✓	✓	✓
Cabergoline	✓	↑	↑
Calcium Aminosalicylate	✓	✓	✓

	Effect	Breakdown	Dose
Calcium Carbimide	✓	✓	✓
Calcium Dobesilate	✓	✓	✓
Calcium Silicate	✓	✓	✓
Camphora	✓	✓	✓
Candicidin	✓	✓	✓
Capreomycin	✓	✓	✓
Carbachol	✓	✓	✓
Carbasalate Calcium	✓	✓	✓
Carbenoxolon	✓	✓	✓
Carbimazole	✓	✓	✓
Carbocromen	✓	✓	✓
Carboquone	✓	✓	✓
Carbuterol	✓	✓	✓
Carindacillin	✓	✓	✓
Carmofur	✓	✓	✓
Carteolol	✓	✓	✓
Casopitant	✓	✓	✓
Cefacetrile	✓	✓	✓
Cefalexin	✓	✓	✓
Cefamandole	✓	✓	✓
Cefazedone	✓	✓	✓
Cefcapene	✓	✓	✓
Cefepime	✓	✓	✓
Cefmenoxime	✓	✓	✓
Cefodizime	✓	✓	✓
Ceforanide	✓	✓	✓
Cefotiam	✓	✓	✓
Cefpiramide	✓	✓	✓
Cefprozil	✓	✓	✓

	Effect	Breakdown	Dose
Calcium Carbonate	✓	✓	✓
Calcium Folinate	✓	✓	✓
Camazepam	✓	✓	✓
Camylofin	✓	✓	✓
Canrenone	✓	✓	✓
Captodiame	✓	✓	✓
Carbamazepine	↑	↑	↓
Carbazochrome	✓	✓	✓
Carbetocin	✓	✓	✓
Carbinoxamine	✓	✓	✓
Carboplatin	✓	✓	✓
Carbromal	✓	✓	✓
Carfecillin	✓	✓	✓
Carisbamate	✓	✓	✓
Carmustine	✓	✓	✓
Carumonam	✓	✓	✓
Caspofungin	✓	✓	✓
Cefaclor	✓	✓	✓
Cefaloridine	✓	✓	✓
Cefapirin	✓	✓	✓
Cefazolin	✓	✓	✓
Cefdinir	✓	✓	✓
Cefetamet	✓	✓	✓
Cefmetazole	✓	✓	✓
Cefonicide	✓	✓	✓
Cefotaxime	✓	✓	✓
Cefoxitin	✓	✓	✓
Cefpirome	✓	✓	✓
Cefradine	✓	✓	✓

	Effect	Breakdown	Dose
Calcium Compounds	✓	✓	✓
Calcium Levofolinate	✓	✓	✓
Camostat	✓	✓	✓
Candesartan	✓	↓	↓
Capecitabine	✓	✗	✗
Captopril	✓	✓	✓
Carbamide	✓	✓	✓
Carbenicillin	✓	✓	✓
Carbidopa	✓	✓	✓
Carbocisteine	✓	✓	✓
Carboprost	✓	✓	✓
Carbutamide	✓	✓	✓
Carglumic Acid	✓	✓	✓
Carisoprodol	✓	✓	✓
Caroverine	✓	✓	✓
Carvedilol	✓	✗	✗
Cathine	✓	✓	✓
Cefadroxil	✓	✓	✓
Cefalotin	✓	✓	✓
Cefatrizine	✓	✓	✓
Cefbuperazone	✓	✓	✓
Cefditoren	✓	✓	✓
Cefixime	✓	✓	✓
Cefminox	✓	✓	✓
Cefoperazone	✓	✓	✓
Cefotetan	✓	✓	✓
Cefozopran	✓	✓	✓
Cefpodoxime	✓	✓	✓
Cefroxadine	✓	✓	✓

	Effect	Breakdown	Dose
Cefsulodin	✓	✓	✓
Ceftezole	✓	✓	✓
Ceftobiprole Medocaril	✓	✓	✓
Celecoxib	✓	✗	✗
Cerivastatin	✓	↑	↑
Cetirizine	✓	↑	↑
Cetylpyridinium	✓	✓	✓
Chinin	✓	↑	↑
Chloralodol	✓	✓	✓
Chlorcyclizine	✓	✓	✓
Chlormadinone	✓	✓	✓
Chlorobutanol	✓	✓	✓
Chlorothiazide	✓	✓	✓
Chlorproethazine	✓	✓	✓
Chlorprothixene	✓	✓	✓
Chlorzoxazone	✓	✓	✓
Choline Fenofibrate	✓	✓	✓
Chondroitin Sulfate	✓	✓	✓
Ciclesonide	✓	↑	↑
Ciclonicate	✓	✓	✓
Cilansetron	✓	✓	✓
Cilostazol	✓	↑	↑
Cinacalcet	✓	↑	↑
Cinepazet	✓	✓	✓
Cinolazepam	✓	✓	✓
Ciprofloxacin	✓	✓	✓
Cisplatin	✓	✓	✓
Citiolone	✓	✓	✓
Clavulans□Ure	✓	✓	✓

	Effect	Breakdown	Dose
Ceftaroline Fosamil	✓	✓	✓
Ceftibuten	✓	✓	✓
Ceftriaxone	✓	✓	✓
Celiprolol	✓	✓	✓
Ceruletide	✓	✓	✓
Cetrorelix	✓	✓	✓
Cevimeline	✓	↑	↑
Chiniofon	✓	✓	✓
Chlorambucil	✓	✓	✓
Chlordiazepoxide	✓	↑	↑
Chlormethine	✓	✓	✓
Chloroprocaine	✓	✓	✓
Chlorotrianisene	✓	✓	✓
Chlorpromazine	✓	✓	✓
Chlortalidone	✓	✓	✓
Cholic Acid	✓	✓	✓
Choline Salicylate	✓	✓	✓
Chromium (51Cr) Edetate	✓	✓	✓
Cicletanine	✓	✓	✓
Ciclosporin	✓	↑	↑
Cilazapril	✓	✓	✓
Cimetidine	✓	✓	✓
Cinchocaine	✓	✓	✓
Cinepazide	✓	✓	✓
Cinoxacin	✓	✓	✓
Cisapride	✓	↑	↑
Citalopram	✓	↑	✓
Cladribine	✓	✓	✓
Clebopride	✓	✓	✓

	Effect	Breakdown	Dose
Ceftazidime	✓	✓	✓
Ceftizoxime	✓	✓	✓
Cefuroxime	✓	✓	✓
Cerium Oxalate	✓	✓	✓
Cetiedil	✓	✓	✓
Cetuximab	✓	✓	✓
Chenodeoxycholic Acid	✓	✓	✓
Chloral Hydrate	✓	✓	✓
Chlorbenzoxamine	✓	✓	✓
Chlorhexidine	✓	✓	✓
Chlormezanone	✓	✓	✓
Chloroquine	↑	✓	↓
Chlorphenamine	✓	✓	✓
Chlorpropamide	✓	↓	↓
Chlortetracycline	✓	✓	✓
Choline Alfoscerate	✓	✓	✓
Choline Theophyllinate	✓	✓	✓
Cibenzoline	✓	✓	✓
Ciclobendazole	✓	✓	✓
Cidofovir	✓	✓	✓
Cilnidipine	✓	✓	✓
Cimetropium Bromide	✓	✓	✓
Cinchophen	✓	✓	✓
Cinnarizin	✓	✓	✓
Ciprofibrate	✓	✓	✓
Cisatracurium	✓	✓	✓
Citicoline	✓	✓	✓
Clarithromycin	✓	↑	↑
Clefamide	✓	✓	✓

	Effect	Breakdown	Dose
Clenbuterol	✓	✓	✓
Clindamycin	✓	✓	✓
Clobetasol	✓	✓	✓
Clocortolone	✓	✓	✓
Clofarabine	✓	✓	✓
Clofenamide	✓	✓	✓
Clofibrate	✓	↑	↑
Clomethiazole	✓	↑	↑
Clomipramine	↑	✓	✓
Clonidine	✓	✓	✓
Cloperastine	✓	✓	✓
Cloranolol	✓	✓	✓
Cloridarol	✓	✓	✓
Clotiazepam	✓	✓	✓
Cloxazolam	✓	✓	✓
Codeine	✓	✓	✓
Colestipol	✓	✓	✓
Colistin	✓	✓	✓
Copper Oleinate	✓	✓	✓
Corticotropin	✓	✓	✓
Creatinolfosfate	✓	✓	✓
Cyamemazine	✓	↓	↓
Cyclobarbital	✓	✓	✓
Cyclofenil	✓	✓	✓
Cyclopentolate	✓	✓	✓
Cyclothiazide	✓	✓	✓
Cypermethrin	✓	✓	✓
Cytarabine	✓	✓	✓
Daclizumab	✓	✓	✓

	Effect	Breakdown	Dose
Clevidipine	✓	✓	✓
Clobazam	✓	↑	↑
Clobetasone	✓	✓	✓
Clodantoin	✓	✓	✓
Clofazimine	✓	✓	✓
Clofenotane	✓	✓	✓
Clofibride	✓	✓	✓
Clometocillin	✓	✓	✓
Clomocycline	✓	✓	✓
Clopamide	✓	✓	✓
Clopidogrel	✓	↑	✓
Clorexolone	✓	✓	✓
Clorindione	✓	✓	✓
Clotrimazole	✓	✓	✓
Clozapine	✓	✓	✓
Colchicine	✓	↑	↑
Colestyramine	✓	✓	✓
Conivaptan	✓	✓	✓
Copper Usnate	✓	✓	✓
Cortisone	✓	↑	↑
Cromoglicic Acid	✓	✓	✓
Cyclandelate	✓	✓	✓
Cyclobenzaprine	✓	✓	✓
Cycloguanil Embonate	✓	✓	✓
Cyclophosphamide	✓	↑	↑
Cyfluthrin	✓	✓	✓
Cyproheptadin	✓	✓	✓
Dabigatran Etexilate	✓	✓	✓
Dactinomycin	✓	✓	✓

	Effect	Breakdown	Dose
Clevudine	✓	✓	✓
Clobenzorex	✓	✓	✓
Clobutinol	✓	✓	✓
Clodronic Acid	✓	✓	✓
Clofedanol	✓	✓	✓
Clofezone	✓	✓	✓
Clofoctol	✓	✓	✓
Clomifene	✓	✓	✓
Clonazepam	✓	↑	↑
Clopenthixol	✓	✓	✓
Cloprednol	✓	✓	✓
Cloricromen	✓	✓	✓
Clotiapine	✓	✓	✓
Cloxacillin	✓	✓	✓
Cobalt (58Co) Cyanocobalamine	✓	✓	✓
Colesevelam	✓	✓	✓
Colfosceril Palmitate	✓	✓	✓
Conjugated Estrogens	✓	✓	✓
Corticorelin	✓	✓	✓
Cortivazol	✓	✓	✓
Crospovidone	✓	✓	✓
Cyclizine	✓	✓	✓
Cyclobutyrol	✓	✓	✓
Cyclopenthiazide	✓	✓	✓
Cycloserine	✓	✓	✓
Cymarin	✓	✓	✓
Cyproterone	✓	✓	✓
Dacarbazine	✓	✓	✓
Dalbavancin	✓	✓	✓

	Effect	Breakdown	Dose
Danaparoid	✓	✓	✓
Dantron	✓	✓	✓
Daptomycin	✓	✓	✓
Daunorubicin	✓	✓	✓
Decamethrin	✓	✓	✓
Deferiprone	✓	✓	✓
Degarelix	✓	✓	✓
Demecarium	✓	✓	✓
Demoxytocin	✓	✓	✓
Desaspidin	✓	✓	✓
Desipramine	✓	✓	✓
Desloratadine	✓	↑	↑
Desonide	✓	✓	✓
Desvenlafaxine	✓	✓	✓
Dexchlorpheniramine	✓	✓	✓
Dexibuprofen	✓	✓	✓
Dexmedetomidine	✓	✓	✓
Dextran	✓	✓	✓
Dextropropoxyphene	✓	✓	✓
Diacerein	✓	✓	✓
Diazoxide	✓	✓	✓
Dibromotyrosine	✓	✓	✓
Dibutylsuccinate	✓	✓	✓
Dichlorophen	✓	✓	✓
Dicloxacillin	✓	✓	✓
Didanosine	✓	✓	✓
Diethyl Ether	✓	✓	✓
Diethyltoluamide	✓	✓	✓
Difenpiramide	✓	✓	✓

	Effect	Breakdown	Dose
Danazol	✓	✓	✓
Dapagliflozin	✓	✓	✓
Darunavir	✓	↑	↑
Deanol	✓	✓	✓
Decitabine	✓	✓	✓
Deferoxamine	✓	✓	✓
Delapril	✓	✓	✓
Demecolcine	✓	✓	✓
Deptropine	✓	✓	✓
Deserpidine	✓	✓	✓
Desirudin	✓	✓	✓
Desmopressin	✓	✓	✓
Desoximetasone	✓	✓	✓
Dexamethasone	✓	↑	↑
Dexetimide	✓	✓	✓
Dexketoprofen	✓	✓	✓
Dexpanthenol	✓	✓	✓
Dextromethorphan	✓	✓	✓
Dextrothyroxine	✓	✓	✓
Diamorphine	✓	✓	✓
Dibekacin	✓	✓	✓
Dibunate	✓	✓	✓
Dichloralphenazone	✓	✓	✓
Diclofenac	✓	✗	✗
Dicoumarol	✓	✗	✗
Dienestrol	✓	✓	✓
Diethylcarbamazine	✓	✓	✓
Difemerine	✓	✓	✓
Difetarsone	✓	✓	✓

	Effect	Breakdown	Dose
Dantrolene	✓	↑	↑
Dapiprazole	✓	✓	✓
Dasatinib	✓	↑	↑
Debrisoquine	✓	✓	✓
Deferasirox	✓	✓	✓
Deflazacort	✓	✓	✓
Delavirdine	✓	↑	↑
Demegestone	✓	✓	✓
Dermatan Sulfate	✓	✓	✓
Desflurane	✓	✓	✓
Deslanoside	✓	✓	✓
Desogestrel	✓	↓	↓
Desoxycortone	✓	✓	✓
Dexbrompheniramine	✓	✓	✓
Dexfenfluramine	✓	✓	✓
Dexlansoprazole	✓	✓	✓
Dexrazoxane	✓	✓	✓
Dextromoramide	✓	✓	✓
Dezocine	✓	✓	✓
Diazepam	✓	↑	↑
Dibenzepin	✓	✓	✓
Dibutylphthalate	✓	✓	✓
Dichlorobenzyl Alcohol	✓	✓	✓
Diclofenamide	✓	✓	✓
Dicycloverine	✓	✓	✓
Dienogest	✓	✓	✓
Diethylstilbestrol	✓	✓	✓
Difenoxin	✓	✓	✓
Diflorasone	✓	✓	✓

	Effect	Breakdown	Dose
Diflucortolone	✓	✓	✓
Digitoxin	✓	↑	↑
Dihydralazine	✓	✓	✓
Dihydroergocristine	✓	✓	✓
Dihydrostreptomycin	✓	✓	✓
Diiodotyrosine	✓	✓	✓
Diloxanide	✓	✓	✓
Dimemorfan	✓	✓	✓
Dimethoxanate	✓	✓	✓
Dimethylcarbate	✓	✓	✓
Dimeticone	✓	✓	✓
Dinoprost	✓	✓	✓
Diosmin	✓	✓	✓
Diphenhydramin	✓	✓	✓
Diprophylline	✓	✓	✓
Dirithromycin	✓	↑	↑
Disulfiram	✓	✓	✓
Dixyrazine	✓	✓	✓
Docusate Sodium	✓	✓	✓
Domiodol	✓	✓	✓
Donepezil	✓	↑	↑
Dorzolamide	✓	✓	✓
Doxapram	✓	✓	✓
Doxepin	✓	✓	✓
Doxorubicin	✓	↑	↑
Dronabinol	✓	↓	↓
Dropropizine	✓	✓	✓
Droxypropine	✓	✓	✓
Dyclonine	✓	✓	✓

	Effect	Breakdown	Dose
Diflunisal	✓	✓	✓
Digoxin	✓	✓	✓
Dihydrocodeine	✓	✓	✓
Dihydroergocryptine Mesylate	✓	✓	✓
Dihydroxialumini Sodium Carbonate	✓	✓	✓
Diisopromine	✓	✓	✓
Diltiazem	✓	↑	↑
Dimercaprol	✓	✓	✓
Dimethyl Sulfoxide	✓	✓	✓
Dimethylphthalate	✓	✓	✓
Dimetofrine	✓	✓	✓
Dinoprostone	✓	✓	✓
Diphemanil	✓	✓	✓
Diphenoxylate	✓	✓	✓
Dipyridamole	✓	✓	✓
Disopyramide	✓	↑	↑
Ditazole	✓	✓	✓
Dobutamine	✓	✓	✓
Dofetilide	✓	↑	↑
Domiphen	✓	✓	✓
Dopexamine	✓	✓	✓
Dosulepin	✓	✓	✓
Doxazosin	✓	✓	✓
Doxercalciferol	✓	✓	✓
Doxycycline	✓	↑	↑
Dronedarone	✓	✓	✓
Drotaverine	✓	✓	✓
Duloxetine	✓	✓	✓
Dydrogesterone	✓	✓	✓

	Effect	Breakdown	Dose
Difluprednate	✓	✓	✓
Dihexyverine	✓	✓	✓
Dihydroemetine	✓	✓	✓
Dihydroergotamine	✓	↑	↑
Diiodohydroxyquinoline	✓	✓	✓
Dilazep	✓	✓	✓
Dimefline	✓	✓	✓
Dimetacrine	✓	✓	✓
Dimethylaminopropionylphenothiazine	✓	✓	✓
Dimethyltubocurarine	✓	✓	✓
Dimetotiazine	✓	✓	✓
Diosmectite	✓	✓	✓
Diphenadione	✓	✓	✓
Dipivefrine	✓	✓	✓
Dipyrocetyl	✓	✓	✓
Distigmine	✓	✓	✓
Dixanthogen	✓	✓	✓
Docetaxel	✓	↑	↑
Dolasetron	✓	✓	✓
Domperidone	✓	✓	✓
Doripenem	✓	✓	✓
Doxacurium Chloride	✓	✓	✓
Doxefazepam	✓	✓	✓
Doxofylline	✓	✓	✓
Doxylamin	✓	✓	✓
Droperidol	✓	↑	↑
Droxicam	✓	✓	✓
Dutasteride	✓	↑	↑
Ebastine	✓	✓	✓

	Effect	Breakdown	Dose
Ecallantide	✓	✓	✓
Efaproxiral	✓	✓	✓
Efloxate	✓	✓	✓
Eltrombopag	✓	✓	✓
Emetine	✓	✓	✓
Enalapril	✓	✓	✓
Enflurane	✓	✓	✓
Enoxaparin	✓	✓	✓
Entacapone	✓	✓	✓
Eperisone	✓	✓	✓
Epimestrol	✓	✓	✓
Epirubicin	✓	✓	✓
Epoprostenol	✓	✗	✗
Eprozinol	✓	✓	✓
Ergoloid Mesylates	✓	✓	✓
Erityrityl Tetranitrate	✓	✓	✓
Escitalopram	✓	↑	✓
Esomeprazole	✓	✓	✓
Estramustine	✓	✓	✓
Eszopiclone	✓	↑	↑
Etallobarbital	✓	✓	✓
Etamsylate	✓	✓	✓
Ethadione	✓	✓	✓
Ethenzamide	✓	✓	✓
Ethisterone	✓	✓	✓
Ethyl Biscoumacetate	✓	✓	✓
Ethylestrenol	✓	✓	✓
Etidronic Acid	✓	✓	✓
Etizolam	✓	✓	✓

	Effect	Breakdown	Dose
Ecothiopate	✓	✓	✓
Efavirenz	✓	↑	↑
Elcatonin	✓	✓	✓
Emedastine	✓	✓	✓
Emtricitabine	✓	✓	✓
Encainide	✓	✓	✓
Enfuvirtide	✓	✓	✓
Enoximone	✓	✓	✓
Entecavir	✓	✓	✓
Ephedrin	✓	✓	✓
Epinastine	✓	✓	✓
Eplerenone	✓	↑	↑
Eprazinone	✓	✓	✓
Eptifibatide	✓	✓	✓
Ergometrine	✓	✓	✓
Erlotinib	✓	↑	↑
Eslicarbazepine	✓	✓	✓
Estazolam	✓	↑	↑
Estriol	✓	✓	✓
Etacrynic Acid	✓	✓	✓
Etamiphylline	✓	✓	✓
Etanercept	✓	✓	✓
Ethambutol	✓	✓	✓
Ethinylestradiol	✓	↑	↑
Ethosuximide	✓	↑	↑
Ethyl Chloride	✓	✓	✓
Ethylmorphine	✓	✓	✓
Etifoxine	✓	✓	✓
Etodolac	✓	✗	✗

	Effect	Breakdown	Dose
Edetates	✓	✓	✓
Eflornithine	✓	✓	✓
Eletriptan	✓	↑	↑
Emepronium	✓	✓	✓
Emylcamate	✓	✓	✓
Endralazine	✓	✓	✓
Enoxacin	✓	✓	✓
Enprostil	✓	✓	✓
Epanolol	✓	✓	✓
Epicillin	✓	✓	✓
Epinephrine	✓	✓	✓
Epomediol	✓	✓	✓
Eprosartan	✓	✓	✓
Erdosteine	✓	✓	✓
Ergotamine	✓	↑	↑
Ertapenem	✓	✓	✓
Esmolol	✓	✓	✓
Estradiol	✓	↑	↑
Estrone	✓	✓	✓
Etafenone	✓	✓	✓
Etamivan	✓	✓	✓
Ethacridinlactat	✓	✓	✓
Ethchlorvynol	✓	✓	✓
Ethionamide	✓	✓	✓
Ethotoin	✓	✓	✓
Ethyl Loflazepate	✓	✓	✓
Etidocaine	✓	✓	✓
Etilefrine	✓	✓	✓
Etofamide	✓	✓	✓

	Effect	Breakdown	Dose
Etofenamat	✓	✓	✓
Etoglucid	✓	✓	✓
Etonogestrel	✓	✓	✓
Etoricoxib	✓	✓	✓
Etretinate	✓	✓	✓
Everolimus	✓	↑	↑
Ezetimibe	✓	✓	✓
Fampridine	✓	✓	✓
Febarbamate	✓	✓	✓
Felbamate	✓	↑	↑
Fenbufen	✓	✓	✓
Fenetylline	✓	✓	✓
Fenoldopam	✓	✓	✓
Fenoverine	✓	✓	✓
Fenpiverinium	✓	✓	✓
Fentanyl	✓	↑	↑
Fenyramidol	✓	✓	✓
Fesoterodine	✓	✓	✓
Fingolimod	✓	✓	✓
Flecainide	✓	✓	✓
Flomoxef	✓	✓	✓
Flubendazole	✓	✓	✓
Fludarabine	✓	✓	✓
Fludroxycortide	✓	✓	✓
Flumazenil	✓	✓	✓
Flumetasone	✓	✓	✓
Flunoxaprofen	✓	✓	✓
Fluocortin	✓	✓	✓
Fluorometholone	✓	✓	✓

	Effect	Breakdown	Dose
Etofibrate	✓	✓	✓
Etohexadiol	✓	✓	✓
Etoperidone	✓	✓	✓
Etozolin	✓	✓	✓
Etybenzatropine	✓	✓	✓
Exemestane	✓	↑	↑
Famciclovir	✓	✓	✓
Fasudil	✓	✓	✓
Febuxostat	✓	✓	✓
Felodipine	✓	↑	↑
Fencamfamin	✓	✓	✓
Fenfluramine	✓	✓	✓
Fenoprofen	✓	✓	✓
Fenozolone	✓	✓	✓
Fenquizone	✓	✓	✓
Fentiazac	✓	✓	✓
Feprazone	✓	✓	✓
Fexofenadine	✓	↑	↑
Fipexide	✓	✓	✓
Fleroxacin	✓	✓	✓
Flosequinan	✓	✓	✓
Fluclorolone	✓	✓	✓
Fludiazepam	✓	✓	✓
Flufenamic Acid	✓	✓	✓
Flumedroxone	✓	✓	✓
Flunarizine	✓	↓	↓
Fluocinolone Acetonide	✓	✓	✓
Fluocortolone	✓	✓	✓
Fluorouracil	✓	✗	✗

	Effect	Breakdown	Dose
Etofylline Nicotinate	✓	✓	✓
Etomidate	✓	✓	✓
Etoposide	✓	↑	↑
Etravirine	✓	✓	✓
Etynodiol	✓	✓	✓
Exenatide	✓	✓	✓
Famotidine	✓	✓	✓
Fazadinium Bromide	✓	✓	✓
Fedrilate	✓	✓	✓
Fenbendazole	✓	✓	✓
Fendiline	✓	✓	✓
Fenofibrate	✓	✓	✓
Fenoterol	✓	✓	✓
Fenpiprane	✓	✓	✓
Fenspiride	✓	✓	✓
Fentonium	✓	✓	✓
Ferric Citrate	✓	✓	✓
Finasteride	✓	↑	↑
Flavoxate	✓	✓	✓
Floctafenine	✓	✓	✓
Fluanisone	✓	✓	✓
Flucloxacillin	✓	✓	✓
Fludrocortisone	✓	✓	✓
Fluindione	✓	✓	✓
Flumequine	✓	✓	✓
Flunitrazepam	✓	↑	↑
Fluocinonide	✓	✓	✓
Fluorescein	✓	✓	✓
Fluostigmine	✓	✓	✓

	Effect	Breakdown	Dose
Fluoxetine	✓	✗	✗
Fluperolone	✓	✓	✓
Fluprednidene	✓	✓	✓
Flurithromycin	✓	✓	✓
Fluticasone	✓	↑	↑
Fomepizole	✓	✓	✓
Formestane	✓	✓	✓
Fosamprenavir	✓	↑	↑
Fosfomycin	✓	✓	✓
Fosphenytoin	✓	✗	✗
Fulvestrant	✓	↑	↑
Furosemide	✓	✓	✓
Gallamine	✓	✓	✓
Gamolenic Acid	✓	✓	✓
Garenoxacin	✓	✓	✓
Gefarnate	✓	✓	✓
Gemeprost	✓	✓	✓
Gepefrine	✓	✓	✓
Gestrinone	✓	✓	✓
Glatiramer Acetate	✓	✓	✓
Gliclazide	✓	✗	✗
Gliquidone	✓	✓	✓
Glutamic Acid Hydrochloride	✓	✓	✓
Glyceryl Trinitrate	✓	✓	✓
Glycopyrronium	✓	✓	✓
Gonadorelin	✓	✓	✓
Granisetron	✓	✓	✓
Guacetisal	✓	✓	✓
Guajazulen	✓	✓	✓

	Effect	Breakdown	Dose
Fluoxymesterone	✓	✓	✓
Fluphenazine	✓	✓	✓
Flurazepam	✓	↑	↑
Fluspirilene	✓	✓	✓
Fluvastatin	✓	✓	✓
Fomivirsen	✓	✓	✓
Formocortal	✓	✓	✓
Fosfestrol	✓	✓	✓
Fosfonet	✓	✓	✓
Fotemustine	✓	✓	✓
Fumagillin	✓	✓	✓
Gabapentin	✓	✓	✓
Gallium (67Ga) Citrate	✓	✓	✓
Ganciclovir	✓	✓	✓
Gatifloxacin	✓	✓	✓
Gefitinib	✓	↑	↑
Gemfibrozil	✓	↑	↑
Gepirone	✓	✓	✓
Gitoformate	✓	✓	✓
Glibenclamide	✓	↓	↓
Glimepiride	↓	✗	✗
Glisoxepide	✓	✓	✓
Glutathione	✓	✓	✓
Glycine	✓	✓	✓
Glycyrrhizic Acid	✓	↑	↑
Goserelin	✓	✓	✓
Grepafloxacin	✓	✓	✓
Guaiacolsulfonate	✓	✓	✓
Guanazodine	✓	✓	✓

	Effect	Breakdown	Dose
Flupenthixol	✓	✓	✓
Flupirtine	✓	✓	✓
Flurbiprofen	✓	✗	✗
Flutamide	✓	↑	↑
Fluvoxamine	✓	✓	✓
Fondaparinux	✓	✓	✓
Formoterol	✓	↓	↓
Fosfocreatine	✓	✓	✓
Fosinopril	✓	✓	✓
Frovatriptan	✓	✓	✓
Furazolidon	✓	✓	✓
Galantamine	✓	↑	↑
Gallopamil	✓	✓	✓
Ganirelix	✓	✓	✓
Gedocarnil	✓	✓	✓
Gemcitabine	✓	✓	✓
Gemifloxacin	✓	✓	✓
Gestonorone	✓	✓	✓
Glafenine	✓	✓	✓
Glibornuride	✓	✗	✗
Glipizide	✓	✗	✗
Glucosamine	✓	✓	✓
Glutethimide	✓	✓	✓
Glycobiarsol	✓	✓	✓
Glymidine	✓	✓	✓
Gramicidin	✓	✓	✓
G-Strophanthin	✓	✓	✓
Guaifenesin	✓	✓	✓
Guanethidine	✓	✓	✓

	Effect	Breakdown	Dose
Guanfacine	✓	✓	✓
Guanoxan	✓	✓	✓
Halcinonide	✓	✓	✓
Haloperidol	✓	↑	✓
Heptabarbital	✓	✓	✓
Hexafluronium	✓	✓	✓
Hexobarbital	✓	✓	✓
Hexoprenaline	✓	✓	✓
Histapyrrodine	✓	✓	✓
Hyaluronidase	✓	✓	✓
Hydrocodone	✓	✓	✓
Hydrocortisone Buteprate	✓	✓	✓
Hydromorphone	✓	↓	↓
Hydrotalcite	✓	✓	✓
Hydroxychloroquine	✓	✓	✓
Hydroxyzine	✓	✓	✓
Hypromellose	✓	✓	✓
Ibritumomab-Tiuxetan	✓	✓	✓
Ibuproxam	✓	✓	✓
Iclaprim	✓	✓	✓
Idebenone	✓	✓	✓
Ifosfamide	↑	↑	↓
Imatinib	↑	↑	↓
Imipenem	✓	✓	✓
Indacaterol	✓	✓	✓
Indinavir	✓	↑	↑
Indometacin	✓	↓	↓
Infliximab	✓	✓	✓
Insulin Aspart	✓	✓	✓

	Effect	Breakdown	Dose
Guanoclor	✓	✓	✓
Gusperimus	✓	✓	✓
Halofantrine	✓	↑	↑
Halothane	✓	✓	✓
Heptaminol	✓	✓	✓
Hexapropymate	✓	✓	✓
Hexobendine	✓	✓	✓
Hexylresorcinol	✓	✓	✓
Histrelin	✓	✓	✓
Hydralazine	✓	✓	✓
Hydrocortisone	✓	↑	↑
Hydrocortisone Butyrate	✓	✓	✓
Hydroquinine	✓	✓	✓
Hydroxybutyric Acid	✓	✓	✓
Hydroxyethylpromethazine	✓	✓	✓
Hymecromone	✓	✓	✓
Ibandronic Acid	✓	✓	✓
Ibudilast	✓	✓	✓
Ibutilide	✓	✓	✓
Idanpramine	✓	✓	✓
Ifenprodil	✓	✓	✓
Iloperidone	✓	✓	✓
Imidapril	✓	✓	✓
Imipramine	✓	✓	✓
Indapamide	✓	✓	✓
Indium (111In) Pentetic Acid	✓	✓	✓
Indoprofen	✓	✓	✓
Inosine Pranobex	✓	✓	✓
Insulin Glargine	✓	✓	✓

	Effect	Breakdown	Dose
Guanoxabenz	✓	✓	✓
Halazepam	✓	✓	✓
Halometasone	✓	✓	✓
Hematin	✓	✓	✓
Hetacillin	✓	✓	✓
Hexetidine	✓	✓	✓
Hexocyclium	✓	✓	✓
Hidrosmin	✓	✓	✓
Homatropine	✓	✓	✓
Hydrochlorothiazide	✓	✓	✓
Hydrocortisone Aceponate	✓	✓	✓
Hydroflumethiazide	✓	✓	✓
Hydroquinone	✓	✓	✓
Hydroxycarbamide	✓	✓	✓
Hydroxyprogesterone	✓	✓	✓
Hyoscyamine	✓	✓	✓
Ibopamine	✓	✓	✓
Ibuprofen	✓	✗	✗
Icatibant	✓	✓	✓
Idarubicin	✓	↓	↓
Ifn-A2A/B	✓	✓	✓
Iloprost	✓	✓	✓
Imidazole Salicylate	✓	✓	✓
Imolamine	✓	✓	✓
Indigo Carmine	✓	✓	✓
Indobufen	✓	✓	✓
Indoramin	✓	✓	✓
Inositol Nicotinate	✓	✓	✓
Insulin Lispro	✓	✓	✓

	Effect	Breakdown	Dose
Insulindetemir	✓	✓	✓
Iodine Iofetamine (123I)	✓	✓	✓
Iodocholesterol (131I)	✓	✓	✓
Ipriflavone	✓	✓	✓
Iproniazide	✓	✓	✓
Isepamicin	✓	✓	✓
Isocarboxazid	✓	✓	✓
Isometheptene	✓	✓	✓
Isopropamide	✓	✓	✓
Isoxsuprine	✓	✓	✓
Itramin Tosilate	✓	✓	✓
Ixabepilone	✓	✓	✓
Kaolin	✓	✓	✓
Ketanserin	✓	✓	✓
Ketoprofen	✓	✓	✓
Krypton (81Mkr) Gas	✓	✓	✓
Lacosamide	✓	✓	✓
Lafutidine	✓	✓	✓
Lanatoside C	✓	✓	✓
Lanthanum Carbonate	✓	✓	✓
Latamoxef	✓	✓	✓
Lenalidomide	✓	✓	✓
Lercanidipine	✓	↑	↑
Leuprorelin	✓	✓	✓
Levetiracetam	✓	✓	✓
Levocarnitine	✓	✓	✓
Levodropropizine	✓	✓	✓
Levomepromazine	✓	✓	✓
Levosimendan	✓	✓	✓

	Effect	Breakdown	Dose
Insulinglulisin	✓	✓	✓
Iodine Ioflupane (123I)	✓	✓	✓
Ipratropiumbromid	✓	✓	✓
Iprindole	✓	✓	✓
Irbesartan	✓	✗	✗
Isoaminile	✓	✓	✓
Isoetarine	✓	✓	✓
Isoniazid	✓	✗	✗
Isosorbide Dinitrate	✓	✓	✓
Isradipine	✓	↑	↑
Ivabradine	✓	↑	↑
Josamycin	✓	✓	✓
Kebuzone	✓	✓	✓
Ketazolam	✓	✓	✓
Ketorolac	✓	✓	✓
Labetalol	✓	✓	✓
Lactitol	✓	✓	✓
Lamivudine	✓	✓	✓
Lanreotide	✓	✓	✓
Lapatinib	✓	↑	↑
Latanoprost	✓	✓	✓
Lentinan	✓	✓	✓
Letosteine	✓	✓	✓
Levacetylmethadol	✓	↑	↑
Levobunolol	✓	✓	✓
Levocetirizine	✓	✓	✓
Levofloxacin	✓	✓	✓
Levomethadone	✓	✓	✓
Levosulpiride	✓	✓	✓

	Effect	Breakdown	Dose
Iodine (131I) Norcholesterol	✓	✓	✓
Iodine Iolopride (123I)	✓	✓	✓
Iprazochrome	✓	✓	✓
Iproclozide	✓	✓	✓
Irinotecan	✓	↑	↓
Isobromindione	✓	✓	✓
Isoflurane	✓	✓	✓
Isoprenaline	✓	✓	✓
Isosorbide Mononitrate	✓	✓	✓
Itraconazole	✓	↑	↑
Ivermectin	✓	↑	↑
Kanamycin	✓	✓	✓
Ketamine	✓	↓	↓
Ketobemidone	✓	↓	↓
Ketotifen	✓	✓	✓
Lacidipine	✓	↑	↑
Lactulose	✓	✓	✓
Lamotrigine	✓	✓	✓
Lansoprazole	✓	↑	✓
Lasofoxifene	✓	✓	✓
Leflunomide	✓	✗	✗
Lepirudin	✓	✓	✓
Letrozole	✓	↑	↑
Levamisole	✓	✓	✓
Levobupivacaine	✓	↑	↑
Levodopa	✓	✓	✓
Levoglutamide	✓	✓	✓
Levonorgestrel	✓	↑	↑
Levothyroxine Sodium	✓	✓	✓

	Effect	Breakdown	Dose
Levoverbenone	✓	✓	✓
Linagliptin	✓	✓	✓
Linezolid	✓	✓	✓
Liothyronine Sodium	✓	✓	✓
Lisinopril	✓	✓	✓
Lodoxamide	✓	✓	✓
Lomefloxacin	✓	✓	✓
Lonidamine	✓	✓	✓
Lopinavir	✓	↑	↑
Lorajmine	✓	✓	✓
Lorcainide	✓	✓	✓
Losartan	✗	↓	✗
Loxapine	✓	✓	✓
Lymecycline	✓	✓	✓
Macrogol	✓	✓	✓
Magnesium Peroxide	✓	✓	✓
Malathion	✓	✓	✓
Mannosulfan	✓	✓	✓
Maribavir	✓	✓	✓
Mazindol	✓	✓	✓
Mebhydrolin	✓	✓	✓
Mecamylamine	✓	✓	✓
Meclofenoxate	✓	✓	✓
Medifoxamine	✓	✓	✓
Medrysone	✓	✓	✓
Mefloquine	✓	↑	↑
Meglumine Antimonate	✓	✓	✓
Melagatran	✓	✓	✓
Melevodopa	✓	✓	✓

	Effect	Breakdown	Dose
Lidocain	✓	✓	✓
Lincomycin	✓	✓	✓
Linopirdine	✓	↑	↑
Liraglutide	✓	✓	✓
Lisuride	✓	↑	↑
Lofepramine	✓	✓	✓
Lomustine	✓	✓	✓
Loperamide	✓	✓	✓
Loprazolam	✓	✓	✓
Loratadine	✓	↑	↑
Lormetazepam	✓	✓	✓
Loteprednol	✓	✓	✓
Lubiprostone	✓	✓	✓
Lynestrenol	✓	✓	✓
Magaldrate	✓	✓	✓
Magnesium Phosphate	✓	✓	✓
Mandelic Acid	✓	✓	✓
Maprotiline	✓	✓	✓
Masoprocol	✓	✓	✓
Mebendazole	✓	✓	✓
Mebutamate	✓	✓	✓
Mecillinam	✓	✓	✓
Meclozin	✓	✓	✓
Medrogestone	✓	✓	✓
Mefenamic Acid	✓	✗	✗
Mefruside	✓	✓	✓
Meglutol	✓	✓	✓
Melarsoprol	✓	✓	✓
Melitracen	✓	✓	✓

	Effect	Breakdown	Dose
Lidoflazine	✓	✓	✓
Lindane	✓	✓	✓
Linsidomine	✓	✓	✓
Lisdexamfetamine	✓	✓	✓
Lithium Succinate	✓	✓	✓
Lofexidine	✓	✓	✓
Lonazolac	✓	✓	✓
Loperamide Oxide	✓	✓	✓
Loracarbef	✓	✓	✓
Lorazepam	✓	✓	✓
Lornoxicam	✓	✗	✗
Lovastatin	✓	↑	↑
Lumiracoxib	✓	↓	↓
Lypressin	✓	✓	✓
Magnesium Oxide	✓	✓	✓
Magnesiumsilicate	✓	✓	✓
Manidipine	✓	✓	✓
Maraviroc	✓	✓	✓
Mazaticol	✓	✓	✓
Mebeverine	✓	✓	✓
Mebutizide	✓	✓	✓
Meclofenamic Acid	✓	✓	✓
Medazepam	✓	✓	✓
Medroxyprogesterone	✓	↑	↑
Mefenorex	✓	✓	✓
Megestrol	✓	✓	✓
Meladrazine	✓	✓	✓
Melatonin	✓	✓	✓
Meloxicam	✓	✗	✗

	Effect	Breakdown	Dose
Melperone	✓	✓	✓
Mepacrine	✓	↑	↑
Mephenesin	✓	✓	✓
Mephenytoin	✓	✓	✓
Mepixanox	✓	✓	✓
Meprobamate	✓	✓	✓
Mequinol	✓	✓	✓
Mercaptopurine	✓	✓	✓
Mesalazine	✓	✓	✓
Mesterolone	✓	✓	✓
Metacycline	✓	✓	✓
Metampicillin	✓	✓	✓
Metenolone	✓	✓	✓
Methadone	✓	↑	↑
Methapyrilene	✓	✓	✓
Methazolamide	✓	✓	✓
Methionine	✓	✓	✓
Methohexital	✓	✓	✓
Methoxamine	✓	✓	✓
Methyclothiazide	✓	✓	✓
Methylcellulose	✓	✓	✓
Methylestrenolone	✓	✓	✓
Methylphenidate	✓	✓	✓
Methylprednisolone Aceponate	✓	✓	✓
Methyltestosterone	✓	✓	✓
Methyprylon	✓	✓	✓
Meticrane	✓	✓	✓
Metirosine	✓	✓	✓
Metoclopramide	✓	✓	✓

	Effect	Breakdown	Dose
Melphalan	✓	✓	✓
Mepartricin	✓	✓	✓
Mephenoxalone	✓	✓	✓
Mepindolol	✓	✓	✓
Mepolizumab	✓	✓	✓
Meprotixol	✓	✓	✓
Mequitazine	✓	✓	✓
Meropenem	✓	✓	✓
Mesna	✓	✓	✓
Mesuximide	✓	✓	✓
Metahexamide	✓	✓	✓
Metandienone	✓	✓	✓
Metergoline	✓	✓	✓
Methallenestril	✓	✓	✓
Methaqualone	✓	✓	✓
Methdilazine	✓	✓	✓
Methiosulfonium Chloride	✓	✓	✓
Methoserpidine	✓	✓	✓
Methoxyflurane	✓	✓	✓
Methyl Aminolevulinate	✓	✓	✓
Methyldopa (Levorotatory)	✓	✓	✓
Methylnaltrexone Bromide	✓	✓	✓
Methylphenobarbital	✓	✓	✓
Methylpropylpropanediol Dinitrate	✓	✓	✓
Methylthioninium Chloride	✓	✓	✓
Methysergide	✓	✓	✓
Metildigoxin	✓	✓	✓
Metisazone	✓	✓	✓
Metolazone	✓	✓	✓

	Effect	Breakdown	Dose
Memantine	✓	✓	✓
Mepenzolate	✓	✓	✓
Mephentermine	✓	✓	✓
Mepivacaine	✓	✓	✓
Meprednisone	✓	✓	✓
Meptazinol	✓	✓	✓
Mercaptamine	✓	✓	✓
Mersalyl	✓	✓	✓
Mesoridazine	✓	✓	✓
Metabutethamine	✓	✓	✓
Metamizole Sodium	✓	✓	✓
Metaraminol	✓	✓	✓
Metformin	↓	✓	✓
Methantheline	✓	✓	✓
Metharbital	✓	✓	✓
Methenamine	✓	✓	✓
Methocarbamol	✓	✓	✓
Methotrexate	✓	✓	✓
Methoxyphenamine	✓	✓	✓
Methylatropine	✓	✓	✓
Methylergometrine	✓	↑	↑
Methylpentynol	✓	✓	✓
Methylprednisolone	✓	↑	↑
Methylscopolamine	✓	✓	✓
Methylthiouracil	✓	✓	✓
Meticillin	✓	✓	✓
Metipranolol	✓	✓	✓
Metixene	✓	✓	✓
Metopimazine	✓	✓	✓

	Effect	Breakdown	Dose
Metoprolol	✓	✓	✓
Metyrapone	✓	✓	✓
Mianserin	✓	✓	✓
Miconazole	✓	↑	↑
Midecamycin	✓	✓	✓
Mifepristone	✓	↑	↑
Milnacipran	✓	✓	✓
Minaprine	✓	✓	✓
Miocamycin	✓	✓	✓
Misoprostol	✓	✓	✓
Mitoguazone	✓	✓	✓
Mitoxantrone	✓	✓	✓
Moclobemide	✓	✓	✓
Mofebutazone	✓	✓	✓
Mometasone	✓	↑	↑
Monoxerutin	✓	✓	✓
Moracizine	✓	✓	✓
Morniflumate	✓	✓	✓
Morpholine Salicylate	✓	✓	✓
Moxestrol	✓	✓	✓
Moxonidine	✓	✓	✓
Mycophenolic Acid	✓	↑	↑
Nabumetone	✓	✓	✓
Naftazone	✓	✓	✓
Nalfurafine	✓	✓	✓
Naloxone	✓	✓	✓
Naproxcinod	✓	✓	✓
Narcobarbital	✓	✓	✓
Natriumhypochlorit	✓	✓	✓

	Effect	Breakdown	Dose
Metrifonate	✓	✓	✓
Mexiletine	✓	✓	✓
Mibefradil	✓	✓	✓
Micronomicin	✓	✓	✓
Midodrine	✓	✓	✓
Miglitol	✓	✓	✓
Milrinone	✓	✓	✓
Minocycline	✓	✓	✓
Mipomersen	✓	✓	✓
Mitiglinide	✓	✓	✓
Mitomycin	✓	✓	✓
Mivacurium Chloride	✓	✓	✓
Modafinil	✓	↑	↑
Molindone	✓	✓	✓
Monobenzone	✓	✓	✓
Montelukast	✓	↓	↓
Morclofone	✓	✓	✓
Moroxydine	✓	✓	✓
Mosapramine	✓	✓	✓
Moxifloxacin	✓	✓	✓
Muronomab	✓	✓	✓
Myristyl-Benzalkonium	✓	✓	✓
Nadolol	✓	✓	✓
Naftidrofuryl	✓	✓	✓
Nalidixic Acid	✓	✓	✓
Naltrexone	✓	✓	✓
Naproxen	✓	✗	✗
Natamycin	✓	✓	✓
Natriumpentosanpolysulfat	✓	✓	✓

	Effect	Breakdown	Dose
Metronidazole	✓	✓	✓
Mezlocillin	✓	✓	✓
Micafungin	✓	✓	✓
Midazolam	↑	↑	↓
Mifamurtide	✓	✓	✓
Miglustat	✓	✓	✓
Miltefosine	✓	✓	✓
Minoxidil	✓	✓	✓
Mirtazapine	✓	✓	✓
Mitobronitol	✓	✓	✓
Mitotane	✓	✓	✓
Mizolastine	✓	↑	↑
Moexipril	✓	✓	✓
Molsidomine	✓	✓	✓
Monoethanolamine Oleate	✓	✓	✓
Moperone	✓	✓	✓
Morinamide	✓	✓	✓
Morphine	✓	✓	✓
Moxaverine	✓	✓	✓
Moxisylyte	✓	✓	✓
Muzolimine	✓	✓	✓
Nabilone	✓	✓	✓
Nafarelin	✓	✓	✓
Nalbuphine	✓	✓	✓
Nalorphine	✓	✓	✓
Nandrolone	✓	✓	✓
Naratriptan	✓	✓	✓
Nateglinide	✓	↓	↓
Nebivolol	✓	✓	✓

	Effect	Breakdown	Dose
Nefazodone	✓	↑	↑
Nelfinavir	✓	↑	↑
Neostigmine	✓	✓	✓
Nesiritide	✓	✓	✓
Nialamide	✓	✓	✓
Nicergoline	✓	✓	✓
Nicofetamide	✓	✓	✓
Nicorandil	✓	✓	✓
Nifedipine	✓	↑	↑
Nifuroxazide	✓	✓	✓
Nifurzide	✓	✓	✓
Nilutamide	✓	✓	✓
Nimodipine	✓	↑	↑
Niperotidine	✓	✓	✓
Nitazoxanide	✓	✓	✓
Nitrendipine	✓	↑	↑
Nitroprusside	✓	✓	✓
Nizofenone	✓	✓	✓
Nordazepam	✓	✓	✓
Norethisterone	✓	↑	↑
Norgestrienone	✓	✓	✓
Noscapine	✓	✓	✓
Obidoxime	✓	✓	✓
Ofloxacin	✓	✓	✓
Oleandomycin	✓	✓	✓
Olsalazine	✓	✓	✓
Omeprazole	✓	✓	✓
Orciprenalin	✓	✓	✓
Ornidazole	✓	↑	↑

	Effect	Breakdown	Dose
Nefopam	✓	✓	✓
Neltenexine	✓	✓	✓
Nepafenac	✓	✓	✓
Netilmicin	✓	✓	✓
Niaprazine	✓	✓	✓
Niceritrol	✓	✓	✓
Nicofuranose	✓	✓	✓
Nicotinic Acid	✓	✓	✓
Niflumic Acid	✓	✓	✓
Nifurtimox	✓	✓	✓
Nikethamide	✓	✓	✓
Nilvadipine	✓	✓	✓
Nimorazole	✓	✓	✓
Niridazole	✓	✓	✓
Nitisinone	✓	✓	✓
Nitrofural	✓	✓	✓
Nitroxoline	✓	✓	✓
Nomegestrol	✓	✓	✓
Norepinephrine	✓	✓	✓
Norfenefrine	✓	✓	✓
Normethadone	✓	✓	✓
Noxytiolin	✓	✓	✓
Octopamine	✓	↑	↑
Olaflur	✓	✓	✓
Olmesartan Medoxomil	✓	✓	✓
Omacetaxine Mepesuccinate	✓	✓	✓
Ondansetron	✓	↑	✓
Oritavancin	✓	✓	✓
Ornipressin	✓	✓	✓

	Effect	Breakdown	Dose
Nelarabine	✓	✓	✓
Neomycin	✓	✓	✓
Nepinalone	✓	✓	✓
Nevirapine	✓	↑	↑
Nicardipine	✓	↑	↑
Niclosamide	✓	✓	✓
Nicomorphine	✓	✓	✓
Nicotinyl Alcohol (Pyridylcarbinol)	✓	✓	✓
Nifuratel	✓	✓	✓
Nifurtoinol	✓	✓	✓
Nilotinib	✓	↑	↑
Nimesulide	✓	✗	✗
Nimustine	✓	✓	✓
Nisoldipine	✓	↑	↑
Nitrazepam	✓	✓	✓
Nitrofurantoin	✓	✓	✓
Nizatidine	✓	✓	✓
Nomifensine	✓	✓	✓
Norethandrolone	✓	✓	✓
Norfloxacin	✓	✓	✓
Nortriptyline	✓	✓	✓
Nystatin	✓	✓	✓
Octreotide	✓	✓	✓
Olanzapine	✓	✓	✓
Olopatadine	✓	✓	✓
Omalizumab	✓	✓	✓
Opipramol	✓	✓	✓
Orlistat	✓	✓	✓
Ornithine Oxoglurate	✓	✓	✓

	Effect	Breakdown	Dose
Orphenadrine (Citrate)	✓	✓	✓
Oxabolone Cipionate	✓	✓	✓
Oxaflozane	✓	✓	✓
Oxamniquine	✓	✓	✓
Oxaprozin	✓	✗	✗
Oxcarbazepine	✓	✓	✓
Oxetacaine	✓	✓	✓
Oxitriptan	✓	✓	✓
Oxolinic Acid	✓	✓	✓
Oxybutynin	✓	↑	↑
Oxyfedrine	✓	✓	✓
Oxyphenbutazone	✓	✓	✓
Oxyphenonium	✓	✓	✓
Paclitaxel	✓	✓	✓
Palonosetron	✓	✓	✓
Pancuronium	✓	✓	✓
Papaveretum	✓	✓	✓
Paraldehyde	✓	✓	✓
Paraoxon	✓	↑	↑
Pargyline	✓	✓	✓
Paroxetine	✓	✓	✓
Pefloxacin	✓	✓	✓
Penamecillin	✓	✓	✓
Pengitoxin	✓	✓	✓
Pentaerithrityl	✓	✓	✓
Pentamidine Isethionate	✓	✓	✓
Pentetrazol	✓	✓	✓
Pentobarbital	✓	✓	✓
Pentoxyverine	✓	✓	✓

	Effect	Breakdown	Dose
Oseltamivir	✓	✓	✓
Oxaceprol	✓	✓	✓
Oxaliplatin	✓	✓	✓
Oxandrolone	✓	✓	✓
Oxatomide	✓	✓	✓
Oxedrine	✓	✓	✓
Oxetorone	✓	✓	✓
Oxitropium Bromide	✓	✓	✓
Oxomemazine	✓	✓	✓
Oxycinchophen	✓	✓	✓
Oxymetholone	✓	✓	✓
Oxyphencyclimine	✓	✓	✓
Oxyquinoline	✓	✓	✓
Paclitaxel Poliglumex	✓	✓	✓
Pamidronic Acid	✓	✓	✓
Panobinostat	✓	✓	✓
Papaverine	✓	✓	✓
Paramethadione	✓	✓	✓
Parathyroid Hormone	✓	✓	✓
Paricalcitol	✓	✓	✓
Pazopanib	✓	✓	✓
Pemetrexed	✓	✓	✓
Penbutolol	✓	✓	✓
Penicillamine	✓	✓	✓
Pentaerithrityl Tetranitrate	✓	✓	✓
Pentamycin	✓	✓	✓
Penthienate	✓	✓	✓
Pentostatin	✓	✓	✓
Perampanel	✓	✓	✓

