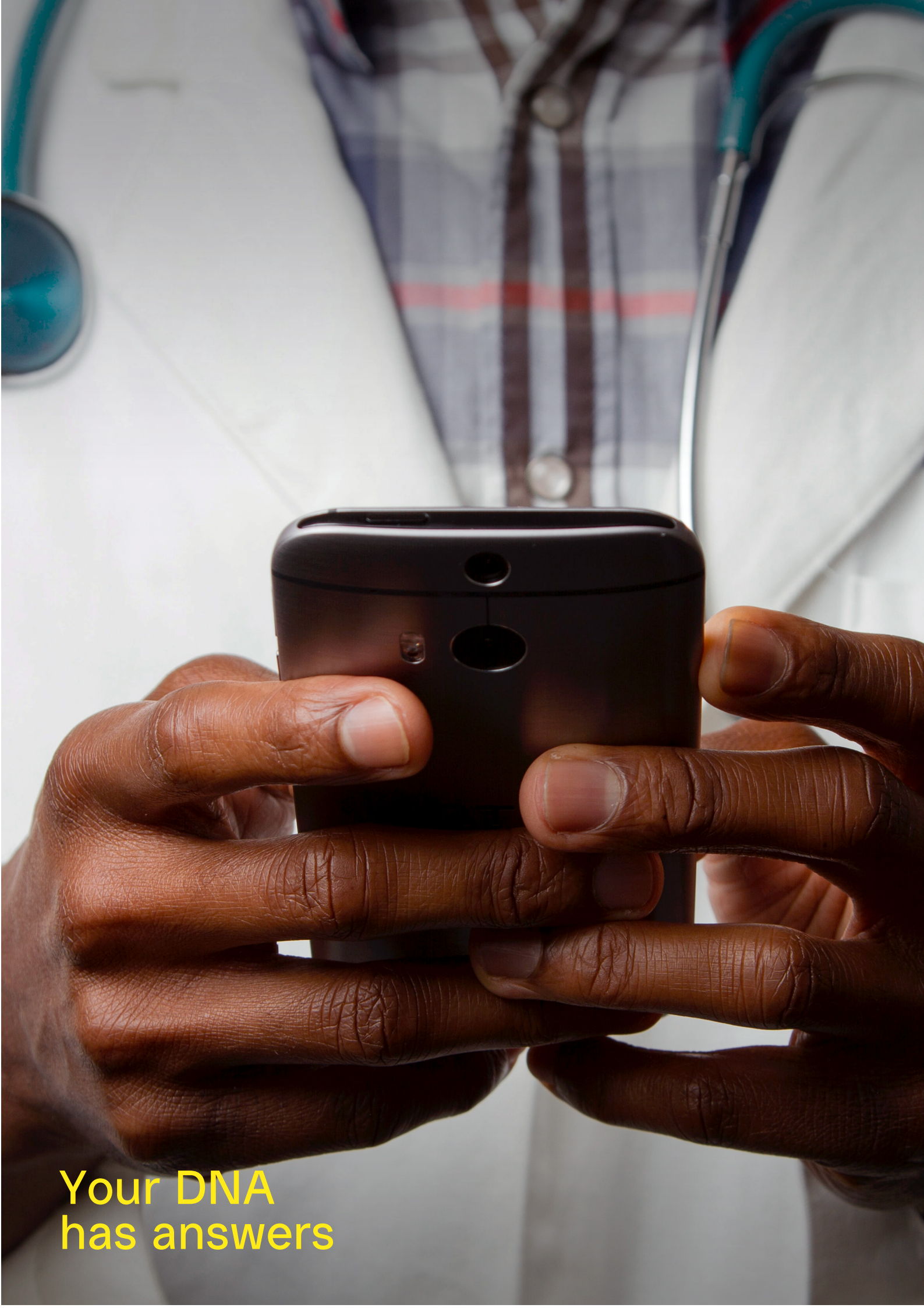
Adnà

Mike, here is your
Genetic Predispositions Report


Your DNA
has answers

1. Introduction

In the following pages, we offer you the Health Report obtained from the analysis of your DNA. In addition, you will find information about your genetic predispositions in several areas of your health (diseases, allergies, viruses, biomarkers, etc.).

Here are some essential aspects to take into account before reading this report.

