



23andMe Genetic Health Overview

Prepared for: **TERO KESKI-VALKAMA**

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What this overview includes

This overview includes brief summaries of your 23andMe results for:

- diseases for which you are at greater than average genetic risk,
- heritable diseases for which you carry one or more genetic variants (carrier status),
- and drugs to which you are likely to have an atypical response based on genetics.

These results are based on your genetic data and any sex and ancestry information you have provided along with population-level risk data for specified age ranges. They do not take into account non-genetic factors, family history, or additional genetic factors that may influence these conditions. Only results for genetic associations that are scientifically well established are included. This overview does not provide details regarding diseases for which you are at typical or lower than average genetic risk, heritable diseases for which you aren't known to carry a variant, or drugs to which you are likely to have a typical response. If you would like more information on any of your 23andMe results, please go to that topic's individual report page on our website at <https://www.23andme.com/you/health/>.

Overview of Genetic Health



Tero Keski-Valkama
Year of Birth: 1982
Northern European

Disease risk results are included in this overview only if your risk based on genetics is greater than 1%. Note that certain conditions may have genetic information applicable only to specific populations.

Components of this test were performed in a clinical laboratory regulated under the Clinical Laboratory Improvement Amendments of 1988 (CLIA) to perform high-complexity testing. The data provided are intended for informational and educational use and are not for diagnostic use.

**All conditions tested are listed at the end of the report. You may not have data for every report.*

Disease risk	Your risk	Average risk
Atrial Fibrillation	46.9%	27.2%
Venous Thromboembolism	17.9%	12.3%
Colorectal Cancer	9.0%	5.6%
Chronic Kidney Disease	5.0%	3.4%
Restless Legs Syndrome	2.5%	2.0%
26 conditions*	Typical or decreased risk	

Carrier status	Status
Hemochromatosis (HFE-related)	Variant Present
52 heritable conditions*	Variant Absent

Drug response	Response
Warfarin (Coumadin®) Sensitivity	Increased
Sulfonylurea Drug Clearance (Type 2 Diabetes Treatment)	Reduced
Phenytoin (Dilantin®) Sensitivity (Epilepsy Drug)	Increased
8 other drugs*	Typical Response

How to read your reports

1

Your risk: The probability of a person with the listed ancestry with your genetics developing the disease between the ages specified based on a specific set of genetic markers.

2

"Genes v. Environment" estimates how much of differences in risk for this condition in a population is due to genetics.

3

Average risk: The probability of a person with the listed ancestry from the general population developing the disease between the ages specified.

4

Your genetic risk is based on the sex and ancestry settings in your profile and your genotype(s) for a specific set of genetic markers. Your risk does not take into account non-genetic factors, family history, or additional genetic factors that may contribute to risk for this disease.

5

Genetic variants (and the genes they're located in or near) used to determine your results for this condition.

Disease Risk reports

Jill's Genetic Risk

41.6%
Jill's risk of developing Alzheimer's Disease between the ages specified

27.2%
Chance that the average person will develop Alzheimer's Disease

1.53x
Jill's risk compared to average

What is my risk based on?

- Age: 65 - 75
- Sex: Male
- European ancestry
- 2 genetic markers: rs207139 (p22.1), rs1803444 (p22.1)

Genes vs. Environment

The heritability of Alzheimer's Disease is estimated to be 60%. This means genetic factors contribute more to differences in risk for this condition than environmental factors. Genetic contributions to Alzheimer's Disease include both inherited factors and known factors such as the APOE4 allele described in this report. There are heritable forms of Alzheimer's Disease caused by rare mutations, but most affected people do not have a family history of the condition. Non-genetic factors that can increase the risk of Alzheimer's Disease are age, obesity, stress, heavy alcohol or caffeine consumption, diabetes, high blood pressure, and high cholesterol.

6

Indicates that you carry a genetic variant linked to an inherited genetic condition.

7

Details of the specific genetic marker(s) for which you carry a mutation linked to the condition. "Coverage" indicates the percentage of mutations for this disease that are covered by this test in specific populations. Other mutations not covered by this test may also be linked to the condition.

8

Your predicted response to this drug based on your genetics.

9

Details of the specific genetic markers tested, the genes in which those markers are located, your genotypes at those markers and other names for your combination of genotypes if any.

Carrier Status reports

Variant Present

Has one mutation in the APOE4 gene linked to Alzheimer's Disease. A person with one of these mutations typically does not have Alzheimer's Disease, but can pass the mutation to offspring. May still have other mutations in the APOE4 gene not reported here.