	Effect	Breakdown	Dose
Otilonium Bromide	✓	✓	✓
Oxacillin	✓	✓	✓
Oxametacin	✓	✓	✓
Oxantel	✓	✓	✓
Oxazepam	✓	↑	↑
Oxeladin	✓	✓	✓
Oxiracetam	✓	✓	✓
Oxolamine	✓	✓	✓
Oxprenolol	✓	✓	✓
Oxycodone	✓	↑	✓
Oxypertine	✓	✓	✓
Oxyphenisatine	✓	✓	✓
Oxytocin	✓	✓	✓
Paliperidone	✓	✓	✓
Pancreozymin (Cholecystokinin)	✓	✓	✓
Pantoprazole	✓	✓	✓
Paracetamol	✓	✓	✓
Paramethasone	✓	✓	✓
Parecoxib	✓	✓	✓
Paromomycin	✓	✓	✓
Pazufloxacin	✓	✓	✓
Pemoline	✓	✓	✓
Penfluridol	✓	✓	✓
Penimepicycline	✓	✓	✓
Pentagastrin	✓	✓	✓
Pentazocine	✓	✓	✓
Pentifylline	✓	✓	✓
Pentoxifylline	✓	✓	✓
Perazine	✓	✓	✓

	Effect	Breakdown	Dose
Pergolide	✓	↑	↑
Perindopril	✓	✓	✓
Peruvoside	✓	✓	✓
Phenacemide	✓	✓	✓
Phenazone	✓	✓	✓
Pheneticillin	✓	✓	✓
Phenglutarimide	✓	✓	✓
Phenobarbital	✓	✓	✓
Phenolsulfonphthalein	✓	✓	✓
Phenoxybenzamine	✓	✓	✓
Phenprocoumon	✓	↓	✓
Phentolamine	✓	✓	✓
Phenylephrine	✓	✓	✓
Pholcodine	✓	✓	✓
Picloxydine	✓	✓	✓
Pilocarpine	✓	✓	✓
Pimozide	✓	↑	↑
Pinazepam	✓	✓	✓
Pipamperone	✓	✓	✓
Pipemidic Acid	✓	✓	✓
Piperazine	✓	✓	✓
Pipobroman	✓	✓	✓
Piprozolin	✓	✓	✓
Pirbuterol	✓	✓	✓
Pirfenidone	✓	✓	✓
Piritramide	✓	✓	✓
Pirprofen	✓	✓	✓
Pivampicillin	✓	✓	✓
Pizotifen	✓	✓	✓

	Effect	Breakdown	Dose
Perhexiline	✓	✓	✓
Permethrin	✓	✓	✓
Pethidine	✓	✓	✓
Phenacetin	✓	✓	✓
Phenazopyridine	✓	✓	✓
Pheneturide	✓	✓	✓
Phenindamine	✓	✓	✓
Phenol	✓	✓	✓
Phenoperidine	✓	✓	✓
Phenoxymethylpenicillin	✓	✓	✓
Phensuximide	✓	✓	✓
Phenyl Salicylate	✓	✓	✓
Phenytoin	✓	✗	↓
Phthalylsulfathiazole	✓	✓	✓
Picotamide	✓	✓	✓
Pimecrolimus	✓	↑	↑
Pinacidil	✓	✓	✓
Pindolol	✓	✓	✓
Pipazetate	✓	✓	✓
Pipenzolate	✓	✓	✓
Piperidione	✓	✓	✓
Pipotiazine	✓	✓	✓
Piracetam	✓	✓	✓
Pirenzepine	✓	✓	✓
Piribedil	✓	✓	✓
Piromidic Acid	✓	✓	✓
Pitavastatin	✓	✓	✓
Pivmecillinam	✓	✓	✓
Pleconaril	✓	✓	✓

	Effect	Breakdown	Dose
Periciazine	✓	✓	✓
Perphenazine	✓	✓	✓
Phanquinone	✓	✓	✓
Phenazocine	✓	✓	✓
Phenelzine	✓	↑	↑
Phenformin	✓	✓	✓
Phenindione	✓	✓	✓
Phenolphthalein	✓	✓	✓
Phenothrin	✓	✓	✓
Phenprobamate	✓	✓	✓
Phentermine	✓	✓	✓
Phenylbutazone	✓	↑	↑
Phloroglucinol	✓	✓	✓
Physostigmine	✓	✓	✓
Pidotimod	✓	✓	✓
Pimethixene	✓	✓	✓
Pinaverium	✓	✓	✓
Pioglitazone	✓	↓	↓
Pipecuronium Bromide	✓	✓	✓
Piperacillin	✓	✓	✓
Piperidolate	✓	✓	✓
Pipradrol	✓	✓	✓
Pirarubicin	✓	✓	✓
Piretanide	✓	✓	✓
Pirisudanol	✓	✓	✓
Piroxicam	✓	✗	✗
Pivagabine	✓	✓	✓
Pixantrone	✓	✓	✓
Plerixafor	✓	✓	✓

	Effect	Breakdown	Dose
Plicamycin	✓	✓	✓
Poly I:C	✓	✓	✓
Polymyxin B	✓	✓	✓
Polythiazide	✓	✓	✓
Potassium Canrenoate	✓	✓	✓
Potassium Lactate	✓	✓	✓
Potassium Salicylate	✓	✓	✓
Pralatrexate	✓	✓	✓
Pramiracetam	✓	✓	✓
Pranoprofen	✓	✓	✓
Pravastatin	✓	✓	✓
Prazosin	✓	✓	✓
Prednisolone	✓	↑	↑
Pregabalin	✓	✓	✓
Prenylamine	✓	✓	✓
Prifinium Bromide	✓	✓	✓
Primidone	✓	✓	✓
Procainamide	✓	✓	✓
Procarbazine	✓	✓	✓
Procyclidine	✓	✓	✓
Progesterone	✓	↑	↑
Proguanil	✓	✓	✓
Promegestone	✓	✓	✓
Propafenone	✓	✓	✓
Propatylnitrate	✓	✓	✓
Propicillin	✓	✓	✓
Propofol	✓	✗	✗
Propyphenazone	✓	✓	✓
Prothipendyl	✓	✓	✓

	Effect	Breakdown	Dose
Poldine	✓	✓	✓
Poly Iclc	✓	✓	✓
Polynoxylin	✓	✓	✓
Porfimer Sodium	✓	✓	✓
Potassium Clorazepate	✓	✓	✓
Potassium Perchlorate	✓	✓	✓
Practolol	✓	✓	✓
Pralidoxime	✓	✓	✓
Pramocaine	✓	✓	✓
Prasterone	✓	✓	✓
Prazepam	✓	↑	↑
Prednicarbate	✓	✓	✓
Prednisone	✓	↑	↑
Prenalterol	✓	✓	✓
Prethcamide	✓	✓	✓
Prilocaine	✓	✓	✓
Probenecid	✓	✓	✓
Procaine	✓	✓	✓
Procaterol	✓	✓	✓
Profenamine	✓	✓	✓
Proglumetacin	✓	✓	✓
Prolintane	✓	✓	✓
Promestriene	✓	✓	✓
Propanidid	✓	✓	✓
Propenidazole	✓	✓	✓
Propiomazine	✓	✓	✓
Propranolol	✓	✓	✓
Proquazone	✓	✓	✓
Protiofate	✓	✓	✓

	Effect	Breakdown	Dose
Polidocanol	✓	✓	✓
Polyestradiol Phosphate	✓	✓	✓
Polystyrene Sulfonate	✓	✓	✓
Posaconazole	✓	✓	✓
Potassium Iodide	✓	✓	✓
Potassium Polysulfide	✓	✓	✓
Prajmaline	✓	✓	✓
Pramipexole	✓	✓	✓
Pranlukast	✓	✓	✓
Prasugrel	✓	✓	✓
Praziquantel	✓	↑	↑
Prednimustine	✓	✓	✓
Prednylidene	✓	✓	✓
Prenoxdiazine	✓	✓	✓
Pridinol	✓	✓	✓
Primaquine	✓	↑	↑
Probucol	✓	✓	✓
Procaine Benzylpenicillin	✓	✓	✓
Prochlorperazine	✓	✓	✓
Progabide	✓	✓	✓
Proglumide	✓	✓	✓
Promazine	✓	✓	✓
Propacetamol	✓	✓	✓
Propantheline	✓	✓	✓
Propentofylline	✓	✓	✓
Propiverine	✓	✓	✓
Propylthiouracil	✓	✓	✓
Proscillaridin	✓	✓	✓
Protionamide	✓	✓	✓

	Effect	Breakdown	Dose
Protirelin	✓	✓	✓
Proxibarbal	✓	✓	✓
Prucalopride	✓	✓	✓
Pyrantel	✓	✓	✓
Pyridostigmine	✓	✓	✓
Pyrithyldione	✓	✓	✓
Pyrvinium	✓	✓	✓
Quinagolide	✓	✓	✓
Quinethazone	✓	✓	✓
Quinupramine	✓	✓	✓
Racecadotril	✓	✓	✓
Raltitrexed	✓	✓	✓
Ranimustine	✓	✓	✓
Ranolazine	✓	✓	✓
Regadenoson	✓	✓	✓
Remoxipride	✓	✓	✓
Reproterol	✓	✓	✓
Reteplase	✓	✓	✓
Ribavirin	✓	✓	✓
Rifampicin	✓	↓	↓
Rifaximin	✓	✓	✓
Riluzole	✓	✓	✓
Rimexolone	✓	✓	✓
Risedronic Acid	✓	✓	✓
Ritonavir	✓	↑	↑
Rizatriptan	✓	✓	✓
Rofecoxib	✓	↓	↓
Rolitetracycline	✓	✓	✓
Ropinirole	✓	✓	✓

	Effect	Breakdown	Dose
Protriptyline	✓	✓	✓
Proxymetacaine	✓	✓	✓
Prulifloxacin	✓	✓	✓
Pyrazinamide	✓	✓	✓
Pyrimethamine	✓	✓	✓
Pyritinol	✓	✓	✓
Quazepam	✓	✓	✓
Quinapril	✓	✓	✓
Quingestanol	✓	✓	✓
Quinupristin/Dalfopristin	✓	✓	✓
Raloxifene	✓	✓	✓
Ramelteon	✓	✓	✓
Ranitidine	✓	✓	✓
Rasagiline	✓	✓	✓
Remifentanil	✓	✓	✓
Repaglinide	✓	↑	↑
Rescinnamine	✓	✓	✓
Retigabine	✓	✓	✓
Ribostamycin	✓	✓	✓
Rifamycin	✓	✓	✓
Rilmenidine	✓	✓	✓
Rimantadine	✓	✓	✓
Rimiterol	✓	✓	✓
Risperidone	✓	✓	✓
Rituximab	✓	✓	✓
Rociverine	✓	✓	✓
Roflumilast	✓	✓	✓
Romidepsin	✓	✓	✓
Ropivacaine	✓	✓	✓

	Effect	Breakdown	Dose
Proxazole	✓	✓	✓
Proxyphylline	✓	✓	✓
Prussian Blue	✓	✓	✓
Pyrethrum	✓	✓	✓
Pyrithione Zinc	✓	✓	✓
Pyrrobutamine	✓	✓	✓
Quetiapine	✓	↑	↑
Quinbolone	✓	✓	✓
Quinidine	✓	↑	↑
Rabeprazole	✓	↑	↑
Raltegravir	✓	✓	✓
Ramipril	✓	✓	✓
Ranitidine Bismuth Citrate	✓	✓	✓
Reboxetine	✓	↑	↑
Remikiren	✓	✓	✓
Reposal	✓	✓	✓
Reserpine	✓	✓	✓
Rhenium (186Re) Etidronic Acid	✓	✓	✓
Rifabutin	✓	↑	↑
Rifapentine	✓	✓	✓
Rilpivirine	✓	✓	✓
Rimazolium	✓	✓	✓
Rimonabant	✓	↑	↑
Ritodrine	✓	✓	✓
Rivastigmine	✓	✓	✓
Rocuronium Bromide	✓	✓	✓
Rokitamycin	✓	✓	✓
Ronifibrate	✓	✓	✓
Roquinimex	✓	✓	✓

	Effect	Breakdown	Dose
Rose Bengal Sodium	✓	✓	✓
Rosuvastatin	✓	✓	✓
Roxithromycin	✓	↑	↑
Rupatadine	✓	✓	✓
Sacrosidase	✓	✓	✓
Salmeterol	✓	↑	↑
Sapropterin	✓	✓	✓
Saxagliptin	✓	✓	✓
Secobarbital	✓	✓	✓
Selegiline	✓	✗	✗
Semustine	✓	✓	✓
Sermorelin	✓	✓	✓
Sevelamer	✓	✓	✓
Sildenafil	✓	↑	↑
Simfibrate	✓	✓	✓
Sirolimus	✓	↑	↑
Sitagliptin	✓	✓	✓
Sodium Acetate	✓	✓	✓
Sodium Aurotiosulfate	✓	✓	✓
Sodium Chloride, Hypertonic	✓	✓	✓
Sodium Feredetate	✓	✓	✓
Sodium Glycerophosphate	✓	✓	✓
Sodium Iothalamate (125I)	✓	✓	✓
Sodium Perborate	✓	✓	✓
Sodium Picosulfate	✓	✓	✓
Sodium Stibogluconate	✓	✓	✓
Sodium Tetradecyl Sulfate	✓	✓	✓
Sorafenib	✓	↑	↑
Sparfloxacin	✓	✓	✓

	Effect	Breakdown	Dose
Rosiglitazone	✓	✗	✗
Rotigotine	✓	✓	✓
Rufinamide	✓	✓	✓
Rutoside	✓	✓	✓
Salbutamol	✓	↑	↑
Salsalate	✓	✓	✓
Saquinavir	✓	↑	↑
Scopolamine	✓	✓	✓
Secretin	✓	✓	✓
Selenium (75Se) Norcholesterol	✓	✓	✓
Senna Glycosides	✓	✓	✓
Sertindole	✓	↑	↑
Sevoflurane	✓	✓	✓
Silodosin	✓	✓	✓
Simvastatin	✓	↑	✗
Sisomicin	✓	✓	✓
Sitaxentan	✓	✓	✓
Sodium Aminosalicylate	✓	✓	✓
Sodium Bicarbonate	✓	✓	✓
Sodium Citrate	✓	✓	✓
Sodium Fluoride	✓	✓	✓
Sodium Iodide (123I)	✓	✓	✓
Sodium Monofluorophosphate	✓	✓	✓
Sodium Phenylbutyrate	✓	✓	✓
Sodium Propionate	✓	✓	✓
Sodium Sulfate	✓	✓	✓
Somatorelin	✓	✓	✓
Sorbitol	✓	✓	✓
Sparteine	✓	✓	✓

	Effect	Breakdown	Dose
Rosoxacin	✓	✓	✓
Roxatidine	✓	✓	✓
Rufloxacin	✓	✓	✓
Saccharated Iron Oxide	✓	✓	✓
Salicylamide	✓	✓	✓
Samarium (153Sm) Lexidronam	✓	✓	✓
Satraplatin	✓	✓	✓
Secnidazole	✓	✓	✓
Sedalipid	✓	✓	✓
Selenium (75Se) Tauroselcholic Acid	✓	✓	✓
Seratrodast	✓	↓	↓
Sertraline	✓	✓	✓
Sibutramine	✓	↑	↑
Silymarin	✓	✓	✓
Sincalide	✓	✓	✓
Sitafloxacin	✓	✓	✓
Sobrerol	✓	✓	✓
Sodium Aurothiomalate	✓	✓	✓
Sodium Borate	✓	✓	✓
Sodium Edetate	✓	✓	✓
Sodium Folinate	✓	✓	✓
Sodium Iodohippurate (123I)	✓	✓	✓
Sodium Nitrite	✓	✓	✓
Sodium Phosphate	✓	✓	✓
Sodium Salicylate	✓	✓	✓
Sodium Tartrate	✓	✓	✓
Somatostatin	✓	✓	✓
Sotalol	✓	✓	✓
Spectinomycin	✓	✓	✓

	Effect	Breakdown	Dose
Spiramycin	✓	↑	↑
Stannous Fluoride	✓	✓	✓
Stepronin	✓	✓	✓
Streptoduocin	✓	✓	✓
Streptozocin	✓	✓	✓
Styramate	✓	✓	✓
Sucralfate	✓	✓	✓
Sulbenicillin	✓	✓	✓
Sulfadicramide	✓	✓	✓
Sulfafurazole	✓	↓	↓
Sulfalene	✓	✓	✓
Sulfamethoxazole	✓	✗	✗
Sulfametoxydiazine	✓	✓	✓
Sulfaphenazole	✓	✓	✓
Sulfathiourea	✓	✓	✓
Sulfobromophthalein	✓	✓	✓
Sulpiride	✓	✓	✓
Sultiame	✓	✓	✓
Sunitinib	✓	↑	↑
Suxamethonium	✓	✓	✓
Tadalafil	✓	↑	↑
Talastine	✓	✓	✓
Tamoxifen	✗	✗	✓
Tasonermin	✓	✓	✓
Tazobactam	✓	✓	✓
Technetium (99Mtc) Disofenin	✓	✓	✓
Technetium (99Mtc) Furifosmin	✓	✓	✓
Technetium (99Mtc) Gluconate	✓	✓	✓
Technetium (99Mtc) Medronic Acid	✓	✓	✓

	Effect	Breakdown	Dose
Spirapril	✓	✓	✓
Stanozolol	✓	✓	✓
Stibophen	✓	✓	✓
Streptokinase	✓	✓	✓
Strontium (89Sr) Chloride	✓	✓	✓
Succinimide	✓	✓	✓
Sufentanil	✓	↑	↑
Sulfacetamide	✓	✓	✓
Sulfadimethoxine	✓	✓	✓
Sulfaguanidine	✓	✓	✓
Sulfamazone	✓	✓	✓
Sulfamethoxypyridazine	✓	✓	✓
Sulfamoxole	✓	✓	✓
Sulfapyridine	✓	✓	✓
Sulfatolamide	✓	✓	✓
Sulindac	✓	✓	✓
Sulprostone	✓	✓	✓
Sultopride	✓	✓	✓
Suprofen	✓	✗	✗
Tacrine	✓	✓	✓
Tafluprost	✓	✓	✓
Talbutal	✓	✓	✓
Tamsulosin	✓	↑	↑
Tasosartan	✓	✓	✓
Technetium (99Mtc) Bicisate	✓	✓	✓
Technetium (99Mtc) Etifenin	✓	✓	✓
Technetium (99Mtc) Galtifenin	✓	✓	✓
Technetium (99Mtc) Lidofenin	✓	✓	✓
Technetium (99Mtc) Mertiatide	✓	✓	✓

	Effect	Breakdown	Dose
Spironolactone	✓	✓	✓
Stavudine	✓	✓	✓
Stiripentol	✓	✓	✓
Streptomycin	✓	✓	✓
Strontium Ranelate	✓	✓	✓
Succinylsulfathiazole	✓	✓	✓
Sulbactam	✓	✓	✓
Sulfadiazine	✓	✗	✗
Sulfadimidine	✓	✓	✓
Sulfaisodimidine	✓	✓	✓
Sulfamethizole	✓	✓	✓
Sulfametomidine	✓	✓	✓
Sulfaperin	✓	✓	✓
Sulfasalazine	✓	✓	✓
Sulfinpyrazon	✓	✗	✗
Suloctidil	✓	✓	✓
Sultamicillin	✓	✓	✓
Sumatriptan	✓	✓	✓
Suramin Sodium	✓	✓	✓
Tacrolimus	✓	↑	↑
Talampicillin	✓	✓	✓
Talinolol	✓	✓	✓
Tapentadol	✓	✓	✓
Taurolidine	✓	✓	✓
Technetium (99Mtc) Butedronic Acid	✓	✓	✓
Technetium (99Mtc) Exametazime	✓	✓	✓
Technetium (99Mtc) Gluceptate	✓	✓	✓
Technetium (99Mtc) Mebrofenin	✓	✓	✓
Technetium (99Mtc) Oxidronic Acid	✓	✓	✓

	Effect	Breakdown	Dose
Technetium (99Mtc) Pertechnetate	✓	✓	✓
Technetium (99Mtc) Sestamibi	✓	✓	✓
Technetium (99Mtc) Tetrofosmin	✓	✓	✓
Teduglutide	✓	✓	✓
Teicoplanin	✓	✓	✓
Telbivudine	✓	✓	✓
Temafloxacin	✓	✓	✓
Temocillin	✓	✓	✓
Temsirolimus	✓	✓	✓
Tenitramine	✓	✓	✓
Tenoxicam	✓	✗	✗
Terconazole	✓	✓	✓
Teriparatide	✓	✓	✓
Terodiline	✓	✓	✓
Testosterone	✓	↑	↑
Tetracosactide	✓	✓	✓
Tetrazepam	✓	✓	✓
Thebacon	✓	✓	✓
Theophylline	✓	✓	✓
Thiazinam	✓	✓	✓
Thiopental	✓	✓	✓
Thioridazine	✓	✓	✓
Thiram	✓	✓	✓
Tiagabine	✓	↑	↑
Tiaprofenic Acid	✓	✓	✓
Tibolone	✓	✓	✓
Ticlopidine	✓	↑	↑
Tienilic Acid	✓	✗	✗
Tilidine	✓	✓	✓

	Effect	Breakdown	Dose
Technetium (99Mtc) Phytate	✓	✓	✓
Technetium (99Mtc) Succimer	✓	✓	✓
Teclozan	✓	✓	✓
Tegafur	✓	✗	✗
Telaprevir	✓	✓	✓
Telithromycin	✓	↑	↑
Temazepam	✓	✓	✓
Temoporfin	✓	✓	✓
Tenidap	✓	✓	✓
Tenofovir Disoproxil	✓	✓	✓
Terazosin	✓	✓	✓
Terfenadine	✓	↑	↑
Terizidone	✓	✓	✓
Tertatolol	✓	✓	✓
Tetrabenazine	✓	✓	✓
Tetracycline	✓	✓	✓
Thalidomide	✓	↓	↓
Theobromine	✓	✓	✓
Thiamazole	✓	✓	✓
Thiethylperazin	✓	✓	✓
Thiopropazate	✓	✓	✓
Thiosulfate	✓	✓	✓
Thymopentin	✓	✓	✓
Tianeptine	✓	✓	✓
Tiazofurine	✓	✓	✓
Ticagrelor	✓	✓	✓
Tidiacic Arginine	✓	✓	✓
Tigecycline	✓	✓	✓
Tiludronic Acid	✓	✓	✓

	Effect	Breakdown	Dose
Technetium (99Mtc) Pyrophosphate	✓	✓	✓
Technetium (99Mtc) Teboroxime	✓	✓	✓
Tedisamil	✓	✓	✓
Tegaserod	✓	✓	✓
Telavancin	✓	✓	✓
Telmisartan	✓	✓	✓
Temocapril	✓	✓	✓
Temozolomide	✓	✓	✓
Teniposide	✓	↑	↑
Tenonitrozole	✓	✓	✓
Terbutaline	✓	✓	✓
Terguride	✓	✓	✓
Terlipressin	✓	✓	✓
Tesamorelin	✓	✓	✓
Tetracaine	✓	✓	✓
Tetramethrin	✓	✓	✓
Thallium (201Tl) Chloride	✓	✓	✓
Theodrenaline	✓	✓	✓
Thiamphenicol	✓	✓	✓
Thiocolchicoside	✓	✓	✓
Thioproperazine	✓	✓	✓
Thiotepa	✓	✓	✓
Tiadenol	✓	✓	✓
Tiapride	✓	✓	✓
Tibezonium Iodide	✓	✓	✓
Ticarcillin	✓	✓	✓
Tiemonium Iodide	✓	✓	✓
Tilbroquinol	✓	✓	✓
Timepidium Bromide	✓	✓	✓

	Effect	Breakdown	Dose
Timolol	✓	✓	✓
Tioclomarol	✓	✓	✓
Tiopronin	✓	✓	✓
Tipepidine	✓	✓	✓
Tiratricol	✓	✓	✓
Tiropramide	✓	✓	✓
Tizanidine	✓	✓	✓
Tofisopam	✓	✓	✓
Tolbutamide	↓	✗	✗
Tolmetin	✓	✓	✓
Tolperisone	✓	✓	✓
Tolvaptan	✓	✓	✓
Torasemide	✓	✗	✗
Tramadol	✓	↑	✓
Tranylcypromine	✓	✓	✓
Travoprost	✓	✓	✓
Trepibutone	✓	✓	✓
Triamcinolone	✓	✓	✓
Triazolam	✓	↑	↑
Triclabendazole	✓	✓	✓
Trifluoperazine	✓	✓	✓
Trifluridine	✓	✓	✓
Trilostane	✓	✓	✓
Trimetaphan	✓	✓	✓
Trimethoprim	✓	✗	✗
Trimipramine	✓	✓	✓
Tritoqualine	✓	✓	✓
Troleandomycin	✓	↑	↑
Tropatepine	✓	✓	✓

	Effect	Breakdown	Dose
Tinidazole	✓	↑	↑
Tioctic Acid	✓	✓	✓
Tiotixene	✓	✓	✓
Tipranavir	✓	↑	↑
Tirilazad	✓	✓	✓
Tisopurine	✓	✓	✓
Tobramycin	✓	✓	✓
Tolazamide	✓	✓	✓
Tolcapone	✓	↑	↑
Tolonidine	✓	✓	✓
Tolrestat	✓	✓	✓
Topiramate	✓	✓	✓
Toremifene	✓	↑	↑
Trandolapril	✓	✓	✓
Trapidil	✓	✓	✓
Trazodone	✓	↑	↑
Treprostinil	✓	✓	✓
Triamterene	✓	✓	✓
Trichlormethiazide	✓	✓	✓
Triclofos	✓	✓	✓
Trifluperidol	✓	✓	✓
Triflusal	✓	✓	✓
Trimazosin	✓	✓	✓
Trimetazidine	✓	✓	✓
Trimethyldiphenylpropylamine	✓	✓	✓
Triprolidine	✓	✓	✓
Trofosfamide	✓	✓	✓
Trolnitrate	✓	✓	✓
Tropicamide	✓	✓	✓

	Effect	Breakdown	Dose
Tiocarlide	✓	✓	✓
Thioguanine	✓	✓	✓
Tiotropium Bromide	✓	✓	✓
Tiracizin	✓	✓	✓
Tirofiban	✓	✓	✓
Tixocortol	✓	✓	✓
Tocainide	✓	✓	✓
Tolazoline	✓	✓	✓
Tolfenamic Acid	✓	✓	✓
Toloxatone	✓	✓	✓
Tolterodine	✓	↑	↑
Topotecan	✓	✓	✓
Trabectedin	✓	✓	✓
Tranexamic Acid	✓	✓	✓
Trastuzumab	✓	✓	✓
Treosulfan	✓	✓	✓
Tretoquinol	✓	✓	✓
Triaziquone	✓	✓	✓
Trichloroethylene	✓	✓	✓
Tridihexethyl	✓	✓	✓
Triflupromazin	✓	✓	✓
Trihexyphenidyl	✓	✓	✓
Trimebutine	✓	✓	✓
Trimethadione	✓	✓	✓
Trimetrexate	✓	✓	✓
Triptorelin	✓	✓	✓
Troglitazone	✓	↓	↓
Trometamol	✓	✓	✓
Tropisetron	✓	✓	✓

	Effect	Breakdown	Dose
Trospium	✓	✓	✓
Troxipide	✓	✓	✓
Tulobuterol	✓	✓	✓
Ulobetasol	✓	✓	✓
Urate Oxidase	✓	✓	✓
Ursodeoxycholic Acid	✓	✓	✓
Valganciclovir	✓	✓	✓
Valpromide	✓	✓	✓
Vancomycin	✓	✓	✓
Vardenafil	✓	↑	↑
Vecuronium	✓	✓	✓
Veralipride	✓	✓	✓
Verteporfin	✓	✓	✓
Vildagliptin	✓	✓	✓
Vinbarbital	✓	✓	✓
Vincamine	✓	✓	✓
Vinflunine	✓	✓	✓
Vinyl Ether	✓	✓	✓
Voclosporin	✓	✓	✓
Vorinostat	✓	✓	✓
Xaliproden	✓	✓	✓
Xibornol	✓	✓	✓
Yohimbin	✓	✓	✓
Zaleplon	✓	↑	↑
Zidovudine	✓	✓	✓
Zinc Chloride	✓	✓	✓
Zofenopril	✓	✓	✓
Zolimidine	✓	✓	✓
Zomepirac	✓	✓	✓

	Effect	Breakdown	Dose
Trovafloxacin	✓	✓	✓
Tryptophan	✓	✓	✓
Tyloxapol	✓	✓	✓
Unoproston	✓	✓	✓
Urofollitropin	✓	✓	✓
Valaciclovir	✓	✓	✓
Valnoctamide	✓	✓	✓
Valrubicin	✓	✓	✓
Vandetanib	✓	✓	✓
Varenicline	✓	✓	✓
Vemurafenib	✓	✓	✓
Verapamil	✓	↑	↑
Vigabatrin	✓	✓	✓
Viloxazine	✓	✓	✓
Vinblastine	✓	↑	↑
Vincristine	✓	↑	↑
Vinorelbine	✓	↑	↑
Vinylbital	✓	✓	✓
Voglibose	✓	✓	✓
Vorozole	✓	✓	✓
Xamoterol	✓	✓	✓
Ximelagatran	✓	✗	✗
Zafirlukast	✓	✗	✗
Zanamivir	✓	✓	✓
Zimeldine	✓	✓	✓
Zipeprol	✓	✓	✓
Zoledronic Acid	✓	✓	✓
Zolmitriptan	✓	✓	✓
Zonisamide	✓	↑	↑

	Effect	Breakdown	Dose
Troxerutin	✓	✓	✓
Tubocurarine	✓	✓	✓
Ubidecarenone	✓	✓	✓
Urapidil	✓	✓	✓
Urokinase	✓	✓	✓
Valdecoxib	✓	✓	✓
Valproic Acid	✓	↓	↓
Valsartan	✓	✗	✗
Vapreotide	✓	✓	✓
Vasopressin	✓	✓	✓
Venlafaxine	✓	✓	✓
Vernakalant	✓	✓	✓
Vilazodone	✓	✓	✓
Viminol	✓	✓	✓
Vinburnine	✓	✓	✓
Vindesine	✓	↑	↑
Vinpocetine	✓	✓	✓
Visnadine	✓	✓	✓
Voriconazole	✓	✗	✓
Warfarin	✓	✗	✗
Xantinol Nicotinate	✓	✓	✓
Xipamide	✓	✓	✓
Zalcitabine	✓	✓	✓
Ziconotide	✓	✓	✓
Zinc Acetate	✓	✓	✓
Ziprasidone	✓	↑	↑
Zolendromat	✓	✓	✓
Zolpidem	✓	↑	↑
Zopiclone	✓	↑	↑


Effect
Breakdown
Dose
Zorubicin
Effect
Breakdown
Dose
Zotepine
Effect
Breakdown
Dose
Zuclopenthixol

PHARMACO GENETICS

ONCOLOGY

CARDIOVASCULAR SYSTEM

NEUROLOGY

METABOLISM

MOVEMENT

DIGESTION

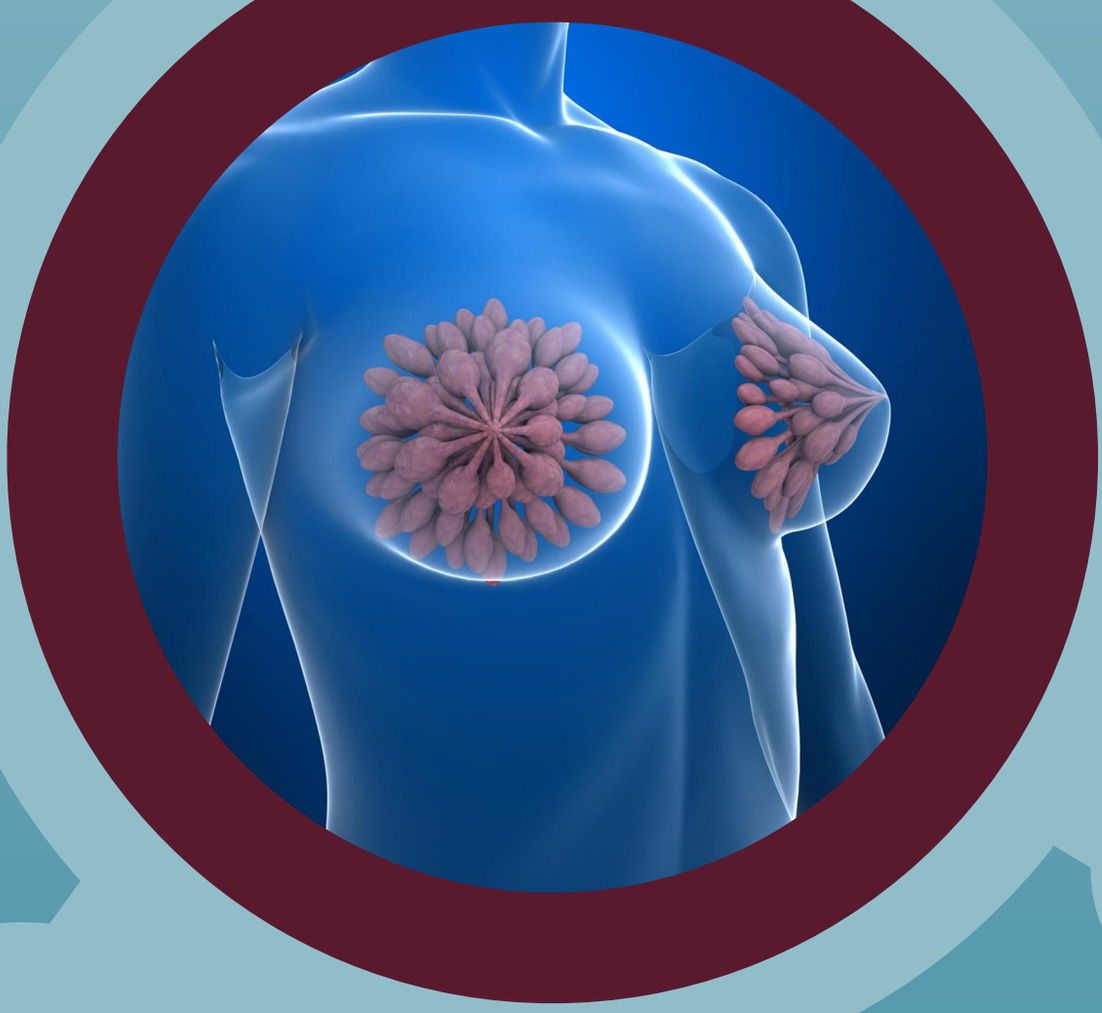
OPHTHALMOLOGY

ODONTOLOGY

OTHERS

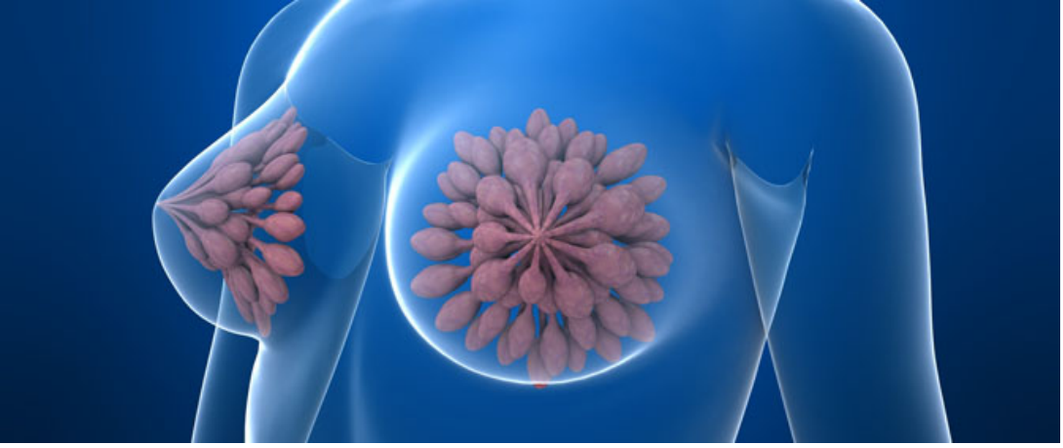
SCIENCE

ADDITIONAL INFORMATION



Breast Health Sensor

Effective prevention and treatment of breast cancer

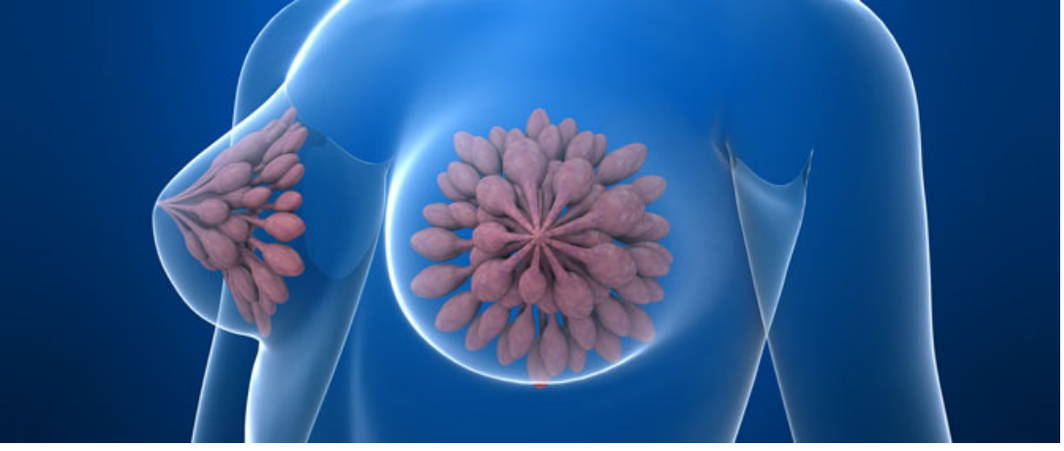


Breast cancer

The number of breast cancer cases has almost doubled since 1970, but the treatment and observation measures have been so effective that the death rate is steadily decreasing. It is however, very important to reduce risk factors so that fewer cancers develop; today's modern genetic diagnostics provide opportunities to do this. Even people who have already been diagnosed with breast cancer will benefit from knowing the various risk factors that they are subject to, in order to eliminate as many risks as possible. Therefore, it is important to know your own genetic predispositions and to make specific lifestyle changes that will maximize the opportunity for a healthy life.

Most cases of breast cancer are caused by an unfortunate interaction of genetic predisposition and environmental triggers. Women who have a high risk of breast cancer are at an even higher risk if they follow an unhealthy lifestyle. It is important for women to know their genetic risk, and if necessary, to take preventive measures and/or make lifestyle changes.

Although some cases of breast cancer occur sporadically with age, it is estimated that genetic predisposition is responsible for about 58% of breast cancer cases. Ten genes associated with breast cancer can now be tested for traits that affect an individual's risk of disease. A person with a strong genetic predisposition to cancer can reduce their overall risk by adopting a balanced diet and avoiding other risk factors. Also, regular checkups will allow the early detection of the disease, and the timely treatment.



Relevant genes for breast cancer

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

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
FGFR2	rs2981582	G>A	C/C
VDR	rs2228570	VDR FokI T/C	T/C
8q24	rs13281615	T>C	G/A
TNRC9	rs3803662	C>T	C/C
MAP3K1	rs889312	A>C	A/C
LSP1	rs3817198	T>C	T/C
CASP8	rs1045485	D302H (G/C)	G/G
2q35	rs13387042	G>A	A/A
XRCC2	rs3218536	A>G	G/G
CYP1A2	rs762551	A>C	C/C

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

Summary of effects

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

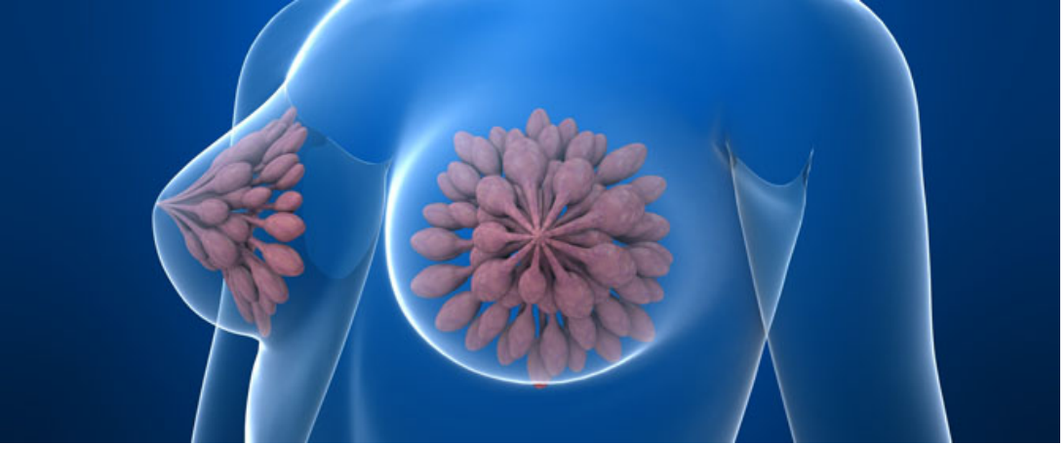
- Your risk of developing breast cancer is lower than the population average.
- 2-5 cups of coffee per day could delay the development of breast cancer by approximately 7 years

How high is your risk of developing breast cancer?

LOW AVERAGE RISK HIGHER

The effect of coffee on breast cancer

EFFECTIVE INEFFECTIVE



Prevention

Based on your genetic profile, you have no higher risk for breast cancer than the average person.
While you do not have a high risk of developing breast cancer, some people with no genetic risk do develop cancer. Therefore, you should follow the usual preventive measures and self-examination. Every person should take the following steps to reduce their risk of breast cancer:

Prevention

Lifestyle plays an important role in the development of breast cancer, and a significant amount of the risk of cancer is based on specific behaviour choices. You can take several steps to reduce your risk of breast cancer.

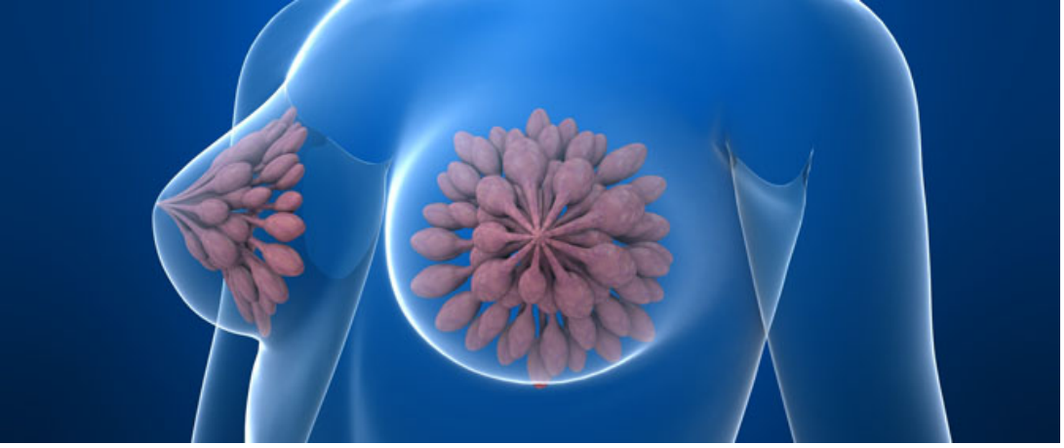
- Excess weight increases the risk of breast cancer 2.5 times. Maintaining a healthy weight is key to reducing your risk of breast cancer, along with many other diseases.
- Tobacco smoking is an equally important risk factor that increases the risk of breast cancer by about 30%, in addition to the many other health problems it causes. Consuming more than 20g of alcohol per day (about 120mL of red wine, or one glass) increases breast cancer risk by about 30% and should therefore be avoided.
- Vitamin D deficiency is a significant risk factor for breast cancer. Vitamin D is normally produced in the presence of UV-B rays from the sun, so deficiencies are more common in countries with limited sunlight or in people who are indoors most of the time. This deficiency is associated with a variety of cancer forms, and so an adequate uptake of vitamin D is highly recommended. Exercise outdoors as much as possible and make sure your diet contains sufficient quantities of vitamin D. Salmon, tuna and mackerel are some of the foods containing vitamin D. However, it is generally advisable to ensure an adequate intake with vitamin supplements.

Early detection

Early detection plays a significant role in every type of cancer, which is why women are encouraged to go for annual breast cancer checkups. Women with your genetic profile should follow the following routine checkups:

- From the age of 20, go for annual breast examinations.
- From the age of 20, perform regular self-examinations of the breast tissue.
- Examine the tissue of your breasts for hard inclusions.
- Should you detect a hard inclusion, talk to your doctor about it immediately.
- From the age of 40, have a mammogram performed every 1 to 2 years.

In this way, a possible cancer is detected immediately and treated in time.



Drug compatibility

DRUGS	139	310	107	262	221	276	524	371	12
GENES	CYP2E1	CYP2D6	CYP2B6	CYP1A2	CYP2C19	CYP2C9	CYP3A4	CYP3A5	NAT2
DEGRADATION	NORMAL	NORMAL	NORMAL	NORMAL	NORMAL	NONE	NORMAL	NORMAL	SLOW

DRUGS	3	
GENES	DPYD	
FUNCTION	NONE	

Effect on relevant medication

	Effect	Breakdown	Dose
Alfentanil	✓	↑	↑
Buprenorphine	✓	↑	↑
Codeine	✓	✓	✓
Dolasetron	✓	✓	✓
Doxorubicin	✓	↑	↑
Erlotinib	✓	↑	↑
Exemestane	✓	↑	↑
Fulvestrant	✓	↑	↑
Goserelin	✓	✓	✓
Ifosfamide	↑	↑	↓
Ixabepilone	✓	✓	✓
Leuprorelin	✓	✓	✓
Megestrol	✓	✓	✓
Methoxyflurane	✓	✓	✓
Oxycodone	✓	↑	✓

	Effect	Breakdown	Dose
Anastrozole	✓	✓	✓
Capecitabine	✓	✗	✗
Cyclophosphamide	✓	↑	↑
Domperidone	✓	✓	✓
Enflurane	✓	✓	✓
Etoposide	✓	↑	↑
Fentanyl	✓	↑	↑
Gefitinib	✓	↑	↑
Halothane	✓	✓	✓
Imatinib	↑	↑	↓
Lapatinib	✓	↑	↑
Levacetylmethadol	✓	↑	↑
Methadone	✓	↑	↑
Metoclopramide	✓	✓	✓
Paclitaxel	✓	✓	✓

	Effect	Breakdown	Dose
Aprepitant	✓	↑	↑
Carboplatin	✓	✓	✓
Docetaxel	✓	↑	↑
Doxorubicin	✓	↑	↑
Epirubicin	✓	✓	✓
Everolimus	✓	↑	↑
Fluorouracil	✓	✗	✗
Gemcitabine	✓	✓	✓
Hydrocodone	✓	✓	✓
Isoflurane	✓	✓	✓
Letrozole	✓	↑	↑
Lidocain	✓	✓	✓
Methotrexate	✓	✓	✓
Nilutamide	✓	✓	✓
Paracetamol	✓	✓	✓

	Effect	Breakdown	Dose
Phenacetin	✓	✓	✓
Sevoflurane	✓	✓	✓
Tamoxifen	✗	✗	✓
Teniposide	✓	↑	↑
Tramadol	✓	↑	✓
Vinblastine	✓	↑	↑
Vindesine	✓	↑	↑

	Effect	Breakdown	Dose
Raloxifene	✓	✓	✓
Sorafenib	✓	↑	↑
Tamoxifen	✗	✗	✓
Thiotepa	✓	✓	✓
Trastuzumab	✓	✓	✓
Vinblastine	✓	↑	↑
Vinorelbine	✓	↑	↑

	Effect	Breakdown	Dose
Ropivacaine	✓	✓	✓
Sunitinib	✓	↑	↑
Temsirolimus	✓	✓	✓
Toremifene	✓	↑	↑
Vemurafenib	✓	✓	✓
Vincristine	✓	↑	↑
Zolmitriptan	✓	✓	✓

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.

Colon Health Sensor

Effective prevention and early detection of colon cancer

Colon cancer

Colon cancer is one of the most common cancers in the Western world, affecting more than 6% of the population during the course of their lifetime. The chances of recovery are relatively good, provided early detection. Regular colonoscopy screening and the removal of benign intestinal polyps (which can turn into malignant cancer at a later stage) often helps detect cancer early on or prevent it altogether.

Men are more likely to be affected by colon cancer than females, with the ratio being around 60:40. About 90% of colon cancer cases occur after the age of 50. Statistics show that about 1 in 100 people between the ages of 45 and 75 has an undetected cancer, while about 3 in 100 have benign polyps in the gut that should be removed as a precautionary measure.

Risk factors

A number of environmental risk factors can increase the likelihood of colon cancer. In general, older people are increasingly affected. Vitamin D deficiency and the presence of colon polyps, as well as genetic predispositions or various disorders of the intestine (ulcerative colitis or Crohn's disease) are further factors that promote the development of the disease.

Diet is a major factor in colon cancer: the daily consumption of red meat increases the risk by 49% per 100g. The risk increases by about 70% per 100g of sausage. The risk can be reduced by 40% by increasing fibre intake.

Obesity, years of smoking, and a lack of sunlight that causes vitamin D deficiency, are also known risk factors for the onset of the disease.

Symptoms of colon cancer

Colon cancer usually remains unrecognized in the early stages, which makes early diagnosis and timely treatment much more difficult. The following symptoms may occur in advanced stages of colon cancer:

- Blood or mucus in the stool
- Intestinal cramps
- Pencil or goat kettle shaped stools
- Diarrhoea and constipation
- Flatulence
- Anemia (due to blood loss)
- Performance degradation
- Fatigue and general weakness
- Weight-loss

Genes relevant to colon cancer

Several genetic variations have been identified that are known to have an impact on the development of colon cancer. If considering these genetic variations as a whole, they can have a significant impact on the likelihood of developing a disease. The analysis of the relevant genetic variations allowed for the following conclusions:

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
CASC8	rs6983267	T>G	G/G
CASC8	rs10505477	G>A	G/G
CASC8	rs10808555	A>G	A/A
CASC8	rs7837328	G>A	G/G
CASC8	rs7014346	G>A	G/G
CCND1	rs9344	G>A	G/G
CDH1	rs16260	C>A	A/A
COLCA1/COLCA2	rs3802842	A>C	A/A
CYP1A1	rs1048943	A>G	A/A
DNMT3B	rs1569686	T>G	G/T
GREM1	rs10318	C>T	C/C
IL8/CXCL8	rs4073	T>A	T/A
IL10	rs1800872	C>A	C/A
MTHFR	rs1801133	C>T	C/C
MTRR	rs1801394	A>G	G/G
SMAD7	rs12953717	C>T	C/C
TGFB1	rs1800469	A>G	A/A

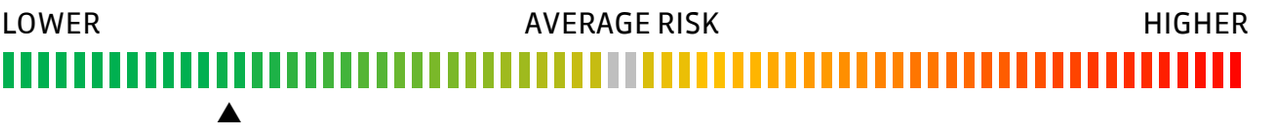
LEGEND: rsNCBI = description of examined genetic variation, POLYMORPHISM = form of the genetic variation, GENOTYPE = personal analysis result

Summary of effects

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

- Your risk of developing colon cancer is lower than that of the population average.

Risk of colon cancer



Prevention

You do not have a genetically increased risk of developing colon cancer, therefore typical prevention and screening measures are sufficient for you. No special measures beyond the general rules of a healthy lifestyle are required.

Recommendations for your diet and lifestyle

According to the latest available information, the following nutritional recommendations apply as prevention against colon cancer:

- Reach and maintain a normal body weight.
- Ensure a balanced diet.
- Foods with large amounts of fat and sugar should only be consumed occasionally and in small quantities.
- Eat foods that are high in fibre.
- Reduce your consumption of red meat.
- Eat fish regularly.
- Alcohol should only be consumed in small quantities.
- Make sure you have enough vitamin D3.
- Do sports regularly.

