Please note that this is a sample report and not a complete report of the test.

Mike, this is your health report

Index

1. Introduction

In this report you will see some of your genetic predispositions related to health.

As usual in our studies, in the first pages you will find an iconographic summary of each of the analyzed values, which we develop more broadly in later pages.

The report is organized in these sections.

1.1. Methodology that we use for your report

Genetic Health Risks: Gwas

In this part we apply GWAS publications, a type of study that compares the DNA markers of people with a disease or trait, to people without this disease or traits. These studies can be very useful for prevention and early diagnosis; it is not a diagnostic tool but it helps us to see in what we have to care a bit more.

The data that will give us, when applying these studies to your genetic information, is your predisposition comparing to the rest of the population. At no time does it mean that you are going to suffer the disease, it only indicates that statistically and according to this study you could have some more predisposition than the majority of the population; we indicate that you have greater predisposition when it is greater than ninety percent of the population, and smaller if your predisposition is less than ninety percent of the population.

It is important to keep in mind that complex diseases are influenced by many factors; genetic ones are only a part; lifestyle, food, etc. are in many cases the most influential factors

Genetic Health Risk: Mutations

In this section we analyze the mutations of the most important genes from the oncological point of view. We look for mutations suspected of being pathogenic, specifically those reported as pathogenic in the ClinVar database.

It is important to note that this test does not sequence the entire genome, we only analyze 700,000 of the 3.2 billion genetic links so, in the case that we do not find any mutation, that does not mean that we are not carriers, since it can be in genetic regions that we are not analyzing. In this section we analyze a small percentage of the genes classified as pathogenic in the consulted databases, so there could be pathogenic mutations in a region that we can not see in this test.

Carrier Status

Hereditary diseases are likely to be passed on to your offspring. In most of the cases we can be carriers and never suffer the disease, but there is a risk that our offspring will suffer if certain conditions. They are mostly monogenic diseases.

In this group we are looking for pathogenic mutations, or likely pathogenic mutations, in the genes involved in these diseases. We look for the mutations that are reported in some of the most important genetic databases worldwide, basically OMIM and ClinVar.

As in the previous section, we do not analyze all the genetic information related to each disease, specifically in this section we were able to analyze on average something less than half of the pathogenic markers reported in the databases consulted (ClinVar), so we could have mutations in the other half and not see them in this report.

If you need a diagnosis of a particular disease, there are genetic tests, which analyze the entire gene or genes involved in given disease, and they are valid for clinical use. If you have a family background related to a disease we recommend that you go to your doctor or geneticist to study the need for this type of test. The results of this report are personal, not applicable to studies on other members of your family.

Biomarkers, biometrics and traits

In this section we use, again, the GWAS statistical analysis to calculate your genetic predisposition to have abnormal levels of certain metabolic parameters.

As in the rest of our GWAS studies, we indicate that you have a greater predisposition when it is greater than ninety percent of the population, and lower if your predisposition is lower than that of ninety percent of the population. Due to the statistical distribution of this analysis, it is normal that several parameters will appear as high or low predisposition.

Pharmacogenomics

In this section we study your genetic predisposition towards certain medications. Depending on the drug, your genetics can affect the level of toxicity, the effectiveness of ore dose needed. Something that a doctor always has to supervise.

The results of this report are personal, and not applicable to studies about other members of your family.

These reports, as well as the scientific research in the genetics field, may vary over time. New mutations are constantly being discovered and we know better the ones we are analyzing today. We make a great effort to periodically apply the consolidated scientific discoveries to our reports.

We remind you that any changes you want to make regarding your health should be guided by your doctor.

1.2. Frequently Asked Questions

If this report shows that I have a genetic predisposition to a specific disease, am I going to suffer it for sure?

Not at all, the genetic reports that we do are based on statistics. You may have genetic predisposition to a particular disease and never develop it, actually it is what it happends in most of the cases . Or you may not have a predisposition to a disease and suffer it in the future. Genetic analysis is just one more tool, the doctors and specialized health proffesionals the ones who must make the interpretation of the available set of health data.

Should I make drastic changes in my health management with the data of this test?

No at all, any changes you want to make in your health management should be analyzed by an expert geneticist and the medical specialists. Any doubts you have about any genetic test should be checked by healthcare experts in Genetic Diagnosis.

Does it all depend on my genes?

No at all, our body responds to many conditions. Our genes are certainly an important parameter. Lifestyle, sport, food, and many other circumstances influence our body. Knowing yourself certainly helps to treat our body in the most appropriate way. And this is what these genetic reports aren all about: more information.

Are all the analyzed genes listed in the sections?

We include most of the genes we analyze; in some sections we are analyzing more genes that we can't show due the to lack of space.

What is this report based on?

This test is based on different genetic studies internationally consolidated and accepted by the scientific community. There are certain scientific databases where studies are published where there is a certain level of consensus. Our genetic tests are carried out by applying these studies to the genotype of our clients. In each section you will see some of the studies publications on which it is based. There are sections where more studies are used than the ones listed.

If the report reflects that I have genetic mutations in an inherited disease, does that mean that I have that disease for sure?

No, we look for both pathogenic mutations and mutations that could be pathogenic (likely pathogenic); if you have any of these your report will indicate that we have detected it. On the other hand, this technology has a reliability greater than 99% but there is no 100% reliability in this type of genotyping technology. If you have any doubt you should talk to your doctor or geneticist.

If the report reflects that I DO NOT have genetic mutations in an inherited disease, does that mean I am free of this disease for sure?