The process with which we obtain your personalized report

The process followed by Adnà's partner laboratory to prepare your health report consists of:

1. Extracting DNA from the saliva sample you sent us.

2. Transforming the biological data contained in DNA into bioinformatics data. This process is called sequencing.

3. Applying the algorithms developed exclusively by Adnà's analysis partner, 24Genetics, to this computer data, which allows us to obtain your personalized report.

As you can see, we combine purely biological processes with computer processes. Without losing an iota of scientific rigor, we can process vast amounts of information and offer you such detailed reports.

What does our algorithm look like?

The algorithms developed by Adnà's sequencing partner are based on the analysis and study of thousands of reports and papers that are compared, validated and recognized by the scientific community at an international level and that add value to our reports.

Thanks to the reliability of our ancestry test, the first step in our genetic analysis is to identify the sex and ancestry of each individual. From there, we exclusively apply the proper studies for each profile whenever it is possible to do so. To obtain the genetic report of a European woman, we do not usually use, for example, studies whose analyzed population has been exclusively male or Asian. At this point, we could apply a single analysis, but we combine a multitude of validated publications, refining the process with artificial intelligence. By doing so we can use all available scientific knowledge to calculate genetic predispositions.

With this, we gain accuracy and reliability in our results.

Methodology

Our genetic reports are obtained based on three types of analysis methodology:

- **GWAS** (Genome-Wide Association Study). This is a type of study in which DNA markers in the whole genome (a person's complete genetic material) of people with a disease or trait are compared with those of people who do not have that disease or feature. It is a study based on statistics, which considers many genes associated with a predisposition in a less direct way, but whose sum offers a relevant conclusion.

- **Multivariate** analysis. In this case, our algorithm analyzes several genetic variants or mutations of one or several genes, which correlate more directly with the predisposition.

- **Monovariate** analysis. In this type of methodology, it is a single variant of a single gene that determines the predisposition due to its strong correlation with the genotype.

Each trait analyzed in this report is based on one of these three types of methodology.

The data and conclusions in this report, like the progress of scientific research in genetics, may evolve. New mutations are continually being discovered and we are learning more about the modifications we are analysing today. At Adnà and 24Genetics we make a great effort to apply new and consolidated scientific findings to our reports.

2. Summary

GWAS Complex Diseases: Neurology

- Parkinson's disease
- Motion sickness
- Multiple sclerosis
- Neuroblastoma
- Glioma
- Intracranial aneurysm
- Alzheimer's disease (late onset)
- Schizophrenia
- Conduct disorder

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.

GWAS Complex Diseases: Circulatory System

- Primary biliary cirrhosis
- Myocardial infarction (early onset)
- Hodgkin's lymphoma
- Follicular lymphoma
- Coronary heart disease
- Chronic lymphocytic leukemia
- Diffuse large B cell lymphoma
- Wilms tumor

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.

GWAS Complex Diseases: Respiratory System

- Upper aerodigestive tract cancers
- Asthma
- Chronic bronchitis and chronic obstructive pulmonary disease

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.

GWAS Complex Diseases: Musculoskeletal System

- Systemic sclerosis
- Rheumatoid arthritis
- Myasthenia gravis
- Osteosarcoma
- Multiple myeloma

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.

GWAS Complex Diseases: Endocrinology

- Type 1 diabetes
- Type 2 diabetes
- Type 1 diabetes nephropathy
- Hypothyroidism

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.

GWAS Complex Diseases: Urogenital System

- Testicular germ cell tumor
- Prostate cancer aggressiveness
- Bladder cancer
- Prostate cancer
- Prostate cancer (early onset)

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.

GWAS Complex Diseases: Dermatology

- Basal cell carcinoma
- Vitiligo
- Psoriasis
- Androgenetic alopecia

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.

GWAS Complex Diseases: Others

- Celiac disease
- Age-related macular degeneration

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.

Complex Diseases: Oncogenic Mutations

- APC: colorrectal and pancreatic cancer
- BARD1: breast cancer
- BMPR1A: colorrectal, gastric and pancreatic cancer
- BRCA2: breast and ovarian cancer
- CDH1: breast and gastric cancer
- CDKN2A: pancreatic cancer
- ATM: breast cancer
- BLM: colorrectal cancer
- BRCA1: breast and ovarian cancer
- BRIP1: breast cancer
- CDK4: Familial melanoma
- CHEK2: breast and colorrectal cancer