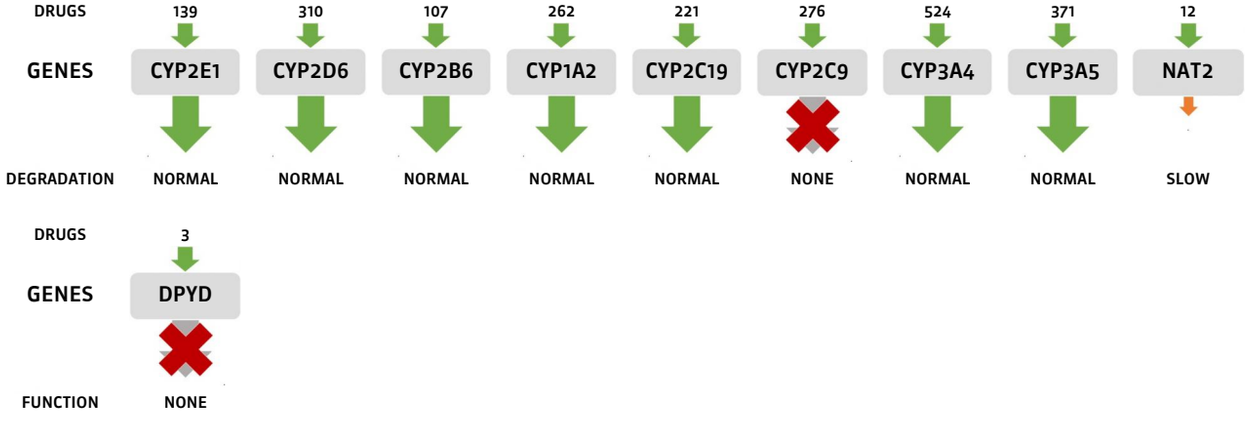
Early diagnosis

Early detection is critical to the successful treatment of any type of cancer. If the cancer is detected early on, it can usually be treated quite well and effectively.

The recommended screening program consists of the following examinations:

- CHEMICAL STOOL TEST: Annually (recommended from the age of 50)
- IMMUNOLOGICAL STOOL TEST: Annually (recommended from the age of 50)
- MAJOR COLONOSCOPY: Every 10 years (recommended from the age of 55)
- MINOR COLONOSCOPY: Every 5 years (recommended from the age of 55)

Drug compatibility



Effect on relevant medication

	Effect	Breakdown	Dose
Alfentanil	✓	↑	↑
Buprenorphine	✓	↑	↑
Codeine	✓	✓	✓
Enflurane	✓	✓	✓
Halothane	✓	✓	✓
Isoflurane	✓	✓	✓
Methadone	✓	↑	↑
Metoclopramide	✓	✓	✓
Paracetamol	✓	✓	✓
Sevoflurane	✓	✓	✓
Trifluridine	✓	✓	✓

	Effect	Breakdown	Dose
Aprepitant	✓	↑	↑
Capecitabine	✓	✗	✗
Dolasetron	✓	✓	✓
Fentanyl	✓	↑	↑
Hydrocodone	✓	✓	✓
Levacetylmethadol	✓	↑	↑
Methotrexate	✓	✓	✓
Oxaliplatin	✓	✓	✓
Phenacetin	✓	✓	✓
Tegafur	✓	✗	✗
Zolmitriptan	✓	✓	✓

	Effect	Breakdown	Dose
Bevacizumab	✓	✓	✓
Cetuximab	✓	✓	✓
Domperidone	✓	✓	✓
Fluorouracil	✓	✗	✗
Irinotecan	✓	↑	↓
Lidocain	✓	✓	✓
Methoxyflurane	✓	✓	✓
Oxycodone	✓	↑	✓
Ropivacaine	✓	✓	✓
Tramadol	✓	↑	✓

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.

Skin Health Sensor

Effective prevention and early detection of skin cancer

Skin cancer

Although skin cancer is visible on the skin and can theoretically be diagnosed and treated easily, some tumours often develop inconspicuously and in concealed areas, only causing symptoms once they have already spread throughout the body.

There are basically two types of skin cancer:

Light skin cancer: This leads to a cancerous reproduction of light skin cells (basal cells or prickle cells). Although this form of cancer is common, it is usually very treatable and rarely fatal.

The black skin cancer: Black skin cancer, also called melanoma, is rarer and more dangerous. It is caused by a cancerous proliferation of pigment cells, which are usually responsible for a sun tan.

Risk factors

In addition to excessive UV radiation from the sun or a solarium, genetic factors play a significant role in skin cancer progression. Thus, individuals with an increased number of skin blemishes, family history of skin cancer, and skin types that don't tan well and easily develop mild sunburns, are at a particularly high risk.

Prevention and early detection

Protecting yourself from excessive UV radiation with sunscreen is a primary precautionary measure against skin cancer. In addition, the early detection of the disease is considerably important for successful treatment. Usually, skin cancer forms from skin blemishes, which can change visually and possibly lead to discomfort. The blemish will be examined visually by your doctor and, if necessary, also in the laboratory to diagnose possible skin cancer. In many cases, the blemish is then excised, effectively curing the cancer.

Self-examination

Regular self-examination is an essential part of early detection, especially for genetically predisposed individuals and those who have had skin cancer previously. Depending on the level of risk, you should examine your skin for any changes every 3 to 6 months (especially where the blemishes occur). If skin lesions are discovered, they should be discussed with the doctor immediately.

Genes relevant to skin cancer

Several genetic variations have been identified that are known to have an impact on the development of skin cancer. If considering these genetic variations as a whole, they can have a significant impact on the likelihood of developing the disease. The analysis of the relevant genetic variations allowed for the following conclusions:

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
ASIP	rs1015362	A>G	G/A
ASIP	rs4911414	G>T	G/T
CDK10	rs258322	C>T	C/C
CLPTM1L	rs401681	T>C	C/T
MC1R	rs11547464	G>A	G/G
MC1R	rs1805005	G>T	G/G
MC1R	rs1805006	C>A	C/C
MC1R	rs1805007	C>T	C/C
MC1R	rs1805009	G>C	G/G
MC1R	rs2228479	G>A	G/G
MC1R	rs885479	G>A	G/G
MTAP	rs7023329	A>G	G/A
MYH7B	rs1885120	G>C	C/G
NCOA6	rs4911442	A>G	A/A
PARP1	rs3219090	A>G	G/G
PIGU	rs910873	G>A	A/A
SLC45A2	rs16891982	C>G	G/G
TYR	rs1393350	G>A	G/A

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

Summary of effects

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

- Your risk of developing malignant melanoma ("black skin cancer") is lower than the population average.
- Your risk of developing non-malignant melanoma ("light skin cancer") is lower than the population average.

Risk of malignant melanoma ("black skin cancer")

Risk of light skin cancer (non-melanoma skin cancer)

Prevention

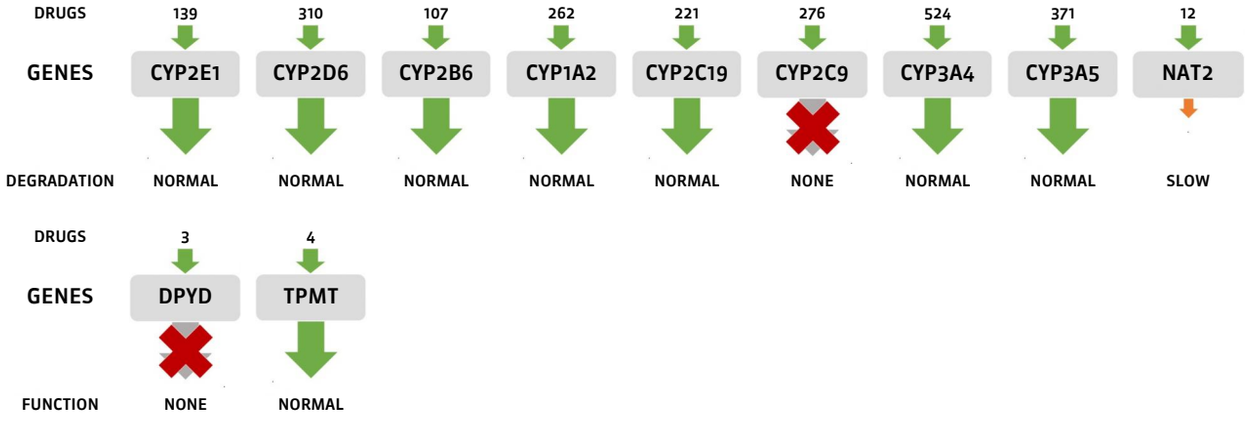
Since you do not have an increased risk of skin cancer, you will not need any precautionary measures beyond the typical recommendations for early skin cancer detection.

Recommendations for prevention and early detection

The following measures are recommended for you:

- Examine all areas of your skin visually and by palpation once a year.
- Talk to your doctor immediately if you notice changes in the skin or blemishes.
- Use sufficient amounts of sunscreen when outdoors.

Drug compatibility



Effect on relevant medication

	Effect	Breakdown	Dose
Alfentanil	✓	↑	↑
Cisplatin	✓	✓	✓
Dolasetron	✓	✓	✓
Fentanyl	✓	↑	↑
Hydrocodone	✓	✓	✓
Lidocain	✓	✓	✓
Metoclopramide	✓	✓	✓
Phenacetin	✓	✓	✓
Tramadol	✓	↑	✓

	Effect	Breakdown	Dose
Aprepitant	✓	↑	↑
Codeine	✓	✓	✓
Domperidone	✓	✓	✓
Fluorouracil	✓	✗	✗
Isoflurane	✓	✓	✓
Methadone	✓	↑	↑
Oxycodone	✓	↑	✓
Ropivacaine	✓	✓	✓
Vemurafenib	✓	✓	✓

	Effect	Breakdown	Dose
Buprenorphine	✓	↑	↑
Dacarbazine	✓	✓	✓
Enflurane	✓	✓	✓
Halothane	✓	✓	✓
Levacetylmethadol	✓	↑	↑
Methoxyflurane	✓	✓	✓
Paracetamol	✓	✓	✓
Sevoflurane	✓	✓	✓
Zolmitriptan	✓	✓	✓

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.

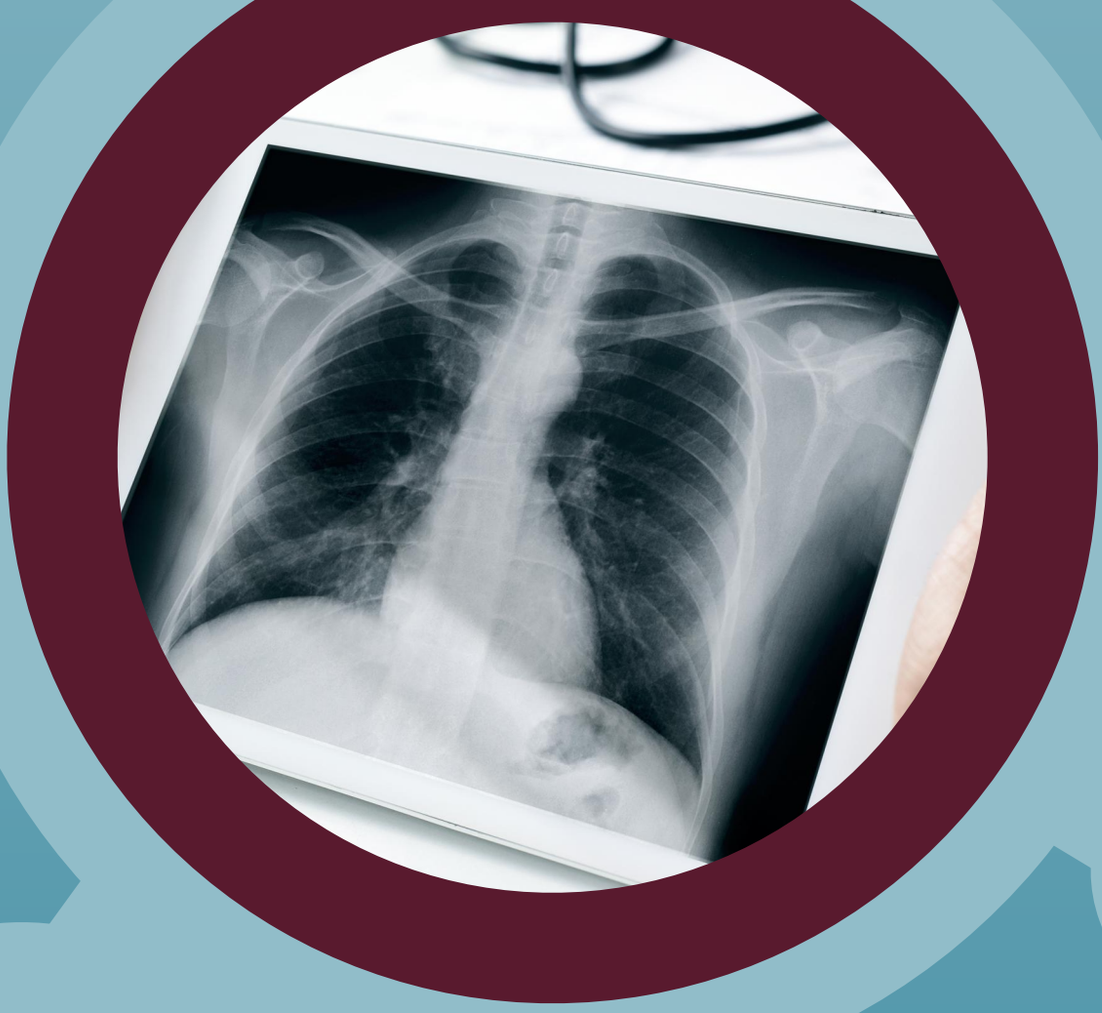
Effect: Normal. Degradation: Slower. Recommendation: Reduce the dosage.

Effect: Normal. Degradation: None. Recommendation: Alternative drug.

Effect: Lower. Degradation: Normal. Recommendation: Normal dosage.

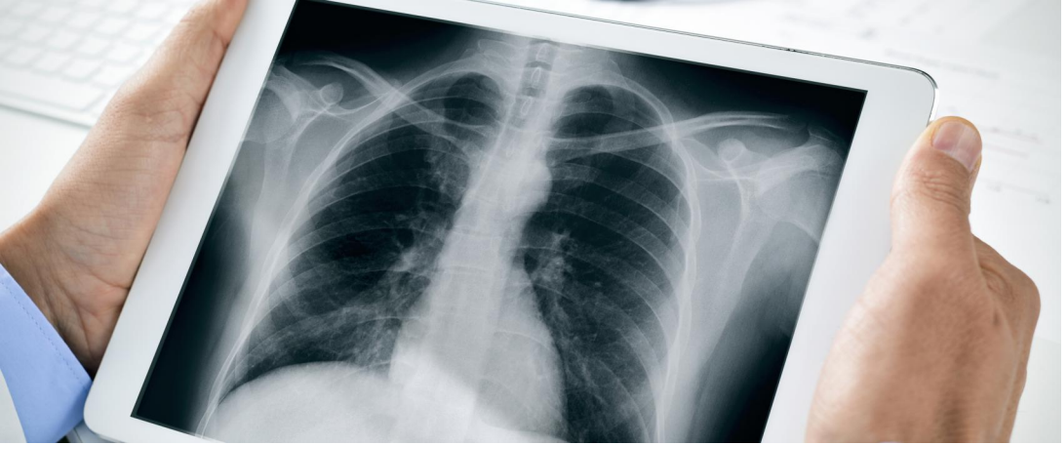
Effect: Lower. Breakdown: Lower. Recommendation: Reduce the dosage.

Effect: Stronger. Degradation: Stronger. Recommendation: Normal dosage.



Lung Health Sensor

Effective prevention and treatment of lung cancer



Lung cancer

Lung cancer, commonly referred to as bronchogenic carcinoma, is a disease of the lung tissue cells. Newly occurring mutations in a cell cause uncontrolled growth that results in a tumour. There are various environmental influences that may damage the DNA of lung cells, such smoking tobacco or regular inhalation of soot, fine dust and exhaust fumes. However, certain genes can usually detect and neutralize these toxins before they are able to do much harm. However, in some people, the genes responsible for such detox-functions are hindered by genetic variations. If people with impaired detoxification systems are exposed to these risk factors, they can develop cancer.

Risk factors

The most significant risk factor is tobacco smoking (for both genetically predisposed individuals and for those with optimally functioning detoxification genes). Smoking is responsible for around 85% of all lung cancer cases. In addition to active smoking, passive smoking accounts for about 3-5% of lung cancer cases. After smoking as a big risk factor, the radioactive gas radon, that can arise from the ground and collect in mines or old cellars, is the other significant risk factor for the development of lung cancer. Thus, adequate ventilation of basements is an effective method of prevention.

Other occupational risk factors include the inhalation of exhaust fumes, particulate matter from construction, mining or metalworking industries. In particular, dust consisting of quartz, arsenic, chromium and nickel compounds, as well as asbestos are particularly problematic.

Early detection is crucial

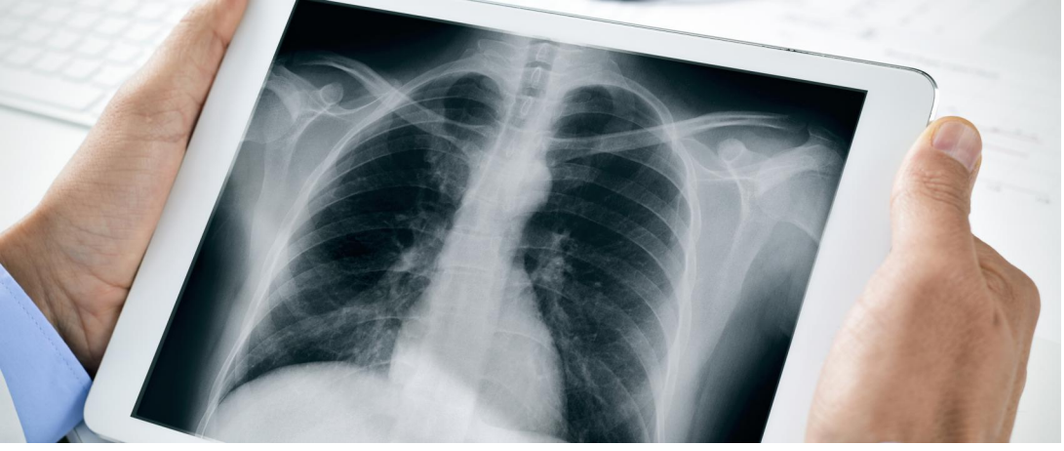
Usually, lung cancer only has noticeable symptoms at a very late stage. At this point, the cancer is often not treatable. People with the highest chance of survival are those who, either by accident or specific preventive screening, recognize and treat the cancer during its asymptomatic stage. For example, a screening study showed that the 5-year survival rate of symptomatic sufferers is only 14%. In contrast, the 5-year survival rate of individuals, in whom the disease was identified in the adenoma-symptomatic stage by means of screening is approximately 80%.

Symptoms at an advanced stage:

Symptoms of lung cancer usually occur relatively late and should immediately be examined by a specialist. Furthermore, symptoms are often very nonspecific and can be triggered by other diseases. Symptoms include:

- A cough that lasts longer than three weeks
- Coughing of blood

- Fatigue and reduced performance
- Weight loss
- Difficulty swallowing or hoarseness
- Bone pain
- Lymph node swelling in the neck region
- Permanent coughing up of mucus
- Fever with no obvious cause
- Constant shortness of breath
- Chest pain



Genes relevant to lung cancer

Several genetic variations have been identified that are known to have an impact on the development of lung cancer. When considering these genetic variations collectively, they can have a significant impact on the likelihood of developing the disease. The analysis of the relevant genetic variations allowed for the following conclusions:

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
CYP1A1	rs4646903	C>T	T/T
CYP1A1	rs1048943	G>A	A/A
GSTM1	Null allele	INS>DEL	INS
GSTT1	Null allele	INS>DEL	DEL
GSTP1	rs1695	G>A	G/A

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

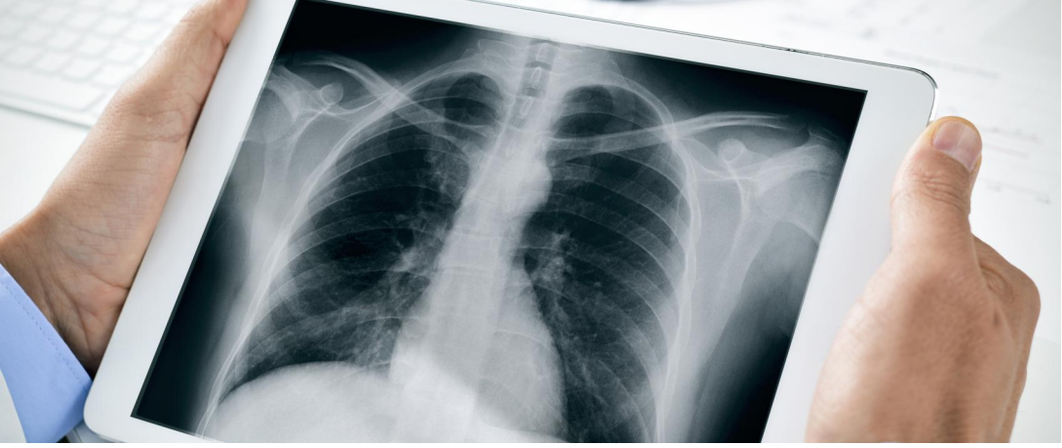
Summary of effects

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

➤ Your risk of suffering lung cancer is higher than the population average. 1.2-fold.

How high is your risk of developing lung cancer?

LOW AVERAGE RISK HIGHER



Prevention

Based on your genetic profile, you have an increased risk of developing lung cancer. In addition to an increased risk, despite a healthy lifestyle, the known risk factors for lung cancer (smoke, fine dust and gases) are significantly more harmful to you than average.

If you are a smoker, do not compare your risk to other smokers. Smoking harms you far more than most other people.

Nutrition

Certain lifestyle choices can drastically reduce the risk of lung cancer and other types of cancers. A diet rich in antioxidants (vitamin C, vitamin E, selenium, zinc, alpha lipoic acid) can help neutralize toxic free radicals and reduce the risk of cancer. Thus, make sure you eat plenty of colourful fruits and vegetables.

A diet rich in minerals such as calcium, zinc and selenium is especially advisable for individuals with limited heavy metal detoxification.

A vitamin D deficiency is another risk factor for the development of various types of cancer. Therefore, spend a safe amount of time exposed to sunlight and eat a lot of Vitamin D3 (contained in fish, dairy and some dietary supplements).

Avoid pollutants

Firstly, tobacco smoke is a toxic pollutant and should therefore be avoided at all costs. This applies to direct smoke from a cigarette, cigar or pipe, but also passive smoking. Even if you have been smoking for decades, stopping now will significantly reduce your risk over time.

If you are exposed to fine dust particles due to professional reasons, it is essential to ensure adequate respiratory protection. This applies to exhaust fumes, fine dust from the construction industry, smoke and dust from metalworking, as well as other types of fine dust.

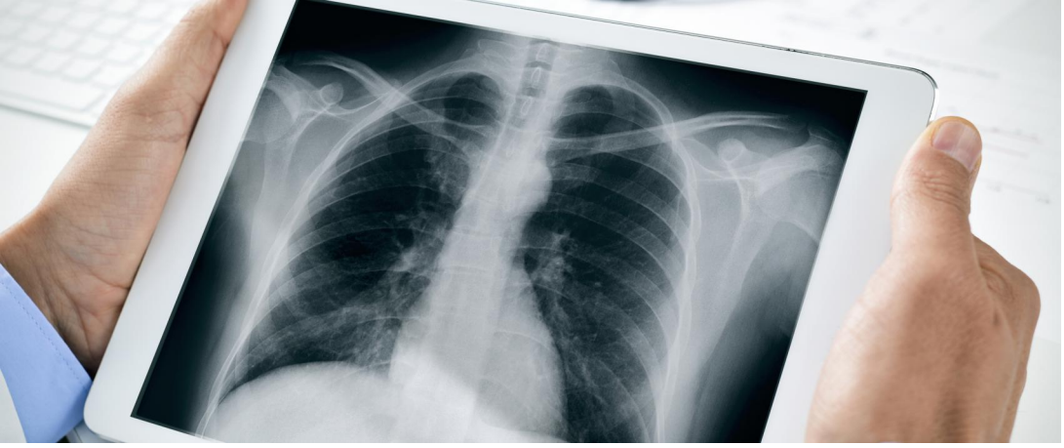
Since radioactive radon gas can accumulate in old cellars and poorly ventilated buildings, make sure that your basement is well ventilated.

Early detection

Although early detection is the most important factor in the treatment of lung cancer, a routine lung examination is not available in many countries. The lungs are only examined with X-ray, MRI or CT and the cancer is only detected after initial symptoms and complaints. By this time, however, only about 12% of all cases can be treated successfully. The best chance of

successful treatment exists for individuals who identify their lung cancer during unrelated x-ray examinations. In such cases, about 88% of those affected will get healthy again.

Currently, European countries do not offer routine lung cancer screenings. Thus, the only option at this time is screening by medical specialists as a private service. Regular lung examinations with CT are recommended for persons who have a genetic predisposition, as well as those who are or were heavy smokers. If the result is negative (i.e. no cancer was detected), it is recommended to repeat this examination every five years.



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
Afatinib	✓	✓	✓
Bevacizumab	✓	✓	✓
Codeine	✓	✓	✓
Domperidone	✓	✓	✓
Erlotinib	✓	↑	↑
Fentanyl	✓	↑	↑
Halothane	✓	✓	✓
Levacetylmethadol	✓	↑	↑
Methotrexate	✓	✓	✓
Oxycodone	✓	↑	✓
Paclitaxel	✓	✓	✓
Phenacetin	✓	✓	✓
Topotecan	✓	✓	✓
Zolmitriptan	✓	✓	✓

	Effect	Breakdown	Dose
Alfentanil	✓	↑	↑
Buprenorphine	✓	↑	↑
Docetaxel	✓	↑	↑
Doxorubicin	✓	↑	↑
Etoposide	✓	↑	↑
Gefitinib	✓	↑	↑
Hydrocodone	✓	✓	✓
Lidocain	✓	✓	✓
Methoxyflurane	✓	✓	✓
Paclitaxel	✓	✓	✓
Paracetamol	✓	✓	✓
Ropivacaine	✓	✓	✓
Tramadol	✓	↑	✓

	Effect	Breakdown	Dose
Aprepitant	✓	↑	↑
Carboplatin	✓	✓	✓
Dolasetron	✓	✓	✓
Enflurane	✓	✓	✓
Everolimus	✓	↑	↑
Gemcitabine	✓	✓	✓
Isoflurane	✓	✓	✓
Methadone	✓	↑	↑
Metoclopramide	✓	✓	✓
Paclitaxel	✓	✓	✓
Pemetrexed	✓	✓	✓
Sevoflurane	✓	✓	✓
Vinorelbine	✓	↑	↑

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.

PHARMACO GENETICS

ONCOLOGY

CARDIOVASCULAR SYSTEM

NEUROLOGY

METABOLISM

MOVEMENT

DIGESTION

OPHTHALMOLOGY

ODONTOLOGY

OTHERS

SCIENCE

ADDITIONAL INFORMATION

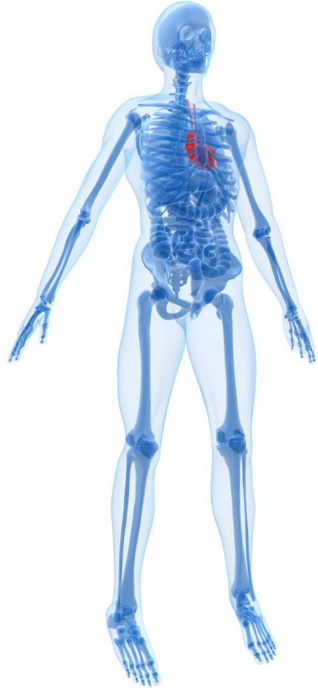
Cardiovascular Sensor

Prevention and treatment of lipid metabolism problems

Cardiovascular Disease

Cholesterol and triglycerides are vital fats our body uses to produce cell membranes, store energy, absorb fat-soluble vitamins and produce hormones. These fats are so essential, that our body even produces cholesterol itself. This cholesterol is then transported and made available for cell growth throughout the body via the bloodstream. In this way, our body produces about 70% of the cholesterol it requires itself. The other 30% comes from what we eat.

The amount of cholesterol in your bloodstream goes up after every meal. To control this, your body has strict regulatory processes that maintain your blood cholesterol at normal levels. If there is too much cholesterol in your bloodstream, your body transports HDL to your liver, which filters it from your blood, lowering your cholesterol level. However, the liver will release LDL cholesterol into your bloodstream, which increases your cholesterol level. High cholesterol levels may cause arteriosclerosis, therefore a low cholesterol level keeps you healthy.



Due to this, HDL cholesterol—the kind transported to the liver—is called "good" cholesterol, whereas LDL cholesterol—which the liver releases into the bloodstream—is called "bad" cholesterol.

That is why it is important for your health to maintain a high HDL and a low LDL cholesterol level.

A number of genes are responsible for regulating cholesterol and triglyceride levels, or for increasing the risk of cardiovascular disease. If you carry an adverse trait in one or more of these genes, you should pay special attention to your fat intake and metabolism. Since diet is the most significant influence on your body's lipid metabolism, it is important to follow a diet tailored to your genes.

Arrhythmia

Long QT syndrome is a life-threatening disease that can lead to sudden cardiac death in people with otherwise perfect health.

The heartbeat is triggered by a recurring electrical pulse which propagates through the heart. The time to initiate a heartbeat up to the point at which the cells are ready for the next heartbeat, is called the QT interval. If this interval is particularly long, it increases the risk of symptoms such as paroxysmal tachycardia, arrhythmia, vertigo or loss of consciousness. In severe cases, such episodes end in cardiac arrest due to ventricular fibrillation. However, most people with this condition have no symptoms until a life-threatening condition develops. The symptoms usually occur during physical exertion or stressful situations. A resting ECG (a measurement of the heart rate in the resting state) and a gene analysis help to better identify the risk.

A long QT interval is usually not noticeable: more than half of the patients with long QT syndrome experience no symptoms. When symptoms do occur, they are caused by potentially life-threatening heart rhythm disorders that are signs of serious disease. Palpitations may be sustained (more than 30 seconds) or intermittent, and sometimes remain unnoticed depending on the following: the duration and pulse rate, body position and the general constitution, dizziness, loss of consciousness or even cardiac arrest. Thus, they may lead to sudden cardiac death. Since tachycardia occurs suddenly and usually during exercise or in stressful situations, the symptoms are often unexpected, and observed because they affect our general state of well-being.

People with this genetic risk should take steps to minimize symptoms. The steps include medical heart rate monitoring in high-risk situations such as: cardiovascular disease, diabetes, morbidly excessive weight, age over 55 years, extreme physical activity, and taking certain medications. If a prolonged QT interval is diagnosed in these situations, medical treatment may be necessary. Thanks to genetic analysis, you can find out if you are in a risk group and take the necessary precautions. Serious consequences, such as sudden death, can usually be prevented.

Relevant genes for cardiovascular disease

The scientific community has linked several genes and polymorphisms to a risk of various cardiovascular diseases. An analysis of these polymorphisms allows us to determine your genetic risk for these diseases, as well as some other genetic traits linked to this disease.

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
CDH13	rs8055236	G>T	T/G
CHDS8	rs1333049	G>C	G/G
APOA5	rs662799	-1131T>C	A/A
PON1	rs662	Q192R	A/A
PON1	rs854560	L55M	A/A
APOB	rs5742904	R3500Q	G/G
SREBF2	rs2228314	Gly595Ala	C/C
NOS3	Ins/Del Int. 4	Ins/Del Intron 4	Ins/Ins
NOS3	rs2070744	-786 T/C	T/T
NOS3	rs1799983	Glu298Asp	G/T
APOA1	rs670	-75G > A	G/G
MTRR	rs1801394	Ile22Met	G/G
MMP3	rs3025058	5A/6A	T/del
GJA4	rs1764391	Pro319Ser	T/T
ITGB3	rs5918	Leu33Pro	T/T
CETP	rs708272	Taq1(B1>B2)	C/T
MTHFR	rs1801133	C>T	C/C
APOE	rs429358	T>C	T/T
APOE	rs7412	T>C	T/C
ApoE type	combination	E2/E3/E4	E2/E3
NOS1AP	rs16847548	T>C	T/T
NOS1AP	rs12567209	G>A	G/G
NOS1AP	rs10494366	T>G	T/T
CYP1A2	rs762551	C/A Pos. -163	A/A

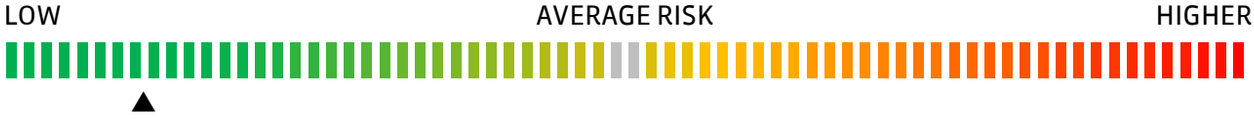
LEGEND: rsNCBI = description of examined genetic variation, POLYMORPHISM = form of the genetic variation, GENOTYPE = personal analysis result

Summary of effects

This analysis examines numerous genes that contribute to risks for several different cardiovascular conditions. Many of these risk variations are common, and almost everyone has some genes that increase the risk of cardiovascular disease. If you have an unusually small number of genes that increase risk, your genes may actually reduce your risk of developing cardiovascular disease. Here you can see a summary of the influence your genetic variations have on your health:

- You do not have an elevated risk of coronary heart disease
- No predisposition to elevated LDL cholesterol levels
- You have a slight predisposition for high triglyceride levels
- Vitamin B2 does not lower your homocysteine levels
- Omega-3 fatty acids reduce your HDL cholesterol levels
- Aspirin can be effective in preventing arterial thrombosis
- Predisposition for slightly lowered HDL cholesterol values
- No predisposition to increased QT-interval duration

Your risk of coronary heart disease, atherosclerosis and heart attack



Your LDL cholesterol levels are likely to be

NORMAL INCREASED

Your HDL cholesterol levels are likely to be

NORMAL LOW

The effect of omega-3 on HDL cholesterol

IMPROVEMENT DETERIORATION

Your triglyceride levels are likely to be

NORMAL INCREASED

Your homocysteine levels are likely to be

NORMAL INCREASED

Effect of vitamin B2 on homocysteine

REDUCTION NO

Effect of aspirin on arterial thrombosis (blood clots)

PROTECTION NO EFFECT

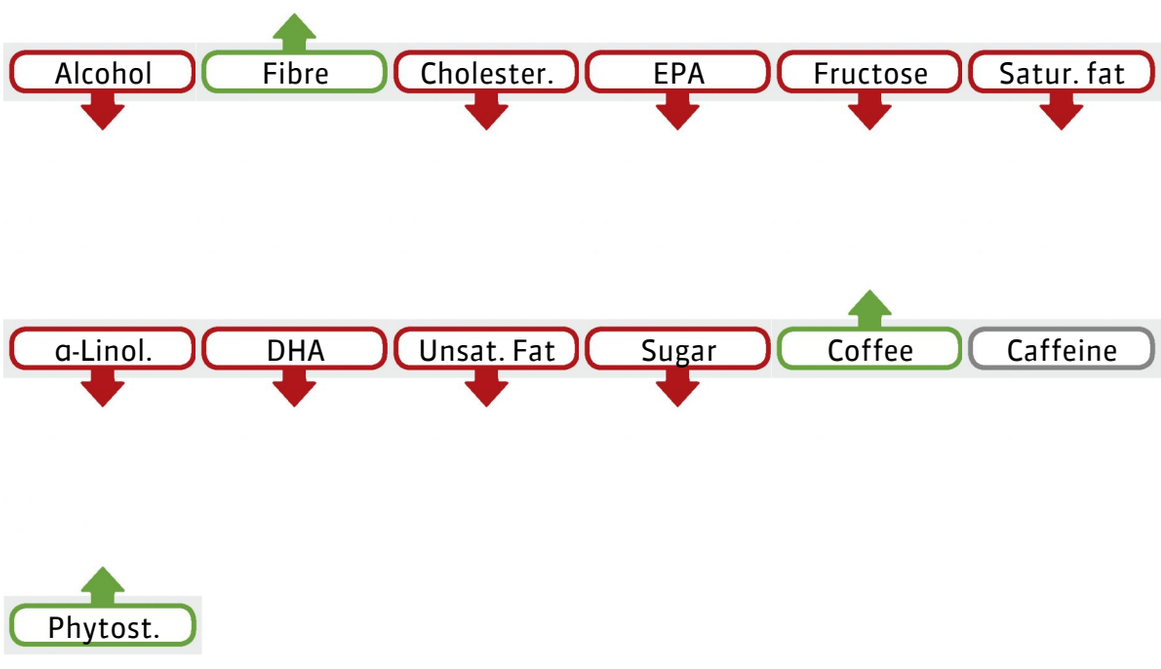
Risk of sudden cardiac death (QT)

NORMAL INCREASED

Nutritional Genes - Heart

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:

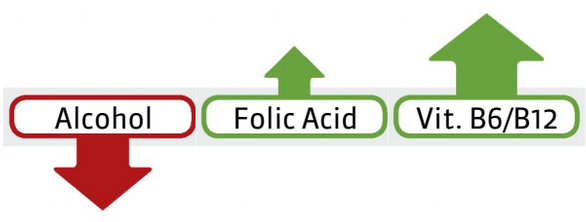


Legend: GREEN ARROWS > this nutrient or substance is classed as healthy for your genetic profile. Try to increase the intake of this substance. RED ARROWS > this substance is classed as unhealthy for your genetic profile. Try to reduce your intake of the substance. NO ARROWS > There is no effect of the nutrient on the genetics of this section. PLEASE NOTE! This interpretation only considers your genetic profile of this section.

Nutritional Genes - Blood

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.

Nutritional Genes - Vitamin B2

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:

Vit B2

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

Based on the genes tested, you have no increased genetic risk for metabolic disorders and your risk is the same as the general population. However, even though you do not have genetic predisposition to these diseases, you can still get sick, especially if you follow an unhealthy lifestyle that leads to high levels of fat in the blood. We recommend you have your cholesterol and triglycerides checked every five years after age 20. If these values increase over time, you should take steps to reduce the risk of atherosclerosis. The specific steps will depend on the test results, so you should follow your doctor's advice.

Preventive measures

- Do sports or regular exercise. The best exercises are endurance sports (walking, Nordic walking, cycling, swimming, weight training, etc.), and watch your weight. Ideally, you should do at least 30 minutes of exercise, 5 days a week.
- Smoking greatly increases your risk for vascular disease along with its many other negative health effects. Quitting smoking is one of the most important ways to improve your health.
- In general, eat low-fat meals (fish, poultry and lean meats are recommended, but fatty meats like sausages, bacon and fat cheese should be reduced).
- You should also eat only low-fat dairy products, e.g. low-fat milk, low-fat cheese and low-fat yogurt.
- Eat as little animal products as possible and use mainly vegetable oils.
- Eat fruits and vegetables several times daily.

Omega-3-fatty acids

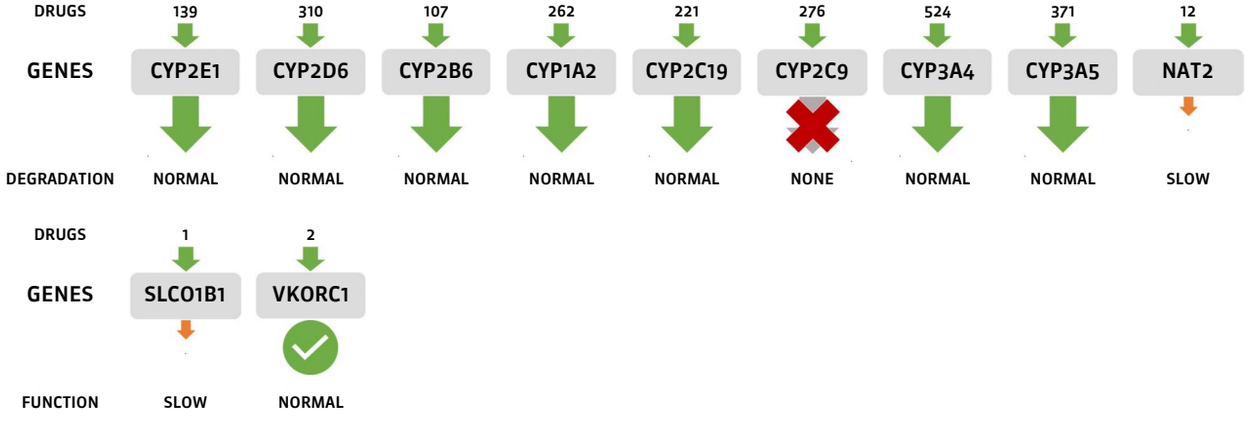
- Omega-3 fatty acids are commonly recommended for high cholesterol; however, because of your APoA1 gene, omega-3 fatty acids could raise your cholesterol levels. Phytosterols can be used as an alternative supplement.

Coffee

- Although coffee contains healthy ingredients, the caffeine contained in the coffee (if not metabolized by CYP1A2 gene) can increase the risk of cardiovascular diseases. Your CYP1A2 gene works normally and therefore moderate coffee consumption (2-5 cups a day) is healthy for your cardiovascular system.

Medical treatment is recommended when dietary changes and exercise do not lower cholesterol and triglycerides to a normal level. There are multiple options: statins, bile acid binders, fibrates, niacin (vitamin B3), and cholesterol synthesis and absorption inhibitors. Your doctor will decide which drug is suitable for you. It is particularly important to take action to prevent heart disease because treatment after symptoms develop can only slow down the progress of the disease.

Drug compatibility



Effect on relevant medication

	Effect	Breakdown	Dose
Abciximab	✓	✓	✓
Alprenolol	✓	✓	✓
Amlodipine	✓	↑	↑
Atorvastatin	✓	↑	↑
Bendroflumethiazide	✓	✓	✓
Bisoprolol	✓	↑	↑
Captopril	✓	✓	✓
Cerivastatin	✓	↑	↑
Cilnidipine	✓	✓	✓
Clopidogrel	✓	↑	✓
Debrisoquine	✓	✓	✓
Dipyridamole	✓	✓	✓
Dorzolamide	✓	✓	✓
Encainide	✓	✓	✓
Eprosartan	✓	✓	✓

	Effect	Breakdown	Dose
Acebutolol	✓	✓	✓
Amiloride	✓	✓	✓
Anagrelide	✓	✓	✓
Barnidipine	✓	✓	✓
Benidipine	✓	✓	✓
Bumetanide	✓	✓	✓
Carvedilol	✓	✗	✗
Chlortalidone	✓	✓	✓
Cilostazol	✓	↑	↑
Colestipol	✓	✓	✓
Digoxin	✓	✓	✓
Disopyramide	✓	↑	↑
Dronedarone	✓	✓	✓
Enoxaparin	✓	✓	✓
Eptifibatide	✓	✓	✓

	Effect	Breakdown	Dose
Acetylsalicylic Acid	✓	✗	✗
Amiodarone	✓	↓	↓
Atenolol	✓	✓	✓
Benazepril	✓	✓	✓
Betaxolol	✓	✓	✓
Candesartan	✓	↓	↓
Celiprolol	✓	✓	✓
Cilazapril	✓	✓	✓
Clevidipine	✓	✓	✓
Cyclopenthiazide	✓	✓	✓
Diltiazem	✓	↑	↑
Dofetilide	✓	↑	↑
Enalapril	✓	✓	✓
Eplerenone	✓	↑	↑
Esmolol	✓	✓	✓

	Effect	Breakdown	Dose
Ezetimibe	✓	✓	✓
Fenofibrate	✓	✓	✓
Fondaparinux	✓	✓	✓
Gemfibrozil	✓	↑	↑
Ibutilide	✓	✓	✓
Isosorbide Mononitrate	✓	✓	✓
Lacidipine	✓	↑	↑
Lisinopril	✓	✓	✓
Manidipine	✓	✓	✓
Metoprolol	✓	✓	✓
Nadolol	✓	✓	✓
Nifedipine	✓	↑	↑
Nisoldipine	✓	↑	↑
Perhexiline	✓	✓	✓
Pitavastatin	✓	✓	✓
Procainamide	✓	✓	✓
Quinapril	✓	✓	✓
Ranolazine	✓	✓	✓
Sotalol	✓	✓	✓
Telmisartan	✓	✓	✓
Ticagrelor	✓	✓	✓
Tocainide	✓	✓	✓
Triamterene	✓	✓	✓
Vernakalant	✓	✓	✓

	Effect	Breakdown	Dose
Felodipine	✓	↑	↑
Flecainide	✓	✓	✓
Fosinopril	✓	✓	✓
Hydralazine	✓	✓	✓
Indapamide	✓	✓	✓
Isradipine	✓	↑	↑
Lercanidipine	✓	↑	↑
Losartan	✗	↓	✗
Methazolamide	✓	✓	✓
Mexiletine	✓	✓	✓
Nebivolol	✓	✓	✓
Nilvadipine	✓	✓	✓
Nitrendipine	✓	↑	↑
Perindopril	✓	✓	✓
Prasugrel	✓	✓	✓
Propafenone	✓	✓	✓
Quinidine	✓	↑	↑
Rosuvastatin	✓	✓	✓
Sparteine	✓	✓	✓
Theobromine	✓	✓	✓
Timolol	✓	✓	✓
Torasemide	✓	✗	✗
Valsartan	✓	✗	✗
Warfarin	✓	✗	✗

	Effect	Breakdown	Dose
Fendiline	✓	✓	✓
Fluvastatin	✓	✓	✓
Gallopamil	✓	✓	✓
Hydrochlorothiazide	✓	✓	✓
Irbesartan	✓	✗	✗
Labetalol	✓	✓	✓
Lidocain	✓	✓	✓
Lovastatin	✓	↑	↑
Metolazone	✓	✓	✓
Moexipril	✓	✓	✓
Nicardipine	✓	↑	↑
Nimodipine	✓	↑	↑
Penbutolol	✓	✓	✓
Pindolol	✓	✓	✓
Pravastatin	✓	✓	✓
Propranolol	✓	✓	✓
Ramipril	✓	✓	✓
Simvastatin	✓	↑	✗
Spironolactone	✓	✓	✓
Theophylline	✓	✓	✓
Tirofiban	✓	✓	✓
Trandolapril	✓	✓	✓
Verapamil	✓	↑	↑
Xipamide	✓	✓	✓

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.

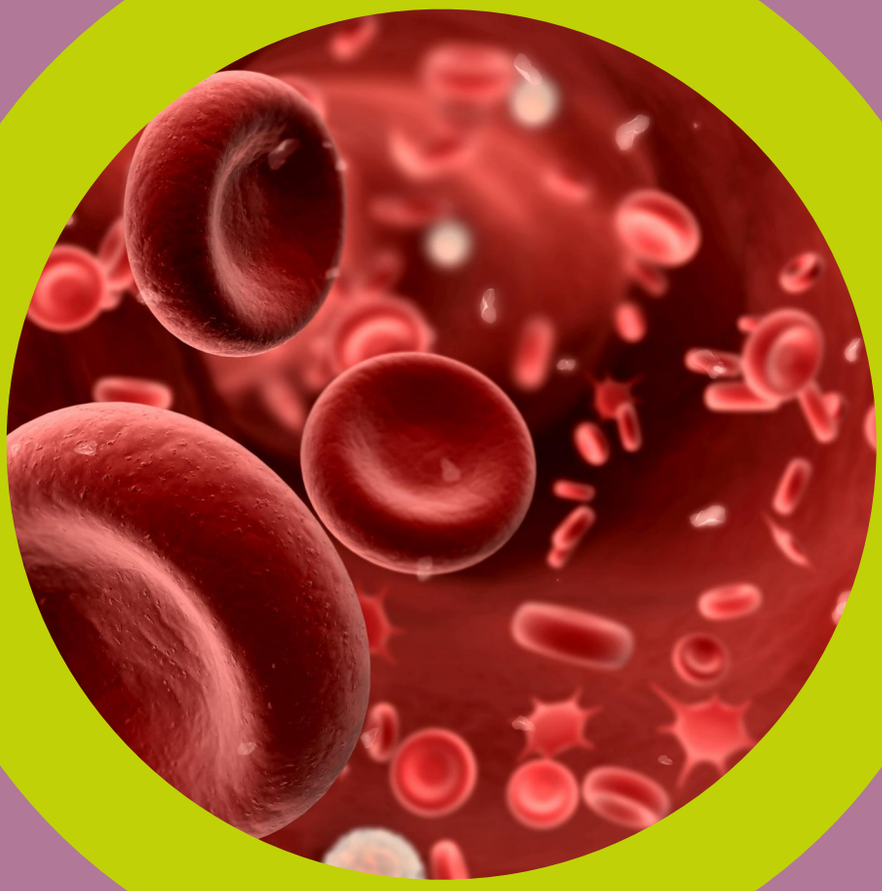

Effect: Normal. Degradation: Slower. Recommendation: Reduce the dosage.


Effect: Normal. Degradation: None. Recommendation: Alternative drug.


Effect: Lower. Degradation: Normal. Recommendation: Normal dosage.

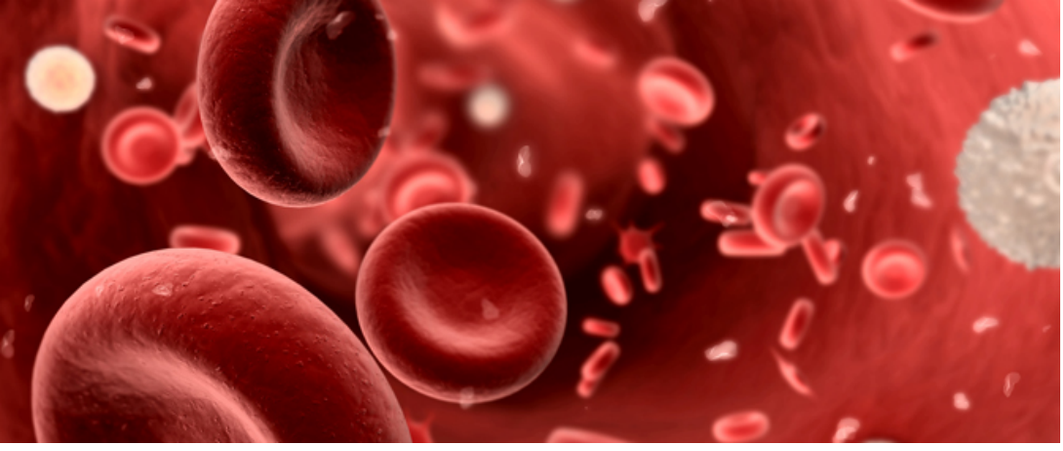

Effect: Lower. Breakdown: Lower. Recommendation: Reduce the dosage.


Effect: Stronger. Degradation: Stronger. Recommendation: Normal dosage.



Thrombo Sensor

Effectively prevent thrombosis



Thrombosis

Thrombosis is a disease where blood clots form in the bloodstream. These clots can obstruct certain blood vessels and reduce blood flow to important organs, like the heart or to areas in the brain. This may lead to damage or even the death of the affected tissue. When blood supply to part of the brain is impaired, a stroke may occur. If blood supply to the heart is affected, it can cause a heart attack. The most common form of thrombosis is decreased blood circulation in the legs caused by a blood clot. The risk in this case is if the clots dissolve they can move along the bloodstream and restrict the blood flow to other important organs ,like the brain, heart or the lungs.

Genetic screening tests for thrombosis are unfortunately rarely performed. Since there are no obvious symptoms until after the event, most people do not know that they are genetically predisposed to thrombosis.

Therefore, a genetic predisposition is usually not detected until after the occurrence of thrombosis; this, however, may have fatal consequences. Genetic screening tests are still performed too infrequently even though they increase awareness of the risk so that the necessary preemptive measures can be taken. In some cases, it may even allow the individual to avoid the disease altogether. Several genes prevent the formation of blood clots in the veins. If one of these genes is defective, it cannot perform its task and the risk of forming a blood clot increases significantly. Everyone has two genes of each type but about 1 in 20 people carries a defect in at least one gene, thus being a carrier with an approximate 8- fold higher risk of thrombosis than the general population. About 1 in 200 people carries an error in both genes of a genotype and has a 80 -fold higher thrombosis risk. Having defective genes does not necessarily mean the patient will suffer from thrombosis because only a fraction of those affected will develop the disease. Other factors also contribute strongly, such as excessive weight, bed rest and inactivity, prolonged air travel, contraceptive pills, pregnancy, etc.

This is why genetic testing is so important; if you know your genetic health risk, you can take precautionary measures and, in most cases, even prevent the occurrence of thrombosis.