Gene	Variant	DNA change	2003 genotype
APOE4	V205X	C/T G/A	AC

Markers tested: 1 Coverage: 80% in Ashkenazi Jews, 40-60% in those with non-Jewish European ancestry

Drug Response reports

Intermediate

Increased warfarin sensitivity. May require decreased warfarin dose.

Marker	2003 Genotype
rs1799965	GG
rs1800745	CC
rs1800745	TT

Markers tested: 3 Genotype combination: CYP2C9*1/*1, VKORC1*1856/907A/A

Atrial Fibrillation

Atrial fibrillation is characterized by chaotic electrical signals in the heart that cause the upper chambers (atria) to quiver. It is the most common type of sustained irregular heart rhythm, and while it is not usually life threatening on its own, it can have deadly complications. Atrial fibrillation can disturb smooth blood flow, increasing the risk of clots that can cause organ damage or stroke. The heart's ability to pump blood can also deteriorate, leading to heart failure. The most common causes of atrial fibrillation are heart abnormalities and heart muscle damage, but in at least 10 percent of cases there is no underlying heart disease that explains the condition.

Tero's Genetic Risk

46.9%

Tero's risk of developing Atrial Fibrillation between the ages specified



27.2%

Chance that the average person will develop Atrial Fibrillation

What is my risk based on?

- Age** 0 - 79
- Sex** Men
- Pop** European ancestry
- SNP** 2 genetic markers
rs2200733 (4q25 (1)), rs10033464 (4q25 (2))

Genes vs. Environment

The heritability of atrial fibrillation is estimated to be 62%. This means genetic factors contribute more to differences in risk for this condition than environmental factors. Genetic contributions to atrial fibrillation include both unknown factors and known factors such as the SNPs described in this report. There are familial forms of atrial fibrillation caused by rare mutations, but most affected people do not have a family history of the condition. Non-genetic factors that can increase the risk of atrial fibrillation are age, obesity, stress, heavy alcohol or caffeine consumption, electrolyte imbalances, severe infections, diabetes and high blood pressure.

Additional Information

Other Medical Conditions

If you have a history of [heart disease](#) (including heart valve problems or a history of heart attack or surgery) your health care provider may work with you to manage these diseases to lower your risk for atrial fibrillation. Other medical problems, such as hyperthyroidism and sleep apnea, can also increase your risk for atrial fibrillation.

Medications and Treatment

If you have atrial fibrillation, your health care provider may [prescribe medications](#) that help control your heart rate and/or rhythm, or to prevent blood clots. If your atrial fibrillation cannot be controlled by medications, your health care provider may suggest a surgical procedure as treatment.

Lifestyle Factors

- **Eat healthy:** A healthy diet will help keep your heart healthy, even if you have no underlying cardiovascular disease. The [American Heart Association](#) has numerous resources and tools to help you make smart choices.
- **Consume in moderation:** [Heavy drinking](#) has been associated with increased risk for atrial fibrillation.

View the full report online for links to resources, references, and more detailed genetic results and information.

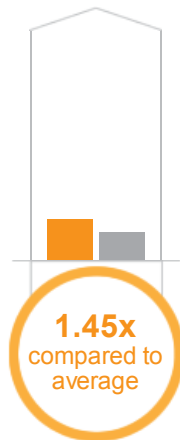
Venous Thromboembolism

Venous thromboembolism (VTE) encompasses two related conditions. The first, deep vein thrombosis or DVT, is the formation of a blood clot in a vein deep within the body, usually in the legs. The second, pulmonary embolism (PE), occurs if the clot breaks free and travels through the circulatory system to the lungs. DVT always precedes PE. It is estimated that about 250,000 people are hospitalized with venous thromboembolism in the United States each year, but the incidence is probably much higher as many cases go undiagnosed. Pulmonary embolism is potentially life threatening if prompt medical attention is not received. Therefore, recognizing the symptoms of venous thromboembolism and avoiding risk factors is of paramount importance.

Tero's Genetic Risk

17.9%

Tero's risk of developing Venous Thromboembolism between the ages specified



12.3%

Chance that the average person will develop Venous Thromboembolism

What is my risk based on?