No, our test does not analyze all the genetic zones where pathogenic mutations may exist and we

do not analyze the deletions, duplications or intergenic zones. We analyze only some markers reported as pathogenic. On average our test covers just under 50% of these markers for a given disease, so there could be pathogenic markers in the other half and we would not be seeing them. There are diagnostic tests with greater coverage in certain pathologies that are valid for clinical use. If you have any doubt you should talk to your doctor or geneticist.

If I am a carrier of a mutation of a hereditary disease, how does that affect my offspring?

Almost all of us are carriers of some mutations of monogenetic diseases, it is normal to find in a person between 5 and 50 significant genetic mutations. However, the risk that our offspring suffer the disease varies greatly depending on the type of inheritance: autosomal dominant, autosomal recessive, multifactorial ..., therefore we advise always to go to your doctor or geneticist. for advice.

2. Summary

Genetic Health Risks: Gwas

- Alopecia areata
- Rheumatoid arthritis
- Prostate cancer
- Prostate cancer (early onset)
- Upper aerodigestive tract cancers
- Motion sickness
- Age-related macular degeneration
- Type 1 diabetes
- Type 2 diabetes
- Alzheimer's disease (late onset)
- Parkinson's disease
- Systemic sclerosis
- Glioma
- Myocardial infarction (early onset)
- Hodgkin's lymphoma
- Follicular lymphoma
- Multiple myeloma
- Osteosarcoma
- Allergic sensitization
- Wilms tumor
- Intracranial aneurysm
- Chronic bronchitis and chronic obstructive pulmonary disease
- Prostate cancer aggressiveness
- Bladder cancer
- Basal cell carcinoma
- Primary biliary cirrhosis
- Conduct disorder
- Type 1 diabetes nephropathy
- Celiac disease
- Coronary heart disease
- Multiple sclerosis
- Schizophrenia
- Hypothyroidism
- Chronic lymphocytic leukemia
- Diffuse large B cell lymphoma
- Myasthenia gravis
- Neuroblastoma
- Psoriasis
- Testicular germ cell tumor
- Vitiligo

Caption:

- According to this study, you have a predisposition similar to most of the population.
- According to this study, you are less likely to suffer from this disease than the majority of the population.
- According to this study, you are more likely to suffer from this disease than most of the population.

Genetic Health Risks: mutations

- APC: colorectal and pancreatic cancer
- BRCA1: breast and ovarian cancer
- BRIP1: breast cancer
- CDKN2A: pancreatic cancer
- MLH1: Lynch syndrome
- MSH6: Lynch syndrome and colorectal cancer
- PALB2: breast and pancreatic cancer
- ATM: breast cancer
- BRCA2: breast and ovarian cancer
- CDH1: breast and gastric cancer
- CHEK2: breast and colorectal cancer
- MSH2: Lynch syndrome and colorectal cancer
- MUTYH: MYH-associated polyposis and colorectal cancer
- PMS2: Lynch syndrome and colorectal cancer

- PTEN: breast, uterine and colorectal cancer
- SMAD4: juvenile polyposis syndrome and colorectal cancer
- VHL: Von Hippel-Lindau syndrome
- SDHB: gastric cancer
- TP53: Li-Fraumeni syndrome, breast cancer and more
- RET: thyroid carcinoma

Caption:

- We have not detected any pathogenic mutation but, since we only analyze a part of the gene, you could have some pathogenic mutation in the non-analyzed genetic regions.
- We have detected at least one mutation that could be pathogenic.

Carrier Status

- 17-BETA HYDROXYSTEROID DEHYDROGENASE III DEFICIENCY
- AARSKOG-SCOTT SYNDROME; AAS
- LEUKEMIA, ACUTE MYELOID; AML
- HYPOPHOSPHATASIA, ADULT
- ALPHA-1-ANTITRYPSIN DEFICIENCY; A1ATD
- ANEMIA, NONSPHEROCYTIC HEMOLYTIC, DUE TO G6PD DEFICIENCY
- ANTITHROMBIN III DEFICIENCY; AT3D
- AURICULOCONDYLAR SYNDROME 1; ARCND1
- BARDET-BIEDL SYNDROME 1; BBS1
- BETA-THALASSEMIA
- BRUGADA SYNDROME 1; BRGDA1
- CARDIOMYOPATHY, DILATED, 1S; CMD1S
- CEROID LIPOFUSCINOSIS, NEURONAL, 1; CLN1
- CHARCOT-MARIE-TOOTH DISEASE, TYPE 4C; CMT4C
- GRANULOMATOUS DISEASE, CHRONIC, X-LINKED; CDGX
- NIGHT BLINDNESS, CONGENITAL STATIONARY, TYPE 1C; CSNB1C
- COSTELLO SYNDROME; CSTLO
- DANON DISEASE
- 3-METHYLCROTONYL-CoA CARBOXYLASE 2 DEFICIENCY; MCC2D
- ACHROMATOPSIA 2; ACHM2
- ADRENOLEUKODYSTROPHY; ALD
- ALLAN-HERNDON-DUDLEY SYNDROME; AHDS
- AMYLOIDOSIS, HEREDITARY, TRANSTHYRETIN-RELATED
- ANGELMAN SYNDROME; AS
- ARRHYTHMOGENIC RIGHT VENTRICULAR DYSPLASIA, FAMILIAL, 10; ARVD10
- HYPOPHOSPHATEMIC RICKETS, AUTOSOMAL DOMINANT; ADHR
- MUSCULAR DYSTROPHY, BECKER TYPE; BMD
- BLOOM SYNDROME; BLM
- CARDIOFACIOCUTANEOUS SYNDROME 1; CFC1
- CARDIOMYOPATHY, FAMILIAL HYPERTROPHIC, 1; CMH1
- CEROID LIPOFUSCINOSIS, NEURONAL, 7; CLN7
- CHONDRODYSPLASIA PUNCTATA 1, X-LINKED RECESSIVE; CDPX1
- ADRENAL HYPOPLASIA, CONGENITAL; AHC
- CORNELIA DE LANGE SYNDROME 1; CDLS1
- CYSTIC FIBROSIS; CF
- DEAFNESS, AUTOSOMAL RECESSIVE 1A; DFNB1A