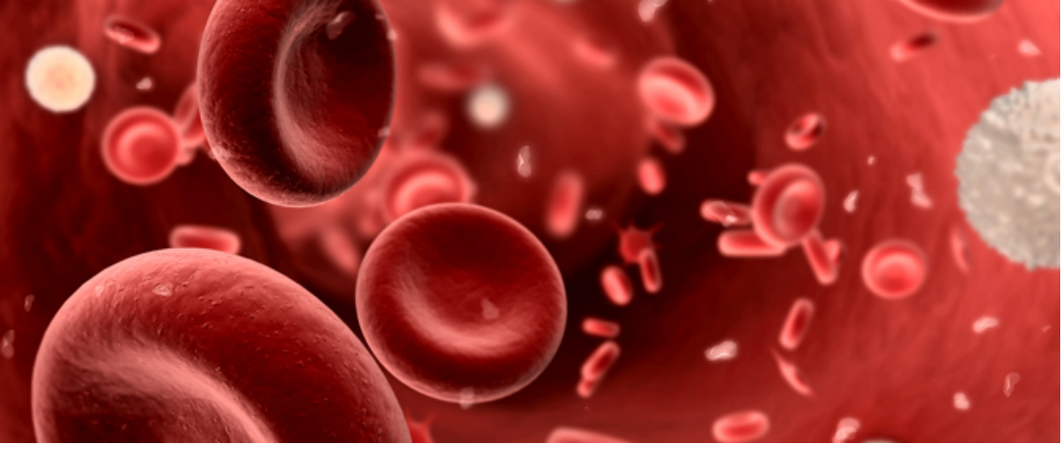
Risk of thrombosis during pregnancy

Studies have shown that the risk of thrombosis during pregnancy is between 4 to 10 times higher than the risk in non-pregnant women. This risk increases further in the months after delivery by between 10- 20 times. What is striking is that most cases occur in young mothers (15-19 years). Approximately 1 in 20 women is genetically predisposed to thrombosis even without pregnancy, and has an approximately 8-80-fold higher risk of thrombosis than the general population. If a genetically predisposed woman is pregnant, the combination of these two risk factors leads to a dangerous combination of genetic defect and risk situation, increasing the risk of thrombosis by 60-fold and leading to life-threatening conditions.

It is estimated that thrombosis is responsible for about one third of deaths in pregnancy, and between 30-60% of women who develop thrombosis are genetically predisposed. Therefore, it is already widely medically accepted that genetically predisposed women should take medication to prevent thrombosis during pregnancy.
Unfortunately, very few women know of their genetic risk of thrombosis and so a significant percentage of these women remain untreated and unprotected. The treatment consists of daily administration of low molecular weight heparin, a drug that prevents the formation of blood clots in the veins. Medication will be carefully chosen so that it does not enter the fetal bloodstream. Heparin can be given to a pregnant woman without harming her fetus, under medical supervision. When used consistently, these drugs ensure that thrombosis is prevented both before and after childbirth.

Risk of thrombosis and the use of hormonal compounds:

Hormone-containing medications are used by many women, either in the form of contraceptives or as treatment for complications caused by reduced hormone production after menopause. Although hormones have numerous benefits, they increase the risk of thrombosis. This risk is very small, unless there are additional risk factors, such as a genetic predisposition. A genetic predisposition for thrombosis significantly increases the risk; in combination with hormonal preparations the risk increases approximately 15 times, and in some cases even to more than 80 times. For women with a genetic predisposition to thrombosis the use of hormone preparations is therefore strongly discouraged, since it can lead to life-threatening conditions.



Relevant genes for thrombosis

Three genetic variations have been identified that can significantly increase the risk of thrombosis. An analysis of these three polymorphisms will determine your risk of developing a thrombosis which can then be reduced with specific preventive measures. The following genes have an impact on your risk of thrombosis:

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
Factor-V	rs6025	G>A	G/G
Factor-II	rs1799963	G>A	G/G
PAI1	rs1799889	G>del	del/del
MTHFR	rs1801133	C>T	C/C
ITGB3	rs5918	T>C	T/T

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

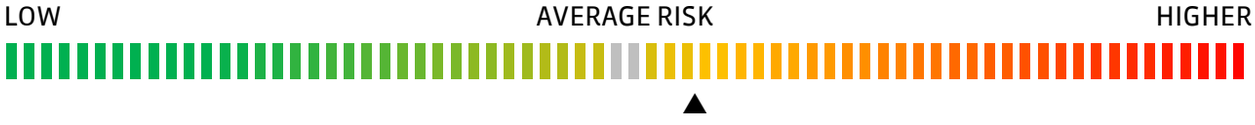
Summary of effects

Certain genes are responsible for preventing blood clot formation in the blood vessels. Variations in these genes may interfere with this process, and therefore increase your risk of blood clot formation and subsequently, thrombosis. Below is a summary of the impact that genetic variations may have on your health:

- Your risk of developing venous thrombosis is not increased
- Therefore, hormonal preparations do not increase your risk of thrombosis.
- Your risk of developing arterial thrombosis is approximately 1.84 -times increased
- Aspirin can be effective in preventing arterial thrombosis

Risk of venous thrombosis

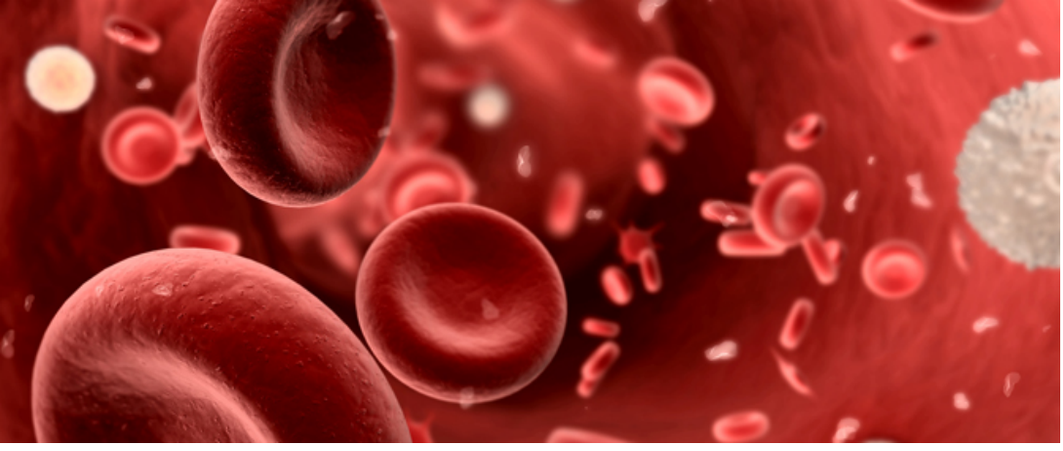
Risk of arterial thrombosis



Risk of gestational thrombosis

Is your risk significantly increased by hormone preparations?

Effect of aspirin on arterial thrombosis (blood clots)



Prevention

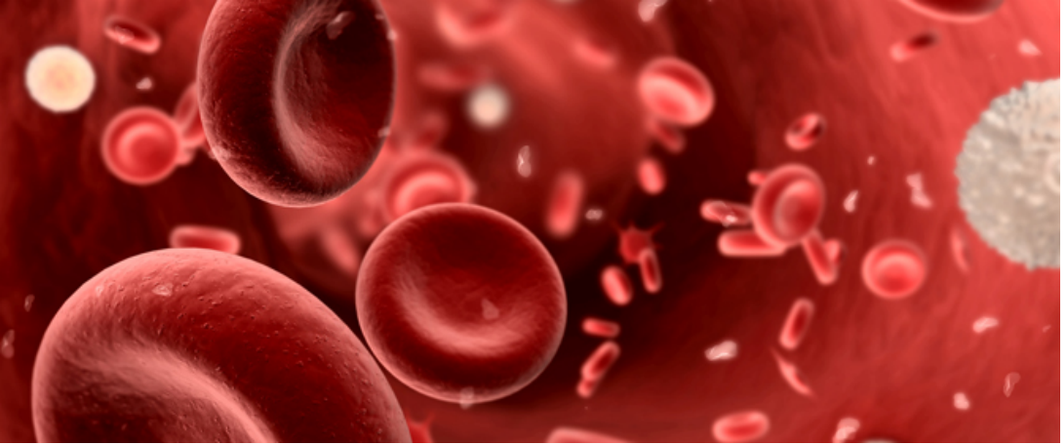
Based on your genetic profile, you have an increased risk of thrombosis. Therefore, it is highly recommended that you take some precautionary measures to reduce your risk. A genetic predisposition to thrombosis only increases your risk of developing a clot but it does not mean that you will definitely experience thrombosis. Complications occur only when a blood clot forms in the blood vessels, which then impairs blood supply to certain parts of the body. The preemptive measures focus on preventing this. You have a significantly higher risk of developing thrombosis and other factors may increase the risk even further.

Special high-risk situations in which precautionary measures should be considered:

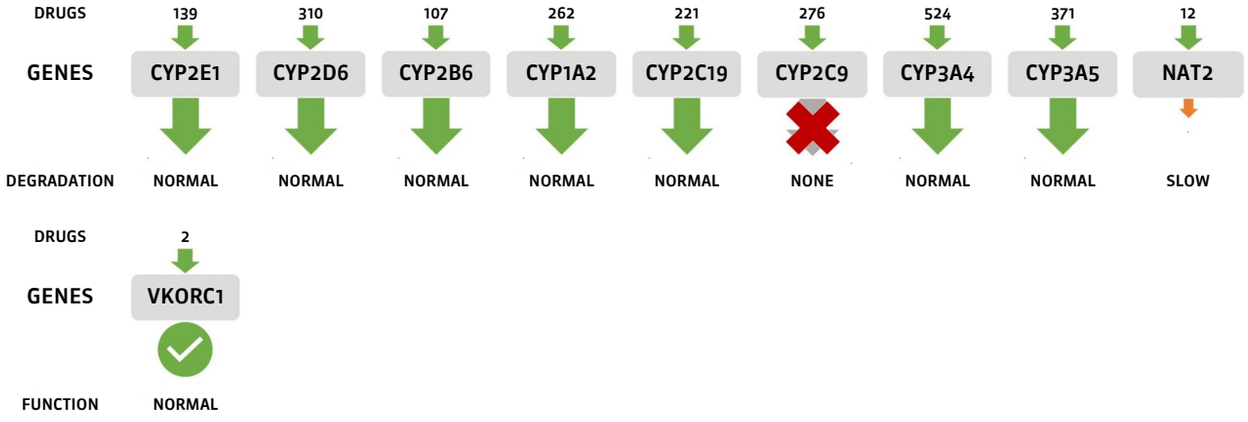
- Bed rest and inactivity, eg. when wearing a plaster cast. In this case, heparin injections are recommended after surgery (especially after surgery in the abdomen, hip or knee surgery).
- Symptoms can frequently occur during pregnancy. However, this increased risk of thrombosis should not be an obstacle to pregnancy, but it will require closer medical supervision and possibly blood-thinning drugs, such as heparin. Heparin does not enter breast milk, so women can take it even while nursing.
- Cancers or diseases that are associated with the loss of fluid (for example, diarrhoea). Varicose veins in the legs. Heart diseases such as heart failure or after a heart attack.

Certain medications can cause additional complications. Discuss the medication you are taking and your genetic risks with your doctor. The following medications may be unsuitable for those affected:

- The "skin pill"- hormonal therapy with ethinylestradiol and cyproterone
- Drugs for the treatment of breast cancer– tamoxifen
- Vaccine for the HPV virus
- Certain blood pressure medications
- Contraceptive pill - ethinylestradiol and drospirenone
- Contraceptive hormone ring - etonogestrel and ethinylestradiol
- Sedative - Thalidomide drug for the treatment of anemia
- Doping drugs – Erythropoietin
- Cortisone
- Menopausal preparations



Drug compatibility



Effect on relevant medication

	Effect	Breakdown	Dose
Acenocoumarol	✓	↓	✓
Clopidogrel	✓	↑	✓
Fondaparinux	✓	✓	✓
Ticagrelor	✓	✓	✓
Warfarin	✓	✗	✗

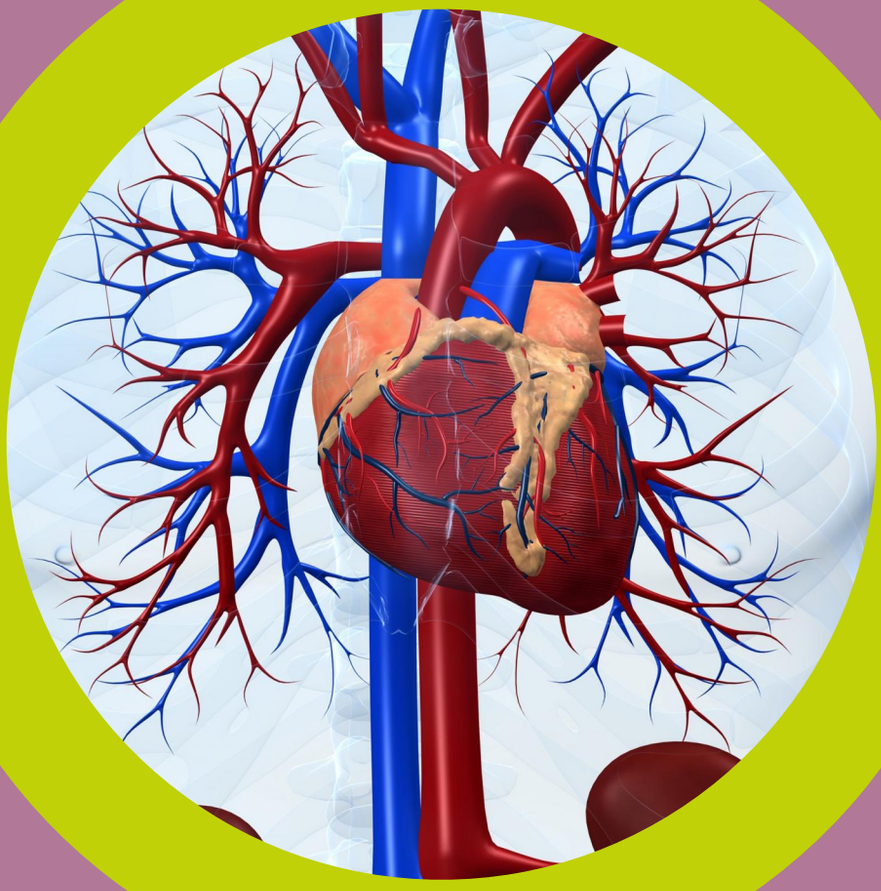
	Effect	Breakdown	Dose
Acetylsalicylic Acid	✓	✗	✗
Desirudin	✓	✓	✓
Prasugrel	✓	✓	✓
Ticlopidine	✓	↑	↑

	Effect	Breakdown	Dose
Bemiparin	✓	✓	✓
Enoxaparin	✓	✓	✓
Reteplase	✓	✓	✓
Urokinase	✓	✓	✓

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!

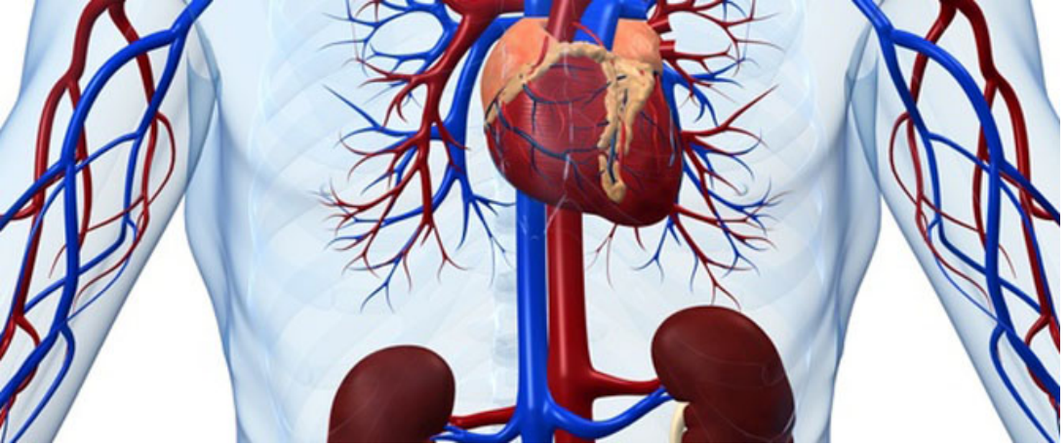
Legend:

- ✓ ✓ ✓ 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.



Hypertension Sensor

Effective prevention and treatment of hypertension



Hypertension

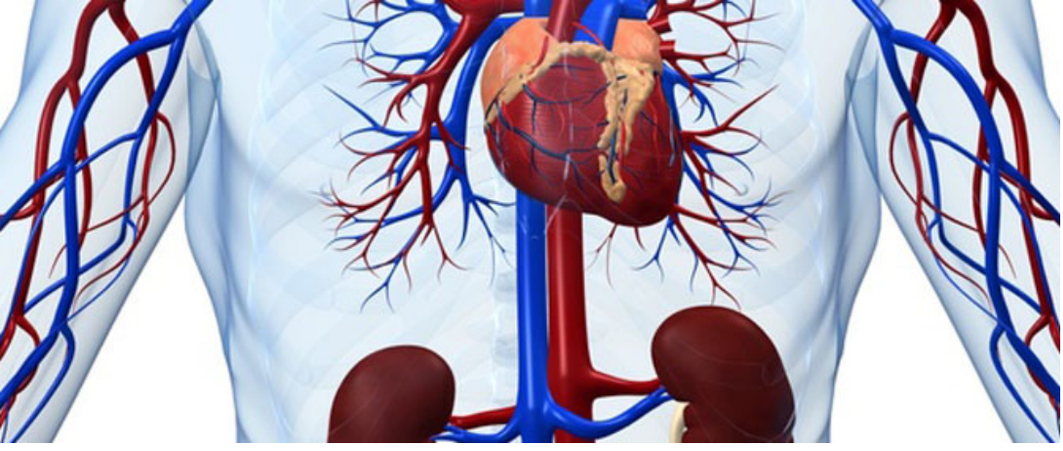
Hypertension is a condition in which the blood pressure of the vascular system is chronically elevated. A chronic systolic blood pressure higher than 140mmHg or a diastolic blood pressure greater than 90 mmHg (both measured after 10 minutes of sitting) are considered high blood pressure. This measurement method is the current standard because the blood pressure often decreases after sitting down, and increases when physical activities are performed.

This disease is very common and it is estimated that 29% of the total population suffers from it. Blood pressure tends to increase with age. High blood pressure is particularly dangerous because it is not always noticeable. Some of the symptoms are: morning headache, dizziness, nausea, nosebleeds, fatigue or insomnia. Often, the disease progresses without symptoms, and it is only identified by the consequential damages, which is why it is also known as the "silent killer".

Hypertension is a major risk factor in the development of atherosclerosis (a hardening of the arteries), especially if other risk factors such as excessive weight, diabetes, or elevated cholesterol/triglyceride levels are present. The resulting cardiovascular diseases, like coronary heart disease (CHD), heart attack, heart failure, kidney failure, stroke and vascular disease cause about 45% of deaths in men and 50% of deaths in women.

Apart from the increased risk of atherosclerosis, a chronically high blood pressure also causes damage to the heart muscle. The muscles become thicker and stiffer so that the heart cannot easily relax in diastole (relaxation phase) and draw in the blood. This causes a poor filling of the heart, and the appearance of heart failure symptoms. If left untreated, high blood pressure can damage the retina causing blindness, or damage the kidneys where kidney function is seriously reduced. Today, treatments exist to lower high blood pressure and alleviate the side effects. These modern drugs increase life expectancy and also improve the quality of life tremendously.

Several genes are responsible for blood pressure regulation. Each one can carry a trait that increases the risk of developing high blood pressure. A person who is aware of their personal risk can take preventative measures to lower blood pressure and also consult a doctor about their risk factors and condition. These steps can usually prevent the severe and often fatal diseases that are caused by long-term high blood pressure.



Relevant genes for hypertension

Several genetic variations have been identified that can significantly increase the risk of hypertension. An analysis of these polymorphisms can determine the risk of developing hypertension and how to reduce it with specific preventive measures. The genetic analyses also help in identifying the most effective therapy for lowering blood pressure. The following genes have an impact on your blood pressure:

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
AGT	rs699	T>C	T/T
ADRB1	rs1801253	G>C	G/C
GNB3	rs5443	C>T	C/T
MTHFR	rs1801133	C>T	C/C

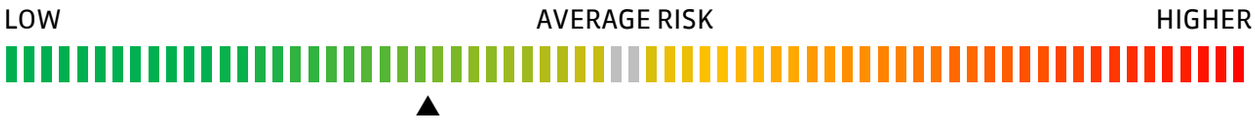
LEGEND: rsNCBI = description of examined genetic variation, POLYMORPHISM = form of the genetic variation, GENOTYPE = personal analysis result

Summary of effects

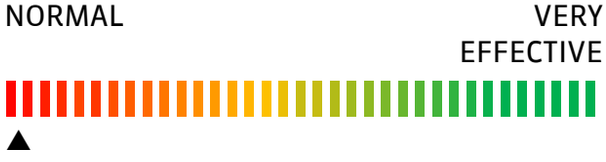
The analysed genes have an influence on your risk of hypertension, which can be also measured in the conventional way. Therefore, the benefit of this analysis is primarily to identify high blood pressure through regular examinations, properly treat it through lifestyle changes, and if necessary, use the most effective drug therapy. Here you can see a summary of the impact your genetic variations have on your health:

- You have no increased risk for elevated blood pressure.
- A reduced dietary salt intake is effective on average to reduce the risk of hypertension
- Taking vitamin B2 has no effect for your blood pressure

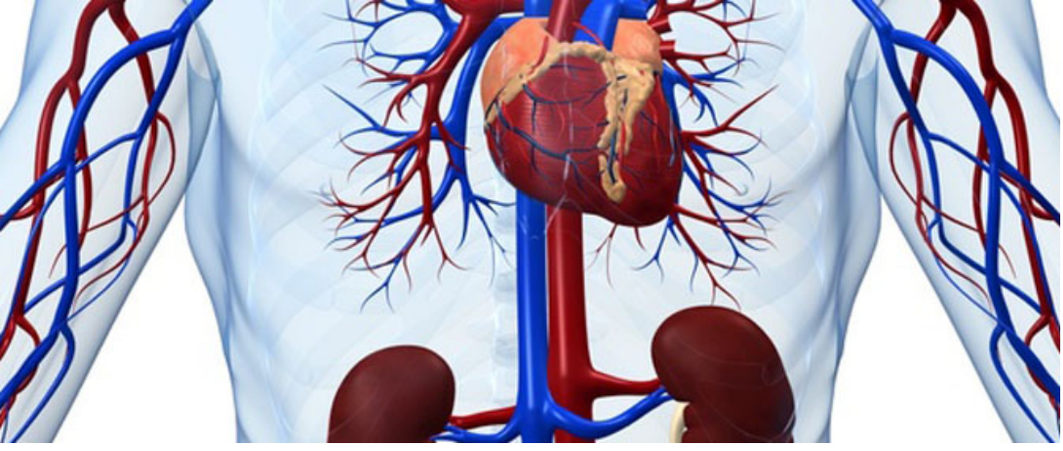
Your risk of hypertension



Lower blood pressure due to reduced salt consumption



Lower blood pressure by taking vitamin B2

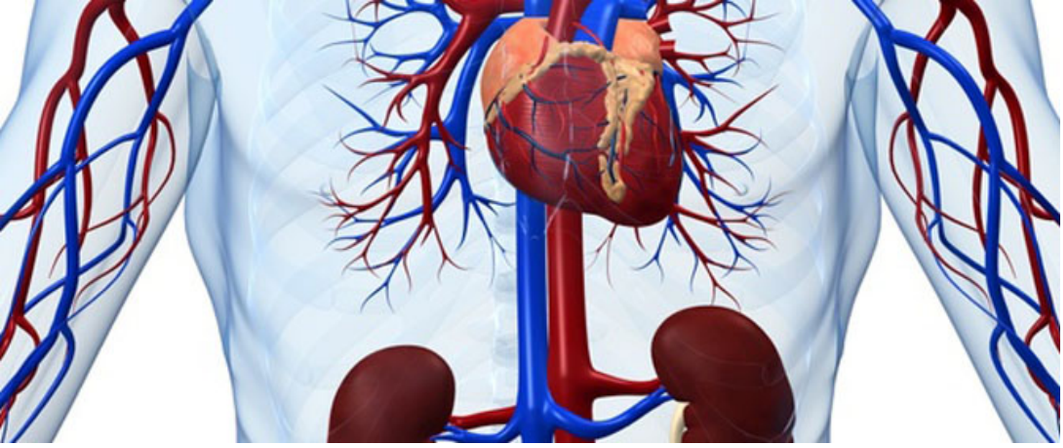


Nutritional Genes - Blood pressure

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

Based on your genetic profile, you have no increased risk of high blood pressure. Therefore, you do not have to follow any specific preventive measures, but only the general guidelines of a healthy lifestyle. However, some people develop high blood pressure even without genetic defects due to their own unhealthy lifestyle. If you suffer from high blood pressure, despite your genetic profile, you can take the following precautionary measures.

Prevention

In addition to genetic factors, environment and lifestyle also play a crucial role in the development of high blood pressure. Therefore, it is important for you to understand the risk factors and to modify your lifestyle to avoid these risk factors.

Giving up smoking is one of the most important ways to lower blood pressure. People who quit smoking in middle age have a similar life expectancy as those who have never smoked. Smoking also affects the effectiveness of blood pressure medication.

After the age of 40, limit the amount of alcohol you consume because alcohol has a direct impact on blood pressure, and moderate or heavy drinking increases the risk of stroke. Light alcohol consumption of 250mL of red wine a day may lower your blood pressure by 2-4mmHg.

Obesity is an important risk factor and also increases your blood pressure. Therefore, reducing your weight will have a major impact and also lower your blood pressure. Losing 10kg of weight will lower your blood pressure by 5-20mmHg. Keep your BMI (Body Mass Index) under 25 to reduce your risk.

Regular physical activity such as swimming, running or walking, even at low intensity, lowers blood pressure by 4-9mmHg. You can reduce blood pressure by getting 30 minutes of exercise several times a week. However, intensive exercise is not recommended.

Salt consumption is also an important risk factor for high blood pressure. You should limit sodium intake to 2500mg or less. This is expected to reduce blood pressure by 8mmHg. Due to a special genetic variation, a decrease in daily salt intake can be particularly effective.

A healthy diet, with a large amount of fruit and vegetables, and low in saturated fats can reduce blood pressure by 8-14mmHg.

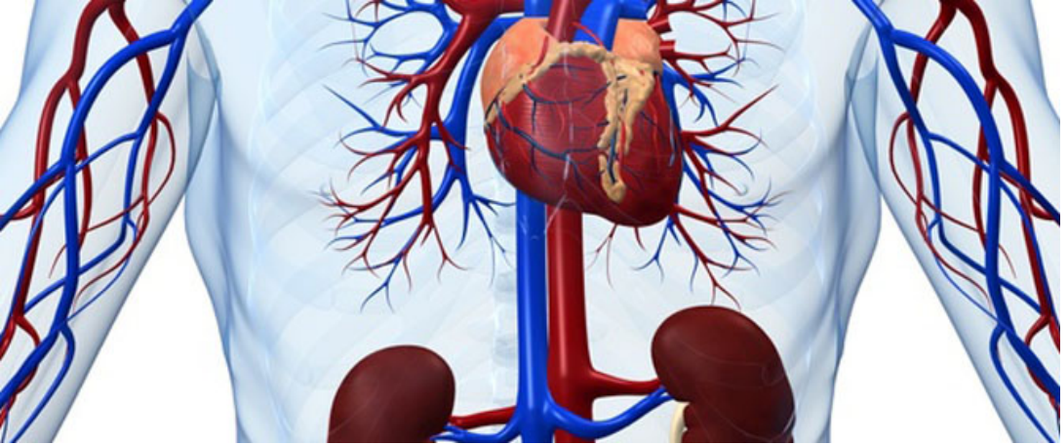
Medical care and treatment

Monitor your blood pressure to see how effective your lifestyle changes are. Ask your doctor to measure your blood pressure regularly or measure it yourself. Use the following guidelines to

determine how often to check:

Blood pressure	Systolic	Diastolic
Optimal blood pressure	under 120	under 80
Normal blood pressure	120-129	80-84
High-normal blood pressure	130-139	85-89
Mild hypertension (stage 1)	140-159	90-99
Moderate hypertension (stage 2)	160-179	100-109
Severe hypertension (stage 3)	under 180	above 110
Isolated systolic hypertension	above 140	under 90

If your blood pressure is normal, check it every week and try to keep it within the normal range by following the measures described above.
If your blood pressure is too high, take steps to lower it. If those measures do not lower your blood pressure to normal levels, talk to your doctor about possible medications to lower blood pressure. The choices are ACE inhibitors, AT1 antagonists, beta blockers, diuretics and calcium antagonists.



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
Acebutolol	✓	✓	✓
Amlodipine	✓	↑	↑
Bisoprolol	✓	↑	↑
Candesartan	✓	↓	↓
Clevidipine	✓	✓	✓
Doxazosin	✓	✓	✓
Eprosartan	✓	✓	✓
Furosemide	✓	✓	✓
Hydrochlorothiazide	✓	✓	✓
Lercanidipine	✓	↑	↑
Metolazone	✓	✓	✓
Nadolol	✓	✓	✓
Nifedipine	✓	↑	↑
Olmesartan Medoxomil	✓	✓	✓
Prazosin	✓	✓	✓
Ramipril	✓	✓	✓
Telmisartan	✓	✓	✓
Triamterene	✓	✓	✓

	Effect	Breakdown	Dose
Aliskiren	✓	↑	↑
Atenolol	✓	✓	✓
Bosentan	✓	↓	↓
Captopril	✓	✓	✓
Clonidine	✓	✓	✓
Enalapril	✓	✓	✓
Felodipine	✓	↑	↑
Guanfacine	✓	✓	✓
Irbesartan	✓	✗	✗
Lisinopril	✓	✓	✓
Metoprolol	✓	✓	✓
Nebivolol	✓	✓	✓
Nisoldipine	✓	↑	↑
Perindopril	✓	✓	✓
Propranolol	✓	✓	✓
Reserpine	✓	✓	✓
Terazosin	✓	✓	✓
Valsartan	✓	✗	✗

	Effect	Breakdown	Dose
Amiloride	✓	✓	✓
Benazepril	✓	✓	✓
Bumetanide	✓	✓	✓
Carvedilol	✓	✗	✗
Diltiazem	✓	↑	↑
Eplerenone	✓	↑	↑
Fosinopril	✓	✓	✓
Hydralazine	✓	✓	✓
Labetalol	✓	✓	✓
Losartan	✗	↓	✗
Minoxidil	✓	✓	✓
Nicardipine	✓	↑	↑
Nitrendipine	✓	↑	↑
Pindolol	✓	✓	✓
Quinapril	✓	✓	✓
Spironolactone	✓	✓	✓
Toremifene	✓	↑	↑
Verapamil	✓	↑	↑

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.

PHARMACO GENETICS

ONCOLOGY

CARDIOVASCULAR SYSTEM

NEUROLOGY

METABOLISM

MOVEMENT

DIGESTION

OPHTHALMOLOGY

ODONTOLOGY

OTHERS

SCIENCE

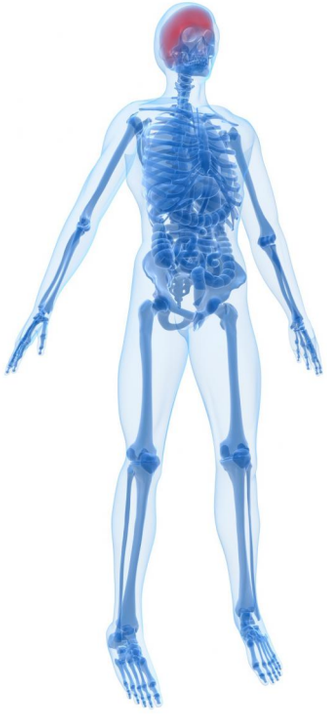
ADDITIONAL INFORMATION

Alzheimer Sensor

Risk assessment, prevention and better treatment

Alzheimer's disease

Alzheimer's disease (often simply called Alzheimer's) is a disease characterized by a progressive loss of certain brain cells. The cause of Alzheimer's is not fully understood. However, certain genetic traits have been clearly linked to a significantly increased risk of developing the disease. These traits cause abnormally folded proteins to accumulate in certain regions of the brain and allow for the development of large numbers of toxic molecules, known as free radicals, that damage brain cells. The damaged brain cells in the affected regions of the brain slowly deteriorate.



Early signs of Alzheimer's can be detected as early as eight years before diagnosis. Examples of early symptoms include short term memory loss and difficulties with language, as well as depression and apathy. The disease is often not recognized until the person develops noticeable learning disorders; short term memory loss increases while long term memory remains unaffected. In advanced stages, persons diagnosed with Alzheimer's completely lose even basic skills and abilities; they may eventually cease to recognize close friends and family, or even day-to-day objects. Irritability and aggression are common and as the disease progresses, the person becomes increasingly dependent on caregivers.

Alzheimer's disease accounts for approximately 60% of the roughly 24 million diagnosed cases of dementia worldwide. The most common form affects individuals over the age of 65. Around 2% of 65-year-olds are affected whereas among 70-year-olds the figure rises to 3%. 6% of 75-year-olds and roughly 20% of 85-year-olds display symptoms of the disease.

So far, the scientific community has not found a cure for Alzheimer's disease. However, there are number of preventative measures that can be effective for people with a genetic predisposition to Alzheimer's. Memory training, changes in lifestyle, an appropriate diet and controlling certain other conditions can all play an important role in preventing Alzheimer's. These measures can delay the development of Alzheimer's for many years, or even prevent it entirely. It is therefore especially important for persons who carry these genetic defects to learn about their risk and begin preventative measures as early as possible.

Genes associated with Alzheimer's disease

A combination of two different polymorphisms plays a role in the development of Alzheimer's disease. There are combinations linked to a 15-fold higher risk of Alzheimer's. Other combinations are linked to a 30% reduction in the risk of Alzheimer's compared to the population average. Your gene analysis shows the following:

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
APOE	rs429358	T>C	T/T
APOE	rs7412	T>C	T/C
ApoE type	combination	E2/E3/E4	E2/E3

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

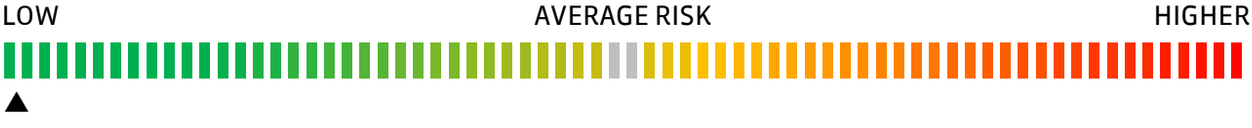
Summary of effects

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

- Based on your genetic profile, you have a lower than average risk of Alzheimer's

Your risk for Alzheimer's disease

LOW AVERAGE RISK HIGHER



Required antioxidants

NORMAL INCREASED

Nutritional Genes - Brain

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:

ß-Carotene	Alcohol	Fibre	High Glyc.	Coffee	EPA

Folic Acid	Fructose	Satur. fat	Potassium	Manganese	Sodium

DHA	Saccharose	Selenium	Unsat. Fat	Vit B2	Vitamin C

Vitamin D3	Vit. B6/B12	Vitamin E	Zinc	Sugar

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 profile shows that you have a lower than normal chance of developing Alzheimer's. However, you can reduce your risk even further by taking measures that reduce the likelihood of developing Alzheimer's. Clinical studies have shown that the following measures reduce the risk of Alzheimer's:

Exercise: studies show that regular physical activity reduces the risk of developing Alzheimer's. At least 15 minutes of physical activity, 3 days per week can reduce the risk by 40%. It is recommended that you follow an exercise program that you enjoy, and also consider joining an exercise group or gym.

Socializing: Studies have found that socially active people have a lower risk of developing dementia. Regular contact with friends or participation in different groups will benefit you. Therefore, keep in touch with friends and stay active in social groups in your community.

Smoking: smoking has many negative health effects and it is always a good idea to quit. Smoking is a risk factor for Alzheimer's, and so if you have a genetic predisposition for Alzheimer's it is absolutely necessary for you to quit smoking.

Diet: diet seems to play an important role in the prevention of Alzheimer's. Since free radicals can cause damage to the brain cells, a diet high in antioxidants is recommended for brain health. Antioxidants include:

- Vitamin C: is found in citrus fruits and various vegetables
- Vitamin E: a fat-soluble vitamin found in cereals, nuts and various vegetable oils
- Beta-carotene: is contained in various fruits and vegetables
- Studies have also found that a Mediterranean diet provides some protection against Alzheimer's and other diseases.

Education and mental stimulation: studies have shown that a high level of education and frequent mentally challenging activities (including puzzles, reading, listening to the radio and cultural activities) reduce the likelihood of Alzheimer's by as much as 75% and can also significantly delay its development. Spending long hours in front of the TV seems to increase the chance of developing Alzheimer's. Pick a challenging hobby that mentally stimulates you (crossword puzzles, chess, art appreciation, etc.) and practice it regularly.

Cholesterol: high cholesterol also contributes to the development of Alzheimer's, and so you should have your cholesterol checked every six months. If your cholesterol is too high, you can lower it with exercise and diet. If these are not effective, your doctor may prescribe cholesterol-lowering drugs. A healthy cholesterol level is important for preventing both atherosclerosis and Alzheimer's.

Blood pressure: high blood pressure is one of the most significant risk factors in the development of Alzheimer's disease. Measure your blood pressure regularly (once a week) after

10 minutes of sitting, and try to keep it within the normal range.

If your blood pressure is too high, the following measures can lower it. If these steps do not lower your blood pressure into the normal range, talk to your doctor about taking medication to lower your blood pressure.

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	✓	✓	✓
Aripiprazole	✓	↑	✓
Bupropion	✓	✓	✓
Carbamazepine	↑	↑	↓
Chlorpromazine	✓	✓	✓
Clomipramine	↑	✓	✓
Cyclobenzaprine	✓	✓	✓
Diltiazem	✓	↑	↑
Duloxetine	✓	✓	✓
Felodipine	✓	↑	↑
Fluvoxamine	✓	✓	✓
Iloperidone	✓	✓	✓
Lercanidipine	✓	↑	↑
Lovastatin	✓	↑	↑
Minaprine	✓	✓	✓
Nefazodone	✓	↑	↑
Nitrendipine	✓	↑	↑
Oxazepam	✓	↑	↑

	Effect	Breakdown	Dose
Amitriptyline	✓	✓	✓
Atorvastatin	✓	↑	↑
Buspirone	✓	↑	↑
Cerivastatin	✓	↑	↑
Citalopram	✓	↑	✓
Clonazepam	✓	↑	↑
Desipramine	✓	✓	✓
Donepezil	✓	↑	↑
Escitalopram	✓	↑	✓
Fluoxetine	✓	✗	✗
Galantamine	✓	↑	↑
Imipramine	✓	✓	✓
Lorazepam	✓	✓	✓
Memantine	✓	✓	✓
Mirtazapine	✓	✓	✓
Nifedipine	✓	↑	↑
Nortriptyline	✓	✓	✓
Oxcarbazepine	✓	✓	✓

	Effect	Breakdown	Dose
Amlodipine	✓	↑	↑
Bosentan	✓	↓	↓
Candesartan	✓	↓	↓
Chloral Hydrate	✓	✓	✓
Clobazam	✓	↑	↑
Clozapine	✓	✓	✓
Diazepam	✓	↑	↑
Doxepin	✓	✓	✓
Eszopiclone	✓	↑	↑
Fluvastatin	✓	✓	✓
Haloperidol	✓	↑	✓
Irbesartan	✓	✗	✗
Losartan	✗	↓	✗
Mianserin	✓	✓	✓
Moclobemide	✓	✓	✓
Nisoldipine	✓	↑	↑
Olanzapine	✓	✓	✓
Paroxetine	✓	✓	✓

	Effect	Breakdown	Dose
Perphenazine	✓	✓	✓
Quetiapine	✓	↑	↑
Risperidone	✓	✓	✓
Tacrine	✓	✓	✓
Trazodone	✓	↑	↑
Venlafaxine	✓	✓	✓
Ziprasidone	✓	↑	↑

	Effect	Breakdown	Dose
Pimozide	✓	↑	↑
Reboxetine	✓	↑	↑
Rivastigmine	✓	✓	✓
Temazepam	✓	✓	✓
Trimipramine	✓	✓	✓
Verapamil	✓	↑	↑
Zolpidem	✓	↑	↑

	Effect	Breakdown	Dose
Protriptyline	✓	✓	✓
Remoxipride	✓	✓	✓
Sertraline	✓	✓	✓
Thioridazine	✓	✓	✓
Valproic Acid	✓	↓	↓
Zaleplon	✓	↑	↑
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.

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

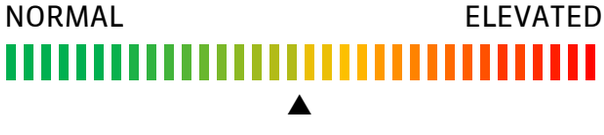
Risk of developing schizophrenia

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

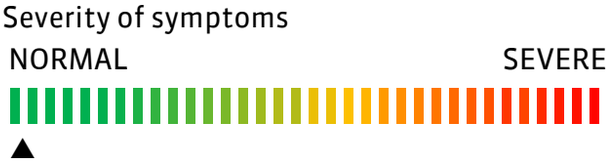


Solving of complex tasks

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

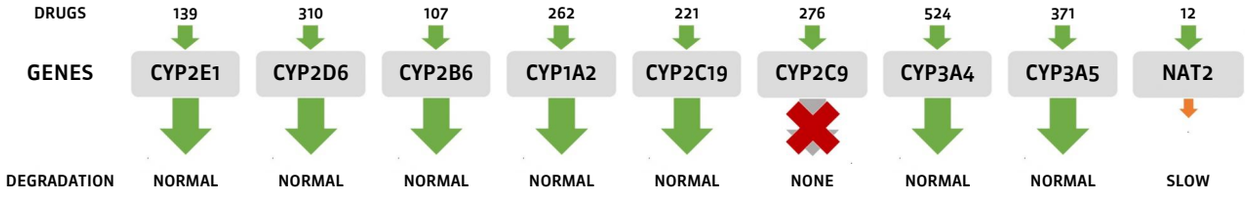
Anti-psychotic medication can control the symptoms by reducing biochemical imbalances in the brain and thereby decreasing the likelihood of relapse.

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



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.

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

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

Suicide risk in depression

Response to antidepressants

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.

PHARMACO GENETICS

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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

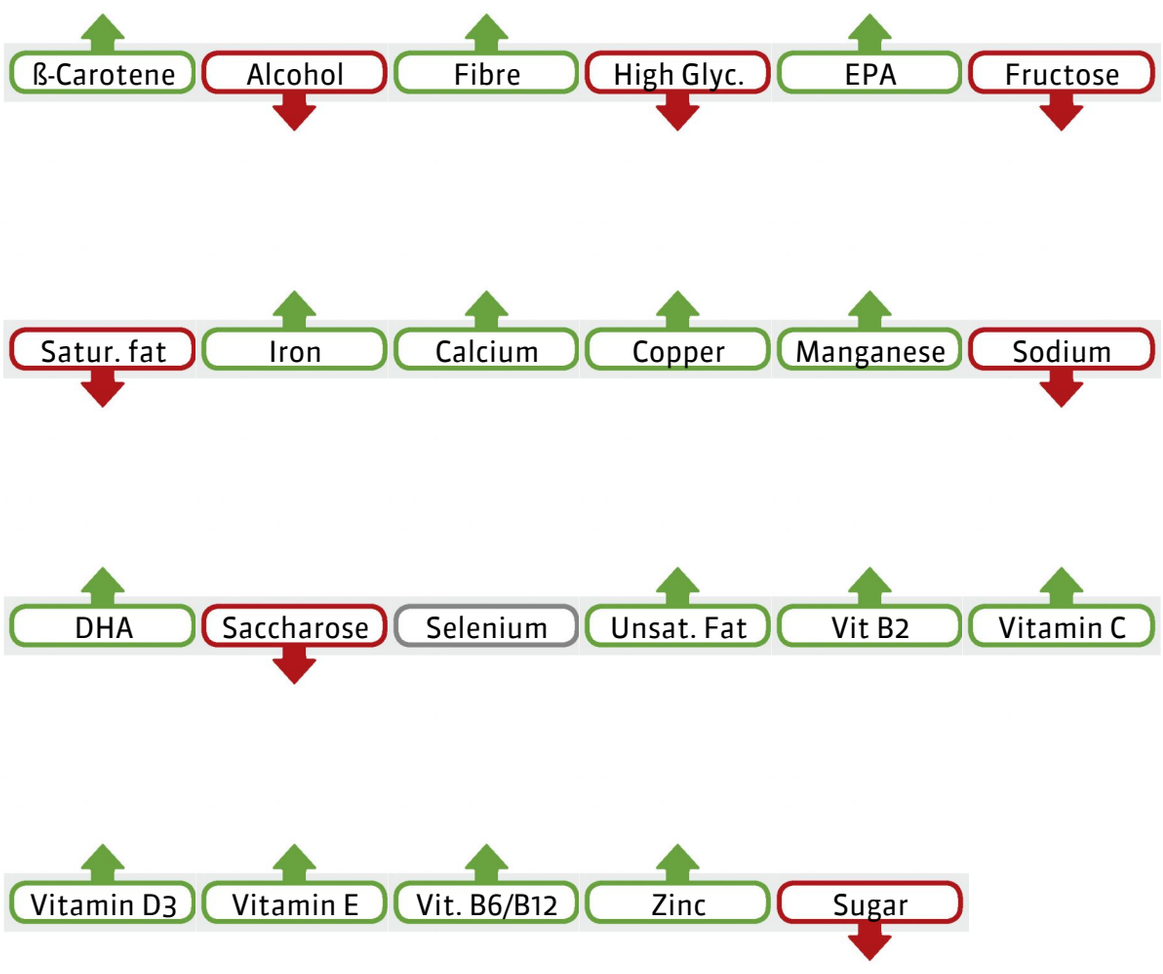
Effectiveness of metformin for prevention

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.

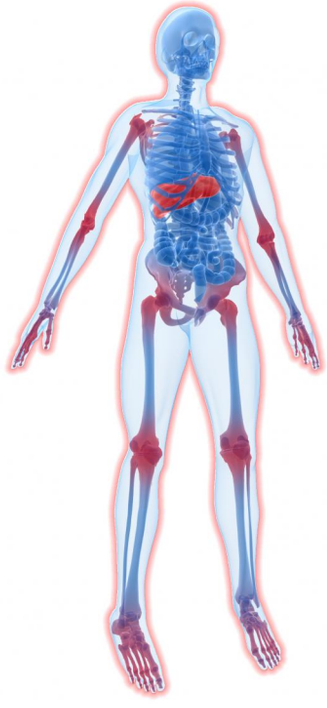
Iron Sensor

Haemochromatosis: easily prevent iron overload

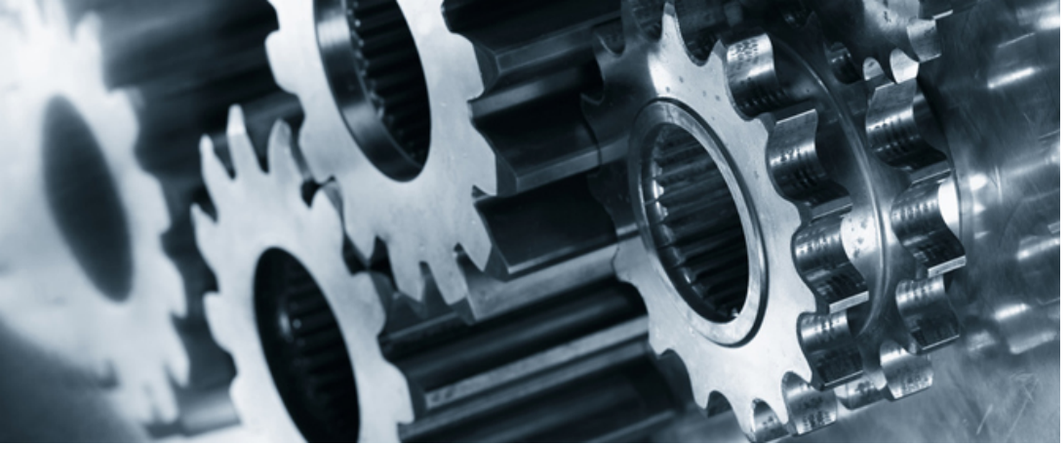
Haemochromatosis (iron overload)

The hereditary condition haemochromatosis, also called iron storage disease, is among the most common inherited metabolic diseases. It is caused by defects in the genes that are responsible for regulating the absorption of iron from food. These defects impair the proper function of these genes and lead to excessive absorption of iron, which over the years is deposited in the organs such as liver, heart, pancreas, pituitary gland and the joints, and damages them. In this case, accompanying diseases such as diabetes and liver cancer may appear.

Haemochromatosis is an "autosomal recessive" disease which usually occurs only when a person has inherited a defective iron-storage gene from both parents. People with only one defective gene have a slightly increased risk of disease, only 5-10% of people with one defective gene have elevated iron levels. The inherited form of haemochromatosis is very common. 1 in 10 persons has a single defective gene and is thus a carrier, while about 1 in 200 people has two defective genes and has a high risk of developing the iron storage disease.



Some symptoms of iron storage disease, eg. elevated liver function, are often misdiagnosed, which leads to wrong treatment and to the worsening of the symptoms. Misdiagnoses is common and, according to experts, 76% of cases are misdiagnosed. If left untreated, this disease can cause early death, but it can be treated and even prevented by regular blood donations (4-6 times per year) or through phlebotomy therapy. It is therefore, helpful to detect a genetic predisposition before symptoms appear. It may be possible to avoid symptoms with the help of preventive measures.



Genes associated with haemochromatosis

Haemochromatosis is usually a recessive disease. This means that the HFE genes from both parents must carry the same polymorphism in order for the disease to develop. In cases where only one gene exhibits a genetic variation affecting its function, the risk of elevated blood iron levels is present but the risk of developing the disease is very low. Your gene analysis shows the following:

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
HFE	rs1800562	C282Y	G/G
HFE	rs1799945	H63D	C/C
HFE	rs1800730	S65C	A/A

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

Summary of effects

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

- Your body absorbs an average proportion of the iron from your food
- You do not have an elevated risk of haemochromatosis

Your risk of haemochromatosis

LOW AVERAGE RISK HIGHER

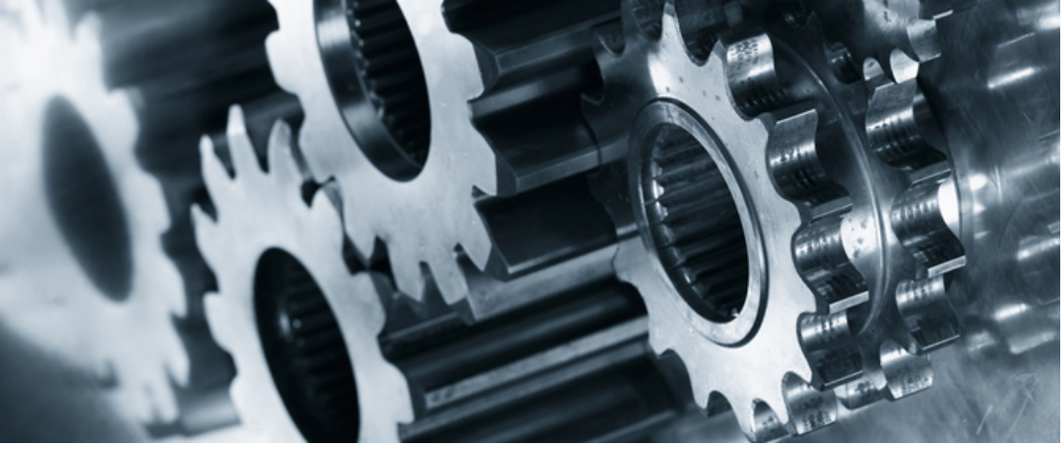
Nutritional Genes - Iron

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:

Iron | Alcohol

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 no increased risk of iron storage disease. Therefore, you do not have to take any special precautions and the following section is simply general information. Affected males develop the first symptoms usually between 20-40 years of age, while women usually develop symptoms after menopause.

- People with an increased genetic risk should donate blood frequently, about 5-6 times per year. When you donate blood, you reduce the iron content in your body. If you develop symptoms of haemochromatosis your blood will not be usable for transfusions.
- Additionally, have your iron level measured twice per year and ask your doctor about how often you should donate blood. If 5-6 donations per year is not a sufficient preemptive measure, your doctor will monitor your iron levels, and if necessary, will start a phlebotomy therapy for you.
- Avoid alcohol and also multivitamins that contain iron.