- Age 0 - 79
- Sex Men
- Pop European ancestry
- SNP 3 genetic markers
rs6025 (F5), i3002432 (F2), rs505922 (ABO)

Genes vs. Environment

The heritability of venous thromboembolism is estimated to be 55%. This means that genetics (including unknown factors and known ones such as the SNPs we describe here) and environment play nearly equal roles in this condition. There are a number of environmental factors of various strengths that contribute to venous thromboembolism. Strong risk factors include hip or leg fractures, hip or knee replacement, major surgery or trauma, and spinal cord injury or surgery. Moderate risk factors include arthroscopic knee surgery, having central venous lines, congestive heart or respiratory failure, hormone replacement or oral contraceptive use, cancer, pregnancy, paralytic stroke, previous venous thromboembolism, and thrombophilia. Weak risk factors include bed rest for more than three days, immobility due to sitting (such as a long car or plane trip), specific types of chemotherapy, increasing age, laparoscopic surgery, obesity, and varicose veins.

Additional Information

Symptoms

Seek out medical attention immediately if you experience any of the [following](#):

DVT (leg clot) symptoms:

- Swelling, usually in one leg
- Leg pain or tenderness
- Reddish or bluish skin discoloration
- Leg warm to touch

PE (lung clot) symptoms:

- Sudden shortness of breath
- Chest pain-sharp, stabbing; may get worse with deep breath
- Rapid heart rate
- Unexplained cough, sometimes with bloody mucus

Medications and Treatment

Estrogen containing oral contraceptives and oral hormone replacement therapy are two commonly used medications that have been linked to increased clotting. Women taking these medications who also have genetic changes in their clotting factors and/or inhibitors are at especially high risk. Read more in the [Oral Contraceptives, Hormone Replacement Therapy and Risk of Venous Thromboembolism](#) Drug Response

Report.

Lifestyle Factors

- **Don't smoke:** [A large Danish study](#) found that women who smoked had a 52% increased risk for venous thromboembolism compared with women who had never smoked. For men, smoking conferred a 32% increase in risk. Heavy smokers had even higher risks.
- **Maintain a healthy weight:** [Obesity](#) increases the risk of venous thromboembolism.
- **Stay active:** Venous thromboembolism is sometimes called "[economy class syndrome](#)" because sitting still for long periods of time, as on a cramped airplane, can cause sluggish blood flow, which in turn increases the risk for the formation of blood clots.

View the full report online for links to resources, references, and more detailed genetic results and information.

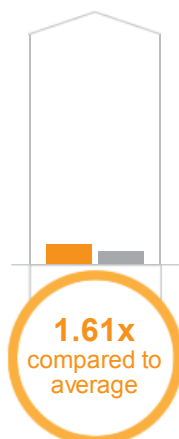
Colorectal Cancer

Colorectal cancer is the third most common cancer (excluding skin cancers) and the second leading cause of cancer-related deaths in the United States. The average lifetime risk of developing colorectal cancer is about 5%. Each year approximately 150,000 people are diagnosed with the disease. The good news is that if caught at an early stage—before it has had a chance to spread to other organs—the chances for survival are extremely high.

Tero's Genetic Risk

9.0%

Tero's risk of developing Colorectal Cancer between the ages specified



5.6%

Chance that the average person will develop Colorectal Cancer

What is my risk based on?

- Age** 15 - 79
- Sex** Men
- Pop** European ancestry
- SNP** 4 genetic markers
rs6983267 (8q24 region), rs4939827 (SMAD7), rs3802842 (LOC120376), rs4779584 (15q13.3 region)

Genes vs. Environment

The heritability of colorectal cancer is estimated to be 35%. This means that environmental factors contribute more to differences in risk for this condition than genetic factors. Genetic factors that play a role in colorectal cancer include both unknown and known factors. Known factors include rare mutations in the MSH2 and MLH1 genes that appear in familial cases of colon cancer (which 23andMe does not genotype), and the SNPs we describe here. Other factors include a history of previous colorectal cancer, colorectal polyps, or inflammatory bowel disease, being an Ashkenazi Jew or of African descent, a diet high in animal fat, physical inactivity, obesity, smoking, heavy alcohol use, and diabetes. (Note: The contribution of the SNPs reported by 23andMe to inherited colorectal cancer risk are minor. If you have a strong family history of early-onset colon cancer, you should consider mutation testing of MSH2 and MLH1.)