- DEAFNESS, AUTOSOMAL RECESSIVE 31; DFNB31
- DEAFNESS, AUTOSOMAL RECESSIVE 7; DFNB7
- DEAFNESS, AUTOSOMAL RECESSIVE 9; DFNB9
- MANNOSIDOSIS, ALPHA B, LYSOSOMAL; MANSA
- CARDIOMYOPATHY, DILATED, 1A; CMD1A
- DUBIN-JOHNSON SYNDROME; DJS
- EPILEPTIC ENCEPHALOPATHY, EARLY INFANTILE, 2; EIEE2
- MYOCLONIC EPILEPSY OF LAFORA
- ERYTHROCYTOSIS, FAMILIAL, 2; ECYT2
- FABRY DISEASE
- FAMILIAL ADENOMATOUS POLYPOSIS 1; FAP1
- CARDIOMYOPATHY, FAMILIAL HYPERTROPHIC, 2; CMH2
- FAMILIAL MEDITERRANEAN FEVER; FMF
- THYROID CARCINOMA, FAMILIAL MEDULLARY; MTC
- FANCONI ANEMIA, COMPLEMENTATION GROUP O; FANCO
- NEPHROTIC SYNDROME, TYPE 1; NPHS1
- GAUCHER DISEASE, TYPE I
- GLUT1 DEFICIENCY SYNDROME 1; GLUT1DS1
- GLUTARIC ACIDEMIA I; GA1
- MULTIPLE ACYL-CoA DEHYDROGENASE DEFICIENCY; MADD
- GLYCOGEN STORAGE DISEASE Ia; GSD1A
- GLYCOGEN STORAGE DISEASE II; GSD2
- HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS, FAMILIAL, 2; FHL2
- HERMANSKY-PUDLAK SYNDROME 3; HPS3
- HISTIOCYTOSIS-LYMPHADENOPATHY PLUS SYNDROME
- ECTODERMAL DYSPLASIA 1, HYPOHIDROTIC, X-LINKED; XHED
- JERVELL AND LANGE-NIELSEN SYNDROME 1; JLNS1
- JOUBERT SYNDROME 14; JBTS14
- JOUBERT SYNDROME 16; JBTS16
- JOUBERT SYNDROME 3; JBTS3
- JOUBERT SYNDROME 5; JBTS5
- JOUBERT SYNDROME 7; JBTS7
- JOUBERT SYNDROME 8; JBTS8
- JOUBERT SYNDROME 9; JBTS9
- KABUKI SYNDROME 1; KABUK1
- LEIGH SYNDROME; LS
- LEOPARD SYNDROME 1; LPRD1
- LEUKOENCEPHALOPATHY WITH VANISHING WHITE MATTER; VWM
- LISSENCEPHALY 1; LIS1
- LOEYS-DIETZ SYNDROME 2; LDS2
- LONG QT SYNDROME 1; LQT1
- MAPLE SYRUP URINE DISEASE; MSUD
- MATURITY-ONSET DIABETES OF THE YOUNG, TYPE 2; MODY2
- MATURITY-ONSET DIABETES OF THE YOUNG, TYPE 3; MODY3
- MECKEL SYNDROME, TYPE 3; MKS3
- MENTAL RETARDATION AND MICROCEPHALY WITH PONTINE AND CEREBELLAR HYPOPLASIA; MICPCH
- METACHROMATIC LEUKODYSTROPHY; MLD
- METHYLMALONIC ACIDURIA AND HOMOCYSTINURIA, cblC TYPE
- METHYLMALONIC ACIDURIA, cblA TYPE
- METHYLMALONIC ACIDURIA, cblB TYPE