- DICER1: ovarian cancer
- EPCAM: Lynch syndrome, breast, ovarian, uterine, colorrectal, gastric and pancreatic cancer
- FH: Hereditary leiomyomatosis and renal cell cancer
- FLCN: Kidney cancer
- MEN1: multiple endocrine neoplasia type 1
- MET: Lung and gastric cancer
- MITF: MITF-related melanoma and renal cell carcinoma predisposition syndrome
- MLH1: Lynch syndrome
- MSH2: Lynch syndrome and colorrectal cancer
- MSH6: Lynch syndrome and colorrectal cancer
- MUTYH: colorrectal cancer
- NBN: breast, ovarian, colorrectal and gastric cancer
- NF1: type 1 neurofibromatosis
- NF2: Familial multiple meningioma
- NTHL1: Attenuated familial adenomatous polyposis
- RAD50: breast and pancreatic cancer
- PMS2: Lynch syndrome and colorrectal cancer
- POLD1: breast, ovarian, uterine and colorrectal cancer
- POLE: ovarian, uterine, colorrectal andpancreatic cancer
- MSH3-related attenuated familial adenomatous polyposis
- POT1: Familial melanoma
- PTCH1: Basal cell carcinoma
- PTEN: breast, uterine and colorrectal cancer
- RB1: Lynch syndrome and retinoblastoma
- RECQL4: Stomach and colon cancer
- RET: thyroid carcinoma
- SDHA: gastric cancer
- SDHAF2: Hereditary pheochromocytoma-paraganglioma
- SDHB: gastric cancer
- SDHC: gastric cancer
- SDHD: breast, uterine and gastric cancer
- BAP1-related tumor predisposition syndrome
- SMAD4: juvenile polyposis syndrome and colorrectal cancer
- SMARCB1: Familial rhabdoid tumor
- SMARCE1: Familial multiple meningioma
- STK11: breast, ovarian, uterine, colorrectal, gastric and pancreatic cancer
- TERT: Familial melanoma
- TP53: Li-Fraumeni syndrome, breast cancer and more
- VHL: Von Hippel-Lindau syndrome
- WT1: Nephroblastoma
- Familial adenomatous polyposis
- Kenny-Caffey syndrome

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.

Complex Diseases: Multivariate Analysis

- Septic shock
- TSC1: tuberous sclerosis complex 1
- TSC2: tuberous sclerosis complex 2

- Bethlem myopathy
- Distal myopathy with anterior tibial onset
- Progressive scapulohumeroperoneal distal myopathy
- Hereditary myopathy with early respiratory failure
- Multiminicore myopathy
- Inclusion body myopathy with Paget disease of bone and frontotemporal dementia
- MODY
- Mucolipidosis type III
- Mucopolysaccharidosis type 2
- Mucopolysaccharidosis type 4
- Mucopolysaccharidosis type 7
- Mitochondrial membrane protein-associated neurodegeneration
- Neurofibromatosis-Noonan syndrome
- Autosomal recessive axonal neuropathy with neuromyotonia
- Autosomal recessive severe congenital neutropenia due to CSF3R deficiency
- Woolly hair nevus
- Obesity due to melanocortin 4 receptor deficiency
- Hypertrichotic osteochondrodysplasia, Cantu type
- Osteopetrosis with renal tubular acidosis
- Osteosarcoma
- Non-acquired panhypopituitarism
- Pachyonychia congenita
- Paramyotonia congenita of Von Eulenburg
- Autosomal dominant spastic paraplegia type 17
- Autosomal dominant spastic paraplegia type 8
- Autosomal recessive spastic paraplegia type 35
- Miyoshi myopathy
- Laing early-onset distal myopathy
- GNE myopathy
- Mitochondrial myopathy with reversible cytochrome C oxidase deficiency
- Severe congenital nemaline myopathy
- Potassium-aggravated myotonia
- MPI-CDG
- Mucopolysaccharidosis type 1
- Mucopolysaccharidosis type 3
- Mucopolysaccharidosis type 6
- Multiple endocrine neoplasia type 2
- Neurofibromatosis type 6
- Navajo neurohepatopathy
- Leber hereditary optic neuropathy
- Autosomal recessive severe congenital neutropenia due to JAGN1 deficiency
- Obesity due to leptin receptor gene deficiency
- Autosomal recessive progressive external ophthalmoplegia
- Multiple osteochondromas
- Albers-Schönberg osteopetrosis
- Hereditary chronic pancreatitis
- Pachydermoperiostosis
- Hypokalemic periodic paralysis
- Autosomal dominant spastic paraplegia type 10
- Autosomal dominant spastic paraplegia type 31
- Autosomal recessive spastic paraplegia type 15
- Autosomal recessive spastic paraplegia type 54