Additional Information

Screening and Risk Assessment

Regular screening can detect polyps, which can be removed before they become cancerous. See the [American Cancer Society's](#) recommendations for colorectal cancer screening. If you have a family history or other risk factors for colorectal cancer, talk to your health care provider about more frequent screening. Use the questionnaire available from [Your Disease Risk](#) to get an estimate of your risk for colorectal cancer.

Lifestyle Factors

The American Cancer Society recommends the following to [reduce the risk](#) of colorectal cancer:

- Exercise regularly
- Maintain a healthy weight
- Drink alcohol in moderation
- Eat a diet rich in whole grains, fruits, and vegetables. Limit intake of processed and red meats.

Family History

Your risk of colorectal cancer is increased if you have one or more family members with the disease. A strong family history of colorectal cancer may indicate that a mutation causing a [cancer syndrome](#) (not included in this report), such as familial adenomatous polyposis or hereditary nonpolyposis colorectal cancer, is being passed down through the generations. Use 23andMe's [Family Health History](#) tool to collect this important information, and consider speaking to a genetic counselor or your health care provider if you have a family history of colorectal cancer.

View the full report online for links to resources, references, and more detailed genetic results and information.

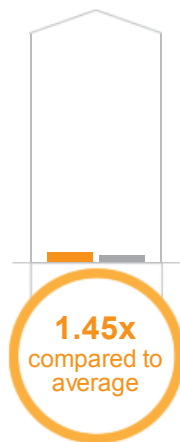
Chronic Kidney Disease

Chronic kidney disease (CKD) develops when damage to the kidneys decreases their ability to perform their many jobs, leading to waste build-up in the body and chemical imbalances. CKD ranges in severity from nearly normal kidney function to complete kidney failure requiring dialysis or kidney transplantation for survival. CKD affects about 26 million adults in the United States and this number is increasing as rates of diabetes and hypertension—the two most common causes of CKD—continue to rise.

Tero's Genetic Risk

5.0%

Tero's risk of developing Chronic Kidney Disease between the ages specified



3.4%

Chance that the average person will develop Chronic Kidney Disease

What is my risk based on?

- Age** 20 - 79
- Sex** Men
- Pop** European ancestry
- SNP** 2 genetic markers
rs4293393 (UMOD), rs7805747 (PRKAG2)

Genes vs. Environment

Although the relative contributions of genetic and non-genetic risk factors for CKD have not been definitively established, there is a clear familial component as having a family member with CKD increases a person's risk of getting the disease. Markers of kidney function—which are used to diagnose CKD—are estimated to be 27-33% heritable, suggesting that environmental risk factors may play a larger role than genetics in determining a person's risk for declining kidney function. The most common causes of CKD are diabetes and high blood pressure, which are responsible for up to two-thirds of CKD cases. Environmental risk factors for CKD include smoking and exposure to certain medications or environmental toxins. However, diabetes and high blood pressure are responsible for up to two-thirds of CKD cases. Other risk factors for CKD include heart disease, high cholesterol, obesity, older age, male gender, and ethnicity.

Additional Information

Other Medical Conditions

[Diabetes and hypertension](#) are the most common causes of CKD. If you have diabetes or high blood pressure, your health care provider may work with you to manage these conditions and lower your risk of CKD.

Screening and Risk Assessment

The [National Kidney Foundation](#) recommends CKD screening if you have any of the following risk factors:

- Diabetes
- High blood pressure
- A family history of CKD
- Older age

Lifestyle Factors

- **Maintain a healthy weight:** [Obesity](#) is associated with increased risk for CKD.
- **Don't smoke:** [Smoking](#) increases the risk of getting CKD and the risk for progression once you have it.

Environmental Factors

Repeated exposure to certain [toxins](#) such as lead, some drugs such as [non-steroidal anti-inflammatory pain medications](#), and certain classes of [antibiotics](#) can cause damage to the kidneys that can lead to CKD. If you are concerned about CKD, talk with your health care provider about your usage of these pain medications and antibiotics.

View the full report online for links to resources, references, and more detailed genetic results and information.

Restless Legs Syndrome

Imagine what it would be like to crawl into bed every night, ready to catch some much-needed Zs, only to be struck by an irrepressible urge to move your legs as soon as you began to relax. No matter how tired you were, instead of drifting off peacefully, you would be compelled to get up and move around. It may sound crazy, but this is exactly the situation people with restless legs syndrome (RLS) experience. Though the symptoms in many people are milder, it is estimated that about 4% of the U.S. population suffers from this puzzling disorder.