- MITOCHONDRIAL COMPLEX III DEFICIENCY, NUCLEAR TYPE 1; MC3DN1
- MUCOPOLYSACCHARIDOSIS TYPE VI; MPS6
- MUCOPOLYSACCHARIDOSIS, TYPE VII; MPS7
- MUCOPOLYSACCHARIDOSIS, TYPE IIIA; MPS3A
- MUCOPOLYSACCHARIDOSIS, TYPE IIIB; MPS3B
- MUCOPOLYSACCHARIDOSIS, TYPE IVA; MPS4A
- MUSCULAR DYSTROPHY-DYSTROGLYCANOPATHY (CONGENITAL WITH BRAIN AND EYE ANOMALIES), TYPE A, 1; MDDGA1
- MYOPATHY, MYOFIBRILLAR, 1; MFM1
- MYOPATHY, CENTRONUCLEAR, 1; CNM1
- MYOPATHY, CENTRONUCLEAR, X-LINKED; CNMX
- NEMALINE MYOPATHY 2; NEM2
- CYSTINOSIS, NEPHROPATHIC; CTNS
- NIEMANN-PICK DISEASE, TYPE C1; NPC1
- NIEMANN-PICK DISEASE, TYPE A
- NIEMANN-PICK DISEASE, TYPE B
- NOONAN SYNDROME 1; NS1
- NOONAN SYNDROME-LIKE DISORDER WITH OR WITHOUT JUVENILE MYELOMONOCYTIC LEUKEMIA; NSLL
- NOONAN SYNDROME 4; NS4
- Obesity due to melanocortin 4 receptor deficiency
- ALBINISM, OCULOCUTANEOUS, TYPE IB; OCA1B
- OSTEOGENESIS IMPERFECTA, TYPE III; OI3
- DIABETES MELLITUS, PERMANENT NEONATAL; PNDM
- PITT-HOPKINS SYNDROME; PTHS
- POLYMICROGYRIA, BILATERAL FRONTOPARIETAL; BFPP
- MICROCEPHALY 5, PRIMARY, AUTOSOMAL RECESSIVE; MCPH5
- RETINITIS PIGMENTOSA; RP
- RUBINSTEIN-TAYBI SYNDROME 1; RSTS1
- SOTOS SYNDROME 1; SOTOS1
- SUPRAVALVULAR AORTIC STENOSIS; SVAS
- TAY-SACHS DISEASE; TSD
- TUBEROUS SCLEROSIS 1; TSC1
- TUBEROUS SCLEROSIS 2; TSC2
- ALBINISM, OCULOCUTANEOUS, TYPE IA; OCA1A
- TYROSINEMIA, TYPE I; TYRSN1
- USHER SYNDROME, TYPE I; USH1
- USHER SYNDROME, TYPE ID; USH1D
- USHER SYNDROME, TYPE IF; USH1F
- USHER SYNDROME, TYPE IIA; USH2A
- USHER SYNDROME, TYPE IIC; USH2C
- USHER SYNDROME, TYPE IID; USH2D
- USHER SYNDROME, TYPE IIIA; USH3A
- ACYL-CoA DEHYDROGENASE, VERY LONG-CHAIN, DEFICIENCY OF; ACADVLD
- VON HIPPEL-LINDAU SYNDROME; VHL
- WEAVER SYNDROME; WVS
- Wilson Disease
- AGAMMAGLOBULINEMIA, X-LINKED; XLA

Caption:

- We have not detected any pathogenic mutation but, since we only analyze a part of the gene, you could have some pathogenic mutation in the non-analyzed genetic regions.
- We have detected at least one mutation that could be pathogenic.

Biomarkers

- Adiponectin levels
- Androgen levels in men
- Beta-2 microglubulin plasma levels
- Bilirubin levels
- C-reactive protein and white blood cell count
- Calcium levels
- Dehydroepiandrosterone sulphate levels
- Eosinophil counts
- Glycated hemoglobin levels
- Homocysteine levels
- IgE levels
- Liver enzyme levels (gamma-glutamyl transferase)
- Liver enzyme levels
- Magnesium levels
- Monocyte count
- Phospholipid levels (plasma)
- Phosphorus levels
- Plasma omega-6 polyunsaturated fatty acid levels (dihomo-gamma-linolenic acid)
- Platelet count
- Red blood cell count
- Serum albumin level
- Sex hormone levels
- Thyroid hormone levels
- Uric acid levels
- Urinary uromodulin levels
- Vitamin B levels in ischemic stroke
- White blood cell count

Caption:

- According to this study, you have a similar predisposition to the majority of the population to have normal levels.
- According to this study, you have a better predisposition than the majority of the population to have normal levels.
- According to this study, you have a greater predisposition than most of the population to suffer abnormal levels.

Biometrics

- Aortic root size
- Bone mineral density
- Heart rate
- Resting heart rate

Caption:

- According to this study, you have a similar predisposition to the majority of the population to have normal levels.
- According to this study, you have a better predisposition than the majority of the population to have normal levels.
- According to this study, you have a greater predisposition than most of the population to suffer abnormal levels.

Traits

- Alcoholism (alcohol dependence factor score)
- Spirometric measure of pulmonary function (Forced vital capacity)
- Smoking behavior

Caption:

- According to this study, you have a predisposition similar to most of the population.
- According to this study, you have less predisposition than the majority of the population.
- According to this study, you have a greater predisposition than the majority of the population.

Pharmacogenomics: Cardiology

- Pravastatin
- Simvastatin

- Warfarin

Caption:

- We have not found anything in your genetics that indicates a predisposition to an abnormal effect of this drug. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype you are more predisposed to have an abnormal effect on you. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype you are more predisposed to have harmful effects on you. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype, you have a greater predisposition to respond positively to this drug. Other non-analyzed and non-genetic genetic factors may play a role.

Pharmacogenomics: Neurology

- Amitriptyline
- Antidepressants
- Bupropion

Caption:

- We have not found anything in your genetics that indicates a predisposition to an abnormal effect of this drug. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype you are more predisposed to have an abnormal effect on you. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype you are more predisposed to have harmful effects on you. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype, you have a greater predisposition to respond positively to this drug. Other non-analyzed and non-genetic genetic factors may play a role.

Pharmacogenomics: Oncology

- Methotrexate
- Vincristine
- Fluorouracil, capecitabine, pyrimidine analogues, tegafur and Neoplasms

Caption:

- We have not found anything in your genetics that indicates a predisposition to an abnormal effect of this drug. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype you are more predisposed to have an abnormal effect on you. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype you are more predisposed to have harmful effects on you. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype, you have a greater predisposition to respond positively to this drug. Other non-analyzed and non-genetic genetic factors may play a role.