- Congenital nephrotic syndrome, Finnish type
- Oculocerebrofacial syndrome, Kaufman type
- Orofaciodigital syndrome type 14
- Orofaciodigital syndrome type 5
- Tumor necrosis factor receptor 1 associated periodic syndrome
- SHORT syndrome
- NPHP3-related Meckel-like syndrome
- Larsen-like syndrome, B3GAT3 type
- Spondylocarpotarsal synostosis
- Deafness with labyrinthine aplasia, microtia, and microdontia
- Microcephalic cortical malformations-short stature due to RTTN deficiency
- Hereditary hemorrhagic telangiectasia
- 46,XY disorder of sex development due to 17-beta-hydroxysteroid dehydrogenase 3 deficiency
- Lethal acantholytic erosive disorder
- Familial progressive cardiac conduction defect
- Nijmegen breakage syndrome-like disorder
- Severe primary trimethylaminuria
- Congenital amegakaryocytic thrombocytopenia
- Severe hereditary thrombophilia due to congenital protein C deficiency
- Desmoid tumor
- Familial cold urticaria
- STING-associated vasculopathy with onset in infancy
- Cerebrotendinous xanthomatosis
- PRUNE1-related neurological syndrome
- Oculocerebrorenal syndrome of Lowe
- Orofaciodigital syndrome type 4
- Otopalatodigital syndrome type 2
- RAPADILINO syndrome
- Congenital intrauterine infection-like syndrome
- Wolfram-like syndrome
- Triple A syndrome
- Sitosterolemia
- Short stature due to GHSR deficiency
- Catecholaminergic polymorphic ventricular tachycardia
- Tyrosinemia type 1
- TELO2-related intellectual disability-neurodevelopmental disorder
- ITPA-related lethal infantile neurological disorder with cataract and cardiac involvement
- Noonan syndrome-like disorder with juvenile myelomonocytic leukemia
- Carney triad
- Glanzmann thrombasthenia
- Paris-Trousseau thrombocytopenia
- Hereditary thrombophilia due to congenital antithrombin deficiency
- Testicular seminomatous germ cell tumor
- Vasculitis due to ADA2 deficiency
- Hereditary xanthinuria
- Xeroderma pigmentosum

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.

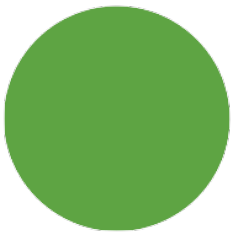
GWAS Complex Diseases: Neurology

Parkinson's disease

Parkinson's Disease (PD) is a type of movement disorder. It happens when nerve cells in the brain don't produce enough of a brain chemical called dopamine. Sometimes it is genetic, but most cases do not seem to run in families. Exposure to chemicals in the environment might play a role. Symptoms begin gradually, often on one side of the body. Later they affect both sides. Genetics is shedding new light on the disease, with the identification of several genes and markers associated with family forms; although these represent just 5 to 10% of cases, their study is key to the knowledge of the disease.

GWAS analysis

What do your genetics tell us?



According to this study, you are less likely to suffer from this disease than most of the population.

More information:

www.ncbi.nlm.nih.gov/pubmed/25064009

Your genetic map

Gene	SNP	Genotype
GBA	rs35749011	GG
NUCKS1	rs823118	TT
SIPA1L2	rs10797576	CC
ACMSD	rs6430538	TT
MCCC1	rs12637471	AG
FAM47E	rs6812193	TC
LOC10537	rs356182	AG
HLA DQB	rs9275326	TC
GPNMB	rs199347	GG
MIR4697	rs329648	TT
LRRK2	rs76904798	CC
CCDC62	rs11060180	GG
GCH1	rs11158026	TC
LOC10798	rs2414739	AA
BCKDK	rs14235	AG
RIT2	rs12456492	AG
SPPL2B	rs62120679	CC

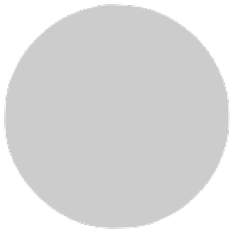
GWAS Complex Diseases: Neurology

Motion sickness

Motion sickness is a common problem in people traveling by car, train, airplanes and boats, especially. Anyone can suffer it, but it is more common in children, pregnant women, and people taking certain medicines. Motion sickness can start suddenly, causing a queasy feeling and cold sweats. It can then lead to dizziness, nausea and vomiting. Your brain senses movement by getting signals from your inner ears, eyes, muscles, and joints. When it receives signals that do not match, you can suffer from 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 its 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.