Tero's Genetic Risk

2.5%

Tero's risk of developing Restless Legs Syndrome between the ages specified



2.0%

Chance that the average person will develop Restless Legs Syndrome

What is my risk based on?

- Age** 0 - 79
- Sex** Men
- Pop** European ancestry
- SNP** 1 genetic markers
rs3923809 (BTBD9)

Genes vs. Environment

The heritability of restless legs syndrome is estimated to be 54%. This means that genetic and environmental factors contribute nearly equally to differences in risk for this condition. Genetic factors that play a role in restless legs syndrome include both unknown factors and known factors such as the SNPs we describe here. Environmental factors include pregnancy. Low iron levels, dialysis for end-stage renal disease, and damage to the nerves of the hands and feet tend to worsen the condition.

Additional Information

Other Medical Conditions

[Chronic diseases](#) such as kidney failure, diabetes, Parkinson's, and peripheral neuropathy can exacerbate symptoms of RLS. If you have RLS, your health care provider may work with you to manage these conditions to reduce your symptoms. [Pregnancy](#) can sometimes trigger symptoms of RLS. If this happens, the symptoms will usually disappear once the pregnancy is completed.

Lifestyle Factors

- **Limit caffeine, alcohol, and tobacco use:** Caffeine, alcohol, and tobacco intake can trigger or aggravate symptoms in predisposed individuals.
- **Get enough iron:** Insufficient iron levels can also trigger or aggravate symptoms.

Medications and Treatment

Taking certain drugs can sometimes cause symptoms of RLS. These symptoms usually disappear once the drug regimen is stopped. Your health care provider can work with you to manage drug regimens that may be triggering RLS.

View the full report online for links to resources, references, and more detailed genetic results and information.

Carrier status: Hemochromatosis (HFE-related)

Iron, an essential mineral, is absorbed via the intestines from food and is important for many bodily functions including red blood cell formation and proper brain function. The iron absorption process must be tightly regulated or else iron can accumulate in the body, possibly causing organ damage. Inherited forms of iron overload, known as hereditary hemochromatosis (HH), are caused by mutations in genes that normally play important roles in regulating iron levels. This report includes three mutations in the HFE gene that are typically found in people with European ancestry and are responsible for most cases of HH. HFE-related HH is inherited in a recessive manner, meaning that a person must receive a mutated copy of the HFE gene from each parent to have the condition. In Europeans, roughly one in 300 individuals has HFE-related HH and at least one in 10 carries a mutation for the condition. Rates are even higher in certain European populations including Irish, Norwegian and Australian. HFE-related HH is much rarer in Asian and African populations.

Tero's Genetic Results

Variant Present

Has one mutation in the HFE gene linked to hemochromatosis. A person with one of these mutations is not typically prone to higher levels of iron in the body, but can pass the mutation to offspring. May have other mutations in the HFE gene (not reported here).

Gene	Variant	DNA change	Tero's genotype
HFE	C282Y	G to A	AG
Markers tested: 3 Coverage: Up to 90%			

What does this test cover?

There are several forms of hereditary hemochromatosis (HH). The most common form is caused by mutations in the HFE gene, of which more than 20 have been documented. 23andMe reports data for the three HFE mutations most commonly linked to hereditary hemochromatosis: the severe C282Y mutation and the milder H63D and S65C mutations.

How is Hemochromatosis (HFE-related) inherited?

HFE-related hemochromatosis is inherited in a recessive manner, meaning that only a child who receives two mutated copies of the HFE gene (one from each parent) is at risk of developing the disease.

How common is this condition?

HFE-related hereditary hemochromatosis is fairly common. Roughly 10-30% of people with European ancestry carry one of the three HFE mutations reported here. About one in 300 individuals has two HFE mutations and is at risk for iron overload; however, only a small fraction of individuals with two mutations go on to develop symptoms.

Additional Information

Other Risk Factors

Men with two mutated copies of the HFE gene are more likely to develop symptoms than pre-menopausal women due to the fact that women eliminate iron through menstruation, pregnancy, and childbirth. Advancing age also raises the likelihood of developing symptoms in those with two mutations. Experts recommend avoiding iron supplements and advise against taking vitamin C supplements or consuming vitamin C-rich juices with meals, as vitamin C aids in the absorption of iron. Alcohol can worsen liver damage in people with hemochromatosis.

Other Medical Conditions

Hemochromatosis can lead to liver disease, arthritis, heart problems, and diabetes. Alcohol can worsen liver damage in people with hemochromatosis.