Pharmacogenomics: Other

- Tacrolimus
- Sildenfail (Viagra)

Caption:

- We have not found anything in your genetics that indicates a predisposition to an abnormal effect of this drug. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype you are more predisposed to have an abnormal effect on you. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype you are more predisposed to have harmful effects on you. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype, you have a greater predisposition to respond positively to this drug. Other non-analyzed and non-genetic genetic factors may play a role.

Pharmacogenomics: Pain

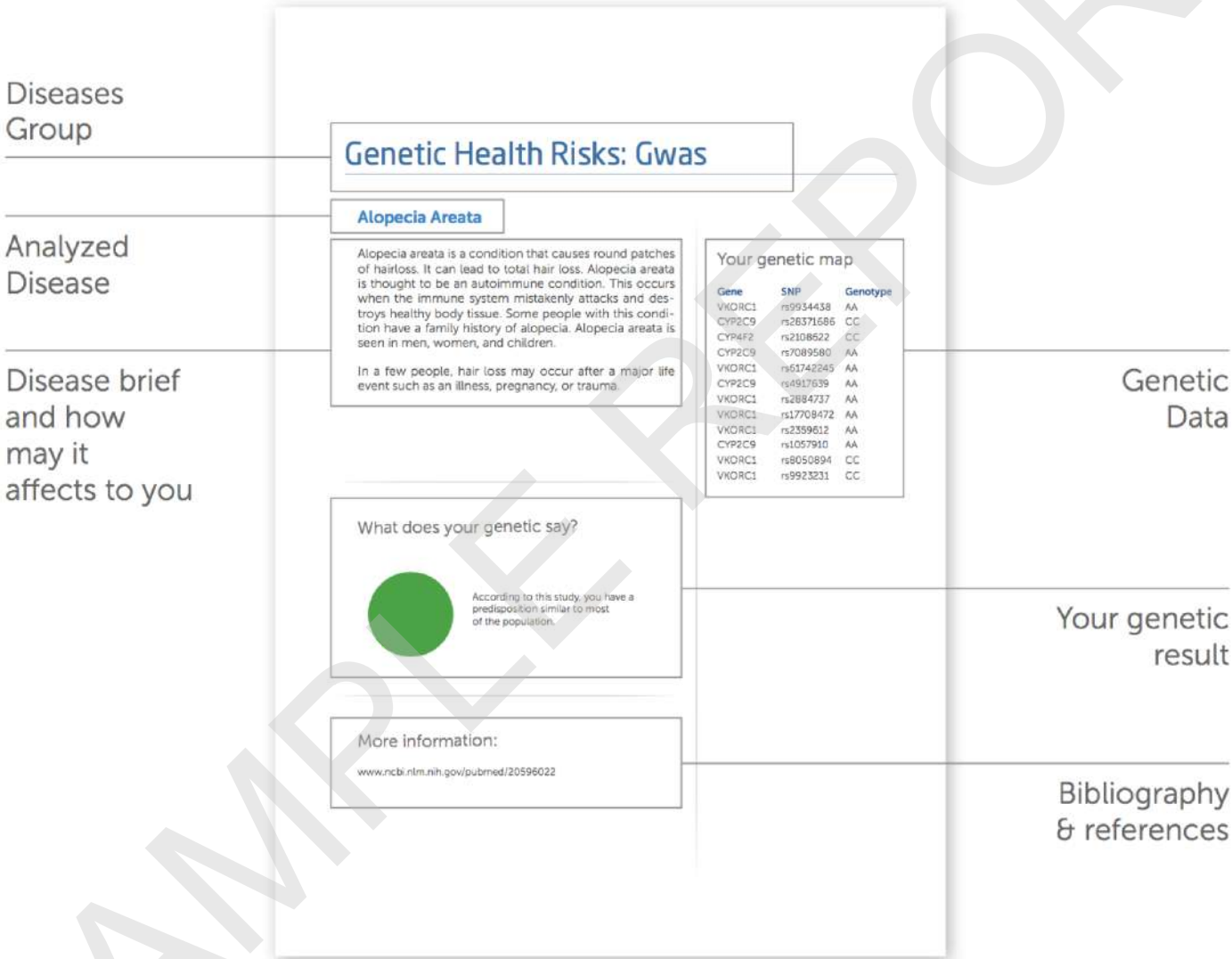
- Meperidine
- Morphine
- Pentazocine
- Aspirin

Caption:

- We have not found anything in your genetics that indicates a predisposition to an abnormal effect of this drug. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype you are more predisposed to have an abnormal effect on you. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype you are more predisposed to have harmful effects on you. Other non-analyzed and non-genetic genetic factors may play a role.
- According to your genotype, you have a greater predisposition to respond positively to this drug. Other non-analyzed and non-genetic genetic factors may play a role.

3. Genetic Results

3.1. What information is included in the results?



3.2. Your genetic results

Genetic Health Risks: Gwas

Alopecia areata

Alopecia areata is a condition that causes round patches of hair loss. It can lead to total hair loss.

Alopecia areata is thought to be an autoimmune condition. This occurs when the immune system mistakenly attacks and destroys healthy body tissue.

Some people with this condition have a family history of alopecia. Alopecia areata is seen in men, women, and children. In a few people, hair loss may occur after a major life event such as an illness, pregnancy, or trauma.

Your genetic map

Gene	SNP	Genotype
ICOS,	rs1024161	TC
IL2, IL21	rs7682241	TG
ULBP3,	rs9479482	TC
IL2RA	rs3118470	TT
PRDX5	rs694739	GG
IKZF4	rs1701704	TT
HLA-	rs9275572	GG

What does your genetics say?