GWAS analysis

What do your genetics tell us?



According to this study, you have a propensity similar to that of 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
LINC0124	rs10970305	CC
AUTS2	rs1195218	GG
LINC0264	rs705145	AC
CBLN4	rs6069325	TT
MUTED	rs2153535	GC
LINGO2	rs2150864	AG
CPNE4	rs9834560	AA
LOC10192	rs1858111	AG
PRDM16	rs61759167	TC
NLGN1	rs11713169	AA
HOXD3	rs2551802	GC
COPS8 DT	rs2318131	AC
TLE4	rs149951341	AA
HOXB3	rs9906289	CC
ST18	rs2360806	AA
SDK1	rs4343996	AG
LINC0092	rs7170668	TC
CELF2	rs10752212	AG
PDZRN4	rs7957589	AA
MCTP2	rs62018380	CC
ARAP2	rs6833641	CC
AUTS2	rs6946969	AG
MAP2K5	rs997295	TG
AGA	rs1378552	TT
POU6F2	rs60464047	AT
LINC0124	rs1782032	GG
GXYLT2	rs1847202	TT
SDK1	rs34912216	AG

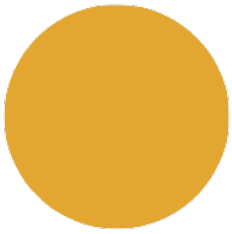
GWAS Complex Diseases: Neurology

Alzheimer's disease (late onset)

Alzheimer's Disease (AD) is the most common form of dementia among older people. Dementia is a brain disorder that seriously affects a person's ability to carry out daily activities. AD begins slowly. It first involves the parts of the brain that control thought, memory and language. People with AD may have trouble remembering things that happened recently, or names of people they know. A related problem, Mild Cognitive Impairment (MCI), causes more memory problems than normal for people of the same age. Many, but not all, people with MCI will develop AD. This section analyses the predisposition to Late-Onset Alzheimer's.

GWAS analysis

What do your genetics tell us?



According to this study, you are more likely to suffer from this disease than most of the population.

More information:

www.ncbi.nlm.nih.gov/pubmed/24162737

Your genetic map

Gene	SNP	Genotype
CR1	rs6656401	GG
LOC10537	rs6733839	TT
CD2AP	rs10948363	AG
EPHA1	rs11771145	GG
CLU	rs9331896	TT
MS4A6A	rs983392	AG
PICALM	rs10792832	AG
INPP5D	rs35349669	TT
MEF2C	rs190982	AA
NME8	rs2718058	AA
ZCWPW1	rs1476679	TT
CELF1	rs10838725	TT
FERMT2	rs17125944	TC
CASS4	rs7274581	TT
HLA DRB5	rs9271192	AA
PTK2B	rs28834970	TC
SORL1	rs11218343	TT
SLC24A4	rs10498633	GG
SQSTM1	rs72807343	CC
LOC10798	rs9381040	TC
CD33	rs3865444	CC

Complex Diseases: Oncogenic Mutations

NTHL1: Attenuated familial adenomatous polyposis

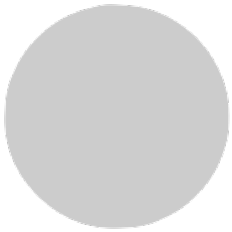
Mutations of the NTHL1 gene may be related to diseases such as familial adenomatous polyposis and colorectal cancer. In addition, some studies associated this gene, to a lesser extent, with other cancers, such as breast cancer.

Your genetic map

Gene	SNP	Genotype
NTHL1	rs146347092	GG
NTHL1	rs779757251	CC

Multivariate analysis

What do your genetics tell us?



We have not detected any pathogenic mutations, but, since we only analyse a part of the gene, you could have a pathogenic mutation in non-analysed genetic regions.

More information:

https://www.ncbi.nlm.nih.gov/gene/4913

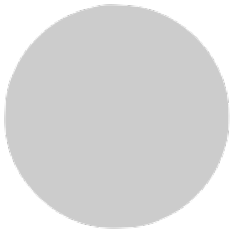
Complex Diseases: Oncogenic Mutations

PMS2: Lynch syndrome and colorrectal cancer

PMS2 gene mutations may be related to diseases such as Lynch Syndrome and colorrectal cancer.

Multivariate analysis

What do your genetics tell us?