Medications and Treatment

Hemochromatosis is treatable and health complications can be avoided if caught early and managed properly through lifestyle modifications. Blood removal on a regular basis (just like donating blood) is the standard treatment. If you are concerned about hereditary hemochromatosis, please consult your health care provider or a genetic counselor.

View the full report online for links to resources, references, and more detailed genetic results and information.

Drug response: Warfarin (Coumadin®) Sensitivity

Each time a doctor writes a prescription for warfarin (Coumadin®), a blood thinner given to about two million people each year in the United States, it's a guessing game. There is no "right" dose of the drug. Everyone is different and it can take weeks of adjustment to find a patient's optimal amount of the medication. Too much puts the patient at risk for bleeding. Too little can lead to clots and in turn, heart attack, stroke or even death. A patient's optimal dose depends not only on age, size, other medications and even diet, but also to a large extent on genetics.

Tero's Genetic Results



Increased warfarin sensitivity. May require decreased warfarin dose.

Marker	Tero's Genotype
rs1799853	CT
rs1057910	AA
rs9923231	TT
Markers tested: 3 Genotype combination: CYP2C9 *1/*2, VKORC1 -1639/3673 AA	

What does this test cover?

Several genes involved in warfarin metabolism play prominent roles in the variable response to warfarin. 23andMe tests for two variants in the CYP2C9 gene (*2, defined using rs1799853, and *3, defined using rs1057910) that are associated with reduced ability to break down warfarin. 23andMe also tests for a variant near the VKORC1 gene (rs9923231) that is associated with increased sensitivity to the drug. [Read more about the genetics.](#)

Additional Information

Other Risk Factors

Many other clinical and demographic factors affect the optimal warfarin dose for an individual, including age, sex, weight, alcohol consumption, smoking status, ethnicity, vitamin K intake, and other medications. Other genetic variations in other genes (not reported here) can also impact a person's response to warfarin. Only a medical professional can determine the optimal dose for an individual.

Medications and Treatment

Warfarin can interact with other medications, including some antibiotics, non-steroidal anti-inflammatory drugs, some antidepressants, cholesterol medications, and chemotherapy drugs. If you are taking one of these drugs, your health care provider can help devise appropriate treatment plans.

View the full report online for links to resources, references, and more detailed genetic results and information.

Drug response: Sulfonylurea Drug Clearance (Type 2 Diabetes Treatment)

Sulfonylurea drugs are commonly used to treat type 2 diabetes, a disease that affects tens of millions of people in the U.S. Genetic as well as non-genetic factors can influence how a person responds to these drugs. This report covers two genetic variants associated with the ability to clear sulfonylurea drugs from the body. Decreased drug clearance can result in better chances for successful treatment but may also increase the risk of side effects.

Sulfonylurea drugs include glyburide (sold as DiaBeta®, Micronase®, and Glynase®), glimepiride (sold as Amaryl®), and glipizide (sold as Glucotrol®).

Tero's Genetic Results

Reduced

Somewhat reduced ability to clear sulfonylurea drugs from the body.

Marker	Tero's Genotype
rs1799853	CT
rs1057910	AA
Markers tested: 2 Genotype combination: CYP2C9 *1/*2	

What does this test cover?

23andMe tests for two variants in the CYP2C9 gene associated with decreased clearance of sulfonylurea drugs. These variants, called *2 (T at rs1799853) and *3 (C at rs1057910), result in reduced clearance of these drugs. Other genetic factors can also influence response to sulfonylurea drugs.

Additional Information

View the full report online for links to resources, references, and more detailed genetic results and information.

Drug response: Phenytoin (Dilantin®) Sensitivity (Epilepsy Drug)

Epilepsy is a neurological condition characterized by seizures. One of the most common epilepsy treatments in the United States is the drug phenytoin (sold as Dilantin®). People with certain versions of the CYP2C9 gene are less able to metabolize the drug. They remove the drug from their body more slowly. Consequently, these people may be at higher risk for serious side effects and may need lower drug doses. This report covers two variants of CYP2C9 associated with increased phenytoin sensitivity: CYP2C9*2 and CYP2C9*3.

Tero's Genetic Results

Increased

Slightly increased sensitivity to phenytoin. May require slightly lower dose.

Marker	Tero's Genotype
rs1799853	CT
rs1057910	AA

Markers tested: 2 Genotype combination: CYP2C9 *1/*2

What does this test cover?