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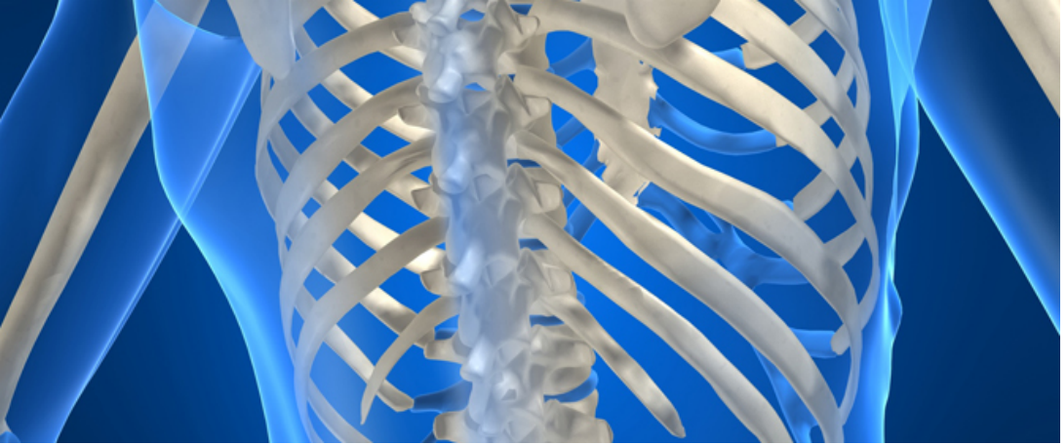
SCIENCE

ADDITIONAL INFORMATION

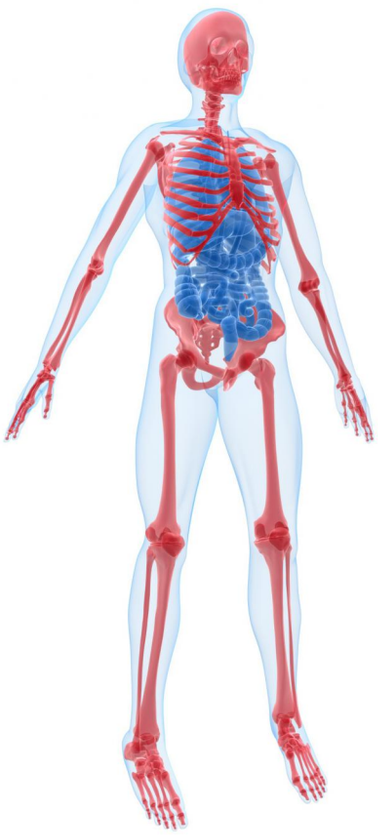
Bone Health Sensor

Stopping bone density loss and improving treatment



Osteoporosis

Osteoporosis is a disease that affects the bones. It causes bones to lose mass and strength, and makes them more fragile and easier to fracture. Even in normal development, bones become more likely to fracture with age. Bones reach maximum strength at about the age of 30 and then bone mass decreases progressively thereafter. However, some genetic traits lead to reduced bone strength, which increases the risk of osteoporosis and bone fractures- especially of the hips, forearms and vertebra. This risk grows with age.



Most fractures involve the hips, forearms and vertebrae. In normal development these bones grow throughout childhood and reach peak strength at about age 30. After that time, bone mass gradually decreases, leading to somewhat more brittle bones. However, some traits in the genes that are responsible for bone formation can cause your bones to become unusually fragile over time. As you age, this leads to increased bone loss and fractures. About 80% of osteoporosis cases occur in post-menopausal women, mainly because the body no longer produces the bone-protective hormone oestrogen. The disease is very common: 1 in 3 women over the age of 50 is diagnosed with osteoporosis. As oestrogen, the female sex hormone, plays a significant role for women in the formation of bone, women who have had lower estrogen levels throughout their life (e.g. due to a late start of menstruation or premature menopause) are particularly at risk.

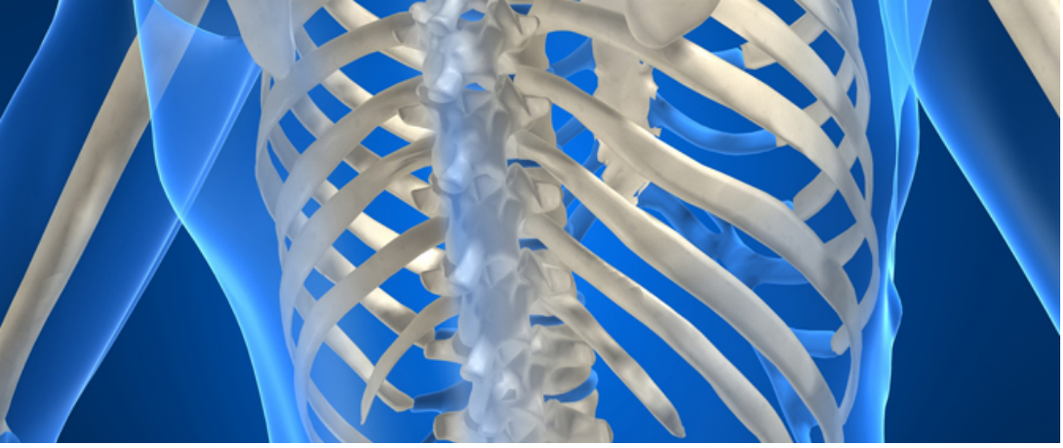
Osteoporosis is a common disease for men over the age of 70. Although women are more often affected by osteoporosis, this disease affects both sexes, and its development is accelerated by certain risk factors such as poor diet and unhealthy lifestyle. In addition to calcium, there are numerous other micronutrients (such as minerals, amino acids and vitamins) which are important in maintaining healthy bones. Bones have the ability to store calcium but these reserves can be depleted with nutritional deficiencies. Calcium is also crucial for other important processes in the body. Vitamin D plays an important role in the absorption of calcium from food.

In the elderly, the conversion to the active form of vitamin D is poor; in addition, the vitamin D from food is low due to poor

nutrition. Therefore, vitamin D deficiency is a widespread problem, but one that can be easily solved.

The incipient phases of the disease are sometimes not associated with any recognizable symptoms, and the diagnosis is made only when the first bone fractures occur. Prior to this point, the bone density is already compromised, and the bones may be fractured even in mild injuries, e.g. when bending or lifting a heavy bag.

If the disease is diagnosed at an advanced stage, treatment is based on preventing falls and increasing bone density as much as possible. This is achieved through a diet rich in calcium and vitamin D, adequate exercise and medication that promotes bone metabolism. The best defense against osteoporosis is prevention. The earlier the disease is diagnosed, the quicker you can take action to stop bone deterioration. Preventing bone loss is always easier than recovering lost bone. That is exactly what makes this gene test so valuable for preventative health care: you learn what your personal risk of disease is and can often completely prevent the disease from developing and follow a prevention program tailored to your individual needs.



Genes relevant in the context of osteoporosis

So far, scientists have identified several genes and polymorphisms which can increase the general risk of osteoporosis. An analysis can determine all relevant polymorphisms, the risk of illness, as well as other pertinent genetic properties. The following genes have an impact on the preservation of bone density:

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
Col1A1	rs1800012	G/T Pos. 1546 (S/s)	T/T
VDR	rs1544410	G/A IVS7 Pos.+283	A/A
ESR1	rs2234693	-397T>C	C/T
LCT	rs4988235	T>C	T/T

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

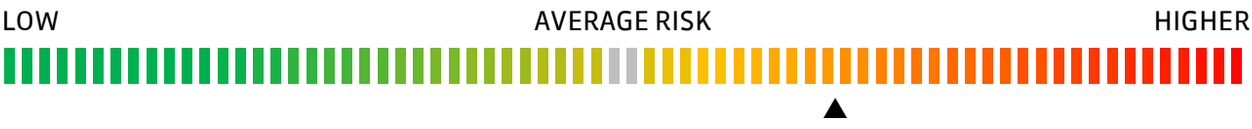
Summary of effects

In the case of osteoporosis, there are polymorphisms that protect against and those that promote its development. Polymorphisms that negatively impact calcium absorption also have an influence on bone density.

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

- Your risk of osteoporosis is 1.8 times increased
- Etidronate therapy is particularly effective
- Clodronate therapy is particularly effective
- Raloxifene therapy is particularly effective
- Your daily calcium uptake is average

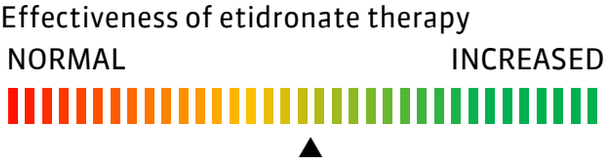
Risk of osteoporosis



Effectiveness of HRT in osteoporosis prevention

Effectiveness of alendronate therapy

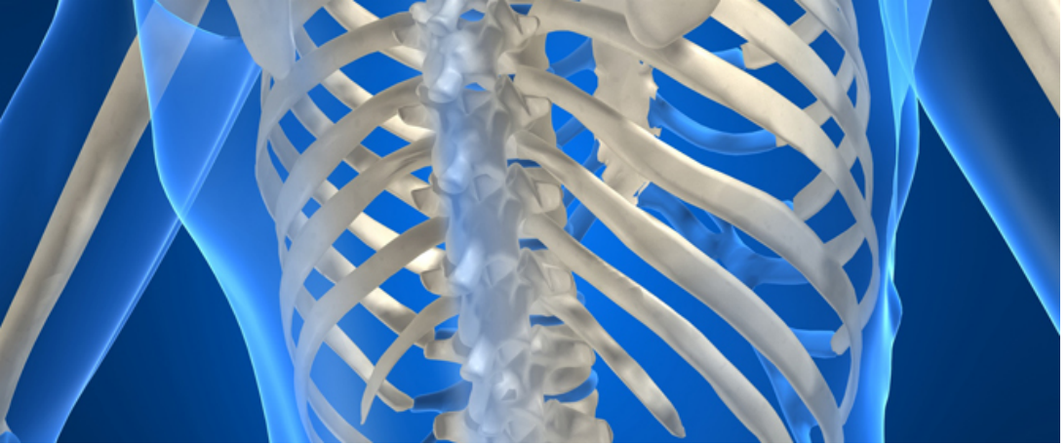
Effectiveness of etidronate therapy



Effectiveness of clodronate therapy

Effectiveness of raloxifene therapy

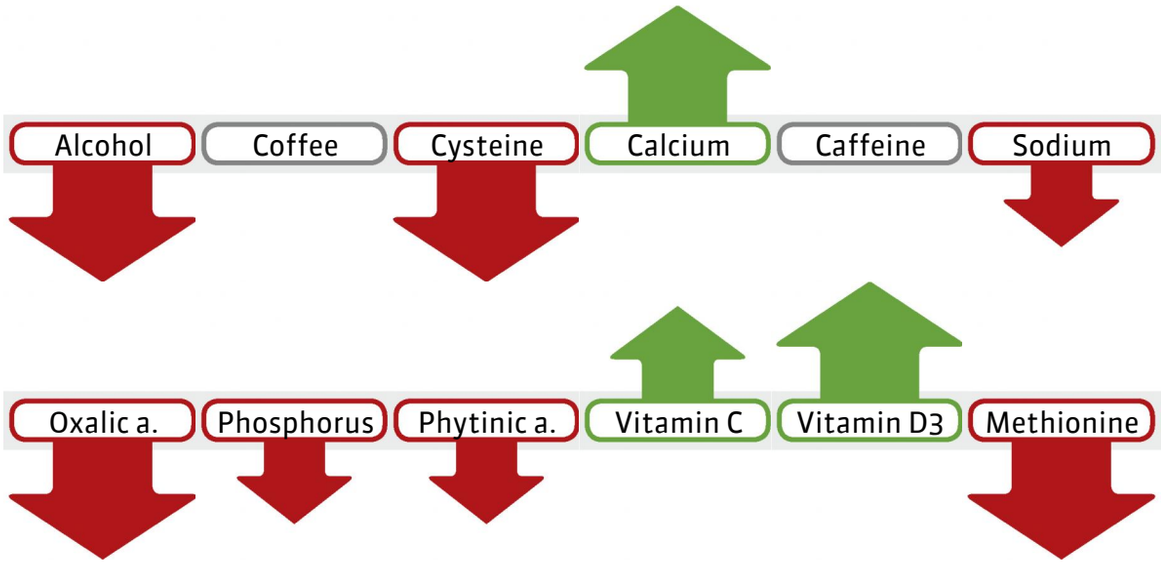
Your typical calcium absorption



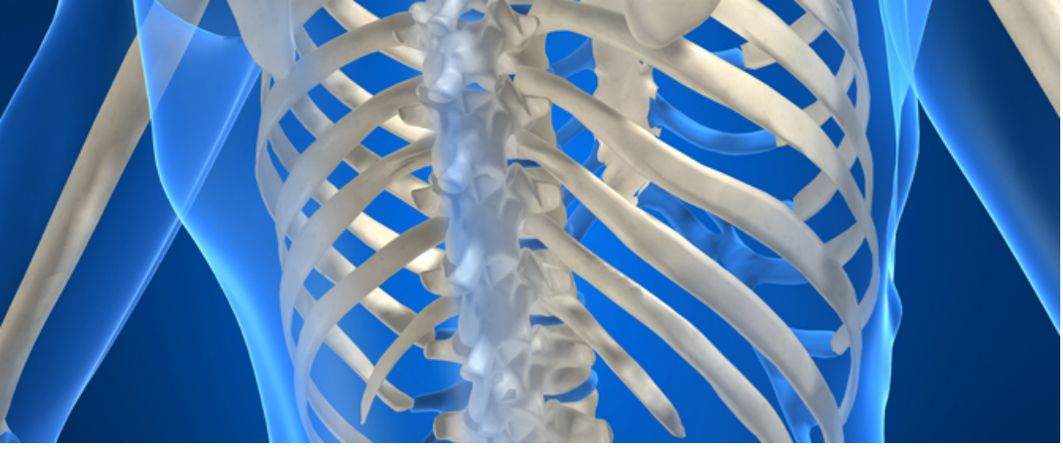
Nutritional Genes - Bones

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:



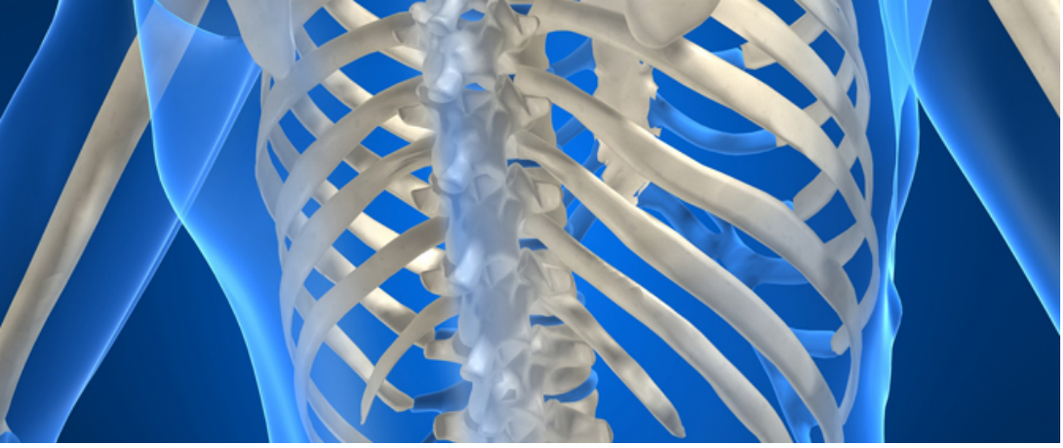
Legend: GREEN ARROWS > this nutrient or substance is classed as healthy for your genetic profile. Try to increase the intake of this substance. RED ARROWS > this substance is classed as unhealthy for your genetic profile. Try to reduce your intake of the substance. NO ARROWS > There is no effect of the nutrient on the genetics of this section. PLEASE NOTE! This interpretation only considers your genetic profile of this section.



Prevention

You have a moderate genetic predisposition for osteoporosis. It is important for you to take preventative measures in order to keep your bones as strong as possible. It is difficult to restore lost bone mass therefore it is better to prevent bone loss in the first place. The following precautions will help you keep your bones strong:

- Make sure you consume enough calcium. Calcium is the raw material necessary for bone regeneration, and it is vital to ensure you are getting sufficient. Dairy products, calcium supplements and some drugs for osteoporosis contain calcium.
- Ensure that you have enough vitamin D. Your body produces this vitamin in the sunlight, so you should spend a safe amount of time outdoors. However, vitamin D is also contained in food products such as fish, smaller quantities in milk, as well as in some nutritional supplements (fish oils).
- You should eat only limited amounts of foods that are high in phosphates, such as sausages, chocolate and meat. Phosphates extract calcium from bone material and weaken bones.
- Any form of exercise, such as jogging or walking, will strengthen your bones by forcing them to develop.
- Several diseases, including hormonal disorders, gastrointestinal, liver, kidney and joint diseases, can cause osteoporosis. These diseases should be treated to reduce symptoms.
- Alcohol and nicotine weaken bones, along with many other negative health effects. Giving up alcohol and cigarettes will reduce your osteoporosis risk.
- Get regular bone density scans from your doctor to track changes in bone density.
- If you have advanced osteoporosis, medication can slow or even stop the progression of the disease. Talk to your doctor about your options for drug therapy.
- Numerous drugs can interfere with bone formation and so people at risk for osteoporosis should consult their doctor before taking any medication. Drugs known to inhibit bone growth are: cortisone, anti-epileptics, oral anticoagulants and heparin, aromatase inhibitors (AI) for breast cancer, androgen deprivation therapy for prostate cancer, calcineurin inhibitors such as those for immunosuppression after organ transplantation, and gastric acid inhibitors.
- Certain foods can also lead to bone mineral density loss and should be avoided if possible. Try to reduce food types that are rich in table salt, phytinic acid, the amino acids cysteine and methionine, and oxalic acid. The excessive consumption of caffeine also leads to a gradual decrease in bone mineral density.



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
Alendronic Acid	✓	✓	✓
Codeine	✓	✓	✓
Fentanyl	✓	↑	↑
Ibuprofen	✓	✗	✗
Lidocain	✓	✓	✓
Oxycodone	✓	↑	✓
Raloxifene	✓	✓	✓
Strontium Ranelate	✓	✓	✓
Zoledronic Acid	✓	✓	✓

	Effect	Breakdown	Dose
Alfentanil	✓	↑	↑
Enflurane	✓	✓	✓
Halothane	✓	✓	✓
Isoflurane	✓	✓	✓
Methadone	✓	↑	↑
Paracetamol	✓	✓	✓
Ropivacaine	✓	✓	✓
Teriparatide	✓	✓	✓
Zolmitriptan	✓	✓	✓

	Effect	Breakdown	Dose
Buprenorphine	✓	↑	↑
Etidronic Acid	✓	✓	✓
Hydrocodone	✓	✓	✓
Levacetylmethadol	✓	↑	↑
Methoxyflurane	✓	✓	✓
Phenacetin	✓	✓	✓
Sevoflurane	✓	✓	✓
Tramadol	✓	↑	✓

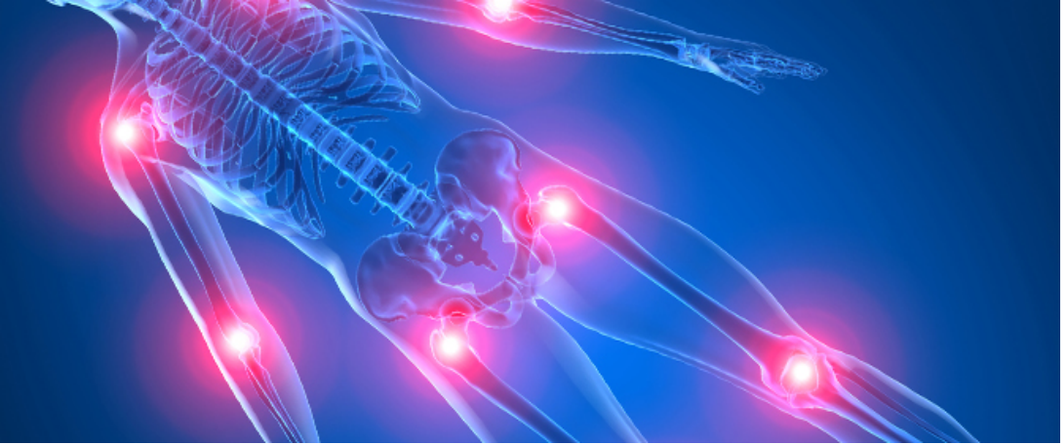
Please note: The right choice and dose of medication is always the responsibility of the doctor. Never make your own decision on whether to stop taking a medication or changing its dose!

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.

Joint Sensor

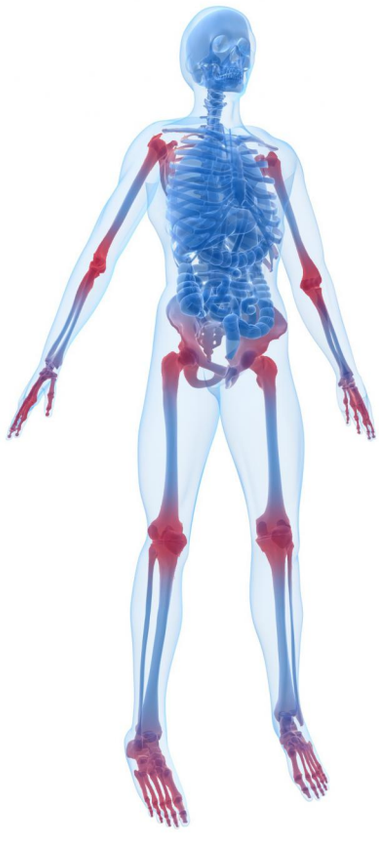
Rheumatoid arthritis: prevention and efficiency



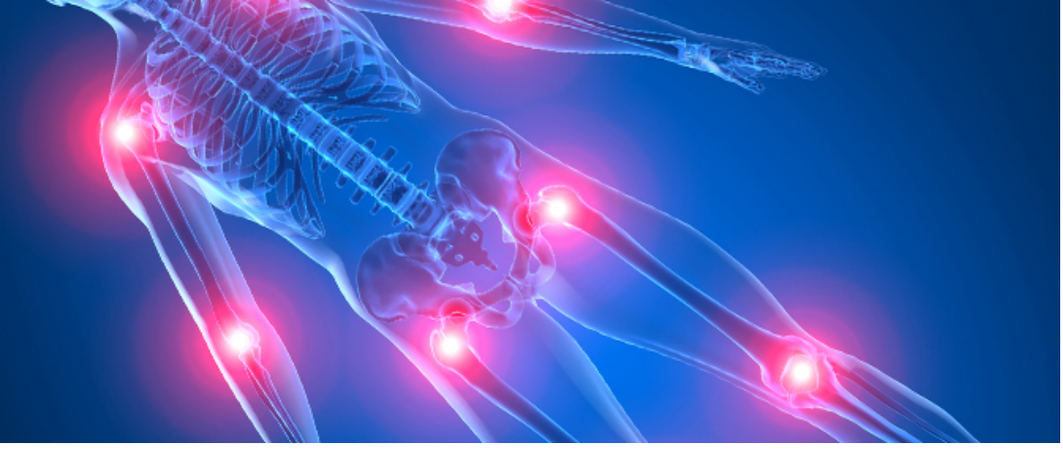
Inflammatory joint diseases

An incorrectly programmed immune system can cause a number of joint diseases. Sometimes the immune system falsely identifies healthy parts of joints as bacterial infections and attacks them by sending immune system cells into the joints where they cause inflammation.

This process can lead to conditions such as arthritis or ankylosing spondylitis (AS), which in advanced stages causes the spine to fuse. This severe disease is estimated to affect 1.6 million people in Germany alone, many of whom are unaware of their condition because the initial symptoms are mild.



The onset of rheumatoid arthritis can take place at an early age. The immune system of affected individuals attacks and destroys joint cartilage due to a (often genetically determined) programming error. In severe cases, the cartilage may be destroyed completely, causing the bones of the joint to rub against one another. This rubbing shortens the bones of the joint, causing them to slowly stop functioning. Patient mobility is increasingly impaired, their joints deform and fuse together. In severe cases, patients can expect increasing disability and eventually incapacitated. Rheumatoid arthritis cannot be cured, but the earlier it is diagnosed and treated, the better its progress can be delayed.



Genes associated with joint diseases

The scientific community has linked several genes and polymorphisms to the risk of various inflammatory diseases. An analysis of these polymorphisms allows us to determine your genetic risk for these diseases as well as some other genetic traits linked to this disease.

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
TNF-α	rs1800629	A>G	A/A
IL1A	rs1800587	C>T	C/C

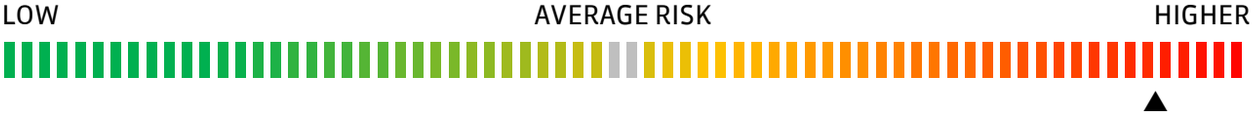
LEGEND: rsNCBI = description of examined genetic variation, POLYMORPHISM = form of the genetic variation, GENOTYPE = personal analysis result

Summary of effects

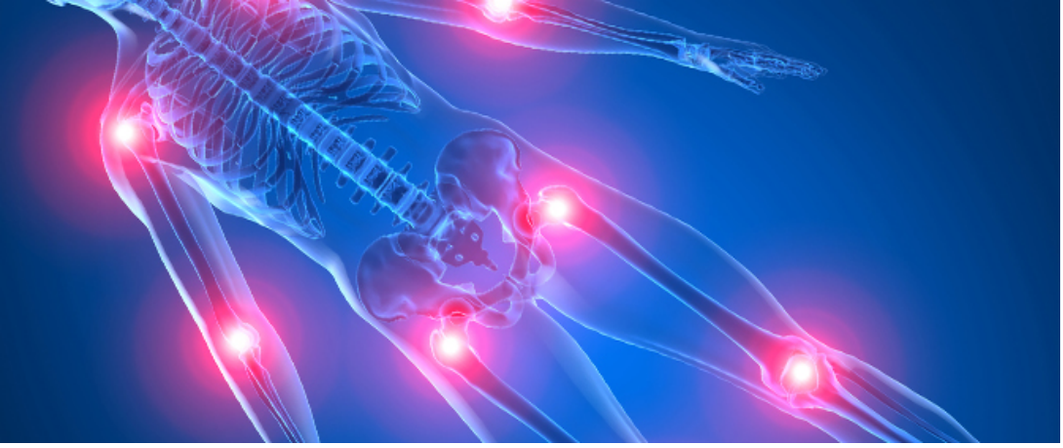
Here you can see a summary of the influence your genetic variations have on your health:

- Based on your genes you have a 4.9 -times higher risk of rheumatoid arthritis
- You do not have an elevated risk of degenerative disc disease

Your risk of rheumatoid arthritis



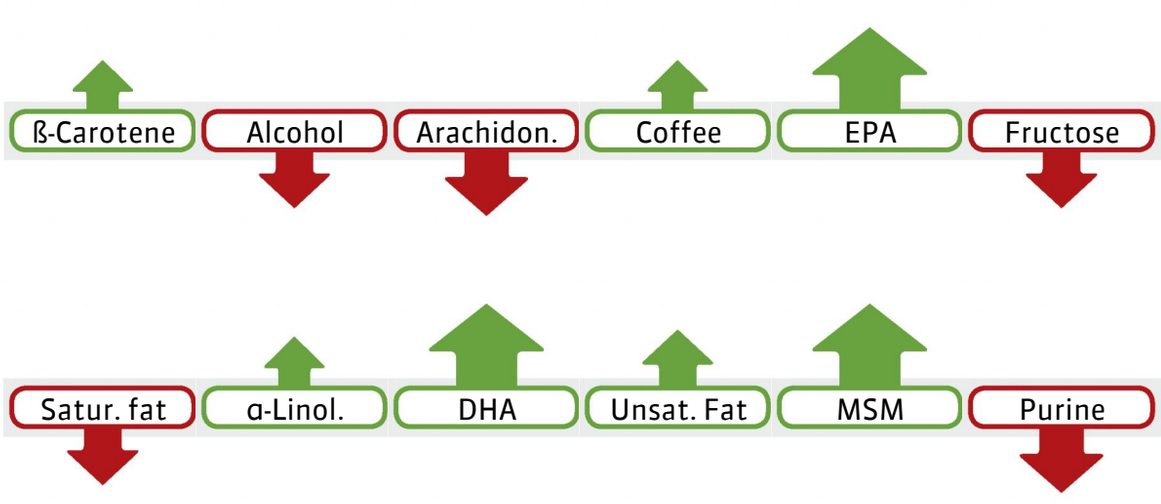
Your risk of degenerative disc disease



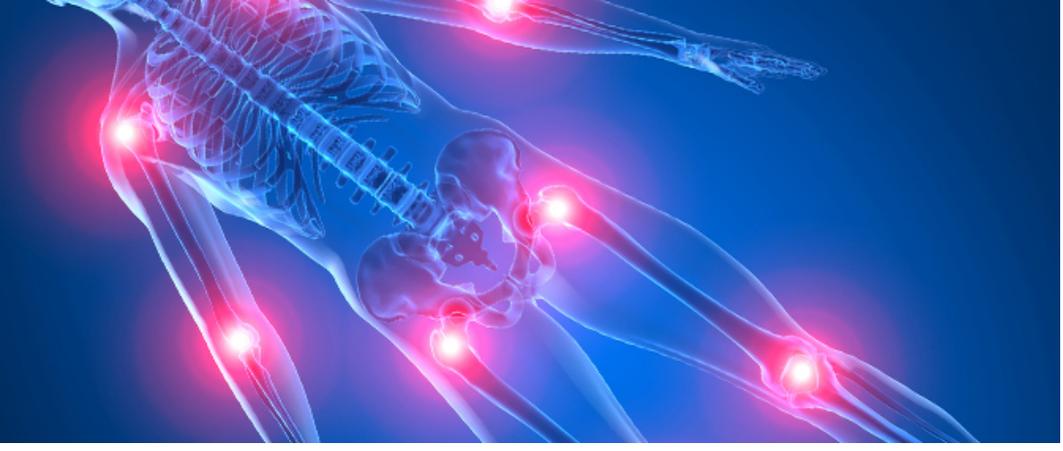
Nutritional Genes - Joints

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

Precautionary measures are now very important for you to stay healthy. The genetic trait that you carry does not have any symptoms, but it significantly increases the risk that you will develop certain diseases of the joints. Your action program consists of three major parts:

Exercise

Although the disease can stiffen the joints, it is important to keep using them. The less you move, the faster your joints will stiffen. Exercise regularly and ensure that all the joints are evenly loaded. Suitable exercises are: daily walking, swimming, cycling and gymnastics. Make sure, however, that you warm-up before increasing the effort. The sooner you start with daily exercises, the better for your joints.

Nutrition

Inflammation produces chemical messengers called cytokines which cause pain, swelling and inflammation of the joints in rheumatoid arthritis. The raw material for these messengers (arachidonic acid) is found in animal fat, such as meat, sausages and dairy products. Therefore, it is advisable to consume meat only once per week and meet your protein requirements by eating fish and vegetable protein. Regular consumption of fish will decrease the symptoms of painful and swollen joints. As an alternative, fish and fish oil capsules can be used as dietary supplements. Studies have shown that fish oil can reduce the number of swollen joints. Test the levels of omega-3 in your blood to see if your diet is effective, and eat foods that contain anti-inflammatory substances.

- Fish (oil)
- Evening primrose oil
- Soy
- Wheat and rapeseed oil
- Black currant

Avoid pro-inflammatory foods that contain large quantities of arachidonic acid. Do not consume more than 300 mg of arachidonic acid daily. Check your consumption based on the list below and adjust your diet if necessary.

- Lard 1700 mg/100g
- Pork liver 870 mg/100 g
- Egg yolk 300 mg/100 g
- Tuna 280 mg/100 g
- Pork 120 mg/100 g
- Beef 70 mg/100g
- Egg (total) 70 mg/100 g
- Veal 53 mg/100g
- Chicken 42 mg/100g
- Camembert 34 mg/100g

- Cow's milk (1.5%) 2 mg/100 g
- Potatoes, fruits, vegetables 0 mg/100g
- Nuts, soy products 0 mg/100g
- Vegetable oils 0 mg/100g

This table shows that only food from animal sources contains this fatty acid. An optimal diet will be vegetarian or with restrictions on meat consumption. A typical vegetarian diet of around 200-400g/day contains only approximately 50 mg of arachidonic acid. In addition, a sulfur-rich diet is recommended for you because organic sulfur (methylsulfonylmethane) is anti-inflammatory. As an alternative, organic sulfur can also be taken as dietary supplements (MSM capsules) or applied in the form of ointments.

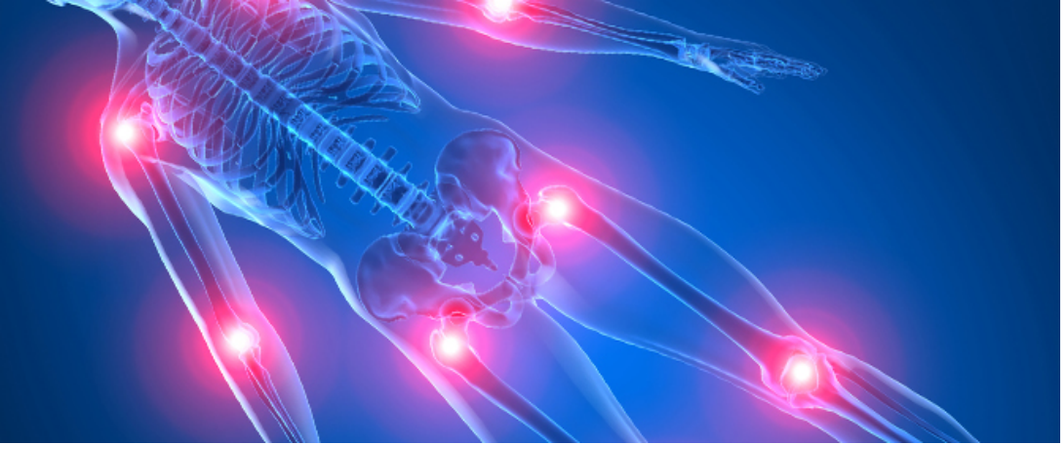
Early detection

The main objective of the preventative measures is to detect the disease as early as possible by paying attention to early symptoms. This will allow for timely medical treatment. Since you are genetically predisposed to the disease, you should note the following warning symptoms:

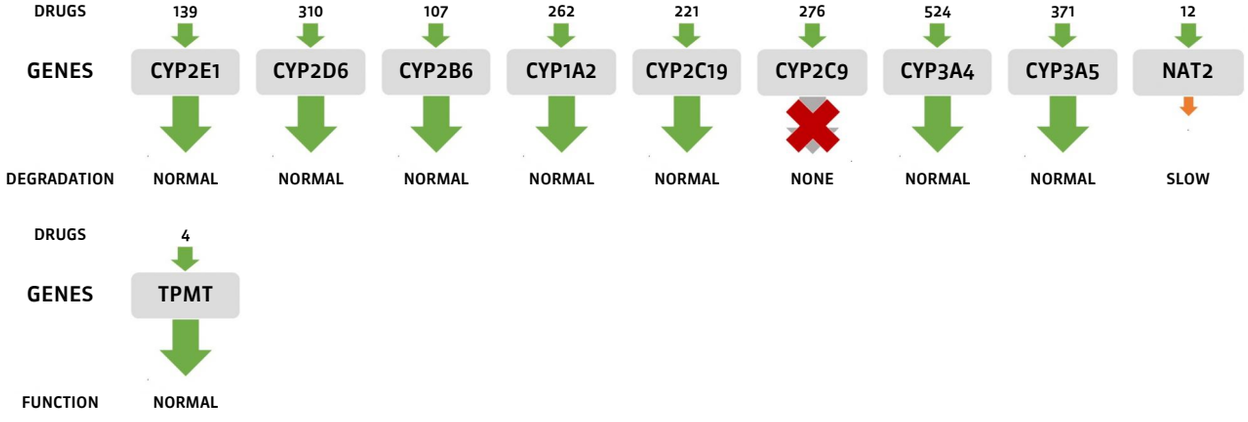
- Morning stiffness in hand and finger joints,
- circulatory disorders of individual fingers,
- back pain

A definitive diagnosis is difficult, especially in the early stages, because there are no unique symptoms of the disease. Furthermore, rheumatoid arthritis presents different manifestations, depending on the patient, which makes it even more difficult to diagnose. Therefore, you should consult an experienced physician as soon as the first symptoms appear to enable him to make the correct diagnosis and initiate treatment. Describe your symptoms as accurately as possible, as this is particularly important for the correct diagnosis.

The current treatment methods are effective for the majority of patients if the joint disease is detected early enough. Symptoms such as inflammation and pain can be controlled if detected early. However, patient commitment is crucial for the success of the treatment.



Drug compatibility



Effect on relevant medication

	Effect	Breakdown	Dose
Aceclofenac	✓	✗	✗
Anakinra	✓	✓	✓
Budesonide	✓	✓	✓
Cortisone	✓	↑	↑
Dexamethasone	✓	↑	↑
Diflunisal	✓	✓	✓
Fenoprofen	✓	✓	✓
Hydrocodone	✓	✓	✓
Hydroxychloroquine	✓	✓	✓
Indometacin	✓	↓	↓
Ketorolac	✓	✓	✓
Mefenamic Acid	✓	✗	✗
Methylprednisolone	✓	↑	↑
Nabumetone	✓	✓	✓

	Effect	Breakdown	Dose
Acetylsalicylic Acid	✓	✗	✗
Azathioprine	✓	✓	✓
Celecoxib	✓	✗	✗
Cyclophosphamide	✓	↑	↑
Diclofenac	✓	✗	✗
Etanercept	✓	✓	✓
Fentanyl	✓	↑	↑
Hydrocortisone	✓	↑	↑
Ibuprofen	✓	✗	✗
Infliximab	✓	✓	✓
Leflunomide	✓	✗	✗
Meloxicam	✓	✗	✗
Minocycline	✓	✓	✓
Naproxen	✓	✗	✗

	Effect	Breakdown	Dose
Adalimumab	✓	✓	✓
Betamethason	✓	✓	✓
Codeine	✓	✓	✓
Cycloserine	✓	✓	✓
Diclofenac	✓	✗	✗
Etodolac	✓	✗	✗
Flurbiprofen	✓	✗	✗
Hydromorphone	✓	↓	↓
Indometacin	✓	↓	↓
Ketoprofen	✓	✓	✓
Lornoxicam	✓	✗	✗
Methotrexate	✓	✓	✓
Morphine	✓	✓	✓
Naproxen	✓	✗	✗

	Effect	Breakdown	Dose
Oxaprozin	✓	✗	✗
Piroxicam	✓	✗	✗
Rituximab	✓	✓	✓
Sulindac	✓	✓	✓
Tolmetin	✓	✓	✓

	Effect	Breakdown	Dose
Oxycodone	✓	↑	✓
Prednisolone	✓	↑	↑
Salsalate	✓	✓	✓
Suprofen	✓	✗	✗
Tramadol	✓	↑	✓

	Effect	Breakdown	Dose
Paracetamol	✓	✓	✓
Prednisone	✓	↑	↑
Sulfasalazine	✓	✓	✓
Tenoxicam	✓	✗	✗

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.

PHARMACO GENETICS

ONCOLOGY

CARDIOVASCULAR SYSTEM

NEUROLOGY

METABOLISM

MOVEMENT

DIGESTION

OPHTHALMOLOGY

ODONTOLOGY

OTHERS

SCIENCE

ADDITIONAL INFORMATION

Gluten Sensor

Early detection and nutritional adjustments

Gluten intolerance

Gluten intolerance, also known as celiac disease, is a widespread food intolerance that affects approximately 1 in 120 humans. While a person can develop gluten intolerance at any age, it is especially prevalent in two stages of life: when an infant is introduced to solid food or between 30-40 years of age. Women are affected more frequently than men.

Gluten is a protein found in many foods. In some people, gluten triggers an immune response in the intestine that attempts to fight gluten as if it were a bacterial infection. 95% of gluten intolerance cases are caused by a hereditary trait in two specific genes that are involved in regulating the immune system. The body's response to gluten usually leads to a chronic condition causing damage to the small intestine and a variety of other symptoms including diarrhoea, loss of appetite and weight loss. If untreated, it can eventually cause malnutrition, fatigue and occasional vomiting. For infants and toddlers, these can cause growth disorders. The symptoms of gluten intolerance are so varied, therefore it is difficult to diagnose and it can go unrecognized for many years. At the same time, some people believe that they are intolerant to gluten when they are actually suffering from other conditions. A genetic test will help you and your doctor determine if you are gluten-intolerant.

Gluten intolerance is often accompanied by other conditions, including type 1 diabetes, anaemia and osteoporosis. Other conditions, such as lactose intolerance can develop. If a gluten-intolerant person continues to consume gluten over a period of years, it can cause serious damage to the intestine. In the worst case, untreated gluten intolerance can cause tumours in different parts of the body. The mortality rate for untreated gluten intolerance is 12%. This risk can generally be eliminated with proper treatment and adjustment to the diet. Damage to intestinal villi prevents the body from absorbing essential nutrients, which can result in vitamin and mineral deficiencies. For this reason, it is important that affected individuals adhere to a balanced, gluten-free diet and take necessary dietary supplements.

There is currently no cure for gluten intolerance and the treatment consists of a lifelong gluten-free diet. Proper treatment usually leads to the regeneration of the intestinal mucosa and complete disappearance of symptoms. Affected individuals must familiarize themselves with the list of foods containing gluten, and also check ingredient lists on food packaging. In rare cases, when the affected person does not respond well to the diet, other medical treatment is possible. Even though gluten intolerance is fairly common, it is often misdiagnosed as a common digestive disorder because its symptoms are so variable. This gene test is a valuable tool for helping you to determine your risk for gluten intolerance. If you have elevated risk, you can adjust your diet accordingly to avoid further discomfort and prevent harmful secondary conditions.

The genetics of gluten intolerance

The development of gluten intolerance is largely dependent on the presence of certain polymorphisms. The analysis of these polymorphisms shows the following:

Genetic traits			
SYMBOL	**rs NCBI**	**POLYMORPH**	**GENOTYPE**
HLA DQ2.5	rs2187668	HLA DQ2.5	G/G
HLA DQ8	rs7454108	HLA DQ8	T/C

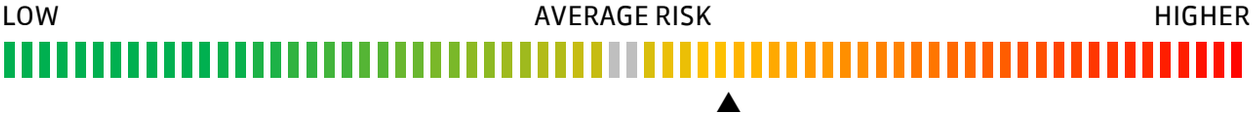
LEGEND: rsNCBI = description of examined genetic variation, POLYMORPHISM = form of the genetic variation, GENOTYPE = personal analysis result

Summary of effects

Gluten intolerance only occurs in people who have specific genes. People who do not have the genetic trait associated with gluten intolerance have virtually no risk of developing it. However, many people with the gluten intolerance gene never actually develop the disease. Even if you are at risk for gluten intolerance, there is a good chance that you will never actually develop it. However, you should pay close attention to your body so you can recognize symptoms early and prevent organ damage by adopting the right diet. Here you can see a summary of the influence your genetic variations have on your health:

- Your genetic profile contains genes associated with gluten intolerance
- You have an elevated risk of gluten intolerance

Risk of gluten intolerance



Nutritional Genes - Cereal

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:

ß-Carotene	Fibre	EPA	Gluten	Iron	Calcium

Copper	Lactose	Manganese	DHA	Selenium	Unsat. Fat

Vit B2	Vitamin C	Vitamin D3	Vitamin E	Vit. B6/B12	Zinc

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

As a result of your genetic analysis, we determined that you have an increased risk of gluten intolerance. If you experience symptoms of gluten intolerance, a gluten-free diet would be advisable. However, keep in mind that your genetic profile does not necessarily cause gluten intolerance. Many people who share your genetic traits consume gluten without any ill effects.

Therefore, it is advisable that you pay attention to see if foods with gluten trigger symptoms. If so, you can correct this and eliminate the symptoms by following a gluten-free diet. A controlled diet can also restore the normal condition of the small intestine even if it has already been damaged, and helps you avoid further complications. If you are gluten intolerant, you should learn about foods that contain gluten and then reduce or, if possible, completely eliminate gluten from your diet. The types and severity of symptoms of gluten intolerance are different from person to person, and mostly depend on how much damage was already caused to the small intestine. Some people are affected by very low gluten levels and must follow a gluten-free diet for their whole lives. It is especially important for them to be familiar with a detailed list of foods containing gluten. In most cases, symptoms will resolve quickly and not recur once gluten is eliminated from the diet. However, if the diet has no effect then the patient must undergo medical treatment. It is especially important to seek treatment to ensure that the symptoms are not caused by another disease. Furthermore, when the small intestine is irritated by gluten, it cannot absorb essential nutrients such as vitamins and minerals. A person with gluten intolerance must take additional measures to ensure that their body has enough vitamins and minerals (in the form of a balanced gluten-free diet or through supplements). Gluten intolerance may lead to lactose intolerance, in which case the patient should avoid milk and dairy products, as well. By following a gluten-free diet, it is possible for the intestine to recover enough to digest milk and dairy products in the future. If you suspect you suffer from gluten intolerance, you should double check with a doctor. Celiac disease can be diagnosed by examining the colon and performing a blood test for specific antibodies. Speak with your doctor as soon as the first symptoms appear.

Typical symptoms of gluten intolerance are:

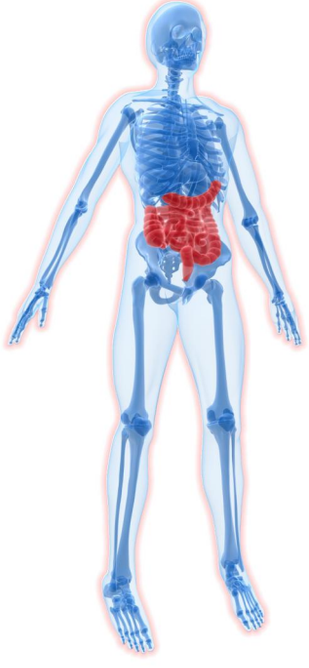
- Diarrhoea or abnormal stools
- Flatulence
- Malaise, fatigue and abdominal cramps
- Iron deficiency anaemia leading to muscle and joint pain
- Occasional vomiting
- Loss of appetite and weight loss, possibly leading to malnutrition
- Painful, itchy blisters
- Growth retardation in infants if the intolerance is undetected and untreated for a long time
- Serious follow-up diseases such as: lactose intolerance, ear, nose and throat tumors (9 times increased risk) or tumors in the lymphatic system (40-80 times increased risk).

Lactose Sensor

Early detection and nutritional adjustments

Lactose intolerance

Lactose intolerance is the most common food intolerance in the European population: 1 in 6 Europeans are affected. Before lactose can be absorbed into the bloodstream, it must first be broken down by lactase, an enzyme in the intestine. The intestine produces lactase in childhood because newborns must be able to digest breast milk. As the child's body prepares to digest other foods, the genes responsible for the production of lactase are gradually deactivated.



As such, even infants who have the ability to digest lactose early on will gradually build up an intolerance to lactose. Eventually, the body is no longer able to digest lactose at all, and consumption of lactose can precipitate a broad range of symptoms. Non-digested lactose is an excellent source of nutrients for intestinal bacteria, which seize the opportunity to multiply rapidly in the digestive tract. The lactose is broken down into different acids and fermentation produces various gases. This process results in diverse symptoms that vary in intensity from person to person. Symptoms include digestive problems such as abdominal bloating, cramps and diarrhoea, as well as a number of nonspecific complaints such as fatigue or skin problems.

Most people in the world are lactose intolerant. However, genes that continued to produce lactase through childhood and adulthood spread through populations of ancient people who raised cattle. As a result, most adults from populations with a history of dairy farming have the ability to digest lactose. Today, 5 out of 6 Europeans can enjoy dairy products. Due to this figure, Europeans view lactose tolerance as the norm, whereas persons that cannot digest lactose are considered to suffer from a food intolerance. Thus, we list lactose intolerance as a disease.

A lactose-free diet can prevent all symptoms of lactose intolerance. Individuals should familiarize themselves with foods that contain lactose. Unfortunately, lactose intolerance is often misdiagnosed for years as the severity of symptoms depends on the amount of lactose one consumes. As symptoms of lactose intolerance are often misinterpreted as general digestive discomfort, gene testing to determine lactose intolerance can help clear up any personal intolerance you may have and prevent further complications.

Genes associated with lactose intolerance

More than 99% of cases of lactose intolerance are caused by a genetic variation of the gene LCT/MCM6. A person with two copies of this variation will most likely develop lactose intolerance in their lifetime. When symptoms arise and their severity depends on many other factors, including the environment. The analysis of associated polymorphisms shows the following:

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
LCT	rs4988235	T>C	T/T

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

Summary of effects

- You do not have an elevated risk of lactose intolerance
- Your daily calcium uptake is average

Your risk of lactose intolerance

LOW AVERAGE RISK HIGHER

Your typical calcium absorption

NORMAL REDUCED

Nutritional Genes - Milk

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:

Calcium | Vitamin D3 | Lactose

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

You do not have an increased genetic risk for lactose intolerance. Therefore, you do not have to take any special precautions or follow a lactose-free diet.

If you do have problems, provide details to your doctor so you can get an accurate diagnosis.

IBD Sensor

Crohn's disease early detection and proper treatment

Crohn's disease

The inflammatory gastro-intestinal disease known as Crohn's disease (named after Burill Bernhard Crohn, the first gastroenterologist who identified and described it) is a chronic and progressive intestinal disease that can affect the entire digestive tract. An abnormal immune reaction causes inflammation of the intestine in multiple locations, which causes digestive problems such as diarrhoea and cramps. The inflammation mostly occurs in the colon and small intestine, and more rarely the mouth and oesophagus. Damage to the intestinal tissue increases if the inflammation persists.

Approximately 1 in 700 people suffer from this inflammatory intestinal disease (Crohn's disease), which can be triggered by an inherited error in the intestinal gene 1 (NOD2). This gene is involved in the function of the immune system. Symptoms most often appear for the first time in people between the ages 16 and 35, or people over 60.

Crohn's disease is usually intermittent, with periods of remission alternating with intensive manifestation of symptoms. However, in some cases, this disease can also be chronically active. In many cases, it can take years to correctly diagnose the disease because the first symptoms are temporary digestive issues. Left untreated, the disease may lead to a variety of conditions which must be properly treated.

The cause of the disease is not fully understood. Better understanding of the disease may lead to improved treatments. Currently, the best treatment consists of alleviating symptoms and using immunosuppressants to reduce the immune reaction. Treatment is aimed at reducing the severity of episodes, preventing further attacks, and treating complications such as strictures, fistulas and perforation of the intestinal tissue. In most cases this leads to a significant improvement in the quality of life of those affected. Because many cases are not diagnosed, this genetic test is recommended for people with recurring digestive problems, as it identifies an increased risk of inflammatory bowel disease, and where applicable, the right diagnosis.

Relevant genes for Crohn's disease

The analysed genes have an influence on your risk of developing Crohn's disease and ulcerative colitis. At present, there is no way to reduce your risk of developing Crohn's disease but an accurate diagnosis and proper medical care can significantly reduce the discomfort. The main benefit of this genetic analysis is to determine your risk for Crohn's disease. Close attention to the early symptoms of the disease will help your doctor make an accurate diagnosis relatively quickly and spare you the long ordeal of searching for the correct diagnosis and treatment. These diseases can be successfully treated by an appropriate diet and genetically-tailored drug therapy.

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
NOD2	rs2066844	C>T	C/C
NOD2	rs2066845	G>C	G/G
NOD2	rs2066847	del>C	del/del

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

Summary of effects

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

➤ Your risk of developing Crohn's disease or ulcerative colitis is not increased.