According to this study, you have a predisposition similar to most of the population.

More information:

www.ncbi.nlm.nih.gov/pubmed/20596022

Genetic Health Risks: Gwas

Motion sickness

Motion sickness is a common problem in people traveling by car, train, airplanes, and especially boats. Anyone can get it, but it is more common in children, pregnant women, and people taking certain medicines. Motion sickness can start suddenly, with a queasy feeling and cold sweats. It can then lead to dizziness and nausea and vomiting. Your brain senses movement by getting signals from your inner ears, eyes, muscles, and joints. When it gets signals that do not match, you can get motion sickness. For example, if you are reading on your phone while riding a bus, your eyes are focused on something that is not moving, but your inner ear senses motion. Despite their high heritability, no associated genetic factors have been discovered. This section is based on a genome association study on motion sickness in 80,494 individuals who were surveyed about this pathology.

What does your genetics say?

According to this study, you have a predisposition similar to most of the population.

More information:

www.ncbi.nlm.nih.gov/pubmed/25628336

Your genetic map

Gene	SNP	Genotype
PVRL3	rs66800491	GG
GPD2	rs56051278	AG
ACO1	rs10970305	CC
AUTS2	rs1195218	GG
GPR26	rs705145	CC
CBLN4	rs6069325	TT
MUTED	rs2153535	GG
LINGO2	rs2150864	AG
CPNE4	rs9834560	AA
RWDD3	rs1858111	AA
PRDM16	rs61759167	CC
NLGN1	rs11713169	AC
HOXD	rs2551802	GG
COPS8	rs2318131	AA
TLE4	rs149951341	AA
HOXB	rs9906289	CC
ST18	rs2360806	AC
SDK1	rs4343996	AG
NR2F2	rs7170668	TC
CELF2	rs10752212	AA
CNTN1	rs7957589	AA
MCTP2	rs62018380	CC
ARAP2	rs6833641	CC
AUTS2	rs6946969	AG
RGS5	rs4076764	TC
MAP2K5	rs997295	TT
AGA	rs1378552	TT
POU6F2	rs60464047	AT
TUSC1	rs1782032	AG
GXYLT2	rs1847202	TT
SDK1	rs34912216	

Genetic Health Risks: mutations

BRCA1: breast and ovarian cancer

Mutations of the BRCA1 gene may be related to diseases such as breast and ovarian cancer. There is some study that relates this gene, on a smaller scale, to other cancers such as colon or pancreatic cancer.

What does your genetics say?

We have not detected any pathogenic mutation but, since we only analyze a part of the gene, you could have some pathogenic mutation in the non-analyzed genetic regions. In this panel we are analyzing less than 10 % of the pathogenic mutations reported in ClinVar.

More information:

https://www.ncbi.nlm.nih.gov/pubmed/7907678

Your genetic map

Gene	SNP	Genotype
BRCA1	rs62625308	GG
BRCA1	rs28897686	CC
BRCA1	rs41293455	GG
BRCA1	rs62625306	CC
BRCA1	rs80357382	TT
BRCA1	rs80358158	CC
BRCA1	rs80356898	GG
BRCA1	rs80357355	TT
BRCA1	rs80358061	AA
BRCA1	rs80358163	TT
BRCA1	rs80357233	GG
BRCA1	rs80356875	CC
BRCA1	rs80356925	GG
BRCA1	rs80357251	CC
BRCA1	rs80357115	AA
BRCA1	rs397507215	GG
BRCA1	rs80357018	CC
BRCA1	rs80357318	GG
BRCA1	rs80357021	CC
BRCA1	rs80358178	CC
BRCA1	rs80358070	CC
BRCA1	rs80357259	CC
BRCA1	rs80356991	CC
BRCA1	rs80358027	CC
BRCA1	rs80357389	CC
BRCA1	rs80356988	CC
BRCA1	rs80356988	CC
BRCA1	rs80357433	GG
BRCA1	rs80358086	AA
BRCA1	rs80358053	CC
BRCA1	rs80358053	CC

Genetic Health Risks: mutations

CDH1: breast and gastric cancer

Mutations of the CDH1 gene may be related to diseases such as breast and gastric cancer. There is some study linking this gene, on a smaller scale, to ovarian and colon cancer.

Your genetic map

Gene	SNP	Genotype
CDH1	rs587780784	CC
CDH1	rs587780787	GG
CDH1	rs587782750	CC
CDH1	rs587782798	CC
CDH1	rs587783047	CC
CDH1	rs587783050	GG
CDH1	rs730881663	CC

What does your genetics say?

We have not detected any pathogenic mutation but, since we only analyze a part of the gene, you could have some pathogenic mutation in the non-analyzed genetic regions. In this panel we are analyzing less than 25 % of the pathogenic mutations reported in ClinVar.

More information:

https://www.ncbi.nlm.nih.gov/pubmed/11729114

Carrier Status

17-BETA HYDROXYSTEROID DEHYDROGENASE III DEFICIENCY

17-beta-hydroxysteroid dehydrogenase isozyme 3 (17betaHSD III) deficiency is a rare disorder leading to male pseudohermaphroditism (MPH), a condition characterized by incomplete differentiation of the male genitalia in 46X,Y males. The estimated incidence of this disease is 1 in 147 000 in The Netherlands. The 17betaHSD III enzyme catalyzes the conversion of androstenedione to testosterone in the testis. Lack of testosterone in the fetal testis leads to genetic males with female external genitalia. Patients usually present at birth with female or ambiguous external genitalia, characterized by clitoromegaly, posterior labioscrotal fusion and perineal blind vaginal pouch. Testes are inguinal or in the labioscrotal folds.