Several genes involved in phenytoin metabolism play prominent roles in the variable response to phenytoin. 23andMe tests for two variants in the CYP2C9 gene (*2, defined using rs1799853, and *3, defined using rs1057910) that are associated with reduced ability to break down phenytoin. [Read more about the genetics.](#)

Additional Information

Other Risk Factors

Many other clinical and demographic factors, such as age and alcohol consumption, affect the optimal phenytoin dose for an individual. Other genetic variations in other genes (not reported here) can also impact a person's response to phenytoin. Only a medical professional can determine the optimal dose for an individual.

Medications and Treatment

Phenytoin can interact with many other medications, including nonsteroidal anti-inflammatory drugs (e.g., aspirin and ibuprofen) and warfarin. Make sure your healthcare provider knows all the medications you take so that they can help devise appropriate treatment plans.





















































View the full report online for links to resources, references, and more detailed genetic results and information.







Tero Keski-Valkama's results for all conditions tested by 23andMe

Conditions and diseases tested by 23andMe: This list is continually expanding as new genetic associations are discovered and reported. Please visit our website at <https://www.23andme.com/health/all/> to view the most up-to-date list of conditions tested by 23andMe.

About Risk Estimates:

23andMe reports results as genotype-specific incidence, which is an estimate of how many individuals in a population composed of people with a customer's genotype are expected to be diagnosed with a condition given a specified ancestry and age range. These estimates are based on well-established genetic associations reported in the biomedical literature and do not account for non-genetic factors, family history, or additional genetic factors that may modify a customer's risk. The genotype-specific incidence estimate combines the odds for a condition for a customer's genotypes at a set of SNPs with data about disease incidence. For more information on how 23andMe calculates these estimates, please see our technical papers available at <https://www.23andme.com/howitworks/>.

Disease risk (31)	Your risk	Average risk
Atrial Fibrillation	  46.9%	27.2%
Venous Thromboembolism	  17.9%	12.3%
Colorectal Cancer	  9.0%	5.6%
Chronic Kidney Disease	  5.0%	3.4%
Restless Legs Syndrome	  2.5%	2.0%
Ulcerative Colitis	  1.00%	0.77%
Multiple Sclerosis	  0.47%	0.34%
Esophageal Squamous Cell Carcinoma (ESCC)	  0.43%	0.36%
Celiac Disease	  0.40%	0.12%
Stomach Cancer (Gastric Cardia Adenocarcinoma)	  0.28%	0.23%
Bipolar Disorder	  0.15%	0.10%
Breast Cancer	  Typical risk	
Gallstones	  Typical risk	
Gout	  Typical risk	
Lung Cancer	  Typical risk	
Lupus (Systemic Lupus Erythematosus)	  Typical risk	
Obesity	  Typical risk	
Parkinson's Disease	  Typical risk	
Primary Biliary Cirrhosis	  Typical risk	
Prostate Cancer	  Typical risk	
Scleroderma (Limited Cutaneous Type)	  Typical risk	
Type 2 Diabetes	  Typical risk	
Age-related Macular Degeneration	  Decreased risk	
Alzheimer's Disease	  Decreased risk	
Coronary Heart Disease	  Decreased risk	
Crohn's Disease	  Decreased risk	

Exfoliation Glaucoma	  Decreased risk
Melanoma	  Decreased risk
Psoriasis	  Decreased risk
Rheumatoid Arthritis	  Decreased risk
Type 1 Diabetes	  Decreased risk

About Carrier Status:

23andMe tests for specific genetic variants that are strongly linked to a number of inherited genetic conditions. These variants are typically the most common ones linked to the condition. Certain variants may be more common in certain populations than others. The absence of specific variants does not rule out the possibility that a customer may carry another variant linked to the condition.