Risk for inflammatory bowel disease

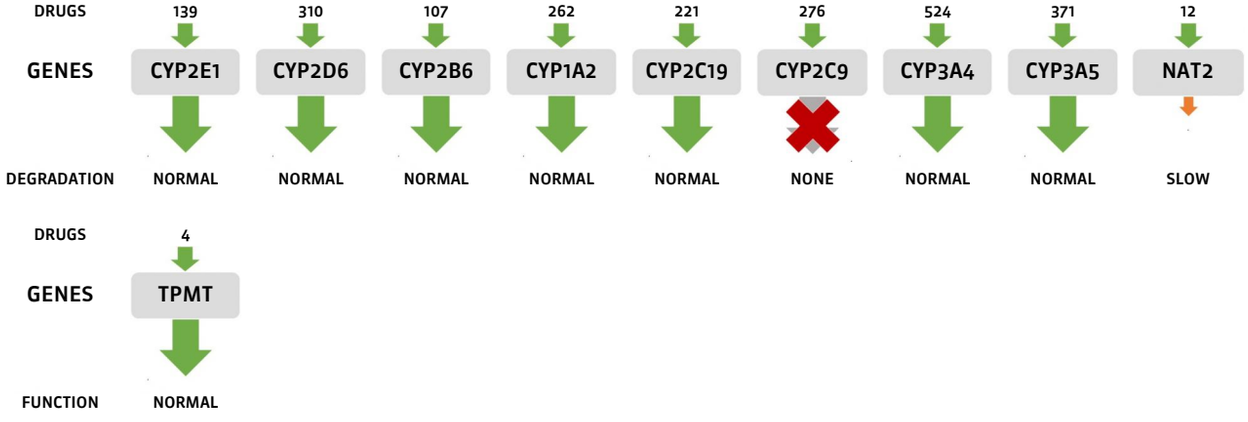
LOW AVERAGE RISK HIGHER

Prevention

Based on the genetic profile, your risk of developing Crohn's disease is not increased.

You do not need to take any special preventative or observational measures since your risk is not increased.

Drug compatibility



Effect on relevant medication

	Effect	Breakdown	Dose
Adalimumab	✓	✓	✓
Budesonide	✓	✓	✓
Corticotropin	✓	✓	✓
Diclofenac	✓	✗	✗
Infliximab	✓	✓	✓
Methylcellulose	✓	✓	✓
Olsalazine	✓	✓	✓

	Effect	Breakdown	Dose
Azathioprine	✓	✓	✓
Budesonide	✓	✓	✓
Cromoglicic Acid	✓	✓	✓
Hydrocortisone	✓	↑	↑
Mercaptopurine	✓	✓	✓
Metronidazole	✓	✓	✓
Prednisone	✓	↑	↑

	Effect	Breakdown	Dose
Balsalazide	✓	✓	✓
Ciprofloxacin	✓	✓	✓
Dexamethasone	✓	↑	↑
Ibuprofen	✓	✗	✗
Methotrexate	✓	✓	✓
Naproxen	✓	✗	✗
Sulfasalazine	✓	✓	✓

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.

Glaucoma Sensor

Detect glaucoma at an early stage and treat it properly

Glaucoma

Glaucoma is a common eye disease and it is one of the leading causes of blindness worldwide. It is estimated that there are currently 500,000 people in Germany suffering from this disease- many unaware. Approximately 10% of them are blind.

Although the disease is easily and effectively treatable with eye drops, most people are unaware of their disease because the symptoms develop slowly and are noticeable only in the advance stages of the disease. Most cases remain untreated for a long time leading to optic nerve damage and, in severe cases, to blindness.

A continuous secretion of clear liquid takes place inside the human eye. This is produced at the posterior part of the eyeball and flows to the anterior parts through the valves. The regulation between the production and secretion creates the correct pressure within the eye, which is important for the its shape and function.
A gene that plays an important role in the function of the vent valves was identified some time ago. Unfavourable genetic variations may interfere with the function of the valves so that the produced fluid cannot be properly drained. This leads to a gradual increase in the intraocular pressure causing pressure on the blood vessels that supply the optic nerve with oxygen and nutrients, thus obstructing the blood flow. If this condition persists untreated, the nerves in the eyes start to gradually wither; in extreme cases it may lead to blindness. The brain combines the image of both eyes and thus will initially compensates for the vision impairment. The disease is usually diagnosed only when both eyes are affected and the patient is experiencing difficulties, for example: overlooking parts of words when reading or having problems while driving. By this time, the optic nerves are often severely damaged resulting in, in most cases, a permanent impairment of the visual field or leading to blindness. After diagnosis, treatment focuses on reducing the eye pressure and on preventing further damage to nerve cells. Damaged nerve cells cannot be repaired.

Preventive genetic testing for glaucoma is recommended because it determines your personal risk for glaucoma. If required, start a medical monitoring program which ensures that the first signs of the disease are immediately recognized and treated properly.

Relevant genes for glaucoma

Science can identify a gene that has an influence on the operation of the drain valves in the eye. As the disease barely manifests itself, and the first vision abnormalities occur only after approximately 95% of the ocular cells have died, it is particularly important to detect the disease as early as possible. The main benefit of this genetic analysis is therefore the recognition of one's own risk, leading to earlier and more accurate eye tests; this will allow for an early diagnosis and proper treatment.

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
LOXL1	rs3825942	T>C	C/C

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

Summary of effects

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

- The risk for glaucoma is approximately 1.6 -times increased
- You should submit to regular eye examinations early on to detect the first signs of glaucoma as early as possible.

Risk for glaucoma

LOW AVERAGE RISK HIGHER

Prevention

Based on your genetic profile, you have a significantly increased risk of glaucoma; therefore, it is important that you take preventative measures in order to reduce your risk of developing the disease. Therefore, it would be advisable to consider the following screening measures, which will allow for early detection of the disease. With immediate treatment, permanent damage can be entirely prevented, in most cases.

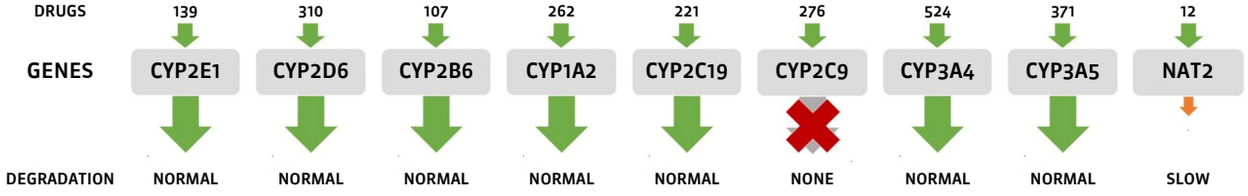
For this reason, the following screening measures apply:

Get both eyes tested by an optometrist at least once a year to identify the first signs of glaucoma. A simple measurement of intraocular pressure will not detect the early stage of glaucoma. Monitoring should include the following tests:

- Intraocular pressure measurement to determine elevated pressure
- A visual field test for both eyes to detect impairments in the visual field
- Gonioscopy, an eye examination which inspects the eye where the drain valves are located
- The examination of the optic nerve in order to detect any abnormalities.

Low blood pressure contributes to the development of the disease because the flowing blood exerts less force on the already compressed blood vessels. The nerve cells located in the eyes will be even more affected by the oxygen and nutrients. Therefore, monitor your blood pressure, and if necessary, ask your doctor about appropriate treatment.
If you follow these recommendations you should be able to detect the first signs of the disease at an early stage and be properly treated before permanent damage to the optic nerve occurs.

Drug compatibility



Effect on relevant medication

	Effect	Breakdown	Dose
Acetazolamide	✓	✓	✓
Bimatoprost	✓	✓	✓
Carteolol	✓	✓	✓
Latanoprost	✓	✓	✓
Metipranolol	✓	✓	✓
Timolol	✓	✓	✓

	Effect	Breakdown	Dose
Apraclonidine	✓	✓	✓
Brimonidine	✓	✓	✓
Dorzolamide	✓	✓	✓
Levobunolol	✓	✓	✓
Pilocarpine	✓	✓	✓
Travoprost	✓	✓	✓

	Effect	Breakdown	Dose
Betaxolol	✓	✓	✓
Brinzolamide	✓	↑	↑
Epinephrine	✓	✓	✓
Methazolamide	✓	✓	✓
Tafluprost	✓	✓	✓

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.

AMD Sensor

Macular Degeneration: effective prevention and early detection for best eye health

Macular degeneration

Macular degeneration is a painless condition affecting the retina of the human eye. The condition usually begins to slowly affect individuals over 50 years of age and impairs the centre of the visual field.

The condition results in a disruptive spot in the centre of the visual field, which can make reading and recognizing details (such as faces) difficult or even impossible without impairing peripheral vision. Macular degeneration is the most common cause of blindness in industrialized countries and roughly 30 million people worldwide are estimated to suffer from the condition. Men and women are equally affected.

The layer of tissue sensitive to light in the human eye is known as the retina. The region of the retina where light is most heavily focused is called the macula. This is the point where your vision is at its highest resolution. Macular degeneration occurs when cells in the macula die with advancing age. It may also be aggravated by the formation of new blood vessels or metabolic waste products that impair macular function. Certain environmental risk factors may accelerate these processes considerably and it is therefore advisable to minimize the risks as much as possible. Risks include: smoking, heart disease and circulatory system conditions, high blood pressure, a poor diet and extreme exposure to light. Preventative measures focus mainly on minimizing such risk factors in order to delay or prevent development of the condition.

Macular degeneration advances slowly over a long period during which symptoms are initially barely noticeable but worsen gradually. Individuals affected usually first experience difficulty reading. Some letters just seem to disappear. Straight lines and edges like window frames appear wavy. This effect can be easily detected and measured by use of a simple test. This is followed by a gradual loss in sharpness of vision, increased difficulty reading, impaired contrast sensitivity and difficulty discerning colour, and increased sensitivity to glare. In advanced stages, the centre of the visual field is often only populated by gray shadows, which themselves disappear as the condition worsens further. As the disease affects only the macula, only the centre of the visual field is affected. Macular degeneration does not cause total blindness because peripheral vision and colour vision remain unaffected. Affected individuals thus retain mobility and orientation. Treatment options for advanced macular degeneration are limited and can usually only slow but not reverse the worsening of symptoms. For this reason, prevention and early detection of macular degeneration are especially important to facilitate timely treatment of the condition.

Genes associated with macular degeneration

So far, science has identified several genes and polymorphisms linked to an increased risk of macular degeneration. By analysing all relevant polymorphisms, we are able to determine the disease risk. The following genes affect the development of macular degeneration.

Genetic traits			
SYMBOL	**rs NCBI**	**POLYMORPH**	**GENOTYPE**
HTRA1	rs11200638	G>A	G/G
CFH	rs1061170	Y402H, T>C	T/C
LOC387715	rs10490924	G>T	G/G

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

Summary of effects

- You do not have an elevated risk of macular degeneration
- Your requirement of antioxidants is average for your symptoms

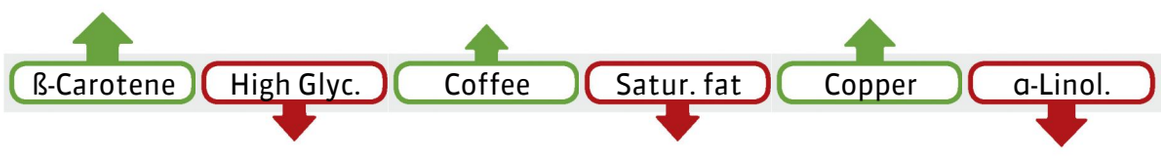
Your risk of macular degeneration

Required antioxidants

Nutritional Genes - Eyes

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

You do not have a genetic predisposition for macular degeneration. You do not need to take special preventive or observational measures because your risk is approximately equal to that of the general population. However, you can still develop macular degeneration, and if you notice symptoms you should discuss them with your doctor.

Even people who have no genetic risk can develop macular degeneration. Therefore, you should have an annual eye test after age 40 to allow for early detection and early treatment of the disease.

- High blood pressure is a risk factor for macular degeneration. Make sure your blood pressure is within the normal range. You can lower your blood pressure by getting more exercise and adopting the right diet. If diet and exercise do not lower your blood pressure, talk with your doctor about using medication to reduce it.
- Smoking is a major risk factor in the development of macular degeneration, and should be avoided.
- Protect your eyes from direct sunlight by wearing UV-protective sunglasses or a hat.
- Make sure that your diet includes sufficient amounts of antioxidants, such as vitamins. These are found in fruit and vegetables, and are also available in concentrated form as dietary supplements.

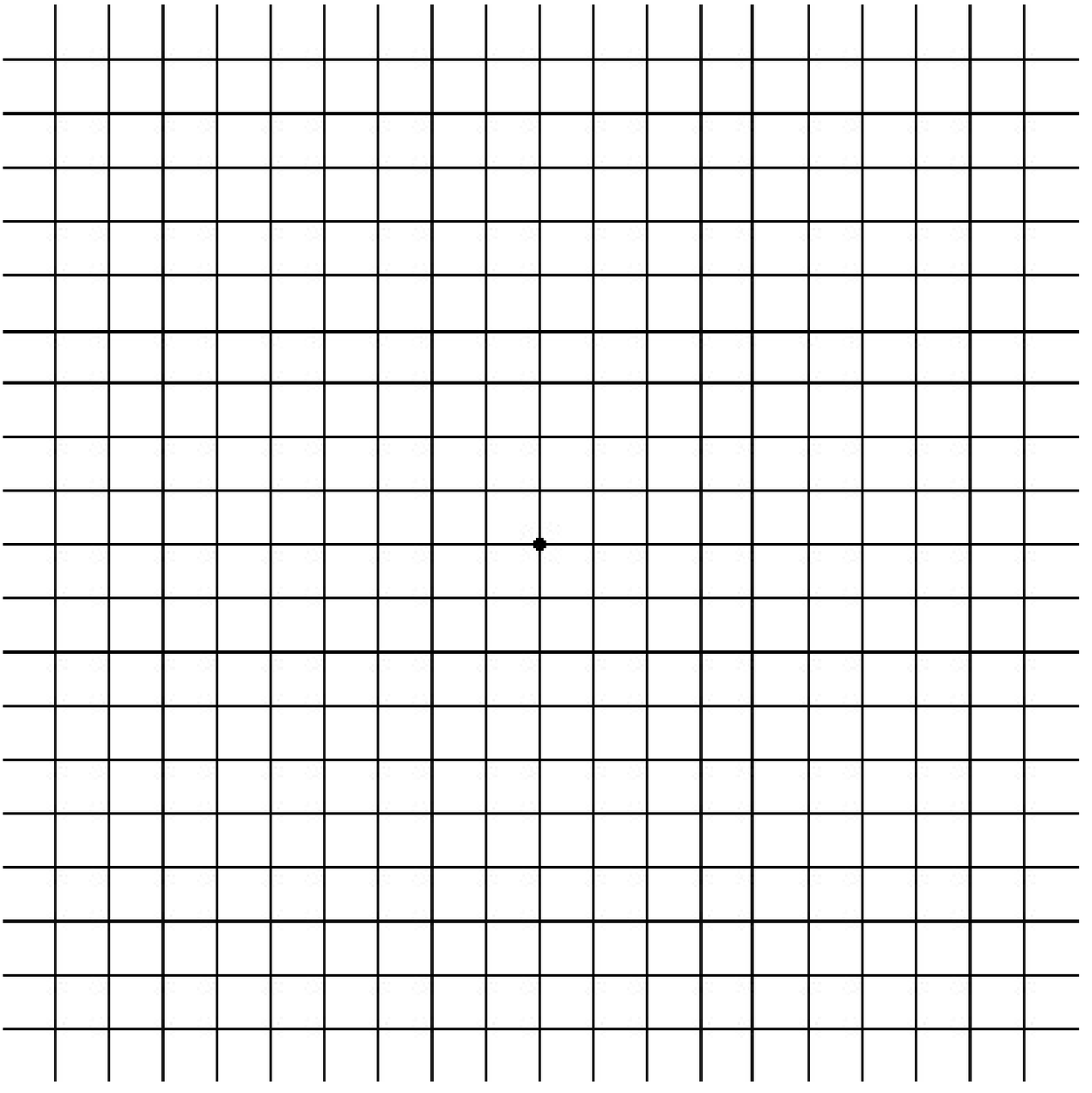
The following sources are recommended:

- Beta carotene
- Copper
- Vitamin C
- Vitamin E (α-Tocopherol)
- Zinc

Macular degeneration is slow to develop and painless, therefore pay particular attention to certain symptoms because early detection plays an important role in determining the best treatment. The symptoms include, amongst others, shadowed or distorted vision (eg. window frames appear wavy), or difficulty in reading (eg. when individual letters disappear). The Amsler grid test will allow you to identify the first signs of distortion in your field of vision. The test can be found on the next page together with instructions on how to take it. If you notice any symptoms, consult your eye doctor immediately.

Instructions for macular degeneration self-examination

- Hold the Amsler grid at a comfortable reading distance.
- Cover one eye (if you have reading glasses, please put them on).
- Focus on the exact point in the middle with the other eye.
- Look for wavy or blurred lines.
- This may indicate symptoms of age-related macular degeneration.
- Repeat the test with the other eye!
- If the you see the irregularities described, contact your optometrist immediately.
- Repeat this self-test once a week.



You do not have a genetic predisposition for macular degeneration. You do not need to take special preventive or observational measures because your risk is approximately equal to that of the general population. However, you can still develop macular degeneration, and if you notice symptoms you should discuss them with your doctor.

PHARMACO GENETICS

ONCOLOGY

CARDIOVASCULAR SYSTEM

NEUROLOGY

METABOLISM

MOVEMENT

DIGESTION

OPHTHALMOLOGY

ODONTOLOGY

OTHERS

SCIENCE

ADDITIONAL INFORMATION

Periodontitis Sensor

Prevent periodontal disease and select the proper implant type

Periodontitis

Inflammatory periodontitis disease affects the gums and the jawbone. Periodontitis disease and tooth decay are the two major oral diseases. More than half of the population between ages 35 and 44 suffers from tooth decay, and about 20% of the population has severe tooth decay. Most cases result from poor oral hygiene. Tooth decay is caused primarily by high consumption of refined sugar but good oral hygiene helps us prevent tooth decay so that more people have their own teeth at an older age. Since the age of the teeth plays an important role in developing periodontitis, this disease is more common in older people. 40% of the population older than 65 suffer from a severe form of the disease.

Plaque is constantly forming in the mouth from a combination of food particles, saliva and bacteria. If this plaque is not removed regularly with brushing and dental care, bacteria breaks the sugar contained in food into acids that attack tooth enamel and cause cavities. Over time, additional material accumulates, which makes the condition worse. In addition to destroying tooth enamel, plaque and tartar penetrate the gums causing the immune system to fight the resulting inflammation. This causes gingivitis, which is the persistent inflammation of the gums. Normally, the immune system can prevent bacteria from spreading further. However, in people with weakened immune systems or other complicating factors, the bacteria are able to spread, infecting part of the jawbone that anchors the teeth. In response, the immune system produces enzymes and chemical mediators to fight the bacterial infection but this also attacks and gradually destroys the oral tissues. This powerful immune reaction causes inflammation in the entire jawbone, which causes the tooth to gradually loosen until it falls out. Often, the whole jaw is affected because the tooth only loosens after the disease has progressed significantly. Periodontitis is caused by a combination of many factors but poor oral hygiene and certain genetic traits play a crucial role. Since most of the damage is caused by an immune response, genetic traits that make the response too aggressive can lead to severe periodontitis. However, the immune system responds only when bacteria penetrate into the tissue. This means that people carrying these genetic traits need to take special care of their teeth by avoiding several risk factors:

- Poor oral hygiene with dental plaque and tartar buildup
- Tobacco use, since smoking increases the risk by 4 to 6 times
- Contracting periodontal disease from other infected people (especially within the family)
- Tooth decay (cavities)
- Mouth breathing
- Teeth Grinding
- Poor diet
- Piercings in the mouth, lips, frenulum or tongue
- Diabetes, especially uncontrolled or poorly controlled
- Pregnancy, hormonal changes loosen connective tissue which makes it easier for bacteria to penetrate into the gums
- Weakened immune system, such as after chemotherapy, organ transplant or HIV

If the disease is detected early, it can usually be treated very effectively. However, the patient must maintain good oral hygiene in

order to prevent a recurrence since people who have previously suffered from periodontal disease are at an increased risk of relapse. If it is not diagnosed and treated in time, periodontal disease usually leads to tooth loss, which causes aesthetic and functional problems. This genetic analysis will inform you if have an increased risk, so that you can take preventive measures and get regular dental checkups to prevent the disease.

Titanium implant loss due to genetic variations:

Titanium is a popular material for dental implants because it causes no allergic reactions and bonds firmly with the surrounding bone within 3-6 months. The success of implant varies from person to person but some people can keep an inserted implant for several decades, while others lose the implant after 4 months. This is difference is caused by the varying inflammatory responses to titanium, which is triggered by four different genetic polymorphisms.

For people with the optimal genetic profile, the implant failure rate is approximately 3%. However, the risk of implant failure can be as high as 60% for some people, depending on the number of unfavourable genetic variations. People with an unfavorable genetic profile, which is determined by the genetic analysis, have the opportunity to choose the most appropriate implant, and thus prevent its premature loss.

Relevant genes for periodontitis

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

Genetic traits			
SYMBOL	**rs NCBI**	**POLYMORPH**	**GENOTYPE**
IL1RN	rs419598	C>T	C/T
IL6	rs1800795	G>C	G/C
IL1A	rs1800587	C>T	C/C
IL 1 Beta	rs1143634	C>T	C/T
TNFa	rs1800629	G>A	A/A

Probability of a titanium implant loss:

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
IL1RN	rs419598	C>T	C/T
IL1A	rs1800587	C>T	C/C
IL 1 Beta	rs1143634	C>T	C/T
TNFa	rs1800629	G>A	A/A

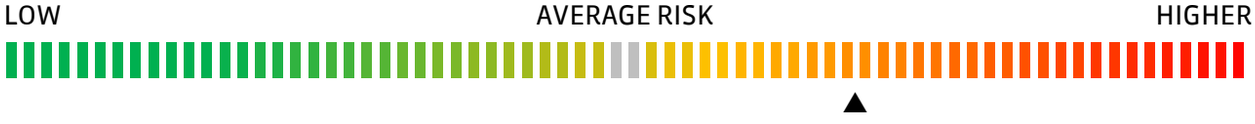
LEGEND: rsNCBI = description of examined genetic variation, POLYMORPHISM = form of the genetic variation, GENOTYPE = personal analysis result

Summary of effects

Several genes have an effect on the aggressiveness of the immune system. Some of these genes may increase the likelihood that an infected jawbone is destroyed by an overactive immune response so that the teeth eventually become loose and fall out. Other genes control how the immune system reacts to titanium implants. If these genes are defective your immune system may reject the titanium implant and you could lose the implant within four weeks.

- Your risk of developing periodontal disease is approximately 3.3 -times increased
- Increased risk of titanium implant loss (OR:4.2)

Risk for periodontitis



Probability of a titanium implant loss

Prevention

Based on your genetic profile, you have an increased risk of developing periodontitis. Preventative measures are very important for you so that you can continue to maintain healthy teeth and gums.

The best way to prevent periodontal disease is to brush your teeth consistently and have regular dental checkups. Disease prevention consists essentially of regular and thorough dental care and medical supervision.

The following points are important for you:

- Brush your teeth in the morning, after every meal and especially in the evening
- Replace your toothbrush regularly
- Use floss or an interdental brush to clean between your teeth.
- Clean the tongue
- Have your teeth cleaned by a dental hygienist every 3 to 6 months.
- Do not eat sugar more than once a day and brush thoroughly immediately afterwards
- Visit the dentist more frequently if you are pregnant, diabetic, or immunodeficient.

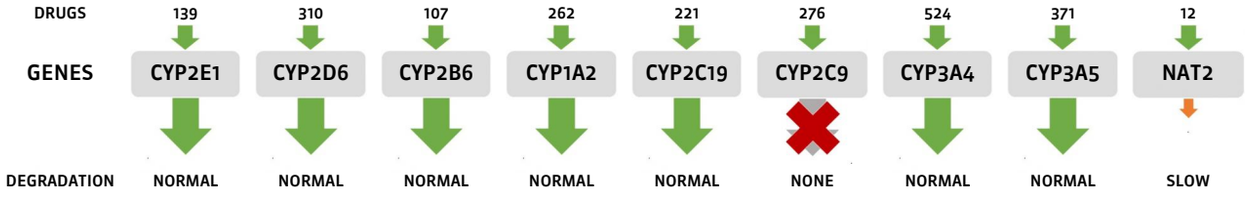
Also avoid, if possible, the following risk factors:

- Poor or improper oral hygiene with dental plaque and tartar
- Tobacco use, since smoking increases the risk 4 to 6-fold
- Contracting periodontal disease from other infected people (especially within the family)
- Tooth decay
- Mouth breathing
- Teeth grinding
- Poor diet
- Piercings in the mouth, lips, frenulum or tongue
- Diabetes, especially uncontrolled or when the blood sugar is poorly controlled
- During pregnancy, hormonal changes loosen the connective tissue and bacteria can easily penetrate into the gums
- A compromised immune system, such as after chemotherapy, organ transplant or HIV

Titanium implant loss due to genetic variations::

Since you have three genetic variations which increase the aggressiveness of your immune system, you have a significantly higher chance of rejecting an implant. You can decide, together with your dentist, if titanium is the appropriate implant material or whether you should choose alternatives such as ceramic implants, zirconium oxide implants, coated titanium implants or removable dentures or bridges.

Drug compatibility



Effect on relevant medication

	Effect	Breakdown	Dose
Amoxicillin	✓	✓	✓
Codeine	✓	✓	✓
Lidocain	✓	✓	✓
Tetracycline	✓	✓	✓

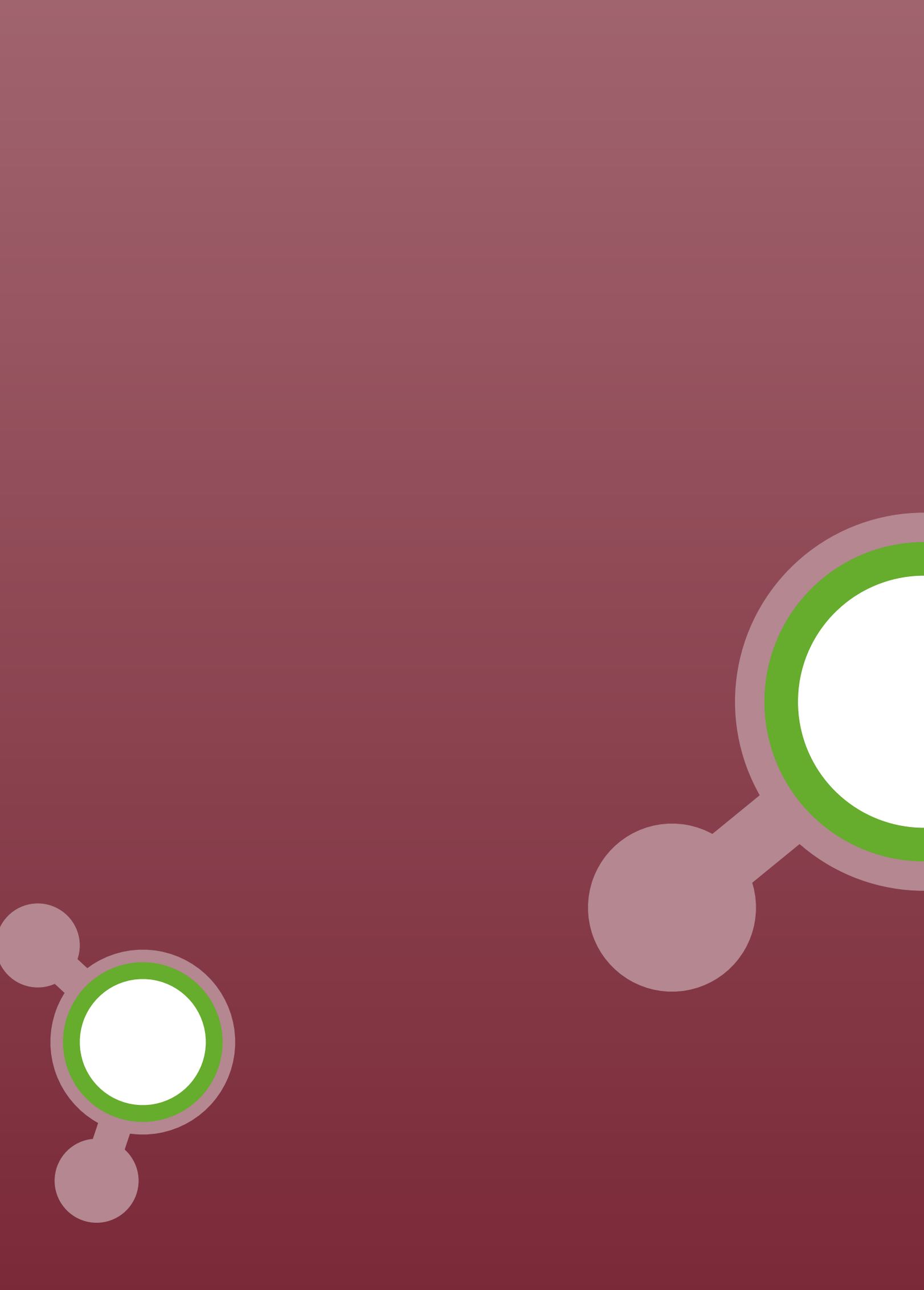
	Effect	Breakdown	Dose
Carbamide	✓	✓	✓
Doxycycline	✓	↑	↑
Metronidazole	✓	✓	✓
Triclofos	✓	✓	✓

	Effect	Breakdown	Dose
Chlorhexidine	✓	✓	✓
Ibuprofen	✓	✗	✗
Minocycline	✓	✓	✓

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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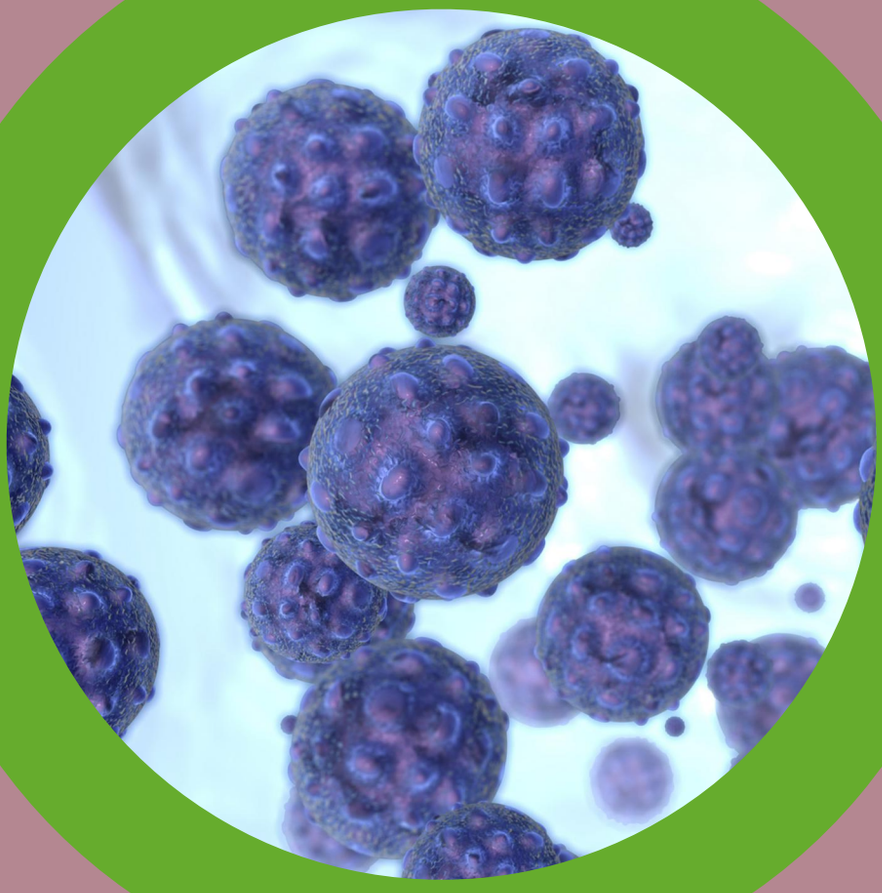
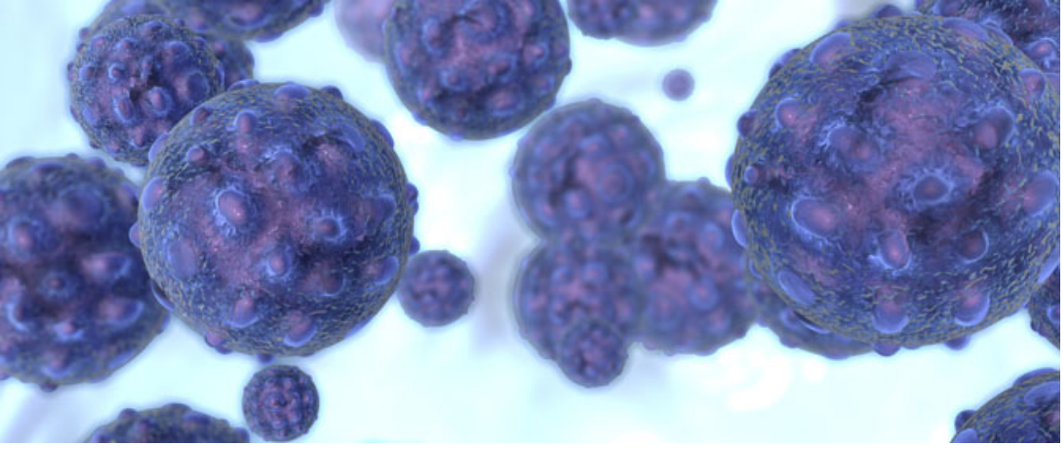


HIV Resistance Sensor

Risk of infection and optimized therapy



HIV - Human Immunodeficiency Virus

The human immunodeficiency virus, also called HIV virus, has become widespread since the 1980s and has infected about 34 million people so far. The infection is usually present for decades or years without noticeable symptoms, until eventually the immune system is weakened to a point where other infections invade the body and eventually cause death.

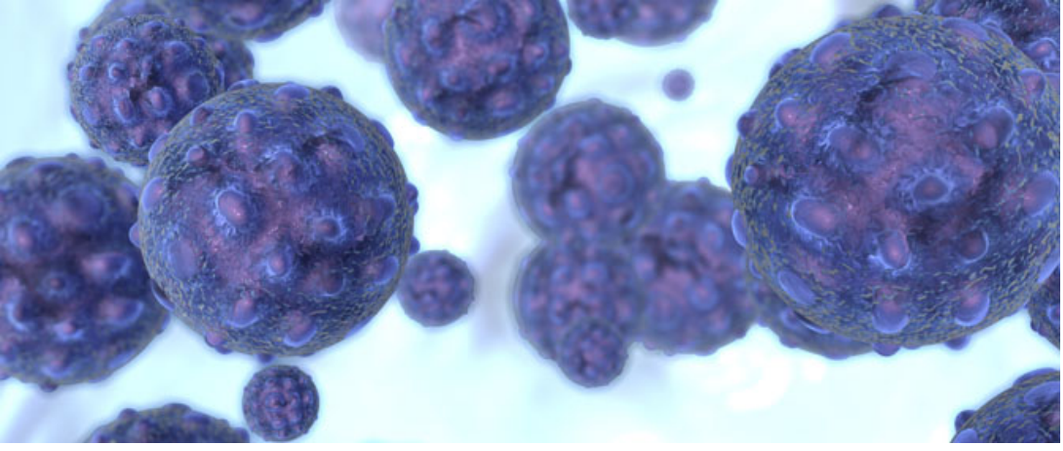
A virus can be described as a self-replicating machine. It usually consists of just a few genes, which are surrounded by a protein capsule. The surface of this capsule has the property of binding to special elements- which are called receptors- of certain cells of the body, and then transfer its genes into the interior of the cell. There, depending on the type of virus, it replicates its genes and sometimes incorporates them into the genome of the cells.

The infected cell cannot differentiate between its own genes and the viral genes, and so it activates them all. Viral genes have different functions. Some of the genes produce the building blocks of the protein capsule, while others copy and carry the viral genes into the new empty capsules. The new viruses then leave the cell and infect new cells, where the same process repeats.

Each form of virus infects only certain cells because each virus needs specific receptors. For HIV, these are the cells of the immune system. The receptors which are required by HIV viruses are CD4 and CCR5. For each of the receptors, a human gene reveals to the cell how to build the receptors. About 20% of the population has a genetic variation in a CR5 gene (CCR5delta32) and therefore produces only about half of the CCR5 receptors. This leads to a lower surface for the virus and considerably reduces the risk of infection. About 1% of the population has this mutation in both CCR5 genes, and is therefore very highly resistant to HIV.

CCR5 receptors are essential for HIV infection and a drug that blocks the CCR5 receptors has already been developed (maraviroc). Other medicines for HIV try to block the replication of the viral genes or interfere in the cycle of the virus in other ways. Without medical treatment, the HIV infection is usually fatal within several years. With drug therapy, however, HIV infection is similar to a chronic disease, and the majority of infected people have a normal life expectancy of over 70 years. Therefore, an effective therapy is of great importance.

Due to genetic differences in the genes that convert drugs in the body, it is possible that certain drugs are either not activated or their efficacy is low, resulting in either over- or under-dosing. Therefore, a genetic analysis of the conversion capability of HIV-related drugs is extremely important for the optimal therapy.



Relevant genes for HIV

An analysis of HIV-related genes determines the risk of HIV infection, estimates the progression of the disease and helps in the optimization of drug therapy. Since there are many other sexually transmitted diseases besides HIV, having a degree of HIV resistance should not to be perceived as freedom to engage in unprotected sexual intercourse.Regardless of the genetic predisposition to HIV, the use of condoms in risky sexual intercourse is recommended.

Genetic traits			
SYMBOL	rs NCBI	POLYMORPH	GENOTYPE

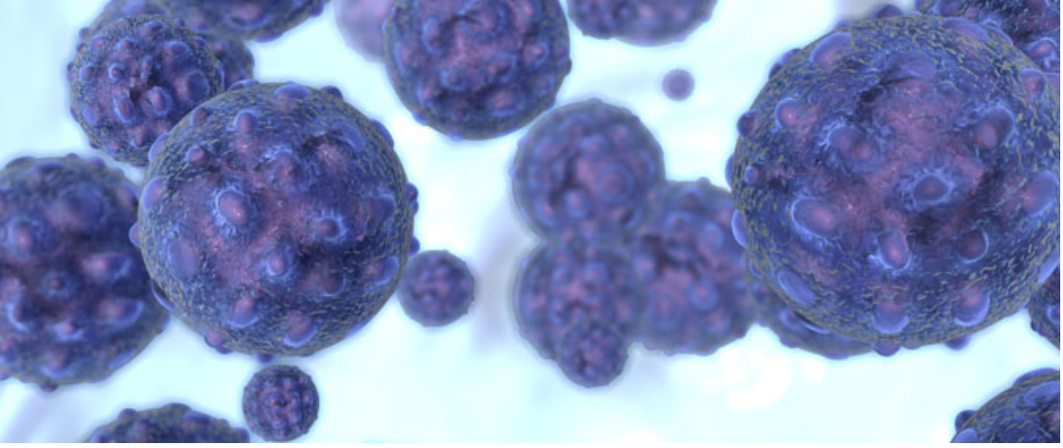
LEGEND: rsNCBI = description of examined genetic variation, POLYMORPHISM = form of the genetic variation, GENOTYPE = personal analysis result

Summary of effects

Based on your genetic profile your cells produce CCR5 receptors needed by the virus. Your infection risk in case of contact is the same as for the general population.

Risk of HIV infection in case of contact

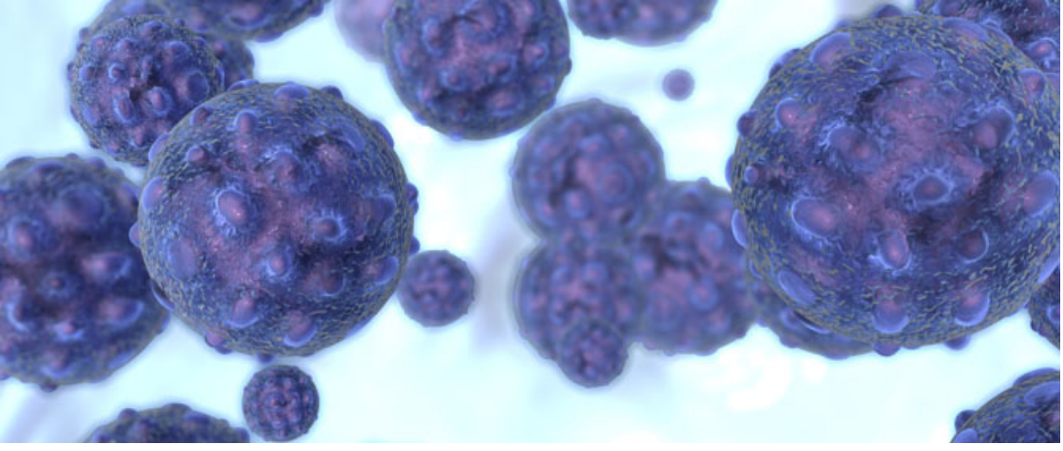
Please note: HIV infection can never be completely excluded even with a favourable genetic CCR5 variant. Some HIV strains do not use the CCR5 receptor, but another called CXCR4. Absolute immunity is therefore not possible.



Prevention

An analysis of HIV-related genes determines the risk of HIV infection, estimates the progression of the disease and helps in the optimization of drug therapy. Since there are many other sexually transmitted diseases besides HIV, having a degree of HIV resistance should not to be perceived as freedom to engage in unprotected sexual intercourse. Regardless of the genetic predisposition to HIV, the use of condoms in risky sexual intercourse is recommended.

Based on your genetic profile your cells produce CCR5 receptors needed by the virus. Your infection risk in case of contact is the same as for the general population.



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
DRUGS									
GENES									
FUNCTION									

Effect on relevant medication

	Effect	Breakdown	Dose
Abacavir	✓	✓	✗
Clarithromycin	✓	↑	↑
Efavirenz	✓	↑	↑
Hydroxychloroquine	✓	✓	✓
Itraconazole	✓	↑	↑
Maraviroc	✓	✓	✓
Proguanil	✓	✓	✓
Rifampicin	✓	↓	↓
Saquinavir	✓	↑	↑
Telithromycin	✓	↑	↑

	Effect	Breakdown	Dose
Atazanavir	✓	↑	✗
Darunavir	✓	↑	↑
Etravirine	✓	✓	✓
Indinavir	✓	↑	↑
Lopinavir	✓	↑	↑
Nelfinavir	✓	↑	↑
Pyrazinamide	✓	✓	✓
Rilpivirine	✓	✓	✓
Sulfadiazine	✓	✗	✗
Tipranavir	✓	↑	↑

	Effect	Breakdown	Dose
Caspofungin	✓	✓	✓
Delavirdine	✓	↑	↑
Fosamprenavir	✓	↑	↑
Isoniazid	✓	✗	✗
Lopinavir	✓	↑	↑
Nevirapine	✓	↑	↑
Raltegravir	✓	✓	✓
Ritonavir	✓	↑	↑
Sulfapyridine	✓	✓	✓
Voriconazole	✓	✗	✓

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.

HRT

Risk assessment and optimized therapy

Hormone replacement therapy (HRT)

Menopause is a natural stage of every woman's life, when the ovaries cease the hormone production of oestrogens, progesterone and androgens. During this time, women may experience hormone deficiency, manifested with symptoms such as hot flashes, sweating at night, insomnia and vaginal dryness. Hormone replacement therapy (HRT) can replace these missing hormones and alleviate the discomfort.

Menopause is a stage in every woman's life, when natural hormonal changes take place. This normally occurs between the ages of 45 and 55, and usually does not require any treatment. However, due to the decrease of female sex hormones, menopause may lead to physical and mental ailments. These menopausal symptoms include:

- Hot flashes
- Sweat during the nights
- Tachycardia
- Sleep disorders
- Dizziness
- Lack of libido
- Vaginal dryness
- Depression
- Apathy

Hormone replacement therapy (HRT) may alleviate the climacteric symptoms, which are triggered by hormone deficiency, and can seriously affect quality of life. It can be either a monotherapy carried out only with oestrogens, or a combined therapy with estrogen and progestin. Comprehensive medical analyses should be carried out before starting the treatment.

Although hormone therapy has many advantages, many scientific studies have shown that HRT also increases the risk of breast cancer and thrombosis slightly. If other risk factors also exist (such as a genetic predisposition), it is recommended to follow alternative therapies.

Please note: Not all HRTs are the same. There are two common types of hormone treatments available: natural bio-identical HRT and synthetic drug-based HRT. Numerous studies have demonstrated an increased risk of breast cancer using synthetic oestrogens and progestins. However, research has revealed that natural oestriol and progesterone have the ability to defend against breast cancer. Compared to synthetic progestin that stimulates breast cell proliferation, natural progesterone has demonstrated a protective effect. So, the latest research suggests that bio-identical hormones are safer than commonly used synthetic versions.

Relevant genes for HRT

Scientific studies have identified several genetic variations that significantly increase the risk of HRT-associated thrombosis. Considering this increase, together with the genetic variations of the breast cancer risk, the risk of hormone replacement therapy (HRT) can be estimated. The gene analysis came to the following conclusion:

Genetic traits

SYMBOL	rs NCBI	POLYMORPH	GENOTYPE
FGFR2	rs2981582	G>A	C/C
VDR	rs2228570	VDR FokI T/C	T/C
8q24	rs13281615	T>C	G/A
TNRC9	rs3803662	C>T	C/C
MAP3K1	rs889312	A>C	A/C
LSP1	rs3817198	T>C	T/C
CASP8	rs1045485	D302H (G/C)	G/G
2q35	rs13387042	G>A	A/A
XRCC2	rs3218536	A>G	G/G
Factor-V	rs6025	G>A	G/G
Factor-II	rs1799963	G>A	G/G
PAI1	rs1799889	G>del	del/del
VDR	rs1544410	G/A IVS7 Pos.+283	A/A
ApoE type	combination	E2/E3/E4	E2/E3

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

Summary of effects

- Your risk of developing a HRT-associated thrombosis is not increased
- Your risk of developing breast cancer is lower than the population average.
- HRT is very effective in lowering the total and the LDL cholesterol levels
- There are no contraindications for ERT/HRT. However, due to possible long-term side effects, it is recommended to avoid preventive, oestrogen-containing HRT therapies for a period of 4-6 years

Your risk of HRT- associated thrombosis

LOW HIGHER

HRT for preventing osteoporosis

NORMAL VERY EFFECTIVE

Your risk of developing breast cancer

LOW HIGHER

HRT for lowering the cholesterol values

NORMAL VERY EFFECTIVE

Prevention

Because your risk of breast cancer or thrombosis is the same as for the general population, there are no contraindications regarding the hormone replacement therapy.

Since it has been shown that a synthetic HRT, over a longer period of time, increases the risk of breast cancer and HRT-associated thrombosis, it should only be used when the symptoms affect quality of life. In addition, the hormone preparations should only be used in the lowest effective dose, and for the shortest period possible.

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SCIENCE

This chapter shows the science behind the test.

Pharmaco Sensor

CYP2D6 - cytochrome P450, family 2, subfamily D, polypeptide 6

Cytochrome P450 2D6 (CYP2D6) is an enzyme that is involved in the metabolism of drugs through oxidation or hydrolysis of various substrates. This process is strongly influenced by the genetic variant of the CYP2D6 gene or allele.

RES	Genotype	POP	Possible results
	UM	9%	Drugs metabolized by this enzyme are degraded too quickly Prodrugs metabolized by this enzyme are activated too quickly
X	EM	70%	Drugs are metabolized by this enzyme as normal Prodrugs are activated by this enzyme as normal
	IM	16%	Drugs are metabolized by this enzyme at a slow rate Prodrugs can barely be activated by this enzyme
	PM	5%	Drugs are metabolized by this enzyme at a very slow rate Prodrugs are not activated by this enzyme

References

Zhou SF. et al. Polymorphism of human cytochrome P450 2D6 and its clinical significance: Part I. Clin Pharmacokinet. 2009,48(11):689-723.

Stüven et al. Rapid detection of CYP2D6 null alleles by long distance- and multiplex-polymerase chain reaction. Pharmacogenetics. 1996 Oct,6(5):417-21.

Hicks JK et al. Clinical Pharmacogenetics Implementation Consortium (CPIC) Guideline for CYP2D6 and CYP2C19 Genotypes and Dosing of Selective Serotonin Reuptake Inhibitors. Clin Pharmacol Ther. 2015 Aug,98(2):127-34.

CYP2B6 - cytochrome P450, family 2, subfamily B, polypeptide 6

CYP2B6 is metabolizing a variety of drugs similar to other Cytochrome P450 enzymes.

RES	Genotype	POP	Possible results
	UM	1%	Drugs metabolized by this enzyme are degraded too quickly Prodrugs metabolized by this enzyme are activated too quickly
	RM	1%	Drugs metabolized by this enzyme are degraded too quickly Prodrugs metabolized by this enzyme are activated too quickly
X	EM	96%	Drugs are metabolized by this enzyme as normal Prodrugs are activated by this enzyme as normal
	IM	1%	Drugs are metabolized by this enzyme at a slow rate Prodrugs can barely be activated by this enzyme
	PM	1%	Drugs are metabolized by this enzyme at a very slow rate Prodrugs are not activated by this enzyme

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Zanger UM et al. Pharmacogenetics of cytochrome P450 2B6 (CYP2B6): advances on polymorphisms, mechanisms, and clinical relevance. Front Genet. 2013 Mar 5,4:24.

Kharasch ED et al. Methadone Pharmacogenetics: CYP2B6 Polymorphisms Determine Plasma Concentrations, Clearance, and Metabolism. Anesthesiology. 2015 Nov,123(5):1142-53.

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Gatanaga H et al. Successful efavirenz dose reduction in HIV type 1-infected individuals with cytochrome P450 2B6 *6 and *26. Clin Infect Dis. 2007 Nov 1,45(9):1230-7.

CYP1A2 - cytochrome P450, family 1, subfamily A, polypeptide 2

CYP1A2 (cytochrome P450 1A2) is a heme protein- enzyme involved in various metabolic processes. It metabolizes various xenobiotics such as caffeine, aflatoxin B1 and medications like paracetamol.

RES	Genotype	POP	Possible results
	UM	14%	Drugs metabolized by this enzyme are degraded too quickly Prodrugs metabolized by this enzyme are activated too quickly
X	EM	53%	Drugs are metabolized by this enzyme as normal Prodrugs are activated by this enzyme as normal
	IM	28%	Drugs are metabolized by this enzyme at a slow rate Prodrugs can barely be activated by this enzyme
	PM	5%	Drugs are metabolized by this enzyme at a very slow rate Prodrugs are not activated by this enzyme

References

Hubacek JA. et al. Drug metabolising enzyme polymorphisms in Middle- and Eastern-European Slavic populations. Drug Metabol Drug Interact. 2014,29(1):29-36.

Kuo HW et al. CYP1A2 genetic polymorphisms are associated with early antidepressant escitalopram metabolism and adverse reactions. Pharmacogenomics. 2013 Jul,14(10):1191-201.

Lin KM et al. CYP1A2 genetic polymorphisms are associated with treatment response to the antidepressant paroxetine. Pharmacogenomics. 2010 Nov,11(11):1535-43.

CYP2C19 - cytochrome P450, family 2, subfamily C, polypeptide 19

The cytochrome P450 2C19 (CYP2C19) enzyme is involved in the oxidative metabolism of various drugs, such as: antidepressants, antipsychotics, tranquilizers and proton pump inhibitors. CYP2C19 provides an alternative metabolic pathway for CPY2D6. Defects in the CYP2C19 gene can increase or decrease the enzymatic activity.

RES	Genotype	POP	Possible results
	UM	5%	Drugs metabolized by this enzyme are degraded too quickly Prodrugs metabolized by this enzyme are activated too quickly
	RM	27%	Drugs metabolized by this enzyme are degraded too quickly Prodrugs metabolized by this enzyme are activated too quickly
X	EM	39%	Drugs are metabolized by this enzyme as normal Prodrugs are activated by this enzyme as normal
	IM	27%	Drugs are metabolized by this enzyme at a slow rate Prodrugs can barely be activated by this enzyme
	PM	2%	Drugs are metabolized by this enzyme at a very slow rate Prodrugs are not activated by this enzyme

References

Sheffield L. J. et al. Clinical use of pharmacogenomic tests in 2009. Clin Biochem Rev. 2009 May,30(2):55-65.

Hodgson K. et al. Genetic differences in cytochrome P450 enzymes and antidepressant treatment response. J Psychopharmacol. 2014 Feb,28(2):133-41.

Hicks JK et al. Clinical Pharmacogenetics Implementation Consortium (CPIC) Guideline for CYP2D6 and CYP2C19 Genotypes and Dosing of Selective Serotonin Reuptake Inhibitors. Clin Pharmacol Ther. 2015 Aug,98(2):127-34.