Your genetic map

Gene	SNP	Genotype
HSD17B3	rs119481077	GG

What does your genetics say?

We have not detected any pathogenic mutation, but you might have some in non analyzed genetic regions.

More information:

https://www.omim.org/entry/264300

Carrier Status

MICROCEPHALY 5, PRIMARY, AUTOSOMAL RECESSIVE; MCPH5

Autosomal recessive primary microcephaly (MCPH) is a rare genetically heterogeneous disorder of neurogenic brain development characterized by reduced head circumference at birth with no gross anomalies of brain architecture and variable degrees of intellectual impairment. Exact prevalence of non-syndromic microcephaly is not known. MCPH is more common in Asian and Middle Eastern populations than in Caucasians, in whom an annual incidence of 1/1,000,000 is reported. It is more common in specific populations, e.g. northern Pakistanis. Consanguinity appears to play a role in incidence.

What does your genetics say?

We have not detected any pathogenic mutation, but you might have some in non analyzed genetic regions.

More information:

https://www.omim.org/entry/608716

Your genetic map

Gene	SNP	Genotype
ASPM	rs199422146	
ASPM	rs137852997	AA
ASPM	rs140602858	GG
ASPM	rs199422165	GG
ASPM	rs199422134	GG
ASPM	rs199422189	GG
ASPM	rs587783220	II
ASPM	rs587783221	
ASPM	rs587783227	GG
ASPM	rs587783228	II
ASPM	rs587783230	AA
ASPM	rs587783238	CC
ASPM	rs587783239	II
ASPM	rs587783247	AA
ASPM	rs587783248	GG
ASPM	rs587783259	II
ASPM	rs587783268	GG
ASPM	rs587783269	II
ASPM	rs587783272	GG
ASPM	rs587783275	GG
ASPM	rs587783277	II
ASPM	rs587783278	II
ASPM	rs587783282	GG
ASPM	rs587783285	CC
ASPM	rs587783287	GG
ASPM	rs587783288	AA
ASPM	rs587783289	II
ASPM	rs759632528	DD
ASPM	rs199422147	II
ASPM	rs199422161	CC
ASPM	rs199422173	

Biomarkers

Homocysteine levels

Homocysteine (HC) is a sulfur amino acid important in the transfer of methyl groups in cell metabolism, this has been considered an influential factor in the development of cardiovascular and cerebrovascular diseases.

Recent studies have focused on the analysis of the relationship between hyperhomocysteinemia (increased plasma homocysteine concentration) and damage to neuronal cells; in neurotoxic mechanisms such as: increase of oxidative stress and generation of homocysteine derivatives as well as the increase in the toxicity of β-amyloid protein, among others.

Homocysteine is synthesized as an intermediate product of the metabolism of methionine by the action of the enzyme methionine adenosyl transferase.

What does your genetics say?

According to this study, you have a similar predisposition to the majority of the population to have normal levels.

More information:

www.ncbi.nlm.nih.gov/pubmed/23824729

Your genetic map

Gene	SNP	Genotype
MTHFR	rs1801133	GG
MTR	rs2275565	GG
EEF1A1P4	rs9369898	
NOX4	rs7130284	CC
DPEP1 -	rs154657	AG
CBS	rs234709	TC
PRDX1	rs4660306	TC
SLC17A3	rs548987	GG
LOC1079	rs42648	AG
RPL12P33	rs2251468	AA
FGF21	rs838133	AG
C1orf167,	rs12134663	
TRDMT1	rs12780845	AA
NOX4	rs957140	GG
CBS	rs2851391	TC

Biomarkers

Magnesium levels

Magnesium, potassium, and sodium, cations commonly measured in serum, are involved in many physiological processes including energy metabolism, nerve and muscle function, signal transduction, and fluid and blood pressure regulation.

Your genetic map

Gene	SNP	Genotype
MUC1	rs4072037	TC
SHROOM	rs13146355	GG
LOC1079	rs7965584	AA
LOC1019	rs3925584	TT
HOXD9 -	rs2592394	AG
MECOM	rs448378	GG

What does your genetics say?

According to this study, you have a similar predisposition to the majority of the population to have normal levels.

More information:

www.ncbi.nlm.nih.gov/pubmed/20700443

Traits

Spirometric measure of pulmonary function (Forced vital capacity)

Forced vital capacity (FVC), a spirometric measure of pulmonary function, reflects lung volume and is used to diagnose and monitor lung diseases.

Your genetic map

Gene	SNP	Genotype
EFEMP1	rs1430193	AA
BMP6	rs6923462	TC
MIR129-2	rs4237643	GG
PRDM11	rs2863171	AC
WWOX	rs1079572	GG

What does your genetics say?

According to this study, you have a predisposition similar to most of the population.

More information:

www.ncbi.nlm.nih.gov/pubmed/24929828

Traits

Smoking behavior

Consistent but indirect evidence has implicated genetic factors in smoking behavior.

Your genetic map

Gene	SNP	Genotype
HECTD2-	rs1329650	TG
RAB4B-	rs3733829	AA
BDNF,	rs6265	CC
FAM163B	rs3025343	GG

What does your genetics say?

According to this study, you have a predisposition similar to most of the population.