We have not detected any pathogenic mutations, but, since we only analyse a part of the gene, you could have a pathogenic mutation in non-analysed genetic regions.

More information:

http://www.orpha.net/consor/cgi-bin/OC_Exp.php?lng=en&Expert=144

Your genetic map

Gene	SNP	Genotype
PMS2	rs63751466	GG
PMS2	rs63750451	GG
PMS2	rs121434629	CC
PMS2	rs267608158	AA
PMS2	rs587778617	GG
PMS2	rs63750490	TT
PMS2	rs63751422	GG
PMS2	rs201451115	TT
PMS2	rs267608172	CC
PMS2	rs587779338	GG
PMS2	rs587779343	GG
PMS2	rs267608153	CC
PMS2	rs200640585	GG
PMS2	rs587780062	GG
PMS2	rs587780064	CC
PMS2	rs587778618	GG
PMS2	rs587780724	GG
PMS2	rs587781339	TT
PMS2	rs730881919	CC
PMS2	rs863224450	CC
PMS2	rs876659736	TT
PMS2	rs1064794577	CC
PMS2	rs1064794083	AA
PMS2	rs988423880	CC
PMS2	rs1458321358	GG
PMS2	rs63750871	GG
PMS2	rs267608161	CC
PMS2	rs63750261	GG
PMS2	rs587779347	TT
PMS2	rs141577476	GG
PMS2	rs786201047	GG

Hereditary Diseases (genetics)

Carnitine palmitoyltransferase II deficiency

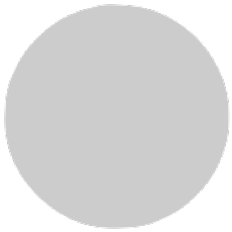
Carnitine palmitoyltransferase II (CPT II) deficiency is an inherited metabolic disorder that affects mitochondrial oxidation of long chain fatty acids (LCFA). Three forms of CPT II deficiency have been described: a myopathic form, a severe infantile form and a neonatal form (see these terms).

Your genetic map

Gene	SNP	Genotype
CPT2	rs28936375	CC
CPT2	rs74315295	TT
CPT2	rs74315296	CC

Multivariate analysis

What do your genetics tell us?



We have not detected any pathogenic mutations, but, since we only analyse a part of the gene, you could have a pathogenic mutation in non-analysed genetic regions.

More information:

http://www.orpha.net/consor/cgi-bin/OC_Exp.php?lng=en&Expert=157

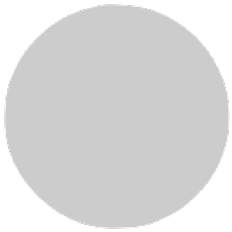
Hereditary Diseases (genetics)

Pendred syndrome

A syndromic genetic deafness clinically variable characterized by bilateral sensorineural hearing loss and euthyroid goiter.

Multivariate analysis

What do your genetics tell us?



We have not detected any pathogenic mutations, but, since we only analyse a part of the gene, you could have a pathogenic mutation in non-analysed genetic regions.

More information:

http://www.orpha.net/consor/cgi-bin/OC_Exp.php?lng=en&Expert=705

Your genetic map

Gene	SNP	Genotype
SLC26A4	rs80338848	TT
SLC26A4	rs80338849	GG
SLC26A4	rs111033244	AA
SLC26A4	rs121908363	CC
SLC26A4	rs111033307	TT
SLC26A4	rs111033348	CC
SLC26A4	rs111033199	GG
SLC26A4	rs111033254	TT
SLC26A4	rs397516414	GG
SLC26A4	rs111033305	GG
SLC26A4	rs111033311	GG
SLC26A4	rs397516416	CC
SLC26A4	rs397516418	TT
SLC26A4	rs111033316	AA
SLC26A4	rs111033312	GG
SLC26A4	rs111033257	GG
SLC26A4	rs397516424	AA
SLC26A4	rs111033318	TT
SLC26A4	rs111033256	TT
SLC26A4	rs397516432	TT
SLC26A4	rs111033454	GG
SLC26A4	rs111033245	GG
SLC26A4	rs727503430	GG
SLC26A4	rs727503431	CC
SLC26A4	rs542620119	GG
SLC26A4	rs146281367	GG
SLC26A4	rs876657722	GG
SLC26A4	rs147952620	CC
SLC26A4	rs111033200	CC
SLC26A4	rs111033302	TT
SLC26A4	rs397516430	CC

Adnà

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