CYP2C9 - cytochrome P450, family 2, subfamily C, polypeptide 9

Cytochrome P450 2C9 (CYP2C9) enzyme is expressed mainly in the liver, where it is involved in the oxidation of xenobiotic and endogenous substances. CYP2C9 plays an important role in the metabolism of various drugs. Defects in the CYP2C9 gene are associated with a reduced enzyme activity.

RES	Genotype	POP	Possible results
	EM	60%	Drugs are metabolized by this enzyme as normal Prodrugs are activated by this enzyme as normal
	IM	35%	Drugs are metabolized by this enzyme at a slow rate Prodrugs can barely be activated by this enzyme
X	PM	5%	Drugs are metabolized by this enzyme at a very slow rate Prodrugs are not activated by this enzyme

References

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CYP3A4 - cytochrome P450, family 3, subfamily A, polypeptide 4

The cytochrome P450 3A4 (CYP3A4) is expressed in the liver, and it is involved in the activation or hydroxylation of various drugs and endogenous substances.

RES	Genotype	POP	Possible results
X	EM	96%	Drugs are metabolized by this enzyme as normal Prodrugs are activated by this enzyme as normal
	IM	3%	Drugs are metabolized by this enzyme at a slow rate Prodrugs can barely be activated by this enzyme
	PM	1%	Drugs are metabolized by this enzyme at a very slow rate Prodrugs are not activated by this enzyme

References

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Okubo M et al. CYP3A4 intron 6 C>T polymorphism (CYP3A4*22) is associated with reduced CYP3A4 protein level and function in human liver microsomes. J Toxicol Sci. 2013,38(3):349-54.

CYP3A5 - cytochrome P450, family 3, subfamily A, polypeptide 5

The cytochrome P450 3A5 (CYP3A5) is expressed in the liver, and it is involved in the activation or hydroxylation of various drugs and endogenous substances.

RES	Genotype	POP	Possible results
X	EM	1%	Drugs are metabolized by this enzyme as normal Prodrugs are activated by this enzyme as normal
	IM	30%	Drugs are metabolized by this enzyme at a slow rate Prodrugs can barely be activated by this enzyme
	PM	69%	Drugs are metabolized by this enzyme at a very slow rate Prodrugs are not activated by this enzyme

References

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CYP2E1 - cytochrome P450, family 2, subfamily E, polypeptide 1

The cytochrome P450 2E1 (CYP2E1) is expressed in the liver, and it is involved in the activation or hydroxylation of various drugs and endogenous substances.

RES	Genotype	POP	Possible results
X	EM	98%	Drugs are metabolized by this enzyme as normal Prodrugs are activated by this enzyme as normal
	IM	1%	Drugs are metabolized by this enzyme at a slow rate Prodrugs can barely be activated by this enzyme
	PM	1%	Drugs are metabolized by this enzyme at a very slow rate Prodrugs are not activated by this enzyme

References

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De Bock L. et al. Quantification of cytochrome 2E1 in human liver microsomes using a validated indirect ELISA. J Pharm Biomed Anal. 2014 Jan 25,88:536-41.

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NAT2 - N-acetyltransferase 2 (arylamine N-acetyltransferase)

The arylamine N-acetyltransferase 2 (NAT2) is involved in the detoxification of drugs and endogenous substances through acetylation. Toxic and carcinogenic substances are converted and can be eliminated. The polymorphisms can alter the enzymatic activity of the NAT2 protein.

RES	Genotype	POP	Possible results
	EM	45%	Drugs are metabolized by this enzyme as normal Prodrugs are activated by this enzyme as normal
X	IM	30%	Drugs are metabolized by this enzyme at a slow rate Prodrugs can barely be activated by this enzyme
	PM	25%	Drugs are metabolized by this enzyme at a very slow rate Prodrugs are not activated by this enzyme

References

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VKORC - Vitamin K epoxide reductase complex (rs9923231)

The vitamin K epoxide reductase-(VKOR) is a membrane protein in the ER (endoplasmic reticulum), and it is involved in the formation of blood clotting factors. The anticoagulant warfarin inhibits the activity of the VKOR protein. This inhibition can be prevented by defects of the VKORC gene.

RES	Genotype	POP	Possible results
X	C/C	40%	No dose adjustments for various drugs
	C/T	40%	Dose adjustments for various drugs
	T/T	20%	Dose adjustments for various drugs

References

Swen JJ et al. Pharmacogenetics: from bench to byte--an update of guidelines. Clin Pharmacol Ther. 2011 May,89(5):662-73.

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International Warfarin Pharmacogenetics Consortium Estimation of the warfarin dose with clinical and pharmacogenetic data. N Engl J Med. 2009 Feb 19,360(8):753-64.

DPYD- Dihydropyrimidine dehydrogenase (rs3918290)

The DPYD gene provides instructions for making an enzyme called dihydropyrimidine dehydrogenase, which is involved in the breakdown of uracil and thymine. Genetic variations in this gene result in an error in pyrimidine metabolism and an increased risk of toxicity in patients receiving special chemotherapy.

RES	Genotype	POP	Possible results
	EM	98%	Drugs are metabolized by this enzyme as normal Prodrugs are activated by this enzyme as normal
	IM	1%	Drugs are metabolized by this enzyme at a slow rate Prodrugs can barely be activated by this enzyme
X	PM	1%	Drugs are metabolized by this enzyme at a very slow rate Prodrugs are not activated by this enzyme

References

Amstutz U et al. Clinical Pharmacogenetics Implementation Consortium (CPIC) Guideline for Dihydropyrimidine Dehydrogenase Genotype and Fluoropyrimidine Dosing: 2017 Update. Clin Pharmacol Ther. 2018 Feb103(2):210-216.

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Mattison LK et al. Implications of dihydropyrimidine dehydrogenase on 5-fluorouracil pharmacogenetics and pharmacogenomics. Pharmacogenomics. 2002 Jul,3(4):485-92.

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	30%	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	44%	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	26%	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.

SLCO1B1 - Solute carrier organic anion transporter family member 1B1 (rs4149056)

The SLCO1B1 gene provides instructions for making a protein called organic anion transporting polypeptide 1B1, or OATP1B1. OATP1B1 is found in the liver and involved in the removal of drug compounds such as statins.

RES	Genotype	POP	Possible results
	EM	84%	Drugs are metabolized by this enzyme as normal Prodrugs are activated by this enzyme as normal
X	IM	15%	Drugs are metabolized by this enzyme at a slow rate Prodrugs can barely be activated by this enzyme
	PM	1%	Drugs are metabolized by this enzyme at a very slow rate Prodrugs are not activated by this enzyme

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UGT1A1 - UDP glucuronosyltransferase family 1 member A1 (rs3064744)

UDP-Glucuronosyltransferase is an enzyme that takes part in bilirubin glucuronidation and metabolism, and degrading a variety of drugs.

RES	Genotype	POP	Possible results
	EM	91%	Drugs are metabolized by this enzyme as normal Prodrugs are activated by this enzyme as normal
	IM	5%	Drugs are metabolized by this enzyme at a slow rate Prodrugs can barely be activated by this enzyme
X	PM	4%	Drugs are metabolized by this enzyme at a very slow rate Prodrugs are not activated by this enzyme

References

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Swen JJ et al. Pharmacogenetics: from bench to byte--an update of guidelines. Clin Pharmacol Ther. 2011 May,89(5):662-73.

TPMT - Thiopurine S-methyltransferase

Thiopurine-methyltransferase is an enzyme that catalyzes the transformation of thiopurine. Genetical variations can alter the activity or the breakdown of certain immunosuppressive and chemotherapeutic drugs.

RES	Genotype	POP	Possible results
X	EM	86%	Drugs are metabolized by this enzyme as normal Prodrugs are activated by this enzyme as normal
	IM	13%	Drugs are metabolized by this enzyme at a slow rate Prodrugs can barely be activated by this enzyme
	PM	1%	Drugs are metabolized by this enzyme at a very slow rate Prodrugs are not activated by this enzyme

References

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Relling MV et al. Clinical pharmacogenetics implementation consortium guidelines for thiopurine methyltransferase genotype and thiopurine dosing: 2013 update. Clin Pharmacol Ther. 2013 Apr,93(4):324-5.

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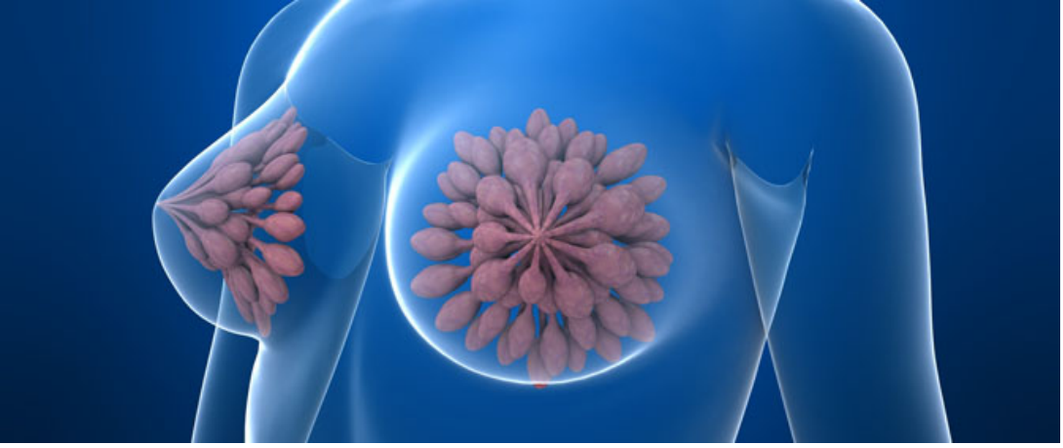
LEGEND: RES = your personal analysis result (marked with an X), GENOTYPE = different variations of the gene (called alleles),
POP = percent of the general population that have this genetic result,
POSSIBLE RESULTS = influence of the genetic variation.



Breast Health Sensor

FGFR2 - fibroblast growth factor receptor 2 (rs2981582)

The receptor protein FGFR2 (fibroblast growth factor receptor 2) is a member of the fibroblast growth factor receptor family, which, among other functions, plays an important role in angiogenesis, wound healing, embryonic development, and various endocrine signaling pathways. Mutations in the FGFR2 gene can affect both bone growth and the development of cancer. It has been repeatedly shown that the carriers of the T-allele have an increased risk of breast cancer.

RES	Genotype	POP	Possible results
	T/T	17%	Increased risk of breast cancer (OR: 1.63)
	C/T	48%	Increased risk of breast cancer (OR: 1.23)
X	C/C	36%	No increased risk of breast cancer

References

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Heterogeneity of breast cancer associations with five susceptibility loci by clinical and pathological characteristics. Garcia-Closas et al. PLoS Genet. 2008 Apr 25,4(4):e1000054.

Common Genetic Variants Associated with Breast Cancer and Mammographic Density Measures That Predict Disease. Cancer Res 2010,70:1449-1458. February 9, 2010.

VDR - vitamin D (1,25- dihydroxyvitamin D3) receptor (rs2228570)

The VDR gene encodes the vitamin D receptor, which is part of the steroid receptor family. It is a transcription factor that regulates the activity of specific target genes and thus affects the metabolism. The rs2228570 polymorphism is associated with an increased risk of breast cancer.

RES	Genotype	POP	Possible results
	T/T	13%	Increased risk of breast cancer (OR: 1.57)
X	C/T	41%	Increased risk of breast cancer (OR: 1.27)
	C/C	47%	No increased risk of breast cancer

References

Anderson et al. Vitamin D-related genetic variants, interactions with vitamin D exposure, and breast cancer risk among Caucasian women in Ontario. Cancer Epidemiol Biomarkers Prev. 2011 Aug,20(8):1708-17.

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8q24 (rs13281615)

The human chromosome segment 8q24 contains risk loci for various epithelial cancers, such as breast, prostate or colon cancer. A variety of studies have shown that the polymorphism of Rs13281615 increases the risk of breast cancer.

RES	Genotype	POP	Possible results
	A/A	27%	No increased risk of breast cancer
X	A/G	48%	No increased risk of breast cancer
	G/G	25%	Increased risk of breast cancer (OR: 1.38)

References

Garcia-Closas et al. Heterogeneity of breast cancer associations with five susceptibility loci by clinical and pathological characteristics. PLoS Genet. 2008 Apr 25

Mcinerney et al. Low penetrance breast cancer predisposition SNPs are site specific. Breast Cancer Res Treat. 2009 Sep,117(1):151-9.

Odefrey et al. Common Genetic Variants Associated with Breast Cancer and Mammographic Density Measures That Predict Disease. Cancer Res 2010,70:1449-1458.

TNRC9 - tenascin R (rs3803662)

The protein encoded by the TNCR9 gene (or TOX3) is a transcription factor that belongs to the family of HMG-box proteins. These proteins can bind DNA and alter the chromatin structure. The mutation of the TNCR9 gene (rs3803662) is one of the most important cancer-associated polymorphisms.

RES	Genotype	POP	Possible results
	T/T	22%	Increased risk of breast cancer (OR: 1.64)
	T/C	44%	Increased risk of breast cancer (OR: 1.23)
X	C/C	34%	No increased risk of breast cancer

References

Stacey et al. Common variants on chromosomes 2q35 and 16q12 confer susceptibility to estrogen receptor-positive breast cancer. Nat Genet. 2007 Jul,39(7):865-9.

Mcinerney et al. Low penetrance breast cancer predisposition SNPs are site specific. Breast Cancer Res Treat. 2009 Sep,117(1):151-9.

Garcia-Closas et al. Heterogeneity of breast cancer associations with five susceptibility loci by clinical and pathological characteristics. PLoS Genet. 2008 Apr 25

MAP3K1 - Mitogen-activated protein kinase kinase kinase 1, E3 ubiquitin protein ligase (rs889312)

The protein encoded by this gene is a serine/threonine kinase and is part of various signal transduction cascades. The polymorphism Rs889312 is located close to the MAP3K1 gene and a comprehensive genome-wide association study associated it with an increased breast cancer risk.

RES	Genotype	POP	Possible results
	A/A	39%	No increased risk of breast cancer
X	A/C	45%	Increased risk of breast cancer (OR: 1.13)
	C/C	16%	Increased risk of breast cancer (OR: 1.27)

References

Huijts et al. Clinical correlates of low-risk variants in FGFR2, TNRC9, MAP3K1, LSP1 and 8q24 in a Dutch cohort of incident breast cancer cases. Breast Cancer Research 2007, 9:R78

Couch et al. Association of Breast Cancer Susceptibility Variants with Risk of Pancreatic Cancer. Cancer Epidemiol Biomarkers Prev. 2009 November 18(11): 3044–3048.

Easton et al. Genome-wide association study identifies novel breast cancer susceptibility loci. Nature. 2007 June 28, 447(7148): 1087–1093.

LSP1 - Lymphocyte-specific protein 1 (rs3817198)

The protein LSP1 (Lymphocyte-specific protein 1) is expressed in lymphocytes, neutrophils, macrophages and endothelium, and it is involved in many regulatory processes. A genome-wide association study, analyzing 4000 breast cancer specimens, has shown that carriers of the rs3817198 polymorphism have an increased risk of breast cancer.

RES	Genotype	POP	Possible results
	T/T	63%	No increased risk of breast cancer
X	T/C	31%	Increased risk of breast cancer (OR: 1.06)
	C/C	6%	Increased risk of breast cancer (OR: 1.17)

References

Odefrey et al. Common Genetic Variants Associated with Breast Cancer and Mammographic Density Measures That Predict Disease. Cancer Res 2010,70:1449-1458.

Long et al. Evaluation of Breast Cancer Susceptibility Loci in Chinese Women. Cancer Epidemiol Biomarkers Prev. 2010 September 19(9): 2357–2365.

Easton et al. Genome-wide association study identifies novel breast cancer susceptibility loci. Nature. 2007 June 28, 447(7148): 1087–1093.

CASP8 - Caspase 8, apoptosis-related cysteine peptidase (rs1045485)

The caspases are a family of proteases and they are the most important enzymes in carrying out the cell death process (apoptosis). In addition to apoptosis, caspases are also involved in the development of the red blood cells and myoblasts. Mutations and the resulting defective caspases are involved in the development of tumours.

RES	Genotype	POP	Possible results
	C/C	0%	No increased risk of breast cancer
	C/G	10%	Increased risk of breast cancer (OR: 1.2)
X	G/G	90%	Increased risk of breast cancer (OR: 1.35)

References

Cox et al. A common coding variant in CASP8 is associated with breast cancer risk. Nat Genet. 2007 Mar,39(3):352-8. Epub 2007 Feb 11.

Shepard et al. A breast cancer risk haplotype in the caspase-8 gene. Cancer Res. 2009 April 1 69(7): 2724–2728.

Couch et al. Association of Breast Cancer Susceptibility Variants with Risk of Pancreatic Cancer. Cancer Epidemiol Biomarkers Prev. 2009 November 18(11): 3044–3048.

2q35 (rs13387042)

The polymorphism rs13387042 on the 2q35 region is associated with an increased risk of breast cancer.

RES	Genotype	POP	Possible results
X	A/A	29%	Increased risk of breast cancer (OR: 1.44)
	A/G	37%	Increased risk of breast cancer (OR: 1.22)
	G/G	34%	No increased risk of breast cancer

References

Reeves et al. Incidence of breast cancer and its subtypes in relation to individual and multiple low-penetrance genetic susceptibility loci. JAMA. 2010 Jul 28,304(4):426-34.

Stacey et al. Common variants on chromosomes 2q35 and 16q12 confer susceptibility to estrogen receptor-positive breast cancer. Nat Genet. 2007 Jul,39(7):865-9.

Odefrey et al. Common Genetic Variants Associated with Breast Cancer and Mammographic Density Measures That Predict Disease. Cancer Res 2010,70:1449-1458.

XRCC2 - X-ray repair complementing defective repair in Chinese hamster cells 2 (rs3218536)

The XRCC2 protein belongs to the RecA/Rad51-related protein family, and it is involved in the homologous recombination and repair of DNA. Studies have shown that the rs3218536 polymorphism has a protective effect against the development of breast cancer.

RES	Genotype	POP	Possible results
	A/A	90%	No increased risk of breast cancer
	G/A	9%	Increased risk of breast cancer (OR: 2.67)
X	G/G	1%	Increased risk of breast cancer (OR: 3.33)

References

Lin et al. A role for XRCC2 gene polymorphisms in breast cancer risk and survival. J Med Genet. Author manuscript, available in PMC Feb 24, 2014.

Silva et al. Breast cancer risk and common single nucleotide polymorphisms in homologous recombination DNA repair pathway genes XRCC2, XRCC3, NBS1 and RAD51. Cancer Epidemiol. 2010 Feb,34(1):85-92.

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CYP1A2 - cytochrome P450, family 1, subfamily A, polypeptide 2 (rs762551)

CYP1A2 (cytochrome P450 1A2) is a heme protein- enzyme involved in various metabolic processes. It metabolizes various xenobiotics such as caffeine, aflatoxin B1 and medications like paracetamol.

RES	Genotype	POP	Possible results
	A/A	41%	The consumption of 2 or more cups of coffee per day delays the appearance of breast cancer by approximately 7 years.
	A/C	43%	Coffee consumption does not delay the appearance of breast cancer
X	C/C	16%	Coffee consumption does not delay the appearance of breast cancer

References

Bågeman et al. Coffee consumption and CYP1A2*1F genotype modify age at breast cancer diagnosis and estrogen receptor status. Cancer Epidemiol Biomarkers Prev. 2008 Apr,17(4):895-901.

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.

Colon Health Sensor

CASC8 - Cancer susceptibility 8 (non-protein coding) (rs6983267)

Cancer susceptibility candidate 8 (CASC8) gene, a long non-coding RNA, is a gene desert region with no ability of protein coding. Recent evidence suggested that several CASC8 gene polymorphisms play important roles in various cancers.

RES	Genotype	POP	Possible results
X	G/G	41%	Increased risk of colon cancer (OR: 1.51)
	G/T	39%	Increased risk of colon cancer (OR: 1.20)
	T/T	20%	No increased risk of colon cancer

References

Montazeri Z et al. Systematic meta-analyses and field synopsis of genetic association studies in colorectal adenomas. Int J Epidemiol. 2016 Feb,45(1):186-205.

Poynter JN et al. Variants on 9p24 and 8q24 are associated with risk of colorectal cancer: results from the Colon Cancer Family Registry. Cancer Res. 2007 Dec 1,67(23):11128-32.

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Li M et al. Genetic variants on chromosome 8q24 and colorectal neoplasia risk: a case-control study in China and a meta-analysis of the published literature. PLoS One. 2011 Mar 24,6(3):e18251.

Yao K et al. Correlation Between CASC8, SMAD7 Polymorphisms and the Susceptibility to Colorectal Cancer: An Updated Meta-Analysis Based on GWAS Results. Medicine (Baltimore). 2015 Nov,94(46):e1884.

Hutter CM et al. Characterization of the association between 8q24 and colon cancer: gene-environment exploration and meta-analysis. BMC Cancer. 2010 Dec 4,10:670.

Li L et al. Association of 8q23-24 region (8q23.3 loci and 8q24.21 loci) with susceptibility to colorectal cancer: a systematic and updated meta-analysis. Int J Clin Exp Med. 2015 Nov 15,8(11):21001-13. eCollection 2015.

CASC8 - Cancer susceptibility 8 (non-protein coding) (rs10505477)

Cancer susceptibility candidate 8 (CASC8) gene, a long non-coding RNA, is a gene desert region with no ability of protein coding. Recent evidence suggested that several CASC8 gene polymorphisms play important roles in various cancers.

RES	Genotype	POP	Possible results
X	C/C	21%	No increased risk of colon cancer
	C/T	42%	Increased risk of colon cancer (OR: 1.13)
	T/T	37%	Increased risk of colon cancer (OR: 1.28)

References

Schafmayer C et al. Investigation of the colorectal cancer susceptibility region on chromosome 8q24.21 in a large German case-control sample. Int J Cancer. 2009 Jan 1,124(1):75-80.

Real LM et al. A colorectal cancer susceptibility new variant at 4q26 in the Spanish population identified by genome-wide association analysis. PLoS One. 2014 Jun 30,9(6):e101178.

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Tan C et al. Risk of eighteen genome-wide association study-identified genetic variants for colorectal cancer and colorectal adenoma in Han Chinese. Oncotarget. 2016 Nov 22,7(47):77651-77663.

CASC8 - Cancer susceptibility 8 (non-protein coding) (rs10808555)

Cancer susceptibility candidate 8 (CASC8) gene, a long non-coding RNA, is a gene desert region with no ability of protein coding. Recent evidence suggested that several CASC8 gene polymorphisms play important roles in various cancers.

RES	Genotype	POP	Possible results
	G/G	11%	Increased risk of colon cancer (OR: 1.28)
	G/A	45%	Increased risk of colon cancer (OR: 1.13)
X	A/A	44%	No increased risk of colon cancer

References

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Tan C et al. Risk of eighteen genome-wide association study-identified genetic variants for colorectal cancer and colorectal adenoma in Han Chinese. Oncotarget. 2016 Nov 22,7(47):77651-77663.

Yang B et al. Genetic variants at chromosome 8q24, colorectal epithelial cell proliferation, and risk for incident, sporadic colorectal adenomas. Mol Carcinog. 2014 Feb,53 Suppl 1:E187-92.

CASC8 - Cancer susceptibility 8 (non-protein coding) (rs7837328)

Cancer susceptibility candidate 8 (CASC8) gene, a long non-coding RNA, is a gene desert region with no ability of protein coding. Recent evidence suggested that several CASC8 gene polymorphisms play important roles in various cancers.

RES	Genotype	POP	Possible results
	A/A	23%	Increased risk of colon cancer (OR: 1.37)
	A/G	45%	Increased risk of colon cancer (OR: 1.17)
X	G/G	32%	No increased risk of colon cancer

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CASC8 - Cancer susceptibility 8 (non-protein coding) (rs7014346)

Cancer susceptibility candidate 8 (CASC8) gene, a long non-coding RNA, is a gene desert region with no ability of protein coding. Recent evidence suggested that several CASC8 gene polymorphisms play important roles in various cancers.

RES	Genotype	POP	Possible results
X	G/G	44%	No increased risk of colon cancer
	G/A	45%	Increased risk of colon cancer (OR: 1.12)
	A/A	11%	Increased risk of colon cancer (OR: 1.25)

References

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CCND1 - Cyclin D1 (rs9344)

Cyclin D1, encoded by the CCND1 gene located on 11q13, plays an important role in the progression of the cell cycle. Cyclins function as regulators of CDK kinases and is required for progression through the G1 phase.

RES	Genotype	POP	Possible results
X	G/G	37%	No increased risk of colon cancer
	G/A	43%	Increased risk of colon cancer (OR: 1.13)
	A/A	20%	Increased risk of colon cancer (OR: 1.17)

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CDH1 - Cadherin 1 (rs16260)

Cadherin-1, a tumour suppressor gene, provides the genetic code for making a protein called epithelial cadherin. E-cadherin is one of the most important molecules in cell-cell adhesion in epithelial tissues.

RES	Genotype	POP	Possible results
	C/C	58%	No protection against colon cancer
	C/A	36%	Protection against colon cancer (OR: 0.92)
X	A/A	6%	Protection against colon cancer (OR: 0.92)

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COLCA - Colorectal cancer associated (rs3802842)

Colorectal cancer associated protein 1 localizes in granular structures and has been associated with colorectal cancer.

RES	Genotype	POP	Possible results
X	A/A	52%	No increased risk of colon cancer
	A/C	39%	Increased risk of colon cancer (OR: 1.15)
	C/C	9%	Increased risk of colon cancer (OR: 1.32)

References

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CYP1A1 - Cytochrome P450 family 1 subfamily A member 1 (rs1048943)

The haeme protein cytochrome P450-1A2 (CYP1A2) belongs to the group of cytochrome P450 enzymes and metabolizes various xenobiotic substances (including caffeine), medications and oestrogen.

RES	Genotype	POP	Possible results
X	A/A	77%	No increased risk of colon cancer
	A/G	19%	Increased risk of colon cancer (OR: 1.26)
	G/G	4%	Increased risk of colon cancer (OR: 1.54)

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DNMT3B - DNA methyltransferase 3 beta (rs1569686)

The DNA methyltransferase 3-beta protein belongs to the group of DNA methyltransferases that can transfer methyl groups to nucleic bases of DNA. The DNA methyltransferase 3-beta protein is able to methylate cytosine de novo, which is particularly important in early embryonic development.

RES	Genotype	POP	Possible results
	G/G	13%	Protection against colon cancer (OR: 0.84)
X	G/T	30%	Protection against colon cancer (OR: 0.84)
	T/T	57%	No protection against colon cancer

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GREM1 - Gremlin 1, DAN family BMP antagonist (rs10318)

Gremlin1 is a protein that inhibits the TGF-beta signaling pathway. It plays a role in the regulation of organogenesis, body patterns and tissue differentiation.

RES	Genotype	POP	Possible results
X	C/C	63%	No increased risk of colon cancer
	C/T	28%	Increased risk of colon cancer (OR: 1.13)
	T/T	9%	Increased risk of colon cancer (OR: 1.28)

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IL8 - Interleukin 8 (rs4073)

CXCL8 (interleukin-8) is a member of the chemokine family and produced by macrophages and other cell types. It acts on chemokine receptors CXCR1 and CXCR2, and is an important mediator in the immune reaction of the innate immune system response.

RES	Genotype	POP	Possible results
	A/A	31%	Increased risk of colon cancer (OR: 1.21)
X	A/T	42%	Increased risk of colon cancer (OR: 1.21)
	T/T	27%	No increased risk of colon cancer

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IL10 - Interleukin 10 (rs1800872)

Interleukin-10 is a cytokine with multiple effects in immunoregulation and inflammation. Studies suggested the function of this cytokine as an immunoregulator in the intestinal tract.

RES	Genotype	POP	Possible results
	A/A	21%	Increased risk of colon cancer (OR: 1.25)
X	A/C	44%	Increased risk of colon cancer (OR: 1.25)
	C/C	35%	No increased risk of colon cancer

References

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MTRR - 5-methyltetrahydrofolate-homocysteine methyltransferase reductase (rs1801394)

Methionine is an essential, sulphur-containing proteinogenic amino acid. The synthesis of methionine is catalyzed by the methionine synthase enzyme, which in turn requires homocysteine. The protein encoded by the MTRR gene (methionine synthase reductase) regenerates the inactive methionine synthase through methylation.

RES	Genotype	POP	Possible results
	A/A	43%	No increased risk of colon cancer
	A/G	41%	Increased risk of colon cancer (OR: 1.11)
X	G/G	16%	Increased risk of colon cancer (OR: 1.23)

References

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SMAD7 - SMAD family member 7 (rs12953717)

SMAD Family Member 7 is an antagonist of the transforming growth factor β (TGF-β) signaling and plays an important role in modulating a large array of biological processes. Dysregulation of Smad7 is associated with a variety of human diseases.

RES	Genotype	POP	Possible results
X	C/C	50%	No increased risk of colon cancer
	C/T	39%	Increased risk of colon cancer (OR: 1.16)
	T/T	11%	Increased risk of colon cancer (OR: 1.35)

References

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MTHFR - Methylenetetrahydrofolate reductase (NAD(P)H) (rs1801133)

Methylenetetrahydrofolate reductase (MTHFR) is involved in many metabolic pathways in the human body. It is responsible for the degradation of homocysteine to methionine in homocysteine metabolism.

RES	Genotype	POP	Possible results
X	C/C	59%	No protection against colon cancer
	C/T	33%	No protection against colon cancer
	T/T	8%	Protection against colon cancer (OR: 0.93)

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TGFB1 - Transforming growth factor beta 1 (rs1800469)

Transforming growth factor beta 1 is a cytokine which is involved in many cellular functions, including the control of cell growth, proliferation, differentiation, apoptosis, and plays an important role in controlling the immune system.

RES	Genotype	POP	Possible results
	C/C	42%	Increased risk of colon cancer (OR: 1.36)
	C/T	43%	Increased risk of colon cancer (OR: 1.18)
X	T/T	15%	No increased risk of colon cancer

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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.

Skin Health Sensor

CDK10 - Cyclin dependent kinase 10 (rs258322)

The protein encoded by this gene belongs to the CDK subfamily of the Ser/Thr protein kinase family. This kinase plays a role in cell cycle regulation. Multiple transcript variants encoding different isoforms have been found for this gene. At least three alternatively spliced transcript variants encoding different isoforms have been reported, two of which contain multiple non-AUG translation initiation sites.

RES	Genotype	POP	Possible results
X	C/C	62%	No increased risk of melanoma
	C/T	28%	Increased risk of melanoma (OR: 1.64)
	T/T	10%	Increased risk of melanoma (OR: 2.69)

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MTAP - Methylthioadenosine phosphorylase (rs7023329)

Methylthioadenosine Phosphorylase is an enzyme that plays a major role in polyamine biosynthesis and the methionine salvage pathway. The enzyme is deficient in many forms of cancer because the gene and the tumour suppressor p16 gene are co-deleted.

RES	Genotype	POP	Possible results
	A/A	33%	No protection against melanoma
X	A/G	45%	Protection against melanoma (OR: 0.83)
	G/G	22%	Protection against melanoma (OR: 0.69)

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MYH7B - Myosin heavy chain 7B (rs1885120)

MYH7B is belongs to the motor domain superfamily. It catalyzes the ATP hydrolysis and interacts with actin. Experiments have shown that MYH7B is not expressed in a large number of melanoma cell lines.

RES	Genotype	POP	Possible results
	G/G	98%	No increased risk of melanoma No increased risk of non-melanoma skin cancer
X	C/G	1%	Increased risk of melanoma (OR: 1.55) Increased risk of non-melanoma skin cancer (OR: 1.46)
	C/C	1%	Increased risk of melanoma (OR: 2.40) Increased risk of non-melanoma skin cancer (OR: 2.13)

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Bishop DT et al. Genome-wide association study identifies three loci associated with melanoma risk. Nat Genet. 2009 Aug,41(8):920-5.

NCOA6 - Nuclear receptor coactivator 6 (rs4911442)

Nuclear receptor coactivator 6 is a multifunctional transcription coregulator that can enhance transactivation by other transcription factors and is involved in cell survival, growth and development. The gene is amplified in several human cancers including breast cancer, colon and lung cancers.

RES	Genotype	POP	Possible results
X	A/A	93%	No increased risk of melanoma
	A/G	6%	Increased risk of melanoma (OR: 1.76)
	G/G	1%	Increased risk of melanoma (OR: 2.73)

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Maccioni L et al. Variants at chromosome 20 (ASIP locus) and melanoma risk. Int J Cancer. 2013 Jan 1,132(1):42-54.

PARP1 - Poly(ADP-ribose) polymerase 1 (rs3219090)

PARP1 (Poly(ADP-Ribose) Polymerase 1) is an enzyme which modifies various nuclear proteins by poly(ADP-ribosyl)ation. The protein is involved in the regulation of various cellular processes such as differentiation, proliferation and tumour transformation.

RES	Genotype	POP	Possible results
	A/A	30%	Protection against melanoma (OR: 0.74)
	A/G	43%	Protection against melanoma (OR: 0.86)
X	G/G	27%	No protection against melanoma

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PIGU - Phosphatidylinositol glycan anchor biosynthesis class U (rs910873)

PIGU is an integral membrane protein that plays a role in cell division control.

RES	Genotype	POP	Possible results
	G/G	97%	No increased risk of melanoma No increased risk of non-melanoma skin cancer
	G/A	2%	Increased risk of melanoma (OR: 1.81) Increased risk of non-melanoma skin cancer (OR: 1.35)
X	A/A	1%	Increased risk of melanoma (OR: 1.81) Increased risk of non-melanoma skin cancer (OR: 1.82)

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SLC45A2 - Solute carrier family 45 member 2 (rs16891982)

The SLC45A2 gene (also called MATP) provides instructions for making a protein that mediates melanin synthesis and has been found to play a role in pigmentation. It is also a melanocyte differentiation antigen that is expressed in a high percentage of melanoma cell lines.

RES	Genotype	POP	Possible results
X	G/G	22%	Protection against melanoma (OR: 0.18)
	G/C	11%	Protection against melanoma (OR: 0.42)
	C/C	67%	No protection against melanoma

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CLPTM1L - Cleft lip and palate transmembrane protein 1-like (rs401681)

CLPTM1L is a membrane protein associated with cisplatin-induced apoptosis. Polymorphisms in this gene have been associated with increased susceptibility to several cancers.

RES	Genotype	POP	Possible results
	C/C	36%	Protection against melanoma (OR: 0.74) Increased risk of non-melanoma skin cancer (OR: 1.28)
X	C/T	45%	Protection against melanoma (OR: 0.86) Increased risk of non-melanoma skin cancer (OR: 1.13)
	T/T	19%	No protection against melanoma No increased risk of non-melanoma skin cancer

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TYR - Tyrosinase (rs1393350)

Tyrosinase catalyzes several steps in the conversion of tyrosine to melanin and other pigments. Polymorphisms in this gene result in skin pigmentation variation.

RES	Genotype	POP	Possible results
	G/G	85%	No increased risk of melanoma
X	G/A	14%	Increased risk of melanoma (OR: 1.21)
	A/A	1%	SP - Increased risk of melanoma (OR: 1.80)

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MC1R - Melanocortin 1 receptor

The MC1R gene provides instructions for making a protein called the melanocortin 1 receptor. The G protein–coupled receptor is located on the surface of melanocytes which produce the pigment melanin through the process of melanogenesis. The MC1R score can be calculated based on the classification of MC1R variants, as implemented by Davies et al.

RES	Genotype	POP	Possible results
X	Score 0	31%	No increased risk of melanoma No increased risk of non-melanoma skin cancer
	Score 1	25%	Increased risk of melanoma (OR: 1.24) Increased risk of non-melanoma skin cancer (OR: 1.41)
	Score 2	27%	Increased risk of melanoma (OR: 1.69) Increased risk of non-melanoma skin cancer (OR: 1.81)
	Score 3	11%	Increased risk of melanoma (OR: 3.28) Increased risk of non-melanoma skin cancer (OR: 2.68)
	Score 4	5%	Increased risk of melanoma (OR: 3.12) Increased risk of non-melanoma skin cancer (OR: 2.68)

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ASIP - Agouti signaling protein (rs4911414/rs1015362)

The agouti-signaling protein is encoded by the agouti gene and influences the distribution of black and yellow pigments in the skin and hair. A haplotype (rs4911414 T and rs1015362 G) in the proximity of ASIP is not only associated with pigmentation features, but is also involved in the development of skin cancer.

RES	Haplotype	POP	Possible results
X	-	99%	No increased risk of melanoma No increased risk of non-melanoma skin cancer
	GG_TT	1%	Increased risk of melanoma (OR: 1.5) Increased risk of non-melanoma skin cancer (OR: 1.45)

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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.

Lung Health Sensor

CYP1A1 (Cytochrome P450 1A1) rs4646903

The haeme protein cytochrome P450-1A1 (CYP1A1) belongs to the group of phase I enzymes, and mediates the metabolism of environmental toxins and various xenobiotic substances. Defects in this gene can alter the enzymatic activity.

RES	Genotype	POP	Possible results
X	T/T	52%	No increased risk of lung cancer
	C/T	37%	Increased risk of lung cancer (OR: 1.3)
	C/C	11%	Increased risk of lung cancer (OR: 1.27)

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GSTM1 - glutathione s-transferase mu1 (null allele)

The glutathione s-transferases are found in the liver and in lymphocytes. They are involved in the detoxification of endogenous and exogenous substances. A defective GSTM1 gene reduces the enzymatic activity of the protein, which leads to a limited cellular detoxification.

RES	Genotype	POP	Possible results
X	INS	56%	No increased risk of lung cancer
	DEL	44%	Increased risk of lung cancer (OR: 1.26)

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GSTT1 - glutathione s-transferase theta 1 (null allele)

The glutathione s-transferases are found in the liver and in lymphocytes. They are involved in the detoxification of endogenous and exogenous substances. A defective GSTM1 gene reduces the enzymatic activity of the protein, which leads to a limited cellular detoxification.

RES	Genotype	POP	Possible results
	INS	74%	No increased risk of lung cancer
X	DEL	26%	Increased risk of lung cancer (OR: 2.4)

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GSTP1 - glutathione s-transferase pi 1 (rs1695)

The glutathione s-transferases are found in the liver and in lymphocytes. They are involved in the detoxification of endogenous and exogenous substances. The GSTP1 enzymes are involved in the metabolism of endogenous metabolites, and protect the cells against oxidative stress- similar to GSTM1 and GSTT1.

RES	Genotype	POP	Possible results
	A/A	43%	No increased risk of lung cancer
X	A/G	43%	Increased risk of lung cancer (OR: 1.38)
	C/C	14%	Increased risk of lung cancer (OR: 3.21)

References

Liu HX et al. Correlation between gene polymorphisms of CYP1A1, GSTP1, ERCC2, XRCC1, and XRCC3 and susceptibility to lung cancer. Genet Mol Res. 2016 Nov 3,15(4).

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CYP1A1 (cytochrome P450 1A1) rs1048943

The haeme protein cytochrome P450-1A1 (CYP1A1) belongs to the group of phase I enzymes, and mediates the metabolism of environmental toxins and various xenobiotic substances. Defects in this gene can alter the enzymatic activity.

RES	Genotype	POP	Possible results
X	A/A	77%	No increased risk of lung cancer
	A/G	19%	Increased risk of lung cancer (OR: 1.22)
	G/G	4%	Increased risk of lung cancer (OR: 3.06)

References

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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.

Cardiovascular Sensor

CDH13 - Cadherin 13 (rs8055236)

The CDH13 gene encodes a protein of the cadherin superfamily. The protein is localized on the cell membrane, and it is expressed, inter alia, in the heart, aortic wall, neurons and in the spinal cord. The polymorphism rs8055236 is associated with an increased risk of heart diseases.

RES	Genotype	POP	Possible results
	T/T	11%	No increased risk of disease
X	T/G	31%	Increased risk of coronary heart disease (OR: 1.91)
	G/G	59%	Increased risk of coronary heart disease (OR: 2.23)

References

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CHDS8 - Coronary heart disease, susceptibility to, 8 (rs1333049)

The polymorphism rs1333049 on gene CHDS8 (Coronary heart disease, susceptibility to, 8) has been repeatedly associated with an increased risk of heart diseases.

RES	Genotype	POP	Possible results
X	G/G	36%	No increased risk of disease
	G/C	45%	Increased risk of coronary heart disease (OR: 1.47)
	C/C	19%	Increased risk of coronary heart disease (OR: 1.9)

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APOA5 - Apolipoprotein A-V (rs662799)

The protein encoded by this gene is an apolipoprotein and an important determinant of plasma triglyceride levels, a major risk factor for coronary artery disease. It is a component of several lipoprotein fractions including VLDL, HDL and chylomicrons. It is believed that apoA-V affects lipoprotein metabolism by interacting with LDL-R gene family receptors. Studies have shown that carriers of the G-allele experience low weight gain when eating a fatty diet.

RES	Genotype	POP	Possible results
X	A/A	71%	No increased risk of disease
	A/G	26%	Increased risk of coronary heart disease (OR: 1.98)/atherosclerosis/heart attack Predisposition to low HDL cholesterol (the good cholesterol) Predisposition to elevated triglyceride levels
	G/G	3%	Increased risk of coronary heart disease (OR: 1.98)/atherosclerosis/heart attack Predisposition to low HDL cholesterol (the good cholesterol) Predisposition to elevated triglyceride levels

References

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PON1 - Paraoxonase 1 (rs662)

Paraoxonase (PON1) is an antioxidant enzyme involved in radical elimination and lipometabolism. The polymorphisms rs854560 and rs662 lead to a reduced catalytic activity and an increased risk of cardiovascular diseases.

RES	Genotype	POP	Possible results
X	A/A	24%	No increased risk of disease
	G/A	43%	Increased risk of coronary heart disease/atherosclerosis/heart attack (OR: 2.3)
	G/G	33%	Increased risk of coronary heart disease/atherosclerosis/heart attack (OR: 3.2)

References

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PON1 - Paraoxonase 1 (rs854560)

Paraoxonase (PON1) is an antioxidant enzyme involved in radical elimination and lipometabolism. The polymorphisms rs854560 and rs662 lead to a reduced catalytic activity and an increased risk of cardiovascular diseases.

RES	Genotype	POP	Possible results
	A/A	68%	Increased risk of coronary heart disease/atherosclerosis/heart attack (OR: 2.25)
	A/T	27%	No increased risk of disease
X	T/T	5%	No increased risk of disease

References

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Oliveira SA et al. PON1 M/L55 mutation protects high-risk patients against coronary artery disease. Int J Cardiol. 2004 Mar,94(1):73-7.

APOB R3500Q - Apolipoprotein B (rs5742904)

Apolipoprotein B (ApoB) is the major protein component of the LDL proteins (low density lipoprotein), which are responsible for the transport of cholesterol in the blood. As such, ApoB regulates the LDL concentration in the individual. Rs5742904 polymorphism leads to an increased LDL cholesterol levels.

RES	Genotype	POP	Possible results
X	G/G	98%	No increased risk of disease
	A/G	1%	Significantly increased risk of coronary heart disease/atherosclerosis/heart attack Significantly increased risk of elevated LDL cholesterol levels (familial Hypercholesterolaemia)
	A/A	1%	Significantly increased risk of coronary heart disease/atherosclerosis/heart attack Significantly increased risk of elevated LDL cholesterol levels (familial Hypercholesterolaemia)

References

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Castillo et al. The apolipoprotein B R3500Q gene mutation in Spanish subjects with a clinical diagnosis of familial hypercholesterolemia. Atherosclerosis. 2002 Nov,165(1):127-35.

NOS3 - Nitric oxide synthase 3 (endothelial cell) (Ins/Del Int. 4)

NO-synthases (NOS) are oxidases which catalyze the reaction of arginine to citrulline and nitric oxide. NOS3 is an endothelial nitric oxide synthase, predominantly expressed in endothelial cells on the inside of the blood vessels, where it indirectly adjusts the blood pressure and the afterload of the heart. Several polymorphisms in the NOS3 gene are associated with an increased risk of cardiovascular diseases.

RES	Genotype	POP	Possible results
X	Ins/Ins	94%	No increased risk of disease
	Ins/Del	6%	No increased risk of disease
	Del/Del	0%	Increased risk of coronary heart disease/atherosclerosis/heart attack (OR: 1.34)

References

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Salimi S et al. Endothelial nitric oxide synthase gene intron4 VNTR polymorphism in patients with coronary artery disease in Iran. Indian J Med Res. 2006 Dec,124(6):683-8.

NOS3 - Nitric oxide synthase 3 (endothelial cell) (rs2070744)

RES	Genotype	POP	Possible results
X	T/T	60%	No increased risk of disease
	C/T	34%	No increased risk of disease
	C/C	7%	Increased risk of coronary heart disease/atherosclerosis/heart attack

References

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NOS3 - Nitric oxide synthase 3 (endothelial cell) (rs1799983)

RES	Genotype	POP	Possible results
	G/G	69%	No increased risk of disease
X	G/T	26%	Increased risk of coronary heart disease/atherosclerosis/heart attack (OR: 1.52)
	T/T	5%	Increased risk of coronary heart disease/atherosclerosis/heart attack (OR: 2.31)

References

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APOA1 - Apolipoprotein A-I (rs670)

Apolipoprotein A1 (ApoA1) is the major protein component of HDL (high density lipoprotein) particles in the blood. These are responsible for the transport of excess cholesterol to the liver, where it is further converted and eliminated. The polymorphism rs670 influences both the impact of polyunsaturated fatty acids on HDL cholesterol levels, as well as the risk of heart disease.

RES	Genotype	POP	Possible results
X	G/G	66%	No increased risk of disease Polyunsaturated fatty acids (such as omega-3) WORSEN the HDL cholesterol levels
	A/G	31%	Increased risk of coronary heart disease (OR: 1.47)/atherosclerosis/heart attack Polyunsaturated fatty acids (such as omega-3) improve the HDL cholesterol levels
	A/A	3%	Increased risk of coronary heart disease (OR: 1.9)/atherosclerosis/heart attack Polyunsaturated fatty acids (such as omega-3) improve the HDL cholesterol levels

References

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Juo SH et al. Mild association between the A/G polymorphism in the promoter of the apolipoprotein A-I gene and apolipoprotein A-I levels: a meta-analysis. Am J Med Genet. 1999 Jan 29,82(3):235-41.

MTRR - 5-methyltetrahydrofolate-homocysteine methyltransferase reductase (rs1801394)

Methionine is a essential, sulfur containing proteinogenic amino acid. The synthesis of methionine is catalyzed by the methionine synthase enzyme, which in its turn requires homocysteine. The protein encoded by the MTRR gene (methionine synthase reductase) regenerates the inactive methionine synthase through methylation.

RES	Genotype	POP	Possible results
	A/A	43%	No increased risk of disease
	A/G	41%	Increased risk of coronary heart disease atherosclerosis/heart attack Predisposition to elevated homocysteine values
X	G/G	16%	Increased risk of coronary heart disease atherosclerosis/heart attack Predisposition to elevated homocysteine values

References

Cai et al. Genetic variant in MTRR, but not MTR, is associated with risk of congenital heart disease: an integrated meta-analysis. PLoS One. 2014 Mar 4,9(3):e89609.

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Zeng W et al. A66G and C524T polymorphisms of the methionine synthase reductase gene are associated with congenital heart defects in the Chinese Han population. Genet Mol Res. 2011 Oct 25,10(4):2597-605.

Yu D et al. Association between methionine synthase reductase A66G polymorphism and the risk of congenital heart defects: evidence from eight case-control studies. Pediatr Cardiol. 2014 Oct,35(7):1091-8.

GJA4 - Gap junction protein, alpha 4, 37kDa (rs1764391)

The GJA4 gene (Gap junction alpha-4 protein) belongs to the connexin gene family. These transmembrane proteins are components of the intercellular channels (the so-called gap junctions), which link the adjacent cells with each other, and facilitate the exchange of ions and small molecules. Gap junctions are mainly found in the heart muscle, in epithelial cells and in the retina.

RES	Genotype	POP	Possible results
X	T/T	14%	No increased risk of disease
	C/T	39%	Increased risk of coronary heart disease (OR: 2.03)
	C/C	47%	Increased risk of coronary heart disease (OR: 2.03)

References

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Wen D et al. Association of Connexin37 C1019T with myocardial infarction and coronary artery disease: a meta-analysis. Exp Gerontol. 2014 Oct,58:203-7.

ITGB3 - Integrin beta 3 (platelet glycoprotein IIIa, antigen CD61) (rs5918)

The integrin beta 3 (ITGB3), or CD61, is a transmembrane protein involved in the signal transmission between cells and the extracellular matrix. It has been proven that carriers of the C-allele (rs5918) have an increased risk of cardiovascular diseases. In addition, the polymorphism influences the blood-thinning effect of the aspirin drug.

RES	Genotype	POP	Possible results
X	T/T	84%	No increased risk of disease Aspirin protects against arterial thrombosis
	T/C	15%	Increased risk of coronary heart disease (OR: 2.8) Aspirin does not provide protection from thrombosis
	C/C	1%	Increased risk of coronary heart disease (OR: 7.84) Aspirin does not provide any protection from thrombosis

References

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Goodman T et al. Pharmacogenetics of aspirin resistance: a comprehensive systematic review. Br J Clin Pharmacol. 2008 Aug,66(2):222-32.

CETP - Cholesteryl ester transfer protein, plasma (rs708272)

The cholesterol ester transfer protein (CETP) is a pore-forming protein involved in lipoprotein metabolism. It is mainly expressed in the liver, and ensures the transfer of cholesterol esters from HDL to LDL or VLDL, in exchange for triglycerides. The polymorphism rs708272 influences the regulation of HDL cholesterol levels.

RES	Genotype	POP	Possible results
	T/T	40%	No predisposition to bad HDL cholesterol values (the good cholesterol)
X	C/T	45%	Predisposition to bad HDL cholesterol values (the good cholesterol)
	C/C	15%	Predisposition to bad HDL cholesterol values (the good cholesterol)

References

Radovica et al. The association of common SNPs and haplotypes in CETP gene with HDL cholesterol levels in Latvian population. PLoS One. 2013 May 13,8(5):e64191.

Agirbasli et al. Multi-locus candidate gene analyses of lipid levels in a pediatric Turkish cohort: lessons learned on LPL, CETP, LIPC, ABCA1, and SHBG. OMICS. 2013 Dec,17(12):636-45.

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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
	T/T	8%	Predisposition to elevated homocysteine values Vitamin B2 lowers the homocysteine levels
	C/T	33%	Predisposition to elevated homocysteine values Vitamin B2 does not lower the homocysteine levels
X	C/C	59%	No predisposition to elevated homocysteine values Vitamin B2 does not lower the homocysteine levels

References

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Hustad et al. Riboflavin and Methylenetetrahydrofolate Reductase. Madame Curie Bioscience Database.

MMP3 - Matrix metallopeptidase 3 (stromelysin 1, progelatinase) (rs3025058)

The matrix metalloproteinase-3 (MMP3) or Stromelysin 1, is a zinc-dependent endopeptidase involved in the degradation of extracellular matrix components. It plays an important role in the remodeling of tissues, wound healing and inflammatory processes. The polymorphism (rs3025058) influences the risk of heart diseases.

RES	Genotype	POP	Possible results
	T/T	26%	No increased risk of disease
X	T/Del	49%	Increased risk of coronary heart disease (OR: 1.26)
	Del/Del	25%	Increased risk of coronary heart disease (OR: 1.59)

References

Abilleira et al. The role of genetic variants of matrix metalloproteinases in coronary and carotid atherosclerosis. J Med Genet. 2006 Dec,43(12):897-901. Epub 2006 Aug 11.

Zee et al. Genetic risk factors in recurrent venous thromboembolism: A multilocus, population-based, prospective approach. Clin Chim Acta. 2009 Apr,402(1-2):189-92.

Wang J et al. Polymorphisms of matrix metalloproteinases in myocardial infarction: a meta-analysis. Heart. 2011 Oct,97(19):1542-6. doi: 10.1136/heartjnl-2011-300342.

NOS1AP - Nitric oxide synthase 1 (neuronal) adaptor protein (rs16847548)

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. NOS1AP polymorphisms are associated with a prolonged QT interval, and an increased risk of sudden cardiac death.

RES	Genotype	POP	Possible results
X	T/T	53%	No increased risk of sudden cardiac death
	T/C	38%	Increased risk of sudden cardiac death (OR: 1.3)
	C/C	9%	Increased risk of sudden cardiac death (OR: 2.6)

References

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Crotti et al.NOS1AP is a genetic modifier of the long-QT syndrome. Circulation. 2009 Oct 27,120(17):1657-63.

Kao et al. Genetic variations in nitric oxide synthase 1 adaptor protein are associated with sudden cardiac death in US white community-based populations. Circulation. 2009 Feb 24,119(7):940-51.

NOS1AP - Nitric oxide synthase 1 (neuronal) adaptor protein (rs12567209)

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. NOS1AP polymorphisms are associated with a prolonged QT interval, and an increased risk of sudden cardiac death.