More information:

www.ncbi.nlm.nih.gov/pubmed/20418890

Pharmacogenomics: Cardiology

Pravastatin

Pravastatin is a cholesterol-lowering agent that belongs to a class of medications known as statins. It was derived from microbial transformation of mevastatin, the first statin discovered. It is a ring-opened dihydroxyacid with a 6'-hydroxyl group that does not require in vivo activation. Pravastatin is one of the lower potency statins; however, its increased hydrophilicity is thought to confer advantages such as minimal penetration through lipophilic membranes of peripheral cells, increased selectivity for hepatic tissues, and a reduction in side effects compared with lovastatin and simvastatin.

Your genetic map

Gene	SNP	Genotype
HMGCR	rs17244841	AA

What does your genetics say?

Patients with the AA genotype who are treated with statins may be more likely to respond as compared to patients with the AT or TT genotype. Other genetic and clinical factors may also influence a patient's response when treated with statins.

More information:

https://www.ncbi.nlm.nih.gov/pubmed/15199031

Pharmacogenomics: Cardiology

Warfarin

Warfarin is an anticoagulant drug normally used to prevent blood clot formation as well as migration. Although originally marketed as a pesticide (d-Con, Rodex, among others), Warfarin has since become the most frequently prescribed oral anticoagulant in North America. Warfarin has several properties that should be noted when used medicinally, including its ability to cross the placental barrier during pregnancy which can result in fetal bleeding, spontaneous abortion, preterm birth, stillbirth, and neonatal death. Additional adverse effects such as necrosis, purple toe syndrome, osteoporosis, valve and artery calcification, and drug interactions have also been documented with warfarin use. Warfarin does not actually affect blood viscosity, rather, it inhibits vitamin-k dependent synthesis of biologically active forms of various clotting factors in addition to several regulatory factors.

Your genetic map

Gene	SNP	Genotype
VKORC1	rs9923231	TT

What does your genetics say?

Patients with the TT genotype may require a lower dose of warfarin as compared to patients with the CC or TC genotype. Other genetic and clinical faTCors may also influence a patient's warfarin dose requirement.

More information:

https://www.ncbi.nlm.nih.gov/gtr/conditions/CN078029

Pharmacogenomics: Other

Tacrolimus

Tacrolimus (also FK-506 or Fujimycin) is an immunosuppressive drug whose main use is after organ transplant to reduce the activity of the patient's immune system and so the risk of organ rejection. It is also used in a topical preparation in the treatment of severe atopic dermatitis, severe refractory uveitis after bone marrow transplants, and the skin condition vitiligo. It was discovered in 1984 from the fermentation broth of a Japanese soil sample that contained the bacteria Streptomyces tsukubaensis. Tacrolimus is chemically known as a macrolide. It reduces peptidyl-prolyl isomerase activity by binding to the immunophilin FKBP-12 (FK506 binding protein) creating a new complex. This FKBP12-FK506 complex interacts with and inhibits calcineurin thus inhibiting both T-lymphocyte signal transduction and IL-2 transcription.

Your genetic map

Gene	SNP	Genotype
CYP3A4	rs2740574	TT

What does your genetics say?

Transplant recipients with the TT (CYP3A4 genotype may require a decreased dose of tacrolimus as compared to patients with the TC or CC genotype. Other genetic and clinical faTCors, such as CYP3A5 (rs776746), may also influence a patient's dose requirements.

More information:

https://www.ncbi.nlm.nih.gov/pubmed/23778326

Pharmacogenomics: Other

Sildenfail (Viagra)

Sildenfail is a vasoactive agent used to treat erectile dysfunction and reduce symptoms in patients with pulmonary arterial hypertension (PAH). Sildenafil elevates levels of the second messenger, cGMP, by inhibiting its breakdown via phosphodiesterase type 5 (PDE5). PDE5 is found in particularly high concentrations in the corpus cavernosum, erectile tissue of the penis. It is also found in the retina and vascular endothelium. Increased cGMP results in vasodilation which facilitates generation and maintenance of an erection.

Your genetic map

Gene	SNP	Genotype
GNB3	rs5443	CC

What does your genetics say?

Patients with the CC genotype and erectile dysfunction who are treated with sildenafil may be less likely to have positive erectile response as compared to patients with the TT genotype. Other genetic and clinical factors may also influence a patient's response to sildenafil.

More information:

https://www.ncbi.nlm.nih.gov/pubmed/12576843

Pharmacogenomics: Pain

Morphine

The principal alkaloid in opium and the prototype opiate analgesic and narcotic. Morphine has widespread effects in the central nervous system and on smooth muscle. In January, 2017, morphine was approved for the treatment of chronic pain.

Your genetic map

Gene	SNP	Genotype
CREB1	rs2952768	TC

What does your genetics say?

Patients with the TC genotype may have decreased opioid analgesic requirements after surgery as compared to patients with the CC genotype. Other genetic and clinical factors may influence a patient's opioid dose requirement.

More information:

https://www.ncbi.nlm.nih.gov/pubmed/23183491

Pharmacogenomics: Pain

Aspirin

Aspirin, also known as acetylsalicylic acid (ASA), is a medication used to treat pain, fever, or inflammation. Specific inflammatory conditions in which aspirin is used include Kawasaki disease, pericarditis, and rheumatic fever. Aspirin is a nonsteroidal anti-inflammatory drug (NSAID) and works similar to other NSAIDs but also suppresses the normal functioning of platelets.

Your genetic map

Gene	SNP	Genotype
PTGS1	rs10306114	AA

What does your genetics say?

Patients with the AA genotype who are treated with aspirin may have a decreased, but not absent, risk for non-response to aspirin as compared to patients with the AG or GG genotype. Other genetic and clinical factors may also influence a patient's response to aspirin.

More information:

https://www.ncbi.nlm.nih.gov/pubmed/16493486