Carrier status (53)	Status
Hemochromatosis (HFE-related)	Variant Present
ARSACS	Variant Absent
Agensis of the Corpus Callosum with Peripheral Neuropathy (ACCPN)	Variant Absent
Alpha-1 Antitrypsin Deficiency	Variant Absent
Autosomal Recessive Polycystic Kidney Disease	Variant Absent
BRCA Cancer Mutations (Selected)	Variant Absent
Beta Thalassemia	Variant Absent
Bloom's Syndrome	Variant Absent
Canavan Disease	Variant Absent
Congenital Disorder of Glycosylation Type 1a (PMM2-CDG)	Variant Absent
Connexin 26-Related Sensorineural Hearing Loss	Variant Absent
Cystic Fibrosis	Variant Absent
D-Bifunctional Protein Deficiency	Variant Absent
DPD Deficiency	Variant Absent
Dihydrolipoamide Dehydrogenase Deficiency	Variant Absent
Factor XI Deficiency	Variant Absent
Familial Dysautonomia	Variant Absent
Familial Hypercholesterolemia Type B	Variant Absent
Familial Hyperinsulinism (ABCC8-related)	Variant Absent
Familial Mediterranean Fever	Variant Absent
Fanconi Anemia (FANCC-related)	Variant Absent
G6PD Deficiency	Variant Absent
GRACILE Syndrome	Variant Absent
Gaucher Disease	Variant Absent
Glycogen Storage Disease Type 1a	Variant Absent
Glycogen Storage Disease Type 1b	Variant Absent
Hereditary Fructose Intolerance	Variant Absent
Hypertrophic Cardiomyopathy (MYBPC3 25bp-deletion)	Variant Absent

LAMB3-related Junctional Epidermolysis Bullosa	Variant Absent
Leigh Syndrome, French Canadian Type (LSFC)	Variant Absent
Limb-girdle Muscular Dystrophy	Variant Absent
Maple Syrup Urine Disease Type 1B	Variant Absent
Medium-Chain Acyl-CoA Dehydrogenase (MCAD) Deficiency	Variant Absent
Mucopolipidosis IV	Variant Absent
Neuronal Ceroid Lipofuscinosis (CLN5-related)	Variant Absent
Neuronal Ceroid Lipofuscinosis (PPT1-related)	Variant Absent
Niemann-Pick Disease Type A	Variant Absent
Nijmegen Breakage Syndrome	Variant Absent
Pendred Syndrome	Variant Absent
Phenylketonuria	Variant Absent
Primary Hyperoxaluria Type 2 (PH2)	Variant Absent
Rhizomelic Chondrodysplasia Punctata Type 1 (RCDP1)	Variant Absent
Salla Disease	Variant Absent
Sickle Cell Anemia & Malaria Resistance	Variant Absent
Sjögren-Larsson Syndrome	Variant Absent
TTR-Related Cardiac Amyloidosis	Variant Absent
TTR-Related Familial Amyloid Polyneuropathy	Variant Absent
Tay-Sachs Disease	Variant Absent
Torsion Dystonia	Variant Absent
Tyrosinemia Type I	Variant Absent
Usher Syndrome Type I (PCDH15-related)	Variant Absent
Usher Syndrome Type III	Variant Absent
Zellweger Syndrome Spectrum	Variant Absent

About Drug Response:

23andMe displays your likely response to a number of drugs based on genetic variants associated with differences in response. These may be differences in sensitivity, in the likelihood or severity of side effects, or differences in disease risk tied to use of a drug. Only a medical professional can determine whether a drug is right for a particular patient. The information contained in this report should not be used to independently establish a drug regimen, or abolish or adjust an existing course of treatment.

Drug response (11)	Response
Warfarin (Coumadin®) Sensitivity	Increased
Sulfonylurea Drug Clearance (Type 2 Diabetes Treatment)	Reduced
Phenytoin (Dilantin®) Sensitivity (Epilepsy Drug)	Increased
Abacavir Hypersensitivity	Typical
Alcohol Consumption, Smoking and Risk of Esophageal Cancer	Typical
Clopidogrel (Plavix®) Efficacy	Typical
Fluorouracil Toxicity	Typical

Oral Contraceptives, Hormone Replacement Therapy and Risk of Venous Thromboembolism	Not Applicable
Pseudocholinesterase Deficiency	Typical
Response to Hepatitis C Treatment	Typical
Thiopurine Methyltransferase Deficiency	Typical

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About the 23andMe Personal Genome Service®

23andMe's Personal Genome Service provides customers with data on nearly 1,000,000 single nucleotide polymorphisms (SNPs) in their genome using a microarray-based genotyping assay. Customers provide saliva samples, which are analyzed by a CLIA-certified laboratory. Results are viewable on the 23andMe website at <https://www.23andme.com/you/> where reports are considered Established or Preliminary Research reports depending on the amount of evidence supporting the associations reported. We currently provide more than 60 Established Research reports on various disease risk, drug response, and carrier status topics, as well as Preliminary Research reports on more than 150 conditions and traits.