RES	Genotype	POP	Possible results
X	G/G	74%	No increased risk of sudden cardiac death
	A/G	23%	Protection against sudden cardiac death (OR: 0.51)
	A/A	3%	Increased risk of sudden cardiac death (OR: 1.31)

References

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Eijgelsheim et al. Genetic variation in NOS1AP is associated with sudden cardiac death: evidence from the Rotterdam Study. Hum Mol Genet. Nov 1, 2009, 18(21): 4213–4218.

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. NOS1AP polymorphisms are associated with a prolonged QT interval, and an increased risk of sudden cardiac death.

RES	Genotype	POP	Possible results
	G/G	37%	Predisposition to increased QT-interval duration (+ 4 to 7,9 ms)
	G/T	43%	Predisposition to increased QT-interval duration (+ 1,7 to 4,6 ms)
X	T/T	20%	No predisposition to increased QT-interval duration

References

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Marjamaa et al. Common candidate gene variants are associated with QT interval duration in the general population. J Intern Med. 2009 Apr,265(4):448-58.

SREBF2 - Sterol regulatory element binding transcription factor 2 (rs2228314)

SREBF2 or SREBP2 (sterol regulatory element-binding protein 2) is a transcription factor involved in the regulation of cholesterol metabolism. The cholesterol concentration is kept in balance through the control of the transcriptional activity of various target genes.

RES	Genotype	POP	Possible results
	G/G	39%	Predisposition to elevated LDL cholesterol levels
	G/C	40%	No predisposition to elevated LDL cholesterol levels
X	C/C	20%	No predisposition to elevated LDL cholesterol levels

References

Fan et al. Expression of sterol regulatory element-binding transcription factor (SREBF) 2 and SREBF cleavage-activating protein (SCAP) in human atheroma and the association of their allelic variants with sudden cardiac death. Published online Dec 30, 2008.

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Mohammad Abdullah et al. The impact of dairy consumption on circulating cholesterol levels is modulated by common single nucleotide polymorphisms in cholesterol synthesis- and transport-related genes. Fasebj, Published Online: 1 Apr 2014 Abstract Number: 1038.4

CYP1A2 - cytochrome P450, family 1, subfamily A, polypeptide 2 (rs762551)

The haeme protein cytochrome P450-1A2 (CYP1A2) belongs to the group of cytochrome P450 enzymes, and metabolizes various xenobiotic substances (including caffeine), medications and oestrogens. The polymorphism rs762551 is associated with the risk of breast cancer.

RES	Genotype	POP	Possible results
X	A/A	41%	Caffeine is broken down normally
	A/C	44%	Caffeine is broken down slowly
	C/C	15%	Caffeine is broken down slowly

References

Bågeman et al. Coffee consumption and CYP1A2*1F genotype modify age at breast cancer diagnosis and estrogen receptor status. Cancer Epidemiol Biomarkers Prev. 2008 Apr,17(4):895-901.

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APOE - apolipoprotein E (E2/E3/E4)

APoE (apolipoprotein E) metabolizes triglyceride-rich lipoprotein constituents, and plays a central role in the lipid metabolism. The ApoE gene is present in three common types, which are called allele E2, E3 and E4. The E4 allele is associated with an increased risk of heart disease and Alzheimer.

RES	Genotype	POP	Possible results
	E2/E2	1%	No increased risk of coronary heart disease/atherosclerosis/heart attack No predisposition to elevated LDL cholesterol levels Predisposition to elevated triglyceride levels
X	E2/E3	6%	No increased risk of coronary heart disease/atherosclerosis/heart attack No predisposition to elevated LDL cholesterol levels Predisposition to elevated triglyceride levels
	E3/E3	66%	No increased risk of coronary heart disease No predisposition to elevated LDL cholesterol levels No predisposition to elevated triglyceride levels
	E2/E4	2%	No increased risk of coronary heart disease No predisposition to elevated LDL cholesterol levels No predisposition to elevated triglyceride levels
	E3/E4	24%	Increased risk of coronary heart disease/atherosclerosis/heart attack Predisposition to elevated LDL cholesterol levels Predisposition to elevated triglyceride levels
	E4/E4	1%	Increased risk of coronary heart disease/atherosclerosis/heart attack Predisposition to elevated LDL cholesterol levels Predisposition to elevated triglyceride levels

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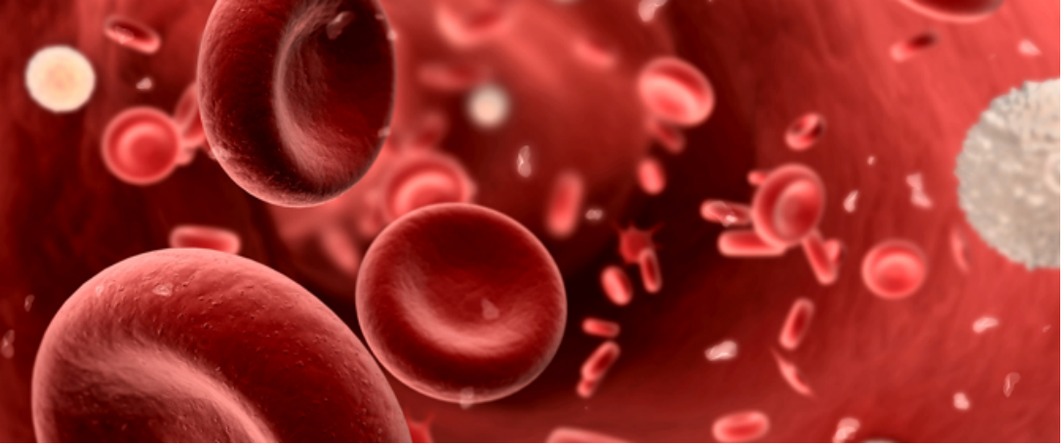
LEGEND: RES = your personal analysis result (marked with an X), GENOTYPE = different variations of the gene (called alleles),
POP = percent of the general population that have this genetic result,
POSSIBLE RESULTS = influence of the genetic variation.



Thrombo Sensor

Factor-V - Coagulation factor V (proaccelerin, labile factor) (rs6025)

The so-called factor V Leiden mutation is a genetically transmitted clotting defect, associated with an increased risk of thrombosis. This defect inhibits the degradation of factor V and the protein retains its coagulant effect.

RES	Genotype	POP	Possible results
	A/A	1%	Increased risk of thrombosis (venous) (OR: 80!)
	A/G	1%	Increased risk of thrombosis (venous) (OR: 7)
X	G/G	98%	No increased risk of thrombosis (venous)

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Factor-II - Coagulation factor II (thrombin) (rs1799963)

The prothrombin mutation (factor II mutation) is a blood coagulant disorder. The risk of venous thrombosis is significantly increased by the polymorphism rs1799963, which allows the creation of too much clotting factor, prothrombin, in the blood.

RES	Genotype	POP	Possible results
	A/A	1%	Increased risk of thrombosis (venous) (OR: 25)
	A/G	1%	Increased risk of thrombosis (venous) (OR: 5)
X	G/G	98%	No increased risk of thrombosis (venous)

References

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Ye et al. Seven haemostatic gene polymorphisms in coronary disease: meta-analysis of 66,155 cases and 91,307 controls. Lancet. 2006 Feb 25,367(9511):651-8.

PAI1 - Phosphoribosylanthranilate isomerase (rs1799889)

Plasminogen activator inhibitor-1 (PAI-1) is a glycoprotein belonging to the group of serine protease inhibitors. It inhibits the fibrinolytic activity, by inactivating tPA and urokinase. A defect in the PAI-1 gene leads to increased transcription and a higher concentration PAI-1. This condition is associated with an increased risk of thrombosis.

RES	Genotype	POP	Possible results
X	Del/Del	24%	Increased risk of thrombosis (arterial) (OR: 1.84)
	Del/G	48%	Increased risk of thrombosis (arterial) (OR: 1.83)
	G/G	28%	No increased risk of thrombosis (arterial)

References

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Ye et al. Seven haemostatic gene polymorphisms in coronary disease: meta-analysis of 66,155 cases and 91,307 controls. Lancet. 2006 Feb 25,367(9511):651-8.

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 of thrombosis (venous)
	C/T	33%	No increased risk of thrombosis (venous)
	T/T	8%	Increased risk of thrombosis (venous) (OR: 3)

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ITGB3 - Integrin beta 3 (platelet glycoprotein IIIa, antigen CD61) (rs5918)

The integrin beta 3 (ITGB3), or CD61, is a transmembrane protein involved in the signal transmission between cells and the extracellular matrix. It has been proven that carriers of the C-allele (rs5918) have an increased risk of cardiovascular diseases. In addition, the polymorphism influences the blood-thinning effect of the aspirin drug.

RES	Genotype	POP	Possible results
X	T/T	84%	Aspirin protects against arterial thrombosis
	T/C	15%	Aspirin does not provide any protection from thrombosis
	C/C	1%	Aspirin does not provide any protection from thrombosis

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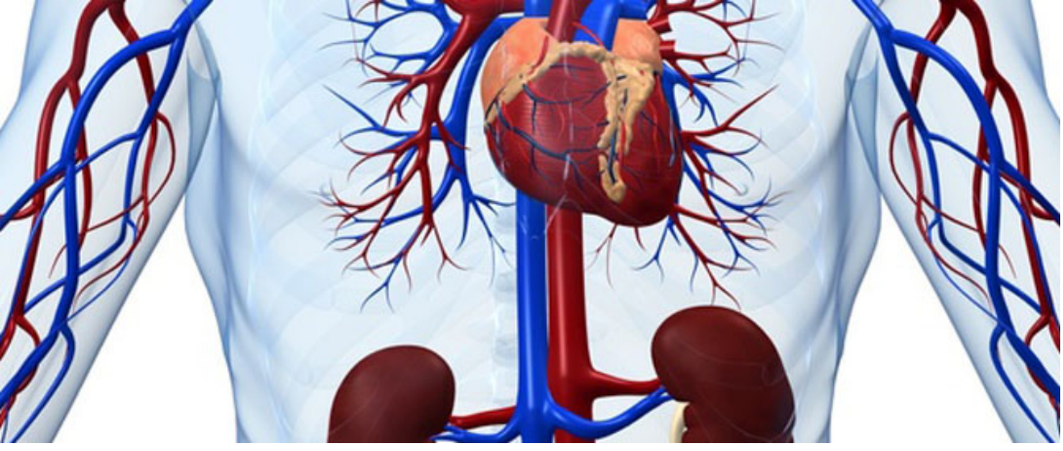
POP = percent of the general population that have this genetic result,
POSSIBLE RESULTS = influence of the genetic variation.



Hypertension Sensor

AGT - Angiotensinogen (serpin peptidase inhibitor, clade A, member 8) (rs699)

The polymorphism rs699 in the angiotensinogen gene (AGT) leads to an increased concentration of angiotensinogen in the blood serum, and thus to a predisposition to high blood pressure.

RES	Genotype	POP	Possible results
X	T/T	54%	No predisposition to high blood pressure/hypertension A reduced salt intake is effective on average for the prevention of hypertension / hypertension
	T/C	34%	Predisposition to high blood pressure/hypertension (OR: 1.2) A reduced salt intake is particularly effective for the prevention of hypertension / hypertension
	C/C	13%	Predisposition to high blood pressure/hypertension (OR: 1.4) A reduced salt intake is particularly effective for the prevention of hypertension / hypertension

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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%	Vitamin B2 does not lower the blood pressure
	C/T	33%	Vitamin B2 does not lower the blood pressure
	T/T	8%	Vitamin B2 does lower the blood pressure

References

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ADRB1 - Adrenoceptor beta 1 (rs1801253)

The β1-adrenoceptor protein encoded by the gene ADRB1 is the main adrenergic receptor of the human heart. It is mainly responsible for the effect of the adrenaline and the target structure of the beta-blockers.

RES	Genotype	POP	Possible results
	G/G	10%	No predisposition to high blood pressure/hypertension
X	G/C	40%	No predisposition to high blood pressure/hypertension
	C/C	50%	Predisposition to high blood pressure/hypertension (OR: 1.9)

References

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GNB3 - Guanine nucleotide binding protein (G protein), beta polypeptide 3 (rs5443)

G-proteins are signal transduction proteins, bonded to the inside of the cell membrane receptors, and involved in a variety of signaling pathways. The polymorphism rs5443 is associated with both high blood pressure and a predisposition to excessive weight.

RES	Genotype	POP	Possible results
	C/C	30%	No predisposition to high blood pressure/hypertension
X	C/T	41%	Predisposition to high blood pressure/hypertension (OR: 6.1)
	T/T	29%	Predisposition to high blood pressure/hypertension (OR: 6.1)

References

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

Alzheimer Sensor

APOE - apolipoprotein E (E2/E3/E4)

APoE (apolipoprotein E) metabolizes triglyceride-rich lipoprotein constituents, and plays a central role in the lipid metabolism. The ApoE gene is present in three common types, which are called allele E2, E3 and E4. The E4 allele is associated with an increased risk of heart disease and Alzheimer.

RES	Genotype	POP	Possible results
	E2/E2	1%	Protection against Alzheimer's disease (OR: 0.7)
X	E2/E3	6%	Protection against Alzheimer's disease (OR: 0.7)
	E3/E3	66%	No increased risk of Alzheimer's disease
	E2/E4	2%	Increased risk of Alzheimer's disease (OR: 2.5)
	E3/E4	24%	Increased risk of Alzheimer's disease (OR: 3.2)
	E4/E4	1%	Increased risk of Alzheimer's disease (OR: 15)

References

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POP = percent of the general population that have this genetic result,
POSSIBLE RESULTS = influence of the genetic variation.

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

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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

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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.

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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

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POSSIBLE RESULTS = influence of the genetic variation.

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

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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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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

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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

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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

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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

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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.

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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

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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

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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.

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

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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

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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

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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

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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.

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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.

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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.

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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

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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

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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.

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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.

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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.

Iron Sensor

HFE C282Y - Haemochromatosis (rs1800562)

The HFE gene encodes the hereditary haemochromatosis-protein. The protein is expressed on the cell membrane and forms a complex that enables the binding of the principal iron transport protein, transferrin. Several polymorphisms in the HFE gene are associated with the occurrence of haemochromatosis.

RES	Genotype	POP	Possible results
X	G/G	97%	No increased risk of haemochromatosis
	G/A	1%	Increased risk of haemochromatosis (in combination)
	A/A	2%	Increased risk of haemochromatosis

References

Vujić et al. Molecular basis of HFE-hemochromatosis. Front Pharmacol. 2014 Mar 11,5:42.

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HFE H63D - Haemochromatosis (rs1799945)

The HFE gene encodes the hereditary haemochromatosis-protein. The protein is expressed on the cell membrane and forms a complex that enables the binding of the principal iron transport protein, transferrin. Several polymorphisms in the HFE gene are associated with the occurrence of haemochromatosis.

RES	Genotype	POP	Possible results
X	C/C	87%	No increased risk of haemochromatosis
	C/G	12%	Increased risk of haemochromatosis (in combination)
	G/G	1%	Increased risk of haemochromatosis

References

Vujić et al. Molecular basis of HFE-hemochromatosis. Front Pharmacol. 2014 Mar 11,5:42.

Carelle et al. Mutation analysis of the HLA-H gene in Italian hemochromatosis patients. Am J Hum Genet. Apr 1997, 60(4): 828–832.

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HFE S65C - Haemochromatosis (rs1800730)

The HFE gene encodes the hereditary haemochromatosis-protein. The protein is expressed on the cell membrane and forms a complex that enables the binding of the principal iron transport protein, transferrin. Polymorphisms in the HFE gene are associated with the occurrence of haemochromatosis.

RES	Genotype	POP	Possible results
X	A/A	97%	No increased risk of haemochromatosis
	A/T	1%	Increased risk of haemochromatosis (in combination)
	T/T	2%	Increased risk of haemochromatosis (in combination)

References

Mura et al. HFE mutations analysis in 711 hemochromatosis probands: evidence for S65C implication in mild form of hemochromatosis. Blood. 1999 Apr 15,93(8):2502-5.

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Crownover BK et al. Hereditary hemochromatosis. Am Fam Physician. 2013 Feb 1,87(3):183-90.

Wallace DF et al. Frequency of the S65C mutation of HFE and iron overload in 309 subjects heterozygous for C282Y. J Hepatol. 2002 Apr,36(4):474-9.

Asberg A et al. Hereditary hemochromatosis: the clinical significance of the S65C mutation. Genet Test. 2002 Spring,6(1):59-62.

Mura C et al. HFE mutations analysis in 711 hemochromatosis probands: evidence for S65C implication in mild form of hemochromatosis. Blood. 1999 Apr 15,93(8):2502-5.

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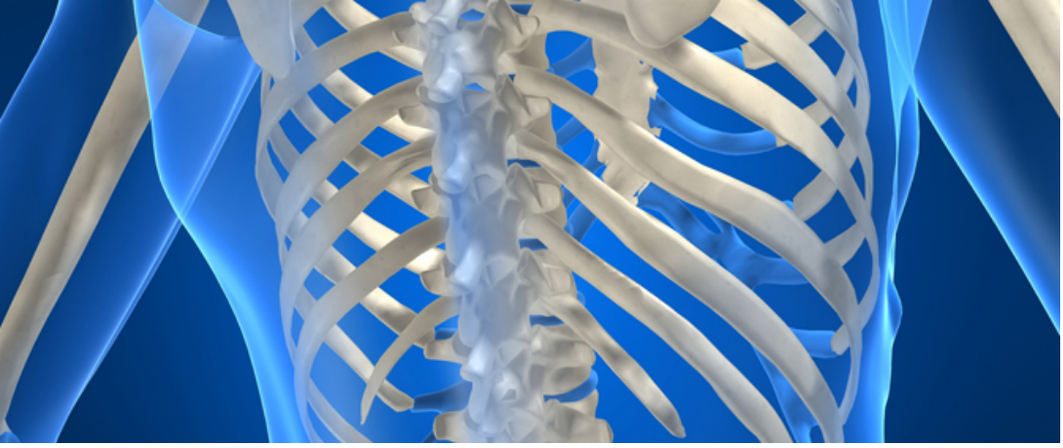

Bone Health Sensor

Col1A1 - Collagen, type I, alpha 1 (rs1800012)

The protein encoded by the COL1A1 gene (collagen type I, alpha 1) is the main protein component of the bone matrix. Defects in the COL1A1 structure lead to changes in the bone matrix.

RES	Genotype	POP	Possible results
	G/G	83%	No increased risk of osteoporosis Etidronate is particularly effective
	G/T	15%	Increased risk of osteoporosis (OR: 1.26)
X	T/T	2%	Increased risk of osteoporosis (OR: 1.78)

References

Mann V et a. Meta-analysis of COL1A1 Sp1 polymorphism in relation to bone mineral density and osteoporotic fracture. Bone. 2003 Jun,32(6):711-7.

Jin et al. Polymorphisms in the 5' flank of COL1A1 gene and osteoporosis: meta-analysis of published studies. Osteoporos Int. 2011 Mar,22(3):911-21.

Qureshi et al. COLIA1 Sp1 polymorphism predicts response of femoral neck bone density to cyclical etidronate therapy. Calcif Tissue Int. 2002 Mar,70(3):158-63. Epub 2002 Feb 19.

VDR - Vitamin D (1,25- dihydroxyvitamin D3) receptor (rs1544410)

The vitamin D receptor protein (VDR) is the most important regulator of calcium and bone metabolism. Vitamin D also controls a large number of important functions, such as calcium absorption, bone growth and the production of hormones. A defect in this gene leads to a change in bone density, amongst other things.

RES	Genotype	POP	Possible results
	G/G	52%	Increased protection against osteoporosis (OR: 0.61) Alendronate is particularly effective HRT is particularly effective as a preventative measure
	A/G	37%	No increased protection against osteoporosis Etidronate is particularly effective Clodronate is particularly effective
X	A/A	11%	No increased protection against osteoporosis Etidronate is particularly effective Clodronate is particularly effective Raloxifene is particularly effective

References

Palomba et al. BsmI vitamin D receptor genotypes influence the efficacy of antiresorptive treatments in postmenopausal osteoporotic women. A 1-year multicenter, randomized and controlled trial. Osteoporos Int. 2005 Aug,16(8):943-52. Epub 2005 Mar 1.

Jia et al. Vitamin D receptor BsmI polymorphism and osteoporosis risk: a meta-analysis from 26 studies. Genet Test Mol Biomarkers. 2013 Jan,17(1):30-4.

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Marc J et al. VDR genotype and response to etidronate therapy in late postmenopausal women. Osteoporos Int. 1999,10(4):303-6.

Creatsa M et al. The effect of vitamin D receptor BsmI genotype on the response to osteoporosis treatment in postmenopausal women: a pilot study. J Obstet Gynaecol Res. 2011 Oct,37(10):1415-22.

Mossetti G et al. Vitamin D receptor gene polymorphisms predict acquired resistance to clodronate treatment in patients with Paget's disease of bone. Calcif Tissue Int. 2008 Dec,83(6):414-24.

ESR1 - Estrogen receptor 1 (rs2234693)

Oestrogens have a positive effect on the human skeleton through regulation of bone metabolism, control of the optimal bone mass and limitation of bone loss. Defects in this gene can impact negatively on these effects.

RES	Genotype	POP	Possible results
	C/C	20%	No increased risk for osteoporosis HRT is particularly effective
X	C/T	49%	Increased risk of osteoporosis (OR: 2)
	T/T	31%	Increased risk of osteoporosis (OR: 4)

References

Gennari L et al. Estrogen receptor gene polymorphisms and the genetics of osteoporosis: a HuGE review. Am J Epidemiol. 2005 Feb 15,161(4):307-20.

van Meurs JB et al. Association of 5' estrogen receptor alpha gene polymorphisms with bone mineral density, vertebral bone area and fracture risk. Hum Mol Genet. 2003 Jul 15,12(14):1745-54.

Herrington DM et al. Estrogen-receptor polymorphisms and effects of estrogen replacement on high-density lipoprotein cholesterol in women with coronary disease. N Engl J Med. 2002 Mar 28,346(13):967-74.

Herrington DM et al. Common estrogen receptor polymorphism augments effects of hormone replacement therapy on E-selectin but not C-reactive protein. Circulation. 2002 Apr 23,105(16):1879-82.

LCT - lactase (rs4988235)

The LCT gene encodes for the protein lactase, an enzyme in the small intestine that splits the milk sugar (lactose) so that it can be absorbed. If the LCT gene is defective, the lactose consumed can either be absorbed insufficiently, or not at all. This is known as lactose intolerance. The avoidance of dairy products usually leads to a reduced absorption of calcium.

RES	Genotype	POP	Possible results
X	T/T	76%	Normal calcium intake from nutrition
	T/C	8%	Normal calcium intake from nutrition
	C/C	16%	Reduced calcium intake from nutrition

References

Koek et al. The T-13910C polymorphism in the lactase phlorizin hydrolase gene is associated with differences in serum calcium levels and calcium intake.

Bácsi Ket al. LCT 13910 C/T polymorphism, serum calcium, and bone mineral density in postmenopausal women. Osteoporosis International, 20(4), 639–645.

Tolonen S et al. Cardiovascular Risk in Young Finns Study Group. (2011). Lactase Gene C/T-13910 Polymorphism, Calcium Intake, and pQCT Bone Traits in Finnish Adults. Calcified Tissue International, 88(2), 153–161.

Laaksonen MM et al. Genetic lactase non-persistence, consumption of milk products and intakes of milk nutrients in Finns from childhood to young adulthood. Br J Nutr. 2009 Jul,102(1):8-17.

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Kuchay RA et al. Effect of C/T -13910 cis-acting regulatory variant on expression and activity of lactase in Indian children and its implication for early genetic screening of adult-type hypolactasia. Clin Chim Acta. 2011 Oct 9,412(21-22):1924-30.

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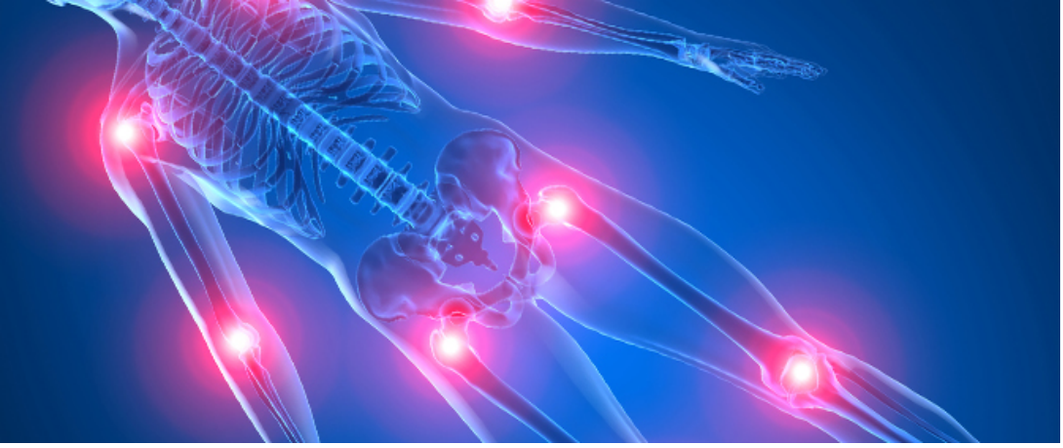

Joint Sensor

TNF-α - tumor necrosis factor a (TNF superfamily, member 2) (rs1800629)

The tumour necrosis factor (TNF or TNF-α) is a cytokine in the human immune system that regulates the activity of immune cells. TNF regulates apoptosis, cell proliferation, cell differentiation and the secretion of various cytokines. The polymorphism rs1800629 leads to a highly increased TNFa expression, and thus to an increased inflammatory capacity.

RES	Genotype	POP	Possible results
	G/G	83%	No increased risk of rheumatoid arthritis
	G/A	17%	Increased risk for rheumatoid arthritis (OR: 2.9)
X	A/A	1%	Increased risk for rheumatoid arthritis (OR: 7.29)

References

Dayer et al. The pivotal role of interleukin-1 in the clinical manifestations of rheumatoid arthritis. Rheumatology 2003,42(Suppl. 2):ii3–ii10

Goldring et al. Pathogenesis of bone and cartilage destruction in rheumatoid arthritis. Rheumatology 2003,42(Suppl. 2):ii11–ii16

Oregón-Romero et al. Tumor necrosis factor alpha-308 and -238 polymorphisms in rheumatoid arthritis. Association with messenger RNA expression and sTNF-alpha. J Investig Med. 2008 Oct,56(7):937-43.

IL1A - interleukin 1 alpha (rs1800587)

The interleukin-1 gene cluster on chromosome 2 contains the genes for IL1A and IL1B. In the presence of these polymorphisms (rs1800587 and rs1143634) the T-allele increases the IL-1 synthesis, leading to an increase of the inflammatory capacity.

RES	Genotype	POP	Possible results
	T/T	10%	Increased risk of rheumatoid arthritis (OR 1.36) Increased risk of degenerative disc disease (OR: 7.87)
	T/C	36%	Increased risk of rheumatoid arthritis (OR 1.17) Increased risk of degenerative disc disease (OR: 1.31)
X	C/C	54%	No increased risk of rheumatoid arthritis

References

Virtanen et al. Occupational and genetic risk factors associated with intervertebral disc disease. Spine (Phila Pa 1976). 2007 May 1,32(10):1129-34.

Dayer et al. The pivotal role of interleukin-1 in the clinical manifestations of rheumatoid arthritis. Rheumatology 2003,42(Suppl. 2):ii3–ii10

Goldring et al. Pathogenesis of bone and cartilage destruction in rheumatoid arthritis. Rheumatology 2003,42(Suppl. 2):ii11–ii16

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Gluten Sensor

HLA DQ 2.5 (rs2187668)

The human leukocyte antigen system (HLA system) is a group of genes that play a central role in the immune system. It has been shown that certain polymorphisms are associated with the celiac disease.

RES	Genotype	POP	Possible results
X	G/G	85%	Gluten intolerance/ celiac disease are practically impossible in the absence of other risk variants.
	A/G	14%	There is a genetic predisposition for gluten intolerance
	A/A	1%	There is a genetic predisposition for gluten intolerance

References

Monsuur et al. Effective Detection of Human Leukocyte Antigen Risk Alleles in Celiac Disease Using Tag Single Nucleotide Polymorphisms. PLoS One. 2008 May 28.3(5):e2270.

Wolters et al. Genetic background of celiac disease and its clinical implications. Am J Gastroenterol. 2008 Jan,103(1):190-5.

Louka et al. A collaborative European search for non-DQA1*05-DQB1*02 celiac disease loci on HLA-DR3 haplotypes: analysis of transmission from homozygous parents. Hum Immunol. 2003 Mar,64(3):350-8.

HLA DQ 8 (rs7454108)

The human leukocyte antigen system (HLA system) is a group of genes that play a central role in the immune system. It has been shown that certain polymorphisms are associated with the celiac disease.

RES	Genotype	POP	Possible results
	T/T	67%	Gluten intolerance/ celiac disease are practically impossible in the absence of other risk variants.
X	C/T	30%	There is a genetic predisposition for gluten intolerance
	C/C	3%	There is a genetic predisposition for gluten intolerance

References

Monsuur et al. Effective Detection of Human Leukocyte Antigen Risk Alleles in Celiac Disease Using Tag Single Nucleotide Polymorphisms. PLoS One. 2008 May 28.3(5):e2270

Wolters et al. Genetic background of celiac disease and its clinical implications. Am J Gastroenterol. 2008 Jan,103(1):190-5.

Louka et al. A collaborative European search for non-DQA1*05-DQB1*02 celiac disease loci on HLA-DR3 haplotypes: analysis of transmission from homozygous parents. Hum Immunol. 2003 Mar,64(3):350-8.

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Lactose Sensor

LCT - lactase (rs4988235)

The LCT gene encodes for the protein lactase, an enzyme in the small intestine that splits the milk sugar (lactose) so that it can be absorbed. If the LCT gene is defective, the lactose consumed can either be absorbed insufficiently, or not at all. This is known as lactose intolerance. The avoidance of dairy products usually leads to a reduced absorption of calcium.

RES	Genotype	POP	Possible results
X	T/T	55%	No increased risk for lactose intolerance Normal calcium intake from food
	C/T	36%	No increased risk for lactose intolerance Normal calcium intake from food
	C/C	9%	Very high risk for lactose intolerance later in life Reduced calcium intake from food

References

Enattah et al. Identification of a variant associated with adult-type hypolactasia. Nat Genet. 2002 Feb,30(2):233-7.

Bersaglieri et al. Genetic Signatures of Strong Recent Positive Selection at the Lactase Gene. The American Journal of Human Genetics, 74(6), 1111–1120.

Rasinperä et al. Transcriptional downregulation of the lactase (LCT) gene during childhood. Gut. Nov 2005, 54(11): 1660–1661.

Matlik L et al. Perceived milk intolerance is related to bone mineral content in 10- to 13-year-old female adolescents. Pediatrics 2007.

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IBD Sensor

NOD2 - nucleotide-binding oligomerization domain containing 2 (rs2066844)

NOD2 (nucleotide-binding oligomerization domain-containing protein 2) is a receptor protein which recognizes bacterial molecules and activates the NF-KB signaling pathway. This is part of the immune response. NOD2 was identified as a Crohn disease associated gene.

RES	Genotype	POP	Possible results
	T/T	1%	Increased risk of Crohn's disease (OR: 2.52)
	T/C	3%	Increased risk of Crohn's disease (OR: 1.59)
X	C/C	96%	No increased risk of Crohn's disease

References

Jung et al. Genotype/phenotype analyses for 53 Crohn's disease associated genetic polymorphisms. PLoS One. 2012,7(12):e52223.

Hugot et al. Association of NOD2 leucine-rich repeat variants with susceptibility to Crohn's disease. Nature. 2001 May 31,411(6837):599-603.

Glas et al. The NOD2 single nucleotide polymorphisms rs2066843 and rs2076756 are novel and common Crohn's disease susceptibility gene variants. PLoS One. 2010 Dec 30,5(12):e14466.

Yazdanyar et al. Penetrance of NOD2/CARD15 genetic variants in the general population. CMAJ. 2010 Apr 20,182(7):661-5.

NOD2 - nucleotide-binding oligomerization domain containing 2 (rs2066845)

RES	Genotype	POP	Possible results
X	G/G	98%	No increased risk of Crohn's disease
	G/C	1%	Increased risk of Crohn's disease (OR: 1.98)
	C/C	1%	Increased risk of Crohn's disease (OR: 3.92)

References

Jung et al. Genotype/phenotype analyses for 53 Crohn's disease associated genetic polymorphisms. PLoS One. 2012,7(12):e52223.

Hugot et al. Association of NOD2 leucine-rich repeat variants with susceptibility to Crohn's disease. Nature. 2001 May 31,411(6837):599-603.

Glas et al. The NOD2 single nucleotide polymorphisms rs2066843 and rs2076756 are novel and common Crohn's disease susceptibility gene variants. PLoS One. 2010 Dec 30,5(12):e14466.

Yazdanyar et al. Penetrance of NOD2/CARD15 genetic variants in the general population. CMAJ. 2010 Apr 20,182(7):661-5.

NOD2 - nucleotide-binding oligomerization domain containing 2 (rs2066847)

RES	Genotype	POP	Possible results
	C/C	1%	Increased risk of Crohn's disease (OR: 15)
	del/C	1%	Increased risk of Crohn's disease (OR: 11)
X	del/del	98%	No increased risk of Crohn's disease

References

Jung et al. Genotype/phenotype analyses for 53 Crohn's disease associated genetic polymorphisms. PLoS One. 2012,7(12):e52223.

Hugot et al. Association of NOD2 leucine-rich repeat variants with susceptibility to Crohn's disease. Nature. 2001 May 31,411(6837):599-603.

Glas et al. The NOD2 single nucleotide polymorphisms rs2066843 and rs2076756 are novel and common Crohn's disease susceptibility gene variants. PLoS One. 2010 Dec 30,5(12):e14466.

Yazdanyar et al. Penetrance of NOD2/CARD15 genetic variants in the general population. CMAJ. 2010 Apr 20,182(7):661-5.

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Glaucoma Sensor

LOXL1 - lysyl oxidase-like 1 (rs3825942)

Lysyl oxidase-like 1 (LOXL1) is a copper-dependent protein that plays an important role in elastogenesis. A genetic defect in LOXL1 gene is associated with an increased risk of glaucoma.

RES	Genotype	POP	Possible results
X	C/C	73%	Increased risk of open-angle glaucoma (OR: 11.19)
	C/T	25%	Increased risk of open-angle glaucoma (OR: 1.66)
	T/T	2%	No increased risk of open-angle glaucoma

References

Chen et al. Ethnicity-based subgroup meta-analysis of the association of LOXL1 polymorphisms with glaucoma. Mol Vis. 2010 Feb 6,16:167-77.

Thorleifsson et al. Common sequence variants in the LOXL1 gene confer susceptibility to exfoliation glaucoma. Science. 2007 Sep 7,317(5843):1397-400. Epub 2007 Aug 9.

Pasutto F et al. Association of LOXL1 Common Sequence Variants in German and Italian Patients with Pseudoexfoliation Syndrome and Pseudoexfoliation Glaucoma. Investigative Opthalmology & Visual Science, 49(4), 1459.

Wang L et al. LOXL1 Gene Polymorphism With Exfoliation Syndrome/Exfoliation Glaucoma: A Meta-Analysis. J Glaucoma. 2016 Jan,25(1):62-94.

Fan BJ et al. DNA sequence variants in the LOXL1 gene are associated with pseudoexfoliation glaucoma in a U.S. clinic-based population with broad ethnic diversity. BMC Med Genet. 2008 Feb 6,9:5.

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AMD Sensor

HTRA1 - HtrA serine peptidase 1 (rs11200638)

The polymorphism rs11200638 in the HTRA1 gene (high temperature requirement protein A1) is associated with an increased risk for age-related macular degeneration. The encoded protein, a serine protease, plays an important role in the quality control of the extracellular matrix proteins. The mutation in the promoter region of the gene leads to overexpression of the pigment epithelium and to an increased risk of disease.

RES	Genotype	POP	Possible results
	A/A	9%	Increased risk of macular degeneration (OR: 8.6)
	A/G	40%	Increased risk of macular degeneration (OR: 2.2)
X	G/G	51%	No increased risk for macular degeneration

References

Yang et al. A variant of the HTRA1 gene increases susceptibility to age-related macular degeneration. Science. 2006 Nov 10,314(5801):992-3.

Chen et al. Meta-analysis of the association of the HTRA1 polymorphisms with the risk of age-related macular degeneration. Exp Eye Res. 2009 Sep,89(3):292-300.

Dewan et al. HTRA1 promoter polymorphism in wet age-related macular degeneration. Science. 2006 Nov 10,314(5801):989-92.

LOC387715 - Age-related maculopathy susceptibility 2 (rs10490924)

The LOC387715 gene locus is located on chromosome 10. The rs10490924 polymorphism is associated with an increased risk of developing age-related macular degeneration.

RES	Genotype	POP	Possible results
X	G/G	51%	No increased risk for macular degeneration
	G/T	40%	Increased risk of macular degeneration (OR: 2.69)
	T/T	9%	Increased risk of macular degeneration (OR: 8.21)

References

Fritsche et al. Age-related macular degeneration is associated with an unstable ARMS2 (LOC387715) mRNA. Nat Genet. 2008 Jul,40(7):892-6.

Rivera et al. Hypothetical LOC387715 is a second major susceptibility gene for age-related macular degeneration, contributing independently of complement factor H to disease risk. Hum Mol Genet. 2005 Nov 1, 14(21):3227-36.

Ross et al. The LOC387715 and age-related macular degeneration: replication in three case-control samples. Invest Ophthalmol Vis Sci. 2007,48:1128–1132.

CFH - Complement factor H (rs1061170)

A defect in the CFH (complement factor H) gene is regarded, in different studies, as the primary risk for the development of AMD. The complement factor H controls the immune response against various pathogens.

RES	Genotype	POP	Possible results
	T/T	55%	No increased risk for macular degeneration
X	T/C	36%	Increased risk of macular degeneration (OR: 4)
	C/C	9%	Increased risk of macular degeneration (OR: 12)

References

Klein et al. Complement Factor H Polymorphism in Age-Related Macular Degeneration. Science. Apr 15, 2005, 308(5720): 385–389.

Haines et al. Complement factor H variant increases the risk of age-related macular degeneration. Science. 2005 Apr 15,308(5720):419-21.

Hageman G et al. From The Cover: A common haplotype in the complement regulatory gene factor H (HF1/CFH) predisposes individuals to age-related macular degeneration. Proceedings of the National Academy of Sciences, 102(20), 7227–7232.

Thakkinstian A et al. Systematic review and meta-analysis of the association between complement factor H Y402H polymorphisms and age-related macular degeneration. Hum Mol Genet. 2006 Sep 15,15(18):2784-90. Epub 2006 Aug 11.

Kondo N et al. Complement factor H Y402H variant and risk of age-related macular degeneration in Asians: a systematic review and meta-analysis. Ophthalmology. 2011 Feb,118(2):339-44.

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Periodontitis Sensor

IL1RN - interleukin 1 receptor antagonist (rs419598)

The interleukin 1 receptor antagonist (IL1RN) is involved in the regulation of immune and inflammatory responses. The rs419598 polymorphism can enhance the inflammatory activity, which leads to an increased risk of periodontitis. In addition, it was shown that carriers of the C-allele have an increased risk of titanium implant loss.

RES	Genotype	POP	Possible results
	T/T	67%	Increased risk of periodontitis (OR: 3.44) No increased risk of titanium implant loss
X	T/C	28%	Increased risk of periodontitis (OR: 3.44) Increased risk of titanium implant loss
	C/C	5%	No increased risk for periodontitis Increased risk of titanium implant loss

References

Braosi et al. Analysis of IL1 gene polymorphisms and transcript levels in periodontal and chronic kidney disease. Cytokine. 2012 Oct,60(1):76-82.

Trevilatto et al. Association of IL1 gene polymorphisms with chronic periodontitis in Brazilians. Arch Oral Biol. 2011 Jan,56(1):54-62.

Baradaran-Rahimi et al. Association of interleukin-1 receptor antagonist gene polymorphisms with generalized aggressive periodontitis in an Iranian population. J Periodontol. 2010 Sep,81(9):1342-6.

Komatsu et al. Association of interleukin-1 receptor antagonist +2018 gene polymorphism with Japanese chronic periodontitis patients using a novel genotyping method. Int J Immunogenet. 2008 Apr,35(2):165-70.

Jacobi-Gresser et al. Genetic and immunological markers predict titanium implant failure: a retrospective study. Int J Oral Maxillofac Surg. 2013 Apr,42(4):537-43.

Laine et al., IL-1RN gene polymorphism is associated with peri-implantitis. Clin Oral Implants Res. 2006 Aug, 17(4):380-5.

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
	G/G	77%	No increased risk for periodontitis
X	G/C	19%	No increased risk for periodontitis
	C/C	5%	Increased risk of periodontitis (OR: 1.89)

References

Nibali et al. Association between periodontitis and common variants in the promoter of the interleukin-6 gene. Cytokine. 2009 Jan,45(1):50-4.

de Sá et al. Association of CD14, IL1B, IL6, IL10 and TNFA functional gene polymorphisms with symptomatic dental abscesses. Int Endod J. 2007 Jul,40(7):563-72.

Babel et al. Analysis of tumor necrosis factor-alpha, transforming growth factor-beta, interleukin-10, IL-6, and interferon-gamma gene polymorphisms in patients with chronic periodontitis. J Periodontol. 2006 Dec,77(12):1978-83.

IL1A - interleukin 1 alpha (rs1800587)

The interleukin-1 gene cluster on chromosome 2 contains the genes for IL1A and IL1B. In the presence of these polymorphisms (rs1800587 and rs1143634) the T-allele increases the IL-1 synthesis, leading to an increase of the inflammatory capacity.

RES	Genotype	POP	Possible results
	T/T	54%	Increased risk of periodontitis (OR: 1.73) Increased risk of titanium implant loss
	T/C	10%	Increased risk of periodontitis (OR: 1.31) Increased risk of titanium implant loss
X	C/C	36%	No increased risk for periodontitis No increased risk of titanium implant loss

References

Jacobi-Gresser et al. Genetic and immunological markers predict titanium implant failure: a retrospective study. Int J Oral Maxillofac Surg. 2013 Apr,42(4):537-43.

Nikolopoulos et al. Cytokine gene polymorphisms in periodontal disease: a meta analysis of 53 studies including 4178 cases and 4590 controls. J Clin Periodontol 2008

Jansson et al., Clinical consequences of IL-1 genotype on early implant failures in patients under periodontal maintenance. Clin Implant Dent Relat Res. 2005, 7(1)51-9.

Feloutzis et al., IL-1 gene polymorphism and smoking as risk factors for peri-implant bone loss in a wellmaintained population. Clin Oral Implants Res. 2003, 14(1)10-7.

IL1B - interleukin 1 beta (rs1143634)

The interleukin-1 gene cluster on chromosome 2 contains the genes for IL1A and IL1B. In the presence of these polymorphisms (rs1800587 and rs1143634) the T-allele increases the IL-1 synthesis, leading to an increase of the inflammatory capacity.

RES	Genotype	POP	Possible results
	T/T	2%	Increased risk of periodontitis (OR: 4.89) Increased risk of titanium implant loss
X	T/C	22%	Increased risk of periodontitis (OR: 2.85) Increased risk of titanium implant loss
	C/C	76%	No increased risk for periodontitis No increased risk of titanium implant loss

References

Gore et al. Interleukin-1beta+3953 allele 2: association with disease status in adult periodontitis. J Clin Periodontol. 1998 Oct,25(10):781-5.

Galbraith et al. Polymorphic cytokine genotypes as markers of disease severity in adult periodontitis. J Clin Periodontol. 1999 Nov,26(11):705-9.

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Feloutzis et al., IL-1 gene polymorphism and smoking as risk factors for peri-implant bone loss in a wellmaintained population. Clin Oral Implants Res. 2003, 14(1):10-7.

TNF-α - tumor necrosis factor a (TNF superfamily, member 2) (rs1800629)

The tumour necrosis factor (TNF or TNF-α) is a cytokine in the human immune system that regulates the activity of immune cells. TNF regulates apoptosis, cell proliferation, cell differentiation and the secretion of various cytokines. The polymorphism rs1800629 leads to a highly increased TNFa expression, and thus to an increased inflammatory capacity.

RES	Genotype	POP	Possible results
	G/G	83%	No increased risk of titanium implant loss
	G/A	17%	Increased risk of titanium implant loss
X	A/A	1%	Increased risk of titanium implant loss

References

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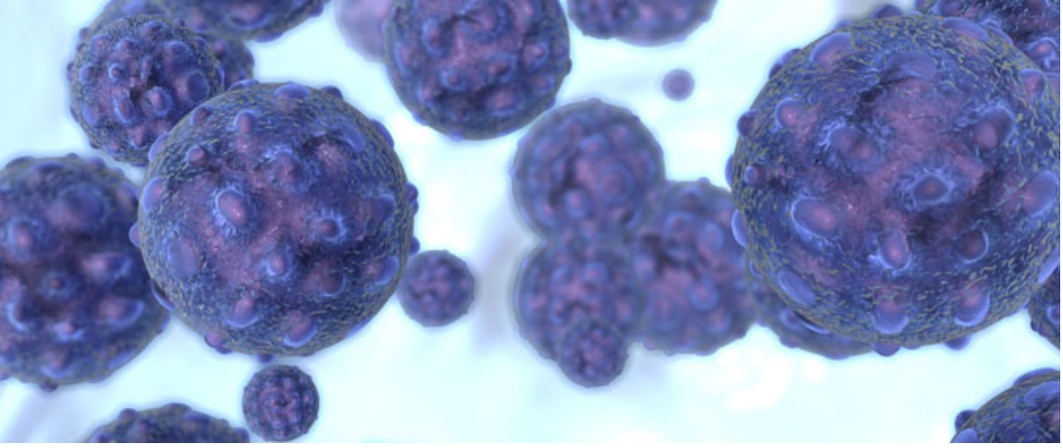
Dereka X et al. A systematic review on the association between genetic predisposition and dental implant biological complications. Clinical Oral Implants Research, 23(7), 775–788.

Nikolopoulos G et al. Cytokine gene polymorphisms in periodontal disease: a meta-analysis of 53 studies including 4178 cases and 4590 controls. Journal of Clinical Periodontology, 35(9), 754–767.

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.



HIV Resistance Sensor

CCR5 - chemokine (C-C motif) receptor 5 (gene/pseudogene) (rs333)

The chemokine receptor CCR5 is widespread in leukocytes and leucoplasts, and plays an important role in a variety of immunological processes. CCR5 is also an essential co-receptor in the sexual transmission of HIV, by allowing the settling of the HIV virus into cells. It has been shown that the so-called CCR5 Delta32 mutation has an effect on both HIV infection risk, as well as on the progression of the disease.

RES	Genotype	POP	Possible results
X	G/G	90%	Normal risk of HIV infection by viral contact
	G/DEL	9%	Lower risk of HIV (CCR5 HIV variant) infection by viral contact Slower progression of the disease
	DEL/DEL	1%	Virtually no risk of HIV infection by viral contact (CCR5 HIV variant) Normal risk of HIV infection by CCR5-independent HIV variants

References

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Fellay et al. NIAID Center for HIV/AIDS Vaccine Immunology (CHAVI). Common genetic variation and the control of HIV-1 in humans. PLoS Genet. 2009 Dec:5(12)

Hütter et al. Coregulation of HIV-1 dependency factors in individuals heterozygous to the CCR5-delta32 deletion. AIDS Res Ther. 2013 Nov 18:10(1):26.

Agrawal et al. CCR5Delta32 protein expression and stability are critical for resistance to human immunodeficiency virus type 1 in vivo. J Virol. 2007 Aug:81(15):8041-9

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

Hormone replacement therapy (HRT)

VDR - Vitamin D (1,25- dihydroxyvitamin D3) receptor (rs1544410)

The vitamin D receptor protein (VDR) is the most important regulator of calcium and bone metabolism. Vitamin D also controls a large number of important functions, such as calcium absorption, bone growth and the production of hormones. A defect in this gene leads to a change in bone density, amongst other things.

RES	Genotype	POP	Possible results
	G/G	52%	Hormone replacement therapy is significantly more effective for osteoporosis prevention
	A/G	37%	Hormone replacement therapy is not more effective for osteoporosis prevention, than in the the average population
X	A/A	11%	Hormone replacement therapy is not more effective for osteoporosis prevention, than in the the average population

References

Jia et al. Vitamin D receptor BsmI polymorphism and osteoporosis risk: a meta-analysis from 26 studies. Genet Test Mol Biomarkers. 2013 Jan,17(1):30-4.

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Chen H et al. Relation of BsmI vitamin D receptor gene polymorphism to bone mineral density and occurrence of osteoporosis in postmenopausal Chinese women in Taiwan. Osteoporosis International : A Journal Established as Result of Cooperation between the European Foundation for Osteoporosis and the National Osteoporosis Foundation of the USA, 12(12), 1036–41.

Kikuchi R et al. Early and late postmenopausal bone loss is associated with BsmI vitamin D receptor gene polymorphism in Japanese women. Calcified Tissue International, 64(2), 102–6.

APOE - apolipoprotein E (E2/E3/E4)

APoE (apolipoprotein E) metabolizes triglyceride-rich lipoprotein constituents, and plays a central role in the lipid metabolism. The ApoE gene is present in three common types, which are called allele E2, E3 and E4.

RES	Genotype	POP	Possible results
	E2/E2	1%	HRT is very effective in lowering the total and the LDL cholesterol levels
X	E2/E3	6%	HRT is very effective in lowering the total and the LDL cholesterol levels
	E3/E3	66%	HRT is very effective in lowering the total and the LDL cholesterol levels
	E2/E4	2%	HRT lowers the total and LDL cholesterol levels, at the same rate as for the average population
	E3/E4	24%	HRT lowers the total and LDL cholesterol levels, at the same rate as for the average population
	E4/E4	1%	HRT lowers the total and LDL cholesterol levels, at the same rate as for the average population

References

Phenotype of apolipoprotein E influences the lipid metabolic response of postmenopausal women to hormone replacement therapy. Tsuda et al. Maturitas. 2001 May 30,38(3):297-304.

Hagberg J et al. APO E gene and gene-environment effects on plasma lipoprotein-lipid levels. Physiological Genomics, 4(2), 101–108.

Tsuda M et al. Phenotype of apolipoprotein E influences the lipid metabolic response of postmenopausal women to hormone replacement therapy. Maturitas, 38(3), 297–304.

ESR1 - Estrogen receptor 1 (rs2234693)

Oestrogens have a positive effect on the human skeleton through regulation of bone metabolism, control of the optimal bone mass and limitation of bone loss. Defects in this gene can impact negatively on these effects.

RES	Genotype	POP	Possible results
	C/C	20%	**Hormone replacement therapy is significantly more effective for osteoporosis prevention**
X	C/T	49%	**Hormone replacement therapy is not more effective for osteoporosis prevention, than in the the average population**
	T/T	31%	**Hormone replacement therapy is not more effective for osteoporosis prevention, than in the the average population**

References

Gennari L et al. Estrogen receptor gene polymorphisms and the genetics of osteoporosis: a HuGE review. Am J Epidemiol. 2005 Feb 15,161(4):307-20.

van Meurs JB et al. Association of 5' estrogen receptor alpha gene polymorphisms with bone mineral density, vertebral bone area and fracture risk. Hum Mol Genet. 2003 Jul 15,12(14):1745-54.

Herrington DM et al. Estrogen-receptor polymorphisms and effects of estrogen replacement on high-density lipoprotein cholesterol in women with coronary disease. N Engl J Med. 2002 Mar 28,346(13):967-74.

Herrington DM et al. Common estrogen receptor polymorphism augments effects of hormone replacement therapy on E-selectin but not C-reactive protein. Circulation. 2002 Apr 23,105(16):1879-82.

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.

Note:

The scientific documentation of the breast cancer and thrombosis associated genes can be found in the relevant chapters.

PHARMACO GENETICS

ONCOLOGY

CARDIOVASCULAR SYSTEM

NEUROLOGY

METABOLISM

MOVEMENT

DIGESTION

OPHTHALMOLOGY

ODONTOLOGY

OTHERS

SCIENCE

ADDITIONAL INFORMATION

ADDITIONAL INFORMATION

In this chapter you will receive useful information

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Technical details

Order number
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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
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Product codes
L1WSS, L3NUT, L5TOX, L6EPI, L7BUR, L8AGE, M0PHA, M1CAR, M1HYP, M1THR, M2GLU, M2IBD, M2LAC, M3DIA, M3IRO, M4BON, M4JOI, M5ALZ, M5DEP, M5SCH, M6PER, M7BRE, M7COL, M7LUN, M7PRO, M7SKI, M8AMD, M8GLA, M9HIV

Current